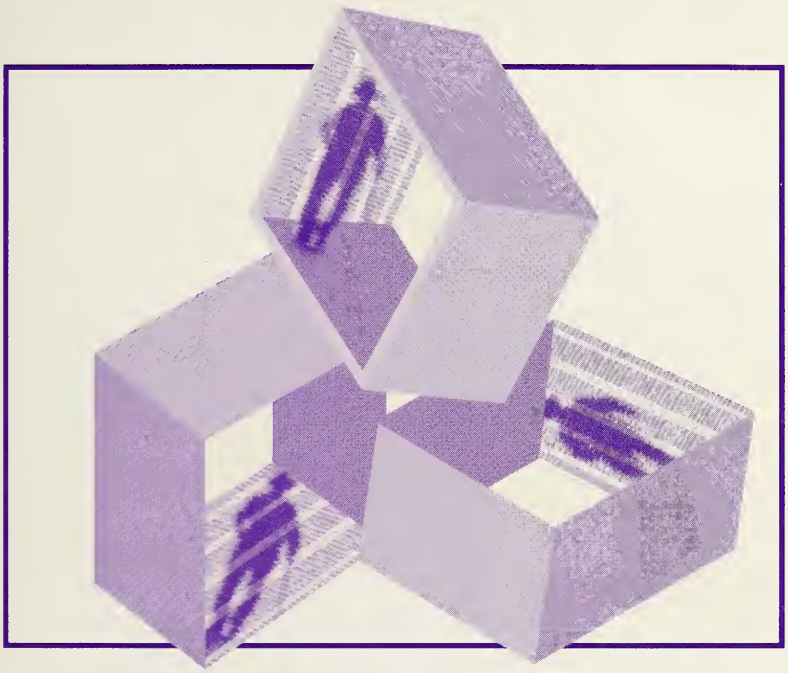
National Institute on Alcohol Abuse and Alcoholism

RESEARCH MONOGRAPH - 33



Alcohol Problems and Aging

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
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Research Monograph No. 33

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Edited by:
Edith S. Lisansky Gomberg, Ph.D.
Andrea M. Hegedus, Ph.D.
Robert A. Zucker, Ph.D.

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Keywords: elderly; aging; AODD (alcohol or other drug use disorder); AODE (effects of alcohol or other drug use, abuse, and dependence); AODR disorder (alcohol or other drug related disorder); biochemical mechanism; disease course; treatment; intervention; AOD prevention.

These descriptors are drawn from *The Alcohol and Other Drug Thesaurus: A Guide to Concepts and Terminology in Substance Abuse and Addiction, Second Edition, 1995* and may be used to retrieve this monograph in the Alcohol and Alcohol Problems Science Database (commonly known as ETOH).

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FOREWORD

One of our main tasks in the National Institute on Alcohol Abuse and Alcoholism (NIAAA) is to identify populations at risk for alcohol-related problems and to develop the science-based knowledge necessary to guide the development of effective treatment and prevention programs. For some time now, we have been concerned with alcohol problems among the elderly, usually defined as persons age 65 and older; compared with younger age groups, relatively little is known about alcohol-related problems in this age group. Yet, several changes in the population composition have occurred that make these problems an increasingly important public health issue. One change is the growing number of people who are in this age group, now the fastest growing segment of the U.S. population. Another is the aging of the large cohort of individuals born during the post–World War II "baby boom." This "baby boomer" generation, the first of whom are now in their fifties, appears to drink more than generations preceding it. This alcohol consumption pattern may herald an increase in alcohol-related problems as the "boomer" population ages.

Older persons are at special risk for some alcohol-related problems due to reduced alcohol tolerance, the presence of age-related health conditions, and potential alcohol/medication interactions. Both episodic and chronic drinking appear to precipitate or aggravate a number of conditions that may be experienced in the later years, including hypertension, stroke, diabetes, cognitive loss, falls and fractures, depression, isolation, and suicide. Additionally, tolerance for alcohol is reduced in older persons; and a level of consumption that is moderate for a young person may be problematic for an older person. Because alcohol problems among older persons often are mistaken for other conditions associated with the aging process, alcohol abuse and alcoholism in this population may go undiagnosed and untreated or be treated inappropriately. As with their younger counterparts, alcoholism among older persons is treatable. Regrettably, there remains a persistent belief among families, friends, and health care providers that older individuals have earned the right to be left alone—if they wish to drink, even excessively, then so be it. However, every stage of life has its own rewards for sobriety, and alcoholism treatment is life-enhancing at any age. The expansion of science-based knowledge of alcohol's impact on the aging process, however, may help to counter these prevailing beliefs, making the diagnosis and treatment of alcoholism among older persons a standard of medical practice.

This monograph, *Alcohol Problems and Aging,* which was developed from papers presented at the NIAAA-sponsored 1996 national conference on alcohol and aging, clearly shows that we understand considerably more about this subject area than we did at the time of our first workshop on this topic in 1983. However, it also is clear that we need to know much more. Some of the scientists whose work is represented in these pages have been actively pursuing research on alcohol and aging for many years. Others are experienced scientists who are relative newcomers to the alcohol research field. This is testimony to the increasingly broad range of basic and biological studies that have been undertaken to understand alcohol's impact on the aging process; on the social, economic, and health status of older Americans; and on society at large. Each author has provided an excellent review of the state of the art on alcohol and aging that I believe will be highly useful to scientists and clinicians alike.

I commend the monograph editors, Drs. Gomberg, Hegedus, and Zucker, and the authors of the various papers for their efforts making this important information on alcohol and aging accessible to a wider audience.

Enoch Gordis, M.D.
Director
National Institute on Alcohol Abuse and Alcoholism

PREFACE

In 1983, the National Institute on Alcohol Abuse and Alcoholism (NIAAA) held a workshop on the nature and extent of alcohol problems among the elderly. The workshop was held in collaboration with the National Institute of Mental Health (NIMH) and the National Institute on Aging, and was held at the Alcohol Research Center of the Washington University School of Medicine in St. Louis, MO. The proceedings of the workshop were published a year later as NIAAA Research Monograph 14 (Maddox, G.; Robins, L.N.; and Rosenberg, N., eds. *Nature and Extent of Alcohol Problems Among the Elderly.* Rockville, MD: National Institute on Alcohol Abuse and Alcoholism, 1984).

In the Foreword, Robert G. Niven stated that the goal of the workshop and the published monograph was to inform "researchers, clinicians, program administrators, . . . about significant findings that may be useful in strengthening alcoholism research and improving alcoholism programs" (p. iii). As stated in the Preface by Ernestine Vanderveen, at that time no one really knew the extent of alcoholism and alcohol-related problems among the elderly, and researchers lacked "a reliable basis for making sound estimates" (p. vi).

In the last decade and a half since that workshop was held, considerable progress has been made in demographic and epidemiologic studies of alcohol use and abuse and in basic and clinical research that has increased our understanding of biological and genetic mechanisms, the impact and consequences of alcohol abuse on the health and well-being of the elderly, and prevention and treatment methods. It is now reasonably accepted that there are indeed alcohol-related problems among older people that have a significant impact on health and the utilization and cost of health care.

Progress has been made, not only in the interest and amount of research reported, but also in the expansion of multidisciplinary research and the production of research reports in a number of different areas: biology, genetics, psychology, treatment, prevention, economics, and policy. With our increased knowledge has come the inevitable expansion of the type and scope of the questions being raised.

In November 1996, a national conference on alcohol and aging was held, sponsored by NIAAA; the University of Michigan Alcohol Research Center, which is mandated to study alcohol and aging; and the Department of Psychiatry of the University of Michigan School of Medicine. Whereas the 1983 conference was divided primarily into discussion of longitudinal studies

and data from NIMH's Epidemiologic Catchment Area Study, the 1996 conference was divided into consecutive sessions covering epidemiology; basic genetic, biological, and developmental mechanisms; the course and consequences of elderly problem drinking; and treatment and prevention.

Conference presentations resulted in renewed interests, expanded knowledge bases, and lively discussions. At the end of the second day, an informal but intense session was initiated that brought together professionals in usually divergent areas: treatment providers applied the research presented to their clinical experiences, and biological researchers found practical applications for their work. We hope that, through this monograph, these dialogs will continue and the dissemination of research findings will be used to inform clinical and prevention practices. Based on conference presentations, the monograph is arranged in the following sections: basic issues, biological mechanisms, course and consequences, treatment and prevention, and a chapter summarizing research issues and priorities.

BASIC ISSUES

The first section of the monograph addresses issues that are integral to the study of alcohol problems in the elderly. In the first chapter, Robert A. Zucker presents a brief review of the epidemiology of changes in the elderly population that are projected for the next several generations; he then uses developmental theory to provide a framework within which to understand the manner in which patterns of alcohol use, abuse, dependence, and related health behaviors are linked to these changing demographics. A. Regula Herzog presents a chapter on methodological issues in survey research with older Americans and discusses the many potential errors that may affect data collection and interpretation. Kathleen K. Bucholz and colleagues present cross-sectional and longitudinal data on the use of alcohol by older individuals in their chapter, including new findings from the Australian Twin Registry. In the final chapter of this section, James S. Jackson and colleagues use life-course theory to account for the differential effects of alcohol and aging on African Americans at various life stages.

BIOLOGICAL MECHANISMS

This section of the monograph provides a discussion of biological mechanisms that are affected by the interaction of alcoholism and agedness. Gerald E.

McClearn summarizes the genetic evidence from both animal and human studies regarding the declining heritability of alcohol use in advancing age. The contribution of behavioral pharmacology in understanding the effects of alcohol and age is presented in the chapter by Harold Kalant, who calls for additional research on their interaction effects. Clive Harper and his co-authors examine the question of whether alcohol affects the aging process of the brain by comparing the neuropathological changes that have been identified in both aging and alcohol studies of brain damage. This question is also approached by the authors of the three final chapters in the section. W. Gibson Wood reviews the literature on the interaction of aging and ethanol on brain membrane structure and the major neurotransmitter systems. Sid Gilman and colleagues present a summary of their recent research on cerebral injury in severe chronic alcoholics, using both neurobehavioral and neuroimaging techniques to localize the areas of damage. In the final chapter of this section, David W. Oslin and Alan M. Mellow review recent advances in the neurobiology of addiction in relation to the process of aging and discuss the implications that such changes may have for differential neurotransmitter-based treatment strategies for the elderly.

COURSE AND CONSEQUENCES

This section provides a review of the medical and psychosocial consequences of the effects of alcohol on the elderly. The chapter by Richard E. Finlayson and Richard D. Hurt is an overview of common patterns of medical illness and injury in the elderly associated with heavy drinking. Sara Jo Nixon's primary focus is on neurocognitive function in the often understudied area of social drinking in the elderly. Along the way, she addresses issues of the relationship of aging to cognitive change and reviews recent findings regarding the effects of social drinking on neurocognitive function.

The relationship among stress, illness, and alcohol has not been well understood among the elderly; John W. Welte presents a synthesis of research findings in his chapter, and also reports findings from his own work on the Erie County Elder Drinking Survey that are pertinent to these issues. Michael R. Levenson and his co-authors examine the complicated issues of the interaction of personality and problem drinking over long spans of time among middle-aged and older men. Using data from the Normative Aging Study and a database that tracked variations in these attributes over an almost 20-year interval, they

found both a personality effect on the likelihood of alcohol problems and an effect of chronic drinking on personality. Rudolf Moos and his co-authors summarize life context factors, treatment, and late-life drinking behaviors, using a stress and coping model to guide the conceptualization.

After briefly describing sleep and its changes with age, Michael S. Aldrich reviews the effects of alcohol on sleep, breathing and apnea, insomnia, and sleep in alcoholics, and summarizes recent findings from his own laboratory in these areas. Of special clinical interest is the relationship between sleep disturbance and relapse, and the possible contribution that sleep pathology may play in this process. Patricia F. Waller presents research on alcohol and driving behavior in the elderly, ranging from injury and crash studies to her group's investigations using driving simulator methodology.

TREATMENT AND PREVENTION

This section covers age-specific topics in alcohol treatment and prevention. Wendy L. Adams discusses the prevalence of alcohol problems in health care settings and the impact of alcohol use on the use of health services among the elderly, with specific reference to how primary care doctors can best address alcohol problems of the elderly in clinical settings. Larry W. Dupree and Lawrence Schonfeld review the literature regarding recurring treatment issues among older adult alcohol abusers and present a set of summary recommendations based upon what the literature indicates are "best" responses. They also offer a brief discussion of empirically validated treatment approaches and make recommendations here as well. Providing information on the little-understood topic of the effects of aging on alcohol withdrawal, Kirk J. Brower examines both animal and human studies and explores possible mechanisms that may explain the age-related differences in alcohol withdrawal observed among alcoholics.

Frederic C. Blow reviews the range of prevention/ intervention strategies available for older adults who are at-risk drinkers, problem drinkers, and those with alcohol abuse/dependence. He encourages broadening the menu of choices for working at these different points along the drinking problem continuum. The chapter includes a discussion of critical issues in the design and application of brief intervention strategies and the design of outcome studies. Blow describes brief intervention studies from the University of Wisconsin and

the University of Michigan as well as a treatment outcome study conducted by his group. Mark B. Sobell and colleagues present their study on natural recovery and describe how this strategy can be used to foster self-change in individuals with late-onset drinking problems. They also discuss differences in late- versus early-onset drinking patterns.

Despite the high levels of comorbidity in older alcoholics, many treatment and prevention programs are ill equipped to address the complexities of these problems. Mark L. Willenbring describes the major types of comorbidities found among older adults with alcohol problems and presents the challenges one faces when developing relevant treatment models that address this clinical diversity. Roland M. Atkinson imparts a different perspective to this issue by addressing the question of whether it is advantageous to segregate older adults in age-specific treatment programs, no matter what form or location of treatment is selected. In the final chapter in this section, Gayle M. Boyd provides a commentary on prevention of alcohol problems in the elderly and discusses the theoretical issues that need to be considered in developing a comprehensive prevention research agenda.

SUMMARY

In the final chapter, the editors discuss and attempt to integrate the research and clinical material presented in this monograph. A review of research issues and priorities is presented in this chapter.

We have come a long way, but each new finding brings more questions in its wake. That is the nature of science. To respond to the future needs of a society in which the elderly will constitute a substantially increased proportion of the population, it is imperative that the research required to answer these questions be carried out.

Edith S. Lisansky Gomberg, Ph.D.
Andrea M. Hegedus, Ph.D.
Robert A. Zucker, Ph.D.

ABBREVIATIONS AND ACRONYMS

AA	Alcoholics Anonymous
AARP	American Association of Retired Persons
ACD	alcoholic cerebellar degeneration
AD	Alzheimer's disease
ADH	alcohol dehydrogenase
AHEAD	Asset and Health Dynamics Among the Oldest Old [survey]
AIDS	acquired immunodeficiency syndrome
ANOVA	analysis of variance
APS	Addiction Potential Scale
ATU	alcohol treatment unit
AUDIT	Alcohol Use Disorders Identification Test
BAC	blood alcohol concentration
BDZ	benzodiazepine
CAD	coronary artery disease
cAMP	cyclic adenosine monophosphate
CDT	carbohydrate-deficient transferrin
CES-D	Center for Epidemiologic Studies—Depression Scale
[^{11}C]FMZ	[^{11}C]flumazenil
CI	confidence interval
CIWA-Ar	Revised Clinical Institute Withdrawal Assessment for Alcohol
CNS	central nervous system
CRI	Coping Responses Inventory
CSAT	Center for Substance Abuse Treatment
CT	computed tomography
d	day(s)
dL	deciliter(s)
DNA	deoxyribonucleic acid
DOI	1-(2,5-demethoxy-4-iodophenyl)-2-aminopropane
DPI	Drinking Problems Index
dpy	drinks per year
DSM-III	*Diagnostic and Statistical Manual of Mental Disorders, Third Edition*
DSM-III-R	*Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised*
DSM-IV	*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*
DTBZ	dihydrotetrabenazine

DTs	delirium tremens
DUI	driving under the influence
DV	distribution volume
DWI	driving while intoxicated
ECA	Epidemiologic Catchment Area
ED	emergency department
EEG	electroencephalogram
EMC	Educational Materials Control
EPESE	Established Populations for Epidemiologic Studies of the Elderly
ERP	event-related potential
[^{18}F]FDG	[^{18}F]fluorodeoxyglucose
FMZ	flumazenil
FSC	Fostering Self-Change
ft	foot (feet)
FWHM	full width at half-maximum
g	gram(s)
GABA	gamma-aminobutyric acid
GAD	glutamic acid decarboxylase
GAP	Gerontology Alcohol Project
HDL	Health and Daily Living [Form Manual]
5-HIAA	5-hydroxyindoleacetic acid
HPLC	high-performance liquid chromatography
5-HT	5-hydroxytryptamine
HVA	homovanillic acid
ICV	intracranial cavity volume
ITS	intelligent transportation systems
kg	kilogram(s)
lCMRglc	local cerebral metabolic rate(s) for glucose
LISRES	Life Stressors and Social Resources Inventory
LS	long sleep [mice]
M	mean
MAC	MacAndrew Alcoholism Scale
MAC-R	MacAndrew Alcoholism Scale, Revised
MANOVA	multivariate analysis of variance
MAO-A	monoamine oxidase A
MAO-B	monoamine oxidase B
MAST	Michigan Alcoholism Screening Test

MAST-G	Michigan Alcoholism Screening Test—Geriatric Version
mCi	millicurie(s)
m-CPP	*m*-chlorophenylpiperazine hydrochloride
mg	milligram(s)
mL	milliliter(s)
mm	millimeter(s)
mM	millimolar
MMPI	Minnesota Multiphasic Personality Inventory
MMPI-2	revised Minnesota Multiphasic Personality Inventory *or* Minnesota Multiphasic Personality Inventory—2
MOS SF-36	Medical Outcomes Study Short Form, 36-item General Health Survey
mph	miles per hour
MR	magnetic resonance
MRI	magnetic resonance imaging
mRNA	messenger ribonucleic acid
ms	millisecond(s)
MSLT	Multiple Sleep Latency Test
µCi	microcurie(s)
µm	micrometer(s)
NAS	Normative Aging Study
NCS	National Comorbidity Survey
NIA	National Institute on Aging
NIAAA	National Institute on Alcohol Abuse and Alcoholism
NMDA	*N*-methyl-D-aspartate
NP	alcohol-nonpreferring
NREM	non-REM [rapid eye movement]
OSA	obstructive sleep apnea
oz	ounce(s)
PBS-EDTA	phosphate-buffered saline–ethylenediaminetetraacetic acid
PC	personal computer
PET	positron emission tomography
PICS	pericerebral space
QFI	Quantity Frequency Index
QTL	quantitative trait locus
REM	rapid eye movement
SAMHSA	Substance Abuse and Mental Health Services Administration

SAS	Senior Adult Services [Clark County, WA, program]
SD	standard deviation
SMAST-G	Short Michigan Alcoholism Screening Test—Geriatric Version
SPM	synaptic plasma membranes
SRC	Survey Research Center [of the Institute for Social Research at the University of Michigan]
SS	short sleep [mice]
SSI	Supplemental Security Income
SSRI	selective serotonin reuptake inhibitor
T	tesla
TAD	Traffic Accident Damage [Scale]
TE	echo time
TR	repetition time
USDA	U.S. Department of Agriculture
VA	[Department of] Veterans Affairs *or* Veterans Administration
VMAT2	vesicular monoamine transporter type 2
VTA	ventral tegmental area
v/v	volume to volume [ratio]
WAIS-R	Wechsler Adult Intelligence Scale—Revised
WHO	World Health Organization
wk	week
WKS	Wernicke-Korsakoff syndrome
WMS	Wechsler Memory Scale

BASIC ISSUES

Chapter 1

Developmental Aspects of Aging, Alcohol Involvement, and Their Interrelationship

Robert A. Zucker, Ph.D.

At this point in historical time the scientific problem being addressed in this monograph is an especially interesting one, and also one of steadily increasing social importance. There are several reasons why this is the case: (1) alcohol, as a drug of celebration and also of everyday use, occupies a special place in the social order that makes patterns of use and abuse heavily tied to other life-cycle variations; (2) major changes in the number, rate of growth, and composition of the elderly population are anticipated over the next generation and a half, that will dramatically alter the character of the U.S. population; and (3) the nature of ongoing changes in health practices and health care, and their subsequent ramifications in extending longevity and changing quality of life, will likely also have impact upon patterns of drinking. Thus, derivative changes in the epidemiologic structure of alcohol use and problems relating to these issues are anticipatable, at least in broad form, and should play out by way of shifts in the extent and type of alcohol problems found among older individuals in the U.S. population during the next generation and thereafter.

After a very brief review of developmental theory, this chapter addresses each of these issues in turn, as they bear upon the broader questions of how alcohol use, abuse, and dependence are likely to show themselves among those over age 65 (i.e., among the elderly). Along the way, where relevant, methodological and definitional

R.A. Zucker, Ph.D., is professor of psychology in the Departments of Psychiatry and Psychology, University of Michigan, and director of the University of Michigan Alcohol Research Center, 400 East Eisenhower Parkway, Suite 2A, Ann Arbor, MI 48108–3318.

issues are also discussed. We begin with some definitional stage setting.

AGE STAGES AND THE CONCEPT OF OLDNESS

Although the concept of age stages as a formal classification system to express systematic age-related variation has largely been rejected by developmentalists (Heatherington 1983), the linking of age ranges to stages in the human life cycle has had a long and useful history (Neugarten and Datan 1973; Levinson 1978; Elder and Caspi 1990). Given that the biological organism has some upper limit to longevity that is substantially younger than Methuselah's, and that all cultures have some chronologically linked classification system to reflect the passage of time and the age gradedness of their members, I offer the labeling structure shown in table 1 as a guide to the territory. Along with the offer comes the stipulation that the categories are fuzzy at their boundaries, and that at the individual level, the linkage of category to chronological age is likely to be imprecise.

DEVELOPMENTAL THEORY, ALCOHOL INVOLVEMENT, AND AGING

Aging is a time-based process that is to a degree irrevocable and to a degree not. Developmental theory provides an analytic framework within which to

Table 1. A Life Stage Chart.

Stage	Age Span
Earlier Life	
Infancy	0–2
Toddlerhood	2–3
Early childhood	3–6
Middle childhood	6–12
Adolescence	12–20
[Early adult transition	18–22]
[Youth	18–25]
Midlife	
Early adulthood	18–40
Middle adulthood	40–60
[Mid-life transition	40–45]
[Late middle adulthood	55–60]
Later Life	
Later adulthood	60 plus
[Late adult transition	60–65]
The elderly	65 plus
The young old	60–75
The old old	75 plus
The frail elderly (or the very old)	80 plus

understand this dialectic, and it also provides a heuristic structure for model building that allows for the disaggregation of this time-based variation. The theory posits the importance of studying earlier behavior as a clue to what unfolds later, and emphasizes the importance of charting process as well as structure (Ford and Lerner 1992; Zucker et al. 1995). The theory also prescribes an evaluation of the interplay between contextual and organismic factors, and of timing and sequencing, in the creation and maintenance of new structures and the dissolution of older ones (Gottlieb 1992; Kraemer et al. 1997). Patterns of adaptation (for example, the achievement of the structure of a disease) are viewed as dynamic systems that evolve in particular contexts, and that have come to a place where a set of patterned interrelationships now exists, that are sufficient to maintain a steady-state adaptation (Fitzgerald et al. 1994; Zucker et al. 1997).

The theory also posits that in order to understand process, it is essential to specify what external as well as organismic characteristics are necessary for the maintenance of apparent stability, as well as to describe those factors that influence the structure's evolution and/or degradation (Featherman and Lerner 1985). Stability of structure is not taken for granted, but rather is regarded as an active homeostasis, that needs to be disaggregated into its component processes, and probed for the degree to which apparent "unchangeability" is accounted for by internal mechanisms, or by way of an active system of relationships between organism and environment. For these reasons, developmental analysis requires a careful specification of the potential maintenance functions of context as well as a description of intraindividual mechanisms (Cicchetti and Cohen 1995).

To understand the interaction of alcoholism-related processes and aging therefore requires an understanding of both the core neurobiological structure of the disorder as well as the contextual factors that encourage the alcoholic display or suppress its development. These factors operating on their own would be regarded as nonspecific causative agents, but they may be vital to the development of and changes in alcoholic symptom structure over the life course. A parallel variable network also needs to be constructed to account for changes in normal patterns of drinking with increasing age.

THE DOMESTICATED NATURE OF ETHANOL USE AND ITS DEVELOPMENTAL IMPLICATIONS

In examining patterns of variation of alcohol use, abuse, and problems over different parts of the lifespan, two attributes of this drug are frequently overlooked. One is that ethanol is the world's most domesticated psychoactive drug. It is widely sought after for its pharmacological attributes, and also, in the form of beer, is one of the world's most common foods. Thus, its use structure is heavily embedded in the life fabric of the majority of modern societies. It is a drug of courting and of grief, and is also a drug with which we sometimes bury people.

The second attribute derives from the first. Because alcohol is so heavily embedded in the fabric of everyday life, its use and abuse are to a degree superimposed upon the ongoing life structure. Therefore, patterns of use, abuse and dependence are likely to differ as a function of the life-course variations upon which the alcohol involvement is overlaid. Marriage, divorce, loss of a job, and change in peer structure have all been related to changes in patterns of consumption (Miller-Tutzauer et al. 1991; Catalano et al. 1993; Wilsnack 1996). Thus, an understanding of alcohol involvement and aging needs to take account of the life variations that co-occur with the age variation, that may make availability greater or lesser, and that to a degree either proscribe or prescribe use with the shifts in role structure that occur. This issue is as relevant for the second half of the life cycle as it is for the first half. In addition, much of the demographic variability that is evident in the existing epidemiologic data on older adulthood is explainable by way of such role variations, but this underlying causal variation has been insufficiently appreciated. This point is revisited later in the chapter. We turn now to the epidemiologic data that address some of these issues.

POPULATION PROJECTIONS FOR THE ELDERLY OVER THE NEXT GENERATION AND THEREAFTER: DEVELOPMENTAL ISSUES

In the following discussion, the primary focus is upon changes occurring in the elderly population (i.e., among persons age 65 and older), as these are set against the backdrop of the larger population and its changing base. I also focus most heavily on changes occurring in approximately the next generation (i.e., through 2020). This is a timeframe within which science can contribute realistically to public policy, and where an understanding of current and impending changes can effectively be characterized and responded to. Changes in alcohol-related outcomes occurring further off than that probably do not have the same immediacy to them, although as the data presented here will indicate, they are likely to continue, and even intensify the trends that are currently becoming evident. Unless otherwise noted, the figures in this section were obtained from three U.S. Bureau of the Census publications: Bernstein (1995), Hobbs (1995), and Day (1996). Population projections were based on U.S. Census "middle series" assumptions about fertility rates, life expectancies, and yearly net immigration.

The composition of the U.S. population has been changing throughout the 20th century, with increasing numbers as well as percentages of elderly individuals entering the population. In 1900, there were 3 million persons over the age of 65, in 1995 there were 34 million, and in 2020, there will be 53 million. Moreover, in 1900 about 1 in 30 were in the elderly group, in 1995 it was 1 in 8, in 2020 it will be 1 in 6, and by 2030, it will be 1 in 5. To give some sense about age-related shifts, median population age is currently 34, but in 2020 it will be almost 38. Figure 1 graphically depicts these

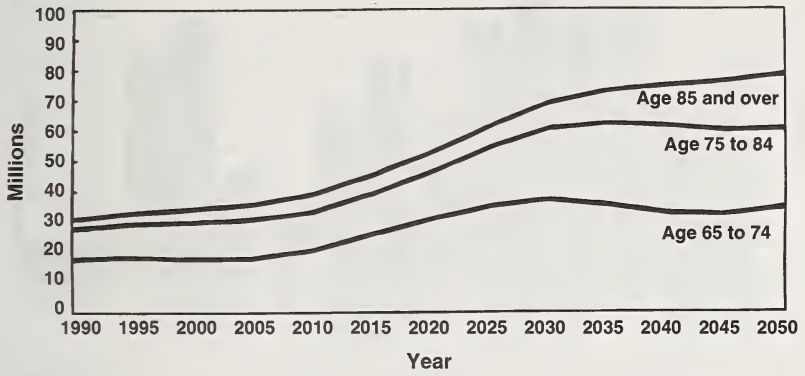

Figure 1. Population of persons age 65 and over, 1990–2050 (middle series beyond 1990). Reprinted from Day, J.C. *Population Projections of the United States by Age, Sex, Race, and Hispanic Origin: 1995 to 2050.* U.S. Bureau of the Census Current Population Reports Series P-25, No. 1130. Washington, DC: U.S. Government Printing Office, 1996. p. 12.

changes by decade subgroup, within the elderly population. As can be seen, the major shifts will start to take place around 2010 and will continue at a high rate until approximately 2030. During this 20-year interval, the number of persons over age 65 will increase by 73 percent, while the population under age 65 will decrease by 3 percent.

Figure 2 presents the rate variations more clearly, and highlights an issue that over the long run is likely to have major clinical ramifications: namely, that the rate of population change will shift from the lowest level in U.S. history to one of the highest (Day 1996, p. 10). In addition, most of these changes will take place among the "old old" (age 75 and older). What is also derivative from, but not so immediately evident in these figures is that the change in population demography is likely to produce a period of rapid social change, involving shifts in the value structure of the society that would be anticipated to occur as median age rises, as the numbers of those requiring dependent care increase, and as the proportion of those under age 18 remains relatively constant. In short, the country is likely to become an older-age–focused society as well as one that is demographically older.

The elderly population of the next generation are projected to be better educated than today's elderly, as younger, better educated cohorts age. In 1993 "only 12 percent of the elderly had college degrees, but 20 percent of 55 to 59 year olds and 27 percent of 45 to 49 year olds did" (Bernstein 1995, p. 8). The better educated tend to stay healthier longer and to be economically more advantaged than their

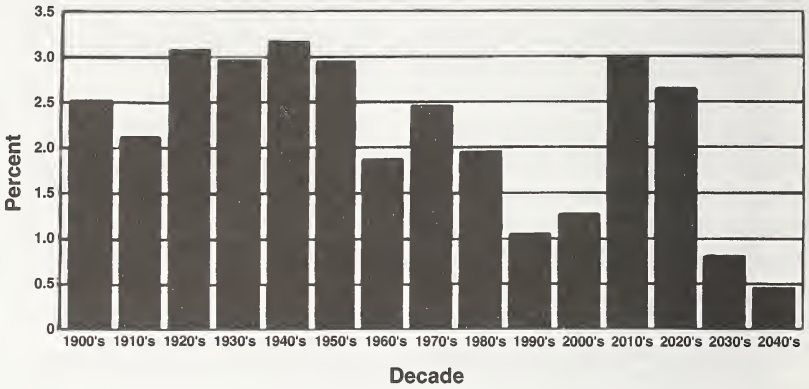

Figure 2. Average annual percent change in population age 65 and over, 1900–2050 (middle series for 1990's and beyond). Reprinted from Day, J.C. *Population Projections of the United States by Age, Sex, Race, and Hispanic Origin: 1995 to 2050.* U.S. Bureau of the Census Current Population Reports Series P-25, No. 1130. Washington, DC: U.S. Government Printing Office, 1996. p. 11.

less educated counterparts. These lifestyle differences will have implications for level of alcohol consumption shifts in the next generation. This issue is discussed further below.

At the same time there is very significant heterogeneity in the elderly population, so heterogeneity of health status outcomes is another ramification of these demographic projections. In fact, substantial income and educational disparities exist among the various subcultural groups (see figure 3). Although the elderly in general have more assets than the non-elderly, poverty rates in the early 1990's continued to be relatively high for elderly African Americans (33 percent) and elderly Hispanics (22 percent), and significantly higher than the overall elderly poverty rate of 13 percent (U.S. Bureau of the Census 1996). Similarly, disparities in educational attainment, as they play out over a life course, are likely to have significant impact not only upon income structure but also upon a number of other life adaptations, including differences in attitude structure about the acceptability of temperate versus intemperate versus abstinent patterns of alcohol use. It is a long-term historical association that less well educated groups have both higher rates of abstinence and also higher rates of problem use, among users, than do those of higher educational level (Cahalan et al. 1969; National Institute on Alcohol Abuse and Alcoholism [NIAAA] 1994). This subcultural group variation appears to be in direct opposition to apparent national trends, with the better educated elderly subgroups likely to sustain drinking into their older years, and with the newer cohorts of less educated elderly having both higher rates of

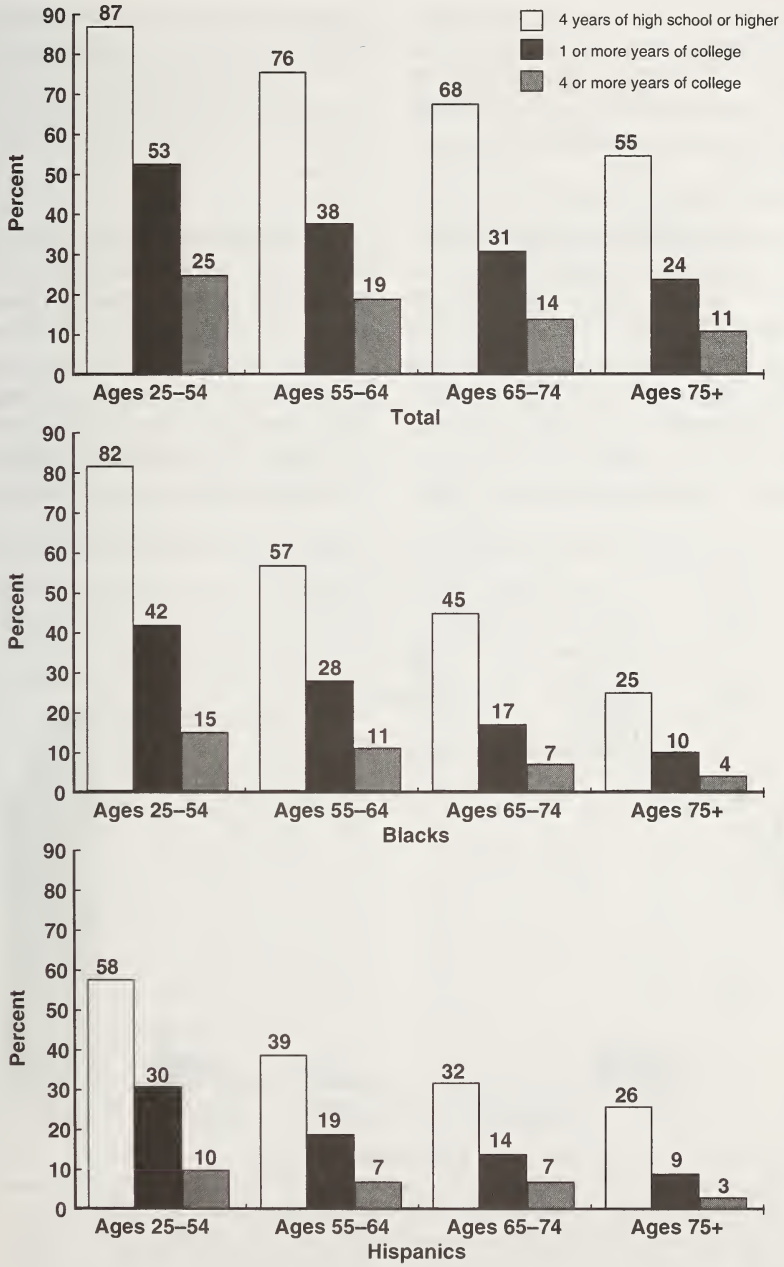

Figure 3. Educational attainment of Americans by age and race/ethnicity, 1994. Reprinted from Treas, J. Older Americans in the 1990s and beyond. *Population Bull* 2:21, 1995.

abstinence as well as problem use. The existing data also indicate that substantially different aging trajectory structures for alcohol use are present for different cultural groups (see also chapter 4).

Heterogeneity of these age structure changes is evident in another way. The white population is projected to continue to be the largest elderly subgroup. It will still comprise approximately three-quarters of the elderly population in 2020, but this is also the group with the slowest growing rate (Day 1996, pp. 13–18). In contrast, the Hispanic-origin population is projected to be the largest contributor to overall population growth, with a doubling in size to 53 million over the 1996 figure of 27 million, and with an overtaking of the black population in absolute size somewhere around 2020. The manner in which these changes are reflected among the elderly is shown in figure 4. Given the major differences in patterns of alcohol use among these groups (Helzer et al. 1991), again it is reasonable to anticipate major shifts in age patterning of both consumption levels and problems within the next generation. This point is elaborated upon later in this chapter.

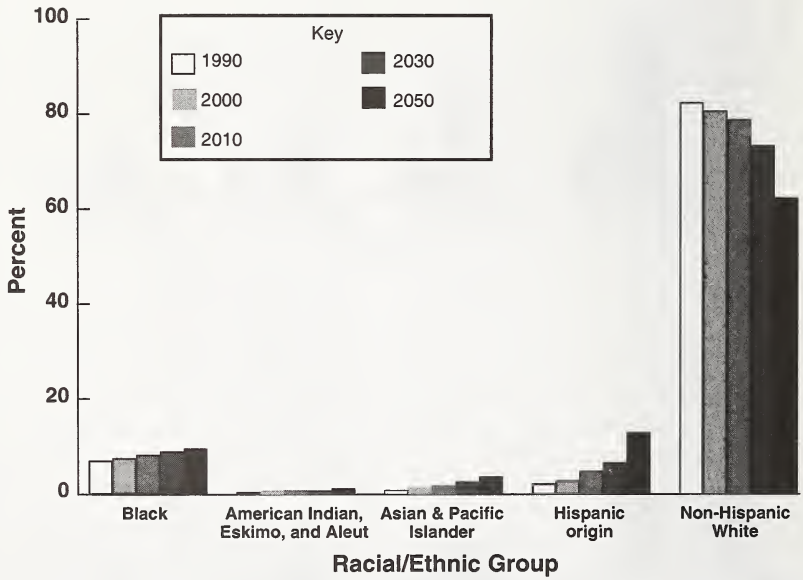

Figure 4. Percent distribution of the population age 65 and over by race and Hispanic origin, 1990–2050 (middle series beyond 1990). Reprinted from Day, J.C. *Population Projections of the United States by Age, Sex, Race, and Hispanic Origin: 1995 to 2050.* U.S. Bureau of the Census, Current Population Reports Series P-25, No. 1130. Washington, DC: U.S. Government Printing Office, 1996. p. 18.

POPULATION VARIATIONS IN ALCOHOL USE, ABUSE, AND DEPENDENCE IN THE LATTER PART OF THE LIFE CYCLE

Variations in Alcohol Use With Increasing Age

Detailed descriptive data on drinking practices and drinking problems among the elderly remain limited. Recent comprehensive reviews of the epidemiologic literature by Adams and Cox (1995) as well as by Bucholz and colleagues (1995; see also chapter 3 in this monograph) indicate that the knowledge base for both consumption variation, as well as for rates of problem drinking and alcohol abuse/dependence have been primarily based upon restricted samples, drawn from sometimes large, but unrepresentative regional populations and clinical/hospital settings. Drawing from 9 population-based and 10 medical studies, Adams and Cox concluded that "the prevalence of alcohol use and misuse decline with age, but misuse remains an important public health problem. Between 2 and 4 percent of the United States elderly population meet criteria for alcohol abuse or dependence. Up to 10 percent are 'heavy' or problem drinkers. Alcohol use and misuse are both more common among men than women." (p. 1469)

The three published data sets that are not restrictive in these ways and that begin to approximate population estimates involve the over age 60 database included in Cahalan and Cisin's American Drinking Practices Survey conducted in 1968 (Cahalan et al. 1969), the primary report on alcohol abuse/dependence from the Epidemiologic Catchment Area (ECA) study (Helzer et al. 1991), and a second set of ECA analyses by Bucholz and colleagues (1995) that explicitly focuses on the elderly.

The ECA data are reviewed later. First, however, to provide some benchmark data on usual levels of consumption to be anticipated in older populations as well as to hone in on the major issue of heterogeneity among the elderly, I reproduce data from a not easily accessible National Institute on Aging (NIA) resource summary volume of the NIA's Established Populations for Epidemiologic Studies of the Elderly (EPESE) project (Cornoni-Huntley et al. 1986). This project, begun in 1980, involved studies in three locations (East Boston, MA; two counties in Iowa; and New Haven, CT), and was designed to investigate the prevalence rates of chronic conditions, social and cognitive functioning, health behavior, and other special problems and needs of the aged. It involved baseline surveys of over 10,000 individuals over age 65 conducted in 1981–82, and yearly followups for several years thereafter. The sampling frame for the East Boston and Iowa samples involved the entire enumerated population of those over 65 in the defined areas, and in New Haven it involved cluster sampling of 18 percent of the enumerated population. Participation rates in all three communities were above 80 percent. Median ages for the three samples were 73 (East Boston), 72 (Iowa), and 71

(New Haven). The Boston and Iowa samples were primarily white, while the New Haven sample included 21 percent who were nonwhite (of whom 89 percent were black).

The data on alcohol consumption levels across the communities are of special interest. Figure 5 (Cornoni-Huntley et al. 1986, p. 205) shows the alcohol consumption (ounces of absolute alcohol per day) across age levels in East Boston and in Iowa and Washington counties in Iowa. Data are for drinkers only, and alcohol use varied substantially across region; 54.7 percent of the East Boston sample consumed alcohol in the last month (70.5 percent in the past year), while only 31.2 percent (46.3 percent in the past year) of the Iowa sample did. In the New Haven sample, 51.9 percent consumed alcohol in the last month (65.8 percent in the past year). The expected gender differences in drinker status existed in all communities. Interestingly, only in the Iowa sample did the proportion of older drinkers (past-year) differ from that observed in younger segments of the population. That is, in 1983, U.S. population estimates derived from the National Health Interview Survey showed that 65 percent of those between 18 and 65 in Iowa were drinkers, while the over 65 data showed 46 percent were drinkers. Among elderly drinkers living in an urban setting, where normative value structure is positive for alcohol use (i.e., in the East Boston sample), slightly more than half used alcohol monthly or more often. In contrast, in an area where value structure can be assumed to be more abstinence oriented (Iowa), less than a third of the population were drinking at this level. In addition, the across-community variation in average *level* of consumption existed primarily among the men; it was present across virtually all age levels and was of the order of 2 to 1. Note that both of these samples were primarily white, but ethnic/religious variation was significantly different between the heavily Italian and Irish Catholic East Boston working-class sample and the heavily rural and small town Protestant Iowa samples.

Figure 6, also from the NIA project but based on the New Haven data, shows within-community but across-racial/ethnic-group variation by age cohort. Differences between the upper and lower figures clearly make the case for primary differentiation of alcohol use patterning by way of subcultural variation, and support the hypothesis that regional variation is a proxy for subculture variation. They also reiterate the major differences in gender role adaptation vis-à-vis alcohol consumption across the two subcultures among the elderly, and simultaneously support a hypothesis that cohort variability relating to heavier versus lighter consumption is historically quite different for different cultural-ethnic groups.

More generally, the levels of alcohol use reported in the EPESE project offer a substantial challenge to the stereotype of nonproblematic drinking among the aged *for specific subsets of the elderly.* Using Barry's (1997) definition of *at risk* drinking (1 or more drinks per day), approximately 50 percent of the East Boston men age 80 and younger who consumed alcohol made this

Figure 5. Age variation in average daily alcohol consumption (ounces of absolute alcohol per day) over a 1-month interval in the EPESE East Boston and Iowa counties samples (drinkers only) from the baseline 1981–82 survey. Participants over 90 years of age were excluded because of small sample size. Alcohol consumption data are presented as moving averages of values for three successive ages. Reprinted from Cornoni-Huntley, J.; Brock, D.B.; Ostfeld, A.M.; Taylor, J.O.; and Wallace, R.B. *Established Populations for Epidemiologic Studies of the Elderly.* NIH Pub. No. 86–2443. Bethesda, MD: National Institute on Aging, 1986. p. 205.

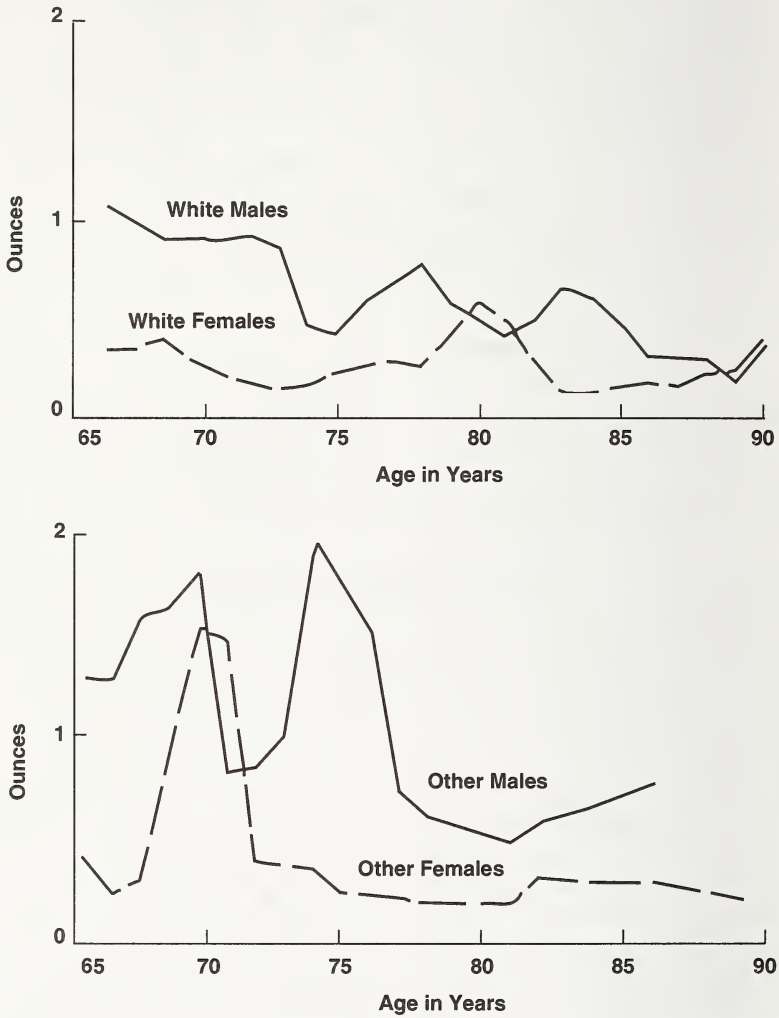

Figure 6. Age variation in average daily alcohol consumption (ounces of absolute alcohol per day) over a 1-month interval in the EPESE New Haven sample for two different racial/ethnic groups (drinkers only) from the baseline 1981–82 survey. Participants over 90 years of age were excluded because of small sample size. For non-white men and women, estimates for subjects age 80 and older are unreliable because of small sample size. Alcohol consumption data are presented as moving averages constructed from population estimates projected from sample data for three successive ages. Reprinted from Cornoni-Huntley, J.; Brock, D.B.; Ostfeld, A.M.; Taylor, J.O.; and Wallace, R.B. *Established Populations for Epidemiologic Studies of the Elderly.* NIH Pub. No. 86–2443. Bethesda, MD: National Institute on Aging, 1986. p. 206.

categorization, as did 50 percent of the age 75 and younger nonwhite New Haven men.

Variations in Alcohol Abuse and Dependence With Increasing Age

The epidemiologic data on prevalence of alcohol problems across the lifespan for different racial/ethnic groups show patterning variability similar to that based on consumption data. Figures 7 and 8 (derived from the ECA data reported in Helzer et al. 1991) show the 1-year, weighted U.S. population prevalence of DSM-III (American Psychiatric Association 1980) diagnoses for men and women in different age groups throughout the life cycle. These data indicate that among white men there is a steady decline in the rate of alcohol abuse/dependence across the life cycle. Among Hispanic men the pattern is the highest of the three subcultural groups through the early middle adult years, with 1 out of 5 men making a 1-year diagnosis through ages 30–44, followed by a dropoff to about 1 in 14 among the elderly. The rate among black men is the lowest in the early adult cohort (about 1 in 14), at precisely the age stage where it is highest for the other two cultural groups. In addition, the age-graded data show an increase to about 1 in 7 in early to middle adulthood, before an age-related decrease. The age patterning for women (figure 8) shows similarly dramatic, across-ethnic-group variation. Thus, a reasonable hypothesis is that nonalcohol-specific but age-stage–related variations of the life structure determine an important part of the symptomatic variability.

Given that these data are cross-sectional rather than longitudinal, it cannot be concluded that the within-culture, across-time trajectory of abuse/dependence will show the same patterning

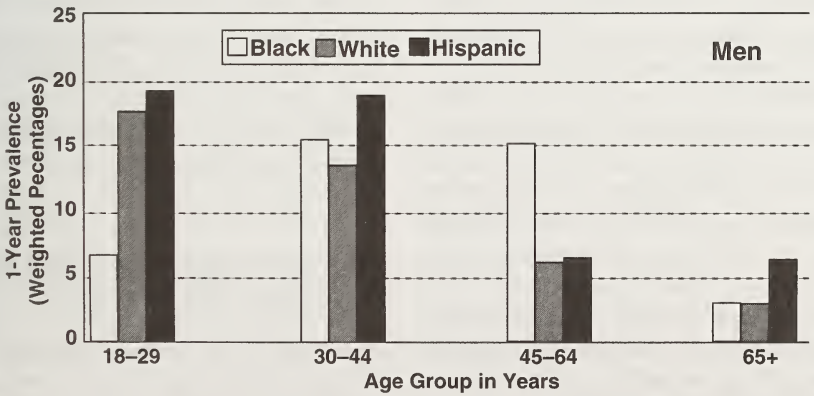

Figure 7. Alcohol abuse/dependence: 1-year prevalence for men. Data from Helzer et al. 1991.

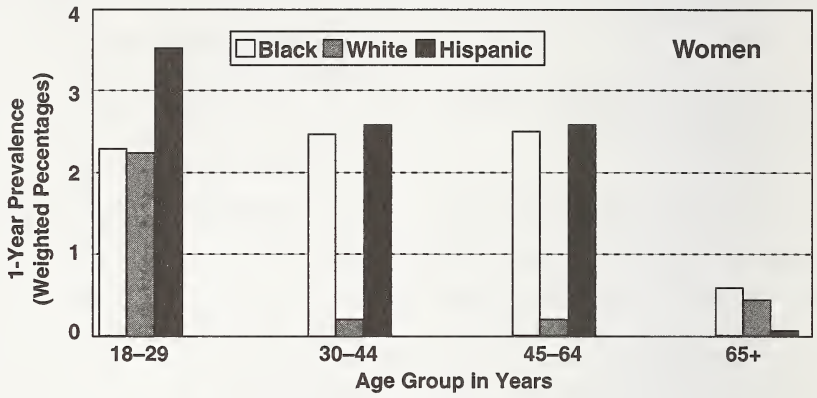

Figure 8. Alcohol abuse/dependence: 1-year prevalence for women. Data from Helzer et al. 1991.

as these cross-sectional snapshots describe. Both historical and cohort effects, including different mortality rates, would be expected to affect across-time relationships differently for the different subcultures (Reich et al. 1988; see also chapter 4 in this monograph). Nonetheless, within each age stage, historical effects and cohort influences (at least at the societal level) are the same across the groups. Thus the cross-sectional data can be interpreted as an indicator of continued subcultural differences in alcohol abuse/dependence over the entire lifespan, with Hispanic males showing the highest rates of alcohol problems in older adulthood. (While this point is accurate as a statement about mean level, there is also very substantial variability within the Hispanic population as a function of Latino subcultural background as well as degree of acculturation [Caetano 1989].)

This conclusion/prediction was derived from the ECA data collected between 1980 and 1984. More recent, albeit also cross-sectional, national data from the 1993 and 1994 Substance Abuse and Mental Health Services Adminstration (SAMHSA) National Household Surveys (Substance Abuse and Mental Health Services Administration 1995, 1996) continues to confirm the prediction that the highest rates of drinking and heavy drinking will be present among Hispanic older males. Note that it is only in the oldest age category that this difference is present; in that regard, the recent SAMHSA figures are an approximate replication of the patterning shown in figure 7.

RECENT CHANGES IN THE CONCEPT OF AGING

The concept of aging, in contrast to those of maturation and growth, is usually understood as a process involving the emergence of both structural and functional deficits, some involving normal degradation of

function, and some involving the emergence of disease and its associated pathologies. In both instances the emergence is chronologically age linked, and is ostensibly also normative (i.e., the underlying presumption is that the observed decrements are inescapable, given the passage of time). In the last generation this concept of aging has increasingly been challenged, and the alternative concepts of successful aging (Rowe and Kahn 1987) and productive aging (American Psychological Association 1993) have begun to drive the empirical literature. This newer framework encourages the disaggregation of the variety of processes taking place as chronological aging occurs. Aging is thus viewed as a composite construction, combining survival, variations in physical and mental health, and variations in life satisfaction. These factors in turn, when operating in concert, lead to the achievement of "successful aging," where normal functional deficits are offset by creative adaptations, which serve to slow or counteract anticipated functional losses.

A related concept is that of successful optimization with compensation (Baltes and Baltes 1990). This perspective emphasizes the lifelong process of age-related increases in specialization, involving increasing selectivity of motivational resources and cognitive skill development, along with increased adaptational capacity, albeit in specified areas (Schulz and Heckhausen 1996). Along with this specialization comes loss in some specific functions and a reduction in general reserve capacity, but the payback is selective optimization of competence. Over the longer timespan, compensatory strategies also take place as a response to the restriction in range of plasticity and adaptive potential. These strategies involve changes in activity level, performance, and even the increased utilization of external aids to sustain effective function. The end result is a transformed, more restrictive but still effective life structure.

These newer concepts of aging have emerged as a result of changes in knowledge about the factors contributing to chronic illness as well as those relating to cognitive decline. Differences in the practice of healthy or strategic behaviors (e.g., exercise, proper diet, the utilization of different cognitive strategies for problem solving), differences in ready and early access to health care, exposure to facilitating rather than damaging physical and social environments, differences in genetic instability (Jazwinski 1996)—all of these factors contribute to differences in adaptational status, and also create differences in functional agedness. The result is that over the life course there is increasing heterogeneity of health status (Hertzman et al. 1994) and increasing lifespan differentiation as earlier differences in adaptive capacity, ability to utilize adaptational strategies, and their consequences play out (Staudinger et al. 1995).

The increased heterogeneity of health status and functioning present among older individuals needs to be underscored. It has major implications for a developmental understanding of the sequelae of alcoholic disorder over time (Zucker et al. 1995), as well as of differences in trajectories of alcohol

use across the lifespan (Brennan and Moos 1995; Schulenberg et al. 1996). Contextual structure, with its related differences in availability of an opportunity base for healthy outcomes (Dannefer 1988), differences in availability of a normative attitudinal structure that supports or does not support nonproblematic drinking, differences in capacity to turn on or turn off the genetic circuitry pertaining to alcohol use and abuse—all of these environmental surrounds have the ability to shape short-term organismic outcomes, which in turn shape a consequent drinking adaptation. But the adaptation, once made, then has the potential to shape longer term outcome. Thus it is reasonable to anticipate an increasingly diverse set of alcohol use trajectories over the forthcoming generation and thereafter.

POPULATION CHANGES: IMPLICATIONS FOR ALCOHOL USE AND ALCOHOL PROBLEMS

The shifts in population composition that have been described earlier in this chapter have a number of implications for changes in drinking practices, in consequences of drinking, and in related outcomes.

(1) The span of human life has increased markedly in the last century, and for this reason the complications of aging are to a greater degree prevalent in a population that would not have survived 200 years ago. Thus the epidemiology of a variety of different illnesses can be expected to continue to change as a function of the changes in "exposure to risk." The very large anticipated increase in the over age 85 population is but one example of this. For the subset of these individuals who continue to drink, and even more for the subset who continue to drink at at-risk levels, the interaction of alcohol with the chronic medical conditions of older life will become an increasing medical problem. Moreover, the pharmacotherapeutic interventions that are effective with younger age groups may have very different effects among the elderly (see chapter 11).

(2) With changes in life expectancy come changes in the span during which useful (and playful) activity is likely to take place. Given that alcohol use continues to be tied to patterns of leisure time activity, a reasonable hypothesis is that sustained patterns of regular and even heavier drinking will persist into older ages than has previously been the case. There are instances of this outside the United States (e.g., where increased disposable income among older persons in mainland China has led to increased levels of alcohol use and alcohol use disorder [Wang et al. 1992]), and the issue is currently being explored with an NIAAA-supported project among elderly persons living in age-segregated and age-integrated housing in California (A.D. Pelham, D. Schafer, and M. Kubik, unpublished grant application, San Francisco State University 1996). Changes in extent of alcohol involvement would be expected to take place as a function of these shifts in the patterning of everyday behavior, and would be predicted to be greater in communities where the existing normative structure is positive

for alcohol use (e.g., where educational level is higher). There is already a substantial literature relating changes in drinking norms and behavior among youth to their transition in living structure (i.e., from home to college) (e.g., Alexander and Campbell 1967; Berkowitz and Perkins 1986; Baer 1994). This work provides an interesting theoretical background for the studies of parallel processes among the aged, and it will also permit a test of the transferability of the theory across life-cycle stages.

(3) When one examines population-level variables as predictors of individual outcomes (i.e., drinking behavior), it is easy to lose sight of the fact that these variables are proxy indicators for processes that ultimately are more proximal to the individual, relating to social support structure, attitudinal support structure, local availability of alcohol, and modeling of alcohol use. This issue continues to emerge in the databases that examine it. Thus, Treno and colleagues (1993) showed that changes in population age structure were related to changes in "daily routine" structure (an index of time spent outside the house), and this factor was more powerful in predicting consumption trends than were economic variables. Similarly, patterns of elder abuse were related not only to substance abuse of the abuser but also of the victim (Hwalek et al. 1996), suggesting that the nesting of patterns of drinking, and perhaps also of abusive behavior, is at least in part familial (see also Zucker et al. 1996).

(4) On the basis of the marked differences in drinking rates, as well as rates of alcohol abuse/dependence observed in the major ethnic/racial subpopulations, coupled with general population trends that predict differences in lifestyle as well as differences in rate of successful aging, it can be anticipated that drinking rates among older adults, and concomitant increases in drinking problems, will be higher than ever before in the history of the country. There are two converging lines of data supportive of this prediction: One is that the educational (and income) composition of the white subpopulation is increasing, and this subgroup still remains, and will continue to remain, the numerically largest subset of the elderly even while its percent share of the overall population is decreasing. These demographic changes are harbingers of greater numbers of drinkers as well as possibly higher levels of consumption, produced by the availability of more leisure time, economic well-being, and the presence of a subculture that to a greater degree values drinking (Grant 1997). The second line of data is that the Hispanic subpopulation is increasing at the highest rate; this is a subpopulation with historically high rates of both drinking and diagnosable problems at both younger and older age stages. As its share of the aged population increases, the prevalence of drinking problems should also increase.

FINAL COMMENTS

These are interesting problems that require focused prospective studies to better define parameters, and focused

cross-sectional and mini-longitudinal studies to establish causal structure. From the foregoing review it is clear that the variable matrix being used to investigate the relationship of alcohol problems to aging is still relatively poorly specified. In addition, current work has not sufficiently benefited from the methodological and conceptual sophistication available in the gerontological literature. Hopefully the present commentary will help in rectifying this situation.

The research areas we have identified are only a small subset of the ones that might be listed, and they are added to by the other contributors to this monograph. Given the increasing magnitude of these problems among older adults as well as the rapid and very large increases in the size of this population occurring in the next generation, a better understanding of the manner in which alcohol problems manifest themselves among older adults, together with the development of methods for both alleviating and preventing such difficulties, becomes an increasing scientific as well as national priority.

ACKNOWLEDGMENT

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REFERENCES

Adams, W.L., and Cox, N.S. Epidemiology of problem drinking among elderly people. *Int J Addict* 30:1469–1492, 1995.

Alexander, C.N., and Campbell, E.Q. Peer influences on adolescent drinking. *Q J Stud Alcohol* 28:444–453, 1967.

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 3d ed. Washington, DC: the Association, 1980.

American Psychological Association. *Vitality for Life: Psychological Research for Productive Aging.* Washington, DC: the Association, 1993.

Baer, J. Effects of college residence on perceived norms for alcohol consumption: An examination of the first year in college. *Psychol Addict Behav* 8:43–50, 1994.

Baltes, P.B., and Baltes, M.M. Psychological perspectives on successful aging: The model of selective optimization with compensation. In: Baltes, P.B., and Baltes, M.M., eds. *Successful Aging: Perspectives From the Behavioral Sciences.* New York: Cambridge University Press, 1990. pp. 1–34.

Barry, K.L. Alcohol and drug abuse. In: Mengel, M., and Holleman, W., eds. *Fundamentals of Clinical Practice: A Textbook on the Patient, Doctor and Society.* New York: Plenum Medical Book Company, 1997.

Berkowitz, A.D., and Perkins, H.W. Problem drinking among college students: A review of recent research. *J Am College Health* 35:21–28, 1986.

Bernstein, R. *Sixty-Five Plus in the United States.* Statistical Brief. Washington, DC: U.S. Census Bureau, May 1995.

Brennan, P.L., and Moos, R.H. Life context, coping responses, and adaptive outcomes: A stress and coping perspective on late-life problem drinking. In: Beresford, T., and Gomberg, E., eds. *Alcohol and Aging.*

New York: Oxford University Press, 1995. pp. 230–248.

Bucholz, K.K.; Sheline, Y.I.; and Helzer, J.E. The epidemiology of alcohol use, problems, and dependence in elders: A review. In: Beresford, T., and Gomberg, E. eds. *Alcohol and Aging.* New York : Oxford University Press, 1995. pp. 19–41.

Caetano, R. Drinking patterns and problems in a national sample of U. S. Hispanics. In: Spiegler, D.L.; Tate, D.A.; Aitken, S.S.; and Christian, C.M., eds. *Alcohol Use Among U. S. Ethnic Minorities.* National Institute on Alcohol Abuse and Alcoholism Research Monograph 18. DHHS Pub. No. (ADM)89–1435. Washington, DC: U.S. Government Printing Office, 1989. pp. 147–162.

Cahalan, D.; Cisin, I.H.; and Crossley, H. *American Drinking Practices: A National Study of American Drinking Behavior and Attitudes.* Monograph 6. New Brunswick, NJ: Rutgers Center of Alcohol Studies, 1969.

Catalano, D.; Dooley, D.; Wilson, G.; and Hough, R. Job loss and alcohol abuse: A test using data from the Epidemiologic Catchment Area project. *J Health Soc Behav* 34:215–225, 1993.

Cicchetti, D., and Cohen, D.J. Perspectives on developmental psychopathology. In: Cicchetti, D., and Cohen, D.J., eds. *Developmental Psychopathology, Volume 1: Theory and Methods.* New York: John Wiley & Sons, 1995. pp. 3–20.

Cornoni-Huntley, J.; Brock, D.B.; Ostfeld, A.M.; Taylor, J.O.; and Wallace, R.B. *Established Populations for Epidemiologic Studies of the Elderly.* NIH Pub. No. 86–2443. Bethesda: National Institute on Aging, 1986.

Dannefer, D. Differential gerontology and the stratified life course: Conceptual and methodological issues. *Annu Rev Gerontol Geriatr* 8:3–36, 1988.

Day, J.C. *Population Projections of the United States by Age, Sex, Race, and Hispanic Origin: 1995 to 2050.* U.S. Bureau of the Census Current Population Reports Series P-25, No. 1130. Washington DC: U.S. Government Printing Office, 1996.

Elder, G.H., and Caspi, A. Studying lives in a changing society: Sociological and personological explorations. In: Rabin, A.I.; Zucker, R.A.; Emmons, R.A.; and Frank, S., eds. *Studying Persons and Lives.* New York: Springer, 1990. pp. 201–247.

Featherman, D.L., and Lerner, R.M. Ontogenesis and sociogenesis: Problematics for theory and research about development and socialization across the lifespan. *Am Sociol Rev* 50:659–676, 1985.

Fitzgerald, H.E.; Davies, W.H.; Zucker, R.A.; and Klinger, M. Developmental systems theory and substance abuse: A conceptual and methodological framework for analyzing patterns of variation in families. In: L'Abate, L., ed. *Handbook of Developmental Family Psychology and Psychopathology.* New York: Wiley, 1994. pp. 350–372.

Ford, D.H., and Lerner, R.M. *Developmental Systems Theory: An Integrative Approach.* Newbury Park, CA: Sage Publications, 1992.

Gottlieb, G. *Individual Development and Evolution: The Genesis of Novel Behavior.* New York: Oxford University Press, 1992.

Grant, B.F. Prevalence and correlates of alcohol use and DSM-IV alcohol dependence in the United States: Results of the

National Longitudinal Epidemiologic Survey. *J Stud Alcohol* 58:464–473, 1997.

Heatherington, M. *Mussen Manual of Child Psychology.* 4th ed. New York: Wiley, 1983.

Helzer, J.E.; Burnam, A.; and McEvoy, L.T. Alcohol abuse and dependence. In: Robins, N.L., and Regier, D.A., eds. *Psychiatric Disorders in America: The Epidemiologic Catchment Area Studies.* New York: Free Press, 1991. pp. 81–115.

Hertzman, C.; Frank, J.; and Evans, R.G. Heterogeneities in health status and the determinants of population health. In: Evans, R.; Barer, M.; and Marmor, T., eds. *Why Are Some People Healthier Than Others?* New York: Aldine, 1994. pp. 67–92.

Hobbs, F.B. *The Elderly Population.* Statistical Brief. Washington, DC: U.S. Census Bureau, [1995].

Hwalek, M.A.; Neale, A.V.; Goodrich, C.S.; and Quinn, K. The association of elder abuse and substance abuse in the Illinois elder abuse system. *Gerontologist* 36: 694–700, 1996.

Jazwinski, S.M. Longevity, genes, and aging. *Science* 273:54–59, 1996.

Kraemer, H.C.; Kazdin, A.E.; Offord, D.R.; Kessler, R. C.; Jensen, P.S.; and Kupfer, D.J. Coming to terms with the terms of risk. *Arch Gen Psychiatry* 54:337–343, 1997.

Levinson, D.J. *Seasons of a Man's Life.* New York: Knopf, 1978.

Miller-Tutzauer, C.; Leonard, K.E.; and Senchak, M. Marriage and alcohol use: A longitudinal study of "maturing out." *J Stud Alcohol* 52:434–440, 1991.

National Institute on Alcohol Abuse and Alcoholism. *Eighth Special Report to the U.S. Congress on Alcohol and Health.* NIH Pub. No. 94–3699. Bethesda, MD: National Institutes of Health, 1994.

Neugarten, B.L., and Datan, N. Sociological perspectives on the life cycle. In: Baltes, P.B., and Schaie, K.W., eds. *Life-Span Developmental Psychology: Personality and Socialization.* New York: Academic Press, 1973. pp. 53–69.

Reich, T.R.; Cloninger, C.R.; Van Eerdewegh, P.; Rice, J.P.; and Mullaney, J. Secular trends in the familial transmission of alcoholism. *Alcohol Clin Exp Res* 12:458–464, 1988.

Rowe, J.W., and Kahn, R.L. Human aging: Usual and successful. *Science* 237:143–149, 1987.

Schulenberg, J.; O'Malley, P.M.; Bachman, J.G.; and Johnston, L.D. Getting drunk and growing up: Trajectories of frequent binge drinking during the transition to young adulthood. *J Stud Alcohol* 57:289–304, 1996.

Schulz, R., and Heckhausen, J. A lifespan model of successful aging. *Am Psychol* 51:702–714, 1996.

Staudinger, U.M.; Marsiske, M.; and Baltes, P.B. Resilience and reserve capacity in later adulthood: Potentials and limits of development across the life span. In: Cicchetti, D., and Cohen, D.J., eds. *Developmental Psychopathology, Volume 2: Risk, Disorder and Adaptation.* New York: John Wiley & Sons, 1995. pp. 801–847.

Substance Abuse and Mental Health Services Administration. *National Household Survey on Drug Abuse: Main Findings 1993.* DHHS Pub. No. (SMA)95–3020. Rockville, MD: the Administration, 1995.

Substance Abuse and Mental Health Services Administration. *National Household Survey on Drug Abuse: Main*

Findings 1994. DHHS Pub. No. (SMA)96–3085. Rockville, MD: the Administration, 1996.

Treas, J. Older Americans in the 1990s and beyond. *Population Bull* 2:2–46, 1995.

Treno, A.J.; Parker, R.N.; and Holder, H.D. Understanding U.S. alcohol consumption with social and economic factors: A multivariate time series analysis, 1950-1986. *J Stud Alcohol* 54:146–156, 1993.

U.S. Bureau of the Census. *Sixty-Five+ in the United States.* Current Population Reports Series P-23, No. 190. Washington, DC: U.S. Government Printing Office, 1996.

Wang, C.; Liu, W.T.; Zhang, M.; Yu, E.S.H.; Xia, Z.; Fernandez, M.; Lung, C.; Xu, C.; and Qu, G. Alcohol use, abuse, and dependency in Shanghai. In: Helzer, J.E., and Canino, G.J., eds. *Alcoholism in North America, Europe, and Asia.* New York: Oxford University Press, 1992. pp. 264–288.

Wilsnack, S.C. Patterns and trends in women's drinking: Recent findings and some implications for prevention. In: Howard, J.M.; Martin, S.E.; Mail, P.D.; and Hilton, M.E., eds. *Women and Alcohol: Issues for Prevention Research.* Research Monograph 32. NIH Pub. No. 96–3817. Bethesda, MD: National Institute on Alcohol Abuse and Alcoholism, 1996. pp. 19–64.

Zucker, R.A.; Fitzgerald, H.E.; and Moses, H.D. Emergence of alcohol problems and the several alcoholisms: A developmental perspective on etiologic theory and life course trajectory. In: Cicchetti, D., and Cohen, D.J., eds. *Developmental Psychopathology, Volume 2: Risk, Disorder and Adaptation.* New York: John Wiley & Sons, 1995. pp. 677–711.

Zucker, R.A.; Ellis, D.A.; Fitzgerald, H.E.; Bingham, C.R.; and Sanford, K.P. Other evidence for at least two alcoholisms. II. Life course variation in antisociality and heterogeneity of alcoholic outcome. *Dev Psychopathol* 8:831–848, 1996.

Zucker, R.A.; Davies, W.H.; Kincaid, S.B.; Fitzgerald, H.E.; Reider, E.E.; and Bingham, D.R. Conceptualizing and scaling the developmental structure of behavior disorder: The lifetime alcohol problems score as an example. *Dev Psychopathol* 9:453–471, 1997.

Chapter 2

Methodological Issues in Survey Research With Older Americans

A. Regula Herzog, Ph.D.

Survey research methods may be defined as those social science research methods that rely on standardized questionnaires or survey instruments to assess a probability sample representing the target population and that utilize multivariate analytical techniques to analyze the resulting data (Rossi et al. 1983). Studied topics may include attitudes, behaviors, health characteristics, socioeconomic status, and many others—essentially every topic about which an individual can be expected to be able to report reasonably accurately. Standardized questionnaires are a mainstay, not only for surveys, but also for clinical and epidemiologic investigations. And whereas the concern about representativeness might seem to be specific to representative surveys, it is increasingly becoming a concern for clinical trials and their often nonrepresentative subject populations. Therefore, methodological issues discussed in this chapter have broader applicability than survey research.

Investigations of survey research methods and their use for surveying older persons are relatively recent (Lawton and Herzog 1989; Rodgers and Herzog 1992; Wallace and Woolson 1992). In this chapter I briefly describe the major sources of error in surveys, summarize the types of errors that are of particular concern in surveys of older persons, and discuss some methods to minimize these errors.

An example from my work in which substantive and methodological issues could potentially intersect sets the stage for this chapter. Robert

A.R. Herzog, Ph.D., is senior research scientist at the Institute for Social Research and the Institute of Gerontology and adjunct professor of psychology, University of Michigan, 1312 ISR Building, Box 1248, Ann Arbor, MI 48106–1248.

Wallace and I participated in the design and the analysis of the Asset and Health Dynamics Among the Oldest Old (AHEAD) nationwide survey of some 7,500 Americans age 70 and over, and we recently analyzed cognitive functioning assessed in this survey by a free recall measure and a mental status–type measure (Herzog and Wallace 1997). One of the significant predictors of cognitive performance among older Americans was alcohol consumption: Those older Americans who reported drinking moderately (i.e., up to 2 drinks per average day, but most of them drank less than 1 drink per day) achieved cognitive scores that were almost half a point better on the 20-point recall score and about one-third of a point better on the 15-point mental status–type score than those who did not drink at all, a statistically significant difference (table 1). Even the heavy drinkers (i.e., 3 or more drinks per average day) scored better than the nondrinkers, although the latter differences were not always statistically significant because of the small numbers of heavy drinkers. This effect was obtained even after controlling for demographic and health factors. In this chapter, I intend to probe whether a result like this can be interpreted at face value or whether alternative methodological interpretations are suggested.

SURVEY ERRORS

The question has often been raised about whether high-quality survey data can be collected from older adults, because physical and cognitive impairment and other age-related differences may affect their ability or willingness to respond. (For a detailed discussion of such age-related differences, see Herzog and Rodgers 1992.) One way to think about the feasibility of a survey is to think about the possible errors that might occur because of the age of the respondent. As originally proposed by Kish (1965), and subsequently developed

Table 1. Alcohol Consumption and Cognitive Functioning in the AHEAD Survey of Americans 70+, 1993–94.

	Cognitive Measures		
Alcohol Consumption	Mental Status (0–15)	Noun Recall (0–20)	Total Cognitive Score (0–35)
No current drinking	reference category	reference category	reference category
Up to 2 drinks/day	0.34**	0.40**	0.74**
3 drinks+/day	0.48*	0.26	0.75

Note: Table entries are unstandardized ordinary least squares regression coefficients of cognitive scores on alcohol consumption. Effects of sociodemographic, socioeconomic, and health status characteristics are statistically controlled.
*$p < 0.05$. **$p < 0.01$.
Source: Adapted from Herzog, A.R., and Wallace, R.B. Measures of cognitive functioning in the AHEAD Study. *J Gerontol Soc Sci* 52B (Special Issue):37–48, 1997.

by Groves (1989), *survey error* may be defined as the deviation of an obtained survey measure from the true value of the population. Survey errors can be categorized into coverage errors, sampling errors, survey nonresponse errors, item nonresponse errors, and response errors. Each of these errors has variable and bias components. A variable error is random with an expected value of zero. A bias is a systematic distortion introduced by survey procedures or personal characteristics, and it adds a constant factor to survey measurement. Each of these error categories is discussed in the following sections.

Errors Related to Drawing a Sample of Older Americans

Coverage errors may occur when a sample to represent a population is drawn from a list or "frame" that does not include all members of the target population (Groves 1989). Although there is not much evidence that sampling frames of housing units typically used in probability samples of the adult American population cover older Americans less completely than they do other age groups, there are two qualifications to this lack of coverage differences. First, household samples exclude institutionalized members of the older population. Approximately 5 percent of Americans over 65 years of age are institutionalized at any one point in time, and this part of the elderly population tends to be more disabled than those living in the community (Rosenwaike 1985). Second, household samples of the older population are relatively expensive to draw. Because only about one-quarter of all U.S. households contain an older person, a great number of households must be screened, a fact that considerably increases costs of drawing a sample of the older population. Luckily, the Medicare enrollment database, which covers over 95 percent of the U.S. older population, provides an alternative frame from which to sample the elderly. A recent paper demonstrates that the Medicare data produce samples of older Americans living in the community that are comparable to area probability household samples at a small fraction of the cost of household samples (Rodgers 1996). An added advantage of the Medicare database is that it also covers institutionalized older adults.

Sampling bias arises when inappropriate (e.g., nonrandom, selective, or convenient) selection procedures are used (Moser and Kalton 1972). For example, small studies of the elderly often recruit their subjects from retirement communities or clinical settings (Camp et al. 1989). These are nonprobability selection procedures that can lead to biased results. *Sampling variance*, on the other hand, is a random source of error that is due to the fact that only a sample and not the entire target population is observed (Groves 1989). Sampling variances for probability samples may be estimated from the survey data and used to develop confidence intervals for estimates of survey statistics. Whereas the size of the sampling variance is inversely related to the sample size, the bias does not decrease with increasing sample size, but only with improved

selection procedures. Both types of sampling errors arise from sampling techniques and are not related systematically to the age of the respondent.

Survey Nonresponse Error and Surveys of the Aged

Nonresponse errors arise when a significant proportion of persons eligible for the sample do not participate in the survey—reflected in a low response rate—and when these persons systematically differ from those who do participate (Moser and Kalton 1972; Platek and Gray 1986).

Surveys of older Americans tend to have more problems with nonresponse than surveys of the rest of the population. First, there is evidence that response rates are lower among older respondents (DeMaio 1980; Herzog and Rodgers 1988*a*). A typical example from Herzog and Rodgers (1988*a*) shows that the response rate in the National Election Study, which was around 70 percent or above among young and middle-age adults, dropped to 65 percent among the 75- to 84-year-olds and to 50 percent among those 85 years old and older.

Second, there are suggestions that older nonrespondents are less healthy than older respondents (Herzog and Rodgers 1982). Although it is difficult to evaluate the nature of nonresponse because nonrespondents do not provide much information about themselves, much imperfect evidence converges on this point. In the 1984 Study of Michigan Generations, health problems were cited by about one-third of all older nonrespondents as the reason for nonparticipation; by comparison, less than one-tenth of younger nonrespondents cited health reasons (table 2). Direct comparisons of older nonrespondents or panel dropouts with older respondents or continuing panel members suggest that the nonrespondents and dropouts are less healthy (Herzog 1987; Cooney et al. 1988; Cohen et al. 1989). Based on these findings, it may be concluded that many of the surveys of the elderly focusing on health and functioning overrepresent healthy individuals and thus portray a positively biased picture of health in the older population. Applying this observation to the AHEAD study finding on drinking and cognitive functioning cited earlier in this chapter (Herzog and Wallace 1997) leads to the suggestions that the most cognitively and physically impaired are not well represented in the AHEAD sample and that a different relationship between cognition and drinking might emerge if the impaired group could be included in a more representative form.

To summarize, sampling frames with good coverage of the survey population, the use of proper sampling techniques, and high response rates are important methodological features for a survey that is expected to provide a representative picture of America's elderly. Unlike in younger segments of the population, samples of the elderly tend to have a "healthy" bias unless special precautionary measures are taken.

Item Nonresponse Error and Surveys of the Aged

Item nonresponse error arises when respondents who do participate in the

Table 2. Reasons Why Some Eligible Respondents Could Not Be Interviewed in the Study of Michigan Generations, 1984.

Reason[a]	Age Group	
	20–59 (n = 379)[b]	60+ (n = 752)[b]
Health problems	8%	32%
Concerns about participation	7%	8%
Concerns about survey	47%	41%
Not available	40%	22%

Note: Table entries are percentages of nonrespondents giving specific reasons.
[a]Multiple reasons are possible.
[b]Number of nonrespondents.
Source: Adapted from Moles, E.L. Perceptions of the interview process. *Gerontologist* 27:41A, 1987.

survey elect not to answer certain survey questions, typically by saying that they do not know the answer or that they prefer not to answer. Item nonresponse may result in biased rates and relationships for questions with missing answers if those respondents who do not respond differ in systematic ways from those who do respond.

Surveys of the elderly typically face a more substantial level of item nonresponse than surveys of younger persons (Francis and Busch 1975; Herzog and Rodgers 1982). Further, older persons who leave more missing answers differ systematically from those elderly with fewer missing answers in terms of their physical, cognitive, and psychological health (Ferber 1966; Glenn 1969; Francis and Busch 1975; Herzog and Rodgers 1982; Colsher and Wallace 1989*a*). For example, in the AHEAD survey the cognitive performance of those respondents who did not provide an answer on the serial 7s test, a particularly challenging cognitive test, was lower on other cognitive tests than the performance of those completing the serial 7s with a good score (table 3) (Herzog and Wallace 1997).

A recent paper by Knauper and her colleagues (Knauper et al. 1997) demonstrates that cognitively impaired older persons are more likely to avoid answering survey questions than are their cognitively more able counterparts and that this difference is particularly pronounced for difficult survey questions—that is, questions that provide minimal introduction, contain relatively ambiguous wording, and require quantitative or retrospective reports. Statistically significant interactions between cognitive impairment and question difficulty confirm this point.

To summarize, older adults are less likely than younger adults to answer a survey question—particularly a complicated question—and those older adults who do not give an answer appear to be more likely to be cognitively and otherwise impaired than those older adults who do answer. This systematic loss of cognitively less able older respondents on answers to certain

questions could introduce further bias into point estimates and relationships unless countermeasures such as imputation are taken. (Imputation, a method of assigning values to missing answers based on other characteristics of the respondent, is discussed later in this chapter.) In the analysis of cognitive performance and alcohol use in the AHEAD survey, Herzog and Wallace (1997) imputed the missing data on cognitive scores due to refusals to submit to the cognitive tests. Assuming the imputation procedures are valid, we believe that the observed relationship is not biased by item nonresponse.

Response Errors and Surveys of the Aged

Response error, or *measurement error,* arises when answers given to certain survey questions are inaccurate, resulting from the reluctance of the survey respondents to provide honest answers or from their inability to give accurate answers (Cannell and Camburn 1991). It is typically hypothesized that response errors are more substantial in older respondents because of the decline in memory and cognitive functioning associated with aging—in other words, because of an inability to report. My colleagues and I have examined the accuracy of many survey answers established by different methods and in different data sets. We have found little evidence that age is an important factor in response error (Herzog and Dielman 1985; Rodgers and Herzog 1987; Rodgers et al. 1992). Table 4 shows the level of disagreement between answers to survey questions on automobile ownership, driver's licensing, and voting behavior in the Study of Michigan Generations, and the relevant public records (Rodgers and Herzog 1987). Contrary to what might be expected, the level of disagreement was actually lower among older than younger respondents in most cases.

These findings should be interpreted in the context of the nonresponse errors

Table 3. Comparison of Respondents Who Refused the Serial 7s Test With Those Who Completed the Test With Low, Medium, and High Performances, AHEAD Survey of Americans 70+, 1993–94.

		Cognitive Measure	
Response to Serial 7s test	*n*	Mental Status (0–10)	Self-Rated Memory (1–5)
Refused first trial	733	6.83	2.84
Refused later trials	669	8.43	2.99
Completed the test with:			
Low scores (0–2)	1,015	8.30	3.07
Medium scores (3–4)	1,841	9.32	3.18
High scores (5)	1,915	9.50	3.29

Note: Table entries are mean scores on cognitive measures.
Source: Adapted from Herzog, A.R., and Wallace, R.B. Measures of cognitive functioning in the AHEAD study *J Gerontol Soc Sci* 52B (Special Issue): 37–48, 1997.

reported in the preceding section: If older people with the worst cognitive functioning do not participate in the survey or fail to answer specific questions, their response errors cannot be evaluated. It is possible that those respondents, had they participated, would have provided lower quality data. In other words, attempts to reduce survey error due to nonresponse may well increase error due to inaccurate responding. However, the findings are also consistent with a larger body of evidence suggesting that respondent characteristics such as gender, race, education, and age are of relatively minor importance in explaining response error compared with features of the study and question design (Bradburn 1983).

Which design features, then, are important? Research has highlighted the significance of question wording (Schuman and Presser 1981); question order and context effects (Schwarz and Sudman 1992); the nature of the information that is requested, particularly its distinctiveness (Means and Loftus 1991), its regularity (Menon 1994), its frequency (Burton and Blair 1991), and its recency (A.F. Smith et al. 1991); and whether response categories are given and how many (Andrews 1984; Rodgers et al. 1992), to name just a few. Comprehensive discussions of the impact of design features on response error are provided by Andrews (1984) and Bradburn (1983). The available evidence suggests that providing answers to survey questions requires cognitive judgment, inference, and memory processes, and that these processes are substantially affected by the design of the study and its instruments (Bradburn et al. 1987).

Regarding the previously cited example of the finding on alcohol use and cognition in the AHEAD survey (Herzog and Wallace 1997), we might hypothesize that the observed cognitive performance differences between heavy, moderate, and nondrinkers could be an artifact of the error-proneness of the measurement of the frequency of drinking. This is

Table 4. Accuracy of Survey Answers, by Age of Respondent, in the Study of Michigan Generations, 1984.

	Age Group		
Question	**< 60**	**60–69**	**70+**
Auto make?	11.2%	5.5%	1.0%
Auto year?	14.7%	7.9%	6.8%
Driver's license?	8.9%	5.5%	4.6%
Voted 1980 presidential election?	22.6%	16.7%	21.7%
Voted 1982 congressional election?	22.3%	18.3%	17.9%

Note: Table entries are percentage disagreement between respondent reports and official records.
Source: Adapted from Rodgers, W.L., and Herzog, A.R. Interviewing older adults: The accuracy of factual information. *J Gerontol* 42:387–394, 1987.

because the number of drinks consumed on an average day may reflect a routine behavior that, for those who drink at all, is not rare or significant enough to be distinctive and thus well remembered. Or, the number of drinks may reflect a behavior that is irregular enough not to lend itself to a simple estimation procedure. Menon (1994) discussed behavioral frequency reports from this perspective in more detail. (See also Herzog 1994 for a similar argument regarding estimates of productive behaviors.)

Another possible response error that needs to be considered in the measurement of alcohol consumption arises from the reluctance of the respondent to report heavy alcohol use. My colleague Robert Wallace hypothesizes that the apparent negative effect of not drinking on cognitive performance indicated by our finding in the AHEAD survey may be due to heavy drinkers who have not admitted to drinking and, thus, are counted and analyzed together with the nondrinkers (Colsher and Wallace 1989*b*).

An example related to another behavior perceived to be undesirable—uncontrolled loss of urine—may be found in one of our recent studies (Herzog et al. 1998). In a telephone survey on urinary incontinence, we experimentally tested whether encouragement to be as forthcoming as possible about this rather disagreeable condition affected the prevalence. A random subset of respondents received a survey question about urine loss that was preceded by an introduction aimed at minimizing reluctance to report by emphasizing the importance of accurate information, while another random subset received the question without introduction. The random subset of respondents who were given the introduction reported a rate about twice as high as the random subset whose question was not likewise introduced. Our interpretation of this finding is that older adults tend not to disclose information about urine loss unless explicitly encouraged to do so because they perceive urine loss as an embarrassing and undesirable behavior.

In summary, while plenty of evidence exists for errors occurring in survey answers, the evidence does not implicate age of respondent as a major factor affecting the level of error.

Survey Procedures to Improve Data Quality

How might survey errors be reduced? Several procedures show promise in reducing survey errors in surveys with older and younger adults.

An important feature in reducing nonresponse in surveys of the elderly is the use of proxy *respondents*—or at least an *assistor*—for those who are too frail or unwilling to report for themselves. Put differently, a higher response rate can be achieved among the old if proxy respondents are accepted. For example, in the 1984 Supplement on Aging (SOA) of persons over age 54, 9 percent were represented by a proxy compared with 27 percent among those over the age of 84 (Fitti and Kovar 1987). This response rate advantage must be balanced against the quality of the proxy's response. Although the qual-

ity of proxy reports is often assumed to be lower than the quality of self-reports, the relevant evidence is equivocal and difficult to evaluate because of design limitations (Moore 1988). The best practice is probably to adopt a standard cognitive screen and automatically switch to an assisted or proxy interview if the respondent appears too impaired by a preestablished criterion. The choice of whom to select as proxy may be as important a decision as whether to use a proxy at all, and this decision should take into consideration the nature of the relationship between proxy and respondent as well as the nature of the information to be collected (Bassett et al. 1990; Herzog and Rodgers 1992).

An extended field period is also important in reaching the broadest range of respondents, because it permits the postponement of interviewing until some of the sick or indisposed have recovered.

A mixed mode strategy of data collection using a combination of face-to-face and telephone interviews might also be helpful in reaching the largest and most representative set of respondents, thereby reducing nonresponse (Tennstedt and McKinlay 1987). A researcher might start with a telephone call—which is cheaper and less imposing—and only conduct a personal interview with respondents who require such a visit. Of course, a mixed mode strategy assumes that the telephone and face-to-face modes are equivalent. Despite widespread concerns over telephone interviews with the elderly, it appears that this mode yields no more response error than a face-to-face survey and that it is not particularly difficult to implement (Groves and Kahn 1979; Herzog and Rodgers 1988). Other design features that are useful in reducing nonresponse are *monetary incentives* (Singer et al. 1996) and *sponsorship* by trusted organizations or individuals (Rodgers and Herzog 1992).

With procedures like these in place, the Survey Research Center (SRC) of the Institute for Social Research at the University of Michigan has been able to attain a 80 percent response rate in the nationwide AHEAD survey of persons 70 years old and older (Soldo et al. 1997). The Bureau of the Census and other survey organizations also have been successful with such procedures (Fitti and Kovar 1987).

In addition, complicated questions need to be simplified in order to reduce item nonresponse and response error. *Bracketing* answers to questions about financial assets has reduced the proportion of "I don't know" answers in the AHEAD study and has facilitated the imputation of missing data. By providing a set of bracketed response categories when respondents could or would not provide an exact dollar amount to a number of questions about assets, item nonresponse was reduced considerably (J.P. Smith 1997). For example, without bracketing, only three-quarters of older respondents could report the exact value of their home, two-thirds the value of their checking and saving accounts, and about half the value of their stocks and mutual funds. With bracketing, another fifth, fourth, and third of respondents reported the respective values.

Answers to a bracketed response format might then be combined with unbracketed answers that are recoded into the same brackets. Alternatively, the brackets might be used as a first step in imputing the actual dollar values for those respondents who offer only a bracketed response.

Imputation of missing answers should be considered for all variables with a substantial proportion of missing values. (See Little and Schenker 1995 for a discussion of imputation procedures of varying complexity.) In the investigation of the relationship between alcohol use and cognitive performance in the AHEAD survey, we observed about 20 percent missing scores on the serial 7s measure. However, based on the evidence of relatively low scores on the remaining mental status–type measures and on self-assessed memory by those respondents with missing recall scores (see table 3), we imputed the missing recall scores with low recall scores. This represents a very simple imputation strategy; a more complex strategy is being developed.

To minimize response error, the *formulation of response categories* must provide sufficient variation (Andrews 1984). One of our studies demonstrates that seven to nine categories are optimal for response quality among those over as well as under 60 years of age (Rodgers et al. 1992). A particular format that has been found

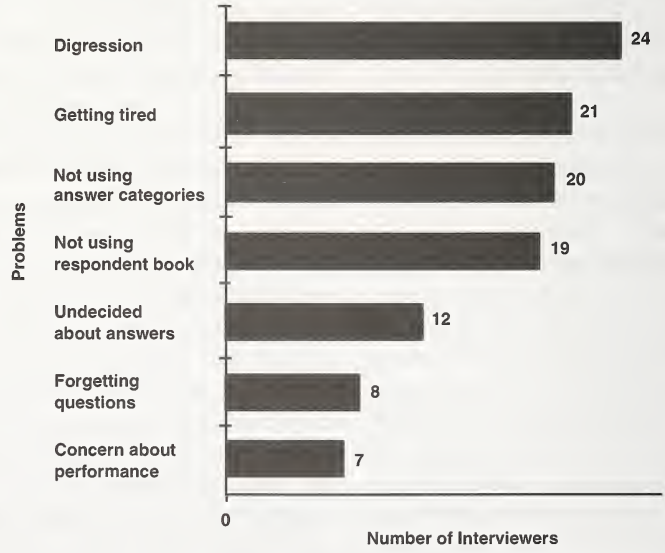

Figure 1. Interviewer reports about problems encountered more frequently with older than with younger respondents in the Study of Michigan Generations, 1984. Adapted from Moles, E.L. Perceptions of the interview process. *Gerontologist* 27:41A, 1987.

to aid in the presentation of a relatively large number of categories, thereby reducing response error, unfolds the categories in a stepwise fashion (Rodgers et al. 1992). Thus, a 7-point question about life satisfaction might ask first whether the respondent is "satisfied," "dissatisfied," or "neither." If the answer to this first question is "satisfied," the next question might then ask whether the respondent is "completely," "very," or "quite" satisfied. A similar followup question is asked if the answer to the first question is "dissatisfied."

Finally, thorough *interviewer training* is necessary, particularly for surveys with older adults. At the SRC, interviewers are prepared for special problems to be expected with older respondents. In the Study of Michigan Generations, my colleague Willard Rodgers and I queried interviewers after completion of the study about problems encountered more frequently with older than with younger respondents. Some of the major problems that the interviewers mentioned include older respondents digressing from the topic of the survey questions, not adhering to the format of the questions, and tiring easily (figure 1). Since then, interviewers of some of SRC's aging studies have been explicitly instructed that more assistance will be needed during the interviews of older adults than during those of younger adults. Interviewers are allowed to provide assistance by repeating questions and answer categories, and they do this much more with older respondents than with younger ones, as shown in findings from the Study of Michigan Generations (table 5). This fact might also explain why we have not observed more response error in interviews with the elderly: The interviewers make up for the increased difficulty experienced by the older respondents by providing more assistance.

CONCLUSION

In conclusion, survey research—defined as methods using standardized instruments and probability samples—includes many potential errors. Except for survey and item nonresponse, current evidence does not

Table 5. Interviewer Assistance by Age of Respondent in the Study of Michigan Generations, 1984.

	Age Group			
Form of Assistance	20–34 (n = 188)	35–59 (n = 287)	60–74 (n = 752)	75+ (n = 264)
Repeat question stem	2%	3%	5%	9%
Repeat answer category	2%	3%	5%	8%
Remind to use respondent book	1%	2%	4%	7%

Note: Tables entries are percentages of respondents who received each type of assistance.
Source: Adapted from Moles, E.L. Perceptions of the interview process. *Gerontologist* 27:41A, 1987.

suggest that such errors are worse in surveys of older adults than in surveys of the general population. Let me qualify this positive conclusion in two ways. First, this is not meant to imply that response errors are minimal, only that they are no worse among the elderly. Second, it is possible that response errors are minimized among the elderly by the operation of nonresponse error and by the increased assistance from interviewers.

Using the documented relationship between alcohol consumption and cognitive performance found in the AHEAD survey as an example, I have identified in this chapter a number of methodological issues in collecting information by survey methods that raise questions about the substantive explanation suggesting that alcohol consumption is positively related to cognitive performance. Other errors potentially emanating from the design and the analysis of the study, such as the need for prospective studies and for proper control for alternative explanations, were not addressed. I have also discussed a number of study features that can be used at the data collection stage to increase data quality. By paying attention to such issues in conducting surveys of the elderly and interpreting the resulting data, we are likely to come to a better understanding of our data and ultimately to conduct better surveys.

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REFERENCES

Andrews, F.M. Construct validity and error components of survey measures: A structural modeling approach. *Public Opinion Q* 48:409–442, 1984.

Bassett, S.S.; Magaziner, J.; and Hebel, J.R. Reliability of proxy response on mental health indices for aged, community-dwelling women. *Psychol Aging* 5:127–132, 1990.

Bradburn, N.M. Response effects. In: Rossi, P.H.; Wright, J.D.; and Anderson, A.B., eds. *Handbook of Survey Research.* San Diego: Academic Press, 1983. pp. 289–328.

Bradburn, N.M.; Rips, L.J.; and Shevell, S.K. Answering autobiographical questions: The impact of memory and interference on surveys. *Science* 236:157–161, 1987.

Burton, S., and Blair, E. Task conditions, response formulation processes, and response accuracy for behavioral frequency questions in surveys. *Public Opinion Q* 55:50–79, 1991.

Camp, C.J.; West, R.L.; and Poon, L.W. Recruitment practices for psychological research in gerontology. In: Lawton, M.P., and Herzog, A.R., eds. *Special Research Methods for Gerontology.* New York: Baywood Publishing, 1989. pp. 163–189.

Cannell, C.F., and Camburn, D. Development of methods to maximize

accuracy of reporting health risk behaviors in a youth survey. Paper presented at the Public Health Conference on Records and Statistics, Washington, DC, July 1991.

Cohen, S.B.; Johnson, A.E.; and Carlson, B.L. An analysis of part-year nonresponse in the household component of the national medical expenditure survey. *J Econ Soc Meas* 15:281–299, 1989.

Colsher, P.L., and Wallace, R.B. Data quality and age: Health and psychobehavioral correlates of item nonresponse and inconsistent responses. *J Gerontol Psychol Sci* 44:P45–P52, 1989*a*.

Colsher, P.L., and Wallace, R.B. Is modest alcohol consumption better than none at all? An epidemiologic assessment. *Ann Rev Public Health* 10:203–219, 1989*b*.

Cooney, T.M.; Schaie, K.W.; and Willis, S.L. The relationship between prior functioning on cognitive and personality dimensions and subject attrition in longitudinal research. *J Gerontol Psychol Sci* 43: P12–P17, 1988.

DeMaio, T.J. Refusals: Who, where and why. *Public Opinion Q* 44:223–233, 1980.

Ferber, R. Item nonresponse in a consumer survey. *Public Opinion Q* 30:399–415, 1966.

Fitti, J.E., and Kovar, M.G. The supplement on aging to the 1984 National Health Interview Survey. *Vital Health Stat* 1 No. 21. DHHS Pub. No. (PHS) 87–1323. Hyattsville, MD: National Center for Health Statistics, 1987.

Francis, J.D., and Busch, L. What we know about "I don't know." *Public Opinion Q* 39:207–218, 1975.

Glenn, N.D. Aging, disengagement, and opinionation. *Public Opinion Q* 33:17–33, 1969.

Groves, R.M. *Survey Errors and Survey Costs.* New York: John Wiley & Sons, 1989.

Groves, R.M., and Kahn, R.L. *Surveys by Telephone.* New York: Academic Press, 1979.

Herzog, A.R. Nonresponse in sample surveys of older adults. *Gerontologist* 27:41A, 1987.

Herzog, A.R. Presentation given at the International Conference on the Measurement and Valuation of Unpaid Work, Ottawa, August 1994.

Herzog, A.R., and Dielman, L. Age differences in response accuracy for factual survey questions. *J Gerontol* 40:350–357, 1985.

Herzog, A.R., and Rodgers, W.L. *Surveys of Older Americans: Some Methodological Investigations. Final Report to the National Institute on Aging (Grant No. AG02038).* Ann Arbor MI: University of Michigan, Survey Research Center and Institute of Gerontology, 1982.

Herzog, A.R., and Rodgers, W.L. Age and response rates to interview sample surveys. *J Gerontol Soc Sci* 43:S200-S205, 1988*a*.

Herzog, A.R., and Rodgers, W.L. Interviewing older adults: Mode comparisons using data from a face-to-face survey and a telephone survey. *Public Opinion Q* 47:405–418, 1988*b*.

Herzog, A.R., and Rodgers, W.L. The use of survey methods in research on older Americans. In: Wallace, R.B., and Woolson, R.F., eds. *The Epidemiologic Study of the Elderly.* New York: Oxford University Press, 1992. pp. 60–90.

Herzog, A.R., and Wallace, R.B. Measures of cognitive functioning in the AHEAD study. *J Gerontol Soc Sci* 52B (Special Issue):37–48, 1997.

Herzog, A.R.; Diokno, A.C.; Fultz, N.H.; and Hsu, W. *Incontinence Epidemiology: Substantive and Method Issues.* Final Report to National Institute of Diabetes and Digestive and Kidney Diseases. Ann Arbor, MI, March 1998.

Kish, L. *Survey Sampling.* New York: John Wiley & Sons, 1965.

Knauper, B.; Belli, R.F.; Hill, D.H.; and Herzog, A.R. Question difficulty and respondents' cognitive ability: The effect on data quality. *J Off Stat* 13:181–199, 1997.

Lawton, M.P., and Herzog, A.R. *Special Research Methods for Gerontology.* New York: Baywood Publishing, 1989.

Little, R.J.A., and Schenker, N. Missing data. In: Arminger, G.; Clogg, C.C.; and Sobel, M.E., eds. *Handbook of Statistical Modeling for the Social and Behavioral Sciences.* New York: Plenum Press, 1995.

Means, B., and Loftus, E.F. When personal history repeats itself: Decomposing memories for recurring events. *Appl Cogn Psychol* 5:297–318, 1991.

Menon, G. Judgments of behavioral frequencies: Memory search and retrieval strategies. In: Schwarz, N., and Sudman, S., eds. *Autobiographical Memory and the Validity of Retrospective Reports.* New York: Springer, 1994. pp. 161–172.

Moles, E.L. Perceptions of the interview process. *Gerontologist* 27:41A, 1987.

Moore, J.C. Self/proxy response status and survey response quality: A review of the literature. *J Off Stat* 4:155–172, 1988.

Moser, C.A., and Kalton, *G. Survey Methods in Social Investigations.* 2d ed. New York: Basic Books, 1972.

Platek, R., and Gray, G.B. On the definition of response rates. *Survey Methodol* 12:17–27, 1986.

Rodgers, W.L. Comparisons of two sampling frames for surveys of the oldest old. In: Warnecke, R.B., ed. *Health Survey Research Methods.* DHHS Publication No. (PHS) 96–1013. National Center for Health Statistics, Centers for Disease Control and Prevention, Public Health Service, U.S. Department of Health and Human Services, 1996. pp.117–122.

Rodgers, W.L., and Herzog, A.R. Interviewing older adults: The accuracy of factual information. *J Gerontol* 42:387–394, 1987.

Rodgers, W.L., and Herzog, A.R. Collecting data about the oldest old: Problems and procedures. In: Suzman, R.M.; Willis, D.P.; and Manton, K.G., eds. *The Oldest Old.* New York: Oxford University Press, 1992. pp. 135–156.

Rodgers, W.L.; Andrews, F.M.; and Herzog, A.R. Quality of survey measures: A structural modeling approach. *J Off Stat* 8:251–275, 1992.

Rosenwaike, I. *The Extreme Aged in America: A Portrait of an Expanding Population.* Westport, CT: Greenwood Press, 1985.

Rossi, P.H.; Wright, J.D.; and Anderson, A.B. Sample surveys: History, current practice, and future prospects. In: Rossi, P.H.; Wright, J.D.; and Anderson, A.B., eds. *Handbook of Survey Research.* San Diego: Academic Press, 1983. pp. 1–20.

Schuman, H., and Presser, S. *Questions and Answers in Attitude Surveys.* New York: Academic Press, 1981.

Schwarz, N., and Sudman, S., eds. *Context Effects in Social and Psychological Research.* New York: Springer-Verlag, 1992.

Singer, E.; Gebler, N.; Raghunathan, T.; Van Hoewyk, J.; and McGonagle, K. The effect of incentives on response rates in face-to-face, telephone, and mixed mode

surveys. Paper presented at the Annual Meeting of the American Association for Public Opinion Research, Salt Lake City, UT, May 1996.

Smith, A.F.; Jobe, J.B.; and Mingay, D.J. Retrieval from memory of dietary information. *Appl Cogn Psychol* 5:269–296, 1991.

Smith, J.P. Wealth inequality among older Americans. *J Gerontol Soc Sci* 52B (Special Issue):74–81, 1997.

Soldo, B.J.; Hurd, M.D.; Rodgers, W.L.; and Wallace, R.B. Asset and health dynamics among the oldest old: An overview of the AHEAD baseline. *J Gerontol Soc Sci* 52B (Special Issue):1–20, 1997.

Tennstedt, S.L., and McKinlay, J.B. Choosing the most appropriate field approach for older populations: The case for mixed-mode surveys. In: *Proceedings of the 1987 Public Health Conference on Records and Statistics.* USDHHS Publication No. (PHS) 88–1214. Hyattsville, MD: 1987.

Wallace, R.B., and Woolson, R.F., eds. *The Epidemiologic Study of the Elderly.* New York: Oxford University Press, 1992.

Chapter 3

Drinking in an Older Population: Cross-Sectional and Longitudinal Data From the Australian Twin Registry

Kathleen K. Bucholz, Ph.D., Andrew C. Heath, D. Phil., Pamela A. F. Madden, Ph.D., Wendy S. Slutske, Ph.D., Dixie J. Statham, M.A., Michael P. Dunne, Ph.D., and Nicholas G. Martin, Ph.D.

Following a discussion of general and methodological issues in research on the use of alcohol by older individuals, this chapter presents new data from a sample of twins age 50 and older from the Australian Twin Registry, from both cross-sectional and longitudinal perspectives. The longitudinal data on alcohol consumption are then analyzed in a biometrical genetic framework, modeling drinking behaviors over time as a function of genetic and environmental effects.

GENERAL ISSUES

The drinking patterns of older individuals and how they change with age are topics stimulating recent research interest, for several reasons. Alcohol use, not just heavy or dependent use, may be viewed as a health risk in older individuals, whose use of prescription medication may interact adversely

K.K. Bucholz, Ph.D., is research associate professor; A.C. Heath, D. Phil., is professor; and P.A.F. Madden, Ph.D., is research instructor in the Department of Psychiatry, Washington University School of Medicine, 4940 Children's Place, St. Louis MO 63110. W.S. Slutske, Ph.D., is assistant professor in the Department of Psychology, University of Missouri, 13 Macalester Hall, Columbia, MO 65211. D.J. Statham, M.A., is clinical psychologist in Epidemiology and Population Health, Queensland Institute of Medical Research, Post Office Royal Brisbane Hospital, Herston, Queensland 4029, Australia. M.P. Dunne, Ph.D., is senior lecturer in the School of Public Health, Queensland University of Technology, Kelvin Grove 4059, Queensland, Australia. N.G. Martin, Ph.D. is professor in Epidemiology and Population Health, Queensland Institute of Medical Research, Post Office Royal Brisbane Hospital, Herston, Queensland 4029, Australia.

with alcohol. Medication use among the elderly is the norm. One study found that 90 percent of elderly persons were using some medication, either prescription or over-the-counter (Dufour et al. 1992). A report from four population-based cohorts of community-dwelling elderly indicated that 60 to 68 percent of men and 68 to 78 percent of women used prescription medications, while slightly lower percentages of each gender (52 to 68 percent of men and 64 to 76 percent of women) used nonprescription medications (Chrischilles et al. 1992). Furthermore, older individuals often take several different medications at the same time. Estimates of the number of medications used concurrently have ranged from 1.5 to 6.1 for prescription and from 1.3 to 4.6 for over-the-counter medications (Pollow et al. 1994). Medications with a high potential for adverse reactions with alcohol that are commonly taken by the elderly include analgesics, antihypertensives, anticoagulants, diuretics, antiarthritics, and psychoactive medications (Forster et al. 1993).

Adverse consequences of alcohol use by the elderly are not limited to interactions with medications. Exacerbation of sleep difficulties, elevation of systolic and diastolic blood pressure, and detrimental effects on bone mineral metabolism are other negative effects (Dufour et al. 1992; Tell et al. 1994). Moreover, because of the decrease in percentage of body water content with age, alcohol may have a more potent effect in the elderly, with high serum ethanol levels being achieved with small amounts of alcohol (Atkinson 1990).

Complicating the interpretation of the potential for adverse consequences of alcohol use in the elderly is evidence of the potential beneficial effects of alcohol use. In contrast to most recreational drugs, abstention from alcohol may not always be beneficial for the elderly. Low to moderate alcohol consumption among people age 60 years and over offers some protection against cardiovascular disease and reduces all-cause mortality, especially for individuals with multiple risk factors for heart disease (Doll et al. 1994; Fuchs et al. 1995; Serdula et al. 1995). This effect is J-shaped, with low to moderate drinkers having a small survival advantage over nondrinkers, but with consumption above one or two drinks per day posing significant risks to health, especially for women. These risks primarily include various cancers, liver cirrhosis, brain damage, and accidents (Friedman and Klatsky 1993; Rehm and Sempos 1995).

Although the findings regarding heart disease have been embraced enthusiastically by some, particularly the alcoholic beverage industry, others have urged caution in promulgating the message that consistent moderate drinking is beneficial for elders (Anderson et al. 1996). Much of the available data are derived from mortality studies; less is known about the impact of drinking by the elderly upon their general morbidity, including those adverse interactions with medications already described. Both positive and negative effects of drinking by the

elderly upon social and psychological functioning ought to be considered, since alcohol use at low to moderate levels may have beneficial effects upon mood and socialization, but moderate levels of drinking may increase the risk that individuals will develop alcohol dependence (Midanik 1995).

The magnitude of late-onset alcoholism and problem drinking, defined as onset after the age of 40 (Atkinson et al. 1990), provides another motivation for studying alcohol use in the elderly.[1] The management of these cases, and in particular, their clinical course and how they respond to treatment, is of clinical and societal interest. A little-appreciated benefit to be derived from studying elderly alcoholics and problem drinkers is that such research can assist health planners and policymakers in anticipating the demands on society's resources placed by younger alcoholics as they age. These aging alcoholics are, by several accounts, a burgeoning group (Reich et al. 1988; Kessler et al. 1994).

METHODOLOGICAL ISSUES

From a research perspective, drinking in the elderly introduces a number of challenging methodological issues. First is the issue of drinking itself. The definitions of drinking and the timeframes over which the drinking has occurred have varied from study to study. Even the definition of "abstainer" has not been uniform. In some surveys, "abstention" has reflected current drinking practices, with those who have not had any alcohol in the last 12 months being classified as abstainers along with those who have never had alcohol (Hilton 1986). In addition, the definition of "abstainer" sometimes includes people who drink a little (up to 11 drinks per year) (Malin et al. 1983; Hilton 1991). These variations in definitions are evident when one views results of surveys of drinking practices side by side, in which substantial shifts in proportions of drinkers from year to year may be observed (figure 1) (Bucholz et al. 1995).

Heavy drinking is another construct that is unsystematically defined and assessed in an older sample. Estimates of heavy drinking in older persons, defined variously by daily amount, weekly consumption, or maximum thresholds, have ranged from 3 to 13 percent in males and from 1 to 8 percent in females. These estimates are typically lower than those obtained for younger groups, consistent with the estimates of drinking at any level as seen in figure 1, in which results from seven surveys of drinking practices conducted from 1964 to 1984 have been compiled (Hilton 1991). The lowest proportions of both drinkers and heavy drinkers were found in the 65 and older group, for both men and women, despite the inconsistency of definitions.

Comparison of findings across studies yields quite variable estimates of current drinking, ranging from a low of 35 percent (in a largely female sample) to a high of 71 percent (for any drinking) (Cahalan et al. 1969; Guttmann 1978; Barnes 1979;

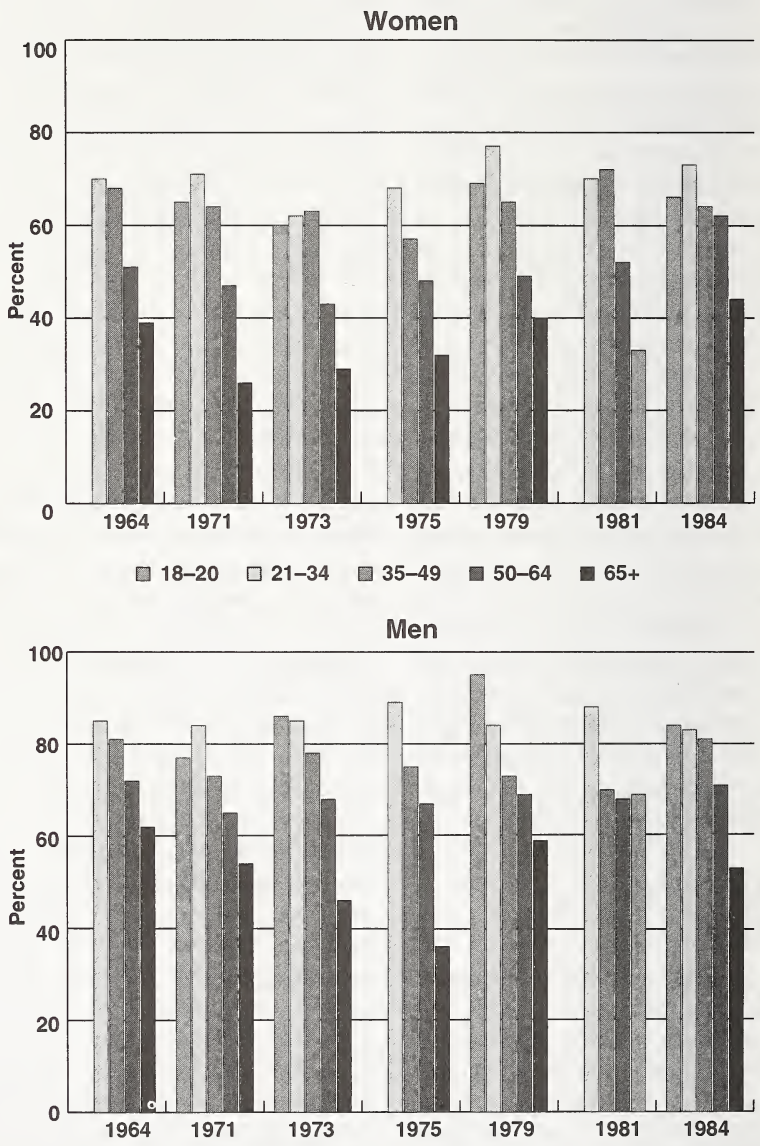

Figure 1. Proportion of drinkers by age group (data for women in top half, data for men in bottom half) from seven cross-sectional surveys conducted from 1964 to 1984. Adapted from Hilton, M.E. Trends in U.S. drinking patterns. Further evidence from the past twenty years. In: Clark, W.B., and Hilton, M.E., eds. *Alcohol in America.* Albany, NY: State University of New York Press, 1991. pp. 121–138.

Meyers et al. 1982; Goodwin et al. 1987; Busby et al. 1988; Douglass et al. 1988; Smart and Adlaf 1988; Sulsky et al. 1990; Iliffe et al. 1991; Welte and Mirand 1994). Some of the variability in these estimates reflects the nature of the study populations. There are, in fact, relatively few studies of representative samples of older individuals. Rather than being sampled as a tenable study population, older individuals often have been studied incidentally as part of a community sample in which they appear. Those studies that specifically have targeted older individuals have limited generalizability due to sampling from a single geographic region or from specialized samples, such as attenders at a particular medical practice or other samples of convenience.

The issue of how drinking changes over time is also by no means settled. Some studies suggest that drinking declines with age (Hilton 1991), while others suggest that drinking patterns are stable (Glynn et al. 1986; Adams et al. 1990) or that there are increases in drinking among older individuals over time (Gordon and Kannel 1983; Gordon and Doyle 1986; Ekerdt et al. 1989). Those studies evincing a decline in drinking with age tend to be cross-sectional in design, while those suggesting that drinking patterns are stable across the lifespan or increase with age are of longitudinal design. In fact, in only one cross-sectional study was drinking observed to increase among a sample of men age 65 or older (Malin et al. 1983), although increases in drinking with age have been observed in some followup studies. For example, the 20-year followup data of Temple and Leino (1989) suggested that drinking patterns increased over time among older cohorts of low-volume drinkers. However, the predominant pattern across all age cohorts of lighter drinkers was that drinking remained stable. Decreases in drinking were prominent only among heavier drinkers.

Although longitudinal data are most promising for untangling the effects of aging from those of historical period on drinking behavior, these studies have had some shortcomings. Some of the evidence on long-term drinking patterns derives from studies not designed specifically to study alcohol use over the long term, and thus the measures of alcohol use are relatively weak. Followup success rates for some of these studies have been low, with 60 percent or less of the target population represented in the followup (Gordon and Kannel 1983; Gordon and Doyle 1986; Adams et al. 1990). The bias due to loss of followup on estimates of drinking is unknown. Finally, women have not been included in many samples, so results from longitudinal data may not be generalizable to females.

AUSTRALIAN TWIN REGISTRY DATA

In this part of the chapter, we will attempt to address some of the shortcomings in the literature described thus far by presenting alcohol consumption data from *both* cross-sectional and longitudinal perspectives, using data from members of the Australian

Twin Registry, an ongoing volunteer registry of twins. Our goals are to present descriptive data from the cross-sectional data on drinking patterns in these older twins; to examine these patterns across time, using reports from twins who were respondents in three of the questionnaire studies; and to conduct a biometrical genetic analysis of the twin data to determine the extent to which genetic, shared, and nonshared environment influences explain drinking behaviors over time.

The twins have participated in several alcohol-related surveys since 1979, when an alcohol challenge study of younger twins (Martin et al. 1985) was undertaken. Since that time, twins from the Australian Twin Registry have been solicited for several alcohol-related surveys, including a mailed questionnaire study (1980–82), a second mailed questionnaire study (1988–89), a telephone interview survey (1992–93), and, most recently, another mailed questionnaire study (1993–95). In this report, we will include data from the three mailed questionnaire surveys, since the identical 7-day drinking diary was used in each.

The Questionnaires

Time 1 Questionnaire

Between 1980 and 1982, a questionnaire was mailed to twins who were members of the Australian Twin Registry and who were 18 years of age or older (Heath et al. 1989). This assessment will be referred to as "Time 1." Included in this questionnaire were items on alcohol consumption reported for the last week in the form of a 7-day diary, with beverage-specific amounts obtained for each day, lifetime smoking behavior, chronic health problems, sleeping patterns, women's health, personality scales, and social and political attitudes. The pairwise response rate, that is, the proportion of twin pairs where both responded, was 63.8 percent (3,808/5,967 pairs); the individual response rate (proportion of respondents among all individuals solicited for study) was 68.5 percent (8,183/11,934).

Time 2 Questionnaire

In 1988, a questionnaire was mailed to all twinships where both twins had completed the 1981 questionnaire ("Time 2") (Heath and Martin 1994). Coverage of this instrument included, in addition to the same 7-day drinking diary that was used in the Time 1 questionnaire, a list of alcohol problems (from which a Feighner diagnosis of alcoholism may be obtained [Feighner et al. 1972]), demographics, family history, cigarette consumption, sleeping patterns, leisure activities, a section on female health problems, and measurement of personality through an abridged 56-item scale. Over 80 percent of the target sample were interviewed successfully; the pairwise response rate was 78.6 percent (2,995/3,808), and the individual response rate was 83 percent (6,327/7,616).

Time 3 Questionnaire

Beginning in 1993 and continuing through 1995, twins age 50 or older who were members of the Australian

Twin Registry and who had not been studied previously in other surveys received a mailed questionnaire, in which the same 7-day drinking diary used in the two previous mailed questionnaires was included ("Time 3"). Drinking frequency and amount in the last 12 months and over different decades in their lifetime also were queried. Additional twins were recruited for this study in 1994–95, all of whom were age 50 or older and had participated in the other previously described surveys. In all, 4,562 twins (2,281 pairs) were recruited for this study, of whom 3,401 individuals (1,443 complete pairs and 515 singletons) responded. The pairwise response rate was 63.3 percent (1,443/2,281); the individual response rate was 74.6 percent (3,401/4,562). A fourth survey, this one a telephone interview rather than mailed questionnaire, was conducted in 1992–93, but it will not be discussed further since the data from this survey were not compatible with those from the mailed questionnaires.[2]

Of the 3,401 respondents at Time 3, 2,022 had not been included in the earlier surveys because they were not members of the Australian Twin Registry in 1981 when the first solicitation occurred. Of the remaining twins, 61 had participated in one other survey and 1,318 had participated in all three.

Measures of Alcohol Consumption

From the data elicited in the 7-day drinking diaries that were included in all three mailed surveys, four measures of quantity and frequency of alcohol consumption were derived. The measures described here reflect all types of alcohol. Although beverage-specific data were available, we elected to combine all types of alcohol in this report. The four measures are as follows: total alcohol consumption in the last 7 days, maximum number of drinks on any one day in the last 7 days, average number of drinks consumed per day over the last 7 days, and number of days on which alcohol was consumed in the last 7 days.

Cross-Sectional Data

Table 1 displays the demographic information from the Time 3 survey. A majority of the sample was female, the average age was 61.1, and most were currently married. The sample is relatively young, especially in comparison with other studies of elderly drinkers, with 50.5 percent ages 50–59, another 31.6 percent ages 60–69, 14.4 percent ages 70–79, and 3.5 percent age 80 or older. In terms of religious affiliation, about one-third were members of the Anglican Church, another third were members of other Protestant sects, and one-fifth were Roman Catholic; less than one-tenth reported no religious affiliation. Approximately one-third attended church frequently, but the majority of the sample reported rare attendance. Thirty-three percent were current smokers, and the majority of the sample (81.7 percent) reported that their current health was either good or very good.

Table 2 contains descriptive data on the drinking patterns of the sample derived from the 7-day drinking diary and other alcohol use questions. Data

Table 1. Demographic Data From the Australian Twin Registry.

Variable	Time 3 Survey	All Three Surveys
Gender	*N* = 3,401	*N* = 1,379
Male	30.5%	26.6%
Female	69.5%	73.4%
Mean age	*N* = 3,385	*N* = 1,371
	61.1 yr	61.6 yr
Marital status	*N* = 3,356	*N* = 1,356
Currently married	73.9%	74.3%
Living as married	1.3%	1.6%
Widowed	12.3%	12.2%
Separated/Divorced	7.4%	6.6%
Single	5.1%	5.2%
Education,	*N* = 3,232	*N* = 1,324
Mean years	10.0	10.2
Religion	*N* = 3,290	*N* = 1,326
Anglican	32.9%	34.3%
Other Protestant	31.4%	32.4%
Roman Catholic	19.6%	15.4%
Other	7.8%	9.1%
None	8.1%	8.8%
Church attendance	*N* = 2,935	*N* = 1,297
Often	34.3%	35.6%
Some	21.6%	22.0%
Rarely	44.1%	42.4%
Current health	*N* = 2,878	*N* = 1,273
Very good	36.2%	36.1%
Good	45.5%	47.4%
Fair	15.4%	13.8%
Poor	2.2%	2.3%
Very Poor	0.7%	0.6%

are reported by gender. The percentages of women and men who reported being lifelong abstainers are low in comparison to reported findings from the United States, where 23.8 percent of women and 12.4 percent of men reported lifelong abstention (Welte and Mirand 1994), although the U.S. sample was somewhat older. However, even when stratified on age, lifelong abstention was relatively rare in the Australian Twin Registry sample; for example, 15 percent of women and 9.2 percent of men ages 60–69 and 19 percent and 7.7 percent, respectively, of women and men ages 70–79 reported being lifelong abstainers. Well over 80 percent of both women and men reported having had something to drink in the last 12

Table 2. Drinking Patterns of the Australian Twin Registry Sample.

	Time 3 Survey		All Three Surveys	
	Women % (*n*)	Men % (*n*)	Women % (*n*)	Men % (*n*)
Abstainers	14.3 (2,119)	9.4 (895)	13.2 (995)	9.0 (354)
Total alcohol consumed				
7-day diary mean	4.9 (1,740)	11.0 (761)	4.9 (821)	9.6 (301)
Maximum drunk last week	1.7 (1,438)	3.4 (677)	1.7 (692)	3.1 (269)
Average number of drinks	1.1 (1,740)	2.1 (761)	1.1 (821)	1.9 (301)
Number of days drank	2.4 (1,817)	3.4 (811)	2.4 (846)	3.4 (322)
Heavy drinking				
(more than 2 per day)	11.9 (2,119)	32.0 (895)	12.2(975)	30.5 (354)
Frequency of drinking in last 12 months[1]				
Daily	21.3	30.2	20.3	26.5
3–4 times/week	13.9	21.9	15.0	23.6
Once a /week	18.0	16.1	18.6	16.4
Once a month	11.4 (1,404)	9.6 (684)	10.3 (661)	11.6 (275)
Less often	27.2	14.6	28.6	14.2
Never in last 12 months	8.2	7.2	7.3	6.9
CAGE items endorsed				
0	84.6	61.3	85.7	65.7
1–2	13.1 (1,653)	27.2 (776)	12.3 (792)	26.5 (309)
3–4	2.3	11.5	2.0	7.8

[1] Nonabstainers excluded.

months, and most reported having had something to drink at least once a week.

Taking the definition of heavy drinking proposed by Welte and Mirand (1994)—drinking more than two drinks daily—nearly 12 percent of women and 32 percent of men were classified as heavy drinkers. These rates were high in relation to a sample from Buffalo, NY (Mirand and Welte 1996), where only 6 percent of the entire sample qualified as heavy drinkers. The high percentages did not appear to be an artifact of the relatively younger sample here, because when stratified on age, the percentages remained high compared with the U.S. sample (i.e., 13.2 percent of women and 33 percent of men ages 50–59, 9.5 percent of women and 23.6 percent of men ages 60–69, and 7 percent of women and 21 percent of men ages 70–79 would be considered heavy drinkers) (figure 2). Even among those 80 or older, 2.3 percent of women and nearly 12 percent of men qualified as heavy drinkers.

On average, women drank nearly five drinks per week, while men drank over twice that amount. Nearly 20 percent of women, compared with 27 percent of men, reported being daily drinkers, and 76 percent of women and 93 percent of men reported

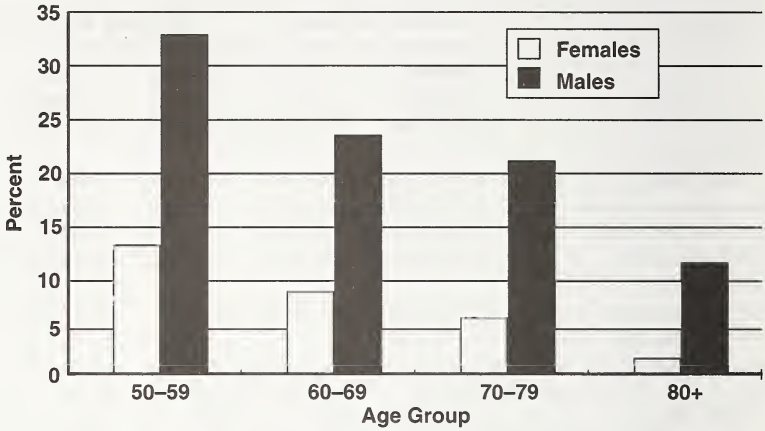

Figure 2. Proportion of heavy drinkers by age and gender in the Time 3 survey. Heavy drinking is defined as drinking more than 2 drinks per day on average.

drinking at least once in the last 12 months (our definition of current drinker). When stratified on age (figure 3), well over half of each age group, including those age 80 or older, qualified as current drinkers. The four-item CAGE was included in the interview, and 15.4 percent of women and 38.7 percent of men endorsed at least one CAGE item in their lifetime, with 23 percent overall so doing. Comparable figures for another general population sample of elderly individuals were unavailable, but in comparison with attenders at a medical clinic, where 43 percent reported one or more CAGE items (Buchsbaum et al. 1992), the proportion among the Australian twins was lower; this is not surprising, given the fact that prevalence of alcohol dependence is higher in a treated compared with an untreated sample.

Longitudinal Data

In addition to a cross-sectional snapshot, this sample also was examined from a longitudinal perspective. In particular, the Time 3 characteristics of the cross-sectional sample were evaluated using those who had participated in at least one other survey from 1981 to 1995; this sample, which consisted of 1,379 individuals, will be referred to as "the longitudinal sample." The columns entitled "All Three Surveys" in tables 1 and 2 display the data from the longitudinal sample.

While the distributions of marital status, age, education, and current health appeared to be similar in both cross-sectional and longitudinal samples, the distribution by gender, education, and religion differed between the two. In the longitudinal sample, fewer men, fewer Roman Catholics, and fewer uneducated per-

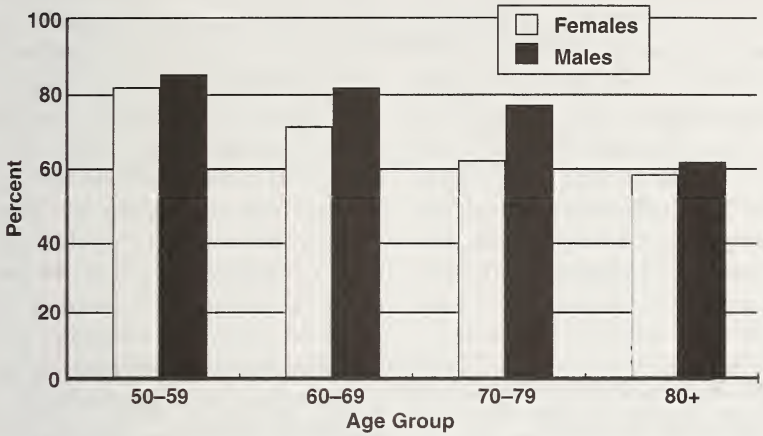

Figure 3. Proportion of current drinkers by age and gender in the Time 3 survey. Current drinking is defined as drinking at least once in the last 12 months.

sons were retained across time. These findings suggested that heavier drinkers might be underrepresented in the longitudinal sample, because heavy drinking, Roman Catholic background, and lower educational attainment all had been linked to a history of alcohol dependence in other analyses on the Australian twin sample (Heath et al. 1997, and unpublished data). The impression was borne out for men when actual drinking characteristics were examined (see table 2).

However, the evidence was different for women. Unlike their male counterparts, women in the cross-sectional sample were quite similar in sociodemographic characteristics to those in the longitudinal sample, with no differences in the four drinking measures observed. The proportion classified as heavy drinkers was slightly higher in the longitudinal (12.2 percent) compared with the cross-sectional (11.9 percent) sample. The distribution of the frequency of drinking was similar in women, whether viewed cross-sectionally or longitudinally.

The data showed that heavier drinking men were *less* likely than their non–heavy-drinking counterparts to be followed across time. This phenomenon was evident in several areas; there were downward shifts in average number of drinks consumed in a week (from 11.0 to 9.6), in maximum number of drinks consumed (from 3.4 to 3.1), and in average number of drinks per day (from 2.1 to 1.9) among men in the longitudinal compared with the cross-sectional sample. Further, decreases in the proportion of men who qualified as heavy drinkers and who were daily drinkers

were observed in the longitudinal compared with the cross-sectional group. Finally, a lower proportion of men in the longitudinal sample reported three or more CAGE items, compared with their cross-sectional counterparts (7.8 percent compared with 11.5 percent). The results of these comparisons indicate that inferences about the male sample from a longitudinal perspective must be tempered by the evidence that heavier-drinking men were less likely to be followed over time.

Correlations Among Drinking Measures Across Time

Table 3 presents the correlations between the four drinking measures across time, for women and men. The correlations suggest that drinking practices as measured by the 7-day diary in this age group were stable, with correlations of 0.5 or better for most of the measures. These data also indicate that male drinking patterns, as reflected in total drinking amounts, maximum amount drunk on any day in the last week, and average amount consumed per day, were more stable than those of women, because the correlations for males for each of these measures exceeded those for women in most instances, with the exception of the Time 2–Time 3 correlations. As would be expected, the correlations over the longer timespan (i.e., Time 1–Time 3) were lower than those for the shorter and more recent timeframe (i.e., Time 2–Time 3). Similar inferences were drawn when data were stratified into

Table 3. Correlations Among Drinking Measures Across Time, Based on Data From the 7-Day Drinking Diaries of the Australian Twin Registry Sample.

	T1 total drinking	T2 total drinking	T3 total drinking
T1 total drinking		**0.62**	**0.62**
T2 total drinking	0.59		**0.69**
T3 total drinking	0.58	0.70	
	T1 drinking max	**T2 drinking max**	**T3 drinking max**
T1 drinking max		**0.52**	**0.49**
T2 drinking max	0.45		**0.51**
T3 drinking max	0.38	0.58	
	T1 avg. consumed	**T2 avg. consumed**	**T3 avg. consumed**
T1 average consumed		**0.52**	**0.56**
T2 average consumed	0.46		**0.49**
T3 average consumed	0.43	0.51	
	T1 days drank	**T2 days drank**	**T3 days drank**
T1 days drank		**0.59**	**0.55**
T2 days drank	0.61		**0.70**
T3 days drank	0.53	0.73	

Note: Correlations for men are in boldface type; correlations for women are in roman type.

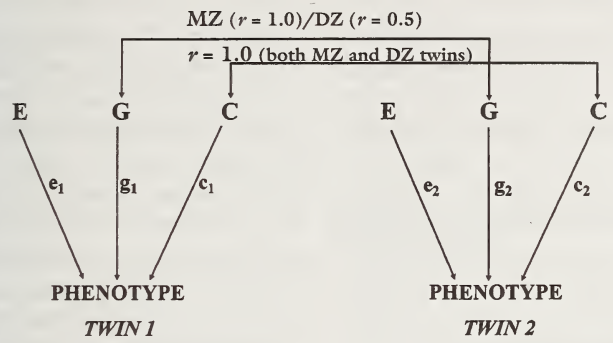

Figure 4. Univariate genetic model describing variance decomposition. Phenotypic variance = $e_2 + h_2 + c_2$; where e_2= nonshared environmental variance, h_2 = genetic variance (additive or nonadditive), and c_2 = shared environmental variance; and where E (nonshared environment), G (genetic effects—additive or nonadditive), and C (shared family environment) are standardized to unit variance. MZ = monozygotic; DZ = dizygotic.

10-year age groups, with the exception of the "oldest-old," where small sample sizes compromised the estimates (data not shown).

Fitting Models of Genetic and Environmental Effects to Longitudinal Data

The longitudinal data were examined from a biometrical genetic approach, that is, studying drinking measures over time as a function of additive shared, and unshared environmental influences. Analyses were carried out for each of the four measures of alcohol consumption (total weekly consumption, maximum number of drinks consumed on one day in that week, average number of drinks consumed per day over the last 7 days, and number of days on which alcohol was consumed in the last 7 days). Male and female twins were analyzed separately. The statistical modeling package used was Mx, a structural equation modeling package (Neale 1994).

The underlying analytical tenets of this strategy are depicted in a schematic in figure 4. The variance of a particular phenotype (in this case, a particular alcohol consumption measure) is equal to the sum of the variances due to (1) genetics (additive and nonadditive) (denoted as "G"); (2) shared environmental effects (denoted as "C"), which are those influences that the twins have in common, such as being reared by the same parents, attending the same school, having the same friends, and eating the same diet; and (3) nonshared, unique or special effects (denoted as "E"), which include those influences that the twins would *not* have in common, such as being married to a particular individual, attending a certain university, and living in a region of the country different from that of the cotwin. This last

term also includes measurement error. In the diagram, it can be seen that the genetic components are perfectly correlated (r = 1.0) for monozygotic twins, who share all of their genes, but they are correlated only 50 percent for dizygotic twins, who are no more alike than ordinary siblings. Further, there is no correlation between twins for nonshared environment, but the shared environment influence is correlated perfectly for both monozygotic and dizygotic twins.

We present the longitudinal version of this model in figure 5 (drawn for one twin for the sake of simplicity, but a straightforward matter to extend it conceptually for the cotwin). Models were fitted to raw data from the three survey time points, allowing for additive genetic, shared environment, and nonshared environment (plus error) to explain the variance observed in the particular alcohol consumption measure ("phenotype") under investigation. As in earlier descriptive analyses, four phenotypes were modeled: total weekly consumption, maximum consumed on any one day during the last week, average number of drinks per day consumed in the last week, and average number of days alcohol was consumed during the last week. The model depicted in figure 5 incorporates both estimates of genetic and environmental effects at each time point (h_1, h_2, h_3, etc.), and longitudinal correlations of these effects (genetic and environmental correlations). P_1, P_2, and P_3

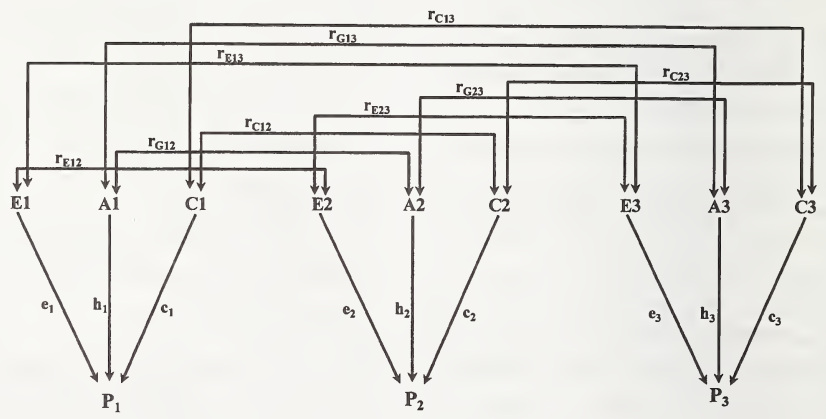

Figure 5. Longitudinal model for biometrical genetic analyses. Only one twin is drawn in this diagram. E = nonshared environment plus error; A = additive genetic effects; C = shared environment. Occasion-specific genetic and environmental effects are indicated by e_1, h_1, c_1; e_2, h_2, c_2; and e_3, h_3, c_3. P_1, P_2, and P_3 refer to the phenotype at three time points.

denote the phenotype at each of the three time points. Two questions may be posed for the longitudinal analyses: (1) how does magnitude of the genetic and environmental (shared and unshared) effects change over time, and (2) are the *same* genetic and environmental (shared and unshared) effects operating over time? We shall focus exclusively on the first question.

For the analyses presented here, data from the 7-day drinking diary were used to define the four alcohol consumption phenotypes. Data were standardized to a mean of 0 and a variance of 1 to facilitate parameter estimation. Extreme values were truncated to avoid the problem of the model failing to converge because of the undue influence of an extreme outlier (for example, extremes in the order of more than 120 drinks per week). Twin pairs were included where there was at least one data point; available for analysis were 744 monozygotic and 478 dizygotic female pairs and 311 monozygotic and 166 dizygotic male pairs.

A series of hierarchical models were fitted to the raw data by the method of maximum likelihood (Neale 1994). The goodness of fit of a full model (the so-called ACE model, where all three contributors to the phenotypic variance were included) was compared with the fit of a more parsimonious model (e.g., AE, CE), in which one term was omitted, by likelihood-ratio (chi-square difference) test. An insignificant chi-square difference indicated that the term that was omitted from the model did not detract from the overall fit of the model, and thus the parsimonious model had an adequate fit. Pending scientific plausibility, the most parsimonious model with adequate fit was to be preferred over others.

Results of Model Fitting for Women

Among women, for each phenotype, the model including additive genetic and nonshared environment effects fit the data well ($p \geq 0.2$ in all cases). (Specifics of the model-fitting statistics may be obtained from Dr. Bucholz upon request.) The interpretation was that, over time, each drinking phenotype may be modeled as a function of additive genetic and nonshared environmental effects. In these data, shared environmental effects did not contribute significantly to the variation in alcohol consumption phenotypes over time.

The proportion of total variance in the phenotype explained by each component was derived for each of the three time points (table 4). As can be seen in the table, for each measure the proportion of variance due to additive genetic influences appeared to be decreasing in women over time. For example, in 1981, the proportion of variance in total alcohol consumption that was explained by additive genetic influences was 65 percent in 1981; 50 percent in 1989, and 46 percent in 1994. Similar decrements were observed for the other three phenotypes.

A formal test was carried out to determine whether the decrement in

Table 4. Proportion of Total Variance in Drinking Phenotypes for Women and Men in the Australian Twin Registry Sample.

	1981 (%)	1989 (%)	1994 (%)	Test of Heterogeneity
		Women		
Variance due to:				
Additive genetics				
Total drinking	65	50	46	$p = 0.003$
Maximum in 7 days	53	45	44	$p = 0.265$
Average number of drinks	52	46	44	$p = 0.230$
Number of days drank	57	47	50	$p = 0.052$
Shared environment[1]				
Nonshared environment				
Total drinking	35	50	54	$p = 0.000$
Maximum in 7 days	47	55	56	$p = 0.001$
Average number of drinks	48	54	56	$p = 0.003$
Number of days drank	43	53	50	$p = 0.178$
		Men		
Variance due to:				
Additive genetics				
Total drinking	51	49	65	$p = 0.339$
Maximum in 7 days	53	56	73	$p = 0.038$
Average number of drinks	50	46	61	$p = 0.262$
Shared environment				
Number of days drank	48	43	32	$p = 0.495$
Nonshared environment				
Total drinking	49	51	35	$p = 0.001$
Maximum in 7 days	47	44	27	$p = 0.003$
Average number of drinks	50	54	33	$p = 0.004$
Number of days drank	52	57	68	$p = 0.032$

[1] This component was eliminated from all model fitting for women because models without this component (that is, more parsimonious) fit equally well.

the influence of additive genetics was statistically significant, by constraining the total additive genetic variance to be equal across the three time points (a test of heterogeneity). When these constraints were included, the model was rejected for two measures—total alcohol consumption and frequency of drinking, as assessed by the number of days on which alcohol was consumed in the last 7 days. The conclusion drawn from these analyses was that there was evidence that a significant decrement in the influence of additive genetics over time existed for women for total alcohol consumption and frequency of consumption, with a nonsignificant trend in the same direction for the other measures. Tests of heterogeneity of the increase

in the influence of nonshared environmental effects revealed that for three of the four drinking phenotypes, the increase over time was significant.

Results of Model Fitting for Men

Similar model-fitting strategies were applied to the data from men. Shared environmental influences did not appear to contribute to the variation for three of the four alcohol consumption phenotypes. Only for number of days on which alcohol was consumed in the last 7 days were shared environmental influences an important contributor to the variation over time. Also of note was the fact that additive genetic influences did not contribute to the variation over time in number of days on which alcohol was consumed. For the other three phenotypes, additive genetic and nonshared environmental influences explained the variation over time.

As was done for the data for women, the proportion of total variance in the phenotype explained by each component was calculated for each of the three time points. The lower half of table 4 displays these estimates. A formal test (test of heterogeneity) was conducted to determine whether the additive genetic influences on alcohol consumption increased over time, by incorporating two constraints in the full model (that the additive genetic variances were equal across the three time points). Evidence for a significant ($p = 0.038$) increase in the proportion of the variance due to additive genetic influences was observed for only one phenotype, maximum number of drinks consumed on any one day in the last 7 days. For number of days drank, additive genetic influences had been eliminated previously from the model. As can be seen, the proportion of variance due to nonshared environmental influences (plus error) appeared to decrease for three out of four phenotypes of alcohol consumption from Time 1 to Time 3, and a formal test was conducted to confirm this impression. Our findings showed that the proportion of variance due to nonshared environmental influences decreased for men for three out of four phenotypes from 1981 to 1994. An increase in the proportion of variance due to nonshared environment was observed for "number of days drank."

CONCLUSIONS

With respect to cross-sectional compared with longitudinal evidence, we observed that women in the cross-sectional samples were similar in terms of both sociodemographic characteristics and alcohol consumption characteristics to those who had completed at least one other previous assessment. The similarity of the two samples establishes confidence in the inferences drawn from the longitudinal data on women. The data for men, on the other hand, were suggestive of an underrepresentation of heavy drinkers in the longitudinal sample and thus warranted caution in drawing inferences based on the longitudinal sample of men.

Substantial proportions of older individuals in the Australian sample were

current drinkers and, in particular, current heavy drinkers, in marked contrast to community samples of older individuals from the United States. This observation held even when the data were age adjusted, since the Australian sample was younger than many of the U.S. samples. For all measures of drinking behavior for women, the predominant patterns were of stability or decrease from 1981 to 1994. There was no evidence for an increase in drinking in women. Stability or decreases in drinking characterized male drinking patterns over time.

The results of the biometrical genetic model fitting indicated that for both men and women, additive genetic and nonshared environment (plus error) explained the variance in most drinking behaviors at each time point. Shared environment was not an important contributor to the variance in drinking phenotypes over time, with the exception of frequency of drinking for men. Further, evidence was mustered to support the contention that the proportion of variance in drinking behaviors due to additive genetic effects was *decreasing* over time for women. In contrast, the evidence from the longitudinal data from men indicated that for at least one drinking measure—maximum number of drinks consumed on any one day in the last week—the proportion of variance due to additive genetic influences was *increasing*. For men, the data indicated a significant decrease over time in the proportion of variance in three out of four drinking behaviors due to nonshared environment effects, while the opposite conclusion—that of a significant increase in the influence of nonshared environmental effects over time—was drawn for women.

These results were unexpected and suggest the following future analyses. The effect of retirement on drinking should be examined more closely. It is possible that spousal drinking patterns—an environmental influence not shared by twin and cotwin—may be more influential for females. This possibility would explain our observation of a decrease in additive genetic effects over time in women, and a significant increase in the influence of nonshared environmental effects. This hypothesis will be tested directly with some new data from spouses of about 3,700 twins, which has been collected recently. Furthermore, living arrangements of elderly twins must be explored, to investigate the possibility that shared accommodations for men late in life might account for the decreasing proportion of variance explained by nonshared environmental influences. The data for men must be interpreted with caution, however, in light of the evidence that men who were heavy drinkers were less likely to be represented in the longitudinal data.

NOTES

1. With respect to alcoholism, some researchers have objected to the appropriateness of the diagnostic criteria for alcohol dependence for older individuals (Graham 1986). Older individuals tend to have diminished social

and familial responsibilities and thus would not be expected to endorse items in the diagnostic criteria (e.g., DSM-III-R [American Psychiatric Association 1987]) that include alcohol's interference with these areas of responsibility. These criticisms are not as relevant to the DSM-IV (American Psychiatric Association 1994) criteria, in which adverse social consequences of drinking are not included in the construct of alcohol dependence. A second criticism is that the thresholds of heavy drinking, often set between five and seven per drinking occasion, are not appropriate for older individuals, who, as noted earlier, have a diminished capacity for alcohol. Also, older individuals may have a more difficult time than their younger counterparts in distinguishing the effects of alcohol from those of normal aging. The lower lifetime prevalence for alcohol dependence among older individuals should be evaluated with these points in mind.

2. The telephone interview survey conducted in 1992–93 assessed psychiatric disorder via a structured interview abridged from an interview used in a multicenter family study of alcoholism in the United States (Bucholz et al. 1994). A markedly different measurement of drinking behavior in the last 12 months was obtained, more appropriate for telephone interview administration than a 7-day drinking diary. Because of the difference in assessing drinking behaviors, the interview study was not included in the analyses reported here. Readers interested in more information about the telephone interview survey may consult several published references (Heath et al. 1994; Slutske et al. 1995; Heath et al. 1997).

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REFERENCES

Adams, W.L.; Garry, P.J.; Rhyne, R.; Hunt, W.C.; and Goodwin, J.S. Alcohol intake in the healthy elderly: Changes with age in a cross-sectional and longitudinal study. *J Am Geriatr Soc* 38:211–216, 1990.

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 3d ed., rev. Washington, DC: the Association, 1987.

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 4th ed. Washington, DC: the Association, 1994.

Anderson, P.; Casswell, S.; Shaper, A.G.; Rehm, J.; Bondy, S.; Room, R.; and Doll, R. Guidelines on sensible drinking. *Addiction* 91:25–33, 1996.

Atkinson, R.M. Aging and alcohol use disorders: Diagnostic issues in the elderly. *Int Psychogeriatr* 2:55–72, 1990.

Atkinson, R.M.; Tolson, R.L.; and Turner, J.A. Late versus early onset prob-

lem drinking in older men. *Alcohol Clin Exp Res* 14:574–579, 1990.

Barnes, G. Alcohol use among older persons: Findings from a western New York State general population survey. *J Am Geriatr Soc* 27:244–250, 1979.

Bucholz, K.K.; Cadoret, R.; Cloninger, C.R.; Dinwiddie, S.H.; Hesselbrock, V.M.; Nurnberger, J.I.; Reich, T.; Schmidt, I.; and Schuckit, M.A. A new, semi-structured psychiatric interview for use in genetic linkage studies; a report of the reliability of the SSAGA. *J Stud Alcohol* 55:149–158, 1994.

Bucholz, K.K.; Sheline, Y.I.; and Helzer, J.E. The epidemiology of alcohol use, problems and dependence in elders: A review. In: Beresford, T., and Gomberg, E., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 19–41.

Buchsbaum, D.G.; Buchanan, R.G.; Welsh, J.; Centor, R.M.; and Schnoll, S.H. Screening for drinking disorders in the elderly using the CAGE questionnaire. *J Am Geriatr Soc* 40:662–665, 1992.

Busby, W.J.; Campbell, A.J.; Borrie, M.J.; and Spears, G.F.S. Alcohol use in a community-based sample of subjects aged 70 years and older. *J Am Geriatr Soc* 36:301–305, 1988.

Cahalan, D.; Cisin, I.; and Crossley, H. *American Drinking Practices: A National Study of Drinking Behavior and Attitudes.* Monograph 6. New Brunswick, NJ: Rutgers Center of Alcohol Studies, Publications Division, 1969.

Chrischilles, E.A.; Foley, D.J.; Wallace, R.B.; Lemke, J.H.; Semla ,T.V.; Hanlon, J.T.; Glynn, R.J.; Ostfeld, A.M.; and Guralnik, J. Use of medications by persons 65 and over: Data from the Established Populations for Epidemiologic Studies of the Elderly. *J Gerontol* 47:M137–M144, 1992.

Doll, R.; Peto, R.; Hall, E.; Wheately, K.; and Gray, R. Mortality in relation to consumption of alcohol: 13 years' observations on male British doctors. *BMJ* 309:911–918, 1994.

Douglass, R.L.; Schuster, E.O.; and McClelland, S.C. Drinking patterns and abstinence among the elderly. *Int J Addict* 23:399–415, 1988.

Dufour, M.; Archer, L.; and Gordis, E. Alcohol and the elderly. *Clin Geriatr Med* 8:127–141, 1992.

Ekerdt, D.J.; DeLabry, L.O.; Glynn, R.J.; and Davis, R.W. Change in drinking behaviors with retirement: Findings from the Normative Aging Study. *J Stud Alcohol* 50:347–353, 1989.

Feighner, J.P.; Robins, E.; Guze, S.B.; Woodruff, R.A.; Winokur, G.; and Munoz, R. Diagnostic criteria for use in psychiatric research. *Arch Gen Psychiatry* 26:57–63, 1972.

Forster, L.E.; Pollow, R.; and Stoller E.P. Alcohol use and potential risk for alcohol-related adverse drug reactions among community-based elderly. *J Community Health* 18:225–239, 1993.

Friedman, G.D., and Klatsky, A.L. Is alcohol good for your health? *N Engl J Med* 329:1882–1883, 1993.

Fuchs, C.S.; Stampfer, M.J.; Colditz, G.A.; Giovannucci, E.L.; Manson, J.E.; Kawachi, I.; Hunter, D.J.; Hankinson, S.E.; Hennekens, C.B.; Rosner, B.; Speizer, F.E.; and Willett, W.C. Alcohol consumption and mortality among women. *N Engl J Med* 332:1245–1250, 1995.

Glynn, R.J.; Bouchard, G.R.; LoCastro, J.S.; and Hermos, J.A. Changes in alcohol

consumption behaviors among men in the normative aging study. In: Maddox, G.; Robins, L.N.; and Rosenberg, N., eds. *Nature and Extent of Alcohol Problems in the Elderly.* New York: Springer, 1986. pp. 101–116.

Goodwin, J.S.; Sanchez, C.J.; Thomas, P.; Hunt, C.; Garry, P.J.; and Goodwin, J.M. Alcohol intake in a healthy elderly population. *Am J Public Health* 77:173–177, 1987.

Gordon, T., and Doyle, J.T. Alcohol consumption and its relationship to smoking, weight, blood pressure and blood lipids. *Arch Intern Med* 143:1366–1374, 1986.

Gordon, T., and Kannel, W.B. Drinking and its relation to smoking, blood pressure, blood lipids and uric acid. *Arch Intern Med* 146:262–265, 1983.

Graham, K. Identifying and measuring alcohol abuse among the elderly: Serious problems with existing instrumentation. *J Stud Alcohol* 47:322–326, 1986.

Guttmann, D. Patterns of legal drug use by older Americans. *Addict Dis* 3:337–356, 1978.

Heath, A.C., and Martin, N.G. Genetic influences on alcohol consumption patterns and problem drinking: Results from the Australian NH & MRC twin panel follow up survey. *Ann NY Acad Sci* 708:72–85, 1994.

Heath, A.C.; Jardine, R.; and Martin, N.G. Interactive effects of genotype and social environment on alcohol consumption in female twins. *J Stud Alcohol* 50:38–48, 1989.

Heath, A.C.; Bucholz, K.K.; Slutske, W.S.; Madden, P.A.F.; Dinwiddie, S.H.; Dunne, M.P.; Statham, D.J.; Whitfield, J.B.; Martin, N.G.; and Eaves, L.J. The assessment of alcoholism in surveys of the general community: What are we measuring? Some insights from the Australian twin panel interview survey. *Int Rev Psychiatry* 6:295–307, 1994.

Heath, A.C.; Bucholz, K.K.; Madden, P.A.F.; Dinwiddie, S.H.; Slutske, W.S.; Statham, D.J.; Dunne, M.P.; Whitfield, J.; and Martin, N.G. Genetic and environmental contributions to alcohol dependence risk in a national twin sample: Consistency of findings in women and men. *Psychol Med* 27:1381–1396, 1997.

Hilton, M.E. Abstention in the general population of the U.S.A. *Br J Addict* 81:95–112, 1986.

Hilton, M.E. Trends in U.S. drinking patterns. Further evidence from the past twenty years. In: Clark, W.B., and Hilton, M.E., eds. *Alcohol in America.* Albany: State University of New York Press, 1991. pp. 121–138.

Iliffe, A.; Haines, A.; Boroff, A.; Goldenberg, E.; Morgan, P.; and Gallivan, S. Alcohol consumption by elderly people: A general practice survey. *Age Ageing* 20:120–123, 1991.

Kessler, R.C.; McGonagle, K.A.; Zhao, S.; Nelson, C.B.; Hughes, M.; Eshleman, S.; Wittchen, H.-U.; and Kendler, K.S. Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States: Results from the National Comorbidity Study. *Arch Gen Psychiatry* 51:8–19, 1994.

Malin, H.; Wilson, R.; Williams, G.; and Aitken, S. Alcohol/health practices supplement. *Alcohol Health Res World* 10:48–50, 1983.

Martin, N.G.; Oakeshott, J.G.; Gibson, J.B.; Starmer, G.A.; Perl, J.; and Wills, A.V. A twin study of psychomotor and physio-

logical response to an acute dose of alcohol. *Behav Genet* 15:305–347, 1985.

Meyers, A.R.; Hingson, R.; Mucatel, M.; and Goldman, E. Social and psychologic correlates of problem drinking in old age. *J Am Geriatr Soc* 30:452–456, 1982.

Midanik, L. Alcohol consumption and consequences of drinking in general health surveys. In: Holden, H., and Edwards, G., eds. *The Scientific Rationale for Alcohol Policy.* Oxford, England: Oxford University Press, 1995.

Mirand, A.L., and Welte, J.W. Alcohol consumption among the elderly in a general population, Erie County, New York. *Am J Public Health* 86:978–984, 1996.

Neale, M.C. *Mx: Statistical Modeling.* Richmond: Department of Psychiatry, Medical College of Virginia, Virginia Commonwealth University, 1994.

Pollow, R.L.; Stoller, E.P.; Forster, L.E.; and Duniho, T.S. Drug combinations and potential for risk of adverse drug reactions among community-dwelling elderly. *Nurs Res* 43:44–49, 1994.

Rehm, J., and Sempos, C. Alcohol consumption and all-cause mortality. *Addiction* 90:471–480, 1995.

Reich, T.; Cloninger, C.R.; Van Eerdewegh, P.; Rice, J.P.; and Mullaney, J. Secular trends in the familial transmission of alcoholism. *Alcohol Clin Exp Res* 12:458–464, 1988.

Serdula, M.K.; Koong, S-L.; Williamson, D.F.; Anda, R.F.; Madans, J.H.; Kleinman, J.C.; and Byers, T. Alcohol intake and subsequent mortality: Findings from the NHANES I follow-up study. *J Stud Alcohol* 56:233–239, 1995.

Slutske, W.S.; Heath, A.C.; Madden, P.A.F.; Bucholz, K.K.; Dinwiddie, S.H.; Dunne, M.P.; Statham, D.J.; Whitfield, J.B.; and Martin, N.G. Is alcohol-related flushing a protective factor for alcoholism in Caucasians? *Alcohol Clin Exp Res* 19:582–592, 1995.

Smart, R.G., and Adlaf, E.M. Alcohol and drug use among the elderly: Trends in use and characteristics of users. *Can J Public Health* 79:236–242, 1988.

Sulsky, S.I.; Jacques, P.F.; Otradovec, C.L.; Hartz, S.C.; and Russell, R.M. Descriptors of alcohol consumption among nonalcoholic elderly. *J Am Coll Nutr* 9:326–331, 1990.

Tell, G.S.; Rutan, G.H.; Kronmal, R.A.; Bild, D.E.; Polak, J.F.; Wong, N.D.; and Borhani, N.O. Correlates of blood pressure in community-dwelling older adults: The Cardiovascular Health Study. *Hypertension* 23:59–67, 1994.

Temple, M.T., and Leino, E.V. Long term outcome of drinking: A 20 year longitudinal study of men. *Br J Addict* 84:889–899, 1989.

Welte, J.W., and Mirand, A.L. Lifetime drinking patterns of elders from a general population survey. *Drug Alcohol Depend* 35:133–140, 1994.

Chapter 4

Aging and Alcohol Use and Abuse Among African Americans: A Life-Course Perspective

James S. Jackson, Ph.D., David R. Williams, Ph.D., and Edith S. Lisansky Gomberg, Ph.D.

The findings of several studies reveal that African Americans now suffer more adverse health consequences from heavy alcohol consumption than do whites (Lex 1987; Warheit et al. 1989; Herd 1991, 1994; Gomberg and Nelson 1995). Epidemiologic studies indicate that there are age differences in alcohol use and abuse between African Americans, Hispanics, and whites (Caetano and Herd 1988; Robins 1989; Herd 1990, 1991; Skarupski et al. 1996). It may be that the adverse health consequences suffered by African American heavy drinkers are linked to the later-in-life development of alcohol abuse among African Americans more generally. There has been speculation that the observed age differences in alcohol use and abuse among African Americans are related to (a) urban migration and second-generation status (i.e., historical events); (b) the lower health status of African Americans compared with whites in the general population; (c) midlife crises and depression among middle-age African Americans; (d) social and psychological factors (e.g., social support, religion, personality traits); or (e) stress that accumulates over the individual life course related to lifetime experiences of racialized maltreatment, low income, and other barriers to mobility.

The purpose of this chapter is to outline a general theoretical and research framework for studying alcohol use and abuse over the individual

J.S. Jackson, Ph.D., is professor of psychology and professor of public health, 5006 Institute for Social Research, P.O. Box 1248, University of Michigan, Ann Arbor, MI 48106–1248. D.R. Williams, Ph.D., is professor of sociology, 2236 Institute for Social Research, University of Michigan. E.S.L. Gomberg, Ph.D., is professor of psychiatry, Alcohol Research Center, 400 East Eisenhower Parkway, Suite A, University of Michigan, Ann Arbor, MI 48104.

life course (Zucker and Gomberg 1986; O'Malley et al. 1988; Gomberg 1990; Zucker et al. 1995; J.S. Jackson 1996). A life-course perspective is conducive to research and practice, as well as the implementation of public health interventions. This framework encompasses consideration of the continuities and discontinuities over the individual life course and focuses on important developmental and aging-related processes, cohort influences, and period events needed to understand physical and psychological health at different points in the individual lifespan.

The main premise of a life-course perspective is that already-born and aging cohorts have been exposed to conditions that will profoundly influence their social, psychological, and health statuses as they reach middle and older ages in the years and decades to come (Baltes 1987; Barresi 1987). Some studies on emotional selectivity (Carstensen 1993) and successful aging (e.g., Mariske et al. 1995) are beginning to provide testable hypotheses of life-course–related processes in human development and aging. Building on decades of work by interdisciplinary scholars, these new models help to organize what has been more a set of assumptions than an organized theory with the power to generate testable predictions (J.S. Jackson 1996).

BIRTH COHORT AND THE LIFE COURSE

> The health of the Negro today is both an expression and the result of the social and economic burdens imposed upon him. His health is inseparably connected with poor housing, unemployment, and inadequate education... Yet there is insufficient data, as well as a paucity of studies, designed to answer many of the questions confronting us. (Cornerly 1968, p. 653)

This quotation is from an article summarizing the first Nationwide Conference on the Health Status of the Negro in 1967 at Howard University. Its content is still applicable today. During the conference, reports on the widening health gap between African Americans and whites pointed specifically to fetal death rates, life expectancy, childhood health risks, and disease-specific causes of death, all indicating significant increased risks in the African American population (Cornerly 1968).

This chapter is also premised upon the context of the changing demography of older populations and the changing age structure. Based upon the middle projection series of the U.S. Census Bureau (1.9 ultimate lifetime births per woman, mortality life expectancy of 79.6 years in 2050, and annual net immigration of 450,000), it is projected that sustained growth will occur in the over 65 age group until 2010. From 2010 through 2030, the postwar baby boom cohorts will increase the over 65 age group from 39 million to 65 million. By the year 2030, every fifth American will be over 65 years of age (Siegel and Taeuber 1986).

As a way of highlighting life-course influences, African Americans have greater disability and morbidity at every point of their individual lifespans (J.J. Jackson 1981). In infancy, this is

marked by higher mortality figures as well as accident and disease rates. In adolescence, young adulthood, and thereafter, African Americans are characterized by comparatively higher homicide deaths than whites. Middle age and early old age show increased disability, earlier retirement, and, ultimately, higher death rates in the African American community as compared with the general population. It is only after the ages of 75 to 80 that African Americans tend to show increased longevity in comparison with whites (Manton et al. 1979; Manton 1982). In support of the substance of this observed crossover, it has been suggested that genetic and environmental factors act in tandem on a heterogeneous African American population to produce hardier older African Americans (Manton et al. 1979; Gibson and Jackson 1987). One direct implication of this explanation is the existence of differential aging processes within African American and white populations (Manton 1982).

Riley and her colleagues (Riley 1994*a*, 1994*b*; Riley and Riley 1994) have proposed that cohort succession and structural lag must be considered in any model of aging and human development, including psychological ones. Their main argument is that as people age, they encounter changing role opportunities and circumstances in society. At the intersections of lives and social structures (e.g., job markets and educational opportunities), lives influence structures, and structures influence lives. They suggest that this interplay between individual lives and role opportunities for individuals can never be in synchrony, there must always be asynchronies, since the factors that influence changes in job markets, educational opportunities, and so on do not keep pace with changes in people's lives. Thus, there must always be structural lags, that is "changes in social structures that provide role opportunities and norms [that] do not keep pace with the 20th century metamorphoses in people's lives" (Riley and Riley 1994, p. 17).

In the past the family has been an important and stable asset for African Americans in a hostile environment, insulating individuals against the continuing, negative effects of discrimination, especially in its institutional forms (e.g., Jim Crow). However, the continuing oppression of prejudice and discrimination interact with structural changes (e.g., loss of manufacturing jobs) to make coping even more difficult. In an ever-increasing technological era, systematic barriers to education for African Americans affect not only the aging cohort members themselves, but also their offspring. Riley (1994*b*) suggested the following:

> However, other segments of older people in future cohorts may be less advantaged than their predecessors, as, for example their lives will reflect their earlier experience with the deteriorating economic conditions of today, the rise of disadvantaged minorities, the loosening family structure, the spreading use of drugs, and the increasing proportions of younger people

who are failing to meet acceptable standards of academic achievement. (p. 1216)

In every example used by Riley (1994*b*), racial and ethnic minority groups are disadvantaged (e.g., deteriorating economic opportunities, weakening of family ties, spreading use of drugs, poor educational and technical training). Significant improvements in the life situations of many groups (Farley 1987), particularly health, have occurred over the last 40 years (J.J. Jackson 1981). On the other hand, various studies (e.g., Gibson 1986; Farley 1987; Jaynes and Williams 1989) document that negative life events and structural barriers still exist for African Americans (Farley 1996; Miller 1996). These problems include the difficulties of single-parent households, high infant mortality and morbidity, childhood diseases, poor diets, lack of preventive health care, deteriorating neighborhoods, poverty, adolescent violence, unemployment and underemployment, teen pregnancy, drug and alcohol abuse, and broken marriages. Although the exact causal relationships are not known (D.W. Williams 1990), these are predisposing factors for poor physical and psychological health across the entire individual lifespan (Haan and Kaplan 1985; Dressler 1991).

Our theoretical interest lies in understanding the interaction and intersection of age-related processes, period events, and cohort-related phenomena as they influence the family and individual experiences. Thus, we have oriented our work to examine how the adaptation and quality of life of individuals, families, and larger groups of Americans are influenced by (a) the age cohort into which individuals are born; (b) the social, political, and economic events that occur to cohorts born together; and (c) the individual aging process at different points in a person's lifespan. For example, African Americans born before the 1940's faced very different environmental constraints and have experienced a very different set of life tasks, events, opportunities, and disappointments than those born in the 1970's (Baker 1987). Health care advances, family changes, urban migration, and macroeconomic influences, in addition to significant changes in the legal structure, all differed dramatically for these very different birth cohorts, as they will for future birth cohorts (Richardson 1991).

In general, members of different racial and ethnic groups evidence varying patterns of disease and limiting health conditions in older ages. These patterns differ by characteristics such as gender and heredity, and they are influenced by cultural patterns and socioeconomic status differences. Although the exact mechanisms of how socioeconomic status and gender may play a role are not known, the negative effects of low socioeconomic status are well documented (Barr et al. 1993; Jones-Webb et al. 1995). Also, an important overlooked factor has been the patterning and co-occurrence of disease and chronic conditions, especially among racial and ethnic groups. For example, it has been reported that for American Indians, dia-

betes is a risk factor, along with malnutrition, fatigue, and crowded living conditions, in the incidence and mortality effects of tuberculosis (McCabe and Cuellar 1994). Alcohol abuse among some American Indians is a well-known phenomenon, although McCabe and Cuellar (1994) reported significant differences among tribes. Research on elders in this group is not extensive (Barker and Kramer 1996). Hypertension has been long recognized as a prevalent disorder among African Americans, but recent work shows a dramatic increase in hypertension among the current generation of American Indians, especially Navajos, suggesting that the next generation over 65 may show an increase in its prevalence (McCabe and Cuellar 1994).

Genetic and biological differences may also play a significant role in disease, morbidity, and mortality in older ages. Differential bone mass between black and white elderly, for example, may play a significant role in predisposition to fractures in older ages (Richardson 1991). Morioka-Douglas and Yeo (1990) indicated racial and ethnic group differences in drug sensitivity and tolerance among Asian Americans in comparison to other groups. There may be significant genetic and physiological components in the observed differential rates of diabetes, cerebrovascular problems, heart problems, and alcoholism among racial and ethnic groups (Barker and Kramer 1996).

Culturally determined differences in beliefs and behaviors related to health may also account for large health status differences between ethnic and racial groups and the general American population. For example, Yu and colleagues (1985) reported that on some measures of preventive health-promoting activities (e.g., well-baby examinations and early physician visits), Asian and Pacific Islander rates exceeded those of the general American population. Kuo (1984) noted, however, that Asian Americans tended to underutilize health services and that the negative consequences of such underutilization differed among specific Asian American groups. Yu and colleagues (1985) reported that cultural factors related to family ties and health beliefs (e.g., use of herbal prescriptions or balance of hot and cold elements of the body) played a significant role in the health status of the elderly among Asian American groups (Barker and Kramer 1996).

How different birth cohorts, historical and current environmental events, and individual differences in aging processes interact with one another must form the overall context of research and interventions on alcohol use and abuse (J.S. Jackson 1996). The examination of physical and psychological health status and functioning has been conducted in a relative life-course vacuum. Several authors have proposed the need for life-course models (e.g., Baltes 1987; Barresi 1987; Manton and Soldo 1985) that include history, cohort, and period effects in the nature of physical and psychological health status and functioning. Few researchers, however, have actually employed these models. We suggest in this chapter that alcohol use and abuse among African Americans provides one important example of the applicability

of this life-course approach (Zucker and Gomberg 1986; Zucker et al. 1995).

ALCOHOL USE AND ABUSE

It has been suggested that alcohol abuse is the number one mental health problem for African Americans (Harvey 1985). There is a high level of alcohol use, heavy drinking, and alcohol problems in the African American community (Lipscomb and Trochi 1981; Caetano and Schafer 1996). Differences in social consequences have changed over time: alcohol-related family problems and arrests for alcohol-impaired driving now show more similar figures for African American and white heavy drinkers than they have in the past (Gomberg and Nelson 1995).

Research Issues

Although there have been some important studies shedding light on alcohol use within the African American population in recent years (e.g., research by Caetano and Herd 1988), there is relatively little empirical research about African Americans and alcohol (Harper and Dawkins 1976; Watts and Wright 1983; Harper and Saifnoorian 1991; Caetano and Schafer 1996). The published literature has largely involved small, selected clinical samples of problem drinkers in treatment and has ignored the majority of African Americans who consume alcohol moderately and the majority of African American women who abstain (Lewis 1955; Liebow 1967; Hannerz 1969; Anderson 1976). The more recent epidemiologic work yields important statistical information and demographic trends but little in-depth data about the psychological, social, and cultural factors that influence drinking, particularly heavy drinking among African Americans. Thus, our understanding of the causes and correlates of drinking problems within the African American population is still in its infancy (Harper 1976). We still know very little about the natural course of alcohol use and abuse and about possible antecedent, etiologic, and prognostic factors that may contribute to heavy/problem drinking among African Americans (Franklin 1989; Russell et al. 1990).

Another reason for our limited knowledge of alcohol use and abuse within the African American population is the dominance of the race comparison paradigm in the research on African American mental health (Neighbors 1984; J.S. Jackson et al. 1986). In this framework, the mental health status of the white population is used as a standard of comparison for the mental health of the African American population. The overreliance of epidemiologic studies on a race comparison paradigm masks the heterogeneity within the African American population. Like any racial or ethnic group, African Americans are not monolithic. For example, some evidence (Gomberg and Nelson 1995) suggests that alcohol is frequently used as a prestige symbol for lower socioeconomic status segments of the African American community.

The consumption of higher content alcoholic beverages may be a way of expressing status among some male groups. As another example, there are gender differences in the patterns and prevalence of alcohol use, abuse, and related problems, and more research in this area is needed (Herd 1997). Alcohol use among women (R.W. Wilsnack et al. 1993), and especially African American women, is a mostly neglected topic in the literature (Harvey 1985; Franklin 1989). African American women are exposed to both racism and sexism, and part of the cultural baggage they carry is the double standard for men and women with regard to alcohol dependence. While alcohol abuse is undesirable for African American men, it is "absolutely socially unacceptable for women" (Harvey 1985, p. 87). Nevertheless, some evidence suggests that there is growing use of alcohol among African American women (Miller and Miller 1988; Gomberg and Nelson 1995).

One of the most important needs in future research on alcohol use and abuse among African Americans is for researchers to give more attention to what race means and why race is related to health status (D.R. Williams and Fenton 1994). Biologists and anthropologists indicate that the concept of races as human populations that differ genetically from others may be without scientific basis (Gould 1977; Polednak 1989; F.L. Jackson 1992). There is more genetic variation within races than between them, and racial categories tend not to represent biological distinctiveness. Racial classification schemes are arbitrary, and race is more of a social category than a biological one (Cooper and David 1986). Research that will advance our understanding of the role of race as a social category in health must seek to identify the ways in which social, economic, political, and cultural forces, as well as racial discrimination, shape the daily experiences of subgroups in ways that promote or retard alcohol usage (Harvey 1985; J.S. Jackson et al. 1986; Herd 1987; D.R. Williams 1992).

Age is a variable routinely employed in epidemiologic studies to represent biological processes. Researchers should consider that age may also be a proxy for important social processes (Geronimus 1988). For example, race differences in blood pressure tend to be evident only in adulthood. This may indicate a lag in the effect of environmental exposures on the sustained elevation of blood pressure, or it may reflect a rapid increase of hypertension in African American young adults, as they are forced to confront the reality of restricted socioeconomic opportunities and truncated options (D.W. Williams 1990). Geronimus (1988) proposed a "weathering hypothesis" to account for the relationship between age and health outcomes among African Americans. Given the chronic and cumulative nature of these adverse conditions, higher risk for poor health, therefore, will increase with age.

Comorbid conditions may influence alcohol use and abuse. For example, rates of depression and depressive symptoms have been found to be very high in samples of alcohol-dependent

respondents (Merikangas and Gelernter 1990). Some evidence suggests that the depression seen in alcoholic patients is due largely to the social stress associated with alcohol abuse and the biological effects of alcohol intoxication or withdrawal from alcohol (e.g., Schuckit and Morrissey 1976, in females only). Consistent with this perspective, some studies find that many depressive symptoms in alcohol-dependent individuals disappear within 2–4 weeks of abstinence (Brown and Schuckit 1988).

Clinical studies have found that 23 to 70 percent of alcoholic patients also meet criteria for the lifetime prevalence of anxiety disorders (Lader 1972; Smail et al. 1984). Clinicians report widespread use of alcohol to reduce the symptoms of anxiety, with Woodruff and colleagues indicating that 50 percent of their patients with anxiety disorders also had secondary alcohol dependence. Data from the National Institute of Mental Health Epidemiologic Catchment Area (ECA) study also reveal high levels of comorbidity between alcohol abuse/dependence and anxiety disorders. Regier and colleagues (1990) indicated a high level of co-occurrence of alcohol abuse or dependence with phobic disorders (odds ratio = 2.4), panic disorder (odds ratio = 4.3), and any anxiety disorder (odds ratio = 1.5). Depression and anxiety disorders were even more strongly associated in the ECA study, with the odds ratios exceeding 15 for both phobia and panic.

Evidence suggests that the onset of anxiety disorder predates the onset of alcohol dependence in persons who manifest both disorders, often by several years (Lader 1972; Cadoret et al. 1985; Kessler et al. 1997). One recent study indicated that 65 percent of persons with both disorders had the anxiety disorders prior to the onset of alcohol dependence (Merikangas et al. 1993). All of these studies were of predominantly white samples, or samples in which racial differences in these processes were not assessed. Other evidence suggests that the causal processes may be reciprocal. Anxiety is a common symptom of alcohol withdrawal, and several studies have found that chronic alcohol intake can produce symptoms of anxiety and depression (Cadoret et al. 1985). A third possibility is that alcohol dependence and anxiety disorders may reflect differing responses to common factors (Cadoret et al. 1985).

Prior research has not identified what types, amounts, and aspects of psychosocial risk factors and resources are most consequential for the initiation and maintenance of alcohol abuse. The mechanisms and processes by which the experience of racial discrimination may produce psychic distress and lead to changes in health behaviors are unknown. There is controversy over the extent to which stress is a cause or a consequence of alcohol use. Likewise, there has been some dispute as to whether psychosocial resources act to insulate people from circumstances that threaten health, to "buffer" them from the adverse consequences of such circumstances to which they have been exposed, or to promote health and well-being by meeting basic needs for social integration

and making sense out of one's world (Antonucci and Jackson 1990).

Research Findings

Cahalan and colleagues (1969), in the first national survey of drinking, concluded that "White and Negro men varied little in their rates of drinking." These authors reported that 29 percent of white males and 24 percent of African American males who drank were heavy drinkers. One of the earliest hints that black/white comparisons were going to be more complex than first indicated appeared in a report from the Research Triangle Institute (Rachal et al. 1975) about drinking by high school students: African Americans were twice as likely to be abstainers as whites, and four times as many whites drank heavily compared with African Americans.

It has been assumed that the peak of heavy/problem drinking occurs early, followed by a gradual decline in heavy drinking as people age, is a universal curve; however, recent work suggests that this pattern holds true for whites but not for some ethnic minorities. With both African American and Hispanic men, frequent/heavy drinking does not show the same trend as whites (Caetano 1984; Herd 1991). For example, work from the Monitoring the Future project (Johnston et al. 1996) indicates that there are differences among minority groups in the frequent or heavy use of alcohol. Almost half of the young white and Native American males reported having five or more drinks in a single sitting once or more during the 2 weeks preceding the survey. The rate was nearly as high for young Mexican American males. Instances of heavy drinking were significantly less common among Puerto Rican and other Latin American males, and even lower among African American and Asian American males.

In a survey of ethnicity/drinking in three northern California counties, Caetano (1984) observed that the patterns of drinking and alcohol problems varied "dramatically" by ethnicity. Among white males, heavy drinking and alcohol-related problems peaked during youth and then dropped linearly; African American males showed "exactly the opposite."

The first national study of drinking patterns, attitudes, and problems in the African American and Hispanic populations was conducted by the Alcohol Research Group in Berkeley, CA, in 1984. As reported by Herd (1990), African American and white men in this study exhibited similar patterns in the aggregate, but a different picture was seen when specific variables were examined. Frequent heavier drinking among whites was associated with youthfulness, high socioeconomic status, and residing in geographic areas with higher concentrations of alcohol use/abuse; these patterns were reversed or absent among African American men, for whom age and income emerged as significant variables. Low-income and high-income African American men were unlikely to drink heavily; rather, middle-income African American men were most likely to be heavier drinkers. While the percentage of frequent/heavy drinkers dropped for

white men from a peak at ages 18–29, for African American men that percentage reached a peak at ages 50–59, surpassing the percentage of white frequent/heavy drinkers in that age group (Herd 1990, 1991). A similar finding was reported for African American women: they showed a considerably lower percentage of heavier drinkers up to age 50, when the white and African American percentages converged (Herd 1989).

There have been several reports from the ECA study (Robins et al. 1988*a*; Robins 1989) of lifetime prevalence alcohol disorder rates for African American men and women that are considerably lower than whites for those under the age of 45 but higher than whites for those age 45 and older. The ECA findings substantiate the validity of different age trajectory curves (Robins et al. 1988*a*; Robins and Regier 1991). Rates of lifetime alcohol disorder for all ECAs combined are shown in table 1. A report from the National Center for Health Statistics on exposure to alcohol abuse within the family (Schoenborn 1991) also supports the validity of black/white differences in the age trajectory of heavy drinking and alcohol-related problems.

A question has been raised whether drugs other than alcohol may be preferred among young African American males. The rate of drug abuse is also lower among young African American males than among young white males, although the gap is not as great as in alcohol abuse. One explanation has been offered by Robins (1989) in terms of recency of urban northward migrations and generational differences in access to the opportunity structure (see the discussion of explanations for the age trajectory of alcohol use in the next section).

Early research suggested that more African American than white women were abstainers but, of those who drank, a higher proportion of African American women were problem drinkers than was true among white women (Bailey et al. 1965). Epidemiologic data suggest, however, that rates of lifetime alcohol disorders are higher for young white than young African American women but are higher for African American than white women

Table 1. Rates of Lifetime Alcohol Disorders for All Epidemiologic Catchment Areas Combined.

Age	Men		Women	
	White %	Black %	White %	Black %
18–29	29	13	7	4
30–59	24	32	4	7
60+	13	24	2	3

Note: Data from Robins 1989.

in middle and later life (Robins 1989; Helzer et al. 1991). This age by race crossover in heavy drinking has also been found in other studies (S.C. Wilsnack and Wilsnack 1991). For example, tables 2 and 3 show data on heavy drinking from the 1991 and 1993 National Household Survey on Drug Abuse (Gerstein et al. 1996). As shown in these tables, peaks in heavy drinking differ by ethnic and racial background. For both African Americans and Hispanics, heavy drinking tends to peak later than that for whites. This tends to be true for both males and females.

Russell and colleagues (1990) found that positive family history of alcohol abuse/dependence interacted with gender, race, and age. Among whites positive family history increased in importance with age and was higher for females than males; but among African Americans it decreased in importance with increasing age and was more important for males than females. The age trajectory may describe social drinking, heavy drinking, problems associated with alcohol, or lifetime alcohol disorder diagnosis.

Explanations for the African American Age Trajectory of Alcohol Use

At least five hypotheses or explanations have been offered for the African American age trajectory of alcohol use. These explanations relate to urban migration and generational differences, health status, midlife crisis, social and psychological factors, and accumulated stress.

The first hypothesis suggests that a crucial causal factor is *urban migration and second-generation status* of the offspring of these migrants (Robins et al. 1988*a*). Specifically, the differences in education and access to the opportunity structure between African Americans migrating northward from the rural South and the generation that followed them are at the heart of the observed age pattern (Robins 1989). This is a sociohistorical, structural explanation which suggests that the phenomenon may or may not appear in the next generation and, if it does appear, it may be attenuated. If this is the case we would expect to find significant differences among third-generation African Americans in the patterns that existed for their parents and grandparents. While we would have to depend upon differences in current and historical reports and short-term changes over 5 years in the younger cohorts, a relatively short longitudinal study may shed light on possible age and cohort differences.

The second explanation focuses on the *declining health status* of African Americans age 40 and older (Gibson 1986; J.S. Jackson 1993) and the associated personal problems, interpersonal problems, and distress (e.g., Gibson 1986). A comparison of health status in 1990 showed a higher proportion of African Americans assessed as in poor/fair health (15.3 percent) than whites (9.4 percent) (National Center of Health Statistics 1994). It is suggested that increasing problems with both acute and chronic ailments in middle age and beyond are related to increased consumption and frequency of alcohol use among African Americans. Although the life

stage emphasized here is middle age, clearly this effect would be seen in other age groups as well if self-medication or co-occurring conditions are the major reasons for the increased use. Similarly, support for this explanation would require us to find relationships among health and other co-occurring conditions with alcohol use, independent of age, combined with a lack of support for the cohort change explanation offered in the urban migration and second-generation status approach.

The third explanation is related to declining health status and associates increased use and abuse of alcohol with life-stage events; specifically, a unique form of *midlife crisis,* particularly among African American males, manifested as depression and despair (Gibson 1986; J.S. Jackson 1993). It has been proposed that African Americans now in middle age, more

Table 2. Percentage Reporting Four or More Drinks in Any Single Day in Past 12 Months, by Age

	12–17	18–25	26–30	31–35	36–40
Males					
White	13.0	51.5	47.3	40.7	34.6
Black	6.3	30.2	37.7	36.3	30.5
Hispanic	10.3	44.7	51.7	46.7	45.3
Females					
White	9.3	31.6	22.0	16.1	15.3
Black	2.5	11.3	15.1	14.5	12.8
Hispanic	6.6	14.2	13.4	14.4	12.6

Note: Question wording was: "What is the *most* you had to drink on any *one* day during the past 30 days?"
Data from the 1994 National Household Survey on Drug Abuse Public Use Files and Codebooks, Office of Applied Statistics, Substance Abuse and Mental Health Services Administration.

Table 3. Percentage Reporting Four or More Drinks in Any Single Day in Past 12 Months, by Age

	12–17	18–25	26–30	31–35	36–40
Males					
White	9.5	52.7	50.8	44.1	34.1
Black	2.8	26.4	42.6	39.9	32.0
Hispanic	9.1	47.8	50.1	46.7	43.7
Females					
White	2.0	6.2	3.5	2.3	3.7
Black	0.4	2.6	3.7	5.8	5.6
Hispanic	0.8	2.8	2.5	2.2	2.9

Note: Question wording was: "What is the *most* you had to drink on any *one* day during the past 30 days?" na = not available.
Data from the 1994 National Household Survey on Drug Abuse Public Use Files and Codebooks, Office of Applied Statistics, Substance Abuse and Mental Health Services Administration.

than any other age group among African Americans, may find their accomplishments falling far short of their aspirations. Middle-aged African Americans may find themselves in the least advantaged position, whether they compare themselves with middle-aged whites or young African American adults. If acute changes with midlife stage is the major associated factor for increased alcohol use and abuse, we would expect to find sharp age differences among middle-aged and older African Americans, as compared with younger African Americans, in alcohol use and abuse. Such a finding alone would not permit us to separate out life-stage from cohort differences, but finding sharp age differences that are independent of the health-related explanations, in combination with a lack of support for generational or cohort effects, would provide some support for this hypothesis.

Group, Gender, and Race, in National Household Survey on Drug Abuse, 1991.

41–45	46–50	51–55	56–60	61–65	66+	Total
27.5	23.7	29.2	16.2	16.9	7.4	32.3
35.6	25.9	30.9	16.9	14.0	10.4	23.0
46.7	38.5	34.9	30.9	27.5	18.8	34.9
14.6	9.5	10.4	7.8	3.3	0.9	17.0
12.0	11.4	9.4	4.7	5.8	0.8	9.6
8.3	7.1	5.7	6.3	4.4	1.7	10.5

Group, Gender, and Race, in National Household Survey on Drug Abuse, 1993.

41–45	46–50	51–55	56–60	61–65	66+	Total
29.2	28.4	21.2	17.6	20.0	11.9	34.4
33.0	18.1	61.5	12.5	16.7	6.5	23.2
39.6	44.2	31.1	24.0	13.6	10.0	34.3
1.6	2.3	0.9	2.4	na	0.3	3.1
3.8	0.7	3.8	0.9	2.9	0.4	2.7
na	3.5	0.8	0.9	1.1	0.6	1.9

The fourth explanation suggests that *social and psychological factors* (e.g., feelings of social integration, religion, social support, personality factors) may serve to inhibit or facilitate alcohol use and abuse. These factors may reflect significant differences in "the cultural environment of drinking" for African Americans and whites (Herd 1990). While these factors could play inhibiting and facilitating roles at all points in the life course and across age cohorts, we might expect to find that they have a similar pattern of relationships to measures of alcohol use/abuse across the different age groups and that the relationships remain fairly consistent within individuals across time. It is possible that the factors may interact with age changes, cohort changes, or life-stage transitions. Evaluation of the validity of this explanation depends heavily on the empirical outcomes of the predictions in the first four hypothesized explanations. In any case, it would be important to know how group-based phenomena such as identity, social support, and religiosity either directly affect use and abuse of alcohol or interact with other structural and life-course–related factors to influence health status and use and abuse of alcohol.

The fifth explanation suggests that a response to *accumulated stress* over the life course due to racism and related structural impediments, including lower incomes and lower social status than whites, accounts for increased use and abuse of alcohol over the latter part of the life course. Among African American men, alcohol dependence and negative drinking consequences are related to lower education (Herd 1991) and also living in poverty-concentrated neighborhoods (Jones-Webb et al. 1997). Assessing the validity of this explanation would depend upon demonstrating the existence of a greater number of stressors among African Americans in older age; ascertaining effects from prior stressors, in terms of both recall of circumstances earlier in life and short-term prospective changes over time; and a relatively important role of age in combination with stressors to influence use and abuse of alcohol. Again, assessing the validity of this explanation depends heavily on finding little support for the first three hypotheses and a relatively smaller inhibiting or facilitating role of social and psychological factors in older ages. This fifth explanation is not independent of the first four but suggests a slightly different mechanism for why African Americans may show a pattern of increased use and abuse of alcohol in older ages. We also must be aware that accumulated stress may not exert direct effects on alcohol use and abuse but instead may have influences through the fostering of other co-occurring conditions.

Future Research

We believe that a new, in-depth community study is needed to (1) investigate the age trajectories in drinking among African American men and women; (2) test specific hypotheses about psychological, social, structural, and cultural factors, and possible mediators, related to age differences in African American drinking and heavy

drinking; and (3) examine possible co-occurring health and mental health conditions among African American high-risk drinkers and alcohol abusers.

Several aspects of the proposed approach would be different from most earlier research. First, we propose to study urban African American populations in defined geographic areas. This strategy is analogous to that used in the ECA study but would contain a larger sample of African Americans than in any of the ECA sites. The African American sample in the ECA study numbered 420 in New Haven, CT, 1,182 in Baltimore, MD, 1,158 in St. Louis, MO, 1,392 in North Carolina, and 135 in Los Angeles. A common problem with race and ethnic comparisons in alcohol and mental health research is the small sample sizes of the minority groups. The ECA study addressed this problem to some degree, but questions have been raised about the extent to which the surveys captured the full socioeconomic spectrum of the African American population (D.R. Williams 1986; Neal and Turner 1991). Even with the relatively large numbers of African Americans in the ECA data, researchers focusing on this population are sometimes forced to aggregate all African Americans across the various sites (D.R. Williams et al. 1992*a*, 1992*b*). The sampling approach we propose would cover a broader range of the sociodemographic spectrum of African Americans than did previous studies.

Second, the study should utilize a broader array of psychosocial risk factors and group, personal, and socioeconomic resources than have been previously used. Previous studies have not adequately considered the potentially unique factors that may exist among African Americans, such as religious beliefs and practices, beliefs of having personal control of one's life, world views, family structure, values, health beliefs and practices, and group identity and consciousness.

Third, the design of the proposed study would yield important cross-sectional and prospective data. The initial data collection would tell us much about the distribution of serious mental disorders and alcohol use/abuse and the relationships of these health outcomes to community stressors, social stress, and family and personal characteristics.

The proposed research would attempt to link disparate literatures to assess the potentially unique factors related to alcohol use among African Americans. This integrative approach is essential to understanding the processes by which social and psychosocial factors affect health status. Alcohol abuse does not occur in isolation but rather is closely associated with a number of deviant behaviors (Jessor and Jessor 1977; Dembo et al. 1985; Herd 1994); low self-efficacy beliefs and nonsupportive disorganized family structures (Chein 1980); school attendance and chronic achievement-related anxieties (Misra 1980; Brunswick and Messeri 1984; Friedman et al. 1985; Bachman et al. 1988); inability to establish future-oriented goals (Ausubel 1980); role strain or deprivation (Brunswick 1979; Winick 1980; R.W. Wilsnack and Cheloha 1987); low employment status (Steinberg and Dorbusch 1991); low religious involvement (Donovan and Jessor 1985;

Selnow 1985; Bachman et al. 1988); and family and environmental factors (Newcomb et al. 1981; Brunswick and Messeri 1984). Most of this prior research, however, has focused on the relationship between a single behavior and alcohol and other drug use rather than an examination of behavioral patterns.

What is needed is a study of how potentially health-enhancing factors (social ties, religious involvement, self-esteem, personal control) are linked to stress and combine to affect alcohol use among African Americans. African Americans are disproportionately exposed to social conditions considered to be important risk factors for mental disorders and alcohol abuse (Helzer et al. 1991; Robins and Regier 1991). This emphasizes the need for renewed attention to identify the cultural strengths and health-enhancing resources that might provide protection from pathogenic risk factors. Much of the prior research has focused only on pathology and deficits (Herd 1987). We believe that an exclusive focus on inadequacy provides a distorted view of alcohol use and abuse in any population group.

Our general hypotheses, discussed earlier in the chapter, could be investigated by focusing on the following areas:

- birthplace of parents and grandparents, year of migration, and whether the migration was rural South to urban North
- health status, practices, and history, including onset of chronic acute illnesses and medical problems in middle age and beyond
- midlife crises, using items derived from subjective well-being studies and direct questions about comparison with younger and older age groups; analyses of these items should permit identification of the stresses described by Gibson (1986)
- identification of the unique stresses and adaptive resources relevant to alcohol use in the African American community, using items about childhood stressors, adolescent stressors, stressors of young adult life, and current stressors; a cumulative stress index can be derived from these data.

We expect that research questions concerning age trajectories can be tested using straightforward analytical methods (e.g., Glenn 1977).

CONCLUSIONS

Older age is not inevitably linked to physical and psychological decline (Rowe 1985; J.S. Jackson 1996; J.S. Jackson et al. 1990; Rowe and Kahn 1998). Changes in lifestyles, reductions in environmental risks, and medical interventions can have profound influences on the quantity and quality of physical and psychological health in late life among older adults, even given the negative life-course experiences suffered by African Americans. Some data (e.g., Gibson and Jackson 1987) show that some African American disadvantaged older groups are free from functional disability and limitations of activity due to chronic illness and disease. For example, after the age of 65, African

Americans and whites, within sex groups, differ very little in years of expected remaining life. Health care has improved significantly for older adults, and consecutive cohorts have been better educated and better able to take advantage of available opportunities.

We are beginning to see a convergence of theory and research derived from resource-based, life-course models of health and effective functioning (e.g., Baltes and Baltes 1990). The literature briefly reviewed in this chapter suggests some directions that theory and research on alcohol use and abuse over the life course might take (Holzberg 1982; Rosenthal 1986; Barresi 1987; Fry 1988; Carstensen 1993; Mariske et al. 1995).

The life-course framework holds great promise. There is a need, however, for a greater infusion of cultural and ethnic aging considerations (Gelfand and Barresi 1987; Fry 1988). Theories, research paradigms, service delivery models, and public policies continue to be developed that are not sensitive to the ever-increasing ethnically and culturally diverse segments of our population. Culture and lifestyle differences are of fundamental importance in psychological constructs, theories, and interventions (Holzberg 1982; J.J. Jackson 1985; Rosenthal 1986). Some studies show that recognition and sensitivity to cultural and racial factors in service delivery programs can increase the effectiveness and reduce the cost of delivering services to some populations (J.S. Jackson et al. 1992). It also has been suggested that the infusion of cultural content has positive effects on the health of the nation more broadly, regardless of whether the direct focus of that work is on specific racial and ethnic groups (Cooper et al. 1981).

A positive future for life-course research, practice, and policy related to aging and age-related changes in physical and psychological health can be seen in the increased emphasis on the important contextual variables of race, culture, ethnicity, gender, and social and economic statuses (J.S. Jackson et al. 1990). A recent Institute of Medicine report on aging research in the behavioral and social sciences (Lonergan 1991) concluded that during the last 40 years an impressive acquisition of knowledge on the nature of aging in sensory, behavioral, and cognitive systems has occurred. These findings indicate that (a) people do not experience declines and increments in age the same way, (b) some aging processes are modifiable, and (c) observed functional differences across individuals are greatly influenced by societal, environmental, and health-related statuses (J.S. Jackson 1996). Intergenerational models of aging and human development are needed to understand individual aging trajectories; there is undoubtedly a causal role of period events and cohort membership on aging-related physical and psychological health processes (J.S. Jackson 1996). We must conceptualize aging-related changes in these processes among African Americans in the context of individual, family, cultural, and societal life-course frameworks.

Our work proposes to extend the life-course framework to encompass an integrated model of alcohol use and abuse that includes historical, cohort, and cultural influences on successful (and unsuccessful) social, physical, and psychological development and aging. The value of comprehensive life-course models in health and psychological research cannot be overstated. It is the key to the development and implementation of comprehensive theory, appropriate research, successful interventions, and programs to ameliorate poor physical and psychological health conditions related to alcohol use and abuse among African Americans at every point in their individual lifespans.

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REFERENCES

Anderson, E. *A Place on the Corner.* Chicago: University of Chicago Press, 1976.

Antonucci, T.C., and Jackson, J.S. The role of reciprocity in social support. In: Sarason, I.G.; Sarason, B.R.; and Pierce, G.R., eds. *Social Support: An Interactional View.* New York: John Wiley and Sons, 1990. pp 173–198.

Ausubel, D. An interactional approach to narcotic addiction. In: Lettieri, D.J.; Sayers, M.; and Pearson, H., eds. *Theories on Drug Abuse.* Rockville, MD.: National Institute on Drug Abuse, 1980. pp 4–7.

Bachman, J.; O'Malley, P.; and Johnston, L. Period, age, and cohort effects on substance abuse among young Americans: A decade of change, 1976–1986. *Am J Public Health* 78:1315–1321, 1988.

Bailey, M.B.; Haberman, P.W.; and Alksne, H. The epidemiology of alcoholism in an urban residential area. *Q J Stud Alcohol* 26:19–40, 1965.

Baker, F.M. The Afro-American life cycle: Success, failure, and mental health. *J Natl Med Assoc* 7: 625–633, 1987.

Baltes, P.B. Theoretical propositions of life-span developmental psychology: On the dynamics between growth and decline. *Dev Psychol* 23:611–626, 1987.

Baltes, P.B., and Baltes, M.M., eds. *Successful Aging: Perspectives From the Behavioral Sciences.* New York: Cambridge University Press, 1990.

Barker, J.C., and Kramer, B.J. Alcohol consumption among older urban American Indians. *J Stud Alcohol* 57:119–124, 1996.

Barr, K.E.M.; Farrell, M.P.; Barnes, G.M.; and Welte, J.W. Race, class, and gender differences in substance abuse: Evidence of middle-class/underclass polarization among black males. *Soc Probl* 40: 314–327, 1993.

Barresi, C.M. Ethnic aging and the life course. In: Gelfand, D.E., and Barresi, C.M., eds. *Ethnic Dimensions of Aging.* New York: Springer, 1987. pp. 18–34.

Brown, S.A., and Schuckit, M.A. Changes in depression among abstinent alcoholics. *J Stud Alcohol* 49:412–417, 1988.

Brunswick, A. Black youths and drug use behavior: An epidemiologic and longitudinal perspective on drugs used and their

users. In: Beschner, G., and Friedman, A., eds. *Handbook of Longitudinal Research in the U.S.* New York: Praeger Press, 1979. pp. 290–314.

Brunswick, A., and Messeri, P. Causal factors in onset of adolescents' cigarette smoking: A prospective study of urban black youth. *Adv Alcohol Subst Abuse* 3:35–52, 1984.

Cadoret, R.; O'Gorman, T.W.; Heywood, E.; and Troughton, E. Genetic and environmental factors in major depression. *J Affect Disord* 9:155–164, 1985.

Caetano, R. Ethnicity and drinking in northern California: A comparison among whites, blacks and Hispanics. *Alcohol Alcohol* 19:31–44, 1984.

Caetano, R., and Herd, D. Drinking in different social contexts among white, black, and Hispanic men. *Yale J Biol Med* 61:243–258, 1988.

Caetano, R., and Schafer, J. DSM-IV alcohol dependence in a treatment sample of white, black, and Mexican-American men. *Alcohol Clin Exp Res* 20:384–390, 1996.

Cahalan, D.; Cisin, I.; and Crossley, H.M. *American Drinking Practices.* Monograph 6. New Brunswick, NJ: Rutgers Center of Alcohol Studies, 1969.

Carstensen, L.L. Motivation for social contact across the life span: A theory of socioemotional selectivity. In: Jacobs, J., ed. *Nebraska Symposium on Motivation: Developmental Perspectives on Motivation.* Vol. 40. Lincoln: University of Nebraska Press, 1993. pp. 209–254.

Chein, I. Psychological, social and epidemiological factors in juvenile drug use. In: Lettieri, D.J.; Sayers, M.; and Pearson, H., eds. *Theories on Drug Abuse.* Rockville, MD: National Institute on Drug Abuse, 1980. pp. 76–82.

Clark, W.B., and Hilton, M.E., eds. *Alcohol in America: Drinking Practices and Problems.* Albany: State University of New York Press, 1991.

Cooper, E.; Steinhaurer, M.; Schatzkin, A.; and Miller, W. Improved mortality among U.S. blacks, 1968-1978: The role of antiracist struggle. *Int J Health Serv* 11:511–522, 1981.

Cooper, R., and David, R. The biological concept of race and its application to public health and epidemiology. *J Health Polit Policy Law* 11:97–116, 1986.

Cornerly, P. B. The health status of the Negro today and in the future. *Am J Pub Health* 58:647–654, 1968.

Dembo, R.; Schmeidler, J.; Burgos, W.; and Taylor, R. Environment setting and early drug involvement among inner-city junior high school youths. *Int J Addict* 20:1239–1255, 1985.

Donovan, J.E., and Jessor, R. The structure of problem behavior in adolescence and in young adulthood. *J Consult Clin Psychol* 56:762–765, 1985.

Dressler, W. Social support, lifestyle incongruity, and arterial blood pressure in a southern black community. *Psychosom Med* 53(2):81–99, 1991.

Farley, R. The quality of life for black Americans twenty years after the civil rights revolution. *Milbank Q* 65(Suppl 1): 9–34, 1987.

Farley, R. *The New American Reality: Who We Are, How We Got Here, Where We Are Going.* New York: Russell Sage Foundation, 1996.

Franklin, J.E., Jr. Alcoholism among blacks. *Hosp Community Psychiatry* 40:1120–1127, 1989.

Friedman, A.; Glickman, N.; and Utada, A. Does drug and alcohol use lead to failure to graduate from high school? *J Drug Educ* 15:353–364, 1985.

Fry, C. Theories of aging and culture. In: Birren, J.E., and Bengston, V.L., eds. *Emergent Theories of Aging.* New York: Springer, 1988. pp 447–481.

Gelfand, D., and Barresi, C. *Ethnic Dimensions of Aging.* New York: Springer Publishing, 1987.

Geronimus, Y. On teenage childbearing and infant mortality in the United States. *Pop Dev Rev* 13:246–279, 1988.

Gerstein, D.R.; Gray, F.; Epstein, J.; and Ghadialy, R. *Mental Health Estimates From the 1994 National Household Survey on Drug Abuse.* Advance Report No. 16: DHHS Pub. No. (SMA) 96–3103. Washington, D.C.: U.S. Government Printing Office, 1996.

Gibson, R. Blacks in an aging society. *Daedalus* 115: 349–372, 1986.

Gibson, R.C., and Jackson, J.S. Health, physical functioning, and informal supports of the black elderly. *Milbank Q* 65(Suppl 1):1–34, 1987.

Glenn, N.D. *Cohort Analysis.* Beverly Hills, CA: Sage Publications, 1977.

Gomberg, E.S.L. Drugs, alcohol and aging. In: Kozlowski, L.; Annis, H.M.; Cappell, H.D.; Glaser, F.B.; Goodstadt, M.S.; Israel, Y.; Kalant, H.; Sellers, E.M.; and Vingilis, E.R., eds. *Research Advances in Alcohol and Drug Problems.* Vol. 10. New York: Plenum Press, 1990. pp. 171–213.

Gomberg, E.S.L. Women and alcohol: Use and abuse. *J Nerv Ment Dis* 181:211–219, 1993.

Gomberg, E.S.L., and Nelson, B. Black and white older men. Alcohol use and abuse. In: Beresford, T.P., and Gomberg, E.S.L., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995.

Gould, S.J. Why we should not name human races: A biological view. In: Gould, S., ed. *Ever Since Darwin.* New York: W.W. Norton, 1977. pp. 231–236.

Haan, M.N., and Kaplan, G.A. The contribution of socioeconomic position to minority health. In: *Cross-cutting Issues in Minority Health.* Report of the Secretary's Task Force on Black and Minority Health. Vol. 2. Washington, DC: U.S. Department of Health and Human Services, 1985.

Hannerz, U. *Soulside: Inquiries Into Ghetto Culture and Community.* New York: Columbia University Press, 1969.

Harper, F.D., ed. *Alcohol Abuse and Black America.* Alexandria, VA: Douglass Publishers, 1976.

Harper, F.D., and Dawkins, M.P. Alcohol and blacks: Survey of the periodical literature. *Br J Addict* 71:327–334, 1976.

Harper, F.D., and Saifnoorian, E. Drinking patterns among black Americans. In: Pittman, D.J., and White, H.R., eds. *Society, Culture, and Drinking Patterns Reexamined.* New Brunswick, NJ: Rutgers Center of Alcohol Studies, 1991. pp 327–338.

Harvey, W.B. Alcohol abuse and the black community: A contemporary analysis. *J Drug Issues* Winter: 81–91, 1985.

Helzer, J.E.; Burnam, A.; and McEvoy, L.T. Alcohol abuse and dependence. In: Robins, L.N., and Regier, D.A., eds.

Psychiatric Disorders in America: The Epidemiologic Catchment Area Study. New York: Free Press, 1991. pp 81–115.

Herd, D. Rethinking black drinking. *Br J Addict* 82:219–233, 1987.

Herd, D. The epidemiology of drinking patterns and alcohol-related problems among U.S. blacks. In: Spiegler, D.; Tate, D.; Aitken, S.; and Christian, C., eds. *Alcohol Use Among U.S. Ethnic Minorities.* National Institute on Alcohol Abuse and Alcoholism Research Monograph 18. DHHS Pub. No. (ADM) 89–1435. Washington, DC: U.S. Government Printing Office, 1989. pp. 3–50.

Herd, D. Subgroup differences in drinking patterns among black and white men: Results from a national survey. *J Stud Alcohol* 51:221–232, 1990.

Herd, D. Drinking patterns in the black population. In: Clark, W.B., and Hilton, M.E., eds. *Alcohol in America: Drinking Practices and Problems.* Albany: State University of New York Press, 1991. pp 308–328.

Herd, D. Predicting drinking problems among black and white men: Results from a national survey. *J Stud Alcohol* 55:61–71, 1994.

Herd, D. Sex ratios of drinking patterns and problems among blacks and whites: Results from a national survey. *J Stud Alcohol* 58:75–82, 1997.

Holzberg, C.S. Ethnicity and aging: Anthropological perspectives on more than just the minority elderly. *Gerontologist* 22:249–257, 1982.

Jackson, F.L. Race and ethnicity as biological constructs. *Ethn Dis* 2: 120–125, 1992.

Jackson, J.J. Urban black Americans. In: Harwood, A., ed. *Ethnicity and Medical Care.* Cambridge, MA: Harvard University Press, 1981. pp 37–129.

Jackson, J.J. Race, national origin, ethnicity, and aging. In: Binstock, R.B., and Shanas, E., eds. *Handbook of Aging and the Social Sciences.* New York: Van Nostrand Reinhold, 1985. pp. 264–303.

Jackson, J.S. African American experiences through the adult years. In: Kastenbaum, R., ed. *The Encyclopedia of Adult Development.* Phoenix, AZ: Oryx Press, 1993. pp. 18–26.

Jackson, J.S. A life-course perspective on physical and psychological health. In: Resnick, R.J., and Rozensky, R.H., eds. *Health Psychology Through the Life Span: Practice and Research Opportunities.* Washington, DC: American Psychological Association, 1996. pp. 39–57.

Jackson, J.S.; Neighbors, H.W.; and Gurin, G. Findings from a national survey of black mental health: Implications for practice and training. In: Miranda, M.M., and Kitano, H.H.L., eds. *Mental Health Research and Practice.* Washington, DC: U.S. Department of Human Services, National Institute of Mental Health, 1986.

Jackson, J.S.; Antonucci, T.C.; and Gibson, R.C. Cultural, racial, and ethnic minority influences on aging. In: Birren, J.E., and Schaie, K.W., eds. *Handbook of the Psychology of Aging.* 3d ed. New York: Academic Press, 1990. pp. 103–123.

Jackson, J.S.; Burns, C.J.; and Gibson, R.C. An overview of geriatric care in ethnic and racial minority groups. In: Calkins, E.; Davis, P.J.; Ford, A.B.; and Katz, P.R., eds. *Practice of Geriatrics.* 2d ed. Philadelphia: Harcourt Brace Jovanovich, 1992. pp. 57–64.

Jaynes, G., and Williams, R. *A Common Destiny: Blacks and American Society.* Washington, DC: National Academy Press, 1989.

Jessor, R., and Jessor, S. *Problem Behavior and Psychosocial Development: A Longitudinal Study of Youth.* New York: Academic Press, 1977.

Johnston, L.D.; O'Malley, P.M.; and Bachman, J.G. *National Survey Results on Drug Use From the Monitoring the Future Study, 1975–1995. Vol. I: Secondary School Students.* DHHS Pub. No. NIH 96–4139. Rockville, MD: National Institute on Drug Abuse, 1996.

Jones-Webb, R.J.; Hsiao, C.; and Hannan, P. Relationship between socio-economic status and drinking problems among black and white men. *Alcohol Clin Exp Res* 19: 623–627, 1995.

Jones-Webb, R.; Snowden, L.; Herd, D.; Short, B.; and Hannan, P. Alcohol-related problems among black, Hispanic, and white men: The contribution of neighborhood poverty. *J Stud Alcohol* 58:539–545, 1997.

Kessler, R.C.; Crum, R.S.; Warner, L.A.; Nelson, C.B.; Schulenberg, J.; and Anthony, J.C. Lifetime co-occurrence of DSM-III-R alcohol abuse and dependence with other psychiatric disorders in the National Comorbidity Survey. *Arch Gen Psychiatry* 54:313–321, 1997.

Kuo, W.H. Prevalence of depression among Asian Americans. J *Nerv Ment Dis* 172: 449–457, 1984.

Lader, M. The nature of anxiety. *Br J Psychiatry* 121: 481–491, 1972.

Lewis, H. *Blackways of Kent.* Chapel Hill: University of North Carolina Press, 1955.

Lex, B.W. Review of alcohol problems in ethnic minority groups. J *Consult Clin Psych*ol 55:293–300, 1987.

Liebow, E. *Tally's Corner.* Boston: Little Brown, 1967.

Lipscomb, W.R., and Trochi, K. *Black Drinking Practices Study Report to the Department of Alcohol and Drug Programs.* Berkeley, CA: Research Triangle Institute, 1981.

Lonergan, E.T., ed. *Extending Life, Enhancing Life.* Washington, DC: National Academy Press, 1991.

Manton, K.G. Differential life expectancy: Possible explanations during the later years. In: Manual, R.C., ed. *Minority Aging: Sociological and Psychological Issues.* Westport, CT: Greenwood Press, 1982. pp 63–70.

Manton, K., and Soldo, B. Dynamics of health changes in the oldest old: New perspectives and evidence. *Milbank Q Health Soc* 63:206–228, 1985.

Manton, K.; Poss, S.S.; and Wing, S. The black/white mortality crossover: Investigation from the perspective of the components of aging. *Gerontologist* 63:177–186, 1979.

Mariske, M.; Lang, F. R.; Baltes, P.B.; and Baltes, M.M. Selective optimization with compensation: Life-span perspectives on successful human development. In: Dixon, R.A., and Backman, L., eds. *Psychological Compensation: Managing Losses and Promoting Gains.* Hillsdale, NJ: Erlbaum, 1995.

McCabe, M., and Cuellar, J. *American Indian/Alaska Native Elders.* 2d ed. Working Paper Series No. 6. Palo Alto, CA: Stanford Geriatric Education Center, 1994.

Merikangas, K.R., and Gelernter, C.S. Comorbidity for alcoholism and depression. *Psychiatr Clin North Am* 13:613–632, 1990.

Merikangas, K.R.; Risch, N.J.; and Weissman, M.M. Comorbidity and cotransmission of alcoholism, anxiety and depression. *Psychol Med* 23:1–12, 1993.

Miller, J.G. *Search and Destroy: African American Males in the Criminal Justice System.* Cambridge, England: Cambridge University Press, 1996.

Miller, J.M., and Miller, J.M. Alcoholism in a black urban area. *J Natl Med Assoc* 80:621–623, 1988.

Misra, R.K. Achievement, anxiety and addiction. In: Lettieri, D.J.; Sayers, M.; and Pearson, H., eds. *Theories on Drug Abuse.* Rockville, MD: National Institute on Drug Abuse, 1980. pp. 212–214.

Morioka-Douglas, N., and Yeo, G. *Aging and Health: Asian/Pacific Island Elders.* Stanford Geriatric Education Center Working Paper Series No. 3. Ethnogeriatric Reviews. Stanford, CA: Stanford Geriatric Education Center, Division of Family and Community Medicine, Stanford University, 1990.

National Center for Health Statistics. *Health United States.* DHHS Pub. No. (PHS)94–1232. Hyattsville, MD: Public Health Service, 1994.

Neal, A.M., and Turner, S.M. Anxiety disorders research with African-Americans: Current status. *Psychol Bull* 109:400–410, 1991.

Neighbors, H.W. The distribution of psychiatric morbidity in black Americans: A review and suggestions for research. *Community Ment Health J* 20:169–181, 1984.

Newcomb, M.D.; Huba, G.J.; and Bentler, P.M. A multidimensional assessment of stressful life events among adolescents: Derivation and correlates. *J Health Soc Behav* 22:400–415, 1981.

O'Malley, P.M.; Bachman, J.G.; and Johnston, L.D. Period, age, and cohort effects on substance use among young Americans: A decade of change, 1976–1986. *Am J Pub Health* 78:1315–1321, 1988.

Polednak, A.P. *Racial and Ethnic Differences in Disease.* New York: Oxford University Press, 1989.

Rachal, J.Z.; Williams, J.R.; Brehm, M.L.; Cavanaugh, B.; Moore, R.P.; and Eckerman, W.C. *A National Study of Adolescent Drinking Behavior: Attitudes and Correlates.* Research Triangle Institute Center for the Study of Social Behavior Project No. RT123U–891. Research Triangle Park, NC: Research Triangle Institute, 1975.

Regier, D.A.; Farmer, M.E.; Rae, D.S.; Locke, B.Z.; Keith, S.J.; Judd, L.L.; and Goodwin, F.K. Comorbidity of mental disorders with alcohol and drug abuse. *JAMA* 264:2511–2518, 1990.

Richardson, J. *Aging and Health: Black Elders.* Stanford Geriatric Education Center Working Paper Series No. 4. Ethnogeriatric Reviews. Stanford, CA: Stanford Geriatric Education Center, Division of Family and Community Medicine, Stanford University, 1990.

Riley, M.W. Aging and society: Past, present, and future. *Gerontologist* 34:436–446, 1994*a*.

Riley, M.W. Changing lives and changing social structures: Common concerns of social science and public health. *Am J Pub Health* 84:1214–1217, 1994*b*.

Riley, M.W., and Riley, J.W. Age integration and the lives of older people. *Gerontologist* 34:110–115, 1994.

Robins, L.N. Alcohol abuse in blacks and whites as indicated in the Epidemiological Catchment Area Program. In: Spiegler, D.; Tate, D.; Aitken, S.; and Christian, C., eds. *Alcohol Use Among U.S. Ethnic Minorities.* National Institute on Alcohol Abuse and Alcoholism Research Monograph 18. DHHS Pub. No. (ADM) 89–1435. Washington, DC: U.S. Government Printing Office, 1989. pp. 75–94.

Robins, L.N., and Regier, D.A., eds. *Psychiatric Disorders in America.* New York: Free Press, 1991.

Robins, L.N.; Helzer, J.E.; Pryzbeck, T.R.; and Regier, D.A. Alcohol disorders in the community: A report from the Epidemiologic Catchment Area. In: Rose, R.M., and Barrett, J. *Alcoholism: Origins and Outcome.* New York: Raven Press, 1988*a*. pp. 15–30.

Robins, L.N.; Wing, L.; Wittchen, H.U.; and Helzer, J.E. The composite international diagnostic interview: An epidemiologic instrument suitable for use in conjunction with different diagnostic systems and in different cultures. *Arch Gen Psychiatry* 45:1069–1077, 1988*b*.

Rosenthal, C.J. Family supports in later life: Does ethnicity make a difference. *Gerontologist* 26:19–24, 1986.

Rowe, J.W. Health care of the elderly. *N Engl J Med* 312:827–835, 1985.

Rowe, J.W., and Kahn, R.L. *Successful Aging.* New York: Pantheon Books, 1998.

Russell, M.; Cooper, M.L.; and Frone, M.R. The influence of sociodemographic characteristics on familial alcohol problems: Data from a community sample. *Alcohol Clin Exp Res* 14:221–226, 1990.

Schoenborn, C.A. Exposure to alcoholism in the family: United States, 1988. *Advance Data From the Vital and Health Statistics of the National Center for Health Statistics* 205:1–13, 1991.

Schuckit, M.A., and Morrissey, E.R. Alcoholism in women: Some clinical and social perspectives with an emphasis on possible subtypes. In: Greenblatt, M., and Schuckit, M.A., eds. *Alcoholism Problems in Women and Children.* New York: Grune & Stratton, 1976. pp 5–35.

Selnow, G. Using a stratified approach in substance intervention and prevention programs among adolescents: An empirical analysis. *J Drug Educ* 15:327–341, 1985.

Siegel, J.S., and Taeuber, C.M. Demographic perspectives on the longlived society. *Daedalus* 115:77–118, 1986.

Skarupski, K.A.; McGee, H.B.; Rafferty, A.P.; and Reeves, M. *Health Risk Behaviors Among African Americans in Michigan.* Lansing: Michigan Department of Community Health and the Michigan Public Health Institute, 1996.

Smail, P.; Stockwell, T.; Canter, S.; and Hodgson, R. Alcohol dependence and phobic anxiety states: A prevalence study. *Br J Psychiatry* 144:53–57, 1984.

Steinberg, L., and Dorbusch, A. Negative correlates of part-time employment during adolescence: Replication and elaboration. *Dev Psychol* 27:304–313, 1991.

Warheit, G.J.; Auth, J.B.; and Black, B.S. Alcohol behaviors among southern blacks and whites: A comparative analysis. In: Spiegler, D.; Tate, D.; Aiken, S.; and Christian, C., eds. *Alcohol Use Among Ethnic Minorities.* National Institute on Alcohol Abuse and Alcoholism Research Monograph 18. DHHS Pub. No. (ADM)

89–1435. Washington, DC: U.S. Government Printing Office, 1989. pp. 95–112.

Watts, T.D., and Wright, R., Jr., eds. *Black Alcoholism: Toward a Comprehensive Understanding.* Springfield, IL: Charles C. Thomas, 1983.

Williams, D.R. The epidemiology of mental illness in Afro-Americans. *Hosp Community Psychiatry* 37:42–49, 1986.

Williams, D.R. Social structure and health behaviors of blacks. In: Schaie, K.W.; Blazer, D.; and House, J.S., eds. *Aging, Health Behaviors and Health Status.* New York: Erlbaum Publishing, 1992. pp. 59–64.

Williams, D.R., and Fenton, B. Psychiatric disorders in African Americans. In: Livingston, I.L., ed. *Handbook of Black American Health: The Mosaic of Conditions, Issues, Policies and Prospects.* New York: Greenwood, 1994. pp. 253–268.

Williams, D.R.; Takeuchi, D.T.; and Adair, R.K. Marital status and psychiatric disorders among blacks and whites. *J Health Soc Behav* 33:140–157, 1992*a*.

Williams, D.R.; Takeuchi, D.T.; and Adair, R.K. Socioeconomic status and psychiatric disorder among blacks and whites. *Soc Forces* 71:179–194, 1992*b*.

Williams, D.W. Socioeconomic differentials in health: A review and redirection. *Soc Psychol Q* 53:280–299, 1990.

Wilsnack, R.W., and Cheloha, L. Women's roles in problem drinking across the life span. *Soc Probl* 35:231–248, 1987.

Wilsnack, R.W.; Harris, T.R.; and Wilsnack, S.C. Changes in U.S. women's drinking: 1981–1991. Presentation at the 19th Annual Alcohol Epidemiology Symposium of the Kettil Brun Society for Social and Epidemiological Research on Alcohol, Krakow, Poland, June 7–11, 1993.

Wilsnack, S.C., and Wilsnack, R.W. Epidemiology of women's drinking. *J Subst Abuse* 3:133–157, 1991.

Winick, M., ed. *The Columbia Encyclopedia of Nutrition.* New York: G.P. Putnam, 1980.

Woodruff, R.A.; Guze, S.B.; Clayton, P.J.; and Carr, D. Alcoholism and depression. *Arch Gen Psychiatry* 28:97–100, 1973.

Yu, E.S.; Liu, W.T.; and Kyrzeia, A. Physical and mental health status indicators for Asian American communities. In: *Cross-cutting Issues in Minority Health.* Report of the Secretary's Task Force on Black and Minority Health. Vol. 2. Washington, DC: U.S. Department of Health and Human Services, 1985.

Zucker, R.A., and Gomberg, E.S.L. Etiology of alcoholism reconsidered: The case for a bio-psychosocial process. *Am Psychol* 41:783–793, 1986.

Zucker, R.A.; Fitzgerald, H.E.; and Moses, H.D. Emergence of alcohol problems and the several alcoholisms: A developmental perspective on etiologic theory and life-course trajectory. In: Cicchetti, D., and Cohen, D.J., eds. *Manual of Developmental Psychopathology.* Vol. 2. New York: Wiley, 1995. pp 677–711.

BIOLOGICAL MECHANISMS

Chapter 5

Genetics, Aging, and Alcohol

Gerald E. McClearn, Ph.D.

It is useful to begin a discussion of genetics, alcohol, and the elderly with an examination of the possible nuances of meaning in various assertions of genetic influence. Perhaps the broadest meaning is implied by observations that some attribute "runs in families." It is, of course, well recognized that family members ordinarily share environments as well as hereditary factors. The simple observation of familiality thus requires some supplementary observation to eliminate the possibility that family resemblance is due to the shared environments rather than shared genes. Adoption studies offer one means of making this distinction. Then comparisons may be made of the similarity of some particular attribute (a phenotype, in genetic parlance) of individuals who share genes but not environments to those who share environments but not genes. Such studies have provided evidence (e. g., Schuckit et al. 1972; Goodwin et al. 1973; Cloninger et al. 1981) that environmental influences alone cannot account for the observed degree of familiality of alcoholism.

QUANTITATIVE GENETICS

The type of evidence found in adoption studies is not very revealing about details of the genetic system. Indeed, the general logic of inference does not even require any understanding of the Mendelian basis of genetics. This conceptual basis, however, with its familiar paired elements, does provide the logical foundation for the general model of *quantitative genetics*, which involves comparisons

G.E. McClearn, Ph.D., is director of the Center for Developmental and Health Genetics and an Evan Pugh Professor of health and human development in the Department of Biobehavioral Health, College of Health and Human Development, The Pennsylvania State University, 101 Amy Gardner House, University Park, PA 16802.

of the degree of similarity of individuals of differing biological relatedness. The theory of quantitative genetics permits the attribution of individual differences to different sources of influence—genetic and environmental in the broadest terms—with different subcategories in each of these main categories estimable from certain comparisons. A frequently used index is *heritability,* which is the proportion of the total variability among individuals of a population with respect to some attribute that is due to genetic differences among them. The index of *environmentality* is simply the flip side—the proportion of total phenotypic variance due to differences among the individuals in the environmental influences they have encountered. In application to the complex phenotypes addressed in alcohol research, it is clearly not appropriate to inquire if some phenotype is "genetic" or not. Only in extreme cases will there be no genetic influence; similarly, only in extreme cases will all of the influence be genetic in origin. The relevant question is, What are the relative proportions of influence of genetic and environmental origin?

The level of understanding provided by quantitative genetics constitutes most of the available evidence on genetics of alcohol-related processes in humans or in animal models. Twin studies, family studies, and adoption studies collectively have provided evidence of genetic involvement in the human encounter with alcohol (see McClearn and Plomin 1995 for a review). Studies of inbred strains and selectively bred lines have produced much of the corpus of knowledge in animal, particularly rodent, models (see Crabbe and Harris 1991 for a review). These studies have revealed significant genetic influence on many alcohol-related processes, but, of course, only the consequences of gene action are assessed; the responsible genes are anonymous, and one does not know how many may be operative, nor where they might be located on the chromosomes.

THE EFFECTS OF SPECIFIC GENES

The default assumption in dealing with polygenic systems is generally that the constituent genes have equal and small effects. It has long been appreciated that this is not necessarily true, although the difficulty in characterizing differential effect size has been, until very recent times, quite substantial. In the limiting case, one of the genes may have so substantial an influence that its presence is detectable in almost any environmental set of circumstances and, for the most part, regardless of other genes present in the organism. Such genes are the stuff of classical Mendelian genetics and are avidly sought in research on complex phenotypes.

There is, to be sure, a significant benefit gained from the identification of a single-locus effect. The predictive efficiency with respect to outcomes of particular matings is more clear-cut, for example, with consequences for genetic counseling as well as for research purposes. In many cases these

benefits are realized simply by virtue of the demonstration that a major locus exists—they do not require any information about the location or structure of the gene. More substantial benefits accrue from knowledge about chromosomal location, and even more substantial ones from knowledge of the molecular structure of the gene.

These single-locus benefits are being sought in the study of alcohol phenotypes as relationships are explored with already known genes that, for one reason or another, are hypothesized to play a mechanistic role in the phenotype. In animal research, for example, Crabbe and colleagues (1996) have examined the effects of a "knock-out" manipulation of a locus affecting serotonin receptors on alcohol consumption in mice. The pursuit of this candidate gene approach is most promising, but, of course, it can only exploit genes that have already been found.

The rapid advance in characterizing the genomes of human beings and of certain model animal species has provided a powerful tool for searching for these intermediate genes, now conventionally called quantitative trait loci (QTLs). The various techniques for the searches have been applied to a wide diversity of phenotypes, and there has been a substantial effort applied to alcohol-related processes. As a result, numerous QTLs have been tentatively identified for various indices of voluntary alcohol consumption, sensitivity to administered alcohol, and withdrawal severity (see Crabbe et al. 1994 for a review). These identifications give a general chromosomal location (the QTL can be said to be somewhere in the neighborhood of a particular chromosomal marker) of some of the genetic influence on a phenotype. This information is more detailed than that derived from quantitative genetic analysis, and less detailed than that from a specific mapped locus. It can be expected to guide and narrow the search for the effective locus, and it is likely that an explosion of research on mechanism will eventuate from the current frenetic period of QTL identification.

DEVELOPMENTAL GENETICS

At all levels of genetic evidence, developmental processes may be apparent. From the early days of genetic research, the existence of classical Mendelian genes that influence a phenotype with late onset (e.g., Huntington's disease) plainly indicated that genetic effects could have a developmental course. It is possible to imagine that the gene product is being produced from the beginning, with a slow buildup of some metabolite that eventually reaches some threshold. Much better explanations have been provided by the dramatic molecular discoveries about gene regulation, however. These discoveries have made clear that genes can be turned on and off developmentally, as well as in response to environmental factors. The mystery of how genes present from conception could fail to show effects until adulthood is now

solved, at least in principle, and the solution offers an exciting new perspective on the "genetics" of a condition.

Thus, with respect to some particular phenotype, the *effective* genotype of an individual can differ substantially from age to age. This is not to say that the genetic information encoded in the DNA changes; it means that the particular assortment of loci being expressed can change. As a consequence, the answer to whatever genetic question we are asking—heritability, QTL, specific base sequence—may well be limited to a particular life stage. Whatever we find out about the genetic architecture of some phenotype in young adults may not be pertinent to the same phenotype in older individuals. On the other hand, it may be. Many genes evidently remain operative through life. So, it is a matter for empirical investigation.

ALCOHOLISM AS A DEVELOPMENTAL PROCESS

There is good reason for believing that a developmental genetic orientation might be a useful one in studies on alcoholism and on alcohol-related processes. Human alcoholism is in many respects a prototype case of a developmental disorder in that it is not present at birth, but has an onset in young adulthood or later. These developmental features have been the subject of considerable behavioral and social research seeking to identify environmental influences that are either risk or protective factors for the developing child or young adult with respect to liability to becoming an alcohol abuser or alcoholic (e.g., Tarter and Vanyukov 1994).

Developmental Genetics of Alcohol-Related Variables

In animal model research, relatively little emphasis has been placed on developmental aspects of the alcohol phenotypes being studied. For the most part, this research has involved measurement of phenotypes at a single age, usually young adulthood. However, a sufficient number of researchers have examined various ages to make clear that a developmental perspective may be highly relevant to alcohol-related processes. Early examples are provided by the work of Parisella and Pritham (1964) in rats and the studies of Kakihana (Kakihana and McClearn 1963; Kakihana 1965; McClearn 1980) in mice.

In Kakihana's studies, developmental genetic processes were implicated both for measures of alcohol consumption and for hypnotic dose sensitivity. Cross-sectional observations of alcohol preference (two-bottle choice, water and 10 percent alcohol solution, with preference ratio defined as proportion of total daily liquid intake from alcohol solution) on male BALB/c mice revealed moderate alcohol consumption (preference ratios of 0.30 to 0.40) from 3 weeks of age to 9 or 10 weeks of age, after which consumption dropped quite abruptly to about 0.15. Longitudinal observations at 4 weeks and 16 weeks of age on BALB/c mice of both sexes gave similar results, with the mean preference ratio

for the former group being 0.50 and that for the latter being 0.12. By contrast to the results for male BALB/c mice reported above, cross-sectional observations on C3H/2, RIII, and DBA/2 mice revealed no systematic changes across the age range from 3 to 12 or 16 weeks. Hypnotic dose sensitivity was assessed by duration of loss of righting response following an intraperitoneal injection of 0.014 mL/g body weight in saline. In a cross-sectional design, male animals of six strains were tested at 4, 8, and 16 weeks of age. The strains were essentially indistinguishable at 4 weeks of age, but increasing differentiation occurred at the later ages, with dramatic differences among the strains at the 16-week measurement.

More recent illustrations of developmental genetic processes are provided by observations from a number of studies. First, the selectively bred long sleep (LS) and short sleep (SS) mice (McClearn and Kakihana 1981) differ in hypnotic dose sensitivity as early as 10 days of age, and the differences increase with age (Smolen et al. 1990). Second, maximum blood alcohol concentrations decrease with age from 1-day-old rats to 60-day-old rats, and the rate of alcohol clearance is slower in the younger animals (Kelly et al. 1987). Third, the stimulatory phase of dose response of gamma-aminobutyric acid and glutamic acid transport systems that is observed in 30- to 40-day-old rats is absent in 60- to 100-day-old animals (Foley and Rhoads 1992). Fourth, stage- and tissue-specific patterns of expression of the alcohol dehydrogenase 1 gene are observed during prenatal and perinatal mouse development (Vonesch et al. 1994).

These examples involve a wide variety of measures and suggest that developmental processes may be a near-ubiquitous feature of alcohol-related phenotypes. They make plausible the hypothesis that a different constellation of genetic factors influences the propensity to consume alcohol and the sensitivity of its effects in older than in younger individuals.

Developmental Processes in Human Alcohol Studies

Genetic factors have also been emphasized in the search for developmental processes in human alcohol studies. For example, studies utilizing young, nonalcoholic offspring of alcoholics (see review by Begleiter and Porjesz 1988; Schuckit 1987) either explicitly or implicitly engage both an assumption of genetically transmissible influence and an assumption of developmental process. A pertinent example of research of this ilk is the proposal of genetic heterogeneity in alcoholism based on age of onset (Cloninger 1987). Devor (1994) has called attention to the developmental complexities of alcoholism in a genetic context.

Not only alcohol abuse and alcoholism but also alcohol consumption within the normal range may be subject to genetic influences that are temporally heterogeneous. Pertinent research on twins of various ages has recently been reviewed by Heller and McClearn (1995). In general, the results of these studies suggest that

between one-third and two-thirds of the population variability in quantity and frequency of alcohol use is heritable. The age ranges differed substantially in the different studies, and in several studies the study population was subdivided to examine possible age differences in heritability. Partanen and colleagues (1966), for example, found heritability to be higher in younger than in older Finnish twins (ages 28–37). Kaprio and colleagues (1981) examined another Finnish sample ranging from 18 to over 60 years of age. Heritability appeared to be about the same up to 59 years of age, with a dramatic decline after 60. In an Australian sample, Jardine and Martin (1984) found that, for males, heritability was much higher in the 18–30 age group than in the over 30 age group. Heller and McClearn (1995), from observations on a Swedish twin sample, suggested a heritability of about 0.25 for females 47–79, but essentially no heritability at all in males. These results are fairly consistent in suggesting a decline in heritability of alcohol use in advanced age. They also raise the prospects of a substantial sex differential in this age-related effect.

CONCLUSIONS

Both theoretical considerations and a small body of literature suggest that the genetic contribution to individual differences in the human response to alcohol may be substantially different at different ages. The utilization of the full range of developmental methods available to animal researchers, in concert with further human studies, could constitute a major research agenda, with very substantial prospects for significant illumination of alcohol-related processes.

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REFERENCES

Begleiter, H., and Porjesz, B. Neurophysiological dysfunction in alcoholism. In: Rose, R.M., and Barrett, J., eds. *Alcoholism: Origins and Outcome.* New York: Raven Press, 1988. pp. 157–172.

Blum, K.; Noble, E.P.; Sheridan, P.J.; Montgomery, A.; Ritchie, T.; Jagadeeswaran, P.; Nogami, H.; Briggs, A.H.; and Cohn, J.B. Allelic association of human dopamine D2 receptor gene in alcoholism. *JAMA* 263:2055–2060, 1990.

Cloninger, C.R. Neurogenetic adaptive mechanisms in alcoholism. *Science* 236:410–416, 1987.

Cloninger, C.R.; Bohman, M.; and Sigvardsson, S. Inheritance of alcohol abuse: Cross-fostering analysis of adopted men. *Arch Gen Psychiatry* 38:861–868, 1981.

Crabbe, J.C., and Harris, R.A. *The Genetic Basis of Alcohol and Drug Actions.* New York: Plenum Press, 1991.

Crabbe, J.C.; Belknap, J.K.; and Buck, K.J. Genetic animal models of alcohol and drug abuse. *Science* 264:1715–1723, 1994.

Crabbe, J.C.; Phillips, T.J.; Feller, D.J.; Hen, R.; Wenger, C.D.; Lessov, C.N.; and Schafer, G.L. Elevated alcohol con-

sumption in null mutant mice lacking 5-HT_{1B} serotonin receptors. *Nat Genet* 14:98–101, 1996.

Devor, E.J. A developmental-genetic model of alcoholism: Implications for genetic research. *J Consult Clin Psychol* 62:1108–1115, 1994.

Foley, T.D., and Rhoads, D.E. Effects of ethanol on Na(+) -dependent amino acid uptake: Dependence on rat age and Na+, K(+) -ATPase activity. *Brain Res* 593:39–44, 1992.

Goodwin, D.W.; Schulsinger, F.; Hermansen, L.; Guze, S.B.; and Winokur, G. Alcohol problems in adoptees raised apart from alcoholic biological parents. *Arch Gen Psychiatry* 28:238–243, 1973.

Heller, D.A., and McClearn, G.E. Alcohol, aging, and genetics. In: Beresford, T., and Gomberg, E., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 99–114.

Jardine, R., and Martin, N.G. Causes of variation in drinking habits in a large twin sample. *Acta Genet Med Gemellol* 33:435–450, 1984.

Kakihana, R.Y. Developmental study of preference for and tolerance to ethanol in inbred strains of mice. Unpublished doctoral dissertation, University of California, Berkeley, 1965.

Kakihana, R., and McClearn, G.E. Development of alcohol preference in BALB/c mice. *Nature* 199:511–512, 1963.

Kaprio, J.; Koskenvuo, M.; and Sarna, S. Cigarette smoking, use of alcohol, and leisure-time physical activity among same-sexed adult male twins. In: Gedda, L.; Parisi, P.; and Nance, W., eds. *Twin Research 3: Epidemiological and Clinical Studies.* New York: Alan R. Liss, 1981. pp. 37–46.

Kelly, S.J.; Bonthius, D.J.; and West J.R. Developmental changes in alcohol pharmacokinetics in rats. *Alcohol Clin Exp Res* 11:281–286, 1987.

McClearn, G.E. Animal models as pharmacogenetic tools: Some initial explorations into alcohol consumption. In: Maloff, D.R., and Levison, P.K., eds. *Issues in Controlled Substance Use. National Academy of Sciences Committee Report.* Washington, DC: National Academy of Sciences, 1980. pp. 142–171.

McClearn, G.E., and Kakihana, R. Selective breeding for ethanol sensitivity: SS and LS mice. In: McClearn, G.E.; Deitrich, R.A.; and Erwin, V.G., eds. *Development of Animal Models as Pharmacogenetic Tools.* National Institute on Alcohol Abuse and Alcoholism Research Monograph 6. DHEW Pub. No. [ADM] 81–1133. Washington, DC: U.S. Government Printing Office, 1981. pp. 147–159.

McClearn, G.E., and Plomin, R. Strategies for the search for genetic influences in alcohol-related phenotypes. In: Begleiter, H., and Kissin, B., eds. *The Genetics of Alcoholism.* New York: Oxford University Press, 1995. pp. 327–352.

Parisella, R.M., and Pritham, G.H. Effect of age on alcohol preference by rats. *Q J Stud Alcohol* 25:248–252, 1964.

Partanen, J.; Bruun, K.; and Markkanen, T. *Inheritance of Drinking Behavior: A Study of Intelligence, Personality, and Use of Alcohol of Adult Twins.* Helsinki, Finland: Finnish Foundation for Alcoholic Studies, 1966.

Schuckit, M.A. Biological vulnerability to alcoholism. *J Consult Clin Psychol* 55:301–309, 1987.

Schuckit, M.A.; Goodwin, D.W.; and Winokur, G. A study of alcoholism in half siblings. *Am J Psychiatry* 128:1132–1135, 1972.

Smolen, T.N.; Smolen, A.; and van de Kamp, J.L. Developmental profile of hepatic alcohol and aldehyde dehydrogenase activities in long-sleep and short-sleep mice. *Alcohol* 7:69–74, 1990.

Tarter, R.E., and Vanyukov, M. Alcoholism: a developmental disorder. *J Consult Clin Psychol* 62:1096–1107, 1994.

Vonesch, J.L.; Nakshatri, H.; Philippe, M.; Chambon, P.; and Dolle, P. Stage and tissue-specific expression of the alcohol dehydrogenase 1 (Adh-1) gene during mouse development. *Dev Dyn* 199:199–213, 1994.

Chapter 6

Pharmacological Interactions of Aging and Alcohol

Harold Kalant, M.D., Ph.D.

IDENTIFICATION OF THE PROBLEM

It is well known that people tend to drink less alcohol as they get older (Gomberg 1990; Dufour et al. 1992), regardless of the manner in which the intake is measured. Thus, the average dose (in milligrams per kilogram) per drinking occasion was reported to decrease steadily from age 20 to age 70 years, in both men and women, and in both alcoholics and social drinkers (Vogel-Sprott 1983). The average daily intake, which reflects both the frequency of drinking occasions and the dose per occasion, peaks in the middle years (ages 30–45) and then declines steadily beyond that point, in both alcoholics and social drinkers (York 1995). A cross-sectional survey at the start of the Normative Aging Study showed that the proportion of abstainers and very infrequent drinkers rose from 12 percent in those age 49 and under to 30 percent in those age 65 or over, while the proportion taking 3 or more drinks per day decreased from 16 percent to 12 percent (De Labry et al. 1992). Similarly, in a 7-year longitudinal study of a group of healthy elderly men and women, the proportion who used alcohol decreased by about 2 percent a year, and the average daily amount consumed by those who initially drank more than 30 g of ethanol per day declined from 46 g/d to 26 g/d over the 7-year period (Adams et al. 1990).

The generally accepted explanation for this decrease is that older persons become more sensitive to the adverse effects of intoxication (Tarter 1995).

H. Kalant, M.D., Ph.D., is director emeritus of the Addiction Research Foundation of Ontario and professor emeritus of the Department of Pharmacology, University of Toronto, Medical Sciences Building, Toronto, Canada M5S 1A8.

At the same time, it is well recognized that some elderly individuals become heavy drinkers or alcoholics after having drunk alcohol moderately throughout their previous adult life (Ades and Lejoyeux 1994). This is illustrated by the findings in a 19-year followup study in San Francisco, in which the proportion who drank two to three times a day increased from 5.9 percent to 10.6 percent over the 19 years; during the same period the proportion who drank once a month or less rose from 17.7 percent to 33.2 percent (Stall 1986).

The contribution of behavioral pharmacology to the resolution of this seeming paradox involves the following issues:

- Are the elderly really more sensitive to alcohol than younger groups?
- If so, is the difference due to pharmacokinetic or to pharmacodynamic factors, or both?
- Does the difference reflect a true increase in base level of sensitivity, or a loss of ability to adapt to intoxication (i.e., decreased ability to develop tolerance)?
- Does the difference apply equally to all effects of alcohol, including reinforcing as well as aversive effects?

ALCOHOL SENSITIVITY IN THE ELDERLY

It has been observed that in both men and women beyond the age of 60, the frequency of alcohol-induced symptoms is directly proportional to the mean number of drinks consumed per day (Chermack et al. 1996). This is scarcely surprising: at any age, one would expect the frequency of symptoms to be related to the amount consumed and the blood alcohol concentrations (BACs) attained. To determine whether elderly people are especially vulnerable to the adverse effects of alcohol, we need to use direct observation and measurement to answer the following questions:

- Do the elderly reach higher BACs than younger drinkers at the same dose of alcohol per kilogram of body weight?
- Do the elderly show greater impairment than younger drinkers at the same BAC?
- If differences are found between the elderly and younger drinkers, are they due to aging per se, or to illnesses, drug interactions, and other factors that can apply at all ages but are more frequent in the elderly?

PHARMACOKINETIC EFFECTS OF AGING

There is very clear evidence that aging does result in higher BACs for a given dose per kilogram of body weight, because of a change in the proportions of lean body mass and fat. With age, the proportion of fat tends to increase relative to the lean body mass, of which a fairly constant proportion is water (Watson et al. 1980). Since ethanol is essentially confined to total body water, the

same dose in grams per kilogram of total body weight will produce a higher BAC in the individual in whom fat makes up a higher percentage of the body weight (Kalant 1996*b*). When young, middle-age, and old rats were given the same dose of ethanol per gram of body weight, the old rats had both the highest BAC curves and the greatest effects on motor performance and body temperature, while the young rats had the lowest BAC curves and the least effect (Abel and York 1979). In contrast, young and old rats had virtually identical BAC curves when ethanol doses were calculated in milligrams per milliliter of total body water (York 1982).

Similarly in humans, the total body water (as reflected in Roe's ponderal index, an index of body mass equal to the height in centimeters divided by three times the body weight in kilograms) was inversely related to age, but when the alcohol dose was calculated on the basis of the ponderal index, the BAC curve came out exactly as predicted, regardless of age (Vogel-Sprott and Barrett 1984). Such findings provide undeniable evidence that change in body water content, rather than age itself, is the major reason for the higher BACs produced by a given amount of ethanol in the elderly (Scott 1989; Dufour et al. 1992; Egbert 1993). The rates of absorption and elimination of ethanol do not appear to be significantly affected by age alone (Vogel-Sprott and Barrett 1984), though they may be altered by various illnesses that often accompany aging.

PHARMACODYNAMIC DIFFERENCES IN ALCOHOL RESPONSE WITH AGE

The evidence cited in the preceding section indicates that the elderly are likely to have higher BACs than the young after the same amount of alcohol per unit of body weight, but this is not the only factor affecting the response to alcohol. There is also good evidence that, at least with respect to some effects of alcohol, the elderly show a greater effect *at the same BAC* (i.e., that the older nervous system is more sensitive to alcohol). One indication of this increased sensitivity is that in fatal cases of accidental alcohol poisoning uncomplicated by other drugs, the fatal BAC was inversely correlated with age (Poikolainen 1984).

Another indication of increased sensitivity of the older nervous system is found in studies of alcohol-induced impairment. Vogel-Sprott and Barrett (1984), for example, found more than twice as much impairment of balance and of fine motor coordination in elderly subjects as in young adults at the same peak BAC. Similarly, in rats the brain ethanol concentration on recovery from narcosis (loss of righting reflex) was progressively lower with increasing age (York 1983; York and Chan 1994). The degree of alcohol-induced impairment of a learned avoidance response was also greater in old rats than in young ones, and this occurred at a lower BAC in the old rats than in the young (Spirduso et al. 1989).

It is worth noting that this apparently greater sensitivity in the elderly is not

unique to ethanol. Patients aged 80 years and over required an average intravenous dose of 10 mg of diazepam to induce sedation for endoscopic or dental purposes, compared with an average dose of 30 mg for those age 20, and the effective plasma levels showed a similar ratio (Cook et al. 1984). There is similar evidence to suggest that there is greater sensitivity of the brain to diazepam in old rats than in young ones, but surprisingly this does not appear to be true for pentobarbital (Guthrie et al. 1987).

The reason for this difference among the corresponding effects of the various drugs is not clear. There is a need for systematic examination of age-related changes in central nervous system (CNS) sensitivity to a considerably broader range of depressant drugs. It is also apparent that one must be on guard against making unfounded assumptions about the generality of the age effect with respect to *all* actions of alcohol on the CNS. It will be necessary to examine whether or not there is a similar increase in sensitivity to alcohol for a broad range of behaviors, including performance of complex cognitive tasks and their separate components. Some research of this type has in fact been done, with results that are not wholly consistent.

EFFECT OF AGE-ALCOHOL INTERACTIONS ON COMPLEX COGNITIVE FUNCTIONS

There is a considerable body of evidence pointing to greater disruption of cognitive task performance by alcohol in the elderly than in the young, but there is by no means unanimity on this point. The following sections review briefly the findings on a variety of such tasks and their component functions.

Flying Simulator Studies

Collins and Mertens (1988) studied two groups of experienced pilots, age 30–39 years and 60–69 years. Each subject was tested on four different occasions: with and without alcohol, both at ground level and at a simulated altitude of 12,500 ft. The multiple test battery included measures of sensory acuity, reaction times, communication skills, arithmetic, and other task-related performances. The composite score was lower in the older pilots than in the younger ones, and though alcohol reduced the score in both groups and at both altitudes, the impairment was greater in the older group.

Similarly, Morrow and colleagues (1990, 1993), using a difficult simulated course-flying task that involved radio communication concurrently with operation of the flight controls, found a greater impairment of older pilots than of younger ones at BACs of both 40 mg/dL and 100 mg/dL. The age range in this study was rather limited (younger group, 21–29 years; older group, 31–51 years), but the direction of the differences is consistent with that found by Collins and Mertens.

Cognitive Test Batteries

On the other hand, several studies with complex but separated tasks, rather than integrated ones, have revealed no clear difference between young and old. Hultsch and colleagues

(1993) gave a battery of cognitive function tests (including tests for memory, verbal fluency, verbal processing time, and others) to subjects ranging from 55 to 86 years in age. The scores were decreased by age and by alcohol, but the effects of both age and alcohol were small relative to those of disease and of the amount of cognitive activity in which the subjects were habitually engaged. Tupler and colleagues (1995) compared three groups of subjects with mean ages of 25, 41.1, and 60.9 years, using a digit-symbol substitution test, keypad reaction time, and a tracking task. They found an age-related decrease in baseline score, but the addition of alcohol changed only the error pattern and not the error magnitude, and there was no age × alcohol interaction.

Given the difference between the apparently clear-cut findings by Collins and Mertens (1988) and by Morrow and colleagues (1990) and the much less clear-cut ones by Hultsch and colleagues (1993) and by Tupler and colleagues (1995), we cannot reject Cutting's (1988) conclusion that the effect of the interaction of age and alcohol on cognitive functions is by no means certain.

Effects on Specific Components of Psychomotor Performance

Although effects of the age × alcohol interactions on complex behaviors are not consistent, it is worth examining the effects on individual components of complex performance to see if they are more consistent. There is, in fact, substantial literature on this aspect of the subject.

Attention

There is good evidence that divided-attention tasks, requiring the simultaneous monitoring of, and response to, more than one source of information, are impaired by alcohol even at BACs as low as 20–30 mg/dL (Koelega 1995). Visual attention and sensitivity appear to be particularly vulnerable, especially in relation to peripheral vision (Gustafson 1986). For example, Roehrs and colleagues (1994), studying the interaction of alcohol and sleep deprivation, found that alcohol decreased the sleep latency (i.e., it increased sleepiness), increased tracking errors, and increased the visual reaction time, especially to peripheral stimuli. This effect is not specific to alcohol: it has been shown, for example, to occur with the hypnotic drug zolpidem (Wilkinson 1995) as well as with other sedative drugs, and their effects may be synergistic with those of alcohol.

Age has a similar effect on divided attention (Jennings and Jacoby 1993), as well as on peripheral visual attention. Owsley and Ball (1993) found a marked age-related decrease in the diameter of the "useful field of view" (i.e., the field in which the individual could pay sufficient attention to unexpected stimuli to make the responses necessary for safe driving). It would therefore be reasonable to expect a significant interaction between age and alcohol, such that the same BAC would produce greater impairment in the elderly than in the young. However, only a small and marginally significant interaction of this type was found in error scores on a continuous tracking task (Linnoila et al. 1980). This finding

cannot be regarded as conclusive, because the age range of the subjects was small (20–25 years in the younger group and 35–45 in the older), and no elderly subjects were studied.

Other Visual Functions

Apart from the functional constriction of the visual fields, which is accompanied by loss of accommodation and convergence (Hill and Toffolon 1990), alcohol has long been known to decrease light discrimination, critical flicker fusion frequency, and rate of recovery from glare (Wallgren and Barry 1970), as well as visual information processing, visuomotor coordination, and visual reaction time (Krueger 1986). It decreases light sensitivity not only in the peripheral but also the central visual field (Wild et al. 1990). Many of these effects are also produced by other drugs such as benzodiazepines (Krueger 1986).

Several studies suggest that age has similar effects on these functions. For example, critical flicker fusion frequency shows a small but progressive decline over the age range of 10 to 90 years (Lachenmayr et al. 1994), and light difference sensitivity similarly decreases with age from 50 years onward, especially in the peripheral part of the visual field (Brenton and Phelps 1986; Casson et al. 1993; Lachenmayr et al. 1994; Johnson and Marshall 1995). Visual information processing, in the computation of visuospatial relations, is decreased in the elderly, for both peripheral and central field stimulus presentations (Hoyer and Rybash 1992). A broad range of other visual functions are affected by age in ways reminiscent of the effects of alcohol, including retinal illuminance, near focus, smooth and saccadic eye movements, and recovery from glare (Kline 1994).

It would therefore again be reasonable to expect that age and alcohol would act synergistically. Surprisingly, however, the interaction of age and alcohol on these functions does not appear to have been studied.

Central Information Processing

Apart from decreasing the sensitivity and response latency of physiological receptors of various types, both age and alcohol might affect the subsequent central processing of the incoming information. This includes such components as stimulus identification, selecting from among available responses, and matching the stimulus to the appropriate response. Central processing has been studied by various methods.

Moskowitz and Burns (1971) measured the effects of alcohol on the "psychological refractory period" (i.e., the difference in reaction times [RT1 and RT2] to two successive stimuli separated by intervals ranging from 50 to 550 ms). The shorter the interstimulus interval, the greater is the amount by which RT2 exceeds RT1, presumably because the second stimulus arrives while the first is still being processed. Alcohol caused a large increase in this delay time at the short interstimulus intervals, but not at the long ones. An unexplained observation, however, is that both RT1 and RT2 were greatly prolonged by alcohol at the short intervals, but

at the long ones they did not differ from the control reaction times.

Another method of studying the speed of information processing is by measurement of the late components of event-related potentials (ERPs) in the electroencephalogram. For example, the "mismatch negativity," found in the auditory ERP when nonconcordant tone stimuli are presented, is decreased by ethanol in doses as low as 0.55 g/kg, and this is taken as evidence of decreased attention (Jaaskelainen et al. 1995). Many studies have found that the P3 latency is increased and the P3 amplitude decreased by ethanol in a dose-dependent manner, in both visual and auditory ERPs (Teo and Ferguson 1986; Rohrbaugh et al. 1987; Murata et al. 1992; Wall and Ehlers 1995), especially for low-frequency unexpected stimuli (Grillon et al. 1995). Similar but not identical changes have been found with diazepam (Krull et al. 1994).

In the elderly, the P3 amplitude in visual ERPs was not changed significantly with age, but the P3 latency was increased (Porjesz and Begleiter 1982), and the increase in P3 latency related linearly to age, over the range of 20 to 90 years (Pfefferbaum et al. 1984; Kutas et al. 1994). These observations are consistent with other findings that changes of cognitive performance in the elderly consist mainly of reduced speed, but not of reduced accuracy, as in studies with Trail Making Tests (Wahlin et al. 1996).

Since significant changes in central information processing are produced both by alcohol and by age, it would obviously be of considerable interest to see how these two factors interact. However, specific studies on this interaction are lacking.

Learning and Memory

There is a large body of research literature on the impairment of short-term memory by alcohol and on the mechanisms by which alcohol affects the formation of memory traces and results in the alcoholic "blackout" (Mello 1972; Overton 1972; Hashtroudi and Parker 1986; Goodwin 1995). The effects of age on learning and memory have also been studied extensively. One such study involved the learning of the water maze task, in which the animal must learn to use environmental cues to locate an invisible (underwater) platform on which it can rest when forced to swim in a tank of water. Old rats learned the task more slowly than young ones, but the old rats could be divided into two groups: (1) a "good learners" group that improved progressively over successive test days and eventually reached the same level of proficiency as the young rats and (2) a "poor learners" group that showed no significant improvement across test days (Lee et al. 1994). These results appear to offer a differentiation between normal aging and senility.

However, the pattern of impairment by alcohol appears to differ from that produced by age. In a study of verbal memory, immediate recall of word sequences was decreased by alcohol but not by age; in contrast, short-term memory was decreased both by age and by alcohol, but there was no

significant interaction of age × memory (Jones and Jones 1980).

The development of tolerance to alcohol and other drugs has been shown to resemble the processes of learning and memory in numerous ways, and interventions that disrupt learning and memory also prevent the development of tolerance in those paradigms in which tolerance is facilitated by either operant or associative learning (Kalant 1996*a*). This applies not only to chronic tolerance but also to acute tolerance, the form that develops within the duration of a single exposure to alcohol. This form is illustrated by the observation that various signs of intoxication, both in experimental animals and in humans, appear at a lower BAC than that at which they disappear (LeBlanc et al. 1975; Portans et al. 1989). Abel and York (1979) found that acute tolerance of this type was readily demonstrable in young rats, but not in middle-aged or elderly animals. This finding appears consistent with other evidence of decreased learning speed in the elderly.

Driving Skills

Research on driving skills provides a clear indication of the practical importance of studying thoroughly the interactions between the effects of age and alcohol. Driving is a very complex performance that calls on all the cognitive functions mentioned in the preceding sections, including attention (especially divided attention), visuospatial skills, information processing, rapid and continuous integration of new information from the constantly changing surroundings, and speed and accuracy of motor skills required for the safe operation of the vehicle. There is a huge literature, much too large to review here, on the impairing effects of alcohol on driving skills and actual driving performance in both simulated and real road operation.

Age has also been shown to impair driving ability, and there is a U-shaped curve relating accident risk to driver age (Williams and Carsten 1989). In the young, the very high risk is probably attributable to a combination of inexperience, overconfidence, aggressiveness, and lack of mature judgment. The risk decreases rapidly as the young drivers mature, and remains fairly constant between the ages of 30 and 60. Above 60, however, the risk again rises rapidly with increasing age and is now attributable to the progressive impairment of cognitive and motor skills already described. This impairment probably accounts for the increased frequency among elderly drivers of side-impact collisions due to driving through an intersection because of missing a traffic signal (Viano et al. 1990). An additional factor may well be the effects of certain pathological states such as cardiovascular disease, or hypoglycemia in diabetics, that may occur more frequently in older subjects (Waller 1967; Koepsell et al. 1994).

Given the major effects of both alcohol and age, it might be anticipated that the detrimental effect of alcohol on driving would be greater in the elderly than in middle-age, and there is indeed some (though limited) evidence that is consistent with this expectation. The graph relating risk of fatal

accidents to blood alcohol level among Canadian drivers indicates a steeper rate of rise with increasing BAC among those over 55 years of age than among any of the younger groups (Simpson 1985). Similarly, among male drivers in the Grand Rapids Study, the risk of involvement in a collision was increased much more among those aged 70 years or over than among the younger groups at the same BAC (Zylman 1973). However, the same study also showed a disproportionately large increase in collisions due to alcohol in very young drivers, which is consistent with the Finnish observation that alcohol-related traffic fatalities showed two age peaks, one in 18- to 20-year-olds with accidents occurring between midnight and 6:00 a.m. and another in drivers aged 56 and over with accidents occurring in the late afternoon (Summala and Mikkola 1994).

Such evidence, however, is insufficient to answer the questions that naturally arise about the reasons for the greater risk in the elderly. It does not indicate whether the apparently greater sensitivity to impairment by alcohol in the elderly applies to all components of driving ability, or only to some that might be amenable to remedial action, such as night vision, or speed of braking or of steering correction. There is still a clear need for more detailed studies of age × alcohol interaction on specific aspects of total driving safety. Moreover, it must be recalled that the elderly are much more likely to be regular users of other drugs, such as benzodiazepines and antidepressants, with which alcohol may act synergistically. Such drugs can themselves contribute significantly to the impairment of driving skills (Kerr et al. 1992) and to the increased risk of driving accidents in the elderly (Ray et al. 1992), and the extent of possible interaction between them and alcohol requires careful study.

CHRONIC EFFECTS OF ALCOHOL IN THE ELDERLY

The preceding sections dealt with the acute effects of alcohol (i.e., the effects on a single drinking occasion). However, it is also of interest to see whether older subjects are more vulnerable to the chronic effects of alcohol (i.e., the effects resulting from cumulative changes produced by regularly repeated use of alcohol). There have been many suggestions, for example, that chronic intake of large amounts of alcohol results in premature aging of the brain (i.e., loss of brain cells at a higher rate than would occur in normal aging). For example, the increase in cerebrospinal fluid space and the decrease in anterior hippocampal volume revealed by magnetic resonance imaging in alcoholics, in comparison with age-matched healthy controls, suggest a greater cell loss in the elderly alcoholics than in the younger ones (Pfefferbaum et al. 1993; Sullivan et al. 1995). Similarly, the deposition of the degenerative pigment lipofuscin that occurs in the rat hippocampus with aging, occurs earlier and more intensely in rats consuming alcohol than in their pair-fed nonalcohol controls (Borges et al. 1986).

Various facts, however, do not support this concept. In a study of 50- to 60-year-old twins, cognitive function as measured by a test battery was lower in former drinkers than in nondrinkers, but in light to moderate drinkers the performance score improved progressively with increasing alcohol intake up to a maximum of a little over two drinks per day (Christian et al. 1995). There was a suggestion of a decrease in performance at higher levels of intake, and it is possible that the lower scores in ex-drinkers reflected damage in those who had formerly been much heavier drinkers and had stopped for health reasons. The findings at the lower levels of consumption, however, clearly did not support the concept of a simple additive effect of alcohol and aging.

A more detailed analysis of performance on a battery of different cognitive function tests, including digit-symbol substitution, block design, and category recognition, showed the expected decline in performance with increasing age, from 25 to 55 years, but there was no difference in the rate of decline among nondrinkers, social drinkers, alcoholics, and abstinent alcoholics (Page and Cleveland 1987). These findings also fail to support the idea that alcohol produces an increased rate of senescence.

Similarly, in animal studies, learning in the water maze was decreased by age, but not by chronic ethanol consumption, and there was no interaction between age and ethanol (Blokland et al. 1993). There was also no sign of an age × ethanol interaction on a variety of physiological measures in AA rats (the genetically selected alcohol-preferring rats of the Alko line in Finland). AA rats of the same generation were divided into two groups, one consuming water and the other drinking ethanol solution, for their entire lifespan. There was no difference between the groups with respect to survival, weight gain, or ethanol-induced motor impairment, hypothermia, or loss of righting reflex when they were tested at the age of 24 months, which is fairly advanced old age for a rat (Hervonen et al. 1992).

In mice, a clear age effect was found in the form of decreased retention of a learned passive avoidance task, but there was no difference between those that had consumed ethanol chronically and those drinking only water (Samorajski et al. 1982). However, in the same mice, the age-related decrease in spontaneous locomotor activity was significantly greater in the ethanol-drinking mice than in the controls.

On the other hand, there is at least a suggestion of a possible age × alcohol interaction in some observations relating to loss of tolerance. As noted earlier, there is a strong association between learning and some forms of alcohol tolerance. It is therefore worthy of note that, just as age decreases the ability to learn and retain new knowledge, it appears to diminish the extent of an already acquired tolerance to alcohol. Thus, rats that had become tolerant to the anesthetizing effect of alcohol (loss of righting reflex), as a result of prolonged expo-

sure to an alcohol-containing liquid diet, gradually lost their tolerance when the same diet was continued over a period of several months (Khanna et al. 1980). This duration of alcohol exposure is similar to that which had earlier been shown to produce loss of hippocampal neurons (Riley and Walker 1978) and impairment of ability to learn a conditioned avoidance response (Walker and Freund 1971) or a food-motivated maze task (Fehr et al. 1976). A similar phenomenon has been reported in humans: alcoholics who have lost their tolerance are older and have a longer duration of alcoholism than those who are still tolerant (Ziolkowski et al. 1995).

However, the morphological changes in neuronal dendrites found at the microscopic level in rats chronically treated with alcohol are different from those seen in aged rats that have not been treated with alcohol (Pentney 1991). The question of whether alcohol accelerates age-induced changes, or whether the changes produced by age and alcohol are independent but additive, must be considered still unresolved.

AGE AND REINFORCEMENT BY ALCOHOL

As noted at the start of this review, the age-related decrease in alcohol intake is commonly attributed to increased sensitivity to the adverse effects of alcohol. In the terminology of operant psychology, alcohol becomes an increasingly potent punisher. However, there is no a priori reason to reject the alternative possibility, that alcohol becomes progressively less effective as a reinforcer. Conversely, in those cases of late-onset alcoholism, can one explain the development of alcohol problems in the elderly entirely in terms of stress reduction (Welte and Mirand 1995), or is it possible that in some individuals there is increased sensitivity to the reinforcing effects of alcohol? It is necessary to see whether there is any empirical evidence to permit a conclusion about these alternative possibilities.

Dopamine is believed to play a central role in the neuronal processes mediating reinforcement (Koob et al. 1994), especially the dopaminergic pathway from the ventral midbrain to the limbic forebrain (from the ventral tegmental area to the nucleus accumbens). Administration of alcohol has been shown to increase the rate of neuronal activity and the release of dopamine in this pathway, just as amphetamine, cocaine, morphine, and other addictive drugs do (Di Chiara et al. 1993; Di Chiara 1995). It has been shown that amphetamine produces less release of dopamine in the nucleus accumbens in old rats than in young ones, both in vivo and in vitro (Huang et al. 1995). A similar decrease has been found in the nucleus accumbens in mice, during the spontaneous motor activity that is part of the anticipatory response to reinforcers (Watanabe 1987). Aged rats also show a decreased rate of synthesis of new dopamine D_1 receptors in the nucleus accumbens after

the previously existing receptors have been irreversibly inactivated (Giorgi et al. 1992). There is a similarly reduced density of D_1 and D_2 dopamine receptors in the nucleus accumbens in older animals (Morelli et al. 1990). However, there does not appear to be any interaction of age and alcohol, because alcohol did *not* accentuate the age-related decrease of D_1 and D_2 receptors in the accumbens (Woods et al. 1995; Tajuddin and Druse 1996).

CONCLUSIONS

The evidence presented in this chapter calls for great caution against overgeneralization about the interaction of alcohol and aging. It is clear that there are many resemblances between the effects of age and of alcohol on cognitive and behavioral functions. However, their effects are not identical. Despite the a priori appeal of the notion that there is at least an additive, and possibly a supra-additive, interaction between alcohol and aging, this is not demonstrably true for all effects. The main problem is the relative lack of empirical evidence derived from studies designed specifically to test for such interactions. There is much evidence concerning the effects of age and of alcohol *separately* on a wide variety of cognitive and behavioral functions, but not nearly enough systematic study of their combined action to permit sweeping general conclusions. There is clearly a need for much additional research specifically targeted toward this topic.

NOTE

The views expressed in this paper are those of the author, and do not necessarily represent the policies of the Addiction Research Foundation of Ontario.

REFERENCES

Abel, E.L., and York, J.L. Age-related differences in response to ethanol in the rat. *Physiol Psychol* 7:391–395, 1979.

Adams, W.L.; Garry, P.J.; Rhyne, R.; Hunt, W.C.; and Goodwin, J.S. Alcohol intake in the healthy elderly. Changes with age in a cross-sectional and longitudinal study. *J Am Geriatr Soc* 38:211–216, 1990.

Ades, J., and Lejoyeux, M. Conduites d'addiction du sujet âgé [Addictive behaviors in the elderly]. *Rev Praticien* 44:1439–1442, 1994.

Blokland, A.; Prickaerts, J.; and Raaijmakers, W. Absence of impairments in spatial and temporal discrimination learning in Lewis rats after chronic ethanol consumption. *Pharmacol Biochem Behav* 46:27–34, 1993.

Borges, M.M.; Paula-Barbosa, M.M.; and Volk, B. Chronic alcohol consumption induces lipofuscin deposition in the rat hippocampus. *Neurobiol Aging* 7:347–355, 1986.

Brenton, R.S., and Phelps, C.D. The normal visual field on the Humphrey field analyzer. *Ophthalmologica* 193:56–74, 1986.

Casson, E.J.; Johnson, C.A.; and Nelson-Quigg, J.M. Temporal modulation perimetry: The effects of aging and eccentricity on sensitivity in normals. *Invest Ophthalmol Vis Sci* 34:3096–3102, 1993.

Chermack, S.T.; Blow, F.C.; Hill, E.M.; and Mudd, S.A. The relationship between alcohol symptoms and consumption among older drinkers. *Alcohol Clin Exp Res* 20:1153–1158, 1996.

Christian, J.C.; Reed, T.; Carmelli, D.; Page, W.F.; Norton, J.A.; and Breitner, J.C.S. Self-reported alcohol intake and cognition in aging twins. *J Stud Alcohol* 56:414–416, 1995.

Collins, W.E., and Mertens, H.W. Age, alcohol, and simulated altitude: Effects on performance and Breathalyzer scores. *Aviat Space Environ Med* 59:1026–1033, 1988.

Cook, P.J.; Flanagan, R.; and James, I.M. Diazepam tolerance: Effect of age, regular sedation, and alcohol. *Br Med J Clin Res Ed* 289:351–353, 1984.

Cutting, J.C. Alcohol cognitive impairment and aging: Still an uncertain relationship. *Br J Addict* 83:995–997, 1988.

De Labry, L.O.; Glynn, R.J.; Levenson, M.R.; Hermos, J.A.; LoCastro, J.S.; and Vokonas, P.S. Alcohol consumption and mortality in an American male population: Recovering the U-shaped curve—findings from the Normative Aging Study. *J Stud Alcohol* 53:25–32, 1992.

Di Chiara, G. The role of dopamine in drug abuse viewed from the perspective of its role in motivation. *Drug Alcohol Depend* 38:95–137, 1995.

Di Chiara, G.; Acquas, E.; Tanda, G.; and Cadoni, C. Drugs of abuse: Biochemical surrogates of specific aspects of natural reward? *Biochem Soc Symp* 59:65–81, 1993.

Dufour, M.C.; Archer, L.; and Gordis, E. Alcohol and the elderly. *Clin Geriatr Med* 8:127–141, 1992.

Egbert, A.M. The older alcoholic: Recognizing the subtle clinical clues. *Geriatrics* 48:63–66, 1993.

Fehr, K. A.; Kalant, H.; and LeBlanc, A.E. Residual learning deficit after heavy exposure to cannabis or alcohol in rats. *Science* 193:1249–1251, 1976.

Giorgi, O.; Pibiri, M.G.; Dal Toso, R.; and Ragatzu, G. Age-related changes in the turnover rates of D_1-dopamine receptors in the retina and in distinct areas of the rat brain. *Brain Res* 569:323–329, 1992.

Gomberg, E.S.L. Drugs, alcohol, and aging. In: Kozlowski, L.T.; Annis, H.M.; Cappell, H.D.; Glaser, F.B.; Goodstadt, M.S.; Israel, Y.; Kalant, H.; Sellers, E.M.; and Vingilis, E.R., eds. *Research Advances in Alcohol and Drug Problems.* Vol. 10. New York: Plenum Press, 1990. pp.171–213.

Goodwin, D.W. Alcohol amnesia. *Addiction* 90:315–317, 1995.

Grillon, C.; Sinha, R.; and O'Malley, S.S. Effects of ethanol on the processing of low-probability stimuli: An ERP study. *Psychopharmacology* 119:455–465, 1995.

Gustafson, R. Visual attentional span as a function of a small dose of alcohol. *Percept Mot Skills* 63:367–370, 1986.

Guthrie, S.; Cooper, R.L.; Thurman, R.; and Linnoila, M. Pharmacodynamics and pharmacokinetics of ethanol, diazepam and pentobarbital in young and aged rats. *Pharmacol Toxicol* 61:308–312, 1987.

Hashtroudi, S., and Parker, E.S. Acute alcohol amnesia: What is remembered and what is forgotten. In: Cappell, H.D.; Glaser, F.B.; Israel, Y.; Kalant, H.; Schmidt, W.; Sellers, E.M.; and Smart, R.S., eds. *Research Advances in Alcohol and Drug Problems.* Vol. 9. New York: Plenum Press, 1986. pp. 179–209.

Hervonen, A.; Jaatinen, P.; Sarviharju, M.; and Kiianmaa, K. Interaction of aging and lifelong ethanol ingestion on ethanol-related behaviors and longevity. *Exp Gerontol* 27:335–345, 1992.

Hill, J.C., and Toffolon, G. Effect of alcohol on sensory and sensorimotor visual functions. *J Stud Alcohol* 51:108–113, 1990.

Hoyer, W.J., and Rybash, J.M. Age and visual field differences in computing visual-spatial relations. *Psychol Aging* 7:339–342, 1992.

Huang, R.L.; Wang, C.T.; Tai, M.Y.; Tsai, Y.F.; and Peng, M.T. Effects of age on dopamine release in the nucleus accumbens and amphetamine-induced locomotor activity in rats. *Neurosci Lett* 200:61–64, 1995.

Hultsch, D.F.; Hammer, M.; and Small, B.J. Age differences in cognitive performance in later life: Relationships to self-reported health and activity life style. *J Gerontol* 48:1–11, 1993.

Jaaskelainen, I.P.; Pekkonen, E.; Alho, K.; Sinclair, J.D.; Sillanaukee, P.; and Naatanen, R. Dose-related effect of alcohol on mismatch negativity and reaction time performance. *Alcohol* 12:491–495, 1995.

Jennings, J.M., and Jacoby, L.L. Automatic versus intentional uses of memory: Aging, attention, and control. *Psychol Aging* 8:283–293, 1993.

Johnson, C.A., and Marshall, D., Jr. Aging effects for opponent mechanisms in the central visual field. *Optom Vis Sci* 72:75–82, 1995.

Jones, M.K., and Jones, B.M. The relationship of age and drinking habits to the effects of alcohol on memory in women. *J Stud Alcohol* 41:179–186, 1980.

Kalant, H. Current state of knowledge about the mechanisms of alcohol tolerance. *Addict Biol* 1:133–141, 1996*a*.

Kalant, H. Pharmacokinetics of ethanol: Absorption, distribution, and elimination. In: Begleiter, H., and Kissin, B., eds. *The Pharmacology of Alcohol and Alcohol Dependence.* New York: Oxford University Press, 1996*b*. pp. 15–58.

Kerr, J.S.; Fairweather, D.B.; Mahendran, R.; and Hindmarch, I. The effects of paroxetine, alone and in combination with alcohol, on psychomotor performance and cognitive function in the elderly. *Int Clin Psychopharmacol* 7:101–108, 1992.

Khanna, J.M.; Kalant, H.; Lê, A.D.; and LeBlanc, A.E. Reversal of tolerance to ethanol—a possible consequence of ethanol brain damage. *Acta Psychiatr Scand* 62 (Suppl 286):129–134, 1980.

Kline, D.W. Optimizing the visibility of displays for older observers. *Exp Aging Res* 20:11–23, 1994.

Koelega, H.S. Alcohol and vigilance performance: A review. *Psychopharmacology (Berl)* 118:233–249, 1995.

Koepsell, T.D.; Wolf, M.E.; McCloskey, L.; Buchner, D.M.; Louie, D.; Wagner, E.H.; and Thompson, R.S. Medical conditions and motor vehicle collision injuries in older adults. *J Am Geriatr Soc* 42:695–700, 1994.

Koob, G.F.; Rassnick, S.; Heinrichs, S.; and Weiss, F. Alcohol, the reward system and dependence. *EXS* 71:103–114, 1994.

Krueger, H. Comparison of the effect of flurazepam, brotizolam and alcohol on psychomotor performance. *Arzneimittel-Forschung* 36:616–620, 1986.

Krull, K.R.; Smith, L.T.; and Parsons, O.A. Simple reaction time event-related potentials: Effects of alcohol and diazepam. *Prog Neuropsychopharmacol Biol Psychiatry* 18:1247–1260, 1994.

Kutas, M.; Iragui, V.; and Hillyard, S.A. Effects of aging on event-related brain potentials (ERPs) in a visual detection task. *Electroencephalogr Clin Neurophysiol* 92:126–139, 1994.

Lachenmayr, B.J.; Kojetinsky, S.; Ostermaier, N.; Angstwurm, K.; Vivell, P.M.; and Schaumberger, M. The different effects of aging on normal sensitivity in flicker and light-sense perimetry. *Invest Ophthalmol Vis Sci* 35:2741–2748, 1994.

LeBlanc, E.A.; Kalant, H.; and Gibbins, R.J. Acute tolerance to ethanol in the rat. *Psychopharmacologia (Berl)* 41:43–46, 1975.

Lee, J.M.; Ross, E.R.; Gower, A.; Paris, J.M.; Martensson, R.; and Lorens, S.A. Spatial learning deficits in the aged rat: Neuroanatomical and neurochemical correlates. *Brain Res Bull* 33:489–500, 1994.

Linnoila, M.; Erwin, C.W.; Ramm, D.; and Cleveland, W.P. Effects of age and alcohol on psychomotor performance of men. *J Stud Alcohol* 41:488–495, 1980.

Mello, N.K. Behavioral studies of alcoholism. In: Kissin, B., and Begleiter, H., eds. *The Biology of Alcoholism, Vol. 2: Physiology and Behavior*. New York: Plenum Press, 1972. pp. 219–291.

Morelli, M.; Mennini, T.; Cagnotto, A.; Toffano, G.; and Di Chiara, G. Quantitative autoradiographical analysis of the age-related modulation of central dopamine D_1 and D_2 receptors. *Neuroscience* 36:403–410, 1990.

Morrow, D.; Leirer, V.; and Yesavage, J. The influence of alcohol and aging on radio communication during flight. *Aviat Space Environ Med* 61:12–20, 1990.

Morrow, D.; Yesavage, J.; Leirer, V.; Dolhert, N.; Taylor, J.; and Tinklenberg, J. The time-course of alcohol impairment of general aviation pilot performance in a Frasca 141 simulator. *Aviat Space Environ Med* 64:697–705, 1993.

Moskowitz, H., and Burns, M. Effect of alcohol on the psychological refractory period. *Q J Stud Alcohol* 32:782–790, 1971.

Murata, K.; Araki, S.; Tanigawa, T.; and Uchida, E. Acute effects of alcohol on cognitive function and central nervous system assessed by auditory event-related potentials. *Nippon Eiseigaku Zasshi* 47:958–964, 1992.

Overton, D.A. State-dependent learning produced by alcohol and its relevance to alcoholism. In: Kissin, B., and Begleiter, H., eds. *The Biology of Alcoholism, Vol. 2: Physiology and Behavior*. New York: Plenum Press, 1972. pp. 193–217.

Owsley, C., and Ball, K. Assessing visual function in the older driver. *Clin Geriatr Med* 9(2):389–401, 1993.

Page, R.D., and Cleveland, M.F. Cognitive dysfunction and aging among male alcoholics and social drinkers. *Alcohol Clin Exp Res* 11:376–384, 1987.

Pentney, R.J. Remodeling of neuronal dendritic networks with aging and alcohol. *Alcohol Alcohol Suppl* 1:393–397, 1991.

Pfefferbaum, A.; Ford, J.M.; Wenegrat, B.G.; Roth, W.T.; and Kopell, B.S. Clinical application of the P3 component of event-related potentials. I. Normal

aging. *Electroencephalogr Clin Neurophysiol* 59:85–103, 1984.

Pfefferbaum, A.; Sullivan, E.V.; Rosenbloom, M.J.; Shear, P.K.; Mathalon, D.H.; and Lim, K.O. Increase in brain cerebrospinal fluid volume is greater in older than in younger alcoholic patients: A replication study and CT/MRI comparison. *Psychiatry Res* 50:257–274, 1993.

Poikolainen, K. Estimated lethal ethanol concentrations in relation to age, aspiration, and drugs. *Alcohol Clin Exp Res* 8:223–225, 1984.

Porjesz, B., and Begleiter, H. Evoked brain potential deficits in alcoholism and aging. *Alcohol Clin Exp Res* 6:53–63, 1982.

Portans, I.; White, J.M.; and Staiger, P.K. Acute tolerance to alcohol: Changes in subjective effects among social drinkers. *Psychopharmacology* 97:365–369, 1989.

Ray, W.A.; Fought, R.L.; and Decker, M.D. Psychoactive drugs and the risk of injurious motor vehicle crashes in elderly drivers. *Am J Epidemiol* 136:873–883, 1992.

Riley, J.N., and Walker, D.W. Morphological alterations in hippocampus after long-term alcohol consumption in mice. *Science* 201:646–648, 1978.

Roehrs, T.; Beare, D.; Zorick, F.; and Roth, T. Sleepiness and ethanol effects on simulated driving. *Alcohol Clin Exp Res* 18:154–158, 1994.

Rohrbaugh, J.W.; Stapleton, J.M.; Parasuraman, R.; Zubovic, E.A.; Frowein, H.W.; Varner, J.L.; Adinoff, B.; Lane, E.A.; Eckardt, M.J.; and Linnoila, M. Dose-related effects of ethanol on visual sustained attention and event-related potentials. *Alcohol* 4:293–300, 1987.

Samorajski, T.; Strong, J.R.; Volpendesta, D.; Miller-Soule, D.; and Hsu, L. The effects of aging, ethanol, and dihydroergotoxine mesylate (hydergine) alone and in combination on behavior, brain neurotransmitter, and receptor systems. In: Wood, W.G., and Elias, M.F., eds. *Alcoholism and Aging: Advances in Research.* Boca Raton: CRC Press, 1982. pp. 115–129.

Scott, R.B. Alcohol effects in the elderly. *Compr Ther* 15:8–12, 1989.

Simpson, H. Polydrug effects and traffic safety. In: Moskowitz, H., ed. *Alcohol, Drugs, and Driving: Abstracts and Reviews.* Vol. 1, Nos. 1–2, p. 23, 1985.

Spirduso, W.W.; Mayfield, D.; Grant, M.; and Schallert, T. Effects of route of administration of ethanol on high-speed reaction time in young and old rats. *Psychopharmacology* 97:413–417, 1989.

Stall, R. Change and stability in quantity and frequency of alcohol use among aging males: A 19-year follow-up study. *Br J Addict* 31:537–544, 1986.

Sullivan, E.V.; Marsh, L.; Mathalon, D.H.; Lim, K.O.; and Pfefferbaum, A. Anterior hippocampal volume deficits in nonamnesic, aging chronic alcoholics. *Alcohol Clin Exp Res* 19:110–122, 1995.

Summala, H., and Mikkola, T. Fatal accidents among car and truck drivers: Effects of fatigue, age, and alcohol consumption. *Hum Factors* 36:315–326, 1994.

Tajuddin, N.F., and Druse, M.J. Effects of chronic alcohol consumption and aging on dopamine D_2 receptors in Fischer 344 rats. *Alcohol Clin Exp Res* 20:144–151, 1996.

Tarter, R.E. Cognition, aging, and alcohol. In: Beresford, T., and Gomberg, E.,

eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 82–98.

Teo, R.K., and Ferguson, D.A. The acute effects of ethanol on auditory event-related potentials. *Psychopharmacology* 90:179–184, 1986.

Tupler, L.A.; Hege, S.; and Ellinwood, E.H., Jr. Alcohol pharmacodynamics in young-elderly adults contrasted with young and middle-aged subjects. *Psychopharmacology* 118:460–470, 1995.

Viano, D.C.; Culver, C.C.; Evans, L.; Frick, M.; and Scott, R. Involvement of older drivers in multivehicle side-impact crashes. *Accid Anal Prev* 22:177–188, 1990.

Vogel-Sprott, M. Response measures of social drinking. Research implications and applications. *J Stud Alcohol* 44:817–836, 1983.

Vogel-Sprott, M., and Barrett, P. Age, drinking habits and the effects of alcohol. *J Stud Alcohol* 45:517–521, 1984.

Wahlin, T.-B.R.; Bäckman, L.; Wahlin, Å.; and Winblad, B. Trail Making Test performance in a community-based sample of healthy very old adults: Effects of age on completion time, but not on accuracy. *Arch Gerontol Geriatr* 22:87–102, 1996.

Walker, D.W., and Freund, G. Impairment of shuttle box avoidance learning following prolonged alcohol consumption in rats. *Physiol Behav* 7:773–778, 1971.

Wall, T.L., and Ehlers, C.L. Acute effects of alcohol on P300 in Asians with different ALDH2 genotypes. *Alcohol Clin Exp Res* 19:617–622, 1995.

Waller, J.A. Cardiovascular disease, aging, and traffic accidents. *J Chronic Dis* 20:615–620, 1967.

Wallgren, H., and Barry, H., III. *Actions of Alcohol.* New York: Elsevier, 1970. pp. 287–298.

Watanabe, H. Differential decrease in the rate of dopamine synthesis in several dopaminergic neurons of aged rat brain. *Exp Gerontol* 22:17–25, 1987.

Watson, P.E.; Watson, I.D.; and Batt, R.D. Total body water volumes for adult males and females estimated from simple anthropometric measurements. *Am J Clin Nutr* 33:27–39, 1980.

Welte, J.W., and Mirand, A.L. Drinking, problem drinking and life stressors in the elderly general population. *J Stud Alcohol* 56:67–73, 1995.

Wild, J.M.; Betts, T.A.; and Shaw, D.E. The influence of a social dose of alcohol on the central visual field. *Jpn J Ophthalmol* 34:291–297, 1990.

Wilkinson, C.J. The acute effects of zolpidem, administered alone and with alcohol, on cognitive and psychomotor function. *J Clin Psychiatry* 56:309–318, 1995.

Williams, A.F., and Carsten, O. Driver age and crash involvement. *Am J Public Health* 79:326–327, 1989.

Woods, J.M.; Ricken, J.D.; and Druse, M.J. Effects of chronic alcohol consumption and aging on dopamine D_1 receptors in Fischer 344 rats. *Alcohol Clin Exp Res* 19:1331–1337, 1995.

York, J.L. Body water content, ethanol pharmacokinetics, and the responsiveness to ethanol in young and old rats. *Dev Pharmacol Ther* 4:106–116, 1982.

York, J.L. Increased responsiveness to ethanol with advancing age in rats. *Pharmacol Biochem Behav* 19:687–691, 1983.

York, J.L. Progression of alcohol consumption across the drinking career in alcoholics and social drinkers. *J Stud Alcohol* 56:328-336, 1995.

York, J.L., and Chan, A.W.K. Absence of acute tolerance to ethanol hypnosis in F-344 and BN/BIRIJ rats. *Alcohol* 11:31–34, 1994.

Ziolkowski, M.; Maludzinska, E.; Gruss, T.; Rybakowski, J.; and Volpicelli, J.R. Decrease in alcohol tolerance: Clinical significance in alcohol dependence. *Drug Alcohol Depend* 39:33–36, 1995.

Zylman, R. Youth, alcohol, and collision involvement. *J Safety Res* 5:58–72, 1973.

Chapter 7

Neuropathological Studies: The Relationship Between Alcohol and Aging

Clive Harper, M.D., Donna Sheedy, Glenda Halliday, Ph.D., Kay Double, Ph.D., Peter Dodd, Ph.D., Joanne Lewohl, and Jillian Kril, Ph.D.

To determine whether alcohol can affect the aging of the brain, one must first establish which pathological changes in the brain are truly age related and which are alcohol related. One of the critical limiting factors in this regard is that it is extremely difficult to obtain material with adequate clinical information for such studies. Most diseases become more common as a population ages, and strict criteria need to be applied in the selection of material for both aging and alcohol studies. In fact, apart from the occasional study that has addressed the question of aging and alcohol (Freund 1982; Wiggins et al. 1988), there are no neuropathological studies of age-related changes that have considered alcohol as a covariate or, more importantly, used excessive alcohol intake as an exclusion criterion. That is, most neuropathological studies of "normal" populations do not consider alcohol as a relevant pathogenetic factor of brain damage. Similarly, in both alcohol and aging studies, many other disorders such as Alzheimer's disease (AD) and cerebrovascular disease are common and can be easily overlooked, especially if there is inadequate clinical

C. Harper, M.D., is professor of neuropathology in the Departments of Pathology at the University of Sydney and the Royal Prince Alfred Hospital, Camperdown, NSW, Australia 2006. D. Sheedy is a research officer in the Department of Pathology, University of Sydney, Camperdown, NSW, Australia 2006. G. Halliday, Ph.D., is a National Health and Medical Research Council senior research fellow and K. Double, Ph.D., is a research officer at The Prince of Wales Medical Research Institute, Randwick, NSW, Australia 2031. P. Dodd, Ph.D., is a National Health and Medical Research Council research fellow and J. Lewohl is a research assistant at the Royal Brisbane Hospital Foundation, Bancroft Centre, Brisbane, Queensland, Australia 4029. J. Kril, PhD., is senior scientific officer in neuropathology in the Departments of Pathology at the University of Sydney and the Royal Prince Alfred Hospital, Camperdown, NSW, Australia 2006.

information and if adequate neuropathological screening is not carried out; for example, silver impregnation techniques or immunoperoxidase stains for beta A4 protein are the minimum requirements for excluding a neuropathological diagnosis of AD. In fact, because "aging" changes (neuritic plaques and neurofibrillary tangles) are similar to those seen in AD, it is a general policy of most neuropathology research groups to carry out quantitative or semiquantitative analyses on relevant cortical regions in order to be more confident about a diagnosis of AD.

The aim of this study was to collate all our research data on quantitative aspects of the neuropathology of aging and alcohol brain damage in order to determine if there are similar patterns of change. This approach was aptly described by Freund and Butters (1982, p. 1) in their study of aging and alcohol: "It is necessary to descriptively compare alterations occurring with ageing and alcohol consumption at all levels of biological organization." This technique has been employed by clinical psychologists and neuroradiologists in trying to determine whether or not there is an interrelationship between alcohol and aging, and the results still leave the question unresolved (Freund 1982). Parameters that were considered include brain weight and volume, pericerebral space volume, cerebral hemisphere and cerebellar volumes, cerebral cortical gray and white matter volumes, and basal ganglia volumes. At the microscopic level, there are data on neuronal counts and densities and dendritic arborization. Studies of the chemistry of the brain were also considered.

MATERIALS AND METHODS

There are many neuropathological changes seen in the brains of heavy drinkers and alcoholic subjects that develop as a result of other medical complications. The most notable examples are thiamin (vitamin B_1) deficiency disorders and cirrhosis of the liver. All control and alcoholic cases in this study were screened for these disorders, and cases with these disorders were excluded from the analyses. In addition, all cases were extensively screened to eliminate neurological abnormalities such as strokes, AD, and severe head injuries. Thus, any changes identified in the alcoholic group can reasonably be considered to be "alcoholic specific." Cases for study were categorized as follows:

- *Controls*—less than 20 g of alcohol per day (most cases had nil intake);
- *Moderate drinkers*—30–80 g/d (most drank > 50 g/d); and
- *Uncomplicated alcoholics*—greater than 80 g/d (most drank > 120 g/d, had a 20- to 30-year drinking history, and had no other medical complications such as cirrhosis or Wernicke-Korsakoff syndrome (WKS).

The definition of moderate drinkers will probably conflict with many other authors' ideas. Parker and Noble (1980) defined "moderate" as much lower levels of alcohol intake. Nevertheless, at least in Australia, a significant

proportion of professional and skilled individuals fit this definition and are not the archetypal "alcoholic" subject (Tuck and Jackson 1991).

Regression analyses and analysis of variance (ANOVA) statistics were calculated separately for the two drinking groups (moderate drinkers and uncomplicated alcoholics). These statistics were found to be similar, thus all regression analyses for these groups are presented as combined data for what we have termed the "alcoholic" group.

In these studies the mean age of the drinking groups was in the sixth decade and their drinking histories were in excess of 25 years. The mean age of the control group was also in the sixth decade. Cases were only included in the control group if there was adequate clinical information on the premorbid mental state of the patient and a suitably negative alcohol history had been obtained.

RESULTS AND DISCUSSION

Although there is usually some variation in the weight and volume of brains before and after fixation in neutral buffered formalin, the differences were found to be minimal and all data were therefore standardized by using fresh weights and volumes. Similarly, the question of the effect of postmortem delay on the various parameters was addressed and was not found to have a significant effect, and regression analyses showed no correlation with age. No consistent differences were found between the right and left cerebral hemispheres (Harper et al. 1985), and in some cases one hemisphere was frozen and used for neurochemical studies. In these cases the cerebral hemisphere volume was calculated by doubling the volume of one hemisphere (calculated by fluid displacement). Previous studies have suggested that the right and left hemispheres are differentially susceptible to the effects of alcohol (Golden et al. 1981).

There were consistent differences found between male and female cohorts in the control and alcoholic groups—the changes in the two study groups were similar and most have been previously documented (Harper and Blumbergs 1982; Harper et al. 1985; Harper and Kril 1991; Double et al. 1996). For all variables that could not be normalized or calculated as a percentage of brain weight or volume (to negate height, weight, and gender effects), only male data were used because there were far more male than female cases in the studies.

Brain Weight and Volume

One of the features common to both aging and the long-term effects of alcohol, which probably gave rise to the hypothesis of a common pathogenesis, is brain shrinkage. These changes were first documented by neuroanatomists and later by neuroradiologists. There has been a dramatic increase in the amount of neuroimaging literature as the resolution of computed tomography (CT) and magnetic resonance imaging (MRI) scans has improved, and the anatomy of the brain can now be visualized almost as well in vivo as by the neuropathologist.

The MRI data are particularly interesting in that the age-related volume

Table 1. Fresh Brain Weight.

Group	*n*	Weight(g)	SEM
Control	56	1,433	17
Moderate	16	1,415	34
Alcoholic	38	1,352*	27

Note: SEM = standard error of the mean.
*$p < 0.01$.

loss of brain tissue in the alcoholic group is over and above that expected in normal aging (Pfefferbaum et al. 1992; Shear et al. 1994). The changes were particularly evident in the temporal cortex and the white matter (see section on white matter later in this chapter). These researchers also noted that the anterior (but not the posterior) portions of the hippocampus were smaller in alcoholics than in controls and the reduction in volume was greater in older than in younger alcoholics (Sullivan et al. 1995). More recently the same research group reviewed these data to examine whether or not seizure activity plays a role in the hippocampal and white matter shrinkage. They showed that the group of alcoholics who had suffered withdrawal seizures had more severe loss of tissue from both hippocampus and white matter. However, the nonseizure group still had a significant reduction in the volume of the frontoparietal white matter (Sullivan et al. 1996).

Pathological confirmation of brain shrinkage in alcoholics came with studies of brain weights (Harper and Blumbergs 1982; Torvik et al. 1982; Lindboe and Loberg 1988). The mean reductions in brain weight in these studies ranged from 31 to 71 g, but each group included a significant number of alcoholic cases with complications such as WKS and cirrhosis. The Scandinavian research groups looked at their data in relation to age and showed that there was no difference in brain weight between controls and alcoholics after 70 years of age (Torvik et al. 1982; Lindboe and Loberg 1988). As shown in table 1, there appears to be a graduated effect on brain weight in the two drinking groups studied.

The relationship between age and fresh brain weight for the alcoholic and control groups was not statistically significant, but the trends for the two groups appear to be similar in that there appears to be a slight reduction in brain weight with age.

Brain volume studies showed similar patterns of change in controls and alcoholics, but regression analyses with age did not show any significant correlation.

Pericerebral Space

Brain weights and volumes exhibit a wide range of variation, even in the healthy nonalcoholic population, and a more reliable parameter to detect brain shrinkage is measurement of the pericerebral space (PICS). The usefulness of the PICS measurement relates to the fact that during childhood the

growth of the brain determines the growth of the skull (Dekaban and Sadowsky 1978) and the intracranial cavity volume (ICV) then remains unchanged during adult life (Davis and Wright 1977). This measure effectively excludes individual variation based on gender differences and on physical parameters such as height and weight. Even secular change (progressive trend toward increasing mean body height and weight during the 20th century) can be negated (Miller and Corsellis 1977). Any reduction in brain volume will cause an increase in the PICS value, which can be calculated from the following equation:

$$\text{PICS} = \frac{\text{intracranial volume} - \text{brain volume}}{\text{intracranial volume}} \times \frac{100\%}{1}$$

There are very few studies of this parameter in either normal populations or pathological states because of the logistic and mechanical difficulties of obtaining these measurements during routine necropsies. Harper and his colleagues (1984) described a relatively simple technique for measuring intracranial volume based on the production of a permanent polyurethane cast of the cranial cavity.

Table 2 gives the mean PICS values and standard errors for the controls, moderate drinkers, and uncomplicated alcoholics. As shown in previous studies (Harper et al. 1985, 1988*b*) there is a statistically significant difference between controls and uncomplicated alcoholics, and even in the moderate drinkers there is a trend suggesting that an intake of less than 80 g of alcohol per day may cause loss of brain tissue.

Regression analyses show that there is a strong correlation between brain shrinkage and age in both control and alcoholic groups (figure 1). Moreover, the older alcoholic cases have much higher PICS values than controls, suggesting that there may be a cumulative effect of age and alcohol.

Volume Changes Within the Cerebral Hemispheres

Although macroscopic examination of the cerebral hemispheres of both the aging and alcoholic populations has been generally unrewarding, Courville (1966) and others have commented on the apparent regional nature of the cortical atrophy in that the frontal lobes seem to be more severely affected. Imaging studies of alcoholics have also shown that the frontal lobes are more shrunken than other brain regions (Jernigan et al. 1991*a*, 1991*b*). One

Table 2. PICS Values in Control and Drinking Groups.

Group	*n*	PICS(%)	SEM
Control	51	7.8	0.5
Moderate	13	8.8	1.2
Alcoholic	32	11.3*	1.0

Note: PICS = pericerebral space; SEM = standard error of the mean.
*$p < 0.001$.

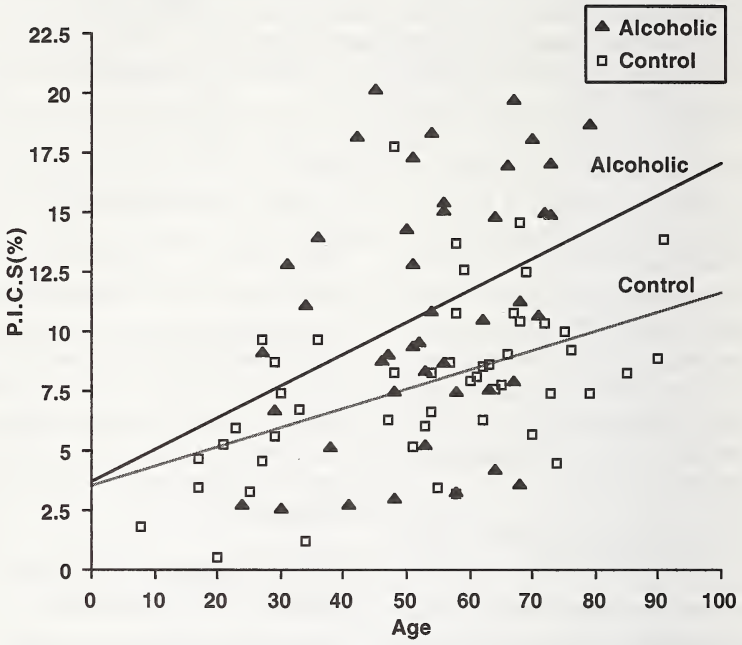

Figure 1. Relationship between brain shrinkage and age in alcoholic and control groups. For the alcoholic group, pericerebral space (P.I.C.S) = 3.715 + 0.134 × age; $R^2 = 0.127$. For the control group, P.I.C.S = 3.529 + 0.081 × age; $R^2 = 0.242$.

reason why these frontal lobe changes may be more evident is that there is a greater proportion of white matter compared with cortical gray matter in frontal regions. The ratio of gray matter to white matter is 1.22 in the frontal region and 1.40 in occipital lobes (Harper et al. 1985). Frontal atrophy has also been commented upon in aging and AD. However, quantitative studies have shown that there is a different pattern of atrophy in aging and AD, the frontal cortex being more severely affected in AD whereas the frontal white matter tends to bear the brunt of the atrophy in aging (Double et al. 1996). This is similar to the documented white matter changes in alcoholics, discussed later in this chapter.

Regression analyses of cerebral cortical volume in control and alcoholic groups with respect to age reflected the brain weight and volume data, but the differences were not statistically significant. Measurements of the thickness of the cortical mantle also showed no difference in the control and alcoholic groups, whereas the cortex is significantly thinned in AD (Terry et al. 1981).

Gray Matter

Cerebral cortical gray matter can be assessed during life with the aid of

MRI and by pathological studies after death. There are a number of human studies of both control and alcoholic groups (see table 3), and these show no significant loss of gray matter. There are also a number of studies of normal aging animals and animal models of alcohol toxicity. The best of the animal studies highlight the relative lack of change of the cerebral cortex of primates (rhesus monkeys) with aging (Albert 1993; Peters et al. 1994, 1996). Several of the studies have made important correlations between the radiological and pathological changes in relation to cognitive function (Albert 1993; Leuchter et al. 1994; Wickelgren 1996). The radiological studies initially gave conflicting results, but it is now generally agreed that aging is not associated with a significant loss of cortical gray matter (Coffey et al. 1992; Wickelgren 1996). Although brain atrophy is a common finding in elderly patients, not all subjects show atrophy, suggesting that it is not an inevitable consequence of aging. One confounding issue is that we may be selecting a "successfully aging population" for our studies by choosing non–cognitively impaired subjects who may not be truly representative of the population.

Our regression analyses of cortical gray matter volume for control and alcoholic groups showed surprisingly little change with respect to age, a finding that has been reported previously in alcoholic groups (Harper et al. 1985, 1988*b*). Pathological studies support the hypothesis that cortical gray matter is relatively intact in "normal" (nonalcoholic) old people (Terry and Hansen 1987; Double et al. 1996). The findings of these studies conflict with traditional beliefs based on previous work done using techniques that are now considered inappropriate (Brody 1955). Many of these old studies describe a progressive decrease in neuronal density with age. There is neuronal loss in certain regions of the brain with age—the basal forebrain (Halliday et al. 1993) and locus coeruleus (Halliday and Baker 1996), for example—but neurons in the cerebral cortex appear to be preserved (Wickelgren 1996). There are a number of other regions previously thought to have neuronal loss with age, but more careful studies using appropriate techniques (e.g., optical dissector technique [Kril et al.

Table 3. Percentage Gray Matter and White Matter in Control and Drinking Groups.

		Gray Matter		White Matter	
Group	***n***	**Volume (%)**	**SEM**	**Volume (%)**	**SEM**
Control	26	53.8	0.6	40.5	0.5
Moderate	13	55.3	0.7	39.4	0.7
Alcoholic	34	54.3	0.5	39.1*	0.4

Note: SEM = standard error of the mean.
*$p < 0.01$.

1997]) show no loss; a good example is the substantia nigra (Halliday et al. 1996).

Carefully controlled studies of alcoholic cases also fail to show significant changes in cortical gray matter volume (see table 3) (Harper et al. 1985; Kril et al. 1997). However, there is now good evidence to show that there are selective regions of the brain in alcoholics where neuronal loss is significant. The superior frontal association cortex has reduced neuronal density (Harper et al. 1987*a*) and reduced total neuronal counts (Kril et al. 1997). Neuronal loss in alcoholics has also been documented in a number of subcortical regions (Baker et al. 1994; Halliday et al. 1994; Cullen and Halliday 1995*a*; Baker et al. 1996*a*, 1996*b*; Harding et al. 1996); table 4 shows a comparison of patterns of neuronal loss in aging and alcoholism. An important point to note is that neuronal losses in the thalamus, basal forebrain, and raphe nuclei tend to be much more severe in those alcoholics with thiamin deficiency (WKS) (Halliday et al. 1994; Cullen and Halliday 1995*a*). It is interesting to note that the correlation between neuronal loss and alcohol consumption is dose related in the supraoptic and paraventricular nuclei of the hypothalamus (Harding et al. 1996). Changes are also well described in the cerebellum, but these will not be discussed in this chapter.

Not all authors agree that alcohol causes cortical neuronal loss. Jensen and Pakkenberg (1993), using unbiased techniques and adhering strictly to stereological principles, found no neuronal loss in their study of alcohol toxicity. However, as Kril and her colleagues have shown, there is a substantial variation in the number of neurons from case to case within cortical regions, and the large variations in neuronal number could mask the small selective loss observed (in frontal association cortex) when regions are averaged (Kril et al. 1997).

A number of other findings have been made in experimental models that suggest links between aging and alcohol toxicity. Lipofuscin is generally considered to be a marker of aging in central nervous system (CNS) neurons (Mrak et al. 1997). Several research groups have found an increase in intracellular lipo-

Table 4. Neuronal Loss in Aging and Alcoholism.

Brain Region	Aging	Alcohol
Cerebral cortex	-	+
Basal ganglia	-	-
Thalamus	-	+*
Substantia nigra	-	-
Basal forebrain	+	+*
Hippocampus	-	-
Locus coeruleus	+	-
Raphe nuclei (dorsal)	-	+*

Note: Plus sign indicates loss; dash indicates no loss. Asterisk indicates neuronal loss mainly in cases with Wernicke-Korsakoff syndrome.

fuscin deposition in the hippocampal and cerebellar neurons of rats given alcohol chronically (Tavares and Paula-Barbosa 1983; Borges et al. 1986).

Given that neuritic plaques and neurofibrillary tangles are a common finding in the brains of aging subjects and that they are two of the pathological hallmarks of AD, it is reasonable to propose that, if alcohol induces premature aging, the brains of alcoholics should have greater numbers of these pathological markers than comparable controls. We have examined this question using quantitative techniques and found no significant difference between controls and alcoholics of the same age (unpublished data). However, Cullen and her colleagues found that the magnocellular neurons of the basal nucleus of Meynert are particularly susceptible to form neurofibrillary tangles in alcoholics who also have WKS, but this was not seen in uncomplicated alcoholics (Cullen and Halliday 1995*b*).

Dendritic arborization of layer III pyramidal neurons from the frontal and motor cortices has been studied in controls and alcoholics. A significant relationship was found between age and total dendritic length, number of branches, and maximal width of dendritic field in both control and alcoholic groups (Harper and Corbett 1990). The dendritic arborization was more severely affected in the alcoholics than in controls. The alcoholic group included some with other medical complications such as WKS and cirrhosis.

White Matter

Although, over a number of years, authors have noted a reduction in the volume of the white matter with aging (Haug and Eggers 1991), this has been largely overlooked. This is partly because both macroscopic and microscopic changes in the white matter of healthy elderly patients are minimal, or at least very subtle. However, it has become evident from recent neuropathological studies that white matter loss is a feature of aging and appears to account for the majority of the cerebral tissue loss (Double et al. 1996). There is also good evidence from MRI studies that one of the main changes in the cerebral hemispheres with aging is a reduction in the volume of the white matter.

A similar finding was noted in studies of alcohol-related brain damage. There is a significant reduction in the volume of the white matter of the cerebral hemispheres (Harper et al. 1985; de la Monte 1988; Kril et al. 1997). As with most of the alcohol-related brain volume changes discussed herein, the loss is most severe in those alcoholics who have additional complications such as WKS and cirrhosis of the liver. Nevertheless, a recent study by Kril and her colleagues (1997) confirmed that alcoholics have reduced amounts of cerebral white matter and there is a significant age effect for both groups (those with and those without complications). The white matter of the cerebellar vermis is also reduced in volume in alcoholics when compared with controls (S.C. Phillips et al. 1990).

The results of regression analyses showing the relationship between cerebral white matter volume and age for the control and alcoholic groups

are shown in figure 2. The pattern is similar to previous studies of alcohol-related brain damage (Harper et al. 1985). There were relatively few alcoholics in the older age groups, which may account for the apparent convergence of the lines in the latter decades. In the previous study (Harper et al. 1985), both controls and alcoholics showed significant decreases in the volume of the white matter with age (controls: slope = −0.08, p = 0.005; alcoholics: slope = −0.11, p = 0.04). However, it should be noted that the alcoholic group included uncomplicated cases and those with cirrhosis and WKS. An important observation was made recently by Kril and her colleagues (1997). They noted that the loss of white matter correlates negatively with alcohol consumption.

Microscopic Changes

Studies using special stains for myelin in controls have been said to show pale staining of the white matter in older cases compared with young cases (Kemper 1994). This has not been quantitated, and the change is not easy to identify unless comparable sections of brains of different ages are available for study. We have recently reviewed myelin-stained sections (Loyez technique) of frontal, temporal, parietal, and occipital cortex in 20 controls and 20 uncomplicated alcoholics. There were significant variations in the pattern of myelinated fibers at

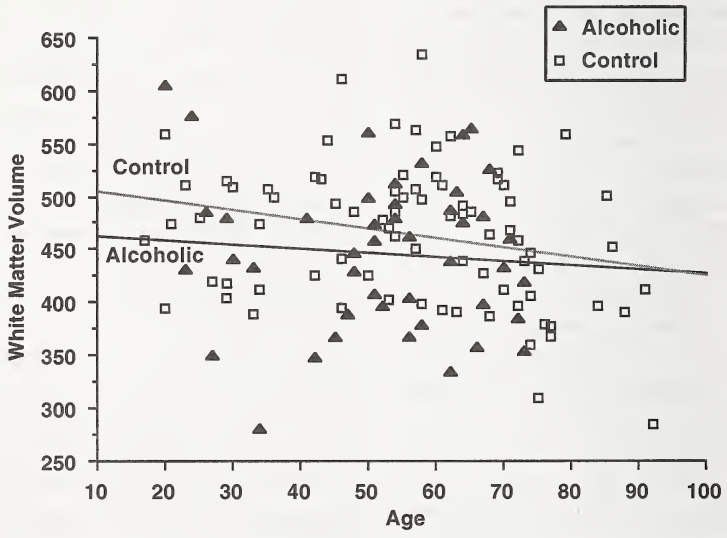

Figure 2. Relationship between cerebral white matter volume and age in alcoholic and control groups. For the alcoholic group, white matter volume = 467.169 − 0.402 × age; R^2 = 7.031E − 3. For the control group, white matter volume = 513.708 − 0.87 × age; R^2 = 0.062.

the crowns of the gyri in different cortical regions (least fibers seen in the temporal cortex), but we were unable to identify consistent differences between the two groups of cases (unpublished data). In 1966 Courville studied the brains of alcoholic subjects and commented that there were "alterations in the structure and a decrease in the number of the nerve fibres" (p. 50). He stated that the most conspicuous changes were observed in myelin-stained sections and that the radiating fibers of the crowns of cerebral gyri were most affected. Other authors have reported diffuse pallor of white matter in myelin-stained preparations, particularly in cases of WKS (Cravioto and Silberman 1961). Alling and Bostrom (1980) showed similar changes in nine chronic alcoholic subjects without WKS, and further analyzed the mamillary body tissue chemically. They found significantly lower concentrations of phospholipids, cholesterol, and cerebroside, implying a loss of myelin in the alcoholic cases. Much of these data needs further evaluation; ideally, techniques such as those used in the rhesus monkey study by Peters and colleagues (1994) should be applied to the problem—a difficult task for a human study.

Studies of aging in rhesus monkeys have also shown a significant loss of white matter with age and a concomitant increase in the size of the ventricles (Peters et al. 1996). In the study of alcoholics by de la Monte (1988) there was a similar increase in the size of the ventricles and this volume was roughly equal to the amount of white matter loss.

A most important part of the aging study in rhesus monkeys was the identification of microscopic and electron microscopic changes in the white matter. Using 1-μm plastic embedded sections, Peters and colleagues noted degeneration of myelinated axons which was most obvious in the deeper layers of the cortex and in the subcortical white matter. In these cases it was shown that there was a good correlation between the extent of myelin degeneration and performance deficits of the monkeys (Peters et al. 1996).

In an animal model of alcohol toxicity/thiamin deficiency, Langlais and his colleagues have also shown degeneration in the white matter (Langlais and Zhang 1996, 1997) using a modified Nauta-Laidlaw method (Eager 1970).

Electron Microscopy

Electron microscopic studies can only be applied to animal models of aging and alcohol-related brain damage because of the poor preservation of myelin sheaths after death in humans. In the rhesus monkey studies of aging there were frequent profiles of large nerve fibers in which the myelin sheaths appeared to be empty or to contain debris. Splitting of the myelin was a common finding and many of the medium-sized nerve fibers had dark, presumably degenerating axons (Peters et al. 1994).

Ultrastructural studies of the effects of alcohol on the structure of developing CNS myelin in an experimental model have been carried out by S.C. Phillips and colleagues (1987, 1990). The alcohol appears to cause a reduction in the relative

thickness of myelin sheaths in rat optic nerve.

Neurochemical Changes

The subtle nature of the white matter changes is borne out by physical and chemical studies of the white matter of alcoholic subjects. The specific gravity of the frontal, parietal, and occipital white matter in alcoholics and age- and sex-matched controls has been measured. There was no significant difference in the specific gravity between the two groups (Harper et al. 1987*b*). In the same cases there was a slight increase in the water content of the white matter, but this only attained statistical significance in the frontal lobe of those alcoholics with WKS (Harper et al. 1988*a*, 1988*b*). Wiggins and his colleagues (1988) found a slight increase in the water content of the white matter with aging. The water gain from 30 to 90 years amounted to 50 mg/g brain. This gain was associated with a decrease in total protein and specifically a decrease in the myelin and myelin-like fractions. The loss of myelin membrane amounted to 43 mg/g brain. Wiggins and colleagues included alcoholics in their study and showed that heavy alcohol consumption was associated with an increase in total protein in the white matter but an acceleration of the age-related loss of myelin. Perhaps more careful selection of alcoholic groups for study would help to explain these data and give us a better understanding of the links between the neurochemical and structural changes in the white matter.

Studies of the lipid profiles of the white matter in the different alcoholic groups (those with WKS, those with cirrhosis, and those with neither) have shown only minor alterations (Lesch et al. 1972; Harper et al. 1991). Even more critical analyses of lipid profiles using high-performance liquid chromatography with evaporative light-scattering detection, wherein all major phospholipid classes were measured, showed no abnormality (Olsson et al. 1996). Nevertheless, minor changes could induce important functional abnormalities, and further structural studies are warranted. In studies of growth and development of the human brain, Dobbing and Sands (1973) measured the cholesterol, water, and DNA content of the brain at different ages. Their findings support the concept that the process of myelination may have two overlapping components: "maturation," consisting of thickening of the myelin lamellae around the axon, and a "growth" process, consisting of myelination concurrent with growth in axon length. They concluded that the former process will cause an increase in cholesterol per unit weight, since the lipid deposition occurs at the expense of water. Although it is generally considered that there is little morphological or biochemical change in myelin lamellae in the adult brain and it has been shown that the metabolic turnover of myelin is relatively low, it is conceivable that subtle changes could occur in various pathological states such as alcoholism. Minor changes in the thickness of the myelin lamellae, such as those seen in the experimental model of alcohol toxicity (D.E. Phillips 1989), would be difficult to identify in human material.

However, such changes would provide an explanation for the apparent reversibility of white matter loss in alcoholics who abstain from drinking for several months (Jacobson 1986; Carlen and Wilkinson 1987).

Cortical neuronal receptor binding sites and uptake activities mediating a number of neurotransmitters have been studied using autopsy material that had also been used in our quantitative morphometric analyses (Dodd et al. 1992, 1996). In the most recent studies Peter Dodd and Joanne Lewohl examined four different $GABA_A$ receptor genes. Quantitation using reverse transcriptase polymerase chain reaction showed no correlation with age in either controls or alcoholics. The slopes of the regression lines were similar in each case (unpublished data).

Reversibility of White Matter Loss

An important difference between the white matter changes in aging and the white matter changes in alcoholism is the fact that, at least in some cases, the loss of white matter in alcoholics appears to be reversible (Carlen and Wilkinson 1987; Shear et al. 1994). Shear and her colleagues used sequential MRIs to show that, after 3 months of abstinence, there was an increase in the volume of the white matter and a decrease in the volume of cerebrospinal fluid (Shear et al. 1994). Young patients with the shortest drinking histories seem to have the greatest chance for reversibility (Jacobson 1986), suggesting that there is probably both a reversible and an irreversible component of the white matter damage in alcoholics. The irreversible component may be similar to the change in aging and is likely to relate to neuronal loss and wallerian degeneration of myelinated axons (Kril et al. 1997). Kril and her colleagues have shown that the white matter loss correlates negatively with lifetime alcohol consumption (Kril et al. 1997). It should be remembered that both afferent and efferent cortical connections could be involved and that degeneration in subcortical structures such as basal forebrain nuclei, substantia nigra, raphe nuclei, and locus coeruleus almost certainly plays an important role in the white matter loss.

Clinical Implications

The most common clinical change in aging patients is a generalized slowing of cognitive processes (Boone et al. 1992; Ylikoski et al. 1993). This slowing may be the result of decreased nerve conduction through the central white matter but is difficult to evaluate until the pathological substrate for the change is identified. An MRI study showed a correlation between the magnitude of white matter loss and cognitive decline (Leuchter et al. 1994). Similar radiological and cognitive changes have been reported in alcoholics (Pfefferbaum et al. 1992).

SUMMARY

The question "does alcohol affect the aging process of the brain" has been addressed by comparing the neuropathological changes that have been identified by our research group and others in both aging and alcohol-related brain damage studies. A large amount of data was available for these studies,

and not all of it has been included in this chapter. One of the disappointing features of this review is the fact that many of the regression analyses failed to reveal statistical significance even though the slopes of the lines were fairly consistent between different parameters (e.g., brain weight, volume, and cerebral hemisphere volume). The reason for this may be the great variability in individual biological measures. Alternatively, as pointed out in an editorial in *Neurobiology of Aging*, in order to identify age-related changes a number of time points must be employed (Coleman et al. 1990). The same thing almost certainly applies to studies of the changes caused by a neurotoxin that appears to act slowly over a long period of time, such as alcohol or one of its metabolites. Moreover, it may well be that both factors (alcohol and aging) act in a nonlinear fashion so that more specialized statistical analyses should be applied to the problem.

Nevertheless, there are a number of conclusions that can be drawn from this review:

- There is loss of brain tissue with aging and alcoholism.
- The loss is greater in alcoholics (PICS data).
- The loss is mainly from the cerebral hemispheres.
- The tissue loss is mainly white matter.
- The loss of white matter is greater in alcoholics.
- There is a correlation between the loss of white matter and alcohol intake.
- There is a correlation between the loss of white matter and cognitive dysfunction in both aging and alcoholism.

Thus it seems that there is considerable evidence to support the hypothesis that there is a link between alcohol and aging. Alcohol may promote the aging effects on the brain, or aging and alcohol may act synergistically. The principal focus of the pathological changes seems to be the white matter of the cerebral hemispheres rather than the gray matter.

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REFERENCES

Albert, M. Neuropsychological and neurophysiological changes in healthy adult humans across the age range. *Neurobiol Aging* 14:623–625, 1993.

Alling, C., and Bostrom, K. Demyelination of the mammillary bodies in alcoholism. A combined morphological and biochemical study. *Acta Neuropathol (Berl)* 50:77–80, 1980.

Baker, K.G.; Halliday, G.M.; and Harper, C.G. Effects of chronic alcohol consumption on the human locus coeruleus. *Alcohol Clin Exp Res* 18:1491–1496, 1994.

Baker, K.; Halliday, G.M.; Kril, J.J.; and Harper, C.G. Chronic alcoholism in the absence of Wernicke-Korsakoff syndrome and cirrhosis does not result in the loss of serotonergic neurons in the median raphe nucleus. *Metab Brain Dis* 11:217–227, 1996*a*.

Baker, K.G.; Halliday, G.M.; Kril, J.J.; and Harper, C.G. Chronic alcoholics without Wernicke-Korsakoff syndrome or

cirrhosis do not lose serotonergic neurons in the dorsal raphe nucleus. *Alcohol Clin Exp Res* 20:61–64, 1996*b*.

Boone, K.B.; Miller, B.L.; Ledder, I.M.; Mehringer, C.; Hill-Gutierrez, E.; Goldberg, M.A.; and Berman, N.G. Neuropsychological correlates of white-matter lesions in healthy elderly subjects. *Arch Neurol* 49:549–554, 1992.

Borges, M.M.; Paula-Barbosa, M.M.; and Volk, B. Chronic alcohol consumption induces lipofuscin deposition in the rat hippocampus. *Neurobiol Aging* 7:347–355, 1986.

Brody, H. Organization of the cerebral cortex III. *J Comp Neurol* 102:511–556, 1955.

Carlen, P.L., and Wilkinson, D.A. Reversibility of alcohol-related brain damage: Clinical and experimental observations. *Acta Med Scand 222* (Suppl 717):19–26, 1987.

Coffey, C.E.; Wildinson, W.E.; Parashos, I.A.; Soady, S.A.R.; Sullivan, R.J.; Patterson, L.J.; Figiel, G.S.; Webb, M.C.; Spritzer, C.E.; and Djang W.T. Quantitative cerebral anatomy of the aging human brain: A cross-sectional study using magnetic resonance imaging. *Neurology* 42:527–536, 1992.

Coleman, P.; Finch, C.; and Joseph, J. The need for multiple time points in aging studies. *Neurobiol Aging* 11:1–2, 1990.

Courville, C.B. Alcoholic atrophy of the cerebral cortex. In: *Effects of Alcohol on the Nervous System of Man*. Los Angeles: San Lucas Press, 1966. pp. 47–52.

Cravioto, H., and Silberman, J. Wernicke's encephalopathy. A clinical and pathological study of 28 autopsied cases. *Arch Neurol* 4:510–519, 1961.

Cullen, K.M., and Halliday, G.M. Mechanisms of cell death in cholinergic basal forebrain neurons in chronic alcoholics. *Metab Brain Dis* 10:81–91, 1995*a*.

Cullen, K.M., and Halliday, G.M. Neurofibrillary tangles in chronic alcoholics. *Neuropathol Appl Neurobiol* 21:312–318, 1995*b*.

Davis, P.J.M., and Wright, E.A. A new method for measuring cranial cavity volume and its application to the assessment of cerebral atrophy at autopsy. *Neuropathol Appl Neurobiol* 3:341–358, 1977.

Dekaban, A.S., and Sadowsky, D. Changes in brain weights during the span of human life: Relation of brain weights to body heights and body weights. *Ann Neurol* 4:345–356, 1978.

de la Monte, S.M. Disproportionate atrophy of cerebral white matter in chronic alcoholics. *Arch Neurol* 45:990–992, 1988.

Dobbing, J., and Sands, J. Quantitative growth and development of the human brain. *Arch Dis Child* 48:757–767, 1973.

Dodd, P.; Thomas, G.; Harper, C.; and Kril, J. Amino acid neurotransmitter receptor changes in cerebral cortex in alcoholism: Effect of cirrhosis of the liver. *J Neurochem* 59:1506–1515, 1992.

Dodd, P.R.; Kril, J.J.; Thomas, G.J.; Watson, W.E.J.; Johnston, G.A.R.; and Harper, C.G. Receptor binding sites and uptake activities mediating GABA neurotransmission in chronic alcoholics with Wernicke encephalopathy. *Brain Res* 710:215–228, 1996.

Double, K.; Halliday, G.; Kril, J.J.; Harasty, J.; Cullen, K.; Brooks, W.S.; Creasey, H.; and Broe, G.A. Topography of brain atrophy during normal aging and Alzheimer's disease. *Neurobiol Aging* 17:513–521, 1996.

Eager, R.P. Selective staining of degenerating axons in the central nervous system by simplified method: Spinal cord projections to external cuneate and inferior olivary nuclei in the cat. *Brain Res* 22:137–141, 1970.

Freund, G. The interaction of chronic alcohol consumption and aging on brain structure and function. *Alcohol Clin Exp Res* 6:13–21, 1982.

Freund, G., and Butters, N. Alcohol and aging: Challenges for the future. *Alcohol Clin Exp Res* 6:1–2, 1982.

Golden, C.J.; Graber, B.; Berg, I.; Coffman, J.; and Block, S. Different brain density between chronic alcoholic and control patients. *Science* 211:508–510, 1981.

Halliday, G., and Baker K. Noradrenergic locus coeruleus neurones [letters]. *Alcohol Clin Exp Res* 20(1):191–192, 1996.

Halliday, G.M.; Cullen, K.; and Cairns, M.J. Quantitation and three-dimensional reconstruction of Ch4 nucleus in the human basal forebrain. *Synapse* 15:1–16, 1993.

Halliday, G.M.; Cullen, K.; and Harding, A. Neuropathological correlates of memory dysfunction in the Wernicke-Korsakoff syndrome. *Alcohol Alcohol Suppl* 2:245–251, 1994.

Halliday, G.M.; McRitchie, D.A.; Cartwright, H.; Pamphlett, R.; Hely, M.A.; and Morris, J.G.L. Midbrain neuropathology in idiopathic Parkinson's disease and diffuse Lewy body disease. *J Clin Neurosci* 3:52–60, 1996.

Harding, A.J.; Halliday, G.M.; Ng, J.L.F.; Harper, C.G.; and Kril, J.J. Loss of vasopressin-immunoreactive neurons in alcoholics. *Neuroscience* 73:699–708, 1996.

Harper, C.G., and Blumbergs, P.C. Brain weights in alcoholics. *J Neurol Neurosurg Psychiatry* 45:838–840, 1982.

Harper, C., and Corbett, D. A quantitative Golgi study of cortical neurons from alcoholic patients. *J Neurol Neurosurg Psychiatry* 53:865–861, 1990.

Harper, C.G., and Kril, J.J. If you drink your brain will shrink. Neuropathological considerations. *Alcohol Alcohol Suppl* 1:375–380, 1991.

Harper, C.; Kril, J.; Raven, D.; and Jones, N. Intracranial cavity volumes: A new method and its potential applications. *Neuropathol Appl Neurobiol* 10:25–32, 1984.

Harper, C.G.; Kril, J.J.; and Holloway, R.L. Brain shrinkage in chronic alcoholics—a pathological study. *Br Med J* 290:501–504, 1985.

Harper, C.; Kril, J.; and Daly, J. Are we drinking our neurones away? *Br Med J* 294:534–536, 1987*a*.

Harper, C.G.; Kril, J.J.; and Daly, J.M. The specific gravity of the brains of alcoholic and control patients: A pathological study. *Br J Addict* 82:1349–1354, 1987*b*.

Harper, C.G.; Kril, J.J.; and Daly, J.M. Brain shrinkage in alcoholics is not caused by changes in hydration: A pathological study. *J Neurol Neurosurg Psychiatry* 51:124–127, 1988*a*.

Harper, C.; Kril, J.; and Daly, J. Does a "moderate" alcohol intake damage the brain? *J Neurol Neurosurg Psychiatry* 51:909–913, 1988*b*.

Harper, C.; Kril, J.; and Daly, J. Cerebral lipids and alcohol abuse in humans. *Aust Drug Alcohol Rev* 8:69–77, 1991.

Haug, H., and Eggers, R. Morphometry of the human cortex cerebri and corpus

striatum during aging. *Neurobiol Aging* 12:336–338, 1991.

Jacobson, R. The contributions of sex and drinking history to the CT brain scan changes in alcoholics. *Psychol Med* 16:547–559, 1986.

Jensen, G.B., and Pakkenberg, B. Do alcoholics drink their neurons away? *Lancet* 342:1201–1204, 1993.

Jernigan, T.L.; Butters, N.; DiTriaglia, G.; Schafer, K.; Smith, T.; Irwin, M.; Grant, I.; Schuckit, M.; and Cermak, L.S. Reduced cerebral grey matter observed in alcoholics using magnetic resonance imaging. *Alcohol Clin Exp Res* 15:418–427, 1991*a*.

Jernigan, T.L.; Schafer, K.; Butters, N.; and Cermak, L.S. Magnetic resonance imaging of alcoholic Korsakoff's patients. *Neuropsychopharmacology* 4:175–186, 1991*b*.

Kemper, T.L. Neuroanatomical and neuropathological changes during aging and dementia. In: Albert, M.L., and Knoefel, J.E., eds. *Clinical Neurology and Aging*. New York and Oxford: Oxford University Press, 1994. pp. 3–67.

Kril, J.J.; Halliday, G.M.; Svoboda, M.D.; and Cartwright, H. The cerebral cortex is damaged in chronic alcoholics. *Neuroscience* 79:983–998, 1997.

Langlais, P.J., and Zhang, S.-X. Cortical white matter damage without Wernicke's encephalopathy following mild thiamine deficiency in rats. *Alcohol Clin Exp Res* 20:76A, 1996.

Langlais, P.J., and Zhang S.-X. Cortical and subcortical white matter damage without Wernicke's encephalopathy after recovery from thiamine deficiency in the rat. *Alcohol Clin Exp Res* 21(3):434–443, 1997.

Lesch, P.; Schmidt, E.; and Schmidt, F.W. Effects of chronic alcohol abuse on the structural lipids in the human brain. Hepatocerebral degeneration, I. *Z Klin Chem Klin Biochem* 10:410–415, 1972.

Leuchter, A.F.; Dunkin, J.J.; Lufkin, R.B.; Anzai, Y.; Cook, I.A.; and Newton, T.F. Effect of white matter disease on functional connections in the aging brain. *J Neurol Neurosurg Psychiatry* 57:1347–1354, 1994.

Lindboe, C.F., and Loberg, E.M. The frequency of brain lesions in alcoholics. *J Neurol Sci* 88:107–113, 1988.

Miller, A.K.H., and Corsellis, J.A.N. Evidence for a secular increase in human brain weight during the past century. *Ann Hum Biol* 4:253–257, 1977.

Mrak, R.E.; Griffin, W.S.T.; and Graham, D.I. Aging-associated changes in human brain. *J Neuropathol Exp Neurol* 56(12):1269–1275, 1997.

Olsson, N.U.; Harding, A.J.; Harper, C.; and Salem, N.J. High-performance liquid chromatography method with light-scattering detection for measurements of lipid class composition: Analysis of brains from alcoholics. *J Chromatogr B Biomed Appl* 681:213–218, 1996.

Parker, E.S., and Noble, E.P. Alcohol and aging process in social drinkers. *J Stud Alcohol* 41:170–178, 1980.

Peters, A.; Leahu, D.; Moss, M.B.; and McNally, K.J. The effects of aging on area 46 of the frontal cortex of the rhesus monkey. *Cereb Cortex* 6:621–635, 1994.

Peters, A.; Rosene, D.L.; Moss, M.B.; Kemper, T.L.; Abraham, C.R.; Tigges, J.; and Albert, M.S. Neurobiological bases of age-related cognitive decline in the rhesus monkey. *J Neuropathol Exp Neurol* 55:861–874, 1996.

Pfefferbaum, A.; Lim, K.O.; Zipursky, R.B.; Mathalon, D.H.; Rosenbloom, M.J.; Lane, B.; Ha, C.N.; and Sullivan, E.V. Brain gray and white matter volume loss accelerates with aging in chronic alcoholics: A quantitative MRI study. *Alcohol Clin Exp Res* 16:1078–1089, 1992.

Phillips, D.E. Effects of limited postnatal ethanol exposure on the development of myelin and nerve fibers in rat optic nerve. *Exp Neurol* 103:90–100, 1989.

Phillips, S.C.; Harper, C.G.; and Kril, J. A quantitative histological study of the cerebellar vermis in alcoholic patients. *Brain* 110:301–314, 1987.

Phillips, S.C.; Harper, C.G.; and Kril, J.J. The contribution of Wernicke's encephalopathy to alcohol-related cerebellar damage. *Drug Alcohol Rev* 9:53–60, 1990.

Shear, P.K.; Jernigan, T.L.; and Butters, N. Volumetric magnetic resonance imaging quantification of longitudinal brain changes in abstinent alcoholics. *Alcohol Clin Exp Res* 18:172–176, 1994.

Sullivan, E.V.; Marsh, L.; Mathalon, D.H.; Lim, K.O.; and Pfefferbaum, A. Anterior hippocampal volume deficits in nonamnesic, aging chronic alcoholics. *Alcohol Clin Exp Res* 19:110–122, 1995.

Sullivan, E.V.; Marsh, L.; Mathalon, D.H.; Lim, K.O.; and Pfefferbaum, A. Relationship between alcohol withdrawal seizures and temporal lobe white matter volume deficits. *Alcohol Clin Exp Res* 20:348–354, 1996.

Tavares, M.A., and Paula-Barbosa, M.M. Lipofuscin granules in Purkinje cells after long-term alcohol consumption in rats. *Alcohol Clin Exp Res 7*:302–306, 1983.

Terry, R.D., and Hansen, L.A. Neocortical cell counts in normal human adult aging. *Ann Neurol* 21:530–539, 1987.

Terry, R.D.; Peck, A.; DeTeresa, R.; Schechter, R.; and Horoupian, D.S. Some morphological aspects of the brain in senile dementia of the Alzheimer type. *Ann Neurol* 10:184–192, 1981.

Torvik, A.; Lindboe, C.F.; and Rodge, S. Brain lesions in alcoholics. A neuropathological study with clinical correlations. *J Neurol Sci* 56:233–248, 1982.

Tuck, R.R., and Jackson, M. Social, neurological and cognitive disorders in alcoholics. *Med J Aust* 155:225–229, 1991.

Wickelgren, I. For the cortex, neuron loss may be less than thought. *Science* 273:48–50, 1996.

Wiggins, R.C.; Gorman, A.; Rolsten, C.; Samorajski, T.; Ballinger, W.E.J.; and Freund, G. Effects of aging and alcohol on the biochemical composition of histologically normal human brain. *Metab Brain Dis* 3:67–80, 1988.

Ylikoski, R.; Ylikoski, A.; Erkinjuntti, T.; Sulkava, R.; Raininko, R.; and Tilvis, R. White matter changes in healthy elderly persons correlate with attention and speed of mental processing. *Arch Neurol* 50:818–824, 1993.

Chapter 8

Interaction of Aging and Ethanol on Brain Membrane Structure and Neurotransmitters

W. Gibson Wood, Ph.D.

Effects of ethanol differ in aged as compared with younger individuals (Beresford and Lucey 1995), and it is generally accepted that ethanol acts differently on the brain of aged individuals than on the brain of younger individuals (Wood 1995). For example, aged mice injected with ethanol sleep longer and wake up at a lower blood and brain ethanol concentration than do younger mice (Ritzmann and Springer 1980; Wood and Armbrecht 1982*a*). It has been reported that chronic ethanol consumption has a greater effect on aged than on younger mice; aged mice did not develop tolerance, and ethanol withdrawal was more severe and of longer duration when compared with younger mice (Wood et al. 1982). One might conclude from these studies that the brain of the aged mice is somehow more sensitive to effects of ethanol than is the brain of younger animals. However, ethanol had less of an effect on inducing hypothermia in aged mice than in younger mice (Wood and Armbrecht 1982*a*). Ethanol-induced changes in body temperature were significantly less in aged mice than such changes in younger mice.

Increasing age modifies the effects of ethanol, with aged organisms being more or less sensitive to effects of ethanol when compared with younger organisms. Potential explanations for

W.G. Wood, Ph.D., is associate director for education and evaluation and acting associate director for research, Geriatric Research, Education and Clinical Center, VA Medical Center, GRECC 11G, Minneapolis, MN 55417. Dr. Wood is also a professor in the Department of Pharmacology, University of Minnesota School of Medicine in Minneapolis.

age differences in response to ethanol include metabolism of ethanol, ratio of body water to body weight, and effects of ethanol on neuronal structure and function. Ethanol metabolism and the ratio of body water to body weight do not fully explain differences in response to ethanol for phenomena such as intoxication, sleep time, righting reflex, and severity of withdrawal (reviewed in Wood and Armbrecht 1982*b*; Wood 1995). Can age differences in response to ethanol be explained by ethanol having a differential effect on neuronal structure and function in aged versus younger individuals? This question is addressed in this chapter by reviewing studies on membrane lipid structure and the gamma-aminobutyric acid (GABA), *N*-methyl-D-aspartate (NMDA), and dopaminergic receptor systems. In addition, there is a discussion of factors that have impeded progress being made in understanding the molecular basis of the actions of ethanol in aged individuals and means that may promote progress in this important area.

MEMBRANE LIPID STRUCTURE

It has been well established that ethanol partitions into membranes and disrupts or fluidizes the membrane lipid environment (reviewed in Goldstein 1986; Wood et al. 1996). Although some have argued that ethanol-induced changes in bulk membrane fluidity are not of primary importance to understanding effects of ethanol, the fact remains that membranes of ethanol-tolerant animals and alcoholic patients are resistant to perturbation by ethanol compared with control subjects (Chin and Goldstein 1977; Beauge et al. 1985; Wood et al. 1987).

Several years ago, my colleagues and I proposed that age differences in response to ethanol might be associated with ethanol-induced changes in membrane fluidity (Armbrecht et al. 1983). Fluidity of synaptic plasma membranes (SPM) from three different age groups of mice (3–5, 11–13, and 22–24 months of age) was determined using the spin-labeled probe 5-doxyl stearic acid and electron spin resonance spectroscopy. Synaptic plasma membranes of the two older groups were resistant to ethanol perturbation when compared with those of the youngest group. We also found similar results using brain microsomes and erythrocyte membranes from the three different age groups of mice (Armbrecht et al. 1983).

Two important conclusions were drawn from this study. First, increasing age modifies the effects of ethanol on membrane fluidity. Second, ethanol-induced changes in membrane bulk fluidity are not predictive of in vivo sensitivity to ethanol. Aged mice sleep longer and awake at a lower blood and brain ethanol concentration than do younger mice (Wood and Armbrecht 1982*a*; Ritzmann and Springer 1980).

Ethanol-induced changes in the bulk fluidity of membranes do not explain age differences in response to ethanol in vivo. Studies in support of the membrane fluidity hypothesis examined the total or average bulk fluidity of membranes (reviewed in Wood and Schroeder

1988, 1992). However, plasma membranes are a bilayer consisting of two individual monolayers or leaflets (reviewed in Op den Kamp 1979; Schroeder 1985; Wood and Schroeder 1992). These leaflets differ in fluidity, lipid distribution, electrical charge, and function. The exofacial or outer leaflet is markedly more fluid than the cytofacial or inner leaflet. Phosphatidylcholine and sphingomyelin are enriched in the exofacial leaflet, whereas phophatidylethanolamine, phosphatidylserine, and phosphatidylinositol are primarily cytofacial leaflet phospholipids. The exofacial leaflet is considered zwitterionic or positively charged, and the cytofacial leaflet is negatively charged. The regulatory activity of certain membrane proteins is associated with the two leaflets.

In view of the fact that the exofacial leaflet is more fluid than the cytofacial leaflet, we proposed that ethanol would have a greater effect on the exofacial leaflet (Wood and Schroeder, 1988; Schroeder et al. 1988). It was determined, using the fluorescence of diphenylhexatriene and quenching procedures, that the exofacial leaflet was significantly more fluid than the cytofacial leaflet. Moreover, we found that ethanol at a concentration as low as 25 mM significantly increased fluidity of the SPM exofacial leaflet and had no effect on the cytofacial leaflet. The cytofacial leaflet was resistant to ethanol even at a concentration as high as 400 mM. In a subsequent study, we reported that chronic ethanol consumption altered the fluidity of mouse SPM leaflets, but this effect was in the opposite direction for each leaflet (Wood et al. 1989). The exofacial leaflet of the chronic ethanol group became significantly less fluid than the exofacial leaflet of the pair-fed control group. The cytofacial leaflet of the chronic ethanol group became more fluid than the cytofacial leaflet of the control group.

This change in the fluidity of the two leaflets was accounted for by a redistribution of cholesterol in SPM of the chronic ethanol–treated mice (Wood et al. 1990). Cholesterol is not evenly distributed in the two membrane leaflets. The SPM exofacial leaflet contains approximately 13 to 15 percent of the total amount of SPM cholesterol, and the cytofacial leaflet contains over 85 percent of the total SPM cholesterol. We found that the transbilayer distribution of cholesterol was altered in SPM of chronic ethanol–treated mice (Wood et al. 1990). There was approximately twice as much cholesterol in the exofacial leaflet of the chronic ethanol mice when compared with the exofacial leaflet of the control mice. There was also a reduction of cholesterol in the cytofacial leaflet of the chronic ethanol mice. The total amounts of SPM cholesterol and the cholesterol-to-phospholipid ratios did not significantly differ between the ethanol and pair-fed groups.

These studies showed that ethanol both in vitro and in vivo had an asymmetric effect on the two leaflets of SPM. The exofacial leaflet was very susceptible to effects of ethanol. This approach was a new way of viewing how ethanol behaves in membranes, and we thought that the asymmetric effects of ethanol may mechanistically

explain sensitivity and tolerance to ethanol. However, recent studies from our laboratory on the transbilayer fluidity and cholesterol distribution of SPM from different age groups of mice weaken the hypothesis that transbilayer fluidity and cholesterol asymmetry are involved in sensitivity and tolerance to ethanol. The exofacial leaflet of aged mice (24–25 months) was significantly less fluid than the exofacial leaflet of mice 3–4 months of age (Igbavboa et al. 1996). Moreover, there was more than a twofold increase in the amount of cholesterol in the exofacial leaflet of the oldest age group of mice when compared with the youngest age group. The transbilayer fluidity and cholesterol distribution in the SPM exofacial leaflet were similar in chronic ethanol–treated mice and aged mice. An exception is the fluidity of the cytofacial leaflet, which became more fluid in the chronic ethanol–treated mice but did not change with increasing age. Ethanol-induced changes in the transbilayer distribution of cholesterol and the fluidity of the individual leaflets may not explain sensitivity and tolerance to ethanol. Instead, these changes may be pathological effects of ethanol that precede neuronal dysfunction. It has been shown, for example, that modification of cholesterol in the exofacial leaflet reduces activity of SPM Ca^{2+} + Mg^{2+}-ATPase (Wood et al. 1995).

NEUROTRANSMITTERS AND RECEPTORS

Ethanol in vitro and in vivo has been shown to act on different neurotransmitters and receptors in the brain, and such effects might explain age differences in response to ethanol. We examined effects of ethanol in vitro on the release of GABA from cortical synaptosomes of mice that were 4, 14, and 28 months of age (Strong and Wood 1984). Potassium-stimulated release of GABA was not affected by increasing age. When ethanol was added, stimulated GABA release was inhibited significantly more in synaptosomes of younger mice than the release of older mice. The IC_{50} (i.e., concentration of ethanol resulting in 50 percent inhibition of GABA release) was twofold higher for the 28-month group compared with the 4-month group. Increasing age reduced effects of ethanol in vitro on the potassium-stimulated release of GABA from mouse synaptosomes, and those results were entirely consistent with the resistance to ethanol-induced fluidization of SPM from aged mice when compared with younger mice that was discussed earlier in this chapter.

Effects of chronic ethanol consumption on GABA release of synaptosomes were also examined (Strong and Wood 1984). Mice 4 and 28 months of age were administered an ethanol liquid diet or a pair-fed control diet for 4 weeks. Potassium-stimulated GABA release was inhibited more by ethanol in vitro in synaptosomes of the 4-month-old pair-fed control group compared with the other groups. Synaptosomes of the 4-month-old chronic ethanol–treated group were resistant to the effects of ethanol in vitro. This resistance to

effects of ethanol in vitro was an indication that cellular tolerance had developed as a result of the 4 weeks of chronic ethanol consumption. Potassium-stimulated GABA release from synaptosomes of the 28-month-old chronic ethanol group did not differ significantly from release of the 28-month-old pair-fed control group. One interpretation of those results is that aged animals are impaired in their capacity to develop cellular tolerance to ethanol. Alternatively, it could be argued that because ethanol in vitro had less of an effect on GABA release of aged control mice, aged animals are intrinsically tolerant to ethanol.

It was reported in another study that the GABA system was less responsive to effects of ethanol in aged rats than younger rats (Lin et al. 1993). Isoproterenol modulation of GABA function in cerebellar Purkinje neurons was less in neurons of rats 24–25 months old than rats 4–6 months old (Lin et al. 1993). Ethanol (750 mM) had less of an effect on the Purkinje neurons of aged rats when compared with young rats.

An injection of ethanol was found to reduce the affinity of the GABA receptor in 12- to 15-month-old rats (Komiskey et al. 1988). However, significant differences in affinity of the GABA receptor were not observed between 3-month-old and 28-month-old rats following an injection of ethanol. It also was reported that the maximum number of binding sites was not altered by ethanol injection or age. An interesting aspect of that study was that a lower dose of ethanol was required in the 28-month-old animals in order to obtain similar blood and brain ethanol levels among the three groups. The young animals received an ethanol dose of 0.6 g/kg, while the 28-month-old animals were injected with 0.45 g/kg. Previously it was proposed that age differences in response to ethanol may result from less body water in aged as compared with younger individuals (York 1982). It was shown that when the concentration of ethanol that was injected was based on estimated body water content, effects of ethanol in aged rats were reduced. Age differences in body water content do not explain findings showing that old mice lose and regain the righting reflex at lower brain and blood ethanol levels as compared with young mice (Ritzmann and Springer 1980; Wood and Armbrecht 1982*a*). Aged rats and mice may differ in response to ethanol. More studies on the interaction among body water, ethanol, and aging are clearly needed.

There has been considerable interest in effects of ethanol on the NMDA receptor system (reviewed in Tabakoff and Hoffman 1996). For example, ethanol inhibits NMDA-induced ion currents of hippocampal neurons and the uptake of calcium into dissociated brain cells. Leslie and his group examined effects of ethanol on NMDA-stimulated release of catecholamines in brain slices from cortex, hippocampus, and striatum of Fischer 344 rats, 3–5 months, 12–14 months, and 24–28 months of age (Brown et al. 1992). NMDA induced release of catecholamines in all of the brain areas examined, and aged animals had reduced

catecholamine release compared with the younger animals. Ethanol inhibited NMDA-stimulated catecholamine release in brain slices of the three different age groups. Ethanol had a greater inhibitory effect on NMDA-stimulated catecholamine release in aged as compared with younger animals. However, the IC_{50} for ethanol inhibition of NMDA-stimulated catecholamine release did not significantly differ among the three age groups. The authors concluded that NMDA-stimulated release of catecholamines in the presence of ethanol was not associated with aging.

Dopaminergic systems have been found to be affected by ethanol or aging. It was recently reported that both chronic ethanol consumption and aging modified dopamine D_2 receptors in brain regions of 5-, 14-, and 24-month-old Fischer 344 rats (Tajuddin and Druse 1996). Ethanol and pair-fed control liquid diets were administered to rats for 6 weeks. The total number of D_2 receptors was reduced in the rostral and caudal striatum of aged rats. However, ethanol did not accelerate the loss of D_2 receptors. Specific binding of [^{3}H]spiperone was lower in the frontal cortex of the aged animals. Chronic ethanol consumption produced an increase in the total number of D_2 receptors in the nucleus accumbens. Increasing age per se did not alter the total number of D_2 receptors in the nucleus accumbens.

CONCLUSIONS

One conclusion is that studies on potential mechanisms that may explain age differences in response to ethanol reveal more information pertaining to what is not known than to what is understood with respect to mechanisms of ethanol's action in general. The GABA, NMDA, and dopamine systems may not be involved in ethanol sensitivity and ethanol tolerance. It could be argued that if those systems were associated with ethanol-induced phenomena then there would have been age differences in effects of ethanol on those neurotransmitter systems. It is certainly well established that aged animals and humans differ in their responses to ethanol in vivo when compared with younger individuals (reviewed in Beresford and Lucey 1995). However, it is important to emphasize that the number of studies that examined those neurotransmitter systems in aged animals is small. It also appears that changes in the transbilayer distribution of cholesterol may not fully explain cellular tolerance to ethanol. Distribution of cholesterol in the exofacial leaflets of young chronic ethanol–treated mice was similar to that of aged mice that were never exposed to ethanol. The body water hypothesis is of potential importance and certainly merits further study. Experiments need to be conducted that determine the total amount of body water, brain water, and the ratios of body water and brain water to body weight and brain weight in both rats and mice of different ages.

There are two major problems that limit progress toward understanding mechanisms that may explain age differences in response to ethanol. The first problem is that molecular mechanisms of ethanol phenomena, such as

intoxication and tolerance, are not well understood, irrespective of age. It might be argued that until such mechanisms have been identified it will be difficult to establish factors contributing to differences in effects of ethanol in aged individuals. Equally important as a major problem is that there simply are not enough investigators who are studying aging and effects of ethanol, and, as a result, very little progress has been made.

The fact that there are so few laboratories pursuing studies of aging and effects of ethanol may be attributed to several different reasons. Aging alone and ethanol alone have systemic effects on the body that can certainly complicate an understanding of neuronal function when effects of ethanol are examined in the aged organisms. Two very practical reasons that may limit interest in pursuing studies on aging and effects of ethanol are the cost and the amount of time involved with such studies.

Animal models of aging are expensive, whether one is raising animals or is purchasing animals from the National Institute on Aging (NIA) contract animal facilities. A 25-month-old C57BL mouse purchased from the NIA contract colony costs approximately $70. Multiply that number by the number of animals used in experiments involving membranes, and the cost certainly becomes a factor if one is considering generating pilot data for a grant application.

The time spent doing aging experiments can be three or four times longer than experiments using only young animals. It is generally recommended that cross-sectional studies use at least three different age groups. Thus, the time involved in examining, for example, different ethanol concentrations on a particular biochemical parameter is markedly increased when studying different age groups. Aging studies also can be very time consuming if the investigator chooses to maintain an aging colony. The median lifespan of a C57BL mouse is approximately 24 months. In addition, the risk of losing the colony to disease or malfunction of heating and cooling equipment is an ever-present concern.

Understanding the complex interaction of aging and ethanol at the molecular level will not proceed unless there are a sufficient number of investigators working in the area. The National Institute on Alcohol Abuse and Alcoholism needs to develop funding mechanisms to promote research on aging and effects of ethanol in order to attract investigators who are using state-of-the-art techniques.

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REFERENCES

Armbrecht, H.J.; Wood, W.G.; Wise, R.W.; Walsh, J.B.; Thomas, B.N.; and Strong, R. Ethanol-induced disordering of membranes from different age groups of C57BL/6NNIA mice. *J Pharmacol Exp Ther* 226:387–391, 1983.

Beauge, F.; Stibler, H.; and Borg, S. Abnormal fluidity and surface carbohydrate content of erythrocyte membrane in alcoholic patients. *Alcohol Clin Exp Res* 9:322–328, 1985.

Beresford, T.P., and Lucey, M.R. Ethanol metabolism and intoxication in the elderly. In: Beresford, T.P., and Gomberg, E.S.L., eds. *Alcohol and Aging*. New York: Oxford University Press, 1995. pp. 117–127.

Brown, L.M.; Trent, R.D.; Jones, T.W.; Gonzales, R.A.; and Leslie, S.W. Alcohol inhibition of NMDA-stimulated catecholamine efflux in aging brain. *Alcohol* 9:555–558, 1992.

Chin, J.H., and Goldstein, D.B. Drug tolerance in biomembranes: A spin label study of the effects of ethanol. *Science* 196:684–685, 1977.

Goldstein, D.B. Effect of alcohol on cellular membranes. *Ann Emerg Med* 15:1013–1018, 1986.

Igbavboa, U.; Avdulov, N.A.; Schroeder, F.; and Wood, W.G. Increasing age alters transbilayer fluidity and cholesterol asymmetry in synaptic plasma membranes of mice. *J Neurochem* 66:1717–1725, 1996.

Komiskey, H.L.; Raemont, L.M.; and Mundinger, K.L. Aging: Modulation of $GABA_A$ binding sites by ethanol and diazepam. *Brain Res* 458:37–44, 1988.

Lin, A.M.-Y.; Bickford, P.C.; and Palmer, M.R. The effects of ethanol on τ-aminobutyric acidinduced depressions of cerebellar Purkinje neurons: Influence of beta adrenergic receptor action in young and aged Fischer 344 rats. *J Pharmacol Exp Ther* 264:951–957, 1993.

Op den Kamp, J.A.F. Lipid asymmetry in membranes. *Annu Rev Biochem* 48:47–71, 1979.

Ritzmann, R.F., and Springer, A. Age differences in brain sensitivity and tolerance to ethanol in mice. *Age* 3:15–17, 1980.

Schroeder, F. Fluorescence probes unravel asymmetric structure of membranes. *Subcell Biochem* 11:51–100, 1985.

Schroeder, F.; Morrison, W.J.; Gorka, C.; and Wood, W.G. Transbilayer effects of ethanol on fluidity of brain membrane leaflets. *Biochim Biophys Acta* 946:85–94, 1988.

Strong, R., and Wood, W.G. Membrane properties and aging: In vivo and in vitro effects of ethanol on synaptosomal GABA-aminobutyric acid release. *J Pharmacol Exp Ther* 229:726–730, 1984.

Tabakoff, B., and Hoffman, P.L. Ethanol and glutamate receptors. In: Deitrich, R.A., and Erwin, V.G., eds. *Pharmacological Effects of Ethanol on the Nervous System*. Boca Raton, FL: CRC Press, 1996. pp. 73–93.

Tajuddin, N.F., and Druse, M.J. Effects of chronic alcohol consumption and aging on dopamine D_2 receptors in Fischer 344 rats. *Alcohol Clin Exp Res* 20:144–151, 1996.

Wood, W.G. Age differences in effects of alcohol on brain membrane structure, neurotransmitters and receptors. In: Beresford, T.P., and Gomberg, E.S.L., eds. *Alcohol and Aging*. New York: Oxford University Press, 1995. pp. 136–149.

Wood, W.G., and Armbrecht, H.J. Age differences in ethanol-induced hypothermia and impairment in mice. *Neurobiol Aging* 3:243–246, 1982*a*.

Wood, W.G., and Armbrecht, H.J. Behavioral effects of ethanol in animals: Age differences and age changes. *Alcohol Clin Exp Res* 6:3–12, 1982*b*.

Wood, W.G., and Schroeder, F. Membrane effects of ethanol: Bulk lipid versus lipid domains. *Life Sci* 43:467–475, 1988.

Wood, W.G., and Schroeder, F. Membrane exofacial and cytofacial leaflets: A new approach to understanding how ethanol alters brain membranes. In: Watson, R.R., ed. *Alcohol and Neurobiology: Receptors, Membranes, and Channels.* Boca Raton, FL: CRC Press, 1992. pp. 161–184.

Wood, W.G.; Armbrecht, H.J.; and Wise, R.W. Ethanol intoxication and withdrawal among three age groups of C57BL/6NNia mice. *Pharmacol Biochem Behav* 17:1037–1041, 1982.

Wood, W.G.; Lahiri, S.; Gorka, C.; Armbrecht, H.J.; and Strong, R. In vitro effects of ethanol on erythrocyte membrane fluidity of alcoholic patients: An electron spin resonance study. *Alcohol Clin Exp Res* 11:332–335, 1987.

Wood, W.G.; Gorka, C.; and Schroeder, F. Acute and chronic effects of ethanol on transbilayer membrane domains. *J Neurochem* 52:1925–1930, 1989.

Wood, W.G.; Schroeder, F.; Hogy, L.; Rao, A.M.; and Nemecz, G. Asymmetric distribution of a fluorescent sterol in synaptic plasma membranes: Effects of chronic ethanol consumption. *Biochim Biophys Acta* 1025:243–246, 1990.

Wood, W.G.; Igbavboa, U.; Rao, A.M.; Schroeder, F.; and Avdulov, N.A. Cholesterol oxidation reduces Ca^{2+} + Mg^{2+}-ATPase activity, interdigitation, and increases fluidity of brain synaptic plasma membranes. *Brain Res* 683:36–42, 1995.

Wood, W.G.; Schroeder, F.; Rao, A.M.; Igbavboa, U.; and Avdulov, N.A. Membranes and ethanol: Lipid domains and lipid-protein interactions. In: Deitrich, R.A., and Erwin, V.G., eds. *Pharmacological Effects of Ethanol on the Nervous System.* Boca Raton, FL: CRC Press, 1996. pp. 13–27.

York, J.L. Body water content, ethanol pharmacokinetics, and the responsiveness to ethanol in young and old rats. *Dev Pharmacol Ther* 4:106–116, 1982.

Chapter 9

Cerebral Injury From Severe Chronic Alcoholism

Sid Gilman, M.D., Kenneth M. Adams, Ph.D., Robert A. Koeppe, Ph.D., Larry Junck, M.D., Doug Johnson-Greene, Ph.D., Karen J. Kluin, M.S., and Mary L. Heumann

The major medical and neurological effects of severe chronic alcoholism, frequently coupled with malnutrition, have been known for many decades; they include cirrhosis, peripheral neuropathy, cerebellar degeneration, and Wernicke-Korsakoff syndrome (Victor et al. 1959; Victor and Ferrendelli 1970; R.D. Adams 1976; Charness et al. 1989; Victor et al. 1989; Lieber 1995). Even in the absence of these disorders, however, and probably occurring more frequently, severe chronic alcoholism results in subtle disorders of higher intellectual function that can be detected with neuropsychological evaluations. These disorders include attentional skills;

S. Gilman, M.D., is the William J. Herdman professor and chair of the Department of Neurology, University of Michigan, 1500 East Medical Center Dr., Ann Arbor, MI 48109–0316, and is a principal investigator in the University of Michigan Alcohol Research Center (UMARC), 400 East Eisenhower, Suite 2A, Ann Arbor, MI 48109–0740. K.M. Adams, Ph.D., is professor of psychology in the Division of Neuropsychology, Department of Psychiatry, University of Michigan; director of the Psychology Service, Ann Arbor Veterans Affairs Medical Center, 2215 Fuller Rd., Ann Arbor, MI 48104–2399; and co-investigator in the UMARC. R.A. Koeppe, Ph.D., is professor and senior research scientist in the Division of Nuclear Medicine, Department of Internal Medicine, University of Michigan, and co-investigator in the UMARC. L. Junck, M.D., is associate professor in the Department of Neurology, University of Michigan, and co-investigator in the UMARC. D. Johnson-Greene, Ph.D., is assistant professor and staff neuropsychologist in the Department of Physical Medicine and Rehabilitation, Johns Hopkins University School of Medicine, 5601 Loch Raven Blvd., Suite 406, Baltimore, MD 21239. K.J. Kluin, M.S., is research investigator in the Department of Neurology, University of Michigan; allied health supervisor in the Division of Speech Pathology, Department of Physical Medicine and Rehabilitation, University of Michigan; and co-investigator in the UMARC. M.L. Heumann is a research associate in the Department of Neurology, University of Michigan.

problem-solving difficulties; abnormalities of concept formation, rule learning, and set shifting; disorders of perceptual-spatial skills; and abnormalities of tactile performance (Ron 1983; Walsh 1983; Grant et al. 1986; Hunt and Nixon 1993).

The neuropathological changes underlying alcoholic cerebellar degeneration (ACD) and Wernicke-Korsakoff syndrome in severe chronic alcoholism are well described (Victor et al. 1959; Victor and Ferrendelli 1970; R.D. Adams 1976; Charness et al. 1989; Victor et al. 1989). The neuropathological changes that might be responsible for the neuropsychological deficits, however, were not known until recently, when the results were reported of several systematic neuropathological examinations of the cerebral cortex and cerebellum in patients with severe chronic alcoholism. These studies demonstrated decreased numbers of large neurons in the superior frontal cortex; shrunken neurons in the superior frontal, precentral, and cingulate cortex; and a reduction of the dendritic arbor of layer III pyramidal neurons in the superior frontal and motor cortex (Harper and Krill 1985; Ferrer et al. 1986; Harper et al. 1987; Harper and Kril 1989; Kril and Harper 1989; Harper and Corbett 1990; Harper and Kril 1990, 1991). Neuropathological studies have also shown asymptomatic degeneration in the cerebellum in up to 27 percent of chronic alcoholics, particularly depletion of neurons in the anterior superior vermis (Torvik et al. 1986; Phillips et al. 1987).

Although these careful neuropathological investigations have revealed the particular vulnerability of the frontal cortex and cerebellum, the specific sites responsible for the individual component neuropsychological disorders have not been determined. The lack of information on the specific sites is largely due to the fact that patients with severe chronic alcoholism who come to postmortem often have not been tested previously, and, in those who have been tested previously, a long delay frequently occurs between the time of testing and the time of death, with uncertain consequences of intervening events upon brain morphology.

The advent of anatomical and functional imaging techniques has made it possible to detect the focal and generalized effects of severe chronic alcoholism in living patients and to begin clarifying the relationship between the sites affected and the types of disorders of neuropsychological function that occur. Generalized cerebral atrophy, particularly involving the frontal lobes, has been recognized for many years (Courville 1955). Anatomical imaging studies, beginning with computed tomography (CT) scanning and later including magnetic resonance (MR) imaging, have shown generalized cerebral atrophy that appears to be partially reversible with abstinence (Carlen et al. 1978, 1981, 1986; Shimamura et al. 1988; Jernigan et al. 1991; Pfefferbaum et al. 1992, 1993; Sullivan et al. 1995). In ACD, this is accompanied by atrophy in the superior aspect of the cerebellar vermis.

Despite the excellent resolution of brain structure with anatomical imaging, particularly MR scanning, this approach alone is unlikely to bring full understanding of the regional vulnerability of the brain to severe chronic alcoholism and the correlation between focal areas of injury and abnormalities on neuropsychological testing. The first functional imaging study to address these issues in alcoholism involved determination of local cerebral metabolic rates for glucose (lCMRglc) utilizing [^{18}F]fluorodeoxyglucose ([^{18}F]FDG) and positron emission tomography (PET) (Samson et al. 1986). The study showed in a small number of cases a decrease of lCMRglc in the medial aspects of the frontal lobes of the cerebral cortex. This seminal paper triggered many additional studies in larger groups of severe chronic alcoholic subjects that not only confirmed the original findings, but also revealed correlations between decreased lCMRglc in subcomponents of the frontal lobes and abnormalities of performance in neuropsychological tests of frontal lobe functions (Sachs et al. 1987; Wik et al. 1988; Gilman et al. 1990; Jagust 1992; Volkow et al. 1992; Wang et al. 1992; K.M. Adams et al. 1993; Wang et al. 1993; Volkow et al. 1994; K.M. Adams et al. 1995*a*). Subsequent studies have concentrated on some of the focal abnormalities of neurotransmitter systems in severe chronic alcoholism (Volkow et al. 1993; K.M. Adams et al. 1995*b*; Gilman et al. 1996*c*). In this chapter, we review the results of structural and functional imaging studies in correlation with clinical neurological and neuropsychological abnormalities in severe chronic alcoholic men investigated by the University of Michigan Alcohol Research Center over the last decade.

METHODS

Patient Sample

The sample size ranged from 6 to 56 patients, ages 33–78. Stringent criteria were used for subject selection. First, patients had to fulfill the DSM-III-R diagnostic criteria for alcohol dependence (American Psychiatric Association 1987). The patients studied had a weekly intake of at least 560 g of ethanol over 2 of the 3 preceding years, but most exceeded this intake for an average of 20 years before entering the study. Admission to the study required at least one hospitalization for alcoholism, and the patients studied usually remained sober for no more than about 1 month at a time from the onset of heavy drinking. These patients are characterized as "heavy" drinkers in duration and intensity of alcohol intake (K.M. Adams et al. 1979).

Case selection required that each patient studied meet the quantitative exclusionary criteria for polydrug abuse of the National Institute on Drug Abuse (Wesson et al. 1978). Patients were excluded if they had suffered from any of a variety of neurological disorders, including closed head injury with loss of consciousness exceeding 30 minutes; stroke; birth complications leading to neurological disturbance; learning disorders; and other acquired or developmental diseases or disorders

and trauma that affect the central nervous system. These exclusionary criteria provided screening for complicating comorbidities that can obscure the interpretation of neuropsychological tests.

Some of the chronic alcoholic subjects studied had a diagnosis of ACD that was based on the criteria described above for alcohol dependence along with supportive evidence from the patients' history, physical examination, neurological examination, and laboratory tests (including anatomical imaging) to exclude other diseases. The diagnosis required the history of a gait disorder occurring in the course of severe chronic alcohol dependence, usually with associated malnutrition, in conjunction with evidence on neurological examination of an ataxia of gait of the cerebellar type, but without severe ataxia of upper extremity movements or speech. The gait disorder could have developed suddenly or slowly. Disorders of sensory function sufficient to cause gait ataxia could not be present.

Patient Evaluation

The patients selected for study were evaluated with a medical history and examination and a neurological history and examination. A battery of neuropsychological tests was performed, with tests that were relevant to the hypotheses under examination and that emphasized intellect, cognition, motor function, and other relevant aspects of behavior. The tests used were those with established reliability, validity, and wide usage in neuropsychological laboratories (Lezak 1986; Reitan and Wolfson 1986; Heaton et al. 1991). The test battery, which provided good coverage of verbal/nonverbal, retrograde/anterograde, procedural/declarative, and spontaneous/primed dimensions of memory, consisted of

- Wechsler Adult Intelligence Scale—Revised (WAIS-R)
- Halstead Category Test
- Tactual Performance Test
- Speech-Sounds Perception Test
- Seashore-Rhythm Test
- Finger-Tapping
- Grooved Pegboard
- Grip Strength
- Trail Making Test
- Aphasia Screening Examination
- Sensory-Perceptual Examination
- Lateral Dominance Examination
- Wechsler Memory Scale—Revised
- Corsi Blocks
- Selective Reminding Test
- Minnesota Multiphasic Personality Inventory—2 (MMPI-2)
- Wisconsin Card Sorting Test

Positron Emission Tomography

The subjects fasted for 4 hours before the PET scan. They were studied lying supine and awake in a quiet room, alert but not speaking, with eyes open from 5 minutes before injection until completion of the scan. A catheter was placed in a radial artery for blood sampling. PET scans were performed following intravenous injection of 10 mCi of [^{18}F]FDG. The subjects were imaged with either a Siemens/CTI 931/08-12 or a Siemens ECAT EXACT-47 scanner. Images from both scanners were reconstructed to a resolution of 8–9 mm full width at half-maximum (FWHM) in-plane. Since the axial sampling of

the EXACT-47 scanner is twice as fine as the CTI 931, two adjacent levels from the EXACT were averaged, providing images with the same axial spacing and nearly the same resolution as the CTI 931 scanner. Attenuation correction was calculated by the standard ellipse method. The PET studies were analyzed with a compartmental model and parameter estimation technique that provides pixel-by-pixel determinations of each measurement, thus creating "functional" images (Koeppe et al. 1991).

PET studies of benzodiazepine receptor binding were performed utilizing intravenous injection of 22 ± 2 mCi of [^{11}C]flumazenil ([^{11}C]FMZ). In these studies, 10 mCi of [^{18}F]FDG were administered 90 minutes after the [^{11}C]FMZ. Flumazenil was labeled with carbon-11 at high specific radioactivity using a methylation process (Mazière et al. 1984). Blood samples were taken as rapidly as possible during the first 2 minutes after tracer injection and then at progressively longer intervals throughout the remainder of the study. A total of 25–30 samples were taken per scan. The samples were centrifuged, and the plasma radioisotope concentrations were measured in a NaI well counter. Plasma levels of radiolabeled metabolites of FMZ were determined by a rapid Sep-Pak C_{18} cartridge chromatographic technique (Frey et al. 1991) in the samples taken at 1 minute and at 2 minutes, and in every sample from 3 minutes until the end of the scan. Dynamic PET scans were performed for 60 minutes beginning immediately after injection. Radioactive fiducials (markers for external landmarks) with 5 μCi of ^{11}C were placed on each subject's scalp. Computer routines automatically determined the locations of these fiducials and used this information to correct for patient motion.

The studies with [^{11}C]FMZ provided quantitative measurement of ligand influx (K_1), which is highly correlated with flow because the single-pass extraction fraction for [^{11}C]FMZ is greater than 50 percent (Koeppe et al. 1991). The studies also provided measurement of FMZ distribution volume, which is linearly related to the density of available receptor sites divided by the ligand dissociation constant (B_{max}'/K_D). The methods for benzodiazepine receptor binding measurement, including the assumptions and limitations and the performance of [^{11}C]FMZ, are published (Frey et al. 1991; Holthoff et al. 1991; Koeppe et al. 1991). In the [^{18}F]FDG studies, data were acquired 30–90 minutes postinjection and quantified utilizing the static scan method (Hutchins et al. 1984). Data acquired with the Siemens/CTI 931/08-12 scanner were obtained as two interleaved image sets.

PET studies of striatal monoaminergic neuronal terminals were performed with a new ligand, (+)[^{11}C] dihydrotetrabenazine (DTBZ). The ligand was prepared by [^{11}C]methylation of α–(+)-9-O-desmethylDTBZ, with a solid-phase supported system that allows purification and isolation of the product [^{11}C]DTBZ without HPLC purification. (+)[^{11}C]DTBZ was given intravenously as a loading bolus (50 percent of total dose over 1

minute) followed by continuous infusion of the remaining 50 percent of the dose. Equilibrium analysis was used to evaluate the density of the vesicular monoamine transporter type 2 (VMAT2) from the (+)[^{11}C]DTBZ studies. In addition, an autoradiographic calculation was performed to assess the ligand blood-brain barrier transport rate constant, K_1, from the initial 5-minute PET scan. Since the tracer was administered as a loading bolus followed by continuous infusion, both the brain concentration and blood concentration of (+)[^{11}C]DTBZ became nearly constant by 30 minutes after the start of the injection.

Arterial blood samples were acquired throughout the first 5 minutes of the study and then at 30, 40, 50, and 60 minutes after the start of the study. Plasma concentrations were measured, and the level of [^{11}C]-labeled metabolites was determined. Blood samples were processed for plasma concentration of [^{11}C] and corrected for metabolites. Plasma samples (0.4 mL) were added to tubes containing [^{3}H]DTBZ (Amersham, Arlington Heights, IL) in 125 mM PBS-EDTA buffer, pH 7.4 (0.6 mL). Samples then were applied to Sep-Pak C_{18} cartridges (Waters, Milford, MA) and polar metabolites of [^{11}C]DTBZ eluted in 9 mL of 1.25 mM PBS-EDTA buffer, 60:40 (v/v). Authentic DTBZ was eluted in 5 mL of absolute ethanol. The plasma free fraction of DTBZ was determined in each subject. The ligand's distribution volume gives a measure of VMAT2 density (Koeppe et al. 1995; Frey et al. 1996; Gilman et al. 1996*b*) The distribution volume is the ratio of the regional PET measure averaged over the late steady-state phase of the study (from 30 to 60 minutes) divided by the metabolite-corrected plasma concentration averaged over the same interval. Following determination of distribution volume, which is equivalent to the ratio of kinetic rate constants K_1/k_2, an estimate of K_1 was obtained using the metabolite-corrected arterial plasma samples acquired through the first 6 minutes of the study in conjunction with the single early scan. The calculation uses an autoradiographic estimation procedure, fixing the K_1/k_2 ratio to the value estimated from the 30- to 60-minute data (Hutchins et al. 1984). For each study, pixel-by-pixel parametric images of ligand distribution volume and K_1 were created.

Magnetic Resonance Imaging

Magnetic resonance scans were performed at 1.5 T utilizing coronal T1 (TR 500–600 ms/TE 18–25 ms) and axial T2 (TR 2,500–3,000 ms/TE 80–90 ms) images. Slice thickness was 5 mm, with 2–2.5 mm interval between slices. Scans were used to evaluate the effects of tissue atrophy on data obtained from PET scans and determine sites for study of volumes of interest in PET studies.

RESULTS

Cerebral Glucose Metabolism in Severe Chronic Alcoholism

Our initial studies of lCMRglc in severe chronic alcoholism were focused upon ACD with the intention of

characterizing the pattern of metabolic abnormalities in clinically symptomatic patients (Gilman et al. 1990). Since a previous PET study with [^{18}F]FDG had revealed hypometabolism in the medial aspects of the frontal lobe (Samson et al. 1986), studies of lCMRglc in the frontal regions were included. The studies were performed in 14 patients with clinical signs of ACD and in 5 patients with severe chronic alcoholism but without clinical signs of cerebellar disorder (non-ACD).

PET studies of the patients with ACD revealed significantly decreased lCMRglc in the superior cerebellar vermis in comparison with eight normal control subjects of the same gender and similar age. The non-ACD patients had no abnormality of lCMRglc in the cerebellum; however, in both the ACD and non-ACD patients, lCMRglc was significantly decreased bilaterally in the medial frontal region of the cerebral cortex in comparison with the normal control subjects. Moreover, frontal lobe lCMRglc was significantly correlated with the results of a neuropsychological test sensitive to frontal lobe function, the Halstead Category Test. The results demonstrated the utility of functional imaging with PET in determining the sites that may be responsible for some of the neuropsychological disorders in chronic alcoholism.

Neuropsychological Changes in Severe Chronic Alcoholism

In efforts to examine further the neuropsychological deficits of patients with severe chronic alcoholism and to relate these deficits to the glucose metabolic abnormalities observed, we utilized neuropsychological tests sensitive to frontal pathology in addition to tests that evaluate general neuropsychological performance, and we compared the results with those from PET [^{18}F]FDG scans (K.M. Adams et al. 1993, 1995*a*). Other investigators have taken similar approaches (Wang et al. 1993; Volkow et al. 1994). Tests known to be sensitive to frontal pathology include the Wisconsin Card Sorting Test, the Halstead Category Test, WAIS-R Similarities, Corsi Blocks, and the Tactual Performance Test. Neuropsychological studies and correlative studies of lCMRglc examined with PET and [^{18}F]FDG were completed in a patient sample of 37 (table 1).

Table 1. Patient Characteristics in Neuropsychological Studies of Severe Chronic Alcoholism in 37 Subjects.

Variable	Mean ± SD
Age (years)	49.8 ± 8.8
Education (years)	12.4 ± 2.1
Duration of heavy drinking (years)	21.6 ± 7.6
Number of drinks during heavy drinking	
Daily average	14.3 ± 5.4
Maximum	24.3 ± 9.3

Neuropsychological studies in these patients revealed that, although the mean IQ level was within normal limits, performance on the Halstead Impairment Index, Halstead Category Test, and Wisconsin Card Sorting Test were within the impaired range (table 2). Correlations of lCMRglc with neuropsychological tests were undertaken by subdividing the frontal lobes into three principal regions: medial frontal, dorsolateral, and orbitomedial (table 3). The results revealed significant correlations between lCMRglc in the dorsolateral and orbitomedial regions and performance on both the Halstead Category Test (VII) and the Wisconsin Card Sorting Test (Categories Achieved). In contrast, lCMRglc in the medial frontal cortex was correlated only with performance on the Wisconsin Card Sorting Test (Perseverative Errors).

Cerebral Benzodiazepine Neurotransmitter Receptors in Severe Chronic Alcoholism

Gamma-aminobutyric acid (GABA) is an inhibitory neurotransmitter found in many structures within the central nervous system (Matsumoto 1989; Haefely 1990). Two types of receptors mediate GABAergic neurotransmission, $GABA_A$ and $GABA_B$. The $GABA_A$ receptor, a membrane glycoprotein

Table 2. Neuropsychological Test Results in 37 Patients With Severe Chronic Alcoholism.

Variable	Mean	Comment
IQ (WAIS-R)	96.2 (± 11.6)	Within normal limits
Halstead Impairment Index	0.6	Moderate level of impairment
Halstead Category Test (Errors)	71.5	Impaired range
Wisconsin Card Sorting Test		
Categories Achieved	4	Impaired range
Perseverative Errors	20	Impaired range

Note: WAIS-R = Wechsler Adult Intelligence Scale—Revised. WAIS-R result is expressed as mean ± standard deviation.

Table 3. Correlations of Neuropsychological Test Results With Local Cerebral Metabolic Rates for Glucose Studied With [^{18}F]fluorodeoxyglucose and Positron Emission Tomography in Patients With Severe Chronic Alcoholism.

Frontal Lobe Subdivision	Halstead Category Test (VII) $n = 59$	Wisconsin Card Sorting Test (Categories) $n = 34$	Wisconsin Card Sorting Test (Perseverative Errors) $n = 34$
Medial frontal	0.08	0.23	-0.45**
Dorsolateral	-0.42***	0.43**	-0.14
Orbitomedial	-0.41***	0.51**	-0.22

Note: Pearson product-moment correlations: **$p < 0.01$; ***$p < 0.001$.

complex belonging to the family of receptor-operated ion channels (Haefely 1990; Olsen et al. 1990), contains several functionally coupled binding sites for pharmacologically specific agents that modulate GABA receptor-chloride channel function (Olsen 1981). These include sites for binding benzodiazepines, barbiturates, and picrotoxin-like convulsants. Located on the oligomeric protein complex that regulates ion permeability, the binding sites are functionally coupled (Matsumoto 1989), and the benzodiazepine, barbiturate, and convulsant receptors appear to be allosterically linked to low-affinity GABA agonist sites (Olsen et al. 1990). These sites are found mostly in layer V of the cerebral cortex, cingulate cortex, hippocampal region, amygdala, anterior and medial hypothalamus, superior colliculus, substantia nigra pars reticulata, periaqueductal gray, and molecular layer of the cerebellum (McCabe and Wamsley 1986). Among other functions, benzodiazepine receptors are thought to be important in influencing anxiety in humans (Smith and Wesson 1985).

Ethanol activates a number of specific receptors and strongly influences $GABA_A$ receptors, stimulating chloride ion flux through channels activated by GABA (Suzdak et al. 1986*b*). Some actions of ethanol may result from increased function of the benzodiazepine-sensitive $GABA_A$/chloride channel complex (Harris and Allan 1987). GABA agonists enhance the effects of ethanol, and both GABA antagonists and GABA inverse agonists reduce ethanol intoxication (Suzdak et al. 1986*a*; Harris and Allan 1987). Chronic exposure to ethanol in experimental animals significantly decreases $GABA_A$ receptor-mediated chloride uptake in cerebral cortical synaptoneurosomes (Morrow et al. 1990). Muscimol and pentobarbital stimulation and ethanol augmentation of muscimol-stimulated chloride flux are decreased with chronic exposure to ethanol. These findings suggest that chronic exposure to ethanol may decrease the level of mRNAs coding for the alpha subunit of the $GABA_A$ receptor. This decrease may reflect altered processing of mRNA encoding $GABA_A$ receptor proteins.

The $GABA_A$/benzodiazepine ($GABA_A$/BDZ) receptor may be important in the pathogenesis of chronic alcoholism. One hypothesis is that central GABAergic hypofunctioning, due to genetic inheritance and/or the result of chronic ethanol intoxication, may lead to further alcohol consumption (Ollat et al. 1988). Thus, an individual may consume alcohol to restore GABAergic neurotransmission and to prevent anxiety. If this notion is correct, a reduced number of GABA neurons or of $GABA_A$/BDZ receptors could decrease the capacity to modulate anxiety in chronically alcohol-dependent persons. This is in keeping with clinical appraisals of severe chronic alcoholics indicating that they have decreased affective control and defective anxiety management (Grant et al. 1986).

We used PET with [^{11}C]FMZ to study $GABA_A$/BDZ receptors and, in the same scanning session, [^{18}F]FDG to examine lCMRglc in 17 males with severe chronic alcoholism—8 with ACD and 9 without ACD (table 4)

(Gilman et al. 1996*c*). In comparison with male normal controls of similar ages, the severe chronic alcoholism group had significantly reduced FMZ ligand influx (K_1), FMZ distribution volume, and lCMRglc bilaterally in the medial frontal lobes, including superior frontal gyrus and rostral cingulate gyrus; the ACD group had significant reductions of K_1, distribution volume, and lCMRglc bilaterally in the same distribution, and also in the superior cerebellar vermis; and the non-ACD group had significant reductions of K_1, distribution volume, and lCMRglc bilaterally in the same regions of the frontal lobes, but not in the superior cerebellar vermis (tables 5 and 6). When compared with the non-ACD group, the ACD group had significant reductions of

Table 4. Subjects for Studies of Benzodiazepine Receptor Binding With [^{11}C]flumazenil (FMZ) and Local Cerebral Metabolic Rates for Glucose With [^{18}F]fluorodeoxyglucose (FDG) and Positron Emission Tomography in Severe Chronic Alcoholism (ALC).

	ALC Subjects		Normal Controls	
	non-ACD	ACD	FDG Studies	FMZ Studies
n	9	8	15	14
Ages (± SD)	49 ± 10	54 ± 6	50 ± 11	46 ± 13

Note: ACD = alcoholic cerebellar degeneration.

Table 5. Local Cerebral Metabolic Rates for Glucose (lCMRglc) in the Cerebral Cortex and Cerebellum Detected With [^{18}F]fluorodeoxyglucose (FDG) and Positron Emission Tomography (PET) in Severe Chronic Alcoholism.

	FDG lCMRglc	
Volume of Interest	non-ACD	ACD
Medial cerebral cortex area 1[a]	0.966	0.951[c]
Medial cerebral cortex area 2[a]	0.960	0.948[c]
Medial cerebral cortex area 3[a,b]	1.007	0.969
Medial cerebral cortex area 4	0.978	1.010
Medial cerebral cortex area 5	0.978	1.053
Frontal convexity	0.974	1.027
Parietal convexity	0.981	1.017
Superior cerebellar vermis[a,b]	1.125[c]	0.847[c,d]
Inferior cerebellar vermis[a]	1.116[c]	0.925[d]
Cerebellar cortex	1.072[c]	1.047

Note: ACD = alcoholic cerebellar degeneration. Cerebral cortical areas 1–3 are in the medial aspects of the frontal lobes; areas 4 and 5 are in the parietal lobe.
[a] Two-factor analysis of variance (ANOVA) significant for differences across groups at $p < 0.05$.
[b] Two-factor ANOVA significant for differences across PET measures at $p < 0.05$.
[c] Two-tailed *t* test significantly different from normal control at $p < 0.05$.
[d] Two-tailed *t* test significantly different between non-ACD and ACD groups at $p < 0.05$.

Table 6. Benzodiazepine Receptor Binding in the Cerebral Cortex and Cerebellum Studied With [^{11}C]flumazenil (FMZ) and Positron Emission Tomography (PET) in Severe Chronic Alcoholism.

	FMZ K_1		FMZ DV	
Volume of Interest	**non-ACD**	**ACD**	**non-ACD**	**ACD**
Medial cerebral cortex area 1[a]	0.954	0.949	0.965	0.961
Medial cerebral cortex area 2[a]	0.926[c]	0.918[a]	0.938[c]	0.925[c]
Medial cerebral cortex area 3[a,b]	0.972	0.927[c,d]	0.937[c]	0.931[c]
Medial cerebral cortex area 4	0.984	1.011	0.992	1.003
Medial cerebral cortex area 5	0.991	1.029	1.029	1.033
Frontal convexity	0.999	1.040	1.013	1.042
Parietal convexity	0.997	1.046	1.021	1.053
Superior cerebellar vermis[a,b]	1.074	0.788[c,d]	0.950	0.772[c,d]
Inferior cerebellar vermis[a]	1.071	0.890[d]	0.993	0.855[c,d]
Cerebellar cortex	1.038	0.976	0.932	1.027

Note: K_1 = ligand influx; DV = distribution volume; ACD = alcoholic cerebellar degeneration. Cerebral cortical areas 1–3 are in the medial aspects of the frontal lobes; areas 4 and 5 are in the parietal lobe.
[a] Two-factor analysis of variance (ANOVA) significant for differences across groups at $p < 0.05$.
[b] Two-factor ANOVA significant for differences across PET measures at $p < 0.05$.
[c] Two-tailed *t* test significantly different from normal control at $p < 0.05$.
[d] Two-tailed *t* test significantly different between non-ACD and ACD groups at $p < 0.05$.

K_1 and distribution volume bilaterally in the superior cerebellar vermis. The results suggest that severe chronic alcoholism damages neurons containing $GABA_A$/BDZ receptors in the superior medial aspects of the frontal lobes and, in patients with clinical signs of ACD, neurons containing $GABA_A$/BDZ receptors in the superior cerebellar vermis.

Cerebral Dopaminergic Neurons in Severe Chronic Alcoholism

Although conflicting information has been published, studies of human postmortem tissue have raised the possibility that, even in the absence of Wernicke-Korsakoff syndrome, severe chronic alcoholism damages not only the cerebral cortex and cerebellum, but also many subcortical neurons, including several nuclear groups utilizing different neurotransmitters such as serotonin, norepinephrine, GABA, and dopamine (Hunt and Nixon 1993). These data have been supported by studies in experimental animals. In an immunohistochemical examination of the brain stems of alcoholic patients, a significant reduction of serotonergic neurons was found in the brain stem raphe nucleus not only in patients with Wernicke-Korsakoff syndrome, but also in a single patient with otherwise uncomplicated severe chronic alcoholism (Halliday et al. 1995). This finding appears now to be incorrect, as the same group reported that study of eight alcoholic subjects without Wernicke-Korsakoff syndrome failed to replicate these findings, but the investigators did find decreased staining intensity of the reaction product in serotonergic

neurons (Baker et al. 1996). Thus, any effects of chronic alcoholism on neurons of the serotonergic system appear to be functional rather than neuropathological (Baker et al. 1996). The notion that alcoholism adversely affects serotonin neurons has been buttressed by studies in experimental animals receiving alcohol showing decreased cerebral levels of serotonin and selective changes in serotonin receptor populations (McBride et al. 1993*a*; Wong et al. 1983).

Postmortem studies have given evidence that noradrenergic neurons are affected in chronic alcoholics, but these studies have proved controversial. Neuronal cell counts in the locus coeruleus in patients with severe chronic alcoholism were reported as decreased (Arango et al. 1994), and reduced norepinephrine concentrations were found in the hippocampus and cingulate cortex of chronic alcoholics (Carlsson et al. 1980). The finding of decreased neurons in the locus coeruleus has not been replicated by an independent group of investigators (Halliday et al. 1992; Baker et al. 1994), who suggest that the previous findings were spurious and resulted from inclusion of a group of young controls (Halliday and Baker 1996).

Human postmortem studies of the effects of severe chronic alcoholism upon dopaminergic neurons are not available, but experimental work in animals suggests that alcohol consumption strongly influences the dopamine neurons that project to the striatum and limbic system. In rats, ethanol increases the extracellular levels of dopamine (Imperato and DiChiara 1986; Khatib et al. 1988; Fadda et al. 1989), and withdrawal from ethanol reduces dopamine outflow during the first 24 hours (Rossetti et al. 1992). Chronic forced ethanol consumption reduces the densities of D_2 receptors in the caudate nucleus, medial and lateral nucleus accumbens, and olfactory tubercle 24 hours after ethanol withdrawal (Rommelspacher et al. 1992), probably because of down-regulation of these receptors due to chronically increased extracellular dopamine. Experiments in selectively bred lines of alcohol-preferring rats suggest that the dopamine system may regulate alcohol-drinking behavior (Gessa et al. 1985; Fadda et al. 1989; McBride et al. 1991; Weiss et al. 1992). Voluntary ingestion of ethanol by the Sardinian alcohol-preferring (P) line increased the tissue levels of the dopamine metabolite 3,4-dihydroxyphenylacetic acid in the caudate nucleus, medial prefrontal cortex, and olfactory tubercle, indicating activation of the substantia nigra and the ventral tegmental area (VTA) dopamine systems (Fadda et al. 1989). Moreover, oral administration of ethanol under operant responding conditions increased the synaptic levels of dopamine significantly more in the nucleus accumbens of the P line of rats than in unselected Wistar rats, suggesting that the VTA dopamine system of the P line is sensitive to the rewarding effects of alcohol (Weiss et al. 1992). The P line of rats, but not the NP line (alcohol-nonpreferring), self-administers alcohol directly into the VTA, probably activating VTA dopamine neurons (McBride et al. 1991; Gessa et al. 1985). Microinjection

of the dopamine D_2 antagonist sulpiride into the nucleus accumbens increases the alcohol consumption of the P line of rats (Levy et al. 1991). The nucleus accumbens and olfactory tubercle of the P rats contain approximately 25–30 percent lower concentrations of dopamine than NP rats, suggesting an innate difference in the VTA dopamine system of the P rat and indicating that this abnormality may be associated with high alcohol-drinking behavior (Murphy et al. 1987; McBride et al. 1993*b*). Dopaminergic innervation is also decreased in the cingulate cortex of the P line of rats (Zhou et al. 1995). In keeping with these findings, the densities of the dopamine D_2 receptor sites are significantly lower in the caudate-putamen, medial and lateral nucleus accumbens, and VTA of the P line than the NP line of rat (Stefanini et al. 1992; McBride et al. 1993*b*).

The findings in studies of experimental animals are intriguing and were sufficiently compelling for us to initiate a pilot project to examine the density of dopaminergic terminals in the striatum of severe chronic alcoholic patients. We are using PET to examine the binding of (+)[^{11}C] DTBZ, an agent that binds to the VMAT2 (Frey et al. 1996; Gilman et al. 1996*b*). Thus far we have studied four normal control subjects and six patients with severe chronic alcoholism. All subjects were male, and the groups were in a similar age distribution (normal controls 61 ± 9 years, alcoholic subjects 55 ± 9 years [mean ± standard deviation]). Both ligand influx (K_1) and ligand distribution volume normalized to the cerebral cortex were significantly decreased in the putamen but not the caudate nucleus or thalamus of the alcoholic subjects as compared with the controls (table 7). Plasma protein levels in both the chronic alcoholic patients (plasma free fraction = 0.22 ± 0.03) and the normal control subjects (0.28 ± 0.10) were within normal limits, indicating that differences in plasma proteins within the subject groups cannot account for the differences found with PET. These preliminary data suggest that the density of monoaminergic terminals is signifi-

Table 7. Ligand Influx (K_1) and Ligand Distribution Volume (DV) From Studies of (+)[^{11}C]dihydrotetrabenazine in Normal Control Subjects Compared With Severe Chronic Alcoholic Patients.

Structure	Controls (n = 4) K_1	Alcoholics (n = 6) K_1	Controls (n = 4) DV	Alcoholics (n = 6) DV
Caudate nucleus	1.12 ± 0.01	1.05 ± 0.09	3.86 ± 0.21	3.70 ± 0.30
Putamen	1.33 ± 0.03	1.23 ± 0.05**	4.01 ± 0.29	3.54 ± 0.36*
Thalamus	1.45 ± 0.08	1.45 ± 0.16	1.30 ± 0.07	1.35 ± 0.09

Note: Both K_1 and DV values were normalized to the cerebral cortex of the whole brain (mean ± standard error). The normalized data were computed directly from the volumes of interest.
*$p < 0.03$; **$p < 0.002$.

cantly diminished in the putamen of the chronic alcoholic subjects, although the significantly decreased normalized K_1 level in the alcoholic group raises the possibility that tissue atrophy in the alcoholic subjects contributes to the findings. The global K_1 values were elevated in the alcoholic group compared with the control subjects, but this may be an artifact of the small number of observations. We have found no other evidence of a disruption of the blood-brain barrier in alcoholic subjects in previous studies, including examination of K_1 values in studies with [^{11}C]FMZ, which were not elevated in the alcoholic group. Moreover, since studies with [^{18}F]FDG measure both transport and phosphorylation effects and lCMRglc is not globally increased in alcoholic subjects, any conclusion concerning global K_1 values at this time would be premature.

Upper Limb Coordination in ACD

Alcoholic cerebellar degeneration results in severe ataxia of lower extremity movements with a marked disturbance of gait and impairment on tests of coordinated movement such as the heel-knee-shin test and rapidly alternating movements of the feet. Involvement of the upper extremities is usually so mild as to be unnoticed, although some patients with this disorder develop difficulties with handwriting and with other motor functions requiring deftness of coordination. In a retrospective study to quantify the abnormalities of upper limb motor performance, we compared the results of the Tactual Performance Test in 13 patients with ACD with those in 43 severe chronic alcoholic patients without ACD. The results showed a significant abnormality of the Tactual Performance Test in total time, though not in memory or location, in the ACD group compared with the non-ACD group (table 8) (Johnson-Greene et al. 1996*a*). These findings indicate that, in addition to the severe ataxia of movements of the lower extremities, ACD does lead to a significant impairment of upper extremity coordination.

Effects of Abstinence and Relapse on Neuropsychological Performance and Local Cerebral Metabolism of Glucose in Severe Chronic Alcoholism

Several studies suggest that at least partial recovery of cerebral cortical function can occur after cessation of alcohol use in patients with severe chronic alcoholism. Anatomical imaging studies have shown increases in the volume of the white matter following 3 months of abstinence (Shear et al. 1994), and continued increases thereafter (Carlen et al. 1978). Similarly, studies with PET have shown positive correlations between lCMRglc in the whole brain and the length of time after cessation of alcohol consumption (Volkow et al. 1992, 1994), and studies with single photon emission computed tomography have shown increases in cerebral blood flow following abstinence (Nicholas et al. 1993). We have begun to explore the effects of abstinence and relapse upon lCMRglc and neuropsychological performance in a small cohort of patients within a pilot

Table 8. Neuropsychological Test Performance in Severe Chronic Alcoholic Patients With Alcoholic Cerebellar Degeneration (ACD) and Without ACD (non-ACD).

Alcoholic Group	WAIS-R	Halstead-Reitan Impairment Index	TPT Total Time	TPT Memory	TPT Location
ACD $n = 13$	97.8±11	0.66±0.21*	24.8±7.5**	6.5±2.0	2.7±1.6
non-ACD $n = 43$	97.4±10	0.49±0.29	16.5±6.2	7.0±1.7	3.3±2.1

Note: WAIS-R = Wechsler Adult Intelligence Scale—Revised; TPT = Tactual Performance Test.
**$p < 0.001$; *$p < 0.02$.

Table 9. Effects of Abstinence on Neuropsychological Performance and Local Cerebral Metabolic Rates for Glucose Detected With Positron Emission Tomography.

Patient	Interscan Interval (months)	Orbitomedial % Change	Medial frontal % Change	Dorsolateral % Change	WAIS-R	H-R II	HCT
1	32	6.4	2.8	0.2	0	0.3	40
2*	10	-0.9	-1.1	-3.8	2	-0.1	16
3	11	10.2	3.2	-2.0	10	0.0	53
4*	27	-3.0	-4.0	1.0	10	0.0	-3
5	20	0.4	0.6	2.8	2	0.5	23
6	12	8.8	1.1	-0.4	5	0.3	17

Note: WAIS-R = Wechsler Adult Intelligence Scale–Revised; H-R II = Halstead-Reitan Neuropsychological Test Battery—II; HCT = Halstead Category Test.
* Patients with repeated relapses during the interscan interval.

study (Johnson-Greene et al. 1996*b*). We examined six patients with severe chronic alcoholism at two intervals ranging from 10 to 32 months (table 9). Four of the patients remained almost completely abstinent, and two relapsed after their initial evaluation. The patients who remained abstinent showed partial recovery of lCMRglc in two of the three divisions of the frontal lobes and improvement on tasks of executive function, but the patients who relapsed showed further declines in these areas. The results indicate the potential for at least some recovery in patients with severe chronic alcoholism who abstain from alcohol.

Relationship of Family History of Alcoholism With Cerebral Glucose Metabolism and Neuropsychological Performance in Patients With Severe Chronic Alcoholism

Patients with severe chronic alcoholism frequently have first-degree relatives with a history of alcohol abuse or alcohol dependence (Merikangas et al. 1985; Merikangas 1990). Several studies have suggested that people with an

alcohol-dependent first-degree relative have neuropsychological abnormalities predating the onset of alcoholism (Goodwin 1983; Schaeffer et al. 1984; Tarter et al. 1984; Moss et al. 1995; Ozkaragoz and Noble 1995). In an attempt to detect differences that might reflect the genetic susceptibility of the severe chronic alcoholic subjects we have studied, we compared the neuropsychological profiles and lCMRglc patterns of 27 patients with a first-degree alcoholic relative with 21 patients with no first-degree alcoholic relative (K.M. Adams et al. 1998). The results revealed no differences between groups for any of the variables studied, possibly because the severity of the disorders equalized any premorbid differences that may have existed between the groups.

Effects of Disulfiram on PET and Neuropsychological Studies in Severe Chronic Alcoholism

Disulfiram is an aldehyde dehydrogenase inhibitor that is frequently used as an adjunctive treatment in patients with severe chronic alcoholism. Some of the alcoholic patients in our recent PET studies of lCMRglc and benzodiazepine receptor binding were receiving disulfiram, and others were not receiving this agent. Since administration of disulfiram could influence the biochemical processes under investigation, we examined the effects of disulfiram on the results of PET studies of lCMRglc and benzodiazepine receptor binding and neuropsychological tests of cognition and executive function in patients with severe chronic alcoholism. PET with [^{18}F]FDG was used to measure lCMRglc in 48 male patients, including 11 receiving and 37 not receiving disulfiram in therapeutic doses. Flumazenil was used to measure benzodiazepine receptor binding in 17 male patients, including 3 receiving and 14 not receiving disulfiram. All patients studied with [^{11}C]FMZ were also examined with [^{18}F]FDG. PET studies of lCMRglc revealed significantly decreased global values in the patients receiving disulfiram as compared with those not receiving disulfiram. PET studies of benzodiazepine receptor binding revealed decreased [^{11}C]FMZ influx and distribution volume in patients receiving disulfiram. The neuropsychological tests demonstrated no differences between the two groups of subjects. The findings suggest that disulfiram may influence the results of PET studies of glucose metabolism and benzodiazepine receptor binding.

SUMMARY AND CONCLUSIONS

Substantial evidence has accumulated indicating that severe chronic alcoholism damages the central nervous system, even in patients without distinctive syndromes such as ACD or Wernicke-Korsakoff syndrome. Our studies, in concert with investigations in other centers, have demonstrated abnormalities in glucose metabolism in the medial aspects of the frontal lobes. Associated abnormalities of binding to benzodiazepine receptors have been found in a similar location. Moreover,

our investigations suggest that the focal area of abnormality is correlated with some of the neuropsychological disorders, particularly those characterized as executive functions. In patients with symptomatic ACD, both glucose metabolism and benzodiazepine receptor binding are abnormally decreased in the vermal portions of the cerebellum. In severe chronic alcoholic patients without symptomatic ACD, however, no abnormalities can be detected in the cerebellum. The injury to the central nervous system is focal, with particular vulnerability in the mesial frontal lobes and the superior cerebellar vermis. The reason for the particular vulnerabilities of these sites is not clear. Although abnormalities of benzodiazepine receptors have been demonstrated, we doubt that these receptors are more vulnerable than others in these locations; studies to verify this notion have not been performed yet.

Recent studies have provided suggestive evidence that the devastating effects of severe chronic alcoholism on the nervous system may occur not only in the cerebral cortex and cerebellum, but also in subcortical structures. Preliminary studies in our laboratories suggest that the dopaminergic projections from the substantia nigra to the striatum may be affected as well. Although involvement of noradrenergic and serotonergic projections is well established in experimental animals, preliminary studies suggesting involvement in humans are inconclusive. These studies indicate that, even in patients with severe chronic alcoholism, abstinence may result in at least partial reversibility of both the metabolic disorders and the neuropsychological abnormalities.

We plan to continue programmatic investigations of alcoholic patients using neurobehavioral and neuroimaging techniques to contribute to a fuller understanding of the cerebral consequences of chronic alcoholism.

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REFERENCES

Adams, K.M.; Grant, I.; Carlin, A.; and Reed, R. Self-reported alcohol consumption in four clinical groups. *Am J Psychiatry* 138:445–449, 1979.

Adams, K.M.; Gilman, S.; Koeppe, R.A.; et al. Neuropsychological deficits are correlated with frontal hypometabolism in positron emission tomography studies of older alcoholic patients. *Alcohol Clin Exp Res* 17:205–210, 1993.

Adams, K.M.; Gilman, S.; Koeppe, R.; et al. Correlation of neuropsychological function with cerebral metabolic rate in subdivisions of the frontal lobes of older alcoholic patients measured with [^{18}F]fluorodeoxyglucose and positron emission tomography. *Neuropsychology* 9:275–280, 1995*a*.

Adams, K.M.; Gilman, S.; Koeppe, R.; et al. Decreased benzodiazepine receptor

binding in the cingulate cortex of chronic alcoholic patients measured with [^{11}C]flumazenil and PET. *Alcohol Clin Exp Res* 19:10A, 1995*b*.

Adams, K.M.; Gilman, S.; Johnson-Greene, D.; Koeppe, R.; Junck, L.; Kluin, K.J.; Martello, S.; Heumann, M.; and Hill, E. The significance of family history status in relation to neuropsychological test performance and cerebral glucose metabolism studied with positron emission tomography in older alcoholic patients. *Alcohol Clin Exp Res* 22:105–110, 1998.

Adams, R.D.: Nutritional cerebellar degeneration. In: Vinken, P.J., and Bruyn, G.W., eds. *Handbook of Clinical Neurology*. Vol. 28. Amsterdam: North-Holland, 1976. pp. 271–283.

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 3d ed., rev. Washington, DC: the Association, 1987.

Arango, V.; Underwood, M.D.; and Mann, J.J. Fewer pigmented neurons in the locus coeruleus of uncomplicated alcoholics. *Brain Res* 650:1–8, 1994.

Baker, K.G.; Halliday, G.M.; and Harper, C.G. Effect of chronic alcohol consumption on the human locus coeruleus. *Alcohol Clin Exp Res* 18:1491–1496, 1994.

Baker, K.G.; Halliday, G.M.; Kril, J.J.; and Harper, C.G. Chronic alcoholics without Wernicke-Korsakoff syndrome or cirrhosis do not lose serotonergic neurons in the dorsal raphe nucleus. *Alcohol Clin Exp Res* 20:61–66, 1996.

Carlen, P.L.; Wortzman, G.; Holgate, R.C.; et al. Reversible cerebral atrophy in recently abstinent chronic alcoholics measured by computerized tomography scans. *Science* 200:1076–1078, 1978.

Carlen, P.L.; Wilkinson, D.A.; Wortzman, G.; et al. Cerebral atrophy and functional deficits in alcoholics without clinically apparent liver disease. *Neurology* 31:377–385, 1981.

Carlen, P.L.; Penn, R.K.; Fornazzari, L.; et al. Computerized tomographic scan assessment of alcoholic brain damage and its potential reversibility. *Alcohol Clin Exp Res* 10:226–232, 1986.

Carlsson, A.; Adolfsson, R.; Aquilonius, S.M.; et al. Biogenic amines in human brain in normal aging, senile dementia and chronic alcoholism. In: Goldstein, M., et al., eds. *Ergot Compounds and Brain Function: Neuroendocrine and Neuropsychiatric Aspects*. New York: Raven Press, 1980. pp. 295–304.

Charness, M.E.; Simon, R.P.; and Greenberg, D.A. Ethanol and the nervous system. *N Engl J Med* 321:442–454, 1989.

Courville, C.B. *Effects of Alcohol in the Nervous System of Man*. Los Angeles: San Lucas Press, 1955.

Fadda, F.; Mosca, E.; Colombo, G.; and Gessa, G.L. Effect of spontaneous ingestion of ethanol on brain dopamine metabolism. *Life Sci* 44:281–287, 1989.

Ferrer, I.; Fabregues, I.; Rairiz, J.; Galofre, E. Decreased numbers of dendritic spines on cortical pyramidal neurons in human chronic alcoholism. *Neurosci Lett* 69:115–119, 1986.

Frey, K.A.; Holthoff, V.A.; Koeppe, R.A.; et al. Parametric in vivo imaging of benzodiazepine receptor distribution in human brain. *Ann Neurol* 30:663–672, 1991.

Frey, K.A.; Koeppe, R.A.; Kilbourn, M.R.; Vander Borght, T.M.; Albin, R.A.; Gilman, S.; and Kuhl, D.E. Presynaptic monoaminergic vesicles in Parkinson's

disease and normal aging. *Ann Neurol* 40:873–884, 1996.

Gessa, G.L.; Muntoni, F.; Collu, M.; et al. Low doses of ethanol activate dopaminergic neurons in the ventral tegmental area. *Brain Res* 348:201–203, 1985.

Gilman, S.; Adams, K.; Koeppe, R.A.; et al. Cerebellar and frontal hypometabolism in alcoholic cerebellar degeneration studied with positron emission tomography. *Ann Neurol* 28:775–785, 1990.

Gilman, S.; Adams, K.M.; Johnson-Greene, D.; Koeppe, R.A.; Junck, L.; Kluin, K.; Martorello, S.; Heumann, M.; and Hill, E. Effects of disulfiram on positron emission tomography and neuropsychological studies in severe chronic alcoholism. *Alcohol Clin Exp Res* 8:1456–1461, 1996*a*.

Gilman, S.; Frey, K.A.; Koeppe, R.A.; Junck, L.; Little, R.; Vander Borght, T.M.; Lohman, M.; Martorello, S.; Lee, L.C.; Jewett, D.M.; and Kilbourn, M.R. Decreased striatal monoaminergic terminals in olivopontocerebellar atrophy and multiple system atrophy demonstrated with positron emission tomography. *Ann Neurol* 40:885–892,1996*b*.

Gilman, S.; Koeppe, R.A.; Adams, K.; Johnson-Greene, D.; Junck, L.; Kluin, K.J.; Brunberg, J.; Martorello, S.; and Lohman, M. Positron emission tomography studies of cerebral benzodiazepine receptor binding in chronic alcoholics. *Ann Neurol* 40:163–171, 1996*c*.

Goodwin, D. Familial alcoholism: A separate entity. *Subst Alcohol Actions Misuse* 4:129–136, 1983.

Grant, I.; Adams, K.M.; and Reed, R. Intermediate-duration (subacute) organic mental disorder of alcoholism. In: Grant, I., ed. *Neuropsychiatric Correlates of Alcoholism*. Washington, DC: American Psychiatric Press, 1986.

Haefely, W.E. The GABA-benzodiazepine interaction fifteen years later. Neurochem Res 15:169–174, 1990.

Halliday, G., and Baker, K. Noradrenergic locus coeruleus neurons. *Alcohol Clin Exp Res* 20:191–192, 1996.

Halliday, G.M.; Ellis, J.; and Harper, C. The locus coeruleus and memory: A study of chronic alcoholics with and without the memory impairment of Korsakoff's psychosis. *Brain Res* 598:33–37, 1992.

Halliday, G.; Baker, K.; and Harper, C. Serotonin and alcohol-related brain damage. *Metab Brain Dis* 10:25–30, 1995.

Harper, C., and Corbett, D. Changes in basal dendrites of cortical pyramidal cells from alcoholic patients—a quantitative Golgi study. *J Neurol Neurosurg Psychiatry* 53:856–861, 1990.

Harper, C.G., and Kril, J. Brain atrophy in chronic alcoholic patients: A pathological study. *J Neurol Neurosurg Psychiatry* 48:211–217, 1985.

Harper, C.G., and Kril, J. Patterns of neuronal loss in the cerebral cortex in chronic alcoholic patients. *J Neurol Sci* 92:81–89, 1989.

Harper, C.G., and Kril, J. Neuropathology of alcoholism. *Alcohol Alcohol* 25:207–216, 1990.

Harper, C., and Kril, J. If you drink your brain will shrink. Neuropathological considerations. *Alcohol Alcohol Suppl* 1:375–380, 1991.

Harper, C.G.; Kril, J.; and Daly, J. Are we drinking our neurones away? *Br Med J* 294:534–536, 1987.

Harris, R.A., and Allan, A.M. Involvement of neuronal chloride channels in ethanol intoxication, tolerance and dependence. In: Galanter, M., ed. *Recent Developments in Alcoholism.* Vol. 5. New York: Plenum, 1987. pp. 313–325.

Heaton, R.K.; Grant, I.; and Matthews, C.G. *Comprehensive Norms for an Expanded Halstead-Reitan Battery: Demographic Corrections, Research Findings, and Clinical Applications.* Odessa, FL: Psychological Assessment Resources, 1991.

Holthoff, V.A.; Koeppe, R.A.; Frey, K.A.; et al. Differentiation of radioligand delivery and binding in the brain: Validation of a two-compartment model for [^{11}C]flumazenil. *J Cereb Blood Flow Metabol* 11:745–752, 1991.

Hunt, W.A., and Nixon, S.J., eds. *Alcohol-Induced Brain Damage.* National Institute on Alcohol Abuse and Alcoholism Research Monograph 22. NIH Pub. No. 93–3549. Bethesda, MD: National Institutes of Health, 1993.

Hutchins, G.D.; Holden, J.E.; Koeppe, R.A.; et al. Alternative approach to single-scan estimation of cerebral glucose metabolic rate using glucose analogs, with particular application to ischemia. *J Cereb Blood Flow Metab* 4:35–40, 1984.

Imperato, A., and DiChiara, G. Preferential stimulation of dopamine release in the nucleus accumbens of freely moving rats by ethanol. *J Pharmacol Exp Ther* 239:219–228, 1986.

Jagust, W.J. PET and SPECT imaging in cognitive disorders of aging and alcoholism. In: Zakhari, S., and Witt, E., eds. *Imaging in Alcohol Research.* National Institute on Alcohol Abuse and Alcoholism Research Monograph 21. Washington, DC: U.S. Department of Health and Human Services, 1992. pp. 333–358.

Jernigan, T.L.; Butters, N.; DiTraglia, G.; Schafer, K.; Smith, T.; Irwin, M.; Grant, I.; Schuckit, M.; and Cermak, L.S. Reduced cerebral grey matter observed in alcoholics using magnetic resonance imaging. *Alcohol Clin Exp Res* 15:418–427, 1991.

Johnson-Greene, D.; Adams, K.M.; Gilman, S.; Kluin, K.; Junck, L.; Martorello, S.; and Heumann, M. Impaired upper limb coordination in alcoholic cerebellar degeneration. *Arch Neurol* 54:436–439, 1997*a*.

Johnson-Greene, D.; Adams, K.M.; Gilman, S.; Koeppe, R.A.; Junck, L.; Kluin, K.; Martorello, S.; Heumann, M.; and Hill, E. Effects of abstinence and relapse upon neuropsychological function and cerebral glucose metabolism in severe chronic alcoholism. *J Clin Exp Neuropsychol* 19:378–385, 1997*b*.

Khatib, S.A.; Murphy, J.M.; and McBride, W.J. Biochemical evidence for activation of specific monoamine pathways by ethanol. *Alcohol* 5:295–299, 1988.

Koeppe, R.A.; Holthoff, V.A.; Frey, K.A.; et al. Compartmental analysis of [^{11}C]flumazenil kinetics for the estimation of ligand transport rate and receptor distribution using positron emission tomography. *J Cereb Blood Flow Metab* 1:735–744, 1991.

Koeppe, R.A.; Frey, K.A.; Vander Borght, T.M.; et al. Kinetic evaluation of [C-11]dihydrotetrabenazine (DTBZ) by dynamic PET: A marker for the vesicular monoamine transporter. *J Cereb Blood Flow Metab* 15:S651 (abstract), 1995.

Kril, J., and Harper, C.G. Neuronal counts from four cortical regions of alcoholic brains. *Acta Neuropathol* 79:200–204, 1989.

Levy, A.D.; Murphy, J.M.; McBride, W.J.; et al. Microinjection of sulpiride into the

nucleus accumbens increases ethanol drinking in alcohol-preferring (P) rats. *Alcohol Alcohol Suppl* 1:417–420, 1991.

Lezak, M. *Neuropsychological Assessment.* 2d ed. New York: Oxford University Press, 1986.

Lieber, C.S. Medical disorders of alcoholism. *N Engl J Med* 333:1058–1065, 1995.

Matsumoto, R.R. GABA receptors: Are cellular differences reflected in function? *Brain Res Rev* 14:203–225, 1989.

Mazière, M.; Hantraye, P.; Prenant, C.; et al. Synthesis of RO 1788 with 11C: A specific radioligand for the in vivo study of central benzodiazepine receptors by positron emission tomography. *Int J Appl Radiat Isot* 35:973–976, 1984.

McBride, W.J.; Murphy, J.M.; Gatto, G.J.; Levy, A.D.; Lumeng, L.; and Li, T.-K. Serotonin and dopamine systems regulating alcohol intake. *Alcohol Alcohol Suppl* 1:411–416, 1991.

McBride, W.J.; Chernet, E.; Rabold, J.A.; et al. Serotonin-2 receptors in the CNS of alcohol-preferring and -nonpreferring rats. *Pharmacol Biochem Behav* 46:631–636, 1993*a*.

McBride, W.J.; Chernet, E.; Dyr, W.; et al. Densities of dopamine D_2 receptors are reduced in CNS regions of alcohol-preferring P rats. Alcohol 10:387–390, 1993*b*.

McCabe, R.T., and Wamsley, J.K. Autoradiographic localization of subcomponents of the macromolecular GABA receptor complex. *Life Sci* 39:1937–1945, 1986.

Merikangas, K.R. The genetic epidemiology of alcoholism. *Psychol Med* 20:11–22, 1990.

Merikangas, K.R.; Leckman, J.F.; Prusoff, B.A.; et al. Familial transmission of depression and alcoholism. *Arch Gen Psychiatry* 42:367–372, 1985.

Morrow, A.L.; Montpied, P.; Lingford-Hughes, A.; and Paul, S.M. Chronic ethanol and pentobarbitol administration in the rat: Effects on $GABA_A$ receptor function and expression in brain. *Alcohol* 7:237–244, 1990.

Moss, H.B.; Vanyukov, M.; Majumder, P.P.; et al. Prepubertal sons of substance abusers: Influence of parental and familial substance abuse on behavioral disposition, IQ, and school achievement. *Addict Behav* 20:345–358, 1995.

Murphy, J.M.; McBride, W.J.; Lumeng, L.; and Li, T.-K. Contents of monoamines in forebrain regions of alcohol-preferring (P) and -nonpreferring (NP) lines of rats. *Pharmacol Biochem Behav* 26:389–392, 1987.

Nicholas, J.M.; Catafau, A.M.; Estruch, R.; et al. Regional cerebral blood flow-SPECT in chronic alcoholism: Relation to neuropsychological testing. *J Nucl Med* 34:1452–1459, 1993.

Ollat, H.; Parvez, H.; and Parvez, S. Review: Alcohol and central neurotransmission. *Neurochem Int* 13:275–300, 1988.

Olsen, R.W. GABA-benzodiazepine-barbiturate receptor interactions. *J Neurochem* 37:1–13, 1981.

Olsen, R.W.; McCabe, R.T.; and Wamsley, J.K. GABAA receptor subtypes: Autoradiographic comparison of GABA, benzodiazepine, and convulsant binding sites in the rat central nervous system. *J Chem Neuroanat* 3:59–76, 1990.

Ozkaragoz, T.Z., and Noble, E.P. Neuropsychological differences between sons of active alcoholic and non-alcoholic fathers. *Alcohol Alcohol* 30:115–123, 1995.

Pfefferbaum, A.; Lim, K.O.; Zipursky, R.B.; et al. Brain gray and white matter volume loss accelerates with aging in chronic alco-

holics: A quantitative MRI study. *Alcohol Clin Exp Res* 16:1078–1089, 1992.

Pfefferbaum, A.; Sullivan, E.V.; Rosenbloom, M.L.; et al. Increase in brain cerebrospinal fluid volume is greater in older than in younger alcoholic patients: A replication study and CT/MRI comparison. *Psychiatry Res* 50:257–274, 1993.

Phillips, S.C.; Harper, C.G.; and Kril, J. A quantitative histological study of the cerebellar vermis in alcoholic patients. *Brain* 110:301–314, 1987.

Reitan, R.M., and Wolfson, D. *The Halstead-Reitan Neuropsychological Test Battery.* Tucson, AZ: Neuropsychology Press, 1986.

Rommelspacher, H.; Raeder, C.; Kaulin, P.; and Bruning, G. Adaptive changes of dopamine-D_2 receptors in rat brain following ethanol withdrawal: A quantitative autoradiographic investigation. *Alcohol* 9:355–362, 1992.

Ron, M.A. The alcoholic brain: CT scan and psychological findings. *Psychol Med Monogr Suppl* 3:1–33, 1983.

Rossetti, Z.L.; Hmaidan, Y.; and Gessa, G.L. Marked inhibition of mesolimbic dopamine release: A common feature of ethanol, morphine, cocaine and amphetamine abstinence in rats. *Eur J Pharmacol* 221:227–234, 1992.

Sachs, H.; Russell, J.A.G.; Christman, D.R.; and Cook, B. Alteration of regional cerebral glucose metabolic rate in non-Korsakoff chronic alcoholism. *Arch Neurol* 44:1242–1251, 1987.

Samson, Y.; Baron, J.C.; Feline, A.; Bories, J.; and Crouzel, C. Local cerebral glucose utilisation in chronic alcoholics: A positron tomography study. *J Neurol Neurosurg Psychiatry* 49:1165–1170, 1986.

Schaeffer, K.W.; Parsons, O.A.; and Yohman, J.R. Neuropsychological differences between male familial and nonfamilial alcoholics and nonalcoholics. *Alcohol Clin Exp Res* 8:347–351, 1984.

Shear, P.K.; Jernigan, T.L.; and Butters, N. Volumetric magnetic resonance imaging quantification of longitudinal brain changes in abstinent alcoholics. *Alcohol Clin Exp Res* 18:172–176, 1994.

Shimamura, A.P.; Jernigan, T.L.; and Squire, L.R. Korsakoff's syndrome: Radiological (CT) findings and neuropsychological correlates. *J Neurosci* 8:4400–4410, 1988.

Smith, D.E., and Wesson, D.R., eds. *The Benzodiazepines: Current Standards for Medical Practice.* Lancaster, England: MTP Press, 1985.

Stefanini, E.; Frau, M.; Garau, M.G.; et al. Alcohol-preferring rats have fewer dopamine D_2 receptors in the limbic system. *Alcohol Alcohol* 27:127–130, 1992.

Sullivan, E.V.; Marsh, L.; Mathalon, D.H.; Lim, K.O.; and Pfefferbaum, A. Anterior hippocampal volume deficits in nonamnesic, aging chronic alcoholics. *Alcohol Clin Exp Res* 19:110–122, 1995.

Suzdak, P.D.; Glowa, J.R.; Crawley, J.N.; et al. A selective imidazobenzodiazepine antagonist of ethanol in the rat. *Science* 234:1243–1247, 1986*a*.

Suzdak, P.D.; Schwartz, R.D.; Skolnick, P.; and Paul, S.M. Ethanol stimulates γ-aminobutryic acid receptor-mediated chloride transport in rat brain synaptoneurosomes. *Proc Natl Acad Sci USA* 83:4071–4075, 1986*b*.

Tarter, R.E.; Hegedus, A.M.; Goldstein, G.; et al. Adolescent sons of alcoholics: Neuropsychological and personality char-

acteristics. *Alcohol Clin Exp Res* 8:216–221, 1984.

Torvik, A.; Torp, S.; and Lindboe, C.F. Atrophy of the cerebellar vermis in ageing. A morphometric and histologic study. *J Neurol Sci* 76:283–294, 1986.

Victor, M., and Ferrendelli, J.A. The nutritional and metabolic diseases of the cerebellum. Clinical and pathological aspects. In: Fields, W.S., and Willis, W.D., eds. *The Cerebellum in Health and Disease*. St. Louis, MO: Green, 1970. pp. 412–449.

Victor, M.; Adams, R.D.; and Mancall, E.L. A restricted form of cerebellar degeneration occurring in alcoholic patients. *Arch Neurol* 1:577–688, 1959.

Victor, M.; Adams, R.D.; and Collins, G.H. *The Wernicke-Korsakoff Syndrome*. 2d ed. Philadelphia: F.A. Davis, 1989.

Volkow, N.D.; Hitzemann, R.J.; Wang, G.J.; et al. Decreased brain metabolism in neurologically intact healthy alcoholics. *Am J Psychiatry* 149:1016–1022, 1992.

Volkow, N.D.; Wang, G.J.; Hitzemann, R.; et al. Decreased cerebral response to inhibitory neurotransmission in alcoholics. *Am J Psychiatry* 150:417–422, 1993.

Volkow, N.D.; Wang, G.J.; Hitzemann, R.; et al. Recovery of brain glucose metabolism in detoxified alcoholics. *Am J Psychiatry* 151:178–183, 1994.

Walsh, K.W. Alcohol related brain damage: An hypothesis. *Aust Drug Alcohol Rev* 2:84, 1983.

Wang, G.J.; Volkow, N.D.; Hitzemann, R.J.; et al. Brain imaging of an alcoholic with MRI, SPECT and PET. *Am J Physiol Imaging* 7:194–198, 1992.

Wang, G.J.; Volkow, N.D.; Roque, C.T.; et al. Functional importance of ventricular enlargement and cortical atrophy in healthy subjects and alcoholics as assessed with PET, MR imaging, and neuropsychologic testing. *Radiology* 186:59–65, 1993.

Weiss, F.; Hurd, Y.L.; Ungerstedt, U.; et al. Neurochemical correlates of cocaine and ethanol self-administration. *Ann NY Acad Sci* 654:220–241, 1992.

Wesson, D.; Carlin, A.; Adams, K.M.; and Beschner, G. *Polydrug Abuse*. New York: Academic Press, 1978.

Wik, G.; Borg, S.; Sjogren, I.; et al. PET determination of regional cerebral glucose metabolism in alcohol-dependent men and healthy controls using ^{11}C-glucose. *Acta Psychiatr Scand* 78:234–241, 1988.

Wong, D.T.; Lumeng, L.; Threlkeld, P.G.; et al. Serotonergic and adrenergic receptors in alcohol-preferring and -nonpreferring rats. *J Neural Transm* 71:207–218, 1983.

Zhou, F.C.; Zhang, J.K.; Lumeng, L.; and Li, T.-K. Mesolimbic dopamine system in alcohol-preferring rats. *Alcohol* 12:403–412, 1995.

Chapter 10

Neurotransmitter-Based Therapeutic Strategies in Late-Life Alcoholism and Other Addictions

David W. Oslin, M.D., and Alan M. Mellow, M.D., Ph.D.

Over the past several years both clinical research and basic science research have increasingly supported a role for specific neurotransmitters in the etiology and treatment of addiction. Almost exclusively, this research has been conducted in young to middle-age adults. The aim of this chapter is to review the current understanding of neurotransmitter involvement in alcoholism and other addictions and the implications of age-associated changes in brain function. The clinical relevance of this discussion is underscored by the dramatic increase in the number of elderly individuals in our population and by the known physiological changes that are associated with aging. Indeed, it is the age-related changes in neurobiology that should raise basic questions regarding the generalizability of research findings about the pharmacotherapy of addictions across the entire lifespan.

The aging process is associated with many biochemical and physiological changes, although many organ systems are able to maintain their function despite advanced age. Physiological changes that occur with age can influence many diseases, including addiction. Age-related changes in physiology may increase (such as pneumonia or hypothermia) or decrease (such as immune disorders) the likelihood of a disease. Age-related changes may also mimic certain diseases (such as glucose intolerance) or

D.W. Oslin, M.D., is assistant professor of psychiatry, Addiction and Geriatric Psychiatry, University of Pennsylvania, Ralston-Penn Center, 3615 Chestnut Street, Philadelphia, PA 19104. A.M. Mellow, M.D., Ph.D., is associate professor of psychiatry and director of the Division of Geriatric Psychiatry at the University of Michigan and is Chief of Psychiatry at Ann Arbor VA Medical Center, 2215 Fuller Road, Ann Arbor, MI 48105.

alter the presentation of a disease (such as diabetes) (Rowe 1996). The best understood age-related physiological changes include decreased pulmonary function, decreased glomerular filtration, menopausal changes, decreased renal blood flow, decreased gastric acid production, decreased hepatic blood flow, decreased hepatic oxidation, changes in body mass, and decreased sensory perception (taste, hearing, vision). Many of these age-related changes in physiology are clinically relevant and necessitate an adjustment in the diagnosis and management of certain diseases. For example, steady-state levels of medications often differ in older versus younger adults because of changes in absorption, volume of distribution, metabolism, excretion, and receptor binding changes. Additionally, in medically debilitated older adults, the diagnosis of diseases like alcoholism may be hampered by the presence of multiple illnesses and normal physiological changes.

The central and peripheral nervous systems also undergo change with advancing age. Changes in the brain that occur with normal aging make elderly individuals especially vulnerable to the effects of a variety of disease states. With aging there is atrophy (or shrinkage) of the brain due to age-related loss of neurons and nerve fibers as well as the frequent accumulation of cerebrovascular and Alzheimer's disease–related pathology (Goldman and Cote 1991). As discussed later in this chapter, aging is also associated with changes in neurotransmitter receptor density and changes in neurotransmitter production and release. One of the most consistent age-related changes is a decrease in protein synthesis that is directly related to central nervous system (CNS) function, because many neurotransmitters, most receptors, and the enzymes and messenger systems that are triggered by receptor-mediated events are protein based (Ratian 1996). Aging is also associated with a host of neurological disorders, including the major neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, and diffuse Lewy body disease, as well as stroke, tumors, and systemic illnesses that affect mental processes. The CNS in particular undergoes age-related changes that make the individual more susceptible to specific disease and increase the individual sensitivity to certain pharmacological therapies.

The neurobiology of addiction has received increased attention as a way to understand the etiology of addiction-related behaviors and as a method for developing "rational" treatment strategies. A notable example of "rationally" developed treatment is the recent Food and Drug Administration approval of the opiate receptor antagonist naltrexone for the treatment of alcoholism. In a preclinical study, Volpicelli and colleagues (1986) demonstrated that blockade of opiate receptors was beneficial in reducing alcohol use, leading to the clinical studies that demonstrated efficacy in the treatment of alcohol dependence (Volpicelli et al. 1992). Ideally, understanding the neurobiological substrates of addiction-related behaviors at a molecular level could lead to further development of

pharmacological agents that target these specific systems.

In addition to the opioid system, several other neurotransmitter systems have been reported to play a role in the etiology and maintenance of addictive behaviors. Most notably, the dopaminergic system has been implicated as the principal neurotransmitter involved in reward mechanisms (Anton 1996). Opioids and other neurotransmitters, such as serotonin, glutamate, and gamma-aminobutyric acid (GABA), are also believed to be involved in uncontrollable drinking and have been demonstrated to modulate dopamine release (Koob 1992; Porras and Mora 1995). The dopaminergic system, the opioid system, the serotonergic system, the glutamatergic system, and the GABAergic system are discussed in this chapter because there is an abundance of data relating them to addictive behavior. Other neurotransmitters may be involved in addiction, but there is currently less evidence for such links. For this review we will address the basic neurochemical and physiological features of each system, their relevance to addictive behavior, age-related changes, and pharmacological strategies in older adults.

DOPAMINERGIC SYSTEM

Dopamine is one of several biogenic monoamine neurotransmitters found throughout the CNS. In the mammalian CNS, there are three major dopaminergic systems: the tuberoinfundibular, the nigrostriatal, and the mesocorticolimbic. The tuberoinfundibular system is involved with the secretion of prolactin. The nigrostriatal system is involved in movement and muscle tone, and loss of neurons from this area often results in Parkinson's disease. The mesolimbic system is thought to be involved in drug-reinforcing behavior as well as locomotor activity. It may also act as a filter or gate for the limbic system (Koob 1992). Neurons in the mesolimbic system originate in the ventral tegmental area (VTA) and project to the forebrain (nucleus accumbens, amygdala, olfactory tubercle, prefrontal cortex, and septal areas). The medial forebrain bundle, sometimes known as the pleasure pathway, consists of the lateral hypothalamus, the VTA, the nucleus accumbens, and the frontal cortex (Koob 1992).

Once released from presynaptic neurons, dopamine acts at one of two principal types of receptors (D_1 and D_2), both of which are linked to the generation of cyclic adenosine monophosphate (cAMP) via the enzyme adenylate cyclase. Three other dopamine receptor types have also been identified (D_3, D_4, and D_5), but their role in the CNS is less clear (Meador-Woodruff 1994). Dopamine is degraded in the CNS by monoamine oxidase B (MAO-B) (Fowler 1982).

Relevance to Addictive Behavior

As reviewed by Robinson and Berridge (1993), the dopamine neurotransmitter system is thought to be involved in the craving for drugs and not necessarily in the pleasure derived from drug use. Researchers have focused on the nucleus accumbens as the area of

the brain most likely to be involved in the craving for substances such as alcohol (Koob 1992). Alcohol has been shown to affect dopamine release. After acute administration of alcohol to rats, significantly higher concentrations of released dopamine have been found in the nucleus accumbens; this effect appears to be dose dependent (Le and Kiianmaa 1988; Weiss et al. 1993). Studies by Gonzales and colleagues suggest that although dopamine release in the nucleus accumbens is a reproducible finding after alcohol administration, the effect is not a direct one, suggesting the involvement of other neurotransmitters (Gonzales 1996). In support of this finding, naltrexone, an opioid antagonist, was found to block the release of dopamine in rats after alcohol ingestion, suggesting an interaction between the dopamine and opioid systems (Spanagel et al. 1992; Benjamin et al. 1993).

In a human study, Balldin and colleagues (1993) demonstrated in a small number of abstinent alcoholics that the growth hormone response to apomorphine, a dopamine receptor agonist, is decreased relative to age-matched controls. Finally, although somewhat controversial, severe alcoholism has been suggested to have a genetic link to the dopamine D_2 receptor subtype (Noble et al. 1994; Blum 1995).

Thus, there is both preclinical and clinical evidence of a significant role for the mesocorticolimbic dopaminergic system in alcoholism. Despite a great deal of effort, pharmacological manipulation of the dopamine system has yielded no significant advances toward the reduction of alcohol drinking (Anton 1995). Part of the reason for this may be the multiplicity of dopaminergic receptors and the lack of specificity of many dopamine receptor agonists and antagonists. Another possible explanation is that dopamine antagonists are only effective in blocking initial craving and are not effective once drug use is established.

Age-Related Changes

The nigrostriatal dopaminergic system is one of the best studied neurotransmitter systems in the human brain and has been shown to undergo several age-related changes. These changes include a decrease in D_2 receptor density, decreased D_2 receptor mRNA levels, decreased striatal dopamine concentrations, decreased tyrosine hydroxylase activity, increased MAO-B activity, and decreased nigrostriatal neurons (Carlsson and Winblad 1976; McGeer and McGeer 1976; Benedetti and Keane 1980; Severson et al. 1982; Wong et al. 1984; Zelnik et al. 1986; DeKeyser et al. 1990; Mesco et al. 1991; Antonini et al. 1993).

Although less well studied, other brain regions also undergo age-specific declines in the dopamine system. Dopamine transporter proteins (reflecting dopamine neurons) in the caudate and putamen are reduced in older adults compared with younger adults (Volkow et al. 1996). However, receptor affinity for dopamine does not undergo age-related changes. The hippocampus has been shown to be particularly vulnerable to aging, with reduced levels of dopamine and homovanillic acid (HVA), a dopamine

metabolite (Adolfsson et al. 1979). Furthermore, there is an age-associated increase in MAO-B activity in temporal cortex, hypothalamus, and frontal cortex (Sparks et al. 1991).

Pharmacological Strategies for Older Adults

Age-related changes in the nigrostriatal dopaminergic system are clinically related to greater susceptibility to diseases such as Parkinson's disease, as well as greater susceptibility to extrapyramidal symptoms during therapy with antipsychotic neuroleptic drugs (Pollock 1995). The changes summarized in the preceding section might suggest that the role of dopamine in other brain regions, such as those involved in alcohol-related reward and craving, may also be altered in older individuals. Unfortunately, no preclinical studies have been conducted examining age-related changes and the interaction between alcohol and the mesocorticolimbic dopaminergic system. However, in preliminary work by Oslin and Katz (unpublished data), older adults appear to be less susceptible to the mood-elevating effects of amphetamine, which acts largely through presynaptic dopamine release. This suggests that the affective response to dopaminergic stimuli may be altered in older adults. The relevance of age-related changes in the dopamine system to the development of pharmacological agents to treat alcoholism is not clear, since direct manipulation of the dopamine system has not produced promising results in animal models or in clinical trials. The relevance of age-related dopaminergic changes is likely to be in the interaction with other neurotransmitter systems.

OPIOID SYSTEM

Opioid neuropeptides are widely distributed throughout the CNS. The opioid system is involved in the modulation of pain, stress, reward, and homeostatic adaptive functions such as food intake, temperature, and water regulation. There are four classes of opioid neurotransmitters—beta-endorphin, mu-enkephalins, leu-enkephalins, and dynorphins (Bissette 1996)—and three main types of opioid receptors—mu, delta, and kappa (Wood 1982). Beta-endorphin appears to be selective for mu sites, enkephalins have high affinity for delta receptors with modest affinity to mu receptors, and dynorphins are selective for kappa receptors (Charness 1989).

Relevance to Addictive Behavior

Preclinical and clinical studies involving the opioid system have produced recent research interest and have led to innovations in the treatment of alcoholism. Several lines of evidence from rodents suggest that alcohol acutely and chronically affects the opioid system. Alcohol administration increases opioid release from the pituitary gland of rodents and humans (Gianoulakis 1990; Gianoulakis et al. 1996). Acute alcohol administration has also been shown to increase endorphin and enkephalin gene expression in the nucleus accumbens as well as the expression of delta opioid receptors and receptor mRNA in

neuronal cell culture (Charness et al. 1986, 1993; Jenab and Inturrisi 1994; Li et al. 1996). Changes in the opioid system may also be genetically transmitted. This is suggested by a study demonstrating a higher density of delta or mu opioid receptors in the VTA and nucleus accumbens in alcohol-preferring rats (Gianoulakis 1996). Finally, long-term drinking decreases beta-endorphin levels in chronic alcoholics (Vescovi et al. 1992).

Pharmacological studies have yielded evidence of opiate effects on alcohol intake in animals. Administration of low doses of mu or delta receptor agonists increased drinking in rats (Reid and Hunter 1984). In contrast, administration of kappa agonists to rats produced aversive drinking states, suggesting that mu and delta receptors are involved in reward systems while kappa receptors provide a balance to the reward and aversion to alcohol (Imperato and DiChiara 1988; Spanagel et al. 1992). Other studies have indicated that both selective and nonselective opioid antagonists reduce alcohol self-administration in rodents and monkeys (Altshuler et al. 1980; Hubbell et al. 1986; Volpicelli et al. 1986; Froehlich et al. 1990; Weiss et al. 1990; Le et al. 1993; Krishnan-Sarin et al. 1995*a*, 1995*b*).

Further evidence for a role of opioids in the etiology of drinking has been found in humans at risk for developing alcohol problems. The basal beta-endorphin levels in adults at risk for developing alcohol problems were low compared with those at less risk for developing alcohol problems (Gianoulakis et al. 1996). Moreover there was a greater release of endorphin in the high-risk subjects after alcohol administration compared with low-risk subjects. This finding, along with studies of twin pairs, suggests a possible genetic contribution of altered opioid function (Froehlich et al. 1995).

There is evidence for a link between the opioid system and the dopaminergic system, which, as noted previously, may have relevance to addiction. Beta-endorphin–producing neurons from the arcuate nucleus innervate dopamine cells in the VTA and the nucleus accumbens (Froehlich 1996). Enkephalin-producing neurons from the nucleus accumbens and striatum innervate dopamine neurons in the accumbens. Thus, ethanol is thought to trigger the release of opioids, which, in turn, stimulate dopamine release in the accumbens.

Perhaps the most encouraging finding regarding the role of the opioid system in excessive drinking is the demonstration of the clinical utility of opioid antagonists in the treatment of alcoholism. Volpicelli and colleagues (1992) and O'Malley and colleagues (1992) have demonstrated that the opioid antagonist naltrexone is efficacious as an adjunct in the treatment of middle-aged patients with alcohol dependence. In both studies, naltrexone was found to be safe and effective in preventing relapse and reducing the craving for alcohol. Similar results have been reported with nalmefene, also an opioid antagonist (Mason et al. 1994).

Age-Related Changes

As with the dopaminergic system, the opioid system has been shown to undergo significant changes with age.

The most consistently demonstrated finding has been a loss of opioid receptor number. Delta and mu receptors are decreased in the cortex, striatum, and hypothalamus of aged rats (Gambert et al. 1980; Petkov et al. 1988). Furthermore, age-dependent decreases in the number of receptors that bind ^{3}H-etorphine have been shown in frontal poles, hippocampus, and striatum of rats (Hess et al. 1981).

The opioid neurotransmitters undergo age-related changes in availability and structure. Miller and colleagues (1991) demonstrated a 30-percent loss of beta-endorphin neurons in aged female mice. Hypothalamic beta-endorphin levels were reduced up to 75 percent in older male rats (Simpkins and Millard 1987). Other studies have shown a 30- to 50-percent loss of beta-endorphin neurons in old mice (Miller et al. 1991; Miller and Zhu 1992). This can be contrasted with increases in beta-endorphin in the anterior pituitary and the neurointermediate lobe of the pituitary gland (Simpkins and Millard 1987). Jiang and colleagues (1989) demonstrated age-related increases in the expression of hippocampal dynorphin. Thus, age-related changes in the opioid neurotransmitters may be specific both to the subclasses of opioids and to selective regions of the brain.

Perhaps one of the more thorough evaluations of beta-endorphin changes across the lifespan demonstrated that age-related posttranslational changes in beta-endorphin result in a change in the ratio of beta-endorphin (1-31) to beta-endorphin (1-27) and (1-26) (Joshi et al. 1995). These latter forms of beta-endorphin have been shown to lose a substantial portion of opioid activity despite being able to displace the more active form (1-31). Thus, beta-endorphin (1-27) and (1-26) in essence become opiate receptor antagonists. In middle-aged female mice there is an increase in beta-endorphin (1-27) and (1-26) that may dramatically alter receptor function. Although such changes may relate more to the onset of menopause, these findings point to the importance of age-related opiate receptor changes across the lifespan. In male rats, Wilkinson and Dorsa (1986) demonstrated an age-related increase in the beta-endorphin (1-27) and (1-26) forms.

Pharmacological Strategies for Older Adults

Age-related changes in opioid receptor function, neuron function, and protein structural changes in beta-endorphin raise important questions regarding the relevance to the opioid compensation hypothesis for alcohol consumption proposed by Volpicelli (1987). Moreover, the clinical efficacy of agents such as naltrexone or nalmefene in older individuals cannot be assumed, given these changes. Indeed, if there are fewer receptors with which to interact, and there is a possibility that the receptors may be competitively blocked by beta-endorphin (1-27) and (1-26), then it is entirely possible that opioid antagonists may be ineffective in older adults.

Despite the theoretical reasons that opioid antagonists may be ineffective, a recent study of adults in late middle age did demonstrate a clinically significant

reduction in alcohol relapse in naltrexone-treated veterans (Oslin et al. 1997). The study was designed as a double-blind placebo-controlled randomized trial of naltrexone, 50 mg/d, in 50- to 70-year-old alcoholics. Half as many naltrexone-treated subjects relapsed to significant drinking compared with those treated with placebo. Although this study did not include many elderly subjects, it does raise the hope that opioid antagonists may have clinical efficacy among older alcoholics. Given the effectiveness of naltrexone in treating younger and middle-aged alcoholics, expanding research to include elderly adults is not only justified but imperative.

SEROTONERGIC SYSTEM

Serotonin is synthesized from tryptophan and found mainly in the dorsal raphe nucleus. Axons from the raphe distribute widely to the forebrain, cerebellum, and spinal cord. The serotonin system has been implicated as a major factor in mood, arousal, sensory perception, and higher cognitive function. Monoamine oxidase A (MAO-A) is the primary enzyme involved in the degradation of serotonin (Fowler 1982). At least 13 different serotonin receptors have been identified (Teitler and Herrick-Davis 1994).

Relevance to Addictive Behavior

As reviewed by Lejoyeux (1996), the serotonin system has been shown in preclinical studies to be involved in drinking behavior as well as the consumption of other palatable or reinforcing substances. Acute administration of alcohol has been shown to cause release of serotonin in the nucleus accumbens (Yoshimoto et al. 1992). However, chronic administration of alcohol results in decreased release of serotonin in the accumbens (Carmichael and Israel 1975). Moreover, alcohol has been shown to potentiate the effect of serotonin at the 5-HT_3 receptor (Lovinger 1991). Evidence also suggests that serotoninergic abnormalities may be genetically transmitted and relevant to drinking. Abnormalities in the serotonergic system have been consistently demonstrated in strains of alcohol-preferring rodents (Wong et al. 1988; Rezvani et al. 1990). In humans, several studies have shown reduced levels of 5-hydroxyindoleacetic acid (5-HIAA), the main metabolite for serotonin, in the cerebrospinal fluid of alcoholics, especially those with heavy, uncontrolled drinking (Ballenger et al. 1979; Roy and Linnoila 1989).

As with other complex neurotransmitter systems, the effects of alcohol on the serotonergic system relate to specific receptors and possibly to specific brain regions. As reviewed by Overstreet and colleagues (1995), preclinical studies have suggested that agonists at 5-HT_{1a} and antagonists at 5-HT_{2a} and 5-HT_3 receptors reduce alcohol intake. The partial 5-HT_{1a} agonist ipsapirone and the agonist 8-OH-DPAT selectively reduce alcohol intake and dopamine release (Yoshimoto and McBride 1992; Schreiber et al. 1993). To complicate this picture, administration of low doses of 8-OH-

DPAT specifically to the median or dorsal raphe nucleus stimulated alcohol intake in rats (Tomkins et al. 1994). Tomkins and colleagues suggested that stimulation of the 5-HT_{1a} receptors in the dorsal raphe in turn inhibited serotonin transmission in other brain regions, thus leading to alcohol intake.

A potentially clinically useful medication is amperozide, which is a selective 5-HT_{2a} antagonist that reduces alcohol intake in a dose-dependent fashion (Overstreet et al. 1996). Clinical trials of this medication are awaited. There have been positive results demonstrated in open-label clinical trials with the use of ritanserin, a 5-HT_2 antagonist (Le et al. 1996). The agonists 1-(2,5-demethoxy-4-iodophenyl)-2-aminopropane (DOI) and *m*-chlorophenylpiperazine hydrochloride (m-CPP) have both been shown to decrease alcohol intake in preclinical studies but have not been demonstrated to be clinically efficacious (Le et al. 1996). The more selective agent ondansetron, a 5-HT_3 receptor antagonist, has shown promise in clinical trials (Le et al. 1993; Sellers et al. 1994). Moreover, an interaction with dopamine has been shown after the use of 5-HT_3 antagonists. Pretreatment with 5-HT_3 antagonists blocks the typical alcohol-induced release of dopamine (Carboni et al. 1989; Wozniak et al. 1991).

Selective serotonin reuptake inhibitors (SSRIs), a class of agents currently in clinical use as antidepressants, showed initial promise in treating alcohol-dependent adults (Naranjo et al. 1984, 1987, 1989, 1990). However, as reviewed by Anton (1995, 1996), several small clinical trials and a few placebo-controlled randomized trials showed only a modest or no effect of these serotonergic agents in reducing alcohol consumption in young and middle-aged adults. One potentially promising area is the use of SSRIs in alcoholic patients who have significant comorbid psychiatric illnesses such as depression or anxiety (Kranzler et al. 1995). Another area of promise is the potential for synergistic effects upon reducing alcohol intake shown in mice and rats when combining naltrexone and ondansetron (Le et al. 1996).

Age-Related Changes

Although not extensive, there is some evidence for age-associated changes in serotonergic systems. In aged rodents, hippocampal serotonin levels are decreased, 5-H_{1b} receptors are lost, neuronal fibers are reduced in number in the dorsal raphe, and fenfluramine, a serotonin reuptake inhibitor, has been reported to be less effective in inducing serotonin release (Gozlan et al. 1990; Lolova and Davidoff 1992; Handa et al. 1993; Venero et al. 1993). In a study of various human brain regions, neither 5-HIAA nor the levels of serotonin demonstrated age-related changes (Wester et al. 1984). However, receptor sites for 5-HT_{1A} have been shown to be decreased in the cerebral cortex of humans and rats, while receptor sites for 5-HT_2 are only slightly decreased in older adults (Maloteaux et al. 1989; Gozlan et al. 1990). Moreover,

there is evidence for an age-related decline in behavioral response to m-CPP (Lawlor et al. 1989).

Pharmacological Strategies for Older Adults

The preclinical evidence for age-related changes in the serotonin system is mixed; the available evidence does suggest a decrease in some receptors. As with the opioid system, the changes in the serotonergic system would suggest that agents that affect this system may be less effective in older adults. Indeed, a study of elderly depressed patients found that SSRIs were less effective in patients with more severe depressive features (Roose et al. 1994). Streim and colleagues (1996) demonstrated a similar phenomenon in frail elderly nursing home residents. Sertraline was ineffective in treating depression in cognitively impaired residents, whereas nortriptyline was modestly effective, suggesting a differential efficacy. Of note, all commercially available SSRIs have been demonstrated effective in treating depression in the elderly compared with placebo.

Closser and colleagues found that sertraline was no more effective than placebo as an adjunctive treatment in preventing relapse in nondepressed, cognitively intact older alcoholics (M.H. Closser, personal communication, 1997). As reviewed above, SSRIs have only demonstrated modest effects in middle-aged adults. Newer, more selective agents may demonstrate a greater effect than the currently available drugs. There is also interest in the efficacy of SSRIs in treating alcoholics with significant affective symptoms. This strategy may also prove efficacious in older adults, but it will need exploration.

GLUTAMATERGIC SYSTEM

Glutamate is an amino acid derived from glutamine and functions in the brain as an excitatory neurotransmitter (Shepherd 1988). Glutamate is the principal neurotransmitter in pyramidal cells in the cerebral cortex and in several hippocampal tracts (Cotman et al. 1987; Collingridge and Lester 1989). Glutamate interacts with five different receptors: the quisqualate receptor, the AP-4 receptor, the metabotropic (or APCD) receptor, the kainate receptor, and the *N*-methyl-D-aspartate (NMDA) receptor (McEntee and Crook 1993; Bissette 1996). It has been speculated that the NMDA receptor is involved in mood and memory function and may play a role in neurodegenerative disorders.

Relevance to Addictive Behavior

Ethanol has been shown to inhibit glutamate function by acting to antagonize its action at the NMDA receptor during acute administration of alcohol (Tsai et al. 1995; Gonzales 1996). However, chronic administration of alcohol leads to up-regulation of NMDA receptor binding sites. Activation of the up-regulated receptors during alcohol withdrawal has been suggested as a possible mechanism for the symptoms of alcohol withdrawal. In support of this idea, NMDA receptor antagonists reduce

the symptoms of withdrawal, especially seizures (Liljequist 1991; Rossetti and Carboni 1995). Moreover, in rats, glutamate levels have been demonstrated to rise in the extracellular fluid during withdrawal and are associated with the occurrence of tremors, rigidity, and seizures (Rossetti and Carboni 1995).

Recently, acamprosate has been studied as a promising agent in the treatment of alcoholism. Although its exact mechanism of action is unknown, acamprosate is thought to reduce glutamate response but not as a typical NMDA blocker (Zeise et al. 1994; Spanagel et al. 1996). The clinical evidence favoring acamprosate is significant. Sass and colleagues (1996) studied 272 alcohol-dependent subjects in Europe for up to 48 weeks in a placebo-controlled randomized trial of acamprosate. Forty-three percent of the acamprosate-treated group were abstinent at the conclusion of the study, compared with 21 percent of the placebo group. A multicenter trial of acamprosate in the United States is currently in progress.

Age-Related Changes

Several studies have demonstrated age-related reductions in glutamate and NMDA receptors in brains of rats (Price et al. 1981; Banay-Schwartz et al. 1989; Tamaru et al. 1991; Ogawa et al. 1992; Nabeshima et al. 1994) and monkeys (Wenk et al. 1991). Antioxidant drugs such as bifemelane, a cerebral metabolic activator, inhibit the age-related decrease in receptors (Ogawa et al. 1992). However, several investigators have failed to demonstrate age-related changes in glutamate release or in vivo extracellular glutamate concentrations in the striatum of aged rats (Fonda et al. 1973; Donzanti et al. 1993; Porras et al. 1993; Sanchez-Prieto et al. 1994).

In human postmortem tissue, binding of MK-801, a NMDA receptor antagonist, has been demonstrated to vary among brain regions, with no age-related change in most regions and increased binding in CA1 and CA2 areas of the hippocampus (Johnson 1996). Interestingly, glutamate is a potential neurotoxin and has been suggested, along with a number of other mechanisms, as a potential toxin involved in neurodegenerative diseases, including Wernicke-Korsakoff syndrome (Rothsein 1996). Glutamate may also be involved in alcohol-related dementia (McEntee and Crook 1993).

Pharmacological Strategies for Older Adults

Acamprosate is particularly interesting in regard to treating older adults. Although less well studied than the monoaminergic systems, glutamatergic systems appear to undergo fewer age-related changes in humans. This would suggest that a compound that has its main effect through the glutamate system and is effective in treating alcoholism may be well suited for older alcoholics. Although the exact mechanism of action of acamprosate is unknown, and more studies of the glutamate system in older adults need to be conducted, there are studies planned to test the efficacy of acamprosate in older alcoholics.

GABAERGIC SYSTEM

The amino acid GABA functions in the CNS generally as an inhibitory neurotransmitter (Shepherd 1988). GABA is synthesized from glutamate by glutamic acid decarboxylase (GAD). GABA receptors have generally been divided into $GABA_A$ and $GABA_B$, based upon properties of the receptor. $GABA_A$ receptors are linked to a Cl^- ion channel, and $GABA_B$ receptors are linked to K^+ and Ca^{2+} ion channels. GABA neurons have been demonstrated to produce a tonic inhibition of dopamine receptors in the nucleus accumbens. Moreover, both dopamine and glutamate agonists have been shown to stimulate the release of GABA (Porras and Mora 1995). Similarly, GABA can affect dopaminergic and glutamatergic tone in the striatum (Porras and Mora 1995).

Relevance to Addictive Behavior

Alcohol is believed to exert its acute behavioral effects by a selective enhancement of $GABA_A$ receptor activity (Little 1991). In support of this, $GABA_A$ receptor antagonists block the ability of alcohol to cause ataxia and anesthesia (Frye and Breese 1982; Liljequist and Engel 1982). Alcohol also potentiates the effects of GABA in the cerebral cortex and cerebellum (Suzdak et al. 1986; Allan and Harris 1987). In rodents, an acute rise in released GABA in the hypothalamus is followed by a decrease in concentration after acute or chronic exposure to intraperitoneal alcohol (Kaneyuki 1995). Although not well studied, there is little evidence for a role of GABA in the craving or reward behavior of alcohol use. This is further supported by a lack of GABA release in the nucleus accumbens after intraperitoneal injections of alcohol (Dahchour et al. 1994).

Age-Related Changes

Decreased levels of GABA, as well as impairment of GABA-mediated synaptic transmission, have been demonstrated in the medial septum of aged rats (Banay-Schwartz et al. 1989; Garcia and Jaffard 1993). In human postmortem studies, age-related changes in GAD have been inconsistent. Investigators have found an increase, a decrease, or no change in the level of GAD in various brain regions (Fonda et al. 1973; Bird and Iversen 1974; McGeer and McGeer 1978; Spokes 1979). Findings with respect to GABA receptors have also been mixed. Wenk and colleagues (1991) found no age-related changes in $GABA_A$ receptor binding of ^3H-muscimol in rats or monkeys. Sundman and colleagues (1997) found no age-related changes in GABA uptake sites in the frontal cortex of suicide victims. However, Gutierrez and colleagues (1994) demonstrated in aged rats a 40-percent decrease in $GABA_A$ receptors and GAD in the inferior colliculus (Gutierrez 1994). This study did not examine the frontal cortex. Araki and colleagues (1996) demonstrated age-related decreased binding of ^3H-muscimol (binds to $GABA_A$ receptors) throughout the entire rat brain.

As already mentioned, the GABAergic system is interconnected with both the dopaminergic and the glutamatergic systems. Indeed, dopamine receptor stimulation with apomorphine causes the release of GABA. Porras and Mora (1995) found that greater doses of apomorphine were required in older rats to yield effects similar to those in young rats. This study suggests that the interaction between dopamine and GABA undergoes important dynamic changes over the lifespan.

Pharmacological Strategies for Older Adults

The loss of apomorphine-induced GABA release, in addition to the reduced levels of GABA demonstrated in older rodents, suggests that medications that alter GABA function may have similar limitations as opioid and serotonergic agents. Whether the GABAergic system is sensitive to manipulations in late-life addictions remains to be studied.

OTHER NEUROTRANSMITTERS

Several other neurotransmitters have been shown to be involved in craving states or to undergo changes in alcoholics. One of the more intriguing neurotransmitters that has particular reference to aging is melatonin. Melatonin has been demonstrated to be involved in circadian rhythms, specifically in the control of sleep and wake cycles. Low levels of melatonin production have been associated with maintenance of alcohol abstinence (Murialdo et al. 1991). Melatonin excretion has also been demonstrated to be reduced in older adults compared with younger adults (Wetterberg et al. 1992; Ferrari et al. 1995). Because disturbances in the sleep-wake cycle are a common symptom in alcohol withdrawal, it is possible that melatonin may be involved in the difficulty many alcoholics have in maintaining abstinence. Given the increases in sleep disturbances with age, dysregulation of melatonin and/or sleep may have special significance in older alcoholics.

Other neurotransmitters may play a role in conditions comorbid with alcoholism; for example, acetylcholine in memory deficits and norepinephrine in mood disturbances. This raises the possibility that pharmacological agents targeting neurotransmitters involved in comorbid illnesses may in fact have beneficial effects in reducing alcohol intake.

CONCLUSIONS

In this chapter we have attempted to briefly review the current understanding of the major neurotransmitter systems involved in alcoholism and other addictions, as well as the significance of age-related changes in these systems. With respect to treatment strategies for older individuals with addictive disorders, this has, of necessity, been largely speculative, because of the dearth of relevant clinical research in this population. Such studies afford unique opportunities, from a developmental perspective, to learn about the interaction between age, addiction, and underlying biochemical vulnerability. Given the tremendous

growth in the elderly population, there is a significant need for further research in the neuropharmacology of addiction and aging, at both the basic science level and with clinical trials. Finally, while psychopharmacology has tremendous potential for the treatment of young and old adults, there remains, in the treatment of addiction, a need to focus on environmental and interpersonal change. Any pharmacological intervention should be considered in the context of the whole range of psychosocial and biological treatments for patients of any age with these disorders.

REFERENCES

Adolfsson, R.; Gottfries, C.G.; Roos, B.E.; and Winblad, B. Post-mortem distribution of dopamine and homovanillic acid in human brain, variations related to age, and a review of the literature. *J Neural Transm* 45:8–105, 1979.

Allan, A.M., and Harris, R.A. Acute and chronic ethanol treatments alter GABA receptor-operated chloride channels. *Pharmacol Biochem Behav* 27:665–670, 1987.

Altshuler, H.L.; Phillips, P.E.; and Feinhandler, D.A. Alteration of ethanol self-administration by naltrexone. *Life Sci* 26:679–688, 1980.

Anton, R.F. New directions in the pharmacotherapy of alcoholism. *Psychiatr Ann* 25:353–362, 1995.

Anton, R.F. Neurobehavioural basis for the pharmacotherapy of alcoholism: Current and future directions. *Alcohol Alcohol* 31 (Suppl 1):43–53, 1996.

Antonini, A.; Leenders, K.L.; Reist, H.; Thomann, R.; Beer, H.-F.; and Locher, J. Effect of age on D_2 dopamine receptors in normal human brain measured by positron emission tomography and ^{11}C-raclopride. *Arch Neurol* 50:474–480, 1993.

Araki, T.; Kato, H.; Fujiwara, T.; and Itoyama, Y. Regional age-related alterations in cholinergic and GABAergic receptors in the rat brain. *Mech Ageing Dev* 88:49–60, 1996.

Balldin, J.; Berggren, U.; Lindstedt, G.; and Sundkler, A. Further neuroendocrine evidence for reduced D2 dopamine receptor function in alcoholism. *Drug Alcohol Depend* 32:159–162, 1993.

Ballenger, J.C.; Goodwin, F.K.; Major, L.F.; and Brown, G.L. Alcohol and central serotonin metabolism in man. *Arch Gen Psychiatry* 36:224–227, 1979.

Banay-Schwartz, M.; Lajtha, A.; and Palkovits, M. Changes with aging in the levels of amino acids in rat CNS structural elements. I. Glutamate and related amino acids. *Neurochem Res* 14:555–562, 1989.

Benedetti, M.S., and Keane, P.E. Differential changes in monoamine oxidase A and B activity in the aging rat brain. *J Neurochem* 35:1026–1031, 1980.

Benjamin, D.; Grant, E.R.; and Pohorecky, L.A. Naltrexone reverses ethanol-induced dopamine relase in the nucleus accumbens of awake, freely moving rats. *Brain Res* 621:137–140, 1993.

Bird, E.D., and Iversen, L.L. Huntington's chorea. post-mortem measurement of glutamic acid decarboxlase, choline acetyltransferase and dopamine in basal ganglia. *Brain* 97:457–472, 1974.

Bissette, G. Chemical messengers. In: Busse, E.W., and Blazer, D.G., eds. *Textbook of Geriatric Psychiatry.*

Washington, DC: American Psychiatric Press, 1996. pp. 73–93.

Blum, K.; Sheridan, P.J.; Wood, R.C.; Braverman, E.R.; Chen, T.J.H.; and Comings, D.E. Dopamine D2 receptor gene variants: Association and linkage studies in impulsive-addictive-compulsive behaviour. *Pharmacogenetics* 5:121–141, 1995.

Carboni, E.; Acquas, E.; Leone, P.; and DiChiara, G. 5-HT3 receptor antagonists block morphine and nicotine but not amphetamine-induced reward. *Psychopharmacology* 97:175–178, 1989.

Carlsson, A., and Winblad, B. Influence of age and time interval between death and autopsy on dopamine and 3-methoxytyramine levels in human basal ganglia. *J Neural Transm* 38:271–276, 1976.

Carmichael, F.J., and Israel, Y. Effects of ethanol on neurotransmitter release by rat brain cortical slices. *J Pharmacol Exp Ther* 193:824–834, 1975.

Charness, M.E. Ethanol and opioid receptor signalling. *Experientia* 45:418–428, 1989.

Charness, M.E.; Querimit, L.A.; and Diamond, I. Ethanol induces the expression of functional delta-opioid receptors in the neuroblastoma x glioma NG108-15 hybrid cells. *J Biol Chem* 261:3164–3169, 1986.

Charness, M.E.; Hu, G.; Edwards, R.H.; and Querimit, L.A. Ethanol increases delta-opioid receptor gene expression in neuronal cell lines. *Mol Pharmacol* 44:1119–1127, 1993.

Collingridge, G.L., and Lester, R.A.J. Excitatory amino acid receptors in the vertebrate central nervous system. *Pharmacol Rev* 40:143–210, 1989.

Cotman, C.W.; Monaghan, D.T.; Ottersen, O.P.; and Storm-Mathisen, J. Anatomical organization of excitatory amino acid receptors and their pathways. *Trends Neurosci* 10:273–280, 1987.

Dahchour, A.; Quertemont, E.; and DeWitte, P. Acute ethanol increases taurine but neither glutamate nor GABA in the nucleus acccumbens of male rats: A microdialysis study. *Alcohol Alcohol* 29:485–487, 1994.

DeKeyser, J.; Ebinger, G.; and Vauquelin, G. Age-related changes in the human nigrostriatal dopaminergic system. *Ann Neurol* 27:157–161, 1990.

Donzanti, B.A.; Hite, J.F.; and Yamamoto, B.K. Extracellular glutamate levels increase with age in the lateral striatum: Potential involvement of presynaptic D-2 receptors. *Synapse* 13:376–382, 1993.

Ferrari, E.; Magri, F.; Dori, D.; Migliorati, G.; Nescis, T.; Molla, G.; Fioravanti, M.; and Solerte, S.B. Neuroendocrine correlates of the aging brain in humans. *Neuroendocrinology* 61:464–470, 1995.

Fonda, M.L.; Acree, D.W.; and Auerbach, S.B. The relationships of gamma-aminobutyrate levels and its metabolism to age in brains of mice. *Arch Biochem Biophys* 159:622–625, 1973.

Fowler, C.J. Selective inhibitors of MAO types A and B and their clinical usefulness. *Drugs Future* 7:501–517, 1982.

Froehlich, J.C. The neurobiology of ethanol-opioid interactions in ethanol reinforcement. *Alcohol Clin Exp Res* 20:181A–186A, 1996.

Froehlich, J.C.; Harts, J.; Lumeng, L.; and Li, T.-K. Naloxone attenuates voluntary ethanol intake in rats selectively bred for high ethanol preference. *Pharmacol Biochem Behav* 35: 385–390, 1990.

Froehlich, J.C.; Rhoades-Hall, C.E.R.; Zink, R.; Li, T.-K.; and Christian, J.C.

Heritability of plasma beta-endorphin response following an alcohol challenge. *Alcohol Clin Exp Res* 19:72A, 1995.

Frye, G.D., and Breese, G.R. GABAergic modulation of ethanol-induced motor impairment. *J Pharmacol Exp Ther* 223:750–756, 1982.

Gambert, S.L.; Garthwaite, T.L.; Pontzer, C.H.; and Hagen, T.C. Age-related changes in central nervous system beta-endorphin and ACTH. *Neuroendocrinology* 31:252–258, 1980.

Garcia, R., and Jaffard, R. A comparative study of age-related changes in inhibitory processes and long-term potentiation in the lateral septum of mice. *Brain Res* 620:229–236, 1993.

Gianoulakis, C. Characterization of the effects of acute ethanol administration on the release of beta-endorphin peptides by the rat hypothalamus. *Eur J Pharmacol* 180:21–29, 1990.

Gianoulakis, C. Implications of endogenous opioids and dopamine in alcoholism: Human and basic science studies. *Alcohol Alcohol* 31(Suppl 1):33–42, 1996.

Gianoulakis, C.; Krishnan, B.; and Thavundayil, J. Enhanced sensitivity of pituitary beta-endorphin to ethanol in subjects at high risk of alcoholism. *Arch Gen Psychiatry* 53:250–257, 1996.

Goldman, J., and Cote, L. Aging of the brain: Dementia of the Alzheimer's type. In: Kandel, E.R.; Schwartz, J.H.; and Jessell, T.M., eds. *Principles of Neural Science*. New York: Elsevier, 1991. pp. 974–983.

Gonzales, R. In vivo links between neurochemistry and behavioral effects of ethanol. *Alcohol Clin Exp Res* 20:203A–209A, 1996.

Gozlan, H.; Daval, G.; Verge, D.; Spampinato, U.; Fattaccini, C.M.; Gallissot, M.C.; elMestikawy, S.; and Hamon, M. Aging associated changes in serotoninergic and dopaminergic pre- and postsynaptic neurochemical markers in the rat brain. *Neurobiol Aging* 11:437–449, 1990.

Gutierrez, A.; Khan, Z.U.; Morris, S.J.; and DeBlas, A.L. Age-related decrease of GABA-A receptor subunits and glutamic acid decarboxylase in the rat inferior colliculus. *J Neurosci* 14:7469–7477, 1994.

Handa, R.J.; Cross, M.K.; George, M.; Gordon, B.H.; Burgess, L.H.; Cabrera, T.M.; Hata, N.; Campbell, D.B.; and Lorens, S.A. Neuroendocrine and neurochemical responses to novelty stress in young and old male F344 rats: Effects of d-fenfluramine treatment. *Pharmacol Biochem Behav* 46:101–109, 1993.

Hess, G.D.; Joseph, J.A.; and Roth G.S. Effects of age on sensitivity to pain and brain opiate receptors. *Neurobiol Aging* 2:49–51, 1981.

Hubbell, C.L.; Czirr, S.A.; Hunter, G.A.; Beaman, C.M.; LeCann, N.C.; and Reid, L.D. Consumption of ethanol solution is potentiated by morphine and attenuated by naloxone persistently across repeated daily administrations. *Alcohol* 3:39–54, 1986.

Imperato, A., and DiChiara, G. Preferential stimulation of dopamine release in the nucleus accumbens of freely moving rats by ethanol. *J Pharmacol Exp Ther* 239:219–228, 1988.

Jenab, S., and Inturrisi, C.E. Ethanol and naloxone differentially upregulate delta opioid receptor gene expression in neuroblastoma hybrid (NG108-15) cells. *Mol Brain Res* 27:95–102, 1994.

Jiang, H.K.; Hong, J.S.; and Gallagher, M. Elevated dynorphin in the hippocampal formation of aged rats: Relation to cognitive impairment on a spatial learning task. *Proc Natl Acad Sci USA* 86:2948–2951, 1989.

Johnson, M.; Perry, R.H.; Piggott, M.A.; Court, J.A.; Spurden, D., Lloyd, S.; Ince, P.G.; and Perry, E.K. Glutamate receptor binding in the human hippocampus and adjacent cortex during development and aging. *Neurobiol Aging* 17:639–651, 1996.

Joshi, D.; Bennett, H.P.J.; James, S.; Tousignant, P.; and Miller, M.M. Hypothalamic processing of beta-endorphin in female C57BL/6J mice is altered at middle age. *J Endocrinol* 144:405–415, 1995.

Kaneyuki, T.; Morimasa, T.; and Shohmori, T. Neurotransmitter interactions in the striatum and hypthalamus of mice after single and repeated ethanol treatment. *Acta Med Okayama* 49:13–17, 1995.

Koob, G.F. Drugs of abuse: Anatomy, pharmacology and function of reward pathways. *Trends Pharmacol Sci* 13:177–184, 1992.

Kranzler, H.R.; Burleson, J.A.; Korner, P.; DelBoca, F.K.; Bohn, M.J.; Brown, J.; and Liebowitz, N. Placebo-controlled trial of fluoxetine as an adjunct to relapse prevention in alcoholics. *Am J Psychiatry* 152:391–397, 1995.

Krishnan-Sarin, S.; Jing, S.-L.; Kurtz, D.L.; Zweifel, M.; Portoghese, P.S.; Li, T.-K.; and Froehlich, J.C. The delta opioid receptor antagonist naltrindole attenuates both alcohol and saccharin intake in rats selectively bred for alcohol preference. *Psychopharmacology (Berl)* 120: 177–185, 1995*a*.

Krishnan-Sarin, S.; Portoghese, P.S.; Li, T.-K.; and Froehlich, J.C. The delta2 opioid receptor antagonist naltriben selectively attenuates alcohol intake in rats bred for alcohol preference. *Pharmacol Biochem Behav* 52:153–159, 1995*b*.

Lawlor, B.A.; Sunderland, T.; Mellow, A.M.; et al. Evidence for a decline with age in behavioral responsivity to the serotonin agonist, m-chlorophenylpiperazine, in healthy human subjects. *Psychiatry Res* 29:1–10, 1989.

Le, A.D., and Kiianmaa, K. Initial sensitivity and the development of ethanol tolerance in alcohol drinking (AA) and alcohol avoiding (ANA) rats. In: Kuriyama, K.; Takada, A.; and Ishii, H., eds. *Biomedical and Social Aspects of Alcohol and Alcoholism.* Amsterdam: Elsevier Science Publishers, 1988. pp. 423–426.

Le, A.D.; Poulos, C.X.; Quan, B.; and Chow, S. The effects of selective blockade of delta and mu opiate receptors on ethanol consumption by C57BL/6 mice in a restricted access paradigm. *Brain Res* 630:330–332, 1993.

Le, A.D.; Tomkins, D.M.; and Sellers, E.M. Use of serotonin (5-HT) and opiate-based drugs in the pharmacotherapy of alcohol dependence: An overview of the preclinical data. *Alcohol Alcohol* 31(Suppl 1):27–32, 1996.

Lejoyeux, M. Use of serotonin (5-hdroxytryptamine) reuptake inhibitors in the treatment of alcoholism. *Alcohol Alcohol* 31(Suppl 1):69–75, 1996.

Li, X.-W.; Li, T.-K.; and Froehlich, J.C. Alcohol alters preproenkephalin mRNA content in the shell and the core of the nucleus accumbens. *Alcohol Clin Exp Res* 20:53A, 1996.

Liljequist, S. The competitive NMDA receptor antagonist, CGP 39551, inhibits ethanol withdrawal seizures. *Eur J Pharmacol* 192:197, 1991.

Liljequist, S., and Engel, J. Effects of GABAergic agonists and antagonists on various ethanol-induced behavioral changes. *Psychopharmacology* 78:71–75, 1982.

Little, H.J. Mechanisms that may underlie the behavioural effects of ethanol. *Prog Neurobiol* 36: 171–194, 1991.

Lolova, I., and Davidoff, M. Age-related changes in serotonin-immunoreactive neurons in the rat nucleus raphe dorsalis and nucleus centralis superior: A light microscope study. *Mech Ageing Dev* 62:279–289, 1992.

Lovinger, D.M. Ethanol potentiation of 5-HT3 receptor-mediated ion current in NCB-20 neuroblastoma cells. *Neurosci Lett* 122: 57–60, 1991.

Maloteaux, J.; Octave, J.N.; and Laterre, E.C. Serotonin receptors in human brain: Classification, localization and changes related to aging and dementia in cerebral insufficiency. In: Carlsson, A.; Kanowski, S.; Allain, H.; and Spiegel, R., eds. *Trends in Research and Treatment.* Carnforth, UK: Parthenon, 1989. pp. 15–31.

Mason, B.J.; Ritvo, E.; Morgan, R.O.; Salvato, F.R.; Goldberg, G.; Welch, B.; and Mantero-Atienza, E. A double-blind, placebo-controlled pilot study to evaluate the efficacy and safety of oral nalmefene HCL for alcohol dependence. *Alcohol Clin Exp Res* 18:1162–1167, 1994.

McEntee, W.J., and Crook T.H. Glutamate: Its role in learning, memory, and the aging brain. *Psychopharmacology (Berl)* 111:391–401, 1993.

McGeer, P., and McGeer, E. Enzymes associated with the metabolism of catecholamines, acetylcholine and GABA in human controls and patients with Parkinson's disease and Huntington's chorea. *J Neurochem* 26:65–76, 1976.

McGeer, P.L., and McGeer, E.G. Aging and neurotransmitter system in Parkinson's disease—II. In: Finch, C.E.; Potter, D.E.; and Kenny, A.D., eds. *Aging and Neuroendocrine Relationships.* New York: Raven Press, 1978. pp. 41–52.

Meador-Woodruff, J.H. Update on dopamine receptors. *Ann Clin Psychiatry* 6:79–90, 1994.

Mesco, E.R.; Joseph, J.A.; Blake, M.J.; and Roth, G.S. Loss of D2 receptors during aging is partially due to decreased levels of mRNA. *Brain Res* 545:355–357, 1991.

Miller, M.M., and Zhu, L. Aging changes in the beta-endorphin neuronal system in the preoptic area of the C57BL/6J mouse: Ultrastructural analysis. *Neurobiol Aging* 13:773–781, 1992.

Miller, M.M.; Joshi, D.; Billiar, R.B.; and Nelson J.F. Loss during aging of beta-endophinergic neurons in the hypothalamus of female C57BL/6J mice. *Neurobiol Aging* 12:239–244, 1991.

Murialdo, G.; Filippi, U.; Costelli, P.; Fonzi, S.; Bo, P.; Polleri, A.; and Savoldi, F. Urine melatonin in alcoholic patients: A marker of alcohol abuse? *J Endocrinol Invest* 14:503–507, 1991.

Nabeshima, T.; Yamada, K.; Hayashi, T.; Hasegawa, T.; Ishihara, S.; Kameyama, T.; Morimasa, T.; Kaneyuki, T.; and Shohmori T. Changes in muscarinic cholinergic, PCP, GABA-A, binding in the brains of aged rats and monkeys. *Neurobiol Aging* 55:1585–1593, 1994.

Naranjo, C.A.; Sellers, E.M.; Roach, C.A.; Woodley, D.V.; Sanchez-Craig, M.; and Sykora, K. Zimelidine-induced variations in alcohol intake in non-depressed

heavy drinkers. *Clin Pharmacol Ther* 35:374–381, 1984.

Naranjo, C.A.; Sellers, E.M.; Sullivan, J.T.; Woodley, D.V.; Kadlec, K.; and Sykora, K. The serotonin uptake inhibitor citalopram attenuates ethanol intake. *Clin Pharmacol Ther* 41:266–274, 1987.

Naranjo, C.A.; Sullivan, J.T.; Kadlec, K.E.; Woodley-Remus, D.V.; Kennedy, G.; and Sellers, M. Differential effects of viqualine on alcohol intake and other consummatory behaviors. *Clin Pharmacol Ther* 46:301–309, 1989.

Naranjo, C.A.; Kadlec, K.E.; Sanhueza, P.; Woodley-Remus, D.V.; and Sellers, E.M. Fluoxetine differentially alters alcohol intake and other consummatory behaviors in problem drinkers. *Clin Pharmacol Ther* 47:490–498, 1990.

Noble, E.P.; Berman, S.M.; Ozkaragoz, T.Z.; and Ritchie, T. Prolonged P300 latency in children with the D2 dopamine receptor A1 allele. *Am J Hum Gen* 54:658–668, 1994.

Ogawa, N.; Mizukawa, K.; Haba, K.; Asanuma, M.; and Mori, A. Effects of chronic bifemelane hydrochloride administration on receptors for N-methyl-D-aspartate in the aged rat brain. *Neurochem Res* 17:687–691, 1992.

O'Malley, S.S.; Jaffe, A.J.; Chang, G.; Schottenfeld, R.S.; Meyer, R.E.; and Rounsaville, B. Naltrexone and coping skills therapy for alcohol dependence: A controlled study. *Arch Gen Psychiatry* 49:881–887, 1992.

Oslin, D.; Liberto, J.; O'Brien, J.; Krois, S.; and Norbeck, J. Naltrexone as an adjunctive treatment for older patients with alcohol dependence. *Am J Geriatr Psychiatry* 5:324–332, 1997.

Overstreet, D.H.; Rezvani, A.H.; Pucilowski, O.; and Janowsky, D.S. 5-HT receptors: Implications for the neuropharmacology of alcohol and alcoholism. *Alcohol Alcohol Suppl* 2:207–212, 1995.

Overstreet, D.H.; McArthur, R.A.; Rezvani, A.H.; and Post, C. Amperozide and FG5974: 5-HT-2A receptor antagonists reduce alcohol intake in three strains of alcohol-preferring rats. *Alcohol Clin Exp Res* 20:15A, 1996.

Petkov, V.D.; Petkov, V.V.; and Stancheva, S. Age-related changes in brain neurotransmission. *Gerontology* 34:14–21, 1988.

Pollock, B.G., and Mulsant, B.H. Antipsychotics in older patients: A safety perspective. *Drugs Aging* 6:312–323, 1995.

Porras, R., and Mora, F. Dopamine-glutamate-GABA interactions and ageing: Studies in the striatum of the conscious rat. *Eur J Neurosci* 7:2183–2188, 1995.

Porras, A.; Gisolfi, C.V.; and Mora F. Release of excitatory amino acid neurotransmitters and glutamine along age in the neostriatum of the conscious rat. *Soc Neurosci Abstr* 19:1535, 1993.

Price, M.T.; Olney, J.W.; and Haft, R. Age-related changes in glutamate concentration and synaptosomal glutamate uptake in adult rat striatum. *Life Sci* 28:1365–1370, 1981.

Ratian, S.I.S. Synthesis, modifications, and turnover of proteins during aging. *Exp Gerontol* 31:33–47, 1996.

Reid, L.D., and Hunter, G.A. Morphine and naloxone modulate intake of ethanol. *Alcohol* 1:33–37, 1984.

Rezvani, A. H.; Overstreet, D.H.; and Janowsky, D.S. Genetic serotonin deficiency and alcohol preference in the fawn hooded rats. *Alcohol Alcohol* 25:573–575, 1990.

Robinson, T.E., and Berridge, K.C. The neural basis of drug craving: An incentive-sensitization theory of addiction. *Brain Res Rev* 18:247–291, 1993.

Roose, S.P.; Glassman, A.H.; Attia, E.; and Woodring, S. Comparative efficacy of selective serotonin reuptake inhibitors and tricyclics in the treatment of melancholia. *Am J Psychiatry* 151:1735–1739, 1994.

Rossetti, Z.L., and Carboni, S. Ethanol withdrawal is associated with increased extracellular glutamate in the rat striatum. *Eur J Pharmacol* 283:177–183, 1995.

Rothsein, J.D. Excitotoxicity hypothesis. *Neurology* 47(Suppl 2):S19–S26, 1996.

Rowe, J.W. Physiological and clinical considerations of the geriatric patient. In: Busse, E.W., and Blazer, D.G., eds. *Textbook of Geriatric Psychiatry.* Washington, DC: American Psychiatric Press, 1996. pp. 49–60.

Roy, A., and Linnoila, M. CSF studies on alcoholism and related behaviours. *Prog Neuropsychopharmacol Biol Psychiatry* 13:505–511, 1989.

Sanchez-Prieto, J.; Herrero, I.; Miras-Portugal, M.T.; and Mora, F. Unchanged exocytotic release of glutamic acid in cortex and neostriatum of the rat during aging. *Brain Res Bull* 33:357–359, 1994.

Sass, H.; Soyka, M.; Mann, K.; and Zieglgansberger, W. Relapse prevention by acamprosate: Results from a placebo-controlled study in alcohol dependence. *Arch Gen Psychiatry* 53:673–680, 1996.

Schreiber, R.; Opitz, K.; Glaser, T.; and DeVry, J. Ipsapirone and 8-OH-DPAT reduce ethanol preference in rats: Involvement of presynaptic 5-HT1A receptors. *Psychopharmacology (Berl)* 112:100–110, 1993.

Sellers, E.M.; Toneatto, T.; Romach, M.K.; Somer, G.R.; Sobell, L.C.; and Sobell, M.B. Clinical efficacy of the 5-HT3 antagonist ondansetron in alcohol abuse and dependence. *Alcohol Clin Exp Res* 18:879–885, 1994.

Severson, J.A.; Marcusson, J.; Winblad, B.; and Finch, C.E. Age-correlated loss of dopaminergic binding sites in human basal ganglia. *J Neurochem* 39:1623–1631, 1982.

Shepherd, G.M. Neurotransmitters and neuromodulators. In: Shepherd, G.M., ed. *Neurobiology.* New York: Oxford University Press, 1988. pp. 145–176.

Simpkins, J.W., and Millard, W.J. Influence of age on neurotransmitter function. *Endocrinol Metab Clin North Am* 16:893–917, 1987.

Spanagel, R.; Herz, A.; and Shippenberg, T.S. Opposing tonically active endogenous opioid systems modulate the mesolimbic dopaminergic pathway. *Proc Natl Acad Sci USA* 89:2046–2050, 1992.

Spanagel, R.; Zieglgansberger, W.; and Hundt, W. Acamprosate and alcohol. III. Effects on alcohol discrimination in the rat. *Eur J Pharmacol* 305:51–56, 1996.

Sparks, D.L.; Woeltz, V.M.; and Markesbery, W.R. Alterations in brain monamine oxidase activity in aging, Alzheimer's disease, and Pick's disease. *Arch Neurol* 48:718–721, 1991.

Spokes, G.S. An analysis of factors influencing measurements of dopamine, noradrenaline, glutamate decarboxylase and choline acetylase in human post-mortem brain tissue. *Brain* 102:333–341, 1979.

Streim, J.E.; DiFilippo, S.; Katz, I.R.; Oslin, D.W.; Hearn, R.; and Boyce, A. Adverse events during antidepressant treatment: A reexamination. Abstract

presented at the Gerontologic Society of America Annual Scientific Meeting, 1996.

Sundman, I.; Allard, P.; Eriksson, A.; and Marcusson, J. GABA uptake sites in frontal cortex from suicide victims and in aging. *Neuropsychobiology* 35:11–15, 1997.

Suzdak, P.D.; Schwartz, R.D.; Skolnick, P.; and Paul, S.M. Ethanol stimulates gamma-aminobutyric acid receptor-mediated chloride transport in rat brain synaptoneurosomes. *Proc Natl Acad Sci USA* 83:4071–4075, 1986.

Tamaru, M.; Yoneda, T.; Ogita, K; Shimizu, J.; and Nagata, Y. Age-related decreases in *N*-methyl-D-aspartate receptor complex in the rat cerebral cortex and hippocampus. *Brain Res* 542:83–90, 1991.

Teitler, M., and Herrick-Davis, K. Multiple serotonin receptor subtypes: Molecular cloning and functional expression. *Crit Rev Neurobiol* 8:175–188, 1994.

Tomkins, D.M.; Sellers, E.M.; and Fletcher, P.J. Median and dorsal raphe injections of the 5-HT1A agonist, 8-OH-DPAT, and the GABA-A agonist, muscimol, increase voluntary ethanol intake in Wistar rats. *Neuropharmacology* 33:349–358, 1994.

Tsai, G.; Gastfriend, D.R.; and Coyle, J.T. The glutamatergic basis of human alcoholism. *Am J Psychiatry* 152:332–340, 1995.

Venero, J.L.; deLaRoza, C.; Machado, A.; and Cano, J. Age-related changes on monoamine turnover in hippocampus of rats. *Brain Res* 631:89–96, 1993.

Vescovi, P.P.; Coiro, V.; Volpi, R.; Giannini, A.; and Passeri, M. Plasma beta-endorphin but not met-enkephalin levels are abnormal in chronic alcoholics. *Alcohol Alcohol* 27:471–475, 1992.

Volkow, N.D.; Ding, Y.-S.; Fowler, J.S.; Wang, G.-J.; Logan, J.; Gatley, S.J.; Hitzemann, R.; Smith, G.; Fields, S.D.; and Gur, R. Dopamine transporters decrease with age. *J Nucl Med* 37:554–559, 1996.

Volpicelli, J.R. Uncontrollable events and alcohol drinking. *Br J Addict* 82:381–392, 1987.

Volpicelli, J.R.; Davis, M.A.; and Olgin, J.E. Naltrexone blocks the post-shock increase of ethanol consumption. *Life Sci* 38:841–847, 1986.

Volpicelli, J.R.; Alterman, A.I.; Hayashida, M.; and O'Brien, C.P. Naltrexone in the treatment of alcohol dependence. *Arch Gen Psychiatry* 49:876–880, 1992.

Weiss, F.; Mithciner, M.; Bloom, F.E.; and Koob, G.F. Free-choice responding for ethanol versus water in alcohol preferring (P) and unselected Wistar rats is differentially modified by naloxone, bromocriptine, and methysergide. *Psychopharmacology* 101:178–186, 1990.

Weiss, F.; Lorang, M.T.; Bloom, F.E.; and Koob, G.F. Oral alcohol self-administration stimulates dopamine release in the rat nucleus accumbens: Genetic and motivational determinants. *J Pharmacol Exp Ther* 267:250–258, 1993.

Wenk, G.L.; Walker, L; Price D.L.; and Cork, L.C. Loss of NMDA, but not GABA-A, binding in the brains of aged rats and monkeys. *Neurobiol Aging* 12:93–98, 1991.

Wester, P.; Hardy, J.A.; Marcusson, J.; Nyberg, P.; and Winblad, B. Serotonin concentrations in normal aging human brains: Relation to serotonin receptors. *Neurobiol Aging* 5:199–203, 1984.

Wetterberg, L.; Aperia, B.; Gorelick, D.A.; Gwirtzman, H.E.; McGuire, M.T.; Serafetinides, E.A.; and Yuwiler, A. Age,

alcoholism, and depression are associated with low levels of urinary melatonin. *J Psychiatr Neurosci* 17:215–224, 1992.

Wilkinson, C.W., and Dorsa, D.M. The effects of aging on molecular forms of beta- and gamma-endorphins in rat hypothalamus. *Neuroendocrinology* 43:124–131, 1986.

Wong, D.F.; Wagner, H.N.; Dannals, R.F.; Links, J.M.; Frost, J.J.; Ravert, H.T.; Wilson, A.A.; Rosenbaum, A.E.; Gjedde, A.; Douglass, K.H.; Petronis, J.D.; Folstein, M.F.; Toung, J.K.T.; Burns, H.D.; and Kuhar, M.J. Effects of age on dopamine and serotonin receptors measured by positron tomography in the living human brain. *Science* 21:1393–1396, 1984.

Wong, D. T.; Lumeng, L.; Threlkeld, P.G.; Reid, L.R.; and Li, T.K. Serotonergic and adrenergic receptors in alcohol-preferring and non-preferring rats. *J Neural Transm* 71:207–218, 1988.

Wood, P.L. Multiple opiate receptors: Support for unique mu, delta and kappa sites. *Neuropharmacology* 21:487–497, 1982.

Wozniak, K.M.; Pert A.; Mele, A.; and Linnoila, M. Focal application of alcohols elevates extracelluar dopamine in rat brain: A microdialysis study. *Brain Res* 540:31–40, 1991.

Yoshimoto, K., and McBride, W.J. Regulation of nucleus accumbens dopamine release by the dorsal raphe nucleus in the rat. *Neurochem Res* 17:401–407, 1992.

Yoshimoto, K.; McBride, W.J.; Lumeng, L.; and Li, T.-K. Alcohol stimulates the release of dopamine and serotonin in the nucleus accumbens. *Alcohol* 9:17–22, 1992.

Zeise, M.L.; Madamba, S.G.; Siggins, G.R.; Putzke, J.; and Zieglgansberger, W. The anti-craving substance acamprosate reduces glutamatergic synaptic transmission and high-threshold calcium current in neocortical and hippocampal pyramidal neurons. *Alcohol Clin Exp Res* 18:36A, 1994.

Zelnik, N.; Angel, I.; Paul, S.M.; and Kleinman, J.E. Decreased density of human striatal dopamine uptake sites with age. *Eur J Pharmacol* 126: 175–176, 1986.

COURSE AND CONSEQUENCES

Chapter 11

Medical Consequences of Heavy Drinking by the Elderly

Richard E. Finlayson, M.D., and Richard D. Hurt, M.D.

There are a number of factors that make the medical consequences of heavy drinking by the elderly difficult to summarize. First, because the amount of alcohol consumed and one's tolerance to it tend to decrease as a person ages, the opportunity to study heavy drinking among this population is uncommon. Second, because people age at different rates, comparisons of the medical effects of alcohol between individuals are difficult to make. Third, drinking patterns later in life usually must be understood within the broader context of lifetime drinking, since late-onset drinking is relatively rare and overall lifetime patterns of drinking can be variable (Welte and Mirand 1994). Finally, the recall of drinking histories by the elderly may be unreliable.

In addition to the problems of studying heavy drinking among the elderly, there are factors that make summarizing study findings regarding alcohol consumption itself problematic. For example, researchers define the terms "heavy" and "excessive" drinking differently, and, although we specify the amounts referred to whenever they are noted in the literature, the amounts of alcohol consumed by subjects in various studies are often not clearly expressed in terms of absolute alcohol.

Nevertheless, this chapter examines aging and biological vulnerability to alcohol, common patterns of injury and illness in the elderly associated with excessive alcohol use, and future research directions. This review defines the term "elderly" as beginning at

R.E. Finlayson, M.D., is associate professor of psychiatry at the Mayo Medical School and consultant in adult psychiatry at the Mayo Clinic, 200 First St., S.W., Rochester, MN 55906. R.D. Hurt, M.D., is professor of medicine at the Mayo Medical School and director of the Mayo Nicotine Dependence Center at the Mayo Clinic.

about the age of 60, but this definition is flexible. We do not purport to offer an exhaustive treatment of the subject; rather, we will discuss some of the common areas in which aging and heavy drinking affect the health of older persons.

BIOLOGICAL VULNERABILITY

Population surveys show that absolute amounts of alcohol consumed decrease with age by all classes of drinkers (Mishara and Kastenbaum 1980; Nordstrom and Berglund 1987; Barker and Kramer 1996). According to Atkinson and Kofoed (1982), however, men continue to drink through their sixties at a rate that differs little from the prior decade, but there is a sharp drop in the prevalence of heavy drinking and problem drinking after their mid-seventies. This is not explained simply by the premature deaths of heavy drinkers. Women have lower rates of problem drinking than men and their alcohol use declines earlier, usually during their fifties and sixties.

Biological factors, rendering elderly persons more sensitive to the effects of alcohol than younger persons, are thought to contribute to this decline in use. To illustrate, healthy men's recordings of their subjective feelings of intoxication (Jones and Neri 1985) indicated that the older men experienced greater effects of alcohol than the younger men. Vogel-Sprott and Barrett (1984) noted that when individual differences in blood alcohol levels were controlled for, the amount of alcohol-induced impairment in task performance was still found to increase significantly with age.

In the absence of diseases known to affect absorption, alcohol is readily absorbed by the gastrointestinal tract in later life. After alcohol enters the bloodstream it is distributed throughout the total body water. Alcohol's volume of distribution is affected by body composition factors such as the percentage of body fat (Vestal et al. 1977). Females and the elderly have lower total body water (because they have less lean body mass [muscle] and a higher percentage of body fat) than males and younger persons (Bienenfeld 1987; Dufour et al. 1992). Therefore, when an older female and a young male consume the same amount of alcohol, the female usually will have a higher blood alcohol level than the male.

Beresford and Lucey (1995) provided data suggesting that mechanisms more complicated than differential volumes of distribution may be at work in the elderly. Their findings were as follows:

1. Elderly persons absorbed significantly more alcohol than did young persons when drinking in a fasted, but not in a fed, state. Younger male subjects required a dose of alcohol 15 percent greater and younger females a dose as much as 30 percent greater than their elderly counterparts to reach similar alcohol levels in a fasted state after a single dose.
2. Feeding lessened the blood alcohol concentration through an un-

known mechanism independent of the route of administration.
3. At low doses, elderly subjects showed differentially increased subjective but not objective intoxication effects relative to younger persons.

Alcohol is absorbed primarily in the small intestine and metabolized in the liver. The liver of an older person, assuming that it is not affected by disease, functions as well as the liver of a young person (Scott 1989). The first step in the metabolism of alcohol is facilitated by the oxidative enzyme alcohol dehydrogenase (ADH), which metabolizes alcohol to the toxic substance acetaldehyde. Acetaldehyde, in turn, is metabolized to acetic acid by the enzyme aldehyde dehydrogenase. Some alcohol is metabolized in the gastric mucosa by gastric ADH. This enzyme is lower in females of all ages than in males and decreases with age and being alcoholic (Frezza et al. 1990). Some prescription drugs, such as H2 blockers, also decrease gastric ADH, resulting in an increased blood level of alcohol per unit ingested (Schuckit 1982; Lieber 1988; Caballeria et al. 1989).

COMMON PATTERNS OF INJURY AND ILLNESS

The literature dealing with aging and alcohol reveals that use of alcohol may confer both harm and benefit, as it does in younger persons. The determination of what constitutes harmful drinking is often difficult to make and cannot be based exclusively on the amount of alcohol consumed. The use of other drugs, such as tobacco, and coexisting medical disorders also help to define what constitutes harmful drinking.

Little has been reported about nicotine use and dependence in elderly alcoholics. Nakamura and colleagues (1990), in a study of 1,034 older persons (defined as age 45 years and older), found that alcohol consumption before the age of 40, smoking, being male, and being unmarried were predictors of severe drinking. These variables were not as robust in their effects on drinking in persons age 65 and older compared with those ages 45–64. Jensen and Bellecci (1987) reported that non-elderly men had a higher prevalence of alcohol abuse, higher current alcohol intake, and greater smoking of cigarettes compared with men ages 65–75. Data from the Mayo Clinic (Finlayson et al. 1988) revealed that among elderly alcoholics, 70 percent of whom were male, the prevalence of reported nicotine dependence was 52 percent. The apparent decline in the prevalence of tobacco use among elderly alcoholics may be related to premature death from smoking-related illness, the phenomenon of late-onset alcoholism, or a decreased prevalence of smoking among alcoholics in general. Orleans and Hutchinson (1993) documented a strong desire to stop smoking and a history of one or more attempts to quit by alcoholics in residential treatment.

The use of tobacco by drinkers has been identified as a cofactor in some of the illnesses associated with heavy alcohol use. The relationship is so close, in fact, that it is often difficult to separate the effects of these two

substances from an epidemiologic perspective. The importance of tobacco use in the clinical outcomes of alcoholics was demonstrated in a recent report on mortality in a population-based cohort (N = 845) following inpatient addictions treatment (Hurt et al. 1996). The patients were followed through the medical record linkage system of the Rochester Epidemiology Project. The cohort was followed up after the index admission for a total of 8,913 person years (mean [SD] 10.5 [5.6] years, range 0.1–21.8 years per patient). Death certificates were obtained on 214 of the 222 patients who died, and the underlying cause of death was classified as to whether it was alcohol or tobacco related. The subjects had an increased cumulative mortality (48.1 percent vs. the expected 18.5 percent) that was associated with tobacco-related illnesses (50.9 percent) more often than alcohol-related illnesses (34.1 percent). Some mortality was found to be associated with factors related to both alcohol and tobacco.

Data have been extracted from the study of Hurt and colleagues (1996) on 104 alcoholics who were 60 years or older at the time of the index admission, and they are reported for the first time in this monograph. There were 66 men and 38 women with an average age of 66 years at the time of admission. As illustrated in table 1, there were 76 deaths observed during the followup period, more than double the expected number of 36.3. Death certificates were available for 74 of these patients, and they were used to tabulate the most common underlying causes of death (table 2). Tobacco-related illnesses accounted for 40 deaths, while alcohol-related illnesses accounted for 23 deaths. As with the whole cohort, coronary artery disease (CAD) (n = 13) and chronic obstructive pulmonary disease (n = 10) were the most common single causes of death in the elderly. It is noteworthy, however, that 17 died of cancer: 2 each with lung, laryngeal, and hepatocellular cancer; 1 each with esophageal, duodenal, pancreatic, prostate, colon, kidney, and breast cancer, leukemia, and melanoma; and 2 with cancers not specified as to type. Unlike the 214 non-elderly alcoholics for whom death certificates were available, suicide was not reported as an underlying cause of death in any of the elderly group. There were 13 such deaths in those who were younger than 60 at the time of their index admission. This is surprising given that suicide rates tend to increase with age, especially in white men (who predominated in this study). Ghosh and Victor (1994) reported data from the literature indicating that suicide rates increase rapidly after age 65 in white men.

The sections that follow focus on broad categories of injury and illness with which alcohol and aging interact. The role of comorbid tobacco use will be discussed whenever information is available.

Injury

The literature regarding the relationship between drinking and falls among the elderly is mixed. Nelson and colleagues (1992) performed a case-controlled study of 320 persons

Table 1. Underlying Causes of Death Among 104 Elderly Olmsted County, Minnesota, Residents First Admitted to an Inpatient Addiction Program From 1972 Through 1983.

	Related to[2]		Deaths		Relative Risk
Cause of Death[1]	Alcohol	Smoking	Observed	Expected	(95% confidence interval)
All causes	"+"	"+"	76	36.3	2.1 (1.7–2.6)
All alcohol related	+	"+"	23	6.5	3.5 (2.2–5.3)
All tobacco related	"+"	+	40	24.6	1.6 (1.2–2.2)
Alcohol but not tobacco related	+	—	16	3.9	4.1 (2.4–6.7)
Tobacco but not alcohol related	—	+	29	20.8	1.4 (0.9–2.0)
Related to both alcohol and tobacco	+	+	11	7.0	1.6 (0.8–2.8)
Neither alcohol nor tobacco related	—	—	18	7.5	2.4 (1.4–3.8)

[1] Causes of death are classified in accord with the *International Classification of Diseases, Ninth Revision* (ICD-9).
[2] A plus sign (+) indicates that this ICD-9 underlying cause of death is related to alcohol or tobacco in the respective columns; a plus sign in quotation marks ("+") indicates that some but not all of the ICD-9 codes included in this group are related to alcohol or tobacco; and a dash (—) indicates that there is no relationship to alcohol or tobacco.

Table 2. Causes of Death as Recorded on Death Certificates of 74 Alcoholics Age 60 Years or Older.

Disorder	Number
Coronary artery disease	13
Cancer[1]	17
Chronic obstructive pulmonary disease	10
Alcoholic liver disease	7
Alcohol dependence syndrome (nonspecified)	3
Cardiomyopathy	3
Stroke	3
Diabetes mellitus	2
Psychosis	2
Pneumonia	2
Miscellaneous	12

[1] See text for types of cancer.

age 65 or older who sought treatment at hospitals in the South Miami Beach area of Florida. The average time from injury to interview was 2.5 months. The authors defined light drinking as 1–3 drinks per week (one drink represented by a can or bottle of beer, a glass of wine, or a cocktail), moderate drinking as 4–13 drinks per week, and heavy drinking as 14 or more drinks per week. No association was found between

injuries due to falls and average weekly alcohol use.

O'Loughlin and colleagues (1993) studied falls among noninstitutionalized elderly in Montreal, Canada. Multivariate analyses revealed that dizziness and being physically active, but having mobility problems, were most significantly related to falls and injuries. Contrary to a common assumption, daily alcohol use had a weakly protective effect against injury due to falls. Data from a level I trauma center (Mosenthal et al. 1995) demonstrated that the elderly accounted for 14 percent of admissions for falls, but for more than 50 percent of the deaths, often after having fallen from low heights. The falls among the elderly group were not identified as being related to alcohol.

Data from the Framingham study, controlled for age, gender, weight, and smoking (Felson et al. 1988) revealed that moderate to heavy alcohol consumption (2–6 oz/wk and ≥ 7 oz/wk, respectively) was only marginally and insignificantly associated with hip fractures in those age 65 and older. Those younger than age 65 had a significant and substantially increased risk of hip fracture.

Another issue is whether alcohol consumption contributes to osteoporosis, a recognized risk factor for bone fracture. A 16-year study of a cohort of men in a midwestern U.S. hospital revealed that men who smoked and used alcohol at levels greater than the median for the population had rates of bone loss twice those of men who were below the median level for both variables (Slemenda et al. 1992). Felson and colleagues (1995) used Framingham data to determine the relationship between alcohol consumption and bone mineral density in the elderly. The association of alcohol intake with bone density was examined after adjustment for age, weight, height, smoking, and, in women, age at menopause and years of estrogen use. The data revealed that bone density in elderly women and men is enhanced by moderate consumption of alcohol (7–13 oz/wk), with the effect being more pronounced in women. The authors speculated that augmentation of endogenous estrogen by alcohol may explain the findings. Ginsburg and colleagues (1996) reported that moderate (0.7g/kg) alcohol ingestion was associated with a threefold increase in circulating estradiol in postmenopausal women on estrogen replacement therapy.

Closed head injuries also are common among the elderly, and alcohol consumption may play a role. Kono and colleagues (1989), in a study of subdural hematoma and hygroma in elderly patients with dementia in Japan, identified male sex, alcohol abuse, and wandering as risk factors for chronic subdural hematoma. Chronic alcoholism and alcohol intoxication also have been identified as risk factors for positional asphyxiation (Bell et al. 1992). These authors noted that elderly, demented persons wearing restraint vests ("poseys") may be at risk due to a hyperflexed position of the head.

Alcohol was identified in the blood screens of 14 percent of 180 elderly drivers admitted to a level 1 trauma

center after motor vehicle crashes (Higgins et al. 1996). Whether older pedestrians who are under the influence of alcohol are at risk of injury in motor vehicle crashes was addressed by Bradbury (1991) in Scotland. A positive correlation was found in only 2 of a group of 34 such pedestrian injuries.

Being elderly and alcoholic may contribute to loss of balance and falling against a space heater, smoking in bed and falling asleep, and other mishaps associated with the risk of fires. Brodzka and colleagues (1985) studied 277 persons admitted consecutively for the inpatient treatment of burns. The most common predisposing factors for these burns were alcohol and drug abuse, physical and mental illness, and advanced age. Cigarette smoking was involved in 14.1 percent of the fires. Burn injury is a very serious problem for the elderly for a variety of reasons. Wound healing seems to be normal in the elderly person without concomitant disease, but disorders that delay or impair the healing of wounds, such as diabetes mellitus and peripheral vascular disease, are common among the elderly (Holt et al. 1992; Van de Kerkhof et al. 1994). Infections as a complication of burn injury may be especially dangerous, for example, for the immune-suppressed elderly person.

Thus, the literature is mixed as to the association between alcohol use and injury among the elderly. Nonetheless, laboratory studies do demonstrate that alcohol has effects in the elderly that could be expected to put them at risk for accidents and injuries. Burke and colleagues (1992), in a study of elderly persons in Australia, found a positive correlation between the consumption of the daily equivalent of 20 mL of ethanol and a postural fall in systolic blood pressure. The latter could, in susceptible individuals, affect consciousness and balance. Vogel-Sprott and Barrett (1984) demonstrated that when a group of male "social drinkers" (ages 19–63) were given 0.72 mL absolute alcohol per kilogram of body weight divided equally in two "drinks" 20 minutes apart and then asked to perform balance beam and bead-stringing tasks, alcohol-induced impairment of task performance increased with age. Other indirect evidence that alcohol may predispose to falls in the elderly is that when attention-dividing reaction time tests were administered to young adult and elderly subjects, both groups had a significantly increased risk of obstacle contact while negotiating obstacles when their attention was divided; however, dividing attention degraded the obstacle avoidance abilities of the older subjects significantly more than those of the younger subjects (Chen et al. 1996). One might hypothesize that the direct effects of alcohol on mental concentration, the social chaos commonly associated with alcohol abuse, and other related factors could contribute to an increased risk of falls and other accidents among the elderly.

Malnutrition

Bonjour and colleagues (1996) cited evidence from the medical literature that a lack of both micronutrients and macronutrients is strongly implicated

in the pathogenesis and consequences of hip fracture in the osteoporotic elderly. The economic and other living circumstances of elderly persons would likely contribute to the vulnerability to malnutrition of older alcohol-consuming persons. Barboriak and colleagues (1978), in a study of elderly men in a Veterans Administration domiciliary, noted that alcohol consumers ate fewer food calories and had lower serum albumin levels than non-users. The lower calorie intake extended to the three main energy sources—carbohydrates, protein, and fat—and occurred even when ample food was available.

The literature does report some data concerning the role of alcohol in reducing specific dietary factors. A review by Iber and colleagues (1982) noted that in the North American population, 5 percent of those older than age 60 suffered thiamin deficiency. The effect was most marked in the poor, those confined in institutions, and those with illness. O'Keeffe and colleagues (1994) found an association between thiamin deficiency and delirium in hospitalized elderly. Ben-Hur and colleagues (1992) reported a series of 13 persons from Israel admitted to a hospital because of complications from thiamin deficiency. Alcohol use was not as important a factor in thiamin deficiency as being solitary, elderly, and of low socioeconomic status. Rosenberg and colleagues (1982), in reporting the findings of a consensus panel, noted that alcohol use is probably the single most important factor in folate deficiency among both the elderly and the non-elderly. The report also cited evidence that the elderly poor as well as other persons of low socioeconomic status are at increased risk of thiamin deficiency.

Many of these reports are limited by their focus on derelict alcoholics. In a study of middle-class alcoholics who received treatment for their alcoholism, Hurt and colleagues (1981) found that the nutrition of these subjects was adequate overall. The mean age of these 58 alcoholics was 44 years, with about 10 percent being 60 years or older. The pretreatment diet histories were generally found to meet recommended daily allowances of vitamins and minerals, and the nutrients derived from the major food groups increased significantly in response to alcoholism treatment.

Infectious Disease

Natural immunity tends to decline with age (Fattal-German 1992; Evans 1995; Licastro et al. 1995). Alcohol ingestion may also play a role in altering the immune response. Bounds and colleagues (1994) evaluated peripheral blood mononuclear cell cytotoxicity before and after a dose of alcohol designed to simulate social drinking. Interleukin-2–induced lymphokine-activated killer activity was significantly reduced after ethanol ingestion compared with preingestion. Natural killer cell activity was found to be suppressed in vitro by 80 mg/dL for 4 hours of alcohol exposure, suggesting the possibility that the direct effect of alcohol on natural killer cell activity may be one of the causes of infections and malignancy in chronic alcoholics (Ochshorn-Adelson et al. 1994).

Ruben and colleagues (1995) recruited 417 noninstitutionalized persons age 65 or older and conducted 24 months of infection surveillance in the Pittsburgh, Pennsylvania, area. A major objective of the study was to identify risk factors for infection in this age group. Alcohol as a risk factor was represented by either current users (n = 121) or those never having used alcohol (n = 184). (The 112 patients not accounted for are presumably former alcohol users, many of whom may have been alcohol abusers. It would be of interest to know if they were at increased risk of having infections.) Only a history of lung problems (pneumonia and bronchitis) and self-reported difficulty with urination remained significant after the analysis. The study did not analyze the association of infection with the amounts of alcohol consumed by these elderly.

The association between alcohol abuse and morbidity and mortality in the elderly due to community-acquired pneumonia, including tuberculosis, has been the most notable example of impaired resistance to infection (Marrie 1990; Koivula et al. 1994). Takada and colleagues (1994) reported data from Japan indicating an association between fulminant Legionnaire's disease and advanced age combined with alcoholism. A Swedish study (Ortqvist 1990) examined prognosis of patients with community-acquired pneumonia treated in the hospital. Factors significantly associated with mortality were male sex, alcoholism, extrapulmonary complications, and the absence of leukocytosis. Surprisingly, the researchers did not find advanced age to be a negative prognostic factor, citing the generally good health of the Swedish population.

Body Temperature Regulation

Accidental hypothermia occurs in all types of climates. Thus the patterns of presentation of this disorder differ regionally both in the United States and internationally. A study by Thomas (1988) of accidental hypothermia cases admitted to a university center in the Deep South of the United States (a warm climate) revealed that being elderly was the greatest risk factor (65 percent), with alcohol use being a less common risk factor (33 percent). Albiin and Eriksson (1984), in a study of 51 cases of fatal accidental hypothermia in northern Sweden, noted that the most common risk factors were being elderly, being under the influence of alcohol, and having chronic alcohol abuse. Data from the Negev desert in the south of Israel revealed that the most common predisposing factors for hypothermia were infections (54.5 percent), renal failure (29.5 percent), and diabetes mellitus (29.5 percent). Alcoholism was relatively uncommon (13.6 percent) as an associated factor for hypothermia.

Being elderly also increases the risk for heatstroke. This was demonstrated in the Kansas City heat wave of 1980, in which the major risk factors for hyperthermia were being elderly, black, and of low socioeconomic status. Alcoholism was present in 21 percent of those afflicted (Tucker et al. 1985). The role of alcoholism in these cases

is likely a combination of physiological, economic, and other factors.

Cardiovascular Disorders

Cardiovascular disease and CAD, in particular, increases with age and is a leading cause of death in the United States. Recent research confirms the benefits of light to moderate drinking for the elderly and younger persons in reducing cardiovascular risk (Scherr et al. 1992; Huijbregts et al. 1995). Klatsky and colleagues (1992) reported that light drinking reduced cardiovascular risk more among the elderly than among the young.

However, heavy use of alcohol increases the risk of cardiovascular and other diseases. Heavy drinking (six or more drinks per day) in the study by Klatsky and colleagues (1992) was associated with increased risk of death from both cardiovascular and noncardiovascular disease, and the effect was most pronounced among women and the young. Smith (1995) noted that the literature to date documents that heavy alcohol use produces direct toxic effects upon the heart (alcoholic cardiomyopathy), contributes to hypertension, and is associated with cardiac arrhythmias. The well-known association between cigarette smoking and alcohol consumption, with the heaviest smokers also being the heaviest drinkers and vice versa, likely accounts for some of the increased mortality with higher levels of alcohol consumption (Craig and Van Natta 1977).

A 1995 review by Smith examined the validity of the concept of a "U-shaped curve," in which moderate drinkers have a lower total and cardiovascular mortality than total abstainers or heavy drinkers. The assessment of "light" daily drinking as possibly being protective for CAD is complicated by confounding factors such as rates of smoking, hypertension, obesity, and physical activity. Former "heavy drinkers," common among the elderly, are reported to have an increased risk of CAD, perhaps because some are persistent heavy smokers. Casswell (1993) argued that the growing public perception that drinking has a protective effect against CAD and will delay mortality among the elderly may encourage individuals to drink more. Several years earlier, Klatsky and colleagues (1986) sounded a similar warning: Although one to two drinks of alcohol per day protects against CAD, this should not be used to justify heavier drinking. Likewise, smokers may mistakenly believe that their drinking will offset the increased risk of CAD known to be associated with smoking.

According to Klatsky (1987), excessive elevation of blood pressure, a major risk factor for cardiac disease, may result from consuming three or more drinks per day. This effect is most prominent in men, Caucasians, and persons over age 55. A study by Criqui and colleagues (1981) identified alcohol consumption of 30 mL or more as having the effect of increasing systolic and diastolic blood pressure independent of age, and in men and women.

Cerebrovascular Disorders

Commonly recognized risk factors for stroke include arterial hypertension, diabetes mellitus, smoking, obesity, hy-

perlipidemia, and previous stroke. The role of alcohol as a risk factor for stroke in the elderly has been explored only to a limited extent. Beghi and colleagues (1992), in a case study of an Italian population, assessed the role of alcohol as a risk factor for cerebral infarction and hemorrhage in 200 middle-age and elderly stroke patients. The role of alcohol as a risk factor was small (odds ratio 1.86) and was practically lost after adjustment for the most common risk factors for cerebrovascular disorders. Lee and colleagues (1995) reported similar results from Taiwan, in a nationwide study of residents age 65 years or older, in which cigarette smoking was noted to be an independent risk factor for stroke, while consumption of 367.5 g/wk or less of alcohol was not. Although excessive drinking (> 367.5 g/wk of alcohol) was a risk factor for cerebral infarction in univariate analysis, this effect was lost after adjustment for other confounding factors.

A study by Hansagi and colleagues (1995) focused on alcohol consumption and stroke mortality in a cohort of 15,077 middle-age and older men and women in Sweden. The subsequent 20-year followup revealed 769 deaths from stroke, of which 574 were ischemic. Relative mortality risks were estimated from logistic regression analyses, with lifelong alcohol abstainers as a reference group. The study was controlled for age and smoking. No association was found between alcohol intake and hemorrhagic stroke. An elevated risk of ischemic stroke was found for men who drank, some to intoxication, but infrequently. Among women, only ex-drinkers, many of whom had been problem drinkers, had an elevated risk of dying from ischemic stroke. One important point taken from the study was that ex-drinkers should not be included with lifelong abstainers in assessment of health risk because the former tend to be at high risk for health problems, including stroke.

Studies of stroke and its relationship to alcohol use have not commonly focused just on the elderly, but certain generalizations about stroke in middle to old age can be drawn. Heavy alcohol consumption is associated as a risk factor with both hemorrhagic and ischemic stroke as well as coronary disease (Palomäki and Kaste 1993; Beghi et al. 1995; Iso et al. 1995; Camargo 1996). Ischemic stroke, in general, occurs several times more often than does hemorrhagic stroke. Its prevention is therefore of greater public health importance. Some literature has suggested that light alcohol consumption has a protective effect against ischemic stroke (Palomäki and Kaste 1993), but studies are divided on this point (Camargo 1996).

Gastrointestinal Disease

Liver cirrhosis in the elderly has received relatively little attention in the medical literature. Tanaka and colleagues (1987) conducted a followup study of 582 cases of cirrhosis in patients of all ages in Japan. Etiologies of the cirrhosis were alcoholism, 39.5 percent; cryptogenic, 39.5 percent; and hepatitis B, 21 percent. The survival rate of patients younger than 50 was significantly higher than those older than

59; however, when corrected for life expectancy in the general population, liver cirrhosis patients survived for 40 to 44 percent of the life expectancy of the general population in Japan in all age groups.

A study by Gruenewald and Ponicki (1995) evaluated the extent to which beverage-specific alcohol sales (beer, wine, and distilled spirits) are associated with cirrhosis mortality rates. Data were drawn from 50 states of the United States taken between 1975 and 1986. After controlling for various covariates, the analyses showed that the sales of distilled spirits had an effect on cirrhosis mortality rates. There were no significant effects for either beer or wine sales. In addition, only one covariate in the analysis proved to be significantly related to cirrhosis mortality rates, and that was the age distribution of state populations over time. Higher mortality rates were observed in populations that had greater proportions of individuals ages 30–59 or older than 75. The upturn in mortality after 75 years of age, according to the authors, may or may not be directly attributable to alcohol-related cirrhosis mortality rates.

Noda and colleagues (1996) studied the progression of chronic hepatitis C first to cirrhosis and then to hepatocellular carcinoma in 115 subjects with hepatitis C and a history of blood transfusion. The time from transfusion to diagnosis in hepatocellular carcinoma patients was 26 ± 6 and 31 ± 9 years for those with alcohol levels of ≥ 46 g/d and < 46 g/d, respectively ($p < 0.05$). The period from transfusion to diagnosis of cirrhosis and/or hepatocellular carcinoma showed a significant negative correlation with the time elapsed since transfusion. The authors concluded from their data that alcohol drinking may promote hepatocellular carcinoma in patients with chronic hepatitis C, and that chronic hepatitis C infection in the elderly promotes cirrhosis and hepatocellular carcinoma. The study also revealed a positive association between the amount of alcohol consumed and the extent of tumor growth.

The literature is mixed as to the relationship between pancreatic cancer and drinking. A 1986 review by Velema and colleagues reported insufficient epidemiologic evidence for a causal relationship, and it did not focus exclusively on the elderly. Fernandez and colleagues (1995) reported data from a population in northern Italy in which the risk of cancer occurring 5 or more years after pancreatitis was more common in persons younger than age 60 than in those who were older. In another Italian study (case controlled) by Tavani and colleagues (1997), no interaction was observed between total alcohol intake and pancreatic cancer in separate strata of age, sex, education, and smoking status. Even high alcohol intake—that is, > 8 drinks per day—did not increase the risk of pancreatic cancer. Shibata and colleagues (1994) reported that there was no association between pancreatic cancer and alcohol or tobacco in a cohort of elderly persons in a retirement community. The question unanswered is whether alcohol adds to pancreatic cancer risk apart

from the causal relationship between alcohol and pancreatitis.

Other Types of Cancer

It would be expected that smoking-related cancers would be common among alcoholics, given their high rate of smoking. However, studies indicate that this may not always be the case. For example, in a 20-year followup of 133 treated alcoholics, 6 percent of the deaths were due to lung cancer (O'Connor and Daly 1985). Similarly, in the large epidemiologic study (n = 845) by Hurt and colleagues (1996), lung cancer was the underlying cause of death in only 6 percent of the deaths in treated alcoholics. The relative risk of dying from lung cancer was not significantly different for age- and gender-matched populations. This rate was also lower than the 25 percent death rate from lung cancer reported in a 20-year followup of 99 alcoholic men (Marshall et al. 1994). The differences may be related to the sample size or the young age of the Hurt cohort, since we would expect to see more cases of lung cancer as the cohort is followed into old age. The most likely cause of the inconsistency is competing causes of death: fewer patients will die of lung cancer if they die earlier from other causes, such as CAD or suicide. Alcoholics who were older at the time of their first admission for alcohol treatment may display a very different spectrum of underlying causes of death than their younger counterparts.

Cancers of the head and neck represent a common and serious problem in later life. Koch and colleagues (1995) stated that squamous cell carcinoma, for example, primarily affects persons in the fifth to seventh decades of life, with occasional cases arising at an older age. Their study group involved persons who developed squamous cell carcinoma after their 75th birthdays and compared them with those who developed this cancer between the ages of 40 and 70 years. The presence or absence of p53 gene mutation was determined from a subset of the two groups. Alcohol and tobacco exposure was assessed. The younger patients were more likely than the older ones to demonstrate the genetic change and to have greater alcohol and tobacco exposure in conjunction with squamous cell carcinoma. The older group had significantly less exposure to alcohol and tobacco and were more susceptible to other cancers, especially cancers outside the upper airway or digestive tract.

Indirect evidence for a lesser role for alcohol and tobacco in the development of cancers of the head and neck at advanced age was reported by Ehlinger and colleagues (1993) in a study of 46 patients 75 years or older who had cancer of the oral cavity. Premalignancies and other primary cancers were common. Fifteen patients had a negative history of tobacco and alcohol consumption. Fourteen of these were women. By contrast, Wray and McGuirt (1993) noted that, from a record review of 128 persons who had used smokeless tobacco exclusive of other carcinogens and developed oral carcinoma, most were elderly white women with a mean age of 78 years. Seventy-eight percent of these

women had used smokeless tobacco for more than 40 years. Alcohol use was not implicated as a factor by the authors. Gaillard-Perera and Gaillard (1992) reported that persons older than 60 who had oral cancer not associated with alcohol and tobacco use had more favorable prognoses than those who had used alcohol and tobacco.

Laboratory Abnormalities

The diagnosis of alcoholism at all ages is generally based upon clinical assessment, with laboratory findings being regarded as ancillary. Laboratory abnormalities may be more helpful in determining alcoholism in older than in younger patients. Hurt and colleagues (1988) reported abnormalities on commonly used hematological and biochemical tests, which were detected more frequently in elderly alcoholics than in non-elderly alcoholics who were hospitalized for treatment of their alcoholism. The laboratory values were compared in two age cohorts: those 64 or younger and those 65 or older. The most significant differences were noted in the *frequency of abnormalities* in mean corpuscular volume (increased), uric acid (increased, females > males), serum albumin (decreased), mean corpuscular hemoglobin (increased in men), and aspartate aminotransferase (increased).

Heavy drinking may be detected by commonly used laboratory tests at any age. The determination of carbohydrate-deficient transferrin (CDT) has been reported to be a useful test for identifying heavy drinking. The literature is mixed as to its sensitivity and specificity when compared, for example, with mean corpuscular volume and gamma-glutamyl transpeptidase. CDT has been found to be elevated primarily in the early stage of alcoholic liver disease (Niemela et al. 1995). We are not aware of any study that has specifically examined the usefulness of CDT in the elderly population; however, in a study by Stibler and colleagues (1988), CDT levels were found to be weakly correlated with age in men. The diminished usefulness of CDT in older persons is suggested by the study of Fagerberg and colleagues (1994), in which the investigators concluded that the sensitivity and specificity of CDT determinations were low in 439 treated hypertensive men, ages 54–77.

FUTURE RESEARCH DIRECTIONS

The medical disorders discussed in this and other chapters of this monograph contribute to a web of comorbidity which is, as yet, poorly understood. Inquiry into the nature of the relationship of these illnesses, aging, and alcohol is a necessary step leading to early recognition and treatment.

One very interesting area for future study is that of the long-term effects of alcohol abuse, including cases in which the elderly person no longer drinks alcohol or drinks very little. The data from Saunders and colleagues (1991), drawn from the Liverpool longitudinal study, raised some very interesting questions. These investigators reported that men with a history of heavy drinking for 5 years or more at any time in their lives have a greater than

fivefold risk of having a mental disorder as they grow older. The observation that alcoholics who have abstained for years are at increased risk of ischemic stroke, compared with lifelong abstainers (Hansagi et al. 1995), raises similar concerns. The same issues apply to tobacco use, which has been an important cause of mortality in both young and old alcoholics, and which deserves more research. The morbidity caused by tobacco is likely to play an important role in the reduced quality of life experienced by elderly alcoholics, even more so than their younger counterparts. More effective interventions are needed for nicotine dependence to produce desired short-term and long-term benefits.

As current cohorts of multiple illicit drug users age, there is a possibility that their use patterns will continue into old age. The interaction of alcohol with other drugs such as cannabis, cocaine, and amphetamine in the aging body will present new challenges on all fronts of investigation. Age will not likely alter the effects of these various substances in a uniform manner. New treatment paradigms will likely be needed to treat the addicted elderly and, with them, methods of assessing treatment outcomes.

Finally, the problems associated with data collection and analysis in the elderly, who often have one or more comorbid medical or drug use problems, present the investigator with a great challenge. More information is needed concerning the reliability of drinking histories that rely on patient self-report. Methods for multivariate analysis must be refined for study of this population.

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REFERENCES

Albiin, N., and Eriksson, A. Fatal accidental hypothermia and alcohol. *Alcohol Alcohol* 19:13–22, 1984.

Atkinson, R.M., and Kofoed, L.L. Alcohol and drug abuse in old age: A clinical perspective. *Subst Alcohol Actions Misuse* 3:353–368, 1982.

Barboriak, J.J.; Rooney, C.B.; Leitschuh, T.H.; and Anderson, A.J. Alcohol and nutrient intake of elderly men. *J Am Diet Assoc* 72: 493–495, 1978.

Barker, J.C., and Kramer, B.J. Alcohol consumption among older urban American Indians. *J Stud Alcohol* 57:119–124, 1996.

Beghi, E.; Bogliun, G.; Cosso, P.; Fiorelli, G.; Lorini, C.; Mandelli, M.; Romano, R.; and Sanguineti, I. Cerebrovascular disorders and alcohol intake: Preliminary results of a case-control study. *Ital J Neurol Sci* 13:209–214, 1992.

Beghi, E.; Boglium, G.; Cosso, P.; Fiorelli, G.; Lorini, C.; Mandelli, M.; and Bellini, A. Stroke and alcohol intake in a hospital population. A case control study. *Stroke* 26(9):1691–1696, 1995.

Bell, M.D.; Rao, V.J.; Wetli, C.V.; and Rodriguez, R.N. Positional asphyxiation in adults: A series of 30 cases from the Dade and Broward County Florida Medical Examiner Offices from 1982 to 1990. *Am J Forensic Med Pathol* 13:101–107, 1992.

Ben-Hur, T.; Wolff, E.; and River, Y. Thiamin deficiency is common in Israel. *Harefuah* 123:382–384, 436, 435, 1992.

Beresford, T.P., and Lucey, M.R. Ethanol metabolism and intoxication in the elderly. In: Beresford, T.P., and Gomberg, E.S.L., eds. *Alcohol and Aging*. New York: Oxford University Press, 1995. pp. 117–127.

Bienenfeld, D. Alcoholism in the elderly. *Am Fam Physician* 36:163–169, 1987.

Bonjour, J.P.; Schurch, M.A.; and Rizzoli, R. Nutritional aspects of hip fractures. *Bone* 18(3 suppl):139S–144S, 1996.

Bonneh, D.Y., and Shvartzman, P. Hypothermia in the elderly in the Negev. *Harefuah* 127:509–511, 576, 1994.

Bounds, W.; Betzing, K.W.; Stewart, R.M.; and Holcombe, R.F. Social drinking and the immune response: Impairment of lymphokine-activated killer activity. *Am J Med Sci* 307:391–395, 1994.

Bradbury, A. Pattern and severity of injury sustained by pedestrians in road traffic accidents with particular reference to the effect of alcohol. *Injury* 22:132–134, 1991.

Brodzka, W.; Thornhill, H.L.; and Howard, S. Burns: Causes and risk factors. *Arch Phys Med Rehabil* 66:746–752, 1985.

Burke, V.; Beilin, L.J.; German, R.; Grosskopf, S.; Ritchie, J.; Puddey, I.B.; and Rogers, P. Postural fall in blood pressure in the elderly in relation to drug treatment and other lifestyle factors. *Q J Med* 84:583–591, 1992.

Caballeria, J.; Baraona, E.; Rodamilans, M.; and Lieber, C.S. Effects of cimetidine on gastric alcohol dehydrogenase activity and blood ethanol levels. *Gastroenterology* 96:388–392, 1989.

Camargo, C.A., Jr. Case-control and cohort studies of moderate alcohol consumption and stroke. *Clin Chim Acta* 246(1–2):107–119, 1996.

Casswell, S. Public discourse on the benefits of moderation: Implications for alcohol policy development. *Addiction* 88:459–465, 1993.

Chen, H.C.; Schultz, A.B.; Ashton-Miller, J.A.; Giordani, B.; Alexander, N.B.; and Guire, K.E. Stepping over obstacles: Dividing attention impairs performance of old more than young adults. *J Gerontol A Biol Sci Med Sci* 51:M116–M122, 1996.

Craig, T.J., and Van Natta, P.A. The association of smoking and drinking habits in a community sample. *J Stud Alcohol* 38(7):1434–1439, 1977.

Criqui, M.H.; Wallace, R.B.; Mishkel, M.; Barrett-Connor, E.; and Heiss, G. Alcohol consumption and blood pressure. The lipids research prevalence study. *Hypertension* 3(5):557–565, 1981.

Dufour, M.C.; Archer, L.; and Gordis, E. Alcohol and the elderly. *Clin Geriatr Med* 8:127–141, 1992.

Ehlinger, P.; Fossion, E.; and Vrielinck, L. Carcinoma of the oral cavity in patients over 75 years of age. *Int J Oral Maxillofac Surg* 22:218–220, 1993.

Evans, J.G. General medicine and geriatrics, where is the difference? The example of infective disease. *Schweiz Med Wochenschr* 125:1847–1854, 1995.

Fagerberg, B.; Agewall, S.; Berglund, A.; Wysocki, M.; Lundberg, P.A.; and Lindstedt, G. Is carbohydrate-deficient transferrin in serum useful for detecting excessive alcohol consumption in hypertensive patients? *Clin Chem* 40(11):2057–2063, 1994.

Fattal-German, M. Immunocompetence in the elderly. *Ann Pharm Fr* 50:13–24, 1992.

Felson, D.T.; Kiel, D.P.; Anderson, J.J.; and Kannel, W.B. Alcohol consumption and hip fractures: The Framingham Study. *Am J Epidemiol* 128:1102–1110, 1988.

Felson, D.T.; Zhang, Y.; Hannan, M.T.; Kannel, W.B.; and Kiel, D.P. Alcohol intake and bone mineral density in elderly men and women. The Framingham Study. *Am J Epidemiol* 142:485–492, 1995.

Fernandez, E.; La Vecchia, C.; Porta, M.; Negri, E.; d'Avanzo, B.; and Boyle, P. Pancreatitis and the risk of pancreatic cancer. *Pancreas* 11:185–189, 1995.

Finlayson, R.E.; Hurt, R.D.; Davis, L.J., Jr.; and Morse, R.M. Alcoholism in elderly persons: A study of the psychiatric and psychosocial features of 216 inpatients. *Mayo Clin Proc* 63:761–768, 1988.

Frezza, M.; di Padova, C.; Pozzato, G.; Terpin, M.; Baraona, E.; and Lieber, C.S. High blood alcohol levels in women. The role of decreased gastric alcohol dehydrogenase activity and first-pass metabolism. *N Engl J Med* 322:95–99, 1990.

Gaillard-Perera, H., and Gaillard, A. Carcinoma of the oral cavity independent of tobacco and alcoholic beverages. Apropos of 23 cases in elderly persons. *Rev Stomatol Chir Maxillofac* 93:58–59, 1992.

Ghosh, T.B., and Victor, B.S. Suicide. In: Hales, R.E.; Yudofsky, S.C.; and Talbott, J.A., eds. *The American Psychiatric Press Textbook of Psychiatry.* 2d ed. Washington, DC: American Psychiatric Press, 1994. pp. 1251–1271.

Ginsburg, E.S.; Mello, N.K,; Mendelson, J.K.; Barbieri, R.L.; Teoh, S.K.; Rothman, M.; Gao, X.; and Sholar, J.W. Effects of alcohol ingestion on estrogens in postmenopausal women. *JAMA* 276(21):1747–1751, 1996.

Gruenewald, P.J., and Ponicki, W.R. The relationship of alcohol sales to cirrhosis mortality. *J Stud Alcohol* 56:635–641, 1995.

Hansagi, H.; Romelsjo, A.; Gerhardsson de Verdier, M.; Andreasson, S.; and Leifman, A. Alcohol consumption and stroke mortality. 20-year follow-up of 15,077 men and women. *Stroke* 26:1768–1773, 1995.

Higgins, J.P.; Wright, S.W.; and Wrenn, K.D. Alcohol, the elderly, and motor and motor vehicle crashes. *Am J Emerg Med* 14(3):265–267, 1996.

Holt, D.R.; Kirk, S.J.; Regan, M.C.; Hurson, M.; Lindblad, W.J.; and Barbul, A. Effect of age on wound healing in healthy human beings. *Surgery* 112:293–297, 1992.

Huijbregts, P.P.; Feskens, E.J.; and Kromhout, D. Dietary patterns and cardiovascular risk factors in elderly men: The Zutphen Elderly Study. *Int J Epidemiol* 24:313–320, 1995.

Hurt, R.D.; Higgins, R.A.; Nelson, R.A.; Morse, R.M.; and Dickson, E.R. Nutritional status of a group of alcoholics before and after admission to an alcoholism treatment unit. *Am J Clin Nutr* 34(3):386–392, 1981.

Hurt, R.D.; Finlayson, R.E.; Morse R.M.; and Davis, L.J., Jr. Alcoholism in elderly persons: Medical aspects and prognosis of 216 inpatients. *Mayo Clin Proc* 63(8 Aug):753–760, 1988.

Hurt, R.D.; Offord, K.P.; Croghan, I.T.; Gomez-Dahl, L.; Kottke, T.E.; Morse, R.M.; and Melton, L.J., III. Mortality following inpatient addictions treatment. *JAMA* 275:1097–1103, 1996.

Iber, F.L.; Blass, J.P.; Brin, M.; and Leevy, C.M. Thiamin in the elderly—relation to alcoholism and to neurological

degenerative disease. *Am J Clin Nutr* 36(5 suppl):1067–1082, 1982.

Iso, H.; Kitamura, A.; Shimamoto, T.; Sanakai, T.; Naito, Y.; Sato, S.; Kiyama, M.; Iida, M.; and Komachi, Y. Alcohol intake and the risk of cardiovascular disease in middle-aged Japanese men. *Stroke* 26(5):767–773, 1995.

Jensen, G.D., and Bellecci, P. Alcohol and the elderly: Relationships to illness and smoking. *Alcohol Alcohol* 22:193–198, 1987.

Jones, A.W., and Neri, A. Age-related differences in blood ethanol parameters and subjective feelings of intoxication in healthy men. *Alcohol Alcohol* 20:45–52, 1985.

Klatsky, A.L. The cardiovascular effects of alcohol. *Alcohol 22*(Suppl 1):117–124, 1987.

Klatsky, A.L.; Armstrong, M.A.; and Friedman, G.D. Alcohol and mortality. *Ann Intern Med* 117(8):646–654, 1992.

Klatsky, A.L.; Armstrong, M.A.; and Friedman, G.D. Relations of alcoholic beverage use to subsequent coronary artery disease hospitalization. *Am J Cardiol* 58:710–714, 1986.

Koch, W.M.; Patel, H.; Brennan, J.; Boyle, J.O.; and Sidransky, D.: Squamous cell carcinoma of the head and neck in the elderly. *Arch Otolaryngol Head Neck Surg* 121:262–265, 1995.

Koivula, I.; Sten, M.; and Makela, P.H. Risk factors for pneumonia in the elderly. *Am J Med* 96:313–320, 1994.

Kono, K.; Endo, H.; Yamamoto, T.; and Kuzuya, F. Clinical study of chronic subdural hematoma and hygroma in two hospitals for elderly patients with dementia. *Nippon Ronen Igakkai Zasshi* 26:367–374, 1989.

Lee, T.K.; Huang, Z.S.; Ng, S.K.; Chan, K.W.; Wang, Y.S.; Liu, H.W.; and Lee, J.J. Impact of alcohol consumption and cigarette smoking on stroke among the elderly in Taiwan. *Stroke* 26:790–794, 1995.

Licastro, F.; Chiricolo, M.; Morini, M.C.; Capri, I.; Davis, L.J.; Conte, R.; Mancini, R.; Melotti, C.; Parente, R.; Serra, R.; et al. Influence of age and health on immune functions and trace elements. *Gerontology* 41:235–241, 1995.

Lieber, C.S. Metabolic effects of ethanol and its interaction with other drugs, hepatotoxic agents, vitamins, and carcinogens: A 1988 update. *Semin Liver Dis* 8:47–68, 1988.

Marrie, T.J. Epidemiology of community-acquired pneumonia in the elderly. *Semin Respir Infect* 5:260–268, 1990.

Marshall, E.J.; Edwards, G.; and Taylor, C. Mortality in men with drinking problems: A 20-year follow-up. *Addiction* 89:1293–1298, 1994.

Mishara, B.L., and Kastenbaum, R. *Alcohol and Old Age.* New York: Grune & Stratton, 1980.

Mosenthal, A.C.; Livingston, D.H.; Elcavage, J.; Merritt, S.; and Stucker, S. Falls: Epidemiology and strategies for prevention. *J Trauma* 38:753–756, 1995.

Nakamura, C.M.; Molgaard, C.A.; Stanford, E.P.; Peddecord, K.M.; Morton, D.J.; Lockevy, S.A.; Zunigan, M.; and Gardner, L.D. A discriminant analysis of severe alcohol consumption among older persons. *Alcohol Alcohol* 25:75–80, 1990.

Nelson, D.E.; Sattin, R.W.; Langlois, J.A.; DeVito, C.A.; and Stevens, J.A. Alcohol as a risk factor for fall injury events among elderly persons living in the community. *J Am Geriatr Soc* 40:658–661, 1992.

Niemela, L.; Sorvajarvi, K.; Blake, J.D.; and Israel, Y. Carbohydrate-deficient transferrin as a marker of alcohol abuse: Relationship to alcohol consumption, severity of liver disease and fibrogenesis. *Alcohol Clin Exp Res* 19(5):1203–1208, 1995.

Noda, K.; Yoshihara, H.; Suzuki, K.; Yamada, Y.; Kashara, A.; Hayashi, N.; Fusamoto, H.; and Kamada, T. Progression of type C hepatitis to liver cirrhosis and hepatocellular carcinoma—its relationship to alcohol drinking and the age of transfusion. *Alcohol Clin Exp Res* 20(1 suppl Feb):95A–100A, 1996.

Nordstrom, G., and Berglund, M. Ageing and recovery from alcoholism. *Br J Psychiatry* 151:382–388, 1987.

Ochshorn-Adelson, M.; Bodner, G.; Toraker, P.; Albeck, H.; and Kreek, M.J. Effects of ethanol on human natural killer cell activity: In vitro and acute, low-dose in vivo studies. *Alcohol Clin Exp Res* 18(6):1361–1367, 1994.

O'Connor, A., and Daly, D. Alcoholics: A twenty year follow-up study. *Br J Psychiatry* 146:645–647, 1985.

O'Keeffe, S.T.; Tormey, W.P.; Glasgow, R.; and Lavan, J.N. Thiamine deficiency in hospitalized elderly patients. *Gerontology* 40:18–24, 1994.

O'Loughlin, J.L.; Robitaille, Y.; Boivin, J.F.; and Suissa, S. Incidence of and risk factors for falls and injurious falls among the community-dwelling elderly. *Am J Epidemiol* 137:342–354, 1993.

Orleans, C.T., and Hutchinson, D. Tailoring nicotine addiction treatments for chemical dependency patients. *J Subst Abuse Treat* 10(2):197–208, 1993.

Ortqvist, A. Prognosis in community-acquired pneumonia requiring treatment in hospital. Importance of predisposing and complicating factors, and of diagnostic procedures. *Scand J Infect Dis Suppl* 65:1–62, 1990.

Palomäki, H., and Kaste, M. Regular light-to-moderate intake of alcohol and the risk of ischemic stroke. Is there a beneficial effect? *Stroke* 24(12):1828–1832, 1993.

Rosenberg, I.H.; Bowman, B.B.; Cooper, B.A.; Halsted, C.H.; and Lindenbaum, J. Folate nutrition in the elderly. *Am J Clin Nutr* 36:(5 suppl) 1060–1066, 1982.

Ruben, F.L.; Dearwater, S.R.; Norden, C.W.; Kuller, L.H.; Gartner, K.; Shalley, A.; Warshafsky, G.; Kelsey, S.F.; O'Donnell, C.; Means, E.; et al. Clinical infections in the noninstitutionalized geriatric age group: Methods utilized and incidence of infections. The Pittsburgh Good Health Study. *Am J Epidemiol* 141:145–157, 1995.

Saunders, P.A.; Copeland, J.R.M.; Dewey, M.E.; Davidson, I.A.; McWilliam, C.; Sharma, V.; and Sullivan, C. Heavy drinking as a risk factor for depression and dementia in elderly men. Findings from the Liverpool longitudinal community study. *Br J Psychiatry* 159:213–216, 1991.

Scherr, P.A.; LaCroix, A.Z.; Wallace, R.B.; Berkman, L., Curb, J.D.; Cornoni-Huntley, J.; Evans, D.A.; and Hennekens, C.H. Light to moderate alcohol consumption and mortality in the elderly. *J Am Geriatr Soc* 40:651–657, 1992.

Schuckit, M.A. A clinical review of alcohol, alcoholism, and the elderly patient. *J Clin Psychiatry* 43:396–399, 1982.

Scott, R.B. Alcohol effects in the elderly. *Compr Ther* 15:8–12, 1989.

Shibata, A.; Mack, T.M.; Paganini-Hill, A.; Ross, R.K.; and Henderson, B.E. A prospective study of pancreatic cancer in the elderly. *Int J Cancer* 58:46–49, 1994.

Slemenda, C.W.; Christian, J.C.; Reed, T.; Reister, T.K.; Williams, C.J.; and Johnston, C.C. Jr. Long-term bone loss in men: Effects of genetic and environmental factors. *Ann Intern Med* 117:286–291, 1992.

Smith, J.W. Medical manifestations of alcoholism in the elderly. *Int J Addict* 30:1749–1798, 1995.

Stibler, H.; Borg, S.; and Beckman, G. Transferrin phenotype and level of carbohydrate-deficient transferrin healthy individuals. *Alcohol Clin Exp Res* 12(3):450–453, 1988.

Takada, N.; Soma, K.; Dobashi, Y.; Kondo, E.; Yamamoto, H.; Kusuhara, N.; Kobayashi, H.; Yanase, N.; Abe, T.; and Tomita, T. A clinical study of fulminant Legionnaires' disease. *Nippon Kyobu Shikkan Gakkai Zasshi* 32: 138–145, 1994.

Tanaka, R.; Itoshima, T.; and Nagashima, H. Follow-up study of 582 liver cirrhosis patients for 26 years in Japan. *Liver* 7:316–324, 1987.

Tavani, A.; Pregnolato, A.; Negri, E.; and La Vecchia, C. Alcohol consumption and risk of pancreatic cancer. *Nutr Cancer* 27(2):157–161, 1997.

Thomas, D.R. Accidental hypothermia in the sunbelt. *J Gen Intern Med* 3:552–554, 1988.

Tucker, L.E.; Stanford, J.; Graves, B.; Swetnam, J.; Hamburger, S.; and Anwar, A. Classical heatstroke: Clinical and laboratory assessment. *South Med J* 78:20–25, 1985.

Van de Kerkhof, P.C.; Van Bergen, B.; Spruijt, K.; and Kuiper, J.P. Age-related changes in wound healing. *Clin Exp Dermatol* 19:369–374, 1994.

Velema, J.P.; Walker, A.M.; and Gold, E.B. Alcohol and pancreatic cancer. Insufficient epidemiologic evidence for a causal relationship. *Epidemiol Rev* 8:28–41, 1986.

Vestal, R.E.; McGuire, E.A.; Tobin, J.D.; Andres, R.; Norris, A.H.; and Mezey, E. Aging and ethanol metabolism. *Clin Pharmacol Ther* 21: 343–354, 1977.

Vogel-Sprott, M., and Barrett, P. Age, drinking habits and the effects of alcohol. *J Stud Alcohol* 45:517–521, 1984.

Welte, J.W., and Mirand, A.L. Lifetime drinking patterns of elders from a general population survey. *Drug Alcohol Depend* 35:133–140, 1994.

Wray, A., and McGuirt, W.F. Smokeless tobacco usage associated with oral carcinoma. Incidence, treatment, outcome. *Arch Otolaryngol Head Neck Surg* 119:929–933, 1993.

Chapter 12

Alcohol, Aging, and Cognition

Sara Jo Nixon, Ph.D.

Aging is accompanied by a variety of physiological changes that may affect both the pharmacokinetics and the pharmacodynamics of various substances, including alcohol (Tupler et al. 1995). In recent years, we have become increasingly aware of older adult alcohol abusers, and a number of studies have examined the prevalence, etiology, and outcome of alcohol use disorders in these persons (Brennan et al. 1993; Geroldi et al. 1994; Krause 1995).

Most older adults, fortunately, do not become alcoholic or even increase their drinking over time (National Institute on Alcohol Abuse and Alcoholism 1994). In fact, studies of drinking patterns have suggested that drinking styles are remarkably stable through middle adulthood, with some reported decreases, perhaps associated with illness (Goodwin et al. 1987; Adams et al. 1990; Gurnack and Hoffman 1992).

These findings do not, however, indicate that alcohol use in aging adults is not an important issue for study. The physiological and metabolic changes associated with chronic health disorders often observed in aging persons, in combination with the frequent use of prescription drugs (Adams 1995) as well as the psychosocial adaptations required with increasing age, may place subgroups of even nonalcoholic, moderate-to-heavy-drinking older adults at increased risk for alcohol-related dysfunction. The list of potential problems resulting from alcohol intake includes an exacerbation of physical symptomatology, untoward drug interactions, interpersonal conflict, and a decline in the ability to perform normal functions. Table 1, taken from Scott and Mitchell (1988), illustrates the substantial interaction

S.J. Nixon, Ph.D., is an associate professor in the Department of Psychiatry and Behavioral Sciences, University of Oklahoma Health Sciences Center; associate director of the Oklahoma Center for Alcohol and Drug Related Studies; and director of the Cognitive Studies Laboratory, Suite 410 Rogers Bldg., 800 NE 15th St., Oklahoma City, OK 73104.

between alcohol and typical medications with hepatic metabolism used by older adults.

Although all of the potential negative outcomes are important, the primary focus of this chapter is neurocognitive

Table 1. Aging and Alcohol Effects on Drugs With Hepatic Metabolism.

Drug	Aging Effects
Acetaminophen	Normal or decreased Cl.
Aspirin	Normal or decreased Cl.
Oral anticoagulants	Pharmacokinetics normal. Increased effect on aging liver.
Heparin	Increased bleeding, especially in elderly women.
Tolbutamide	Decreased hypoglycemic response.
Antidepressants	Increased blood levels (imipramine). Generally increased CNS effect.
Benzodiazepines	Diazepam and chlordiazepoxide: Normal to decreased Cl; half-life increased. Increased plasma levels. Generally, increased sedation and psychomotor impairment. Lorazepam and oxazepam: Cl affected little or none.
Chloral hydrate	Increased sedation.
Barbiturates	Increased CNS effects. Half-life increased.
Antihistamines	Increased sedation.
Phenothiazines	Increased blood levels.
Tagamet	Decreased (renal) Cl. Mental confusion more common.
Inderal	Normal to decreased Cl. Decreased heart rate response.
Dilantin	Increased Cl. Hydroxylation rate decreased.
Opiates	Decreased plasma binding increases blood level.

Note: Cl = plasma clearance; CNS = central nervous system.
Source: Scott , R.B., and Mitchell, M.C. Aging, alcohol, and the liver. *J Am Geriatr Soc* 36:255–265, 1988.

function in social drinking older adults. Before moving to this literature, I will discuss some general issues of aging and cognition and some recent findings regarding the effects of social drinking on neurocognitive function.

Alcohol Effects
Enhanced toxicity in heavy drinkers. Overall Cl unchanged.
Prolongs bleeding time. Aggravates chronic gastrointestinal blood loss.
Potentiation of effect acutely. Chronic alcohol increases Cl.
Acute alcohol increases half-life. Alcohol may exaggerate hypoglycemia.
Potentiation of effect acutely. Increased risk of hypothermia.
Diazepam: acute alcohol decreases Cl. Lorazepam: pharmacokinetics normal. General increased sedation.
Acute alcohol decreases Cl. Enhanced CNS effects.
Potentiation. Acute alcohol increases half-life. Chronic alcohol increases Cl.
Potentiation.
Increased extrapyramidal effects. Decreases alcohol metabolism.
Slight increase in peak blood alcohol level.
Acutely decreases Cl.
Acute alcohol: decreased Cl. Chronic alcohol: increased Cl.
Acute alcohol: decreased Cl. Chronic alcohol: increased Cl.

AGING AND COGNITION

There is a large literature on the effects of aging on cognitive function (e.g., Craik and Salthouse 1992). This body of work now extends across decades of research and provides considerable insight regarding the myths and realities of cognitive change over time.

Moscovitch and Winocur (1992) used both contemporary cognitive theory and neuropsychology to address the effects of aging on cognitive function. After a careful review of the literature, these authors concluded that aging-related cognitive deficits are limited largely to those processes associated with either hippocampal or frontal lobe functions. Accordingly, they concluded that damage to the hippocampus would result in memory problems with four defining characteristics: (a) memory processes are affected, independent of general intelligence; (b) short-term memory is less affected than long-term memory; (c) recent memories are more affected than are remote memories; and (d) explicit memory (or memory with awareness) deficits are more likely to be observed than are implicit memory (or memory without awareness) deficits. This last point should be qualified, however, by noting that this conclusion applies only to those tasks that are primarily perceptual repetition-priming tests that do not involve either the hippocampus or the frontal lobes. In fact, it is hypothesized that when age-related deficits in implicit memory tasks are observed, they are associated with frontal lobe dysfunction (Wright and Payne 1985; Cohen et al. 1990; Dywan and Jacoby 1990).

In addition to playing a role in implicit memory tasks requiring organization or search, the frontal lobes are implicated in problem solving, set shifting, and working with memory (as opposed to working memory) (Goldman-Rakic 1987; Moscovitch and Winocur 1992; Kolb and Whishaw 1996). In short, they are implicated in tasks that require that existing information or memories be manipulated (e.g., inferences drawn from memory, temporal ordering of information, placement of memory in context, and implementing acquisition [or encoding] and retrieval strategies). These types of deficits are reported repeatedly in the aging literature (e.g., Kausler and Puckett 1979; Salthouse 1982; Craik et al. 1990; Light 1991).

While recognizing that cognitive deficits are often associated with aging, it should be noted that there is also considerable heterogeneity in aging studies. There are a number of variables that may mitigate the effects of aging on cognitive functions, including an active lifestyle (Hultsch et al. 1993); specific personality characteristics (Poon et al. 1992); and higher intelligence and affect (Poon et al. 1992). In addition, the observed severity of cognitive deficits may be reduced with the use of ecologically valid test instruments (i.e., those that are based on everyday experiences) (Poon et al. 1992) and the absence or availability of retrieval strategy cues (Levine et al. 1995). Other work has suggested that the primary predictor of poor cognitive performance is not age, per se, but rather dependence as opposed to independence (e.g., Moscovitch

and Winocur 1992; Aronson and Vroonland 1993).

In summary, cognitive decline with advancing age is observed frequently. The severity of these impairments is not uniform (see previous references; Salthouse 1994). Severity is affected by both experimental factors, such as the ecological validity of the test, and a number of individual traits (e.g., intelligence, personality, lifestyle) as well as other factors, such as the degree of dependence of the older adult.

SOCIAL DRINKING

One might ask, why bother to discuss the area of social drinking? It has been studied repeatedly, at least with young adults, and the results are far from consistent or conclusive. However, it is because of these inconsistencies and the usually restricted sample (i.e., young healthy males) that this literature must be revisited for a critical examination of the individual studies. Furthermore, if social drinking in aging groups is to be studied, it is important that the larger literature be fully appreciated.

A review of the social drinking literature was conducted by Parsons in 1986. He concluded that the evidence was weak in regard to social drinking's negative effect on neurocognitive function. However, he urged that continued research be conducted in order to eliminate the inconsistencies and to ascertain which of several hypotheses might best account for any observed deficit.

Significant inconsistencies were again found in a recent review of the social drinking research conducted between 1986 and 1996 (Parsons and Nixon 1998). Table 2 includes a list of these studies as well as information regarding the typical drinking patterns reported in 17 of the 18 studies. A review of this information reveals that significant effects of social drinking (i.e., positive results) were observed only in those studies with exceptionally high "social" drinking patterns. Interestingly, significant negative consequences of social drinking were observed in the two studies reporting event-related potentials (ERPs) (Nichols et al. 1993; Fox et al. 1995 [Fox and colleagues addressed both memory and ERP functions]).

We also wanted to examine the possibility that age might play a role in the observed findings. Therefore, we divided the studies into two groups: a young group ($M < 32$ years of age) and an older group ($M > 32$, roughly equivalent to our male alcoholic samples). Perhaps not surprisingly, given the young age of the samples, age was not an important factor.

SOCIAL DRINKING AND COGNITION IN AGING PERSONS

Few systematic studies of social drinking, aging, and cognition have been completed. One of the more comprehensive studies was conducted by Goodwin and colleagues (Goodwin et al. 1987). In this study, they examined the relation between alcohol consumption and social, psychological/emotional, and cognitive status in 270 men and women ranging in age

Table 2. Comparison of Positive and Negative Results of Studies on Number of Standard Drinks per Occasion, Weekly Frequency, and Weekly Total for Heavy Drinkers.

Studies With Negative Results[a]	Drinks/ Occasion	Weekly Frequency	Weekly Total
Alterman and Hall 1989	6.5	2.5	16
Arbuckle et al. 1994	4	7	28
Bates and Tracy 1990	5	2	10
Bowden et al. 1988	4	4.5	18
Carey and Maisto 1987	3	1	3
Emmerson et al. 1988	4	3	12
Hebert et al. 1993	2	7	14
Launer et al. 1996	3	7	21
Page and Cleveland 1987	2[b]	7	14
Salamé 1991	4	7	28
Sum	37.5	48	164
Mean	3.75	4.8	16.4
Studies With Positive Results			
Christian et al. 1995	3[c]	7	21
Fox et al. 1995	5	5	25
Martin et al.1991	5	6[d]	30
Nichols et al. 1993	3	7	21
Parker et al. 1991	5	7	35
Waugh et al. 1989	8	7	56
Williams and Skinner 1990	15	7	105
Sum	44	46	293
Mean	6.28	6.57	41.9[e]

Note: A standard drink refers to approximately one 12 oz beer, one mixed drink (1.5 oz alcohol), or one 4 oz glass of wine.

[a] The Windle and Blane (1989) study is not included because data in number of drinks and frequency of drinking were not given. Negative results refer to nonsignificant effects of drinking.

[b] Number of drinks based on < 3 per day given in article.

[c] Estimate based on article's definition of > 2 drinks per day.

[d] Exact frequency not given in paper; estimate based on subjects having met research criteria for alcohol abuse or dependence.

[e] Weekly number of drinks are significantly higher for positive than negative results ($U = 7$, $p = 0.01$). Omitting the extreme value of 105 in the positive results group, the mean number of weekly drinks is 30.5 (almost double the mean for the negative results group), and the U remains significant at $p = 0.01$ level.

from 65 to 89, with a mean age of 74. All of these participants lived independently and were part of a larger longitudinal study. Males comprised 46 percent of the sample. To determine group assignment for this study, subjects were asked to keep a diary of alcohol intake over 3 days in the middle of a week. This period was selected to avoid weekend activities which might not be representative of typical drinking behaviors. Those subjects who reported drinking during any of these 3 days were assigned to the drinking group (130/270, 48 percent); the remainder were assigned to the nondrinker group. Most of the drinkers (56 percent) consumed less

than 15 g of alcohol per day (a little over 1 drink); 26 percent consumed 15–30 g/d (about 1 to 2.5 drinks); and 17 percent drank more than 30 g/d.

In this study, emotional status was measured by a 92-item self-rating checklist that gives scores for depression, anxiety, hostility, and somatic complaints as well as a composite distress score adapted from Kellner and Sheffield (1973). Cognitive abilities were measured with several instruments: a 30-item mental status questionnaire administered verbally by the interviewer (Jacobs et al. 1977); the Halstead Category Test, a nonverbal test of abstract thinking (Reitan and Davidson 1974); and the Wechsler Memory Scale (WMS) (Russell 1975; Haaland et al. 1983). Social competency/interaction was assessed with a revised form of the Interview Schedule for Social Interaction (Henderson 1980; Thomas et al. 1985).

Consistent with other work, the results indicated that drinking levels were negatively related to age. They were positively related to sex, with males being more likely to drink, although men were not necessarily more likely to drink larger quantities. Higher income and higher educational levels were also associated with increased probability of drinking. Again, the amount of alcohol consumed was not necessarily related to either of these variables.

There was no relation between emotional status and drinking, either when all drinkers were compared with nondrinkers or when heavy drinkers (> 30 g/d) were compared with nondrinkers. Similarly, drinking status failed to indicate differences in social interaction (i.e., adequacy and amount of social support).

Contrary to some expectations, drinkers performed better than nondrinkers on the Halstead Category Test, the mental status test, and both the immediate and delayed visual memory subscales of the WMS. However, when the relation between amount consumed and performance was examined, incorporating appropriate controls for age, sex, education, and income, significant associations (i.e., correlations) were eliminated. Thus, this study reveals no detrimental effects of light to heavy social drinking in this sample of healthy, independently living adults. It does not speak to the effect of alcohol consumption on other neurobiological systems or with other types of samples (e.g., medically compromised).

We recently conducted two studies of social drinking in older adults. The first study was a preliminary assessment of 32 men who were divided into two drinking groups based on the median split for the sample (Nees 1994). This study failed to distinguish heavy from light drinkers on the basis of cognitive performance. Heavy drinkers did, however, report more lifetime medical problems. They did not report greater medical symptomatology over the past 12 months.

Most interesting was the fact that despite being unable to detect significant differences in cognitive function, the groups did demonstrate a significant difference in their reported methods of coping. Heavy drinkers were less likely to use information seeking to solve problems and reported greater

social isolation when approaching stressful events. This pattern of isolation was also observed in their family and home environment assessment.

There were several limitations to that study: only males were studied, the sample was relatively young (M = about 60 years), and only a cross-sectional analysis could be conducted. Thus, undaunted by the previous null findings, we addressed some of these concerns in a second study which applied a longitudinal design to the study of both male and female nonalcoholic persons between the ages of 55 and 65 (N = 67), again divided into two drinking groups—light and heavy. The primary objective of the study was to conduct a cross-sectional, longitudinal assessment of the effects of continued social drinking in older adults who had not yet developed significant confounding disease states and who were not likely to have significantly altered their drinking patterns over the course of the previous 2 years. The sample is described in table 3.

Participants completed a battery of tests that included the Michigan Alcoholism Screening Test—Geriatric Version (MAST-G) (Blow 1991), the Geriatric Depression Scale (Yesavage et al. 1983), trait and state anxiety scales (Spielberger 1983), and a modified life events inventory (Paykel et al. 1971). The descriptive statistics for these psychological measures (except the life events, discussed later in this section) are shown in table 4.

Cognitive tests were selected because of their sensitivity to alcohol effects in other studies. They included a difficult verbal fluency test (requiring the

Table 3. Demographic Information on Social Drinking Sample.

Variable	Mean	SD	Range
Age	60.25	(3.73)	53–67
Years of education	14.36	(2.32)	12–20
QFI	0.72	(0.95)	0–5.14

Note: The racial/ethnic composition of the sample was as follows: 91% (n = 61) Caucasian, 3% (n = 2) African American, 4.5% (n = 3) American Indian, and 1.5% (n = 1) Hispanic. QFI = Quantity Frequency Index, accounting for both amount and frequency of alcoholic drinking.

Table 4. Psychological Measures.

Variable	Mean	SD	Range
GDS	5.94	5.68	0–24
State AI	49.39	11.15	36–78
Trait AI	49.81	11.58	34–81
MAST-G	4.51	3.87	0–16

Note: GDS = Geriatric Depression Scale (Yesavage et al. 1983); State AI = State Anxiety Scale (Spielberger 1983); Trait AI = Trait Anxiety Scale (Spielberger 1983); MAST-G = Michigan Alcohol Screening Test—Geriatric Version (Blow et al. 1992).

Table 5. Affective and Drinking Measures: Time 1 (T1) vs. Time 2 (T2).

		GDS	State AI	MAST-G	QFI
Light	T1	5.00	51.20	3.60	0.14
	T2	7.78	50.00	3.33	0.08
Heavy	T1	5.10	48.00	6.20	1.91
	T2	4.54	46.64	6.36	2.09

Note: GDS = Geriatric Depression Scale (Yesavage et al. 1983); MAST-G = Michigan Alcohol Screening Test—Geriatric Version (Blow et al. 1992); State AI = State Anxiety Scale (Spielberger 1983); QFI = Quantity Frequency Index.

generation of items to six different categories), two WMS stories (with a required learning criterion of 85 percent correct), the California Card Sorting Test (Delis et al. 1989), and a test of face-name learning (Schaeffer and Parsons 1987).

Participants were contacted monthly and retested after 1 year. Our initial recruitment effort was very difficult and delayed the conduct of the followup visits; therefore, the entire sample could not be retested in the designated timeframe.

The results indicated no significant effects of light versus heavy drinking on any of the demographic variables or psychological measures except for the Quantity Frequency Index (QFI) and the MAST-G, with heavy drinkers indicating significantly higher scores on both ($p = 0.0001$).

The groups did differ in the number of life events that they endorsed having occurred in the previous year. However, neither group endorsed very many, with the light-drinking group endorsing significantly more ($M = 4.41$ vs. 4.00, $p = 0.02$). Thus, these data, consistent with other work (e.g., La Greca et al. 1988; Ekerdt et al. 1989), do not support the hypothesis that older drinkers increase their drinking in response to life stressors. We also considered affective state measures and QFIs at both time 1 and time 2. As illustrated in table 5, these measures remained remarkably stable over the 12-month followup period.

Data from three of the cognitive tasks at time 1 and time 2 are illustrated in figures 1–3. In these figures, a standard group x trials repeated measures design was applied. The verbal fluency figure refers to the total number of correct items generated across the six categories. The dependent variable in the WMS figures is not the one typically used (i.e., number of items correctly recalled); instead, it is the ratio of the total number correct divided by the number of trials required to achieve this level of learning. The group (light vs. heavy drinkers) effect failed to achieve statistical significance. As expected, subjects generally improved their performance over time. An exception is noted in regard to the subjects' performance on the WMS story focusing on the injury of children. Respondents typically found this story emotionally disturbing, and their distress may have affected the long-term storage or retrieval of this story.

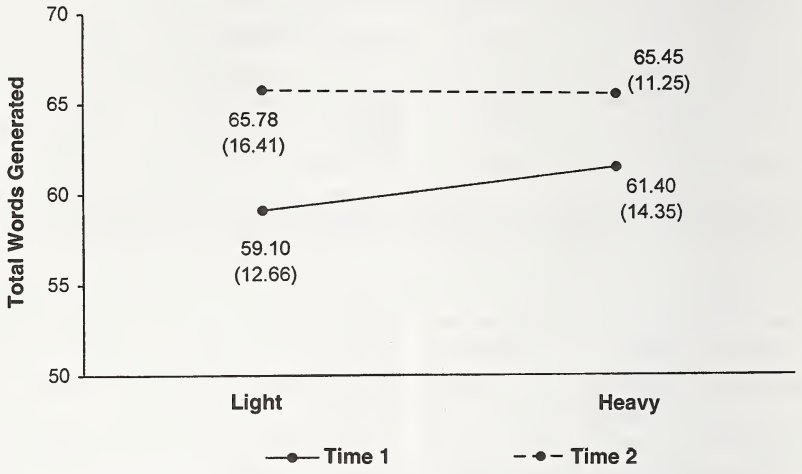

Figure 1. Verbal fluency: Aging project. Time main effects: $F = 4.23$, $p = 0.055$.

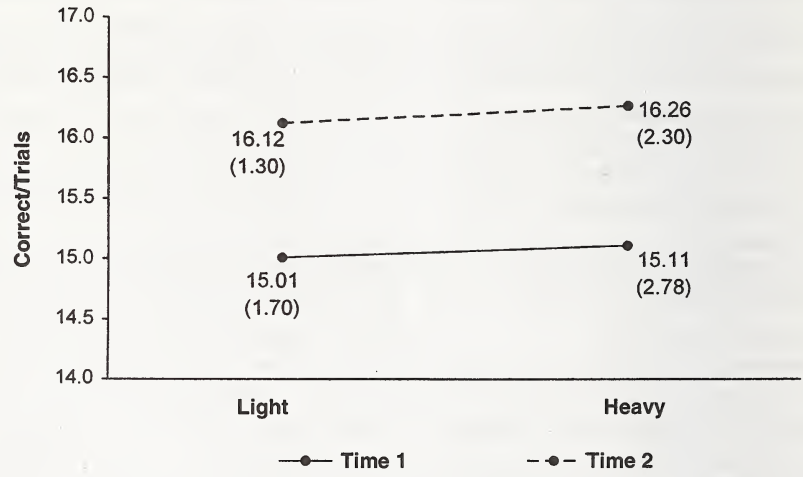

Figure 2. Correct responses/trials to criterion: Wechster Memory Scales stories, dog story. Time main effects: $F = 4.66$, $p = 0.046$.

The data analysis phase of this project is still underway, but the initial data do not support the hypothesis that social drinking in young-old adults is necessarily associated with cognitive decline. There are, however, several

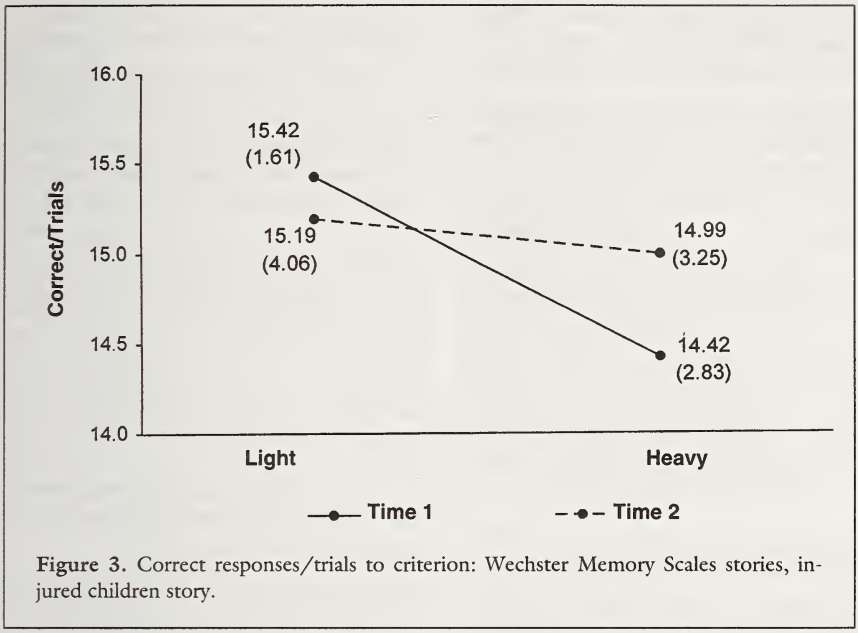

Figure 3. Correct responses/trials to criterion: Wechster Memory Scales stories, injured children story.

caveats. First, the sample size was very small; only 10 subjects were retested in some cells. (It is important to note that analyses to assess the comparability of those who either were or were not retested have not revealed meaningful differences between the two.) Second, the sample was comprised of extremely healthy adults. Individuals were not included if they had medical or psychiatric histories that might interfere with cognitive performance. This strict exclusion criterion was applied because the focus of the study was on the effect of alcohol on brain function, independent of comorbid disorders. However, in applying this criterion, the sample became less representative of the reference population. Third, the followup period was relatively brief. One year may not be sufficient to detect subtle changes in brain function, particularly in a relatively robust sample or population. Fourth, the sample was comprised largely of relatively low to moderate drinkers. A few subjects did report MAST-G scores that might draw concern, but none were classified as alcoholic using a cutoff of 13. If a wider range of drinking had been sampled, differences might have been observed. More longitudinal research is needed before we can conclude that moderate alcohol consumption does not negatively affect neurocognitive function in older adults.

IMPLICATIONS FOR RESEARCH

Given findings that indicate that drinking patterns remain quite stable across most of adulthood, it is important that systematic work be conducted regarding

the effects of continued social drinking in older adults. Existing studies do not reveal consistent negative effects. However, most of these studies have significant limitations that restrict their application. For example, many of the studies are limited to cross-sectional designs, to a restricted subject selection (e.g., only males or only young-old adults), and/or to incomplete assessment batteries. If we are to understand more completely the effects of continued social drinking on aging processes, we must conduct longitudinal studies with representative older male and female drinkers who may have chronic disorders.

ACKNOWLEDGMENTS

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REFERENCES

Adams, W.L. Potential for adverse drug-alcohol interactions among retirement community residents. *J Am Geriatr Soc* 43:1021–1025, 1995.

Adams, W.L.; Garry, P.J.; Rhyne, R.; Hunt, W.C.; and Goodwin, J.S. Alcohol intake in the healthy elderly: Changes with age in a cross-sectional and longitudinal study. *J Am Geriatr Soc* 38:211–216, 1990.

Alterman, A.I., and Hall, S.G. Effects of social drinking and familial alcoholism risk on cognitive functioning: Null findings. *Alcohol Clin Exp Res* 13:799–803, 1989.

Arbuckle, T.Y.; Chaikelson, J.S.; and Gold, D.P. Social drinking and cognitive functioning revisited: The role of intellectual endowment and psychological distress. *J Stud Alcohol* 55:352–361, 1994.

Aronson, H., and Vroonland, J.P. The residual cognitive competence of elderly females. *J Clin Psychol* 49:724–731, 1993.

Bates, M.E., and Tracy, J.I. Cognitive impairment in young "social drinkers": Is there impairment to detect? *J Abnorm Psychol* 99:242–249, 1990.

Blow, F.C. *Michigan Alcoholism Screening Test—Geriatric Version.* Ann Arbor, MI: University of Michigan, 1991.

Bowden, S.C.; Walton, N.H.; and Walsh, K.W. The hangover hypothesis and the influence of moderate social drinking on mental ability. *Alcohol Clin Exp Res* 12:25–29, 1988.

Brennan, P.L.; Moos, R.H.; and Kim, J.Y. Gender differences in the individual characteristics and life contexts of late-middle-aged and older problem drinkers. *Addiction* 88:781–790, 1993.

Cahalan, D.; Cisin, I.; and Crossley, H.M. *American Drinking Practices: A National Study of Drinking Behaviors and Attitudes.* Monograph 6. New Brunswick, NJ: Rutgers Center of Alcohol Studies, 1969.

Carey, K.B., and Maisto, S.A. Effect of change in drinking pattern on the cognitive function of female social drinkers. *J Stud Alcohol* 48:236–241, 1987.

Christian, J.C.; Reed, T.; Carmelli, D.; Page, W.F.; Norton, J.A.; and Breitner, J.C.S. Self-reported alcohol intake and cognition in aging twins. *J Stud Alcohol* 56:414–416, 1995.

Cohen, A.; Ivry, R.I.; and Keele, S.W. Attentional structure in sequence learning. *J Exp Psychol Learn Mem Cogn* 16:17–30, 1990.

Craik, F.I.M., and Salthouse, T.A., eds. *The Handbook of Aging and Cognition.* Hillsdale, NJ: Lawrence Erlbaum Associates, 1992.

Craik, F.I.M.; Morris, L.W.; Morris, R.G.; and Loewen, E.R. Relations between source amnesia and frontal lobe functioning in older adults. *Psychol Aging* 5:148–151, 1990.

Delis, D.C.; Bihrle, A.M.; Janowsky, J.S.; Squire, L.R.; and Shimamura, A.P. Fractionation of problem-solving deficits in frontal lobe patients. *J Clin Exp Neuropsychol* 11:50, 1989.

Dywan, J., and Jacoby, L.L. Effects of aging on source monitoring: Differences in susceptibility to false fame. *Psychol Aging* 5:379–387, 1990.

Ekerdt, D.J.; DeLabry, L.O.; Glynn, R.J.; and Davis, R.W. Change in drinking behaviors with retirement: Findings from the Normative Aging Study. *J Stud Alcohol* 50:347–353, 1989.

Emmerson, R.Y.; Dustman, R.E.; Heil, J.; and Shearer, D.F. Neuropsychological performance of young non-drinkers, social drinkers and long- and short-term alcoholics. *Alcohol Clin Exp Res* 12:625–629, 1988.

Fox, A.M.; Michie, P.T.; Coltheart, M.; and Solowij, N. Memory functioning in social drinkers: A study of event-related potentials. *Alcohol Alcohol* 30:303–310, 1995.

Geroldi, C.; Rozzini, R.; Frisoni, G.B.; and Trabucchi, M. Assessment of alcohol consumption and alcoholism in the elderly. *Alcohol* 11:513–516, 1994.

Goldman-Rakic, P.S. Circuitry of primate prefrontal cortex and regulation of behavior by representational memory. In: Plum, F., ed. *Handbook of Physiology. Vol. 5: The Nervous System.* Bethesda, MD: American Physiological Society, 1987.

Goodwin, J.S.; Sanchez, C.J.; Thomas, P.; Hunt, C.; Garry, P.J.; and Goodwin, J.M. Alcohol intake in a health elderly population. *Am J Public Health* 77:173–177, 1987.

Gurnack, A.M., and Hoffman, N.G. Elderly alcohol misuse. *Int J Addict* 27:869–878, 1992.

Haaland, K.Y.; Linn, R.T.; Hunt, W.C.; and Goodwin, J.S. A normative study of Russell's variant of the Wechsler Memory Scale in a healthy elderly population. *J Consult Clin Psychol* 51:878–881, 1983.

Hebert, L.E.; Scherr, P.A.; Beckett, L.A.; Albert, M.S.; Rosner, B.; Taylor, J.O.; and Evans, D.A. Relation of smoking and low-to-moderate alcohol consumption to change in cognitive function: A longitudinal study in a defined community of older persons. *Am J Epidemiol* 137:881–891, 1993.

Henderson, S. A development in social psychiatry: The systematic study of social bonds. *J Nerv Ment Dis* 168:63–69, 1980.

Hultsch, D.F.; Hammer, M.; and Small, B.J. Age differences in cognitive performance in later life: Relationships to self-reported health and activity life style. *J Gerontol* 48:P1–P11, 1993.

Jacobs, J.W.; Bernhard, M.R.; and Delgado, A. Screening for organic mental syndromes in the medically ill. *Ann Intern Med* 86:40–46, 1977.

Kausler, D.H., and Puckett, J.M. Effects of word frequency on adult age differences in word memory span. *Exp Aging Res* 5:161–169, 1979.

Kellner, R., and Sheffield, R.T. A self-rating scale of distress. *Psychol Med* 3:88–100, 1973.

Kolb, B., and Whishaw, I.Q. *Fundamentals of Human Neuropsychology.* New York: W.H. Freeman, 1996.

Krause, N. Stress, alcohol use and depressive symptoms in later life. *Gerontologist* 35:296–307, 1995.

La Greca, A.J.; Akers, R.L.; and Dwyer, J.W. Life events and alcohol behavior among older adults. *Gerontologist* 28:552–558, 1988.

Launer, L.J.; Feskens, E.J.; Kalmyn, S.; and Kronhout, D. Smoking, drinking and thinking: The Zutphen Elderly Study. *Am J Epidemiol* 143:219–227, 1996.

Levine, B.; Stuss, D.T.; and Milberg, W.P. Concept generation: Validation of a test of executive functioning in a normal aging population. *J Clin Exp Neuropsychol* 17:740–758, 1995.

Light, L.L. Memory and aging. *Annu Rev Psychol* 42:333–376, 1991.

Martin, E.; Sher, K.J.; and Wood, P.K. Substance use and abuse and cognitive functioning in college students. Paper presented at the annual meeting of the Association for the Advancement of Behavior Therapy, New York, NY, 1991.

Moscovitch, M., and Winocur, G. The neuropsychology of memory and aging. In: Craik, F.I.M., and Salthouse, T.A., eds. *The Handbook of Aging and Cognition.* Hillsdale, NJ: Lawrence Erlbaum Associates, 1992. pp. 315–372.

National Institute on Alcohol Abuse and Alcoholism. *Eighth Special Report to the U.S. Congress on Alcohol and Health.* NIH Pub. No. 94–3699. Bethesda, MD: National Institutes of Health, 1994.

Nees, R.M. Cognitive performance in elderly, heavy social drinkers. Unpublished M.S. thesis, University of Oklahoma Health Sciences Center, 1994.

Nichols, J.M.; Martin, F.; and Kirkby, K.C. A comparison of the effect of lorazepam on memory in heavy and low social drinkers. *Psychopharmacology (Berl)* 112:475–482, 1993.

Page, R.D., and Cleveland, M.F. Cognitive dysfunction and aging among male alcoholics and social drinkers. *Alcohol Clin Exp Res* 11:376–384, 1987.

Parker, E.S.; Parker, D.A.; and Harford, T.C. Specifying the relationship between alcohol use and cognitive loss: The effects of frequency of consumption and psychological distress. *J Stud Alcohol* 52:366–373, 1991.

Parsons, O.A. Cognitive functioning in sober social drinkers: A review and critique. *J Stud Alcohol* 47:101–114, 1986.

Parsons, O.A., and Nixon, S.J. Cognitive functioning in sober social drinkers: A review of the research since 1986. *J Stud Alcohol* 59:180–190, 1998.

Paykel, E.S.; Prusoff, B.A.; and Uhlenhuth, E.H. Scaling of life events. *Arch Gen Psychiatry* 25:340–347, 1971.

Poon, L.W.; Martin, P.; Clayton, G.M.; Messner, S.; Noble, C.A.; and Johnson, M.A. The influences of cognitive resources on adaptation and old age. *Int J Aging Hum Dev* 34:31–46, 1992.

Reitan, R., and Davidson, L., eds. *Clinical Neuropsychology: Current Status and Applications.* New York: John Wiley & Sons, 1974.

Russell, E.W. A multiple scoring method of assessment of complex memory function. *J Consult Clin Psychol* 43:800–809, 1975.

Salamé, R. The effects of alcohol on learning as a function of drinking habits. *Ergonomics* 34:1231–1241, 1991.

Salthouse, T.A. *Adult Cognition.* New York: Springer-Verlag, 1982.

Salthouse, T.A. Age-related differences in basic cognitive processes: Implications for work. *Exp Aging Res* 20:249–255, 1994.

Schaeffer, K.W., and Parsons, O.A. Learning impairment in alcoholics using an ecologically relevant test. *J Nerv Ment Dis* 175:213–218, 1987.

Scott, R.B., and Mitchell, M.C. Aging, alcohol, and the liver. *J Am Geriatr Soc* 36:255–265, 1988.

Spielberger, C. *Manual for the State-Trait Anxiety Inventory—Revised.* Palo Alto, CA: Consulting Psychologists Press, 1983.

Thomas, P.D.; Garry, P.J.; Goodwin, J.M.; and Goodwin, J.S. Social bonds in a healthy elderly sample: Characteristics and associated variables. *Soc Sci Med* 20:365–369, 1985.

Tupler, L.A.; Hege, S.; and Ellinwood, E.H., Jr. Alcohol pharmacodynamics in young-elderly adults contrasted with young and middle-aged subjects. *Psychopharmacology (Berl)* 118:460–470, 1995.

Waugh, M.; Jackson, M.; Fox, G.A.; Hawke, S.H.; and Tuck, R.R. Effect of social drinking on neuropsychological performance. *Br J Addict* 84:659–667, 1989.

Williams, C.M., and Skinner, A.E.G. The cognitive effects of alcohol abuse: A controlled study. *Br J Addict* 85:911–917, 1990.

Windle, M., and Blane, H.T. Cognitive ability and drinking behavior in a national sample of young adults. *Alcohol Clin Exp Res* 13:43–48, 1989.

Wright, B.M., and Payne, R.B. Effects of aging on sex differences in psychomotor reminiscence and tracking proficiency. *J Gerontol* 40:179–184, 1985.

Yesavage, J.A.; Brink, T.L.; Rose, T.L.; Lum, O.; Huang, V.; Adey, M.; and Leirer, V.O. Development and validation of a Geriatric Depression Screening Scale: A preliminary report. *J Psychiatr Res* 17:37–49, 1983.

Chapter 13

Stress and Elderly Drinking

John W. Welte, Ph.D.

The proportion of the population age 60 or older is growing, and will continue to grow well into the 21st century. Older persons experience some unique problems associated with drinking: age can increase vulnerability to alcohol's effects, diagnosis of alcohol abuse can be confused because the symptoms are mistaken for conditions related to aging, and alcohol can interact negatively with medications used frequently by the elderly (Ganikos et al. 1988). Research on the etiology of elder alcohol abuse is therefore an increasing national concern.

THE STRESS HYPOTHESIS AND ELDER ALCOHOL ABUSE

Although elder Americans drink less than younger ones (Bucholz et al. 1995; Wilsnack et al. 1995), problems associated with elder alcohol abuse are nonetheless significant, and their causes are not well understood. Continuation of problem drinking established early in life and late-onset problem drinking are both known to occur. Research findings (Bahr 1969; Atkinson et al. 1985; Hurt et al. 1988) suggest that late-onset alcoholics tend to be more socialized and less deviant than the early-onset, antisocial, genetically influenced type described by Zucker (1987). The speculation in the scientific literature about the causes of late-onset heavy drinking often centers on the concept of stress (Glass et al. 1995).

The measurement of stressful conditions as a contributing factor to physical or mental illnesses has played a prominent role in the history of social medicine. Stressors may take the form of discrete life events, such as death of a spouse (Dohrenwend and Dohrenwend 1974), or chronic life

J.W. Welte, Ph.D., is a senior research scientist at the Research Institute on Addictions, 1021 Main St., Buffalo, NY 14203.

strains and daily "hassles," such as concern about the safety of your neighborhood (Folkman and Lazarus 1980). Older persons face many potentially stressful life events, both discrete and chronic; these events may include illness or death of family members and friends, the decline of their own physical and mental abilities, and retirement, which may be seen as the loss of opportunity for work and recognition. It is important to measure the perceived impact of a life event on the respondent (Vinokur and Selzer 1975), as well as the resources that an individual possesses that may buffer the effect of stress. These resources may include social supports (House and Kahn 1985) and cognitive and behavioral methods of coping with stress (Moos et al. 1983).

There is a long history of research on coping with stress as a motive for drinking, and the notion of drinking to relieve stress has powerful intuitive appeal. As Peyser (1982) commented, "Everyday observation and popular opinion agree that alcohol relieves tension and enables one to cope with stress." However, the use of the amelioration of stress as an explanation of heavy drinking has a checkered history. In an influential review of the literature, Pohorecky (1981) concluded that the evidence was clouded, and that "more research needs to be done to assess the validity of the anxiety-reducing theory of alcohol abuse." In a second review (Pohorecky 1991, p. 438), she stated that "although still insufficiently documented, stress does not appear to play a significant role in alcohol ingestion by women and the elderly." Cappell and Herman (1972, p. 33) concluded that "much of the evidence is negative, equivocal and often contradictory." Nevertheless, surveys have shown evidence of drinking in the presence of stress in assorted populations, such as adolescents (Wills 1985) and young and middle-aged males (Welte 1985).

A REVIEW OF THE RESEARCH

Clinical Studies

Clinical studies of stress and late-onset problem drinking have yielded mixed results. Rosin and Glatt (1971) found that most late-onset alcoholics in a clinical sample gave stressful situations such as retirement or bereavement as causes for their problem; few elderly early-onset alcoholics cited stressful situations as instrumental in their drinking. Among clients of an outreach program, Hubbard and colleagues (1979) found that the problems of late-onset alcoholics could be traced back to a specific event such as loss of job or death of a spouse. Brown and Chiang (1984), in an analysis of older alcoholics and community controls, found that alcohol abuse was associated with living alone. Finlayson and colleagues (1988) compared late-onset versus early-onset alcoholics among 216 elderly patients being treated for alcoholism. More late-onset alcoholics mentioned a stressful life event in connection with their drinking problem, but specific events were not statisti-

cally related to the early onset–late onset distinction. The authors acknowledged that a "heightened need to rationalize" may be involved in elder alcoholics implicating a stressful life event in their alcohol dependence.

Schonfeld and Dupree (1991) performed a matched case-control study of elderly early-onset versus late-onset alcoholics who were in a treatment program for older alcoholics. The losses and stresses associated with aging (e.g., widowhood, reduced social network) did not distinguish these groups. In studies with the same design, Gurnack and Hoffman (1992) and Adams and Waskel (1991, 1993) also failed to support the notion that late-onset alcohol abuse was driven by stresses of aging.

Dufour and Fuller (1995) and Liberto and Oslin (1995) reviewed some of the studies examining stressful life events that may be implicated in late-onset versus early-onset alcohol dependence. Dufour and Fuller found mixed results, with some reason to believe that stress exacerbates an already existing late-onset alcohol problem. Liberto and Oslin also noted that stressful events might be implicated in relapse for late-onset alcoholics, but concluded that "there is little additional support to suggest that one group experiences stressful life events more so than the other group" (p. 1807). The pattern that appears in the studies reviewed in this section is that the earlier researchers were somewhat positive about the role of stress as a cause of late-onset alcohol dependence, but articles from recent years are mostly negative. This may be because the more recent studies used more thorough methods, including careful matching of controls and multivariate statistical techniques, such as discriminant function analysis and logistic regression, to compare the late-onset and early-onset groups.

Case-Control Studies With Community Controls

Case-control studies with community controls have also yielded mixed results. Several studies have been performed comparing older subjects with serious alcohol involvement or a stressful life event with controls from the general population. Wells-Parker and colleagues (1983) conducted a case-control study in which cases were elder Mississippians who were first offenders for DWI, with census tract controls who were both drinkers and drivers. Stressful life events were more frequent among cases, but the cases also had more extensive histories of alcohol involvement. In another case-control study, Valanis and colleagues (1987) compared bereaved elders located by death certificate with matched controls obtained from church lists, and also conducted a longitudinal followup. Alcohol use did not increase in the 9 months after the death of a spouse, providing no support for bereavement as a cause of heavy drinking. Bristow and Clare (1992) compared older hospital patients who were heavy drinkers with matched controls. At-risk drinkers were more likely to be unmarried and not have a close friend.

General Population Studies

Unlike clinical studies, general population studies of elder drinking often

include probability samples of the elder general population and allow comparison with elders who are not heavy or problem drinkers. These studies have usually yielded negative results. Barnes (1982), in an often-cited study of an Erie County, NY, general population sample, found that there was no correlation between elder heavy drinking and marital status (e.g., married vs. widowed), retirement, degree of satisfaction with one's life, degree of social activity, or general state of health. Kasl and colleagues (1987) conducted a large general population survey with elders in both age-segregated and age-integrated housing that included longitudinal followup. The cross-sectional results from the first wave showed total volume of alcohol consumed to be negatively correlated with poor health and size of social network, and not correlated with stressful life events or depression. They found slight indications of a positive relationship between drinking and neighborhood safety, but otherwise negative results. Their longitudinal analysis failed to demonstrate drinking increases after illness or death of spouse. La Greca and colleagues (1988) used a sample from two age-segregated and two age-integrated communities in different parts of the country. There was no main effect of life events on quantity of alcohol consumption, and no partial correlation between alcohol consumption and stress from life events while controlling for social support and coping resources.

Brennan and Moos (1990) studied a medical population of elders and found no correlation between stressful events and quantity of alcohol consumed. Other general population surveys that failed to find a relationship between elder drinking and stress were reported by Meyers and Goldman (1980), Joyce (1984), Kivela and colleagues (1988), Huffine and colleagues (1989), and Romelsjo and colleagues (1991). Finally, a survey was conducted by the Research Institute on Addictions (Welte and Mirand 1995) of 2,325 general population elders in Erie County, NY. No relationship between acute or chronic life stresses and likelihood of heavy drinking, regardless of coping strategy and quantity or quality of social supports, was found.

Krause (1991) has pointed out that research on elder drinking and stress may not have achieved positive results because in many cases various stressors are summed to form a scale, implicitly assuming that they *all* cause drinking. Since poor health and financial difficulties have been shown to be negatively related to drinking, a positive relationship with other life stressors might be masked. He performed a structural equation modeling analysis on a national survey, using *abstinence* as the dependent variable. He found that health problems were positively related to abstinence, and this relationship was mediated by religiosity. Financial difficulties were not significantly related. Krause concluded that we should not assume all stresses are positively related to drinking.

A few surveys have yielded ambiguous or positive results. Jennison (1992) tested the stress and buffering

theory of elder problem drinking using the National Opinion Research Center General Social Survey, including 1,418 respondents age 60 or older. Using a regression predicting drinking quantity with interaction terms between stress and various possible stress-buffering agents, she found a positive relationship between stressful life events and drinking behavior. This effect was buffered by sibling support, church attendance, quality of marriage, and friendship. Soderstrom (1992) interviewed 40 independent-living adults age 65 or older. No significant correlation was found between alcohol consumption and the Stokes/Gordon stress scale, but there were positive correlations between alcohol consumption and several individual stress items—death of loved one, concern about aging and (one's own) death, and work-related stress.

Stressful Events and Alcohol Consequences

Even if stress does not cause drinking, stressful events may contribute to alcohol consequences. Moos and colleagues (1990) screened 5,000 elderly people who were patients at two large medical centers and obtained 1,884 drinkers, who completed a mail questionnaire. The problem drinkers reported more negative life events and used more avoidance coping. The authors suggested that the *consequences* are caused by ineffective coping, not necessarily the drinking itself. The Research Institute on Addictions study of 2,325 general population elders in Erie County, NY (Welte and Mirand 1995), which failed to find a relationship between stress and alcohol consumption, also found a positive relationship between stress and negative consequences of drinking.

Alcohol as a Stress Buffer

A study by Krause (1995) provides a different and interesting approach to the subject. He examined the stress-buffering role of alcohol in a national survey of elders by using life stressors as the independent variable, depression as the dependent variable, and alcohol as a moderating or interaction variable. He found no main effect of stressors on alcohol consumption, but a buffering effect for nonsalient events, and an *increasing* effect for salient events. In the most important areas of life, drinking exacerbated the effects of stress on depression.

THE ERIE COUNTY ELDER DRINKING SURVEY—A SECOND LOOK

Findings from this study have been published elsewhere (Mirand and Welte 1994; Welte and Mirand 1994, 1995; Mirand and Welte 1996), and the primary findings pertaining to drinking and stress in the elderly already have been mentioned. However, the recent literature, as well as the perspective provided by the passage of time, has suggested additional investigation of the data. For this chapter, the analyses will include an examination of (1) the predictors of elder alcohol consumption, including interactions between the stress variables and other predictors to iden-

tify subgroups of the elder population in which stress may influence drinking; (2) the relationship between individual stress variables and drinking, as suggested by the work of Soderstrom (1992) and others who found relationships between elder drinking and individual stressful events or chronic stresses; (3) the relationship between stress and alcohol consequences or dependence, as suggested by the work of Moos and colleagues (1990) and others who implicated stressful events and chronic stresses as antecedents to drinking problems; and (4) drinking as a stress buffer, as suggested by the work of Krause (1995) and Neff and Husaini (1985).

Study Design and Methodology

The Erie County Elder Drinking Survey was designed in the late 1980's specifically to address the relationship between stress and elderly drinking in the general population. It included a large sample of elders with an oversample of heavy drinkers, probability sampling that allowed weighting for an unbiased estimate of the general elder population, thorough measurement of alcohol consumption and consequences, specific measurements of acute and chronic stressors, a variety of measures of social supports, and some commonly used measures of coping methods.

From May 1990 through June 1991 telephone interviews were conducted with 2,325 Erie County, NY, residents age 60 or older. These respondents were a representative sample of the elderly people living in the county. Sampling was done by random-digit dialing and was stratified by 10 geographic districts, with allocation of the sample proportional to the total number of phone numbers existing in those districts. Interviews were conducted using the computer-assisted telephone interviewing facility of the Research Institute on Addictions. Seven attempts were made to reach an eligible household before a number was abandoned. If a household contained more than one person age 60 or older, a respondent was selected at random using a selection table. Potential respondents were screened by being asked if they had drunk an average of two drinks per day at any time in their life. If they answered yes, the interviewer attempted to recruit them for a full interview. If they answered no, they were recruited a random one-third of the time. Interviews averaged 42 minutes in length. If an elderly respondent tired during the interview, a callback was scheduled.

Since a respondent was selected at random from those age 60 or over in the household, respondents who lived alone had a greater chance of selection. Because of our oversampling of heavy drinkers, those who answered positively to the screening question had a three times greater chance of being interviewed. A weight variable inversely proportional to sampling probability was constructed, so that the weights of screened "heavy drinkers" were one-third of the weights of others, and the weights of those who lived in a household with another elder were one-half of the weights of those who lived

alone. All analyses reported here are weighted accordingly.

Questionnaire Content

Demographic questions included gender, age, race, marital status, religion, family income, source of family income, education, occupation during working life, education of spouse, occupation of spouse, current employment status, and household composition (who lives with respondent).

Drinking questions included quantity/frequency of beer, wine, and liquor; signs of alcohol dependence (binge, shakes, morning drinking, tolerance, drinking rules, and/or drinking despite consequences); and alcohol-related consequences (accidents, DWI, liver disease, memory problems, and/or psychological distress). A condensed version of this series of questions was also asked with respect to the respondent at age 20, age 40, and the time in the respondent's life that the most drinking occurred. The quantity/frequency questions are a type frequently used in survey research (Russell et al. 1991). Current alcohol consumption was computed by multiplying quantity by frequency by alcohol content for each beverage and summing the results. The signs of dependence and alcohol-related consequences questions are taken from the Diagnostic Interview Schedule (Robins et al. 1988); they are also among the questions used for DSM-III-R (American Psychiatric Association 1987) diagnoses of alcohol abuse and alcohol dependence.

The following **psychosocial scales** were used:

- *Health orientation.* This scale, which was adapted from the work of Walker and colleagues (1987), measures the extent of engaging in good health practices (e.g., checking foods for nutrients). The scale has eight items and a reliability coefficient of 0.58. All items have positive item-to-total correlations.
- *Stressful life events (acute stresses).* The Elders Life Stress Inventory (Aldwin et al. 1989) measures the extent to which elders are distressed by specific life events such as retirement or death of a spouse. These events were selected and tested for relevance to the life of the elderly. Respondents were asked the extent to which they were bothered by each event: not at all, somewhat, or very much. This scale has 18 items and a reliability coefficient of 0.76. All items have positive item-to-total correlations.
- *Chronic stresses.* Our scale was a subset of the Daily Hassles scale of Lazarus (1980), selected to be especially relevant to the elderly. It measures stress from ongoing conditions such as caring for a sick family member, loneliness, or side effects of medication. Respondents were asked the extent to which they were bothered by each ongoing condition: not at all, somewhat, or very much. This scale has 29 items and a reliability coefficient of 0.88.
- *Active cognitive coping.* This scale uses coping methods from the Health and Daily Living Form Manual (HDL manual) (Moos et al. 1983) to measure the extent to which stress is dealt with by active

cognitive methods, such as trying to see the positive side of a problem. Active cognitive methods are considered productive coping methods. This scale has eight items and a reliability coefficient of 0.59. All item-to-total correlations are positive.

- *Active behavioral coping.* This scale, also from the HDL manual, measures the extent to which stress is dealt with by active behavioral methods, such as making a plan and following it. Active behavioral methods are considered productive coping methods. This scale has six items and a reliability coefficient of 0.66. All item-to-total coefficients are positive.
- *Avoidance coping.* This is another scale from the HDL manual; it measures the extent to which stress is dealt with by avoidance methods, such as pretending the problem doesn't exist. Avoidance methods are generally considered unproductive coping methods. This scale has five items. All item-to-total correlations are positive, but the coefficient of reliability is an unimpressive 0.37 because of the small number of items.
- *Social network.* This scale was adapted from the work of Dr. Lisa Berkman at the California State Department of Health Services Human Population Laboratory (Berkman and Breslow 1983). It measures the quantity of social supports such as friends, relatives, and group membership. This scale has four items and a reliability coefficient of 0.58, with strongly positive item-to-total coefficients.
- *Instrumental Social Support.* This scale, from Barerra and colleagues (1981), measures the extent to which others give you practical help, such as providing transportation. The scale has 11 items and a reliability coefficient of 0.75.
- *Emotional Social Support.* This scale, also from Barerra and colleagues (1981), measures emotionally supportive behaviors, such as expressing respect or showing affection. The scale has eight items and a reliability coefficient of 0.78.
- *Depression.* Depression was measured by the Geriatric Depression Scale (Sheikh et al. 1991). This scale has eight items and a reliability coefficient of 0.69. All item-to-total coefficients are positive.
- *Medical conditions.* Respondents were asked to indicate illnesses by responding to a checklist of conditions taken from the HDL manual.
- *Medical symptoms.* Respondents were asked to indicate if they suffered from common symptoms, such as indigestion or poor appetite, by responding to a checklist of symptoms taken from the HDL manual.

Results of the "Second Look" Analyses

Alcohol Consumption and Aggregated Stress Scales

The first step in this analysis was a linear regression predicting current average alcohol consumption. The dependent variable was log-transformed to partly normalize its distribution by correcting the positive skewness. The main effects model included all of the variables listed in table 1, which were forced in and then eliminated backward

if they failed to be significant at the 0.01 level. The "beta," or normalized regression coefficient, gives an idea of the strength of the effect. Table 1 shows that average consumption at age 40 is the dominant predictor, and that acute or chronic stress has no effect. Active lifestyle, socioeconomic status, and male gender are positively related to current alcohol consumption. Those who have a health orientation drink less, as do those who have more medical conditions.

The second phase of this regression analysis (not shown in the table) was the insertion of all the main effects, followed by the interaction terms between the two stress variables and all the other variables, and backward elimination of the interaction variables. The purpose was to test for subgroups of the elder population in which stress and alcohol consumption might be related, particularly those with inadequate coping resources or social support. None of the interaction terms survived the backward elimination. As before with a somewhat less comprehensive predictive model (Welte and Mirand 1995) and a dichotomous "heavy drinking" dependent variable, we found no relationship between acute or chronic stress and alcohol consumption regardless of coping method or social support. In this more extensive

Table 1. Linear Regression Predicting Current Average Alcohol Consumption (N=1,909 Erie County Residents Age 60 or Older).

	Initial Main Effects Model		Model After Backward Elimination	
Predictor	**Beta**	***p***	**Beta**	***p***
Average consumption age 20	0.05	0.03		
Average consumption age 40	0.52	<0.0001	0.55	<0.0001
Gender (1 = M, 0 = F)	0.04	0.04	0.06	0.0008
Age	-0.01	0.59		
Race (1 = White)	0.02	0.25		
Marital status (1 = Married)	0.00	0.96		
Employment (1 = Employed)	0.00	0.93		
Socioeconomic status	0.08	0.0001	0.08	<0.0001
Chronic stress	0.03	0.31		
Acute stress	0.00	0.99		
Active behavioral coping	-0.01	0.74		
Active cognitive coping	-0.05	0.05	-0.05	0.02
Avoidance coping	-0.02	0.39		
Size of social network	0.05	0.10		
Instrumental social support	-0.02	0.50		
Emotional social support	0.00	0.93		
Health-oriented lifestyle	-0.06	0.01	-0.05	0.01
Depression	-0.03	0.15		
Medical conditions	-0.08	0.0002	-0.09	<0.0001
Medical symptoms	-0.01	0.81		
Active lifestyle	0.10	<0.0001	0.13	<0.0001

analysis we also found negative results with health status, depression, and lifestyle factors as interaction variables.

Alcohol Consumption and Individual Stressors

Several researchers have pointed out that important relationships might be missed by using aggregated stress scales as the sole measure of stress. We calculated correlation coefficients between the log transform of average alcohol consumption (the same variable used as the dependent variable in the above regression) and each of the 47 items that make up the acute and chronic stress scales. Those that were significant at the 0.01 level are shown in table 2. They have been grouped to reflect their common themes: financial problems, loneliness, and poor health. The fourth group is a catchall, but might reflect difficulty with access to the world outside the home. The reader will quickly note that all of these significant coefficients, with the sole exception of "sexual problems," are negative. More stress is associated with less drinking. It is also worth noting that the effect sizes are very small, with coefficients that explain 2 percent or less of total variance.

Alcohol Problems and Stress

Among the implications of our past work and that of other researchers is that there might be a relationship between life stresses and alcohol dependence and consequences. (Our "problems" variables reflect both adverse alcohol consequences and signs or symptoms of alcohol dependence,

Table 2. Correlations Between Average Alcohol Consumption and Individual Stress Variables (N = 2,200 Erie County Residents Age 60 or Older).

Chronic and Acute Stressors	r	p
Not enough money for necessities	-0.15	<0.001
Financial insecurity	-0.09	<0.001
Deterioration of financial state	-0.12	<0.001
Move to less desirable residence	-0.06	0.005
Too much time on hands	-0.07	0.002
Loneliness	-0.06	0.004
Friends or relatives too far away	-0.06	0.006
Loss of friendship	-0.06	0.003
Side effects of medications	-0.08	<0.001
Sexual problems	0.12	<0.001
Major illness	-0.10	<0.001
Decrease in activities you enjoy	-0.06	0.007
Transportation problems	-0.12	<0.001
Difficulty shopping	-0.07	0.001
Crime	-0.07	0.001

Note: Correlations displayed are significant at the 0.01 level.

as discussed earlier in this chapter. For convenience, they will often be referred to by the simplified terms "problems" or "consequences"). We measured the respondent's alcohol-related consequences and dependence, currently and at ages 40 and 20. Summary scales were constructed by simply counting the number of consequences and signs of dependence. In our earlier work (Welte and Mirand 1995) we discovered that there was a positive correlation between stress and alcohol problems in the absence of a positive correlation with alcohol consumption itself, and we speculated that life stresses might exacerbate the consequences or at least make them seem worse. In the current analyses, we are examining the stress/drinking problem connection in more detail.

Table 3 shows the simple bivariate correlations between the (current) stress variables and drinking problems currently and at ages 40 and 20. As we reported before, there is a small but significant positive correlation between current drinking and current problems. However, the correlations with past problems are slightly stronger, casting doubt on our conjecture that stress was influencing problems, and making it seem more likely that past problems might be influencing current stress.

For a deeper examination of this matter, we performed a regression predicting current problems. This analysis had a structure very similar to the prediction of current consumption described earlier: backward elimination of main effects, followed by reinsertion of the main effects and backward elimination of interaction terms. Once again, all possible two-way interactions involving the stress variables were employed. Table 4 shows the surviving main effects: current problems are positively related to current consumption, past problems, and male gender. They are negatively related to active lifestyle. A peculiar finding is that they are negatively related to consumption at age 20, keeping in mind that other predictors are held constant.

The interaction analysis (not shown in table 4) found one significant interaction term: chronic stress by problems at age 20. Table 5 displays the meaning of this interaction. For those who had no alcohol problems at age 20, there is no correlation between stress and current problems. However, for those who did report one or more problems at age 20, there is a substantial positive correlation of 0.31. Thus,

Table 3. Correlations Between Alcohol-Related Problems and Stress (N = 2,246 Erie County Residents Age 60 or Older).

	Acute Stress		Chronic Stress	
	r	p	r	p
Current problems	0.04	0.07	0.06	0.004
Problems age 40	0.05	0.009	0.11	<0.001
Problems age 20	0.07	<0.001	0.10	<0.001

Table 4. Linear Regression Predicting Current Alcohol-Related Problems (N = 1,909 Erie County Residents Age 60 or Older).

	Initial Main Effects Model		Model After Backward Elimination	
Predictor	**Beta**	***p***	**Beta**	***p***
Problems age 20	0.17	<0.0001	0.18	<0.0001
Problems age 40	0.17	<0.0001	0.16	<0.0001
Current average alcohol consumption	0.32	<0.0001	0.28	<0.0001
Average consumption age 20	-0.09	0.004	-0.10	<0.0001
Average consumption age 40	-0.05	0.13		
Gender (1 = M, 0 = F)	0.10	0.0001	0.08	0.001
Age	0.00	0.91		
Race (1 = White)	-0.02	0.33		
Marital status (1 = Married)	-0.02	0.51		
Employment (1 = Employed)	-0.02	0.42		
Socioeconomic status	-0.04	0.08		
Chronic stress	-0.01	0.75		
Acute stress	0.02	0.58		
Active behavioral coping	-0.03	0.35		
Active cognitive coping	0.01	0.63		
Avoidance coping	0.00	0.84		
Size of social network	0.00	0.90		
Instrumental social support	-0.03	0.33		
Emotional social support	0.01	0.62		
Health-oriented lifestyle	0.00	0.89		
Depression	0.01	0.61		
Medical conditions	0.04	0.14		
Medical symptoms	0.03	0.34		
Active lifestyle	-0.05	0.06	-0.08	0.0001

the interaction analysis has revealed that the modest but highly significant (0.06) correlation between chronic stress and current problems all comes from the 7 percent of the respondents who had problems at age 20.

Alcohol as a Stress Buffer for Depression

The Krause (1995) study as well as earlier work by Neff and Husaini (1985) suggested a different role for alcohol—that of moderator of the effect of stress on depression. To explore this possibility, we conducted a regression with depression as the dependent variable and with current alcohol consumption as well as acute and chronic stress as independent variables. Interactions between alcohol consumption and the stress variables were also tested. Table 6 shows the results.

Chronic stress had a strong positive association with depression, acute stress had a positive association, and quantity of alcohol consumption had a negative association. The interactions did not achieve statistical significance. However, we took the liberty of exploring the interaction of alcohol

Table 5. Display of Interaction Term From Regression Predicting Current Alcohol-Related Problems: Chronic Stress by Problems at Age 20.

	r	Current Problem and Chronic Stress
No problems age 20 (n = 2,050)	0.00	Not significant
Problems age 20 (n = 150)	0.31	Significance = <0.001

Table 6. Linear Regression Predicting Current Depression (N = 2,146 Erie County Residents Age 60 or Older).

Predictor	Beta	*p*
Current alcohol consumption (CA)	-0.08	<0.0001
Chronic stress (CS)	0.39	<0.0001
Acute stress (AS)	0.11	<0.0001
CA x CS	-0.05	0.06
CA x AS	0.02	0.33

Display of Interaction: Alcohol Consumption by Chronic Stress

Level of Consumption	Slope of Depression on Stress
Abstainer	0.26
Less than ½ drink per day	0.29
½ to 1 drink per day	0.18
1 to 2 drinks per day	0.16
More than 2 drinks per day	0.18

consumption and chronic stress, which had a p level of 0.06. The bottom half of table 6 shows the regression slopes of depression on chronic stress at five levels of alcohol consumption. For those elders who drank more than ½ drink per day on the average, the impact of chronic life stressors on depression was less. Alcohol consumption may have served as a buffer.

CONCLUSIONS

Our "second look" at the Erie County Elder Drinking Survey led to several conclusions. First, *stress is not an important antecedent of alcohol consumption of the elderly.* These analyses reveal no evidence of a relationship between chronic and acute life stresses and amount of drinking in our elderly sample. This is true even for those with weak social supports and inadequate coping methodology. It is also true for various other subgroups of elders: males and females, elderly persons across the socioeconomic spectrum, the retired and the employed, and so on. As our earlier review demonstrated, this negative result is consistent with a large number of studies. Although several studies report indications that drinking might be related to bereavement or loneliness, the major general popu-

lation survey that reported a positive relationship between a variety of life stresses and alcohol consumption is that of Jennison (1992). She also found this relationship to be buffered by several support factors such as religiosity and friendship.

Our investigation of the relationship between individual stressors (as opposed to a total stress score) and drinking quantity revealed negative correlations: the sicker, poorer, lonelier, and more isolated our respondents were, the less they drank. The connection between less drinking and lower income or poor health is consistent with the results of other research and also with common sense. The affluent use more alcohol as with any other consumer good. Sick people drink less because alcohol may aggravate their conditions or interact with their medications. Our prediction model for current alcohol consumption revealed consumption at age 40 to be by far the strongest predictor of current consumption, with active lifestyle as the second strongest. It is not the beleaguered elderly who drink the most, rather it is the active and vigorous, and most of all, those who drank substantial amounts earlier in life.

Second, *chronic stress may aggravate drinking consequences for those who had negative consequences when younger.* We found a small correlation between chronic stress and current problems, and a somewhat stronger correlation with past problems. This contemporary correlation comes from the small fraction of the sample that had problems in the past. These results must also be interpreted in light of the fact that chronic stress is not positively related to either past or current alcohol consumption. Our combination of findings is not consistent with several conspicuous explanations. If it were true that chronic stress was making current problems worse, one would expect the correlation of stress with current problems to be stronger than with past problems. If current drinking was causing problems which in turn were causing the stress, there would be a correlation between current drinking and chronic stress. Likewise, if it were true that past drinking was causing both current stress and current problems, then there would be a correlation between past drinking and current stress. It seems that for those who had already in past years established their vulnerability to drinking consequences, current life stressors will revive that vulnerability, holding constant the amount of alcohol consumption.

Third, *moderate drinking may buffer the negative effects of stress.* The depression-buffering analysis was suggested to us by the work of Krause (1995) and Neff and Husaini (1985). They investigated the possibility that drinking might buffer or protect against the effects of life stresses on depression. Neff and Husaini made a distinction between coping and buffering. *Coping* refers to specific responses to stressors, and drinking to cope may have deleterious consequences. An alcohol *buffer* represents typical patterns of alcohol consumption that would "serve to alleviate the tension and pressure caused by minor problems encountered in daily life" (p. 208). We found some evidence to this effect, as the regres-

sion slope of depression on chronic stress—that is, the additional "units" of depression acquired with each additional "unit" of stress—was lower for those who drank more (although this interaction had a p level of 0.06). This result hints at a favorable effect of regular moderate drinking.

Finally, some results surfaced during these analyses that were tangential to the main subject, but worthy of brief mention. The first of these is the role of active lifestyle, which proved to be positively related to amount of alcohol consumption, and negatively related to adverse consequences holding constant the amount of consumption. *A vigorous lifestyle is associated with more drinking, but has some protective effects against negative consequences.* A second noteworthy result is that *current problems are negatively related to amount of consumption at age 20*, holding constant current consumption and past problems. The better one could withstand drinking-related repercussions in the past, the less likely one is to suffer them in the present.

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REFERENCES

Adams, S.L., and Waskel, S.A. Late onset of alcoholism among older midwestern men in treatment. *Psychol Rep* 68:432–434, 1991.

Adams, S.L., and Waskel, S.A. Late onset alcoholism: Stress or structure. *J Psychol* 127(3):329–334, 1993.

Aldwin, C.M.; Levenson, M.R.; Spiro, A., III; and Bosse, R. Does emotionality predict stress? Findings from the normative aging study. *J Pers Soc Psychol* 56(4):618–624, 1989.

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 3d ed., rev. Washington, DC: the Association, 1987.

Atkinson, R.M.; Turner, J.A.; Kofoed, L.L.; and Tolson, R.L. Early versus late onset alcoholism in older persons: Preliminary findings. *Alcohol Clin Exp Res* 9(6):513–515, 1985.

Bahr, H. Lifetime affiliation patterns of early-and late-onset heavy drinkers on skid row. *Q J Stud Alcohol* 30:645–656, 1969.

Barerra, M., Jr.; Sandler, I.N.; and Ramsey, T.B. Preliminary development of a scale of social support: Studies on college students. *Am J Community Psychol* 9(4):435–447, 1981.

Barnes, G.M. Patterns of alcohol use and abuse among older persons in a household population. In: Wood, W.G., and Elias, M.F., eds. *Alcoholism and Aging: Advances in Research.* Boca Raton, FL: CRC Press, 1982.

Berkman, L.F., and Breslow, L. *Health and Ways of Living: The Alameda County Study.* New York: Oxford University Press, 1983.

Brennan, P.L., and Moos, R.H. Life stressors, social resources, and late-life problem drinking. *Psychol Aging* 5(4):491–501, 1990.

Bristow, M.F., and Clare, A.W. Prevalence and characteristics of at-risk drinkers among elderly acute medical inpatients. *Br J Addict* 87:291–294, 1992.

Brown, B.B., and Chiang, C.P. Drug and alcohol abuse among the elderly: Is being

alone the key? Int *J Aging Hum Dev* 18(1):1–12, 1984.

Bucholz, K.K., Sheline, Y.I., and Helzer, J.E. The epidemiology of alcohol use, problems, and dependence in elders: A review. In: Beresford, T., and Gomberg, E., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 19–41.

Cappell, H., and Herman, C.P. Alcohol and tension reduction: A review. *Q J Stud Alcohol* 33:33–64, 1972.

Dohrenwend, B.S., and Dohrenwend, B.P. *Stressful Life Events: Their Nature and Effects.* New York: Wiley. 1974.

Dufour, M., and Fuller, R.K. Alcohol in the elderly. *Annu Rev Med* 46:123–132, 1995.

Finlayson, R.E.; Hurt, R.D.; Davis, L.J., Jr.; and Morse, R.M. Alcoholism in elderly persons: A study of the psychiatric and psychosocial features of 216 patients. *Mayo Clin Proc* 63:761–768, 1988.

Folkman, S., and Lazarus, R.S. An analysis of coping in a middle-aged community sample. *J Health Soc Behav* 21:219–239, 1980.

Ganikos, M.; Blow, F.C.; and Clark, L. *Alcohol and Older People: New Challenges for Mental Health Workers.* Rockville, MD: National Institute on Alcohol Abuse and Alcoholism, 1988.

Glass, T.A.; Prigerson, H.; Kasl, S.V.; and Mendes de Leon, C.F. The effects of negative life events on alcohol consumption among older men and women. *J Gerontol* 50B(4):S205-S216, 1995.

Gurnack, A.M., and Hoffman, N.G. Elderly alcohol misuse. *Int J Addict* 27(7):869–878, 1992.

House, J.S., and Kahn, R.L. Measures and concepts of social support. In: Cohen, S., and Syme, S.L., eds. *Social Support and Health.* San Diego, CA: Academic Press, 1985. pp. 83–105.

Hubbard, R.W.; Santos, J.F.; and Santos, M.A. Alcohol and older adults: Overt and covert influences. *Soc Casework* 60:166–170, 1979.

Huffine, C.L.; Folkman, S.; and Lazarus, R.S. Psychoactive drugs, alcohol and stress and coping processes in older adults. *Am J Drug Alcohol Abuse* 15(1):101–113, 1989.

Hurt, R.D.; Finlayson, R.E.; Morse, R.M.; and Davis, L.J. Alcoholism in elderly persons: Medical aspects and prognosis of 216 inpatients. *Mayo Clin Proc* 63:753–760, 1988.

Jennison, K.M. The impact of stressful life events and social support on drinking among older adults: A general population survey. *Int J Aging Human Dev* 35(2):99–123, 1992.

Joyce, K. Alcohol use and event-related stress among community elderly. (Ph.D. dissertation, Case Western Reserve University, 1984.) *Dissertation Abstr Int* 46(6), 1985.

Kasl, S.V.; Ostfeld, A.M.; Berkman, L.F.; and Jacobs, S.C. Stress and alcohol consumption: The role of selected social and environmental factors. In: Gottheil, E.; Druley, K.A.; Pashko, S.; and Weinstein, S.P., eds. *Stress and Addiction.* New York: Brunner/Mazel, 1987. pp. 40–60.

Kivela, S.L.; Nissinen, A.; Punsar, S.; Puska, P.; and Karvonen, M. Determinants and predictors of heavy alcohol consumption among aging Finnish men. *Compr Gerontol B* 2:103–109, 1988.

Krause, N. Stress, religiosity, and abstinence from alcohol. *Psychol Aging* 6(1):134–144, 1991.

Krause, N. Stress, alcohol use, and depressive symptoms in later life. *Gerontologist* 35(3):296-307, 1995.

La Greca, A.J.; Akers, R.L.; and Dwyer, J.W. Life events and alcohol behavior among older adults. *Gerontologist* 28(4):552–558, 1988.

Lazarus, R.S. The stress and coping paradigm. In: Eisdorfer, C.; Cohen, D.; and Kleinman, A., eds. *Conceptual Models for Psychopathology.* New York: Spectrum, 1980. pp. 173–209.

Liberto, J.G., and Oslin, D.W. Early versus late onset of alcoholism in the elderly. *Int J Addict* 30(13&14): 1799–1818, 1995.

Meyers, A.; and Goldman, E. Life stress, life satisfaction, and drinking in old age. *Gerontologist* 20:161, 1980.

Mirand, A.L., and Welte, J.W. Total body water adjustment of mean alcohol intakes. *J Subst Abuse* 6:419–425, 1994.

Mirand, A.L., and Welte, J.W. Alcohol consumption among the elderly in a general population, Erie County, New York. *Am J Public Health* 86(7):978–984, 1996.

Moos, R.H.; Cronkite, R.C.; Billings, A.G.; and Finney, J.W. *Health and Daily Living Form Manual.* Palo Alto, CA: Veterans Administration and Stanford University Medical Centers, 1983.

Moos, R.H.; Brennan, P.L.; Fondacaro, M.R.; and Moos, B.S. Approach and avoidance coping responses among older problem and nonproblem drinkers. *Psychol Aging* 5(1):31–40, 1990.

Neff, J.A., and Husaini, B.A. Stress-buffer properties of alcohol consumption: The role of urbanicity and religious identification. *J Health Soc Behav* 26:207–222, 1985.

Peyser, H. Stress and alcohol. In: Goldberger, L., and Breznitz, S., eds. *Handbook of Stress: Theoretical and Clinical Aspects.* New York: Free Press, 1982.

Pohorecky, L.A. The interaction of alcohol and stress: A review. *Neurosci Biobehav Rev* 5:209–229, 1981.

Pohorecky, L.A. Stress and alcohol interaction: An update of human research. *Alcohol Clin Exp Res* 15:438–459, 1991.

Robins, L.; Helzer, J.; Cottler, L.; and Goldring, E. *NIMH Diagnostic Interview Schedule. Version III Revised (DIS–III–R).* St. Louis, MO: Washington University, 1988.

Romelsjo, A.; Lazarus, N.B.; Kaplan, G.A.; and Cohen, R.D. The relationship between stressful life situations and changes in alcohol consumption in a general population sample. *Br J Addict* 86:157–169, 1991.

Rosin, A.J., and Glatt, M.M. Alcohol excess in the elderly. *Q J Stud Alcohol* 32:53–59, 1971.

Russell, M.; Welte, J.W.; and Barnes, G.M. Quantity-frequency measures of alcohol consumption: Beverage-specific vs. global questions. *Br J Addict* 86:409–417, 1991.

Schonfeld, L., and Dupree, L.W. Antecedents of drinking for early-and late-onset elderly alcohol abusers. *J Stud Alcohol* 52(6):587–592, 1991.

Sheikh, J.I.; Yesavage, J.A.; Brooks, J.O.; Friedman, L.; and Gratzinger, P. Proposed factor structure of the geriatric depression scale. *Int Psychogeriatr* 3(1):23–66, 1991.

Soderstrom, M. The use of alcohol by community-based older adults as a response to stress experienced: A quantita-

tive and qualitative study. *Dissertation Abstr Int* 54:2338, 1992.

Valanis, B.; Yeaworth, R.C.; and Mullis, M.R. Alcohol use among bereaved and nonbereaved older persons. *J Gerontol Nurs* 13(5):26–32, 1987.

Vinokur, A., and Selzer, M.L. Desirable versus undesirable life events: Their relationship to stress and mental disease. *J Pers Soc Psychol* 32(2):329–339, 1975.

Walker, S.N.; Sechrist, K.R.; and Pender, N.J. The health-promoting lifestyle profile: Development and psychometric characteristics. *Nurs Res* 36(2):76–81, 1987.

Wells-Parker, E.; Miles, S.; and Spencer, B.: Stress experiences and drinking histories of elderly drunken-driving offenders. *J Stud Alcohol* 44(3):429–437, 1983.

Welte, J.W. Alcohol use and trait anxiety in the general population. *Drug Alcohol Depend* 15:105–109, 1985.

Welte, J.W., and Mirand, A.L. Lifetime drinking patterns of elders from a general population survey. *Drug Alcohol Depend* 35:133–140, 1994.

Welte, J.W., and Mirand, A.L. Drinking, problem drinking and life stressors in the elderly general population. *J Stud Alcohol* 56(1):67–73, 1995.

Wills, T.A. Stress, coping, and tobacco and alcohol use in early adolescence. In: Shiffman, S., and Wills, T.A., eds. *Coping and Substance Use.* New York: Academic Press, 1985.

Wilsnack, S.; Vogeltanz, N.; Diers, L.; and Wilsnack, R. Drinking and problem drinking in older women. In: Beresford, T., and Gomberg, E., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 263–292.

Zucker, R.A. The four alcoholisms: A developmental account of the etiologic process. In: Rivers, P.C., ed. *Nebraska Symposium on Motivation. Alcohol and Addictive Behaviors.* Lincoln: University of Nebraska Press, 1987. pp. 27–83.

Chapter 14

Personality and Problem Drinking in Middle-Aged and Older Men: Longitudinal Findings From the Normative Aging Study

Michael R. Levenson, Ph.D., Carolyn M. Aldwin, Ph.D., Avron Spiro III, Ph.D., and Leanne J. Friedman

Light to moderate consumption of alcoholic beverages is normative in the United States, as well as many other countries. However, many other patterns of alcohol-related behavior can also be found, including problem drinking, heavy drinking, and lifetime abstention from drinking when such abstention is not normative in a given subculture. Deviant behavior associated with alcohol consumption is typically thought of as "excessive" consumption or "abuse," but these constructs are not at all well defined. For example, "abuse" can mean, among other things, drinking in quantities that threaten health, antisocial behavior associated with alcohol consumption (without regard to amount consumed), financial problems caused by the purchase of alcoholic beverages (which are necessarily related to wealth or income), and family disturbances arising from spousal differences in standards of normal and excessive consumption. All of these problems associated with alcohol consumption are represented in commonly used

M.R. Levenson, Ph.D., is a research psychologist and C.M. Aldwin, Ph.D., is a professor in the Department of Human and Community Development, One Shields Ave., University of California at Davis, Davis, CA 95616. A. Spiro III, Ph.D., is a research statistician for the Normative Aging Study at the Department of Veterans Affairs Medical Center, 200 Springs Rd., Bedford, MA 01730. L.J. Friedman is a programmer in the Department of Human and Community Development, University of California at Davis.

self-report questionnaires (Cahalan et al. 1969; Midanik 1988). Taken together, such item sets constitute a rough but useful measure of drinking problems.

Recognized problems constitute only one category of deviant behavior related to alcohol. Another is relatively heavy drinking without reported problems. This is a matter of special concern in older persons with decreased capacity to process alcohol physiologically (Atkinson 1987) and increased use of medication which may interact with alcohol (Williams 1984). Thus, a level of consumption that was not problematic earlier in life may become problematic with aging. There is evidence that there are fewer heavy drinkers among the elderly (Fillmore et al. 1991; Liberto et al. 1992); however, Neve and colleagues (1993) found that, although abstention increased with age, nonabstainers did not generally decrease their consumption with age. Evidence from our own sample indicates that there were cohort, age, and period (time of measurement) effects in alcohol consumption, with period effects proving strongest (Levenson et al. in press).

Another issue in the consideration of both problem and heavy drinking is that of chronicity. Indeed, chronicity—the stability of a drinking problem over time—may be one index of seriousness of risk for alcohol-related physical and mental health problems. Yet the scarcity of longitudinal data (Fillmore 1988) has limited direct examination of this matter.

Another form of alcohol deviance is lifetime abstention. Although a fairly large percentage of Americans report abstention at any one assessment, consistent abstention through the life course is probably infrequent, especially for men who do not belong to a religious or ethnic group in which such abstention is normative. Many in the treatment field might view this as a very benign form of deviance, but as part of the picture of alcohol-related behavior it is intrinsically interesting. For instance, the possibility exists that the refusal to ever sample alcoholic beverages might be related to some form of psychological distress. To our knowledge, there are no personality studies of teetotalers, but there is some evidence from a study of marijuana abstainers. Shedler and Block (1990) found more psychological distress among both heavy users and nonusers of marijuana than among (presumably normative) moderate users in an adolescent sample. This suggested that individuals who abstain from a legal substance might also be likely to show higher levels of psychological distress. Moreover, the potential beneficial effects of light to moderate alcohol consumption cannot be dismissed (Blackwelder et al. 1980; Klatsky et al. 1981).Teetotalers appear to be at higher risk for coronary heart disease (De Labry et al. 1992).

There is a long-standing research tradition that seeks to find psychological predictors of alcohol problems, especially in the domain of personality. However, Levenson and colleagues (1990) examined one major limitation of most studies that show personality traits to be associated with alcohol abuse: most were conducted

on samples of persons already identified as having such problems (e.g., clinical samples of alcoholics). We used the MAC scale (MacAndrew Alcoholism Scale), a personality instrument that had proved to be very high in criterion level validity (i.e., in classifying alcoholics in treatment) (MacAndrew 1965, 1981), to try to identify problem drinkers in a nonclinical (i.e., noncriterion-based) sample. The false-positive rate, reflecting the presence of a score at or above the clinical cutting score for predicting alcoholism among those who had no behavior problems, was over 70 percent. The problem of predicting low base rate phenomena such as drinking problems in an unselected population is one of specificity or predictive validity.

Another problem with studies associating personality and drinking problems is that they are likely to be cross-sectional. Thus, one important issue in the assessment of a potential relationship between personality and alcohol abuse cannot be examined—the issue of causal directionality. It is possible that drinking problems result in part from personality traits, but it is also possible that personality traits reflecting psychological distress result from drinking problems (Vaillant 1983), especially chronic ones. It seems probable that both kinds of association would be found. It is unlikely that any extant data set contains lifespan personality and drinking data in sufficient detail to answer this question completely. Yet a study that assesses both variables over a significant part of the adult lifespan can take some significant steps in that direction, if not exactly toward causal directionality, at least toward increased reliability of associations between personality traits and alcohol abuse.

In other words, without longitudinal research the degree of stability of relationships between personality traits and deviant alcohol-related behavior cannot be assessed. Stability of these relationships over some period of time would increase confidence that they reflect an underlying psychological reality.

Another question that has been raised in the literature on personality and problem drinking is whether or not there are consistent personality differences between those whose drinking problems began early in life and late-onset problem drinkers. Cross-sectional data suggest that early onset of problem drinking is frequently associated with antisocial personality, while late onset is not (Varma et al. 1994; Schuckit et al. 1995).

PRESENT STUDY

We began with an examination of the level of stability in problem and heavy drinking in the Boston Department of Veterans Affairs Normative Aging Study (NAS) population. The study further examined differences in personality trait structure among different patterns of drinking behavior. We examined Minnesota Multiphasic Personality Inventory (MMPI) clinical and validity scales plus special alcohol scales in four groups: chronic problem drinkers assessed by self-report items covering a variety of alcohol-related problems (see table 1); lifetime abstainers (teetotalers);

Table 1. Items Assessing Problem Drinking the Past Year.

1. I got drunk too often. (2)
2. I felt sick upon awakening. (2)
3. I had memory lapses or blackouts. (2)
4. I had the shakes. (2)
5. I had difficulty sleeping. (3)
6. I became hostile when I drank. (2)
7. I was skipping meals. (3)
8. It made me more depressed. (2)
9. I was arrested for driving while intoxicated. (1)
10. I was arrested for disturbing the peace. (1)
11. I hurt myself physically when drunk. (2)
12. It affected my health. (2)
13. It affected my relationship with my family. (2)

Note: A respondent was considered to have a drinking problem if he reported one or more items at the level noted by the number in parentheses after each item: (1) = once a year, (2) = once a month, (3) = once a week.

chronic heavy drinkers who did not report problems; and light to moderate nonproblem drinkers. We also contrasted early- and late-onset problem drinkers. We hypothesized that alcohol deviance of any kind would be associated with personality traits reflecting more psychological distress than would be found among light to moderate nonproblem drinkers.

We also hypothesized that early- and late-onset problem drinking would be reflected in personality differences, with early-onset problem drinkers being characterized by a greater degree of antisociality. We further hypothesized that men reporting alcohol problems at all three points would differ from those reporting problems only in 1973 in ways similar to the differences between the former group and those who never reported problems.

METHOD

Study Population

The overall study population consisted of 1,056 men from the Normative Aging Study who responded to alcohol surveys administered by mail at three time points (1973, 1982, and 1991) and who completed the MMPI administered by mail in 1986 and 1991. The original NAS sample was composed of 2,280 men ages 20–80 who were screened for the absence of chronic or life-threatening illness, blood pressure at or below 140/90, and geographic stability, defined as having extensive social ties in the Boston area and stated intentions to remain there. They were enrolled between 1961 and 1968 (cf. Bossé et al. 1984). The men were not screened for problem drinking or alcoholism, although those with cirrhosis, pancreatitis, and gout, which are often associated with alcoholism, were excluded (Glynn et al. 1985).

Procedures

Drinking surveys were administered by mail in 1973, 1982, and 1991 and were returned with a response rate of over 80 percent on each. The interview was a modified form of the one developed for a national survey of drinking practices (Cahalan et al. 1969). Identical questions were used at all three time points. Respondents were given a list of 16 types of alcoholic beverages and asked to indicate the average number of drinks of each they were currently consuming per day, week, month, or year, as they felt appropriate. These

data were used to calculate an estimated drinks per year (dpy), computed using the Agricultural Research Service standard of 3/5 (0.60) oz of alcohol per drink. Those consuming between 30 and 1,094 dpy were considered light to moderate drinkers. *Heavy drinking* described the heaviest drinking 10 percent of the population. Among those who responded to the personality survey in 1986 and 1991, only 13 and 14 respondents, respectively, were heavy drinkers at all three assessment points. These respondents were characterized as chronic heavy drinkers. The heavy drinkers averaged 4.1 drinks per day.

In the 1973, 1982, and 1991 drinking surveys, respondents were also given a list of 13 items which asked about the current negative effects of drinking and were asked to indicate how frequently they experienced each of these effects (see table 1). As noted in the table, a respondent was considered to have a drinking problem if he reported at least one of the effects once a year, month, or week. Interestingly, there was almost no overlap between the chronic heavy drinkers and the chronic problem drinkers. Therefore, we created discrete categories for individuals who were only chronic heavy drinkers or only chronic problem drinkers, but not both, for all but one of the analyses (reported in table 7). Only four individuals were excluded by this criterion.

As table 2 indicates, 223 men reported a problem at one or more assessment points, but "turnover" rates in those experiencing problems were quite high from one assessment to another. For example, only half of those reporting problems in 1973 reported them in 1982. Only 25 of the 223 reported problems at all three times. Because the number of men reporting problems at all three assessment points was so small, we conducted the same analyses for men who reported problem or heavy drinking at only two assessment points. No participants who failed to respond to all of the surveys were included in this second set of analyses.

Personality Measures

The revised Minnesota Multiphasic Personality Inventory (MMPI-2) was administered to the NAS men in 1986

Table 2. Stability and Change in Problem Drinking (N = 1,056).

Problem Group	*n*	%
No problem	833	78.9
1973 only	56	5.3
1982 only	53	5.0
1973 and 1982	28	2.7
1991 only	24	2.3
1973 and 1991	10	0.9
1982 and 1991	27	2.6
1973, 1982, and 1991	25	2.4

Note: Data are given for subjects who responded at all three assessment points.

and 1991. Although the MMPI-2 was first published in 1989 (Butcher et al. 1989), the 1986 administration of this instrument was derived from an experimental form called the MMPI-AX, which was used to develop the MMPI-2. All MMPI-2 items were included in the MMPI-AX and were extracted for use in this study. We used the MMPI's 3 validity scales (L, F, and K) and 10 clinical scales (Hs, D, Hy, Pd, Mf, Pa, Pt, Sc, Ma, and Si). (See table 3 for an explanation of these scales.) MMPIs were mailed to 1,887 active participants in the NAS in 1986 and to 1,706 participants in 1991. Response rates, excluding invalid questionnaires, were 78 percent (n = 1,472) in 1986 and 70 percent (n = 1,194) in 1991.

We also computed two additional scales: the MacAndrew Alcoholism Scale, Revised (MAC-R) (MacAndrew 1981), the new version of one of the most widely used scales in alcohol research (MacAndrew 1965); and the more recently developed Addiction Potential Scale (APS) (Weed et al. 1992). Although space does not permit a discussion of each scale here (see Graham 1994 for more information), note the caveat reported earlier in this chap-

Table 3. Multivariate Analyses of Variance Examining Alcohol Problem Groups by 1986 MMPI-2 Scores:Purified Sample.

	Group						
Scale	1	2	3	4	Univariate F (3, 74)	p	Scheffé's ($p < 0.05$)
L	3.38	4.15	5.42	4.72	3.35	0.023	3 > 1
F	6.86	4.23	2.47	3.36	13.21	0.000	1 > (2,4,3)
K	14.62	15.08	17.63	17.72	2.07	—	
1 (Hs)	7.81	6.54	5.84	3.92	3.23	0.027	1 > 4
2 (D)	22.71	19.23	19.90	18.80	1.74	—	
3 (Hy)	21.76	22.39	22.58	21.36	0.19	—	
4 (Pd)	18.05	15.69	14.63	13.92	3.50	0.020	1 > 4
5 (Mf)	24.24	25.85	21.79	22.64	2.84	0.044	—
6 (Pa)	10.48	10.00	8.32	8.44	2.69	0.052	—
7 (Pt)	15.29	12.62	8.16	7.88	4.06	0.010	1 > 4
8 (Sc)	15.05	11.54	7.16	6.80	6.53	0.001	1 > (3, 4)
9 (Ma)	17.05	15.08	15.16	14.04	1.42	—	
0 (Si)	28.91	29.62	27.53	25.76	0.73	—	
MAC-R	22.24	22.54	21.11	21.04	0.65	—	
APS	23.81	22.54	20.00	21.96	4.09	0.010	1 > 3

Note: Multivariate F (S = 3, M = 5.5, N = 29) = 1.84, $p < 0.01$. Groups: 1 = chronic problem drinkers (n = 21); 2 = chronic heavy drinkers (n = 14); 3 = teetotalers (n = 22); 4 = nonproblem drinkers (n = 25). The validity scales can serve as personality trait indicators when the scores are below the level that invalidates the MMPI; specifically, L represents excessively positive self-presentation ("fake good"), F represents deviant response style ("fake bad"), and K represents defensiveness. The full names of the clinical scales are as follows: 1 (Hs) = Hypochondriasis; 2 (D) = Depression; 3 (Hy) = Hysteria; 4 (Pd) = Psychopathic Deviate; 5 (Mf) = Masculinity-Femininity; 6 (Pa) = Paranoia; 7 (Pt) = Psychasthenia; 8 (Sc) = Schizophrenia; 9 (Ma) = Mania; 0 (Si) = Social Introversion.

ter concerning the MAC scale. The same types of analyses have not yet been conducted with the APS.

RESULTS

We first selected a "purified" sample of those individuals who were totally consistent at all three time points. A multivariate analysis of variance (MANOVA) comparing MMPI-2 scale scores of chronic problem drinkers (n = 21), chronic heavy drinkers (n = 14), teetotalers (n = 22), and a comparative sample of light to moderate nonproblem drinkers (n = 25) was computed for the 1986 administration. Scheffé's post hoc range tests were used to identify which groups differed at the $p <$ 0.05 level. As shown in table 3, on most MMPI-2 scales only chronic problem drinkers showed significantly higher scores than nonproblem drinkers. The one exception to this pattern found teetotalers higher than chronic problem drinkers on scale L (excessively positive self-presentation). Chronic problem drinkers were significantly higher than nonproblem drinkers on scales 1 (Hs), 4 (Pd), and 7 (Pt) and were higher than teetotalers on the APS. They were higher than both nonproblem drinkers and teetotalers on scale 8 (Sc) and were higher than all three other groups on scale F (deviant response style). It should be noted that, although scale F is a validity scale, scores below those that invalidate the MMPI can be inter-

Table 4. Multivariate Analyses of Variance Examining 1991 MMPI-2 Scores by Alcohol Problem Group Scores: Purified Sample.

	Group				Univariate F		Scheffé's
Scale	1	2	3	4	(3, 74)	p	($p < 0.05$)
L	3.62	3.93	5.59	4.12	3.42	0.021	3 > 1
F	6.10	5.21	3.05	3.40	10.00	0.000	1 > (2,3,4)
K	14.71	15.29	18.46	18.04	3.47	0.020	1 < 4
1 (Hs)	7.24	8.07	6.64	5.20	1.45	—	
2 (D)	21.48	21.64	19.96	19.52	0.96	—	
3 (Hy)	21.81	22.79	23.59	21.76	0.60	—	
4 (Pd)	16.95	16.36	14.91	14.80	1.37	—	
5 (Mf)	23.81	24.71	22.46	22.12	1.54	—	
6 (Pa)	9.33	8.79	8.68	8.68	0.24	—	
7 (Pt)	13.33	13.64	7.91	8.60	3.54	0.018	1 > 4
8 (Sc)	13.38	11.79	8.27	7.64	4.58	0.005	1 > (3,4)
9 (Ma)	16.81	16.07	16.18	13.08	4.28	0.008	1 > 4
0 (Si)	26.57	30.21	25.73	25.04	1.17	—	
MAC-R	23.10	23.21	21.50	20.52	2.56	0.061	
APS	22.76	22.71	20.14	21.32	3.06	0.033	—

Note: Multivariate F (S = 3, M = 5.5, N = 31) = 1.68, $p < 0.01$. Groups: 1 = chronic problem drinkers (n = 21); 2 = chronic heavy drinkers (n = 14); 3 = teetotalers (n = 22); 4 = nonproblem drinkers (n = 25). For explanation of scales, see table 3.

preted as clinical scale scores, with elevations indicating antisocial tendencies (Graham 1994).

A MANOVA for the MMPI-2 administered in 1991 produced a similar pattern of results. Again, as shown in table 4, only chronic problem drinkers had significantly higher scale scores, with the exception of the higher scores of teetotalers on scale L. However, at this time, chronic problem drinkers were not significantly higher on scale 1 (Hs), scale 4 (Pd), or the APS. They were higher than nonproblem drinkers on scale K and scale 7 (Pt). Again, they were higher than both nonproblem drinkers and teetotalers on scale 8 (Sc) and higher than all three other groups on scale F. Scores for chronic heavy drinkers were much more similar to those of chronic problem drinkers than to those of both nonproblem groups at both MMPI-2 administrations, while the latter two groups' means were very similar to each other.

Only a relatively small proportion of individuals were highly consistent across time in chronic problem or heavy drinking. Therefore, we repeated these analyses for men reporting problem or heavy drinking at two or more time points (tables 5 and 6). The resulting larger sample sizes gave the analyses increased power. As can be seen in tables 5 and 6, the problem drinkers reported higher scores on nearly all of the scales indicating psychopathology, while the chronic heavy drinkers did not.

Table 5. Multivariate Analyses of Variance Examining Alcohol Problem Groups by 1986 MMPI-2 Scores: More Inclusive Sample.

	Group				Univariate *F*		Scheffé's
Scale	1	2	3	4	(3, 595)	*p*	($p < 0.05$)
L	3.55	4.53	5.74	4.85	10.00	0.000	(3,4) > 1
F	5.36	3.49	2.53	3.13	22.49	0.000	1 > (2,3,4)
K	14.61	17.93	18.35	17.72	12.76	0.000	(2,3,4) > 1
1 (Hs)	6.13	4.24	5.47	4.37	5.99	0.001	1 > 4
2 (D)	20.44	19.31	20.06	18.32	6.34	0.000	1 > 4
3 (Hy)	21.37	21.36	22.11	21.68	0.31	ns	—
4 (Pd)	17.04	15.44	13.62	14.18	14.02	0.000	1 > (3,4)
5 (Mf)	23.62	23.58	22.03	22.87	1.67	ns	—
6 (Pa)	9.33	9.00	8.09	8.66	2.37	0.069	—
7 (Pt)	12.98	8.71	7.74	7.08	23.07	0.000	1 > (2,3,4)
8 (Sc)	11.98	7.00	7.03	6.40	24.57	0.000	1 > (2,3,4)
9 (Ma)	16.01	13.60	14.94	14.42	4.83	0.003	1 > (2,4)
0 (Si)	28.61	26.84	28.18	23.99	10.00	0.000	(1,3) > 4
MAC-R	22.52	21.89	20.21	21.14	4.69	0.003	1 > (3,4)
APS	23.12	20.64	19.44	21.14	10.30	0.000	1 > (2,3,4)

Note: Multivariate F (S = 3, M = 5.5, N = 29) = 1.84, $p < 0.01$. Groups: 1 = chronic problem drinkers ($n = 84$); 2 = chronic heavy drinkers ($n = 45$); 3 = teetotalers ($n = 34$); 4 = nonproblem drinkers ($n = 436$). ns = not significant. For explanation of scales, see table 3.

A comparison of chronic problem drinkers with those reporting problems in 1973 only (nonchronic) is shown in table 7. This analysis found chronic problem drinkers with higher scores on scales F, 2 (D), 7 (Pt), and 8 (Sc). These differences are quite similar to those found between chronic and nonproblem drinkers. Statistical power was insufficient to produce a

Table 6. Multivariate Analyses of Variance Examining Alcohol Problem Groups by 1991 MMPI-2 Scores: More Inclusive Sample.

	Group						
Scale	1	2	3	4	Univariate F (3, 616)	p	Scheffé's ($p < 0.05$)
L	3.48	4.77	5.89	4.94	12.58	0.000	(2,3,4) > 1
F	5.34	3.55	2.97	3.32	20.34	0.000	1 > (2,3,4)
K	15.44	18.70	18.26	18.27	9.87	0.000	(2,3,4) > 1
1 (Hs)	6.77	5.55	6.05	5.01	5.28	0.001	1 > 4
2 (D)	21.14	19.77	20.24	18.82	7.16	0.000	1 > 4
3 (Hy)	22.16	22.43	22.95	22.18	0.60	ns	—
4 (Pd)	16.81	15.32	13.92	14.31	11.38	0.000	1 > (3,4)
5 (Mf)	23.18	23.18	22.34	22.91	0.47	ns	—
6 (Pa)	8.92	8.55	8.45	8.62	1.14	ns	—
7 (Pt)	12.37	8.64	8.00	7.16	18.82	0.000	1 > (2,3,4)
8 (Sc)	11.99	7.70	7.76	6.91	20.47	0.000	1 > (2,3,4)
9 (Ma)	14.94	13.93	15.32	14.35	1.40	ns	—
0 (Si)	27.77	26.59	26.87	23.78	7.47	0.000	1 > 4
MAC-R	21.58	22.09	20.84	21.27	2.61	0.050	—
APS	23.20	21.77	19.74	21.25	10.91	0.000	1 > (3,4)

Note: Multivariate F (S = 3, M = 5.5, N = 31) = 1.68, $p < 0.01$. Groups: 1 = chronic problem drinkers ($n = 50$); 2 = chronic heavy drinkers ($n = 44$); 3 = teetotalers ($n = 38$); 4 = nonproblem drinkers ($n = 455$). ns = not significant. For explanation of scales, see table 3.

Table 7. Significant Differences on 1986 MMPI-2 Scores Between Chronic and Nonchronic Problem Drinkers.

	Group			
Scale	1973 only ($n = 50$)	1973, 1982, 1991 ($n = 25$)	Univariate F (1, 73)	p
F	4.66	6.68	10.42	0.002
2 (D)	19.08	22.20	5.27	0.025
7 (Pt)	11.08	14.44	3.21	0.077
8 (Sc)	10.58	14.24	3.73	0.057

Note: Multivariate F (S = 1, M = 6.5, N = 28.5) = 1.14, $p = 0.342$. $n = 25$ for chronic problem drinkers in this analysis because chronic heavy drinkers were not excluded. For explanation of scales, see table 3.

Table 8. Significant Scale Differences on 1986 MMPI-2 Scales for Early- and Late-Onset Problem Groups.

	Group			
Scale	Early (n = 35)	Late (n = 20)	Univariate F (1, 53)	p
F	5.63	3.85	6.23	0.016
4 (Pd)	18.23	14.40	9.57	0.003

Note: Multivariate F (S = 1, M = 6.5, N = 18.5) = 2.00, p = 0.041. Mean age of early-onset group in 1991 = 54.2; mean age of late-onset group in 1991 = 65.4. For explanation of scales, see table 3.

significant multivariate F, but, with this caveat in mind, significant univariate F's were reported as suggestive.

Finally, respondents reporting problems earlier in life (defined as having reported a problem in 1973 with age under 40) were compared with those whose first reported problem was later (having first reported a problem in 1982 with age equal to or older than 40 in 1973). Early- and late-onset problem drinkers differed significantly on only two scales, F and 4 (Pd), the two MMPI scales most closely associated with antisocial behavior (table 8).

DISCUSSION

Although 21.1 percent of our sample reported a drinking problem (independent of reported alcohol consumption) at least once over the three assessment points, only 2.4 percent of the men reported problems at all three times. Only 13 or 14 of the three-time respondents (depending on the year of MMPI administration) reported heavy drinking at all three points, while 19 or 22 men who responded to all three surveys consistently reported that they were lifetime abstainers. Thus, as so often happens, a large research sample suddenly becomes a small one for certain specific questions. These data clearly indicate that moderate drinking is the norm, yet quite a few people have a drinking problem at some point in their lives. However, for a great majority of these people, such problems do not appear to be persistent.

Contrary to expectations, only chronic problem drinkers showed higher scores on MMPI-2 scales than nonproblem drinkers. In two cases (scales 7 and 8), the mean scores for chronic problem drinkers were twice as high as those of nonproblem drinkers and lifetime teetotalers. The one exception to this pattern found teetotalers significantly higher than chronic problem drinkers on scale L, which is a validity scale reflecting an unsophisticated attempt at favorable self-presentation (Graham 1994). Elevations on this scale at a level below that which would invalidate the MMPI suggest conventional moralism and rigidity. However, our teetotalers' mean score was not exceptionally high from a clinical point of view.

Chronic problem drinkers were significantly higher than nonproblem drinkers on scale 4 for the 1986 administration. It is common to find scale 4 elevations in problem drinkers (Graham and Strenger 1988). Since scale 4 is reputed to be a general measure of antisociality and contains items that directly reflect a history of alcohol abuse, this finding is not surprising. It is worthy of some note that chronic problem drinkers were not significantly higher on scale 4 in 1991.

The great differences between chronic problem drinkers and the nonproblem groups on scales 7 and 8 are of considerable interest. These differences suggest psychopathology characterized by depression, worry, tension, and feelings of inadequacy (Graham 1994). One point to bear in mind about MMPI clinical scales is that there is considerable item overlap among them. Studies examining individual items would be useful in this context. Unfortunately, with small sizes of the target groups, we lack the statistical power to make such analyses.

Vaillant (1983) suggested that personality disorders in alcoholics may be a result rather than a cause of alcohol abuse. We cannot test this hypothesis conclusively in our sample, since we do not have MMPI scores that antedate the onset of drinking problems. Chronic heavy drinkers who did not report problems had higher scores on scales 7 and 8 than did the teetotalers and the nonproblem drinkers, although, with one exception (scale 7 in 1991), not as high as the chronic problem drinkers. Perhaps the level of worry, depression, and sense of inadequacy reflect less adequate psychological defenses among those who report drinking problems than among those heavy drinkers who do not. Only a minority of self-reported problem drinkers in our sample were among the heaviest drinkers. Of course, the problem drinkers may have misrepresented their levels of alcohol consumption. However, it seems somewhat implausible that they would have done so while still reporting problems. In any case, the higher scale 4 scores among chronic problem drinkers suggest a more complex picture in which personality traits may be risk factors for problem drinking, but, when drinking persists, other symptoms (e.g., depression) may come to predominate.

The suggestion that scale 4 elevations may reflect risk for alcohol abuse in younger people is strengthened by a comparison of MMPI scale scores of respondents reporting drinking problems of relatively early onset (problems reported in 1973 with age under 40) with those of respondents with late-onset problems (problems beginning in 1982 with age equal to or older than 40 in 1973). Early-onset problem drinkers had significantly higher scores on scales F and 4. This finding supported our hypothesis of greater antisociality among early-onset problem drinkers, since these are the two scales in our analysis which are most closely associated with antisocial tendencies.

It should be noted that one item in scale 4 is circular with respect to problem drinking: "I have used alcohol excessively." More significantly, many of the items in scales 4 and F reflect negative affectivity. Actually, scale 4

has 14 items in common with scale 7, scale 8, or both.

A comparison of respondents reporting drinking problems in 1973 only with those reporting problems at all three assessments found that the chronic problem drinkers had significantly higher scores on scales F and 2, while their scores on scales 7 and 8 approached significance (0.08 and 0.06, respectively). As hypothesized, the general pattern of differences between the chronic and nonchronic problem drinkers was similar to that between chronic problem and nonproblem drinkers.

There are several limitations to this study. The first is the relatively small numbers of men reporting chronic heavy or chronic problem drinking. Obviously this is a low base rate problem. However, most studies of this type are conducted on clinical samples, and thus it is important to investigate psychological correlates of the "natural history" of alcohol problems in non–treatment-seeking populations (only a handful of our men have reported seeking treatment).

Another limitation has to do with the problem of response bias. Given that the chronic problem drinkers reported elevations on nearly all of the MMPI scales in the expanded sample, one could interpret these findings as indicating that some individuals simply "fake bad" (try to present themselves in a negative way) and thus report more problem drinking and more psychopathology. However, it should be noted that individuals were excluded from the study whose MMPI F scale scores were high enough to invalidate the MMPI; the questionnaires should be considered valid by standard criteria. In addition, the variability in overall problem drinking status argues that respondents are not just "faking bad." If they were, then one would reasonably conclude that they should have reported problem drinking at all three times, but only 10 percent of the problem drinking group did so. Instead, it would appear that men, especially those who score high on psychasthenia and schizophrenia scales, are more likely to get into trouble with alcohol when they do drink.

Finally, the sample is limited by its inclusion primarily of white men and so may not necessarily be generalizable to either women or minorities.

The findings reported in this chapter are suggestive, but a more thorough understanding of the psychological antecedents of alcohol abuse and the psychological consequences of such abuse requires more than psychometrically assessed personality traits. It may be that the overarching construct of negative affectivity reflected in several MMPI scales is a predictor of problem drinking and is exacerbated by chronic problem drinking. Further research is required to reveal the sources of negative affectivity. In this connection, deeper knowledge of the life courses, significant life events and influences, occupational and other stresses, and cognitive styles of problem drinkers is needed.

ACKNOWLEDGMENTS

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REFERENCES

Atkinson, R.L. Alcohol problems of the elderly. *Alcohol Alcohol* 22:415–417, 1987.

Blackwelder, W.C.; Yano, K.; Rhoads, G.G.; Kagan, A.; Gordon, T.; and Palesch, Y. Alcohol and mortality: The Honolulu heart study. *Am J Med* 68:164–169, 1980.

Bossé, R.; Ekerdt, D.; and Silbert, J.E. The Veterans Administration Normative Aging Study. In: Mednick, S.A.; Harway, M.; and Finello, K.M., eds. *Handbook of Longitudinal Research.* Vol. 2. New York: Praeger, 1984. pp. 273–289.

Butcher, J.N.; Dahlstrom, W.G.; Graham, J.R.; Tellegen, A.; and Kaemmer, B. *Minnesota Multiphasic Personality Inventory (MMPI-2). Manual for Administration and Scoring.* Minneapolis: University of Minnesota Press, 1989.

Cahalan, D.; Cisin, I.H.; and Crossley, H.M. *American Drinking Practices.* New Brunswick, NJ: Rutgers Center for Alcohol Studies, 1969.

De Labry, L.O.; Glynn, R.J.; Levenson, M.R.; Hermos, J.A.; LoCastro, J.S.; and Vokonas, P.S. Alcohol consumption and mortality in an American male population: Recovering the U-shaped curve: Findings from the Normative Aging Study. *J Stud Alcohol* 53:25–32, 1992.

Fillmore, K.M. *Alcohol Use Across the Life Course: A Critical Review of 70 years of International Longitudinal Research.* Toronto: Addiction Research Foundation, 1988.

Fillmore, K.M.; Hartka, E.; Johnstone, B.M.; Leino, E.V.; Motoyoshi, M.; and Temple, M.T. A meta-analysis of life course variation in drinking. *Br J Addict* 86:1221–1267, 1991.

Glynn, R.J.; Bouchard, G.R.; LoCastro, J.S.; and Laird, N.M. Aging and generational effects on drinking behaviors in men: Results from the Normative Aging Study. *Am J Pub Health* 75:1413–1419, 1985.

Graham, J.R. MMPI-2: *Assessing Personality and Psychopathology.* New York: Oxford University Press, 1994.

Graham, J.R., and Strenger, V.E. MMPI characteristics of alcoholics: A review. *J Consult Clin Psychol* 56:197–205, 1988.

Klatsky, A.L.; Friedman, G.D.; and Siegelaub, A.B. Alcohol and mortality: A 10-year Kaiser-Permanente experience. *Ann Intern Med* 95:139–145, 1981.

Levenson, M.R.; Aldwin, C.M.; Butcher, J.N.; De Labry, L.; Workman-Daniels, K.; and Bossé, R. The MAC scale in a normal population: The meaning of "false positives." *J Stud Alcohol* 51:457–462, 1990.

Levenson, M.R.; Aldwin, C.M.; and Spiro, A., III. Age, cohort and period effects on alcohol consumption and problem drinking: Findings from the Normative Aging Study. *J Stud Alcohol,* in press.

Liberto, J.G.; Oslin, D.W.; and Ruskin, P.E. Alcoholism in older persons: A review of the literature. *Hosp Community Psychiatry* 43:975–984, 1992.

MacAndrew, C. The differentiation of male alcoholic outpatients from nonalcoholic psychiatric outpatients by means of the MMPI. *Q J Stud Alcohol* 26:238–246, 1965.

MacAndrew, C. Similarities in the self-depictions of men alcoholics and psychiatric outpatients: An examination of Eysenck's

dimension of emotionality. *J Stud Alcohol* 42:421–431, 1981.

Midanik, L. Validity of self-reported alcohol use: A literature review and assessment. *Br J Addict* 83:1019–1029, 1988.

Neve, R.J.M.; Diederiks, J.P.M.; Knibbe, R.A.; and Drop, M.A. Developments in drinking behavior in the Netherlands from 1958 to 1989, a cohort analysis. *Addiction* 88:611–621, 1993.

Schuckit, M.A.; Tipp, J.E.; Smith, T.L.; Shapiro, E.; Hesselbrock, V.M.; Buchholz, K.; Reich, T.; and Nurnberger, J.I., Jr. An evaluation of Type A and B alcoholics. *Addiction* 90:1189–1203, 1995.

Shedler, J., and Block, J. Adolescent drug use and psychological health: A longitudinal inquiry. *Am Psychol* 45:612–630, 1990.

Vaillant, G.E. *The Natural History of Alcoholism*. Cambridge, MA: Harvard University Press, 1983.

Varma, V.K.; Basau, D.; Malhotra, A.; Sharma, A.; and Matoo, S.K. Correlates of early- and late-onset alcohol dependence. *Addict Behav* 19:609–619, 1994.

Weed, N.C.; Butcher, J.N.; McKenna, T.; and Ben-Porath, Y.S. New measures for assessing alcohol and drug abuse with the MMPI-2: The APS and AAS. *J Pers Assess* 58:389–404, 1992.

Williams, M. Alcohol and the elderly. *Alcohol Health Res World* 8:3–9, 1984.

Chapter 15

Life Context Factors, Treatment, and Late-Life Drinking Behavior

Rudolf Moos, Ph.D., Penny Brennan, Ph.D.,
and Kathleen Schutte, Ph.D.

More than a decade ago, we reviewed existing research on the prevalence of late-life excessive drinking and highlighted the need to develop better measures of problem drinking among older adults (Finney and Moos 1984; Moos and Finney 1984). Although at that time there was considerable speculation linking life stressors to the onset and continuation of late-life drinking problems, evidence in support of this idea was sparse. In addition, researchers had not examined potential mediators or moderators of the relationship between stressors and problem drinking, such as prior drinking history, social resources, and coping skills. To facilitate the development of more dependable knowledge in this area, we described a conceptual framework to guide research on late-life problem drinking and alcohol dependence.

In this chapter, we describe the stress and coping model that has guided our work on late-life problem drinking. After focusing on the role of stress and coping in problem drinking among older adults, we introduce some new measures to assess the key constructs in this area: late-life problem drinking, life stressors and social resources, and coping responses. We then describe our program of longitudinal research on late-life problem drinking, including the rates and predictors of remission, risk factors for continued problem drinking, comparisons of early- and late-onset problem drinkers, gender differences in problem drinkers' life contexts and functioning, and treatment-seeking

R. Moos, Ph.D., P. Brennan, Ph.D., and K. Schutte, Ph.D., are health science specialists with the Program Evaluation and Resource Center and the Health Services Research and Development Center for Health Care Evaluation, Department of Veterans Affairs Health Care System, 3801 Miranda Ave., Palo Alto, CA 94304, and with Stanford University Medical Center in Palo Alto.

predictors. Finally, with respect to the course of late-life alcohol dependence, we cover the role of treatment, the use of health services and predictors of readmission, and the characteristics of effective treatment programs for late-life alcohol dependent persons.

THE ROLE OF STRESS AND COPING IN LATE-LIFE PROBLEM DRINKING

The stress and coping perspective that guides our work on late-life problem drinking is shown in figure 1. We specify that the current life context (panel I) consists of ongoing stressors and social resources in several life domains. Personal factors (panel II) include a person's demographic and diagnostic characteristics, baseline drinking-related and psychosocial functioning, and recent and lifetime drinking history. Panel III includes negative life events, such as age-related loss events (e.g., retirement and widowhood) and other acute stressors. The model posits that these sets of factors influence how people cope with and seek help (panel IV) to alleviate stressful life circumstances.

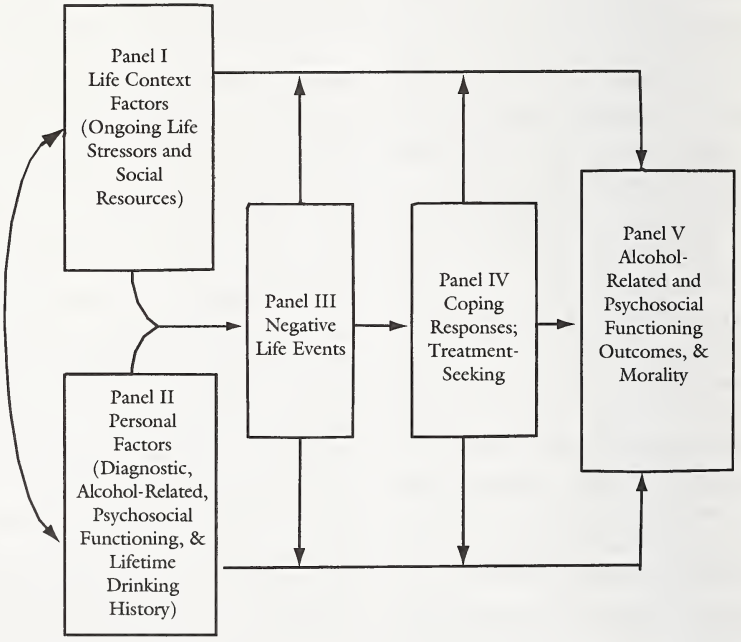

Figure 1. A general stress and coping model of late-life problem drinking.

All four sets of factors affect a person's adaptational outcomes, such as substance use and psychosocial functioning (panel V). Although not shown in the figure, we assume that adaptational outcomes can subsequently influence each of the other sets of factors in a reciprocal, longitudinal process (Brennan and Moos 1995).

Life Stressors

The idea that stressful life circumstances trigger late-life problem drinking has intuitive appeal, but has received inconsistent empirical support (Krause 1995; Welte and Mirand 1995). One reason for the inconsistent findings may be some researchers' reliance on measures of acute life events; in fact, chronic stressors have a stronger influence than acute life events on health outcomes. In addition, some types of stressors elicit alcohol consumption, whereas other stressors, such as financial difficulties or new health problems, may dampen it (Krause 1991; Glass et al. 1995). Moreover, personal risk factors, such as male gender and maladaptive coping responses, moderate the connection between stressors and drinking behavior (Cooper et al. 1992; Peirce et al. 1994).

Social Resources and Coping Responses

Social resources tend to enhance older adults' health outcomes, but there has been little examination of the role of social resources in late-life problem drinking. In a cross-sectional study, Jennison (1992) showed that family and friendship support moderated the association between life stressors and alcohol use. High family support also promotes stable remission in mixed-aged samples of treated alcoholics (Moos et al. 1990). Family involvement in treatment, an indicator of support, predicts better treatment compliance and outcomes (Atkinson et al. 1993).

Consistent with the idea that social relationships can be powerful motivators of behavior, heightened social support for problem drinking may encourage heavier alcohol consumption. In this regard, Alexander and Duff (1988) showed that older adults who were more active socially in residential communities also drank more heavily. As with stressor effects, social influences may be altered by personal risk factors, such as past history of drinking and maladaptive coping responses.

An important link exists between coping responses and excessive alcohol use among mixed-aged groups. Although researchers have begun to examine the structure and outcomes of coping among older adults (e.g., Zautra and Wrabetz 1991; Aldwin et al. 1996), we still know relatively little about maladaptive coping as a risk factor for late-life alcohol abuse.

MEASUREMENT DEVELOPMENT

To examine the role of stress and coping in late-life problem drinking, we first developed measures of the key constructs we sought to study.

The Drinking Problems Index

Current drinking problems were assessed by the Drinking Problems Index (DPI), which includes items

that reflect physical dependence, such as craving for a drink and skipping meals because of drinking, as well as items that assess negative consequences of drinking, such as social isolation, neglecting the appearance of one's self and/or living quarters, and sustaining a fall or being involved in an accident. The DPI has high internal consistency reliability, is relatively stable over time, and shows good construct validity in that people who have more drinking problems experience more depression, have less self-confidence, and engage in fewer social activities (Finney et al. 1991).

The Life Stressors and Social Resources Inventory

The Life Stressors and Social Resources Inventory (LISRES) measures chronic stressors in each of eight life domains—(1) physical health, (2) home and neighborhood, (3) finances, (4) work, (5) spouse or partner, (6) children, (7) extended family, and (8) friends and social groups—and the ongoing resources in the last six of them. The LISRES also includes indices of new life events in the past year. The LISRES subscales have moderate to high internal consistencies (average alpha = 0.80), are moderately intercorrelated (average $r = 0.20$), and are relatively stable over 1 year (average $r = 0.68$) and 4 years (average $r = 0.59$). The LISRES indices have reasonable construct validity; they are associated concurrently and predictively with alcohol-related and other functioning outcomes, help seeking and treatment entry, and both participation in and outcome of mutual help groups (Moos and Moos 1994).

The Coping Responses Inventory

The Coping Responses Inventory (CRI) measures four sets of approach coping responses and four sets of avoidance coping responses. Two of the four sets of approach responses reflect cognitive coping (i.e., logical analysis and positive reappraisal) and the other two sets of approach responses reflect behavioral coping (i.e., seeking guidance and support and engaging in problem solving. The four sets of avoidance coping responses are also divided into cognitive coping (i.e., cognitive avoidance and resigned acceptance) and behavioral coping (i.e., seeking alternative rewards and emotional discharge). These indices have moderate internal consistencies (average alpha = 0.66) and intercorrelations (average $r = 0.27$) and are moderately stable over 1 year (average $r = 0.45$) and 4 years (average $r = 0.38$). In general, the CRI subscales are associated in expected ways with functioning outcomes among alcoholic and depressed patients, problem and nonproblem drinkers, and normal persons and their spouses (Moos 1993).

THE COURSE OF LATE-LIFE PROBLEM DRINKING

We are using our conceptual framework and these measures in a longitudinal study of the forces that elicit and perpetuate problem drinking, as

well as those forces that promote remission, among late-middle-aged and older adults. Specifically, we conducted a baseline assessment (N = 1,884) and both 1-year and 4-year followups of a community sample of late-life problem and nonproblem drinkers. At baseline, respondents were 61 years old on average; approximately 40 percent were women and approximately two-thirds were married. About 70 percent had completed high school. The problem drinkers in the sample consumed an average of 5.2 oz of alcohol on a heavy drinking day and had an average of 3.9 drinking problems as assessed by the DPI.

At 1-year followup, we followed 95 percent of the respondents who were still living (1,755 of 1,838; 46 of the original 1,884 had died). At 4-year followup, we found that 169 of the original 1,884 respondents had died. Of the remaining 1,715 participants, we obtained complete followup information from 1,620, or 94.5 percent. Both the baseline and followup assessments included measures of alcohol consumption and drinking problems, smoking and symptoms attributable to smoking, use of prescription and nonprescription medications, social activities with family and friends, religious and social group memberships, and indices of personal functioning and help seeking. We also used the LISRES and the CRI to obtain information about participants' life context and coping responses (for more details, see Brennan and Moos 1995).

We have used the baseline and followup data to address a number of issues: (1) What is the rate of remission among problem drinkers and what factors at baseline predict remission 1 year and 4 years later? (2) What are the personal and environmental risk factors for continued problem drinking? (3) How do early- and late-onset problem drinkers differ? (4) How do female and male problem drinkers differ? (5) What personal and environmental factors motivate problem drinkers to seek treatment?

Remission and Predictors of Remission

At 1 year, 29 percent of the individuals who were problem drinkers at baseline were remitted—that is, they had no drinking problems in the 12 months before the 1-year followup. To identify precursors of 1-year remission, we compared the remitted individuals with nonremitted problem drinkers (i.e., problem drinkers who continued to have drinking problems at followup) and with consistent nonproblem drinkers. At baseline, to-be-remitted problem drinkers—compared with those individuals who would continue to experience drinking problems—consumed less alcohol, reported fewer drinking problems, had friends who approved less of drinking, and were more likely to have sought help from a mental health practitioner. They also reported less spousal social support. Thus, less severe drinking problems, active help seeking, having friends who view heavy drinking less favorably, and apparent spousal pressure

for change all foreshadowed short-term remission (Moos et al. 1991).

At the 4-year followup, 21 percent of the baseline problem drinkers were stably remitted—that is, they reported no drinking problems at either the 1- or 4-year followup. At baseline, the drinkers who would be remitted at 4 years consumed less alcohol and reported fewer drinking problems but were more depressed and less self-confident than were the individuals who would continue to have drinking problems. These findings support the idea that psychosocial distress motivates people to change and predicts better long-term outcomes. The to-be-remitted drinkers also had less spousal support and fewer friends who approved of drinking and were more likely to have sought help from a mental health professional (Schutte et al. 1994). In general, these results replicate our findings at 1-year followup and strengthen our conclusion that pressure to change from one's spouse and friends and active help seeking foreshadow remission.

Risk Factors for Continued Problem Drinking

We also identified personal and environmental risk factors that predicted poorer 1-year and 4-year outcomes among late-life problem drinkers. Risk factors associated with more alcohol consumption and drinking problems included male gender, early onset, more prior alcohol consumption and drinking problems, and more friends who approved of drinking. Health-related negative events, such as a newly diagnosed medical condition, predicted less alcohol consumption. Similarly, several longitudinal studies have shown that health-related stressors predict reduced alcohol consumption over intervals that range from 1 to 10 years (e.g., Hermos et al. 1984; Glass et al. 1995). Consistent with most cross-sectional studies, both the 1- and 4-year followups showed no link between the number of non-health-related negative life events (e.g., retirement) and subsequent alcohol consumption (Brennan et al. 1994; Brennan and Moos 1996*b*).

Drinking problems are more readily affected by life stressors than is alcohol consumption. Problem drinkers who experienced more health-related stressors were more likely to be in remission from drinking problems at 1- and 4-year followups (Moos et al. 1991; Schutte et al. 1994). Other types of stressors, however, seem to elicit increased drinking problems in later life. For example, people who reported more non-health-related negative life events at baseline were at heightened risk for increased drinking problems 1 year later (Brennan et al. 1994). Heightened spouse and friend stressors at baseline foreshadowed more drinking problems 4 years later (Brennan and Moos 1996*b*). Similarly, in a followup of participants in the Normative Aging Study, Ekerdt and colleagues (1989) found that compared with men who had not retired, retirees were three times more likely to report new alcohol problems.

Personal risk factors altered the influence of some life context factors on outcomes. For example, among lighter drinkers, chronic health stressors were

associated with a decline in alcohol consumption; among heavier drinkers, chronic health stressors were linked to an increase in alcohol consumption (see figure 2). This suggests that health stressors encourage people with less severe drinking problems to curtail their drinking, but may elicit heavier, perhaps self-medicating, drinking among people with more severe alcohol problems.

Other longitudinal studies support the idea that drinking history moderates the relationship between life context and drinking behavior. For example, Glass and associates (1995) showed that initial heavier alcohol consumption increased the likelihood that certain stressful life events, such as a relative's recent sickness or injury, would cause older drinkers to subsequently increase or show a smaller-than-expected decrease in their alcohol consumption.

Avoidance coping also appears to moderate the effect of life context on late-life drinking behavior. For instance, at 1-year followup, friends' approval of drinking had little influence on alcohol consumption among problem drinkers who used few avoidance coping strategies to manage

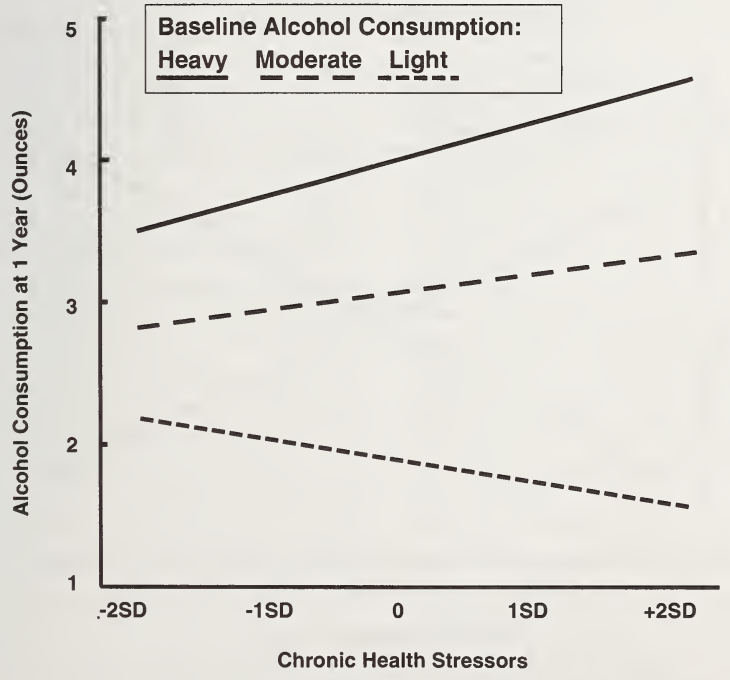

Figure 2. Effect of baseline alcohol consumption and chronic health stressors on alcohol consumption 1 year later.

life stressors. Among individuals who relied more heavily on avoidance coping, however, friends' approval of drinking was associated with elevated alcohol consumption (Brennan et al. 1994). Among those who relied heavily on avoidance coping, more non-health-related negative events at baseline predicted elevated drinking problems at the 4-year followup (see figure 3). And finally, among individuals who relied less on avoidance coping, more non-health-related negative events predicted a decline in drinking problems (Brennan and Moos 1996*b*).

Differences Between Early- and Late-Onset Problem Drinkers

About 30 percent of older problem drinkers are late-onset problem drinkers, in that they first experienced alcohol-related difficulties after age 50. In contrast, early-onset problem drinkers experience continual or intermittent drinking problems throughout their adult life. In our sample, the baseline prevalence of late-onset problem drinkers was 33 percent.

Consistent with distinctions between type I and type II alcoholics (Cloninger 1987) and the closely

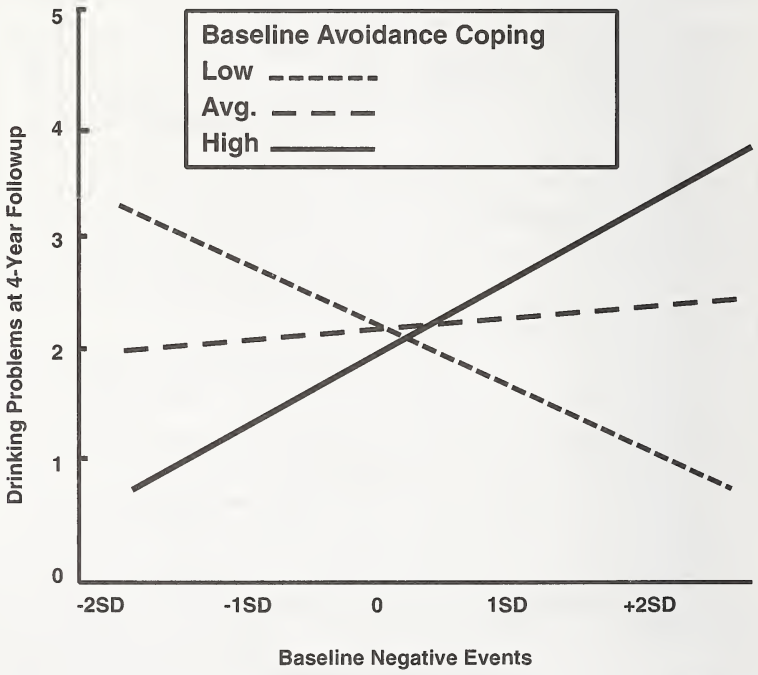

Figure 3. Effect of baseline negative life events and reliance on avoidance coping on drinking 4 years later.

aligned type A–type B distinction (Babor et al. 1992), our early-onset problem drinkers reported heavier alcohol consumption and more drinking problems than the late-onset problem drinkers, and more early- than late-onset problem drinkers reported symptoms of dependence (Brennan and Moos 1991). Other studies have shown that late-onset problem drinkers are in better health, have better psychosocial functioning, and are less likely to have been in treatment for substance abuse than early-onset problem drinkers (Schonfeld and Dupree 1991; Atkinson 1995; Liberto and Oslin 1995).

In addition, time of onset of drinking problems influenced the short-term process of remission: Compared with early-onset problem drinkers, late-onset problem drinkers were more likely to remit over the 1-year interval (i.e., 41 percent vs. 24 percent). Late-onset problem drinkers reduced their drinking in response to chronic health stressors and health-related negative events, friends' disapproval of drinking, and lack of spousal support. In contrast, help seeking and participation in treatment for emotional and/or drinking problems were associated with remission among early-onset drinkers. These findings imply that people with less enduring alcohol problems are more reactive to health-related stressors and informal social influences, whereas people with more enduring problems may require more formal sources of help (Moos et al. 1991).

At the 4-year followup, late-onset problem drinkers were more likely than early-onset problem drinkers to be stably remitted (i.e., 31 percent vs. 16 percent). Fewer financial resources, less approval from friends for drinking, and less spousal support at baseline predicted stable remission among late-onset problem drinkers. In contrast, among early-onset problem drinkers, help seeking and participation in treatment for emotional and/or drinking problems in the year before baseline predicted better outcomes. These results reinforce the idea that health-related stressors and informal social influences contribute more strongly to remission among people who experience fewer chronic alcohol problems, whereas formal sources of help may contribute more to remission among people who experience more persistent alcohol problems (Schutte et al. 1994).

Gender Differences in Problem Drinkers' Life Contexts and Functioning

Although an increasing amount of research literature shows that problem-drinking women differ from their male counterparts, little is known about gender differences in late-life problem drinkers' functioning and life contexts. We therefore compared problem-drinking women and men at baseline and at 1-year followup. Compared with men, women at baseline consumed less alcohol, had fewer drinking problems, and were more likely to have had a recent onset of drinking problems. However, women were more depressed and used more psychoactive medications.

Over the 1-year interval, problem-drinking women experienced a decline in spousal stressors, such as conflict and

criticism, and men with ongoing drinking problems experienced a reduction in conflicts with friends. Although men with ongoing drinking problems lost support from their children, the social resources of women with ongoing drinking problems remained relatively stable (Brennan et al. 1993). Thus, contrary to expectation, ongoing drinking problems did not adversely affect these older women's life contexts. In fact, their use of alcohol may have had the short-term "benefit" of reducing interpersonal conflict and facilitating family functioning (Steinglass et al. 1987).

Remission from drinking problems should help improve the life contexts of women and men. In fact, remission had little influence on men's life contexts; women who remitted experienced a loss of support from extended family members over a 1-year interval and, at followup, reported more family stressors than did remitted men. Thus, for men, remission may portend a slow process of improvement in life context, whereas for women, it may entail costly changes in family context.

Older adults' drinking behavior also affected their psychological well-being in an unexpected way. That is, among women, heavier initial alcohol consumption predicted fewer subsequent depressive symptoms, suggesting that women may use alcohol to alleviate depression. Among men, more depression at baseline predicted less alcohol consumption 1 year later. In addition, more drinking problems at 1 year predicted reduced depression 3 years later. These results support the idea that, among men, psychological distress may foreshadow efforts to reduce alcohol consumption and thus may be associated with improved psychological functioning (Schutte et al. 1995).

Together, these results suggest that in the short term, older adults' drinking behavior may have unexpected effects on subsequent life context and psychological well-being. Longer term followups are needed to determine the stability of these effects and their consequences. Do the short-term reductions in interpersonal stressors found here give way over time to more stressful life contexts? Do adverse family contexts or self-medication to avoid depression pose a risk for older, remitted women? The answers might have treatment implications, such as the need for extra support for women in early remission to cope with family conflict and depression.

Predictors of Seeking Treatment

Older adults make relatively little use of formal help to enhance their emotional well-being. Consistent with this finding, very few problem drinkers sought help for their alcohol-related problems. At initial assessment, approximately 4 percent of late-onset problem drinkers and 12 percent of early-onset problem drinkers had sought help in the past year specifically for drinking problems. However, about 25 percent of the problem drinkers sought help for a personal or emotional problem from a mental health professional or spiritual advisor (Brennan and Moos 1991).

Among these community residents, prior treatment seeking, heightened

health-related and spousal stressors, more non–health-related negative life events, and friends' disapproval of drinking at baseline foreshadowed more treatment seeking 4 years later (Brennan and Moos 1996b). More severe drinking problems and depression as well as fewer social resources were also associated with seeking treatment (Brennan and Moos 1991). These results are consistent with research on treatment entry among younger alcoholics, which shows that stressful life circumstances, heightened need or distress, and more prior treatment trigger formal service use (Finney and Moos 1995). These findings imply a need to engage older problem drinkers in treatment before drinking-related problems generate intolerable health crises and living situations.

Conclusions About Late-Life Problem Drinking

The longitudinal research in this area suggests that the way in which life context influences late-life drinking depends on personal risk factors, such as the chronicity and severity of alcohol-related problems, the specific domains in which stressors occur (especially health vs. interpersonal stressors), and the type of drinking behavior being assessed (i.e., alcohol consumption vs. drinking problems). In general, health-related stressors are associated with less subsequent alcohol consumption and fewer drinking problems, whereas stressors in relationships with spouse and friends can foreshadow more subsequent drinking problems. Problem drinkers who consume more alcohol, however, may increase their alcohol intake when confronted by chronic health stressors. Among problem drinkers who rely more on avoidance coping, negative life events are associated with more drinking problems.

With respect to the chronicity of drinking problems, fewer resources from one's spouse and/or friends, which may reflect disapproval of drinking, predict remission among late-onset problem drinkers. Seeking help for either drinking or emotional problems is a more robust predictor of remission among early-onset problem drinkers. Thus, acute and chronic health-related stressors, as well as stressors arising from relationships with one's spouse and friends, may enhance remission by increasing the likelihood that a problem drinker will seek treatment.

THE ROLE OF TREATMENT IN LATE-LIFE ALCOHOL DEPENDENCE

Although we believe that the stress and coping process is conceptually similar in both treated and community populations, we employ a somewhat different model to focus on treated problem drinkers in order to highlight the associations between patients' personal characteristics and life context before treatment, the characteristics of treatment, and treatment outcome (Brennan and Moos 1996*a*).

Conceptual Framework

The model shown in figure 4 specifies that the treatment outcome (panel V) is influenced by patients' characteristics at intake, including demographic

and diagnostic factors, and the severity and chronicity of substance abuse and other aspects of functioning (panel II). Treatment outcome is also influenced by life context factors before intake (i.e., at baseline) (panel I) and those that occur during the treatment and posttreatment interval (panel IV), as well as by patients' treatment experiences (panel III). In addition, the model depicts both personal and life context factors as determinants of entry into treatment and of the amount and type of treatment required. This model encourages the careful study of treatment, including an examination of the associations between specific treatment components and outcome. It also explicitly considers factors outside of treatment, such as a person's life stressors and social resources and how they influence treatment entry, treatment experiences, and treatment outcome (Moos et al. 1990).

Treatment Seeking and Treatment Outcome

It still is not clearly understood why relatively few late-life problem drinkers seek help specifically for alcohol abuse. The two most popular frameworks for explaining health services use—the health beliefs model (Becker et al. 1977) and Andersen's (1995) schema of predisposing, enabling, and need characteristics—have seldom been extended to describe help seeking specifically for drinking problems. Moreover, these frameworks could be strengthened by considering how changes in life contexts precede help seeking (Weisner 1993) and how personal characteristics, such as the severity of alcohol dependence, combine with contextual factors to predict help seeking (Hasin 1994).

We also know relatively little about treatment outcomes among older alcohol-dependent patients. Many older patients improve after treatment, but there are high relapse and readmission rates (Atkinson 1995; Schonfeld and Dupree 1995). Some findings indicate that remission may be fostered by specialized treatment for elderly clients, nonconfrontational counseling oriented toward harm reduction, and spouse involvement in treatment (Kofoed et al. 1987; Atkinson et al. 1993; Graham et al. 1994). More definitive information is needed about the role of comorbidity and the time of onset in predicting outcome, how specific program characteristics affect outcome, and whether outpatient mental health care facilitates better psychological functioning and prevents relapse among older alcohol patients.

THE COURSE OF TREATED LATE-LIFE ALCOHOLISM

To complement our work on the community sample of late-life problem drinkers, and because of the lack of information about treated late-life alcohol dependence, we identified a nationwide cohort of more than 22,600 substance abuse patients in Department of Veterans Affairs (VA) Medical Centers who were age 55 or older. Almost 99 percent of these older patients were men; approximately 80 percent were Caucasian and 12 percent were African

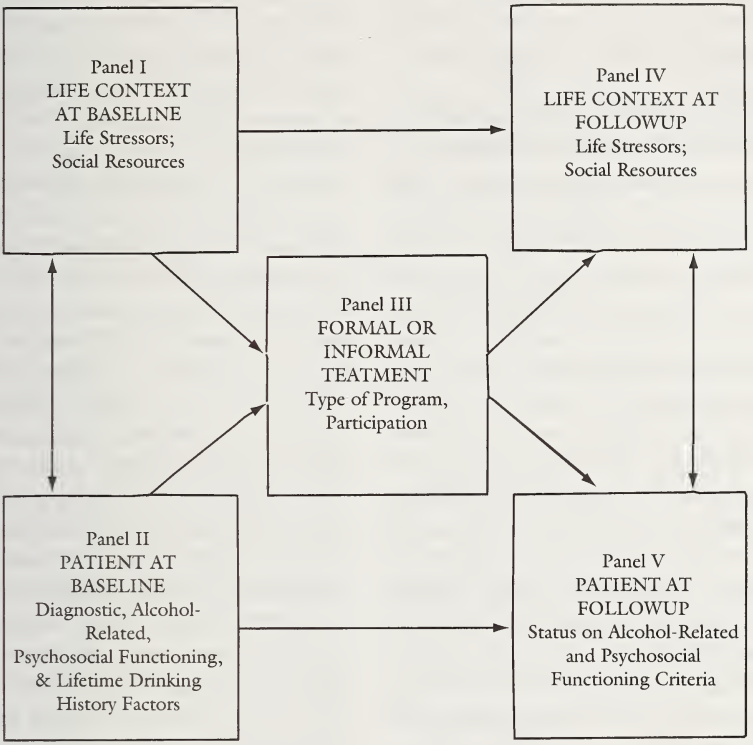

Figure 4. A general model of personal, contextual, and treatment factors that affect the course of treated late-life alcoholism.

American. On average, the patients were 62 years old, and approximately 35 percent were married. More than 95 percent of them had been diagnosed with alcohol dependence and/or alcohol psychosis. Almost 30 percent had a concurrent psychiatric disorder (Moos et al. 1993).

Both diagnostic and treatment information about these patients were obtained from nationwide VA inpatient and outpatient databases. These databases contained information on some of the variables in panels II, III, and V of the model shown in figure 4. For a separate sample of more than 5,600 older patients, we obtained information about the treatment programs in which they participated (Moos et al. 1995).

Our work with these patients addressed two sets of questions: (1) What is the extent of older substance abuse patients' use of health services and what factors predict heavier service use in this group? (2) To what

extent is substance abuse treatment effective for this group? What specific program characteristics predict better outcomes for these older patients?

Use of Health Services and Predictors of Readmission

Older substance abuse patients made heavy use of health care services. In the 4 years before their index episode of care, these patients received more than 920,000 days of inpatient care for a substance abuse or psychiatric disorder; in the 4 years following, they received 1.2 million days of care (Moos et al. 1994*c*). Readmission rates in this group were quite high. For example, the 1-year readmission rate for older patients with only a substance abuse diagnosis at baseline was 37 percent; the 4-year rate was 57 percent (Moos et al. 1994*a*, 1994*c*).

The treatment of older patients was complicated by the chronicity of their alcohol-related problems. Readmission rates were higher among more chronic substance abuse patients than in the sample overall (Moos et al. 1994*c*). This is consistent with our finding that remission is less likely to occur among early-onset than among late-onset problem drinkers (see also Atkinson 1995; Schonfeld and Dupree 1995).

Psychiatric disorders also complicated the treatment of these patients. At the index episode of care, almost 30 percent of the older patients had a concomitant psychiatric disorder, most frequently depressive disorders, personality disorders, or schizophrenia (Moos et al. 1993). Patients who had a dual diagnosis used more health care resources. For example, during the 4 years after the index episode, 57 percent of patients with only a substance abuse diagnosis had a subsequent episode of inpatient care, whereas more than 70 percent of patients with a concomitant psychiatric diagnosis did so.

Compared with younger substance abuse patients, older patients received less specialized substance abuse and psychiatric care and more short-term care, such as detoxification, that focused primarily on their medical needs (Moos et al. 1993). Moreover, despite the complexity and chronicity of their substance abuse problems, less than 25 percent of older patients received mental health aftercare; they were less likely to receive this care than were younger patients (Moos et al. 1995).

At both the 1- and 4-year followups, being unmarried predicted higher readmission, as did more prior service use, more severe and complex psychiatric diagnoses, and disruption of treatment as reflected by a shorter length of stay (Moos et al. 1993, 1994a). Prompt mental health aftercare, such as individual or group counseling following discharge, reduced the likelihood of readmission (Moos et al. 1995). Moreover, intensive mental health aftercare and remission of substance abuse seemed to reduce premature mortality, even among these older patients with long-standing substance abuse problems (Moos et al. 1994b).

Characteristics of Effective Treatment Programs

The high readmission rates among late-life alcoholic patients raise impor-

tant questions: What aspects of treatment programs are associated with better outcomes for older alcoholic patients? Do certain program characteristics differentially affect older compared with younger patients? To address these issues, which focus on the connections between panels II, III, and V in the model shown in figure 4, we compared older alcoholic patients with middle-aged and younger patients before, during, and after an episode of treatment in one of 88 substance abuse programs (Moos et al. 1995).

Among older patients, more structured program policies, more acceptance of problem behavior, and more comprehensive patient assessment were associated with lower readmission rates. More intensive treatment, as reflected in more individual and group counseling sessions, was associated with higher readmission. This finding may be attributable to the confrontational nature of the treatment in many of these alcoholism programs. Consistent with our earlier findings, older alcoholic patients who had longer episodes of inpatient care and immediate outpatient mental health aftercare had lower readmission rates. In contrast, younger patients did better in programs that emphasized more family involvement and consultation with community agencies in planning treatment and in treatment focusing on social and work skills.

These findings suggest that a more supportive treatment regimen in a well-organized program and prompt outpatient mental health aftercare may be especially helpful for older patients. Similarly, clinical observation shows that older substance abuse patients respond well to attempts to meet their special needs, such as slower paced treatment sessions to accommodate cognitive decline, and more supportive counseling approaches (Atkinson 1995). Consistent with this, Rice and colleagues (1993) found that older adults did best in individual-focused therapy, whereas younger patients did better in relationship-focused treatment.

Overall, family involvement and life-skills training may be less effective for older than for younger patients, perhaps because older patients with a long history of substance abuse have eroded family and community support. Moreover, the development of work and social skills that is often emphasized in the treatment of younger patients may not fit the developmental needs of older patients, many of whom are retired and have fewer family responsibilities.

FUTURE DIRECTIONS

Considerable progress has been made in the past decade toward understanding late-life problem drinking and alcohol dependence. Contrary to the idea that late-life alcohol problems have a good prognosis, we identified relatively low rates of stable 4-year remission (i.e., 21 percent) among late-life problem drinkers and relatively high rates of relapse (i.e., 60 percent or more) among late-life alcoholic patients. On a more hopeful note, treatment is associated with better 4-year outcomes among both problem drinkers and alcoholics. Moreover, health stressors

and disapproval of drinking by spouses and friends seem to foreshadow remission, especially among late-onset problem drinkers.

Consistent with other research, late-onset problem drinkers drank less and had fewer drinking problems, had somewhat more benign life contexts, and were more likely to remit from drinking problems than were people identified as early-onset problem drinkers. Further longitudinal research is needed to identify new cases of late-onset problem drinking and to determine the predictors of drinking problem onset and remission in this group. One promising area to examine is the role of "informal treatment" (i.e., disapproval from spouse and friends) in promoting remission among late-onset problem drinkers.

Our findings also show that older substance abuse patients heavily use health care services, and service use is even heavier among those people whose treatment is complicated by a long history of alcohol abuse and psychiatric comorbidity. These findings imply a need to study treatment approaches that combine elements of effective psychiatric and substance abuse care (Swindle et al. 1995). More work is also needed to develop and evaluate supportive counseling approaches that incorporate a focus on harm reduction, which may be especially beneficial for older alcoholic patients.

Continuity of care and outpatient mental health aftercare promoted better outcomes among older patients. These factors may be especially important for older patients who have few informal social resources. Accordingly, an important question to pursue is how much and what kinds of mental health aftercare are optimal for maintaining remission. Some evidence suggests that it may be best to provide low-intensity treatment for a longer duration (Finney and Moos 1997)—for example, monthly or bimonthly outpatient services delivered over an extended period.

As our conceptual model implies, the work on treated late-life alcohol dependence should be extended to examine how older patients' life stressors, social resources, and coping responses influence the course of late-life alcohol dependence. We also need to consider the effectiveness of treatment with respect to a broader range of outcomes, such as remission, abstinence, and other indices of psychosocial functioning. Finally, because self-help groups such as Alcoholics Anonymous may provide a stable source of structure and support, which are lacking in many older patients' lives, it is important to examine their role in maintaining remission.

With more dependable information in these areas, we can fulfill our primary goal, which is to specify personal and contextual factors that can guide the formulation of interventions to prevent the onset and persistence of excessive drinking in later life.

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REFERENCES

Aldwin, C.M.; Sutton, K.J.; Chiara, G.; and Spiro, A. Age differences in stress, coping, and appraisal: Findings from the Normative Aging Study. *J Gerontol: Psychol Sci* 51B:179–188, 1996.

Alexander, F., and Duff, R.W. Social interaction and alcohol use in retirement communities. *The Gerontol* 28:632–636, 1988.

Andersen, R.M. Revisiting the behavioral model and access to medical care: Does it matter? *J Health Soc Behav* 36:1–10, 1995.

Atkinson, R. Treatment programs for aging alcoholics. In: Beresford, T., and Gomberg, E., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 186–210.

Atkinson, R.M.; Tolson, R.L.; and Turner, J.A. Factors affecting outpatient treatment compliance of older male problem drinkers. *J Stud Alcohol* 54:102–106, 1993.

Babor, T.F.; Hoffman, M.; DelBoca, F.K.; Hesselbrock, V.; Meyer, R.E.; Dolinsky, Z.S.; and Rounsaville, B. Evidence for an empirically derived typology based on indicators of vulnerability and severity. *Arch Gen Psychiatry* 49:599–608, 1992.

Becker, M.H.; Haefer, D.P.; Kasl, S.V.; Kirscht, J.P.; Maiman, L.A.; and Rosenstock, I.L. Selected psychosocial models and correlates of individual health-related behaviors. *Med Care* 15:27–36, 1977.

Brennan, P.L., and Moos, R.H. Functioning, life context, and help-seeking among late-onset problem drinkers: Comparisons with nonproblem and early-onset problem drinkers. *Br J Addict* 86:1139–1150, 1991.

Brennan, P.L., and Moos, R.H. Life context, coping responses, and adaptive outcomes: A stress and coping perspective on late-life problem drinking. In: Beresford, T.P., and Gomberg, E., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 230–248.

Brennan, P., and Moos, R. Late-life drinking behavior: The influence of personal characteristics, life context, and treatment. *Alcohol Health Res World* 20:197–204, 1996*a*.

Brennan, P.L., and Moos, R.H. Late-life problem drinking: Personal and environmental risk factors for 4-year functioning outcomes and treatment-seeking. *J Subst Abuse* 8:167–180, 1996*b*.

Brennan, P.L.; Moos, R.H.; and Kim, J.Y. Gender differences in the individual characteristics and life contexts of late-middle-aged and older problem drinkers. *Addiction* 88:781–790, 1993.

Brennan, P.L.; Moos, R.H.; and Mertens, J.R. Personal and environmental risk factors as predictors of alcohol use, depression, and treatment-seeking: A longitudinal analysis of late-life problem drinkers. *J Subst Abuse* 6:191–208, 1994.

Cloninger, C.R. Neurogenetic adaptive mechanisms in alcoholism. *Science* 236:410–416, 1987.

Cooper, M.L.; Russell, M.; Skinner, J.B.; Frone, M.R.; and Mudar, P. Stress and alcohol use: Moderating effects of gender,

coping, and alcohol expectancies. *J Abnorm Psychol* 101:139–152, 1992.

Ekerdt, D.J.; De Labry, L.O.; Glynn, R.J.; and Davis, R.W. Change in drinking behaviors with retirement: Findings from the Normative Aging Study. *J Stud Alcohol* 50:347–353, 1989.

Finney, J., and Moos, R. Life stressors and problem drinking among older adults. In: Galanter, M., ed. *Recent Developments in Alcoholism.* Vol. 2. New York: Plenum, 1984. pp. 267–288.

Finney, J., and Moos, R. Entering treatment for alcohol abuse: A stress and coping model. *Addiction* 90:1223–1240, 1995.

Finney, J., and Moos, R. Effective psychosocial treatment for alcohol use disorders. In: Nathan, P.E., and Gorman, J.M., eds. *Treatments That Work.* New York: Oxford University Press, 1997. pp. 156–166.

Finney, J.W.; Moos, R.H.; and Brennan, P.L. The Drinking Problems Index: A measure to assess alcohol-related problems among older adults. *J Subst Abuse* 3:395–404, 1991.

Glass, T.A.; Prigerson, H.; Kasl, S.L.; and Mendes de Leon, C.F. The effects of negative life events on alcohol consumption among older men and women. *J Gerontol: Soc Sci* 4:S205–S216, 1995.

Graham, K.; Saunders, S.; and Flower, M. *Addiction Treatment for Older Adults: Evaluation of an Innovative Client-Centered Approach.* New York: Haworth, 1994.

Hasin, D.S. Treatment/self-help for alcohol-related problems: Relationship to social pressure and alcohol dependence. *J Stud Alcohol* 55:660–666, 1994.

Hermos, J.A.; LoCastro, J.S.; Bouchard, G.R.; and Glynn, R.J. Influence of cardiovascular disease on alcohol consumption among men in the Normative Aging Study. In: Maddox, G.; Robins, L.N.; and Rosenberg, N., eds. *The Nature and Extent of Alcohol Problems Among the Elderly.* New York: Springer, 1984. pp. 117–132.

Jennison, K.M. The impact of stressful life events and social support on drinking among older adults: A general population survey. *Int J Aging Hum Dev* 35:99–123, 1992.

Kofoed, L.L.; Tolson, R.L.; Atkinson, R.M.; Toth, R.L.; and Turner, J.A. Treatment compliance of older alcoholics: An elderly-specific approach is superior to "mainstreaming." *J Stud Alcohol* 48:47–51, 1987.

Krause, N. Stress, religiosity, and abstinence from alcohol. *Psychol Aging* 6:134–144, 1991.

Krause, N. Stress, alcohol use, and depressive symptoms in later life. *Gerontologist* 35:296–307, 1995.

Liberto, J.G., and Oslin, D.W. Early versus late onset of alcoholism in the elderly. *Int J Addict* 30:1799–1818, 1995.

Moos, R. *Coping Responses Inventory: Adult Form Manual.* Odessa, FL: Psychological Assessment Resources, 1993.

Moos, R., and Finney, J. A systems perspective on problem drinking among older adults. In: Maddox, G.; Robins, L.; and Rosenberg, N., eds. *The Nature and Extent of Alcohol Problems Among the Elderly.* New York: Springer, 1984. pp. 151–172.

Moos, R., and Moos, B. *Life Stressors and Social Resources Inventory: Adult Form Manual.* Odessa, FL: Psychological Assessment Resources, 1994.

Moos, R.; Finney, J.; and Cronkite, R. *Alcoholism Treatment: Context, Process, and Outcome.* New York: Oxford, 1990.

Moos, R.H.; Brennan, P.L.; and Moos, B.S. Short-term processes of remission

and nonremission among late-life problem drinkers. *Alcohol Clin Exp Res* 15: 948–955, 1991.

Moos, R.H.; Mertens, J.R.; and Brennan, P.L. Patterns of diagnosis and treatment among late-middle-aged and older substance abuse patients. *J Stud Alcohol* 54:479–487, 1993.

Moos, R.H.; Brennan, P.L.; and Mertens, J.R. Diagnostic subgroups and predictors of one-year readmission among late-middle-aged and older substance abuse patients. *J Stud Alcohol* 55:173–183, 1994*a*.

Moos, R.H.; Brennan, P.L.; and Mertens, J.R. Mortality rates and predictors of mortality among late-middle-aged and older substance abuse patients. *Alcohol Clin Exp Res* 18:187–195, 1994*b*.

Moos, R.H.; Mertens, J.R.; and Brennan, P.L. Rates and predictors of four-year readmission among late-middle-aged and older substance abuse patients. *J Stud Alcohol* 55:561–570, 1994*c*.

Moos, R.H.; Mertens, J.R.; and Brennan, P.L. Program characteristics and readmission among older substance abuse patients: Comparisons with middle-aged and younger patients. *J Ment Health Admin* 22:332–345, 1995.

Peirce, R.S.; Frone, M.R.; and Russell, M. Relationship of financial strain and psychosocial resources to alcohol use and abuse: The mediating role of negative affect and drinking motives. *J Health Soc Behav* 35:291–308, 1994.

Rice, C.; Longabaugh, R.; Beattie, M.; and Noel, N. Age group differences in response to treatment for problematic alcohol use. *Addiction* 88:1369–1375, 1993.

Schonfeld, L., and Dupree, L.W. Antecedents of drinking for early- and late-onset elderly alcohol abusers. *J Stud Alcohol* 52:587–592, 1991.

Schonfeld, L., and Dupree, L.W. Treatment approaches for older problem drinkers. *Int J Addict* 30:1819–1842, 1995.

Schutte, K.K.; Brennan, P.L.; and Moos, R.H. Remission of late-life drinking problems: A 4-year follow-up. *Alcohol Clin Exp Res* 18:835–844, 1994.

Schutte, K.K.; Moos, R.H.; and Brennan, P.L. Depression and drinking behavior among women and men: A three-wave longitudinal study of older adults. *J Consult Clin Psychol* 63:810–822, 1995.

Steinglass, P.; Bennett, L.; Wolin, S.J.; and Reiss, D. *The Alcoholic Family.* New York: Basic Books, 1987.

Swindle, R.; Phibbs, C.; Paradise, M.; Recine, B.; and Moos, R. Inpatient treatment for substance abuse patients with psychiatric disorders: A national study of determinants of readmission. *J Subst Abuse* 7:79–97, 1995.

Weisner, C. Toward an alcohol entry treatment model: A comparison of problem drinkers in the general population and in treatment. *Alcohol Clin Exp Res* 17:746–752, 1993.

Welte, J.W., and Mirand, A.L. Drinking, problem drinking and life stressors in the elderly general population. *J Stud Alcohol* 56:67–73, 1995.

Zautra, A.J., and Wrabetz, A.B. Coping success and its relationship to psychological distress for older adults. *J Pers Soc Psychol* 61:801–810, 1991.

Chapter 16

Effects of Alcohol on Sleep

Michael S. Aldrich, M.D.

Alcohol has a range of effects on sleep, the consequences of which vary with age. Young persons tend to sleep well, and their sleep mechanisms are able to withstand stresses. Older persons sleep less well and are more susceptible to sleep-disrupting effects of a variety of factors, including alcohol. This chapter provides a brief description of sleep and its changes with age, followed by a review of the effects of alcohol on sleep architecture and on sleepiness and alertness, the effects of alcohol on breathing during sleep in normal persons and in those with obstructive sleep apnea (OSA), the effects of alcohol on insomnia, and the effects of alcohol on sleep in alcoholics.

SLEEP AND CHANGES IN SLEEP WITH AGE

Sleep consists of two fundamentally different sleep states: rapid eye movement (REM) sleep and non-REM sleep. Non-REM (NREM) sleep is further divided into four stages. Stage 1 is the lightest stage, and stage 2 is the intermediate stage characterized by sleep spindles and K-complexes on the electroencephalogram (EEG). Stages 3 and 4 are deeper stages characterized by high-voltage slow waves on the EEG called delta waves; consequently these stages are often referred to collectively as slow wave sleep or delta sleep. Sleep usually begins with stage 1 sleep followed by rapid progression to deeper stages of NREM sleep. After about 80 minutes, sleep lightens and a change in body position may occur, followed by a few minutes of light sleep and then entry into the first period of REM sleep. For the remainder of the night, REM sleep and NREM sleep alternate with an 80–90 minute cycle (figure 1).

Although the function of sleep remains a mystery, both REM and NREM sleep are essential processes

M.S. Aldrich, M.D., is director of the Sleep Disorders Center and associate professor of neurology, Department of Neurology, UH 8820/0117, University of Michigan Medical Center, 1500 E. Medical Center Dr., Ann Arbor, MI 48109–0117.

that are homeostatically regulated. Deprivation of REM sleep leads to increased pressure to enter REM sleep, and when REM sleep is permitted to occur, there is a rebound increase in its amount. Similar effects are observed with slow wave sleep deprivation. In animals, total sleep deprivation, or selective deprivation of REM sleep or slow wave sleep, leads to death within a few weeks (Rechtschaffen et al. 1989).

Patterns of sleep and wakefulness change over the lifespan. Sleep patterns develop during infancy and childhood, and the stages of sleep are well defined by age 2 years. The circadian variation between sleep at night and wakefulness during the day also develops early in life. By the age of 6 years, most children are excellent sleepers: they fall asleep easily, sleep continuously, awaken refreshed, and remain alert throughout the day. After age 12, there is a gradual decline in the amount of slow wave sleep that continues for several decades. However, the reduction in slow wave sleep is due partly to increased thickness of the skull, which attenuates the amplitude of slow waves recorded at the scalp. It is unknown whether the need for slow wave sleep also declines or whether the restorative process that accompanies slow wave sleep continues unabated despite the reduction in the number and amplitude of slow waves.

In adulthood, there is also a gradual increase in the amount of time spent awake at night and in the

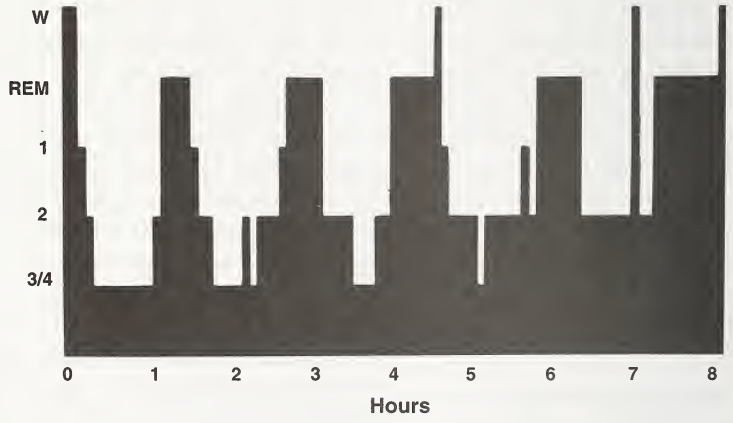

Figure 1. Idealized sleep hypnogram of a normal night of sleep in a young adult, including periods of wakefulness (W), REM sleep (REM), and stages 1, 2, and 3/4 of NREM sleep. Brief arousals and awakenings have been omitted for the sake of clarity. Sleep onset occurs shortly after lights out and is followed by a rapid progression into stage 3/4 (slow wave) sleep. After about 80 minutes of sleep, the first REM sleep period begins. For the remainder of the night, NREM sleep and REM sleep alternate with about five cycles during the night. The depth of NREM sleep tends to decrease and the duration of REM sleep periods tends to increase with successive cycles.

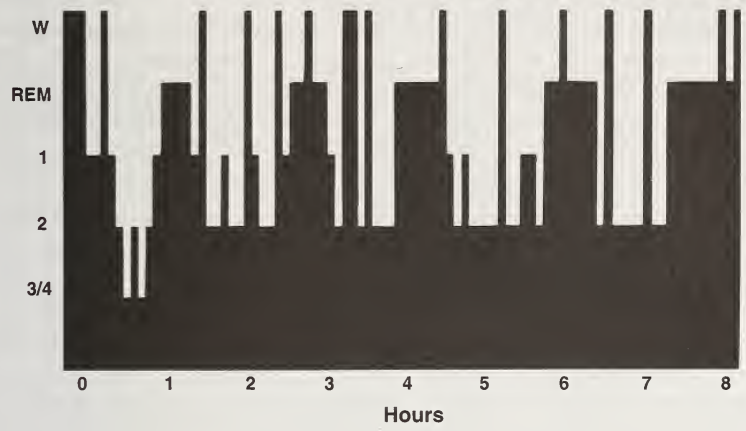

Figure 2. Idealized sleep hypnogram of a night of sleep in an adult over age 70 years, including periods of wakefulness (W), REM sleep (REM), and stages 1, 2, and 3/4 of NREM sleep. Brief arousals and awakenings have been omitted for the sake of clarity. Compared with younger adults, sleep onset is delayed, the amount of stage 3/4 sleep is reduced, and the amount of stage 1 sleep is increased. Frequent awakenings disrupt sleep continuity and reduce the amount of time spent asleep.

amount of stage 1 sleep. This trend toward increasing wakefulness at night appears to accelerate after age 65 years (figure 2). Elderly persons often awaken 10–20 or more times throughout the night, and the fragmentation of sleep produced by these awakenings leads to sleep that is less restful and restorative. Thus, the elderly need to spend more time in bed to get the same amount of sleep.

EFFECTS OF ALCOHOL ON SLEEP ARCHITECTURE AND ON SLEEPINESS AND ALERTNESS

Alcohol consumed in the evening alters sleep/wake patterns in several ways. This section presents a discussion of, first, its effect on the amount of sleep and the distribution of stages of sleep (sleep architecture) and, second, its effect on the degree of alertness during wakefulness.

Sleep Architecture

The effects of alcohol on sleep architecture are a function of the dose of alcohol and the amount of alcohol consumed on prior evenings. Three principal acute effects on nighttime sleep have been observed: effects on REM sleep, effects on slow wave sleep, and effects on sleep time and sleep continuity (table 1). The effects of alcohol on the time required to fall asleep, or sleep latency, are discussed in the next section.

Inhibition of REM sleep by alcohol was first observed more than 30 years ago (Gresham et al. 1963; Yules et al. 1966; Knowles et al. 1968). The reduction in the amount of REM

Table 1. Effects of Alcohol on Sleep in Normal Subjects.

Subjects	Dose	Effects	Reference
17 adults	1.1 mL/kg	↓ TST, ↓ REM	Block and Hellard 1987
78 adults	1 mL/kg	↓ TST in older men and postmenopausal women but not in young men and women	Block et al. 1986
4 young men	1 g/kg	↓ REM	Yules et al. 1967
7 adults	1 g/kg	↓ REM	Gresham et al. 1963
10 young adults	0.25, 0.75, 1.0 g/kg	↑ SWS in 1st 3 hr	MacLean and Cairns 1982
10 young adults	0.9 g/kg	↓ REM during 1st half of night, ↑ REM during 2nd half	Rundell et al. 1972
6 young adults	0.8 g/kg	↑ REM latency, ↓ awakenings	Scrima et al. 1982
5 young men	0.8 g/kg	↑ SWS, ↓ REM during 1st half of night	Prinz et al. 1980
6 young adults	0.16, 0.32, 0.64 g/kg	↑ TST at 0.16 g/kg, ↓ SWS	Stone 1980
8 young men	0.6 g/kg	↑ SWS in 1st 2 hr of sleep	Dijk et al. 1992
11 young women	0.5, 0.75 g/kg	↓ REM and ↑ SWS in 1st 3 hr, ↑ late night sleep disturbance	D.L. Williams et al. 1983
48 adults	0.5 mL/kg	↓ TST	Aldrich et al. 1995
8 young men–naps	0.8 mL/kg	↑ SWS	Van et al. 1995
14 adults–naps	0.25 g/kg	↓ TST, ↓ REM	Rouhani et al. 1989

Note: REM = rapid eye movement sleep; SWS = slow wave sleep; TST = total sleep time.

sleep during the night is most prominent with alcohol doses of 1 g/kg or more, but even at lower doses alcohol may prolong the time to the onset of REM sleep (see table 1). Inhibition of REM sleep is reduced with regular alcohol use, probably because the loss of REM sleep leads to an increased need for REM sleep. For example, Yules and colleagues (1967) found that 1 g/kg alcohol (about 5 oz of 100 proof vodka) consumed 4 hours before bedtime reduced the amount of REM sleep on the first two nights but had no effect on subsequent nights. On the first night without alcohol, the amount of REM sleep increased, similar to the "rebound"

increase in REM sleep that occurs following experimental REM sleep deprivation. This rebound increase suggests that alcohol interferes with the biologically essential REM sleep process.

The amount of slow wave sleep usually increases with bedtime alcohol administration, particularly during the first third of the night (Prinz et al. 1980; Dijk et al. 1992). This effect is most apparent with moderate or high doses; in one study that used low doses of alcohol, the amount of slow wave sleep declined (Stone 1980). The effect on slow wave sleep diminishes with repeated nights of alcohol consumption (Prinz et al. 1980). However, the increased slow wave sleep is probably not an indication of an increase in the homeostatically regulated restorative process that underlies natural slow wave sleep. Cortical dysfunction from a variety of causes may lead to increased amounts of slow waves on the waking EEG and on the EEG recorded during sleep. It is likely that the increased amount of sleep accompanied by slow waves is due to toxic effects of alcohol or its metabolites rather than to an increase in activity of generators of normal slow wave sleep.

The effects of alcohol on sleep continuity and on the total amount of sleep are more variable, but they also appear to be dose related. D.L. Williams and colleagues (1983) noted an increase in late-night sleep disturbance following evening alcohol consumption, but others have found a reduction in the number of awakenings for the night as a whole (Scrima et al. 1982). In studies that used a bedtime dose of 0.5 g/kg or more, total sleep time decreased or was unchanged; however, Stone (1980) noted an increase in total sleep time after doses of 0.16 and 0.32 g/kg. Low doses may have principally sedative effects that increase sleep time, whereas higher doses may produce not only sedative effects but also toxic effects or short-term withdrawal effects associated with increased sympathetic activity that are more potent than the sedative effects and lead to sleep disruption, especially during the second half of the night.

Less is known about the chronic effects of moderate alcohol consumption on sleep, although EEG changes induced by alcohol on one night tend to be less apparent on subsequent nights, suggesting that some tolerance to the effects of alcohol occurs (Rundell et al. 1972). Prinz and colleagues (1980) administered 0.8 g/kg alcohol to five subjects for nine consecutive nights. Slow wave sleep increased on the first alcohol night but returned to baseline values by the ninth night; the amount of REM sleep was modestly reduced on the ninth night, but the amounts of REM sleep and slow wave sleep on the first night without alcohol were not significantly different from nights recorded prior to alcohol administration. For persons who drink more heavily, chronic REM sleep suppression during the first half of the night, when blood alcohol levels are substantial, may lead to REM sleep rebound during the second half of the night, when blood alcohol levels are close to zero, or during subsequent nights without alcohol. The increased REM sleep, combined with late-night sleep disruption, may lead to increased

dream recall as a result of awakenings during REM sleep.

Changes in the effects of alcohol on sleep as a function of age have not been examined in detail. The findings summarized in table 1 indicate that total sleep time generally is not reduced significantly by alcohol in young people. Block and colleagues (1986), however, found that total sleep time was reduced by 1 mL/kg alcohol in older men and postmenopausal women. On the other hand, our laboratory (Aldrich et al. 1995), using a smaller dose of 0.5 mL/kg, found that total sleep time was reduced in subjects under age 40 but not in those over age 40 years. Differences in these results may relate to the dose of alcohol or to other factors. Elderly persons are more likely to have insomnia and other sleep disorders than younger persons, and the effects of alcohol on their sleep may vary as a result of the sleep disruption.

Sleepiness and Alertness

That alcohol can lead to sleepiness has been known for centuries. Although sleepiness and alertness can be assessed in a variety of ways, the Multiple Sleep Latency Test (MSLT) is the most widely used method for objective assessment of sleepiness (Carskadon et al. 1986). The test is based on the principle that the time required to fall asleep in a setting conducive to sleep provides a measure of physiological sleepiness, or sleep need. With the MSLT, the time to fall asleep (sleep latency) is measured at intervals throughout the day. The mean sleep latency of the repeated opportunities for sleep provides an overall measure of sleepiness for that day.

Alcohol can have either a stimulating effect that increases sleep latency or a sedating effect that reduces sleep latency. In general, stimulating effects are produced at low doses and as blood alcohol levels are rising; sedative effects occur at high doses and as blood levels are falling (Petrucelli et al. 1994; O'Boyle et al. 1995). Thus, the effect of alcohol on sleep latency depends partly on the duration of the interval between alcohol consumption and bedtime. Stimulating effects of alcohol taken right at bedtime may increase sleep latency, but alcohol consumed an hour or more before bedtime is likely to reduce sleep latency. The sedating effects are dose dependent for moderate levels of alcohol consumption (0.4–0.8 g/kg), and a dose of alcohol comparable to two to three drinks (0.6 g/kg alcohol) leads to increased sleepiness for several hours (Zwyghuizen-Doorenbos et al. 1988; Roehrs et al. 1993). Furthermore, sedating effects and associated impairments of reaction time and performance persist for at least an hour or two after blood levels have dropped to zero (Roehrs et al. 1993, 1994*a*, 1994*b*).

The effect of alcohol on alertness also depends on the time of day and on basal levels of sleepiness, with greater reductions in sleep latencies in subjects who are more sleepy (Zwyghuizen-Doorenbos et al. 1990). Alcohol given at 8 or 9 a.m. has a greater sedating effect than the same dose given at 4 or 5 p.m. (Roehrs et al. 1992; O'Boyle et al. 1995). When sleepiness is induced by sleep restriction, the sedating effects

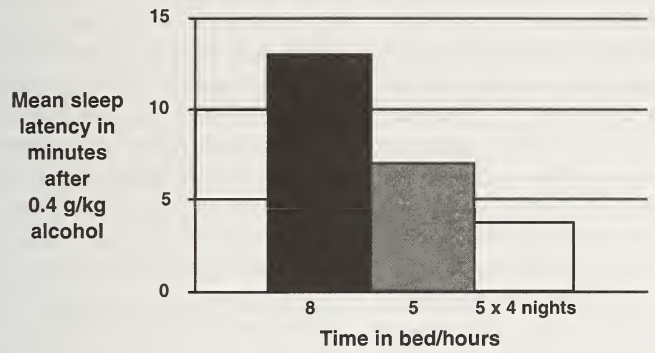

Figure 3. Effects of sleep restriction on the sedating effects of alcohol. After 8 hours in bed, the daytime mean sleep latency following a moderate dose of alcohol is within the normal range. Sleepiness induced by alcohol increases significantly after one night of 5 hours in bed and increases even more after four nights of 5 hours in bed. Data from Zwyghuizen-Doorenbos et al. 1988.

of alcohol increase (figure 3). A dose of 0.4 g/kg of alcohol administered to normal subjects after their time in bed was reduced to 5 hours per night for four nights had a sedative effect equal to that of 0.8 g/kg of alcohol administered to individuals who had spent 8 hours in bed (Zwyghuizen-Doorenbos et al. 1988). The increase in sleepiness appears to have significant functional consequences. For example, sleepiness induced by partial sleep deprivation and low-dose alcohol combines to impair simulated automobile driving and reaction time (Krull et al. 1993; Roehrs et al. 1994*a*).

The sedating effects of alcohol are reduced by sleep extension and are partially reversible with naps (Roehrs et al. 1989, 1993). Roehrs and colleagues (1989) found that subjects who drank 0.75 g/kg alcohol after spending 10 hours per night in bed for 1 week had levels of sleepiness comparable to subjects who spent 8 hours in bed and received no alcohol. Even one night of 11 hours in bed can reduce the degree of sleepiness associated with alcohol use (Lumley et al. 1987).

EFFECTS OF ALCOHOL ON BREATHING DURING SLEEP IN NORMAL PERSONS AND IN PERSONS WITH OSA

The association of alcohol use with snoring has also been recognized for centuries. However, snoring was generally considered a harmless annoyance rather than a health concern prior to the discovery of obstructive sleep apnea in the 1960's. With increased recognition of the high prevalence of OSA and accumulating evidence that it has significant health consequences, the effects of alcohol on breathing have taken on added significance.

Obstructive sleep apnea is characterized by periods of airway closure

or partial closure during sleep. During wakefulness, upper airway dilator muscles help to maintain airway patency. During sleep, these muscles become less active and the diameter of the airway decreases. The pharyngeal airway is most susceptible to narrowing and closure because it lacks the skeletal and cartilaginous support that is present in more proximal and distal segments. As the airway narrows, resistance to breathing increases. The increased force generated by the diaphragm to overcome the added resistance can lead to further narrowing or collapse of the pharyngeal airway. Once the airway closes, the individual generally must awaken partially or completely in order to activate the upper airway dilator muscles and reopen the airway. The repeated arousals to reopen the airway lead to sleep disruption and to complaints of daytime sleepiness. With severe OSA, the cycle of apnea during sleep followed by arousal followed by return to sleep with airway closure and apnea occurs hundreds of times each night. Excessive daytime sleepiness, loud snoring, and periods of apnea witnessed by the bed partner are the usual presenting symptoms of OSA. The prevalence of symptomatic OSA is estimated to be about 2 percent in adult women and 4 percent in adult men (Young et al. 1993).

Alcohol has two major effects on breathing during sleep: it relaxes upper airway dilator muscles and it prolongs the time required to arouse or awaken after an apnea occurs. The relaxation of upper airway dilators leads to upper airway narrowing, increased resistance to breathing, increased airflow velocity and turbulence, and increased likelihood of snoring. Robinson and colleagues (1985) observed a greater than 50 percent increase in nasal and pharyngeal resistance during wakefulness after alcohol consumption by normal male subjects. With evening alcohol consumption, similar increases in airway resistance occur during sleep, particularly during the first 2 hours of sleep when blood alcohol levels are highest (Mitler et al. 1988; Dawson et al. 1993).

The increased airway resistance produced by alcohol leads to increased inspiratory effort, which may in turn lead to airway collapse and obstructive apneas in persons who do not usually have them. The likelihood of developing obstructive apneas after alcohol consumption is increased in persons who snore because their pharyngeal airways tend to be smaller than those of nonsnorers. Men appear to be more susceptible than women to the effects of alcohol on breathing, probably because they tend to have narrower pharyngeal airways. Block and colleagues (Block 1984; Block et al. 1986) found that 1 mL/kg of alcohol had a detrimental effect on nocturnal oxygenation in men but not in women. Furthermore, persons who already have OSA appear to be more susceptible to the effects of alcohol on breathing than those who do not (Issa and Sullivan 1982; Scrima et al. 1982, 1989). In 14 patients with mild OSA, the rate of respiratory events per hour of sleep doubled after consumption of 0.5 mL/kg alcohol, and the lowest nocturnal oxygen saturation was 79 percent compared with 85 percent on the baseline night

(Collop 1994). In persons who are not at high risk for OSA, the effects on breathing are less (Scrima et al. 1982). Scrima and colleagues (1989) found that alcohol doses of 0.32, 0.65, and 0.81 g/kg had little effect on sleep-disordered breathing in men who did not have OSA and were not obese.

Once apnea has occurred, the impairment of arousal mechanisms produced by the central nervous system depressant effects of alcohol leads to increased duration of apneas, which in turn leads to increased hypoxemia. Taasen and colleagues (1981) found that bedtime ingestion of 2 mL/kg of alcohol increased arterial oxygen desaturation during sleep, and the increase in arterial oxygen desaturation persisted for an additional night, even when no additional alcohol was consumed. High doses of alcohol, up to 3.0 g/kg, resulted in a marked increase in the degree of hypoxemia in the first hour of sleep in five patients with OSA (Issa and Sullivan 1982). Although the precise mechanisms that lead to arousal from apnea are not fully delineated, increasing respiratory effort, hypoxemia, and hypercapnia with acidosis all appear to contribute. Berry and colleagues (1992) found that 1 mL/kg of alcohol led to an increase in the time to arousal after experimental airway occlusion by increasing the threshold of inspiratory effort associated with arousal and by decreasing the rate of increase in the magnitude of inspiratory efforts (figure 4).

Alcohol use and OSA can interact to affect daytime function in at least three ways. First, evening alcohol use may increase the frequency and severity of OSAs and thereby lead to greater sleep disruption and more pronounced daytime effects. Second, the

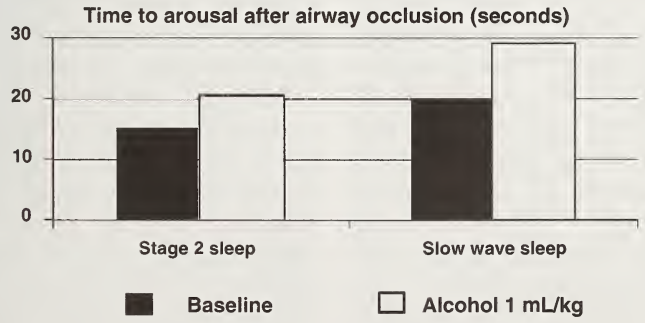

Figure 4. Effect of alcohol on time to arousal from NREM sleep after experimental airway occlusion in adult male subjects. For occlusions during stage 2 sleep, the time to arousal increased from 15 seconds to 21 seconds; for those during slow wave sleep, the time to arousal increased from 20 seconds to 29 seconds. Prior treatment with supplemental oxygen to blunt hypoxemia during apnea led to even longer times to arousal after occlusion: 64 seconds during slow wave sleep after alcohol compared with 32 seconds during slow wave sleep without alcohol. Data from Berry et al. 1992.

sleepiness induced by OSA may add to sedative effects of alcohol. Third, sleep disruption unrelated to OSA that occurs in association with evening alcohol use may add to sleep disruption associated with apneas to cause even greater impairment of daytime function.

Driving performance is one area in which the interaction of alcohol and OSA may be particularly significant. Obstructive sleep apnea is associated with impaired performance on a driving simulator, an increased rate of all motor vehicle accidents, and an increased rate of sleep-related motor vehicle accidents (George et al. 1987; Findley et al. 1988; Aldrich 1989; Findley et al. 1995). Aldrich and Chervin (1997) assessed self-reports of sleep-related accidents in subjects with OSA and in a group of subjects recruited from the general population. The proportion of subjects with one or more sleep-related motor vehicle accidents was 6.7 percent in the control group and 16 percent in the OSA group. Among subjects who consumed 14 or more alcoholic drinks per week, the proportion with one or more sleep-related accidents was 13 percent in the control group and 26 percent in those with severe OSA. Logistic regression analysis indicated that diagnosis of sleep apnea and heavy alcohol use were independent contributors to the increased risk of sleep-related accidents.

Obstructive sleep apnea has significant physiological consequences in addition to its effects on sleep and alertness. During the periods of apnea, body oxygen stores are reduced and oxyhemoglobin saturation falls. Cardiac output may fall as a result of increased negative intrathoracic pressure associated with attempts to breathe against a closed airway that produce right-to-left shifts of the cardiac interventricular septum. Increased vagal tone associated with fluctuations in intrathoracic pressure and stimulation of the carotid body by hypoxemia contribute to arrhythmias during the apneas, including bradycardia, periods of asystole, and atrioventricular block. Hypoxemia and acidosis lead to arteriolar constriction that leads in turn to increased pulmonary and systemic arterial blood pressure despite the fall in cardiac output. Blood pressure reaches a peak with the arousal and the resumption of ventilation. Increased sympathetic nervous system activity accompanying the arousals contributes to increased blood pressure and to tachycardias that commonly occur with arousals. Ventricular premature contractions and dangerous ventricular tachyarrhythmias may also occur.

Although there is no conclusive evidence that sleep apnea alone can lead to daytime systemic hypertension, it seems likely that it can occur in some patients as a result of severe hypoxemia or altered sympathetic activity. The incidence of OSA is greater than 25 percent in patients with hypertension; in untreated severe OSA, the prevalence of hypertension may be as high as 50 percent (Kales et al. 1984; Lavie et al. 1984; Fletcher et al. 1985; A.J. Williams et al. 1985). The risk of hypertension is almost fourfold greater in persons with sleep-disordered breathing

compared with nonsnorers (Olson et al. 1995).

The hemodynamic changes during apneas and subsequent arousals may contribute to cardiovascular and cerebrovascular disease (Jennum and Borgesen 1989; Siebler et al. 1990). Snoring—and by inference OSA—is associated with myocardial infarction, stroke, and sudden death (Hung et al. 1990; Palomaki 1991; Seppala et al. 1991). The risk for ischemic heart disease and stroke is 2- to 3.5-fold greater for habitual snorers than for nonsnorers and remains elevated after controlling for the effects of other cardiovascular risk factors (Koskenvuo et al. 1987; Smirne et al. 1993; Olson et al. 1995). Patients with OSA who are treated conservatively have a greater risk of vascular morbidity than patients treated with tracheostomy (Partinen and Guilleminault 1990). In one series, patients with untreated OSA and more than 20 apneas per hour of sleep had an estimated 37 percent chance of dying within 8 years compared with 4 percent for treated patients (He et al. 1988).

Since alcohol is also a risk factor for vascular disease, the combination of alcohol and OSA may increase the risk for vascular morbidity. The increased risk of hypoxemia and the alterations in sympathetic activity that are associated with alcohol use appear to increase the risk that cardiac arrhythmias will occur in association with sleep-disordered breathing, particularly in those with other risk factors (Dolly and Block 1983; Easton et al. 1987). Alcohol use and OSA may also interact to increase the risk of stroke. In one series of 75 patients who presented with acute stroke or a transient ischemic attack, 23 percent had a history of habitual snoring. In the subgroup with habitual snoring, apnea severity correlated positively with alcohol use and with the number of drinks in the 24 hours preceding the vascular event (Bassetti and Aldrich 1996).

EFFECTS OF ALCOHOL ON SLEEP IN PERSONS WITH INSOMNIA

Alcohol can affect sleep in persons with insomnia. Because of its sedative effects, alcohol is often used by insomniacs to promote sleep onset. Some insomniacs developed conditioned associations of alcohol use with falling asleep and eventually come to believe that they cannot sleep without a "nightcap." Anxiety and symptoms of mild alcohol withdrawal that may occur if alcohol is not consumed may contribute to their impression that they cannot sleep without alcohol. With chronic bedtime use, however, the sleep-inducing effect of alcohol may be reduced while its effects on late-night sleep disturbance continue or are increased. The insomniac may experience sweaty restless sleep during the second half of the night and may have difficulty returning to sleep after awakening from dreams. The sleep disruption that occurs during the second half of the night may lead to daytime fatigue and sleepiness.

Effects of alcohol on insomnia are of particular importance in the elderly because insomnia and sleeping pill use increase with age. Approximately 25 percent of persons over age 65 complain

of frequent nighttime awakenings, and about 15 percent report sleeping less than 5 hours per night. Thus, elderly persons who drink alcohol in the evening or during the night are more likely to be doing so in the setting of poor sleep with delayed sleep onset and frequent nocturnal awakenings. The reduction in sleep latency that occurs with alcohol use may encourage continued alcohol use at bedtime and during the night. Furthermore, the lower volume of distribution for a given body weight in elderly persons leads to a higher blood alcohol level, and alcohol consumed just before bedtime or during the night may lead to unsteadiness during nighttime trips to the bathroom, with increased risk of falls and associated injuries.

Alcohol use also may affect the likelihood of having periodic leg movements of sleep. These movements, which consist of extension of the toe and ankle with varying degrees of lower and upper leg extension or flexion, typically occur every 20–30 seconds in series lasting for several minutes to an hour or more. Their prevalence increases with age, and up to one-third of persons over the age of 65 may have 30 or more such movements during each night of sleep. The movements may have no effect on sleep, or they may be associated with arousals that disrupt sleep continuity and lead to complaints of insomnia and daytime fatigue and drowsiness. Aldrich and Shipley (1993) found that the likelihood of having a clinically significant number of periodic leg movements (more than 20 per hour of sleep) was increased threefold in women who consumed 2 or more alcoholic drinks per day compared with those who did not (25 percent vs. 8 percent). A similar but less strong relation was found among men (22 percent vs. 13 percent). Thus, chronic alcohol use could lead to periodic leg movements, or periodic leg movements could result from sleep disturbance induced by alcohol, or alcohol use could be a result of sleep disturbance associated with periodic leg movements.

EFFECTS OF ALCOHOL ON SLEEP IN ALCOHOLICS

Alcoholics, when they are not drinking, tend to sleep poorly with decreased amounts of slow wave sleep, increased amounts of stage 1 sleep, and increased amounts of time spent awake at night. Heavy alcohol use among alcoholics leads to pronounced effects on this baseline level of poor sleep. With resumption of heavy drinking, acute effects include reduced REM sleep, increased slow wave sleep, and reduced amounts of stage 1 sleep and wakefulness, at least during the first 2 days of drinking (Lester et al. 1973; Zarcone et al. 1977; Skoloda et al. 1979; Zarcone et al. 1980). The decline in wakefulness and light stage 1 sleep during the first couple of nights after relapse probably contributes to the subjective impression of some alcoholics that sleep improves with resumption of drinking. Furthermore, the high prevalence of poor sleep in alcoholics suggests that the use of alcohol to promote sleep may be one factor that leads to relapse. On the other hand, the increase in slow wave sleep that

occurs in some alcoholics when they resume drinking probably reflects toxic effects on the EEG (slowing) rather than an increase in the restorative process that normally accompanies slow wave sleep.

Chronic effects of heavy alcohol use include reduced slow wave sleep, disturbed REM sleep, frequent awakenings and arousals, and a decrease in subjective sleep quality that is accompanied by daytime fatigue (Johnson et al. 1970). Nocturnal gastrointestinal symptoms and other systemic symptoms associated with alcohol abuse, as well as toxic effects of alcohol on the nervous system, probably contribute to sleep disturbance. Secondary depression or other psychological disturbances also play a role in some alcoholics.

Sleep is severely disturbed during alcohol withdrawal. Pronounced insomnia with marked sleep fragmentation is common, particularly during the first week of withdrawal (Johnson et al. 1970; Thompson et al. 1995). The amount of slow wave sleep is often reduced, and the severe REM sleep deprivation that accompanies heavy drinking can lead to "pressure" for REM sleep that results in reduced REM sleep latency, high frequency of rapid eye movements during REM sleep (high REM density), and increased amounts of REM sleep during the night (Johnson et al. 1970). The rebound increase in REM sleep during withdrawal may contribute to hallucinations, and in patients with delirium tremens sleep episodes may consist almost entirely of brief periods of REM sleep interrupted by numerous awakenings (Johnson et al. 1970). Furthermore, muscle atonia during REM sleep may be impaired, leading to a tendency to "act out" dreams and to the REM sleep behavior disorder (Tachibana et al. 1977; Kotorii et al. 1980; Schenck et al. 1987). Sleep disturbance continues during the second week of withdrawal but is less severe.

Sleep in abstinent alcoholics is characterized by delayed sleep onset, frequent awakenings, and reduced amounts of slow wave sleep (Adamson and Burdick 1973; Gillin et al. 1990), although insomnia usually is less severe than during withdrawal. After 3 months of abstinence, sleep in general remains abnormal although in comparison to the first weeks of withdrawal, the amount of slow wave sleep is increased, sleep latency is reduced, and sleep efficiency is improved (H.L. Williams and Rundell 1981; Drummond et al. 1995).

It is unknown whether sleep patterns ever return to normal in alcoholics, even among those who remain abstinent for years. In one study of alcoholics who had been abstinent for more than 1 year, the amount of slow wave sleep was below expected values (Adamson and Burdick 1973). In another study of five alcoholics who remained abstinent for 21 months or more, sleep latency was longer than expected for age (H.L. Williams and Rundell 1981). Alcoholics appear to be at increased risk for sleep apnea, particularly those who are over age 40 (Aldrich et al 1993). These findings suggest that at least some of the changes in sleep patterns may be permanent.

The pathophysiological basis for the changes in sleep that occur in alco-

holics is not well established. Acute effects on REM sleep reflect REM suppressant effects that may be mediated by monoaminergic mechanisms. The increase in REM sleep "pressure" that occurs during withdrawal is at least partly a result of REM sleep suppression during heavy alcohol use, but the REM sleep abnormalities are similar to those that can occur with depression and other psychiatric disorders and thus are nonspecific; similar changes in REM sleep occur during withdrawal in alcoholics with and without secondary depression (Clark et al. 1996). Circadian rhythm abnormalities or abnormal function of cholinergic or monoaminergic systems may also contribute to REM sleep abnormalities.

The cause and significance of the reduced amount of slow wave sleep that occurs in some abstinent alcoholics are also uncertain. Slow wave sleep appears to be generated primarily in the frontal lobes, and frontal lobe dysfunction from a variety of causes can lead to reduced amounts of slow wave sleep. It is possible that frontal lobe dysfunction caused by toxic effects of long-term high-dose alcohol use leads to reduced slow wave sleep. Abstinent alcoholics with low amounts of slow wave sleep appear to develop functional tolerance to alcohol and to reacquire physical dependence to alcohol more quickly than those with normal amounts of slow wave sleep (Gross and Best 1975; Allen et al. 1980), perhaps because of alterations in frontal lobe function that are related to slow wave sleep generation. In addition, the reduced amount of slow wave sleep could make sleep less restorative and contribute to a sense of daytime fatigue.

Although the precise mechanisms responsible for changes in sleep that occur during withdrawal and abstinence have not been established, subjective sleep quality appears to predict relapse potential, and the striking abnormalities of REM sleep and slow wave sleep have led investigators to assess the value of objective sleep measures for predicting relapse potential. Gillin and colleagues (1994) found that a measure of "REM pressure" derived from a combination of REM sleep latency, the proportion of total sleep that consisted of REM sleep (REM sleep percent), and REM density at the time of admission to an inpatient treatment program could accurately predict abstinence or relapse at 3-month followup in 80 percent of alcoholic men. We used sleep variables after 1 month of abstinence to predict outcome at 6 months and found that eventual relapsers had a higher proportion of REM sleep and a lower proportion of slow wave sleep (Aldrich et al. 1994). After 3 months of abstinence, outcome can still be predicted to some degree based on sleep findings, although sleep abnormalities have less predictive power than sleep changes during the first month of rehabilitation, perhaps because sleep has improved to the extent that other factors are more prominent risk factors for relapse (Drummond et al. 1996). Although these data are promising, it is unknown whether a

prediction of relapse based on sleep findings is more accurate than a prediction based on clinical data.

SUMMARY

Alcohol has a number of effects on sleep. After an initial stimulating effect, it has a sedating effect that can reduce the time required to fall asleep. The reduced time to sleep onset may contribute to use of alcohol by insomniacs. However, with low to moderate doses of alcohol, there is often increased sleep disruption toward the end of the night. Elderly persons, who may already have sleep disturbance or who may have sleep that is easily interrupted, are probably particularly susceptible to sleep-disrupting effects of alcohol. High doses of alcohol suppress REM sleep, and when alcohol use is discontinued, there is a "rebound" increase in the amount and intensity of REM sleep. Daytime alcohol use leads to daytime sleepiness, particularly in persons who are already sleepy as a result of partial sleep deprivation. Alcohol facilitates the occurrence of obstructive apneas during sleep by relaxing upper airway dilator muscles and depressing the central nervous system response to airway occlusion. As a result, alcohol use in the evening is associated with an increased risk of OSA, increased severity of hypoxemia in individuals who already have OSA, and increased risk for functional consequences of disrupted sleep, such as sleep-related motor vehicle accidents.

Alcoholics often have poor sleep during periods of abstinence, during episodes of heavy drinking, and during withdrawal from alcohol. Sleep disturbance, with increased pressure for REM sleep and decreased amounts of slow wave sleep, may contribute to relapse or may be a marker for relapse potential, or both. Better understanding of the causes of sleep disturbance with alcohol use and in alcoholics may provide clues to the pathogenesis of alcoholism and to the reasons for relapse in treated alcoholics.

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REFERENCES

Adamson, J., and Burdick, J.A. Sleep of dry alcoholics. *Arch Gen Psychiatry* 28:146–149, 1973.

Aldrich, M.S. Automobile accidents in patients with sleep disorders. *Sleep* 12:487–494, 1989.

Aldrich, M.S., and Chervin, R.D. Alcohol use, obstructive sleep apnea, and sleep-related motor vehicle accidents. *Sleep Res* 26:308, 1997.

Aldrich, M.S., and Shipley, J.E. Alcohol use and periodic limb movements of sleep. *Alcohol Clin Exp Res* 17:192–196, 1993.

Aldrich, M.S.; Shipley, J.E.; Tandon, R.; Kroll, P.D.; and Brower, K.J. Sleep disordered breathing in alcoholics: Association with age. *Alcohol Clin Exp Res* 17:1179–1183, 1993.

Aldrich, M.S.; O'Neal, E.A.; Eiser, A.S.; Kroll, P.; Brower, K.; and Shipley, J.E. Slow wave sleep decrement and relapse tendency in alcoholics in treatment. *Sleep Res* 23:185, 1994.

Aldrich, M.S.; O'Neal, E.A.; and Shipley, J.E. Sleep and low doses of alcohol: Age and gender effects. *Sleep Res* 24:67, 1995.

Allen, R.P.; Wagman, A.M.; Funderburk, F.R.; and Wells, D.T. Slow wave sleep: A predictor of individual differences in response to drinking? *Biol Psychiatry* 15:345–348, 1980.

Bassetti, C., and Aldrich, M.S. Alcohol consumption and sleep apnea in patients with TIA and ischemic stroke. *Sleep Res* 25:400, 1996.

Berry, R.B.; Bonnet, M.H.; and Light, R.W. Effect of ethanol on the arousal response to airway occlusion during sleep in normal subjects. *Am Rev Respir Dis* 145:445–452, 1992.

Block, A.J. Alcohol ingestion does not cause sleep-disordered breathing in premenopausal women. *Alcohol Clin Exp Res* 8:397–398, 1984.

Block, A.J., and Hellard, D.W. Ingestion of either scotch or vodka induces equal effects on sleep and breathing of asymptomatic subjects. *Arch Intern Med* 147:1145–1147, 1987.

Block, A.J.; Hellard, D.W.; and Slayton, P.C. Effect of alcohol ingestion on breathing and oxygenation during sleep. Analysis of the influence of age and sex. *Am J Med* 80:595–600, 1986.

Carskadon, M.A.; Dement, W.C.; Mitler, M.M.; Roth, T.; Westbrook, P.R.; and Keenan, S. Guidelines for the multiple sleep latency test (MSLT): A standard measure of sleepiness. *Sleep* 9:519–524, 1986.

Clark, C.P.; Gillin, J.C.; Golshan, S.; Demodena, A.; Smith, T.L.; Danowski, S.; Irwin, M.; and Schuckit, M. The relationship of sleep abnormalities to short-term sobriety in primary alcoholics with secondary depression. *Sleep Res* 25:155, 1996.

Collop, N.A. Medroxyprogesterone acetate and ethanol-induced exacerbation of obstructive sleep apnea. *Chest* 106: 792–799, 1994.

Dawson, A.; Lehr, P.; Bigby, B.G.; and Mitler, M.M. Effect of bedtime ethanol on total inspiratory resistance and respiratory drive in normal nonsnoring men. *Alcohol Clin Exp Res* 17:256–262, 1993.

Dijk, D.J.; Brunner, D.P.; Aeschbach, D.; Tobler, I.; and Borbely, A.A. The effects of ethanol on human sleep EEG power spectra differ from those of benzodiazepine receptor agonists. *Neuropsychopharmacology* 7:225–232, 1992.

Dolly, F.R., and Block, A.J. Increased ventricular ectopy and sleep apnea following ethanol ingestion in COPD patients. *Chest* 83:469–472, 1983.

Drummond, S.P.A.; Gillin, J.C.; Irwin, M.; Smith, T.; Golshon, S.; Demodena, A.; Danowski, S.; and Schuckit, M. Sleep changes in patients with primary alcoholism after one year of abstinence. *Sleep Res* 24:159, 1995.

Drummond, S.P.A.; Gillin, J.C.; Smith, T.; Demodena, A.; and Schuckit, M. Can sleep after 3-months of abstinence predict treatment outcome at 1-year in patients with pure primary alcoholism? *Sleep Res* 25:159, 1996.

Easton, P.A.; West, P.; Meatherall, R.C.; Brewster, J.F.; Lertzman, M.; and Kryger, M.H. The effect of excessive ethanol ingestion on sleep in severe chronic ob-

structive pulmonary disease. *Sleep* 10:224–233, 1987.

Findley, L.; Unverzagt, M.E.; and Suratt, P.M. Automobile accidents involving patients with obstructive sleep apnea. *Am Rev Respir Dis* 138:337–340, 1988.

Findley, L.; Unverzagt, M.; Guchu, R.; Fabrizio, M.; Buckner, J.; and Suratt, P. Vigilance and automobile accidents in patients with sleep apnea or narcolepsy. *Chest* 108:619–624, 1995.

Fletcher, E.C.; DeBehnke, R.D.; Lavoi, M.S.; and Gorin, A.B. Undiagnosed sleep apnea in patients with essential hypertension. *Ann Intern Med* 103:190–194, 1985.

George, C.F.; Nickerson, P.W.; Hanly, P.J.; Millar, T.W.; and Kryger, M.H. Sleep apnoea patients have more automobile accidents. *Lancet* 2:447, 1987.

Gillin, J.C.; Smith, T.L.; Irwin, M.; Kripke, D.F.; and Schuckit, M. EEG sleep studies in "pure" primary alcoholism during subacute withdrawal: Relationships to normal controls, age, and other clinical variables. *Biol Psychiatry* 27:477–488, 1990.

Gillin, J.C.; Smith, T.L.; Irwin, M.; Butters, N.; Demodena, A.; and Schuckit, M. Increased pressure for rapid eye movement sleep at time of hospital admission predicts relapse in nondepressed patients with primary alcoholism at 3-month follow-up. *Arch Gen Psychiatry* 51:189–197, 1994.

Gresham, S.C.; Webb, W.B.; and Williams, R.C. Alcohol and caffeine: Effect on inferred visual dreaming. *Science* 140:1226–1227, 1963.

Gross, M.M., and Best, S. Behavioral concomitants of the relationship between baseline slow wave sleep and carry-over of tolerance and dependence in alcoholics. *Adv Exp Med Biol* 59:633–643, 1975.

He, J.; Kryger, M.H.; Zorick, F.J.; Conway, W.; and Roth, T. Mortality and apnea index in obstructive sleep apnea. Experience in 385 male patients. *Chest* 94:9–14, 1988.

Hung, J.; Whitford, E.G.; Parsons, R.W.; and Hillman, D.R. Association of sleep apnoea with myocardial infarction in men. *Lancet* 336:261–264, 1990.

Issa, F.G., and Sullivan, C.E. Alcohol, snoring and sleep apnoea. *J Neurol Neurosurg Psychiatry* 45:353–359, 1982.

Jennum, P., and Borgesen, S.E. Intracranial pressure and obstructive sleep apnea. *Chest* 95:279–283, 1989.

Johnson, L.C.; Burdick, J.A.; and Smith, J. Sleep during alcohol intake and withdrawal in the chronic alcoholic. *Arch Gen Psychiatry* 22:406–418, 1970.

Kales, A.; Bixler, E.O.; Cadieux, R.J.; Schneck, D.W.; Shaw, L.C., III; Locke, T.W.; Vela-Bueno, A.; and Soldatos, C.R. Sleep apnoea in a hypertensive population. *Lancet* 2:1005–1008, 1984.

Knowles, J.B.; Laverty, S.G.; and Kuechler, H.A.; Effects of alcohol on REM sleep. *Q J Stud Alcohol* 29:342–349, 1968.

Koskenvuo, M.; Kaprio, J.; Telakivi, T.; Partinen, M.; Heikkila, K.; and Sarna, S. Snoring as a risk factor for ischeamic heart disease and stroke in men. *Br Med J* 294:16–19, 1987.

Kotorii, T.; Nakazawa, Y.; Yokoyama, T.; Kurauchi, H.; Sakurada, H.; Ohkawa, T.; Nonaka, K.; Hasuzawa, H.; Dainoson, K.; and Inanaga, K. The sleep pattern of chronic alcoholics during the alcohol withdrawal period. *Folia Psychiatr Neurol Jpn* 34:89–95, 1980.

Krull, K.R.; Smith, L.T.; Sinha, R.; and Parsons, O.A. Simple reaction time event-related potentials: Effects of alcohol and sleep deprivation. *Alcohol Clin Exp Res* 17:771–777, 1993.

Lavie, P.; Ben-Yosef, R.; and Rubin, A.E. Prevalence of sleep apnea among patients with essential hypertension. *Am Heart J* 108:373–376, 1984.

Lester, B.K.; Rundell, O.H.; Cowden, L.C.; and Williams, H.L. Chronic alcoholism, alcohol and sleep. *Adv Med Exp Biol* 35:261–279, 1973.

Lumley, M.; Roehrs, T.; Asker, D.; Zorick, F.; and Roth, T. Ethanol and caffeine effects on daytime sleepiness/alertness. *Sleep* 10:306–312, 1987.

MacLean, A.W., and Cairns, J. Dose-response effects of ethanol on the sleep of young men. *J Stud Alcohol* 43:434–444, 1982.

Mitler, M.M.; Dawson, A.; Henriksen, S.J.; Sobers, M.; and Bloom, F.E. Bedtime ethanol increases resistance of upper airways and produces sleep apneas in asymptomatic snorers. *Alcohol Clin Exp Res* 12:801–805, 1988.

O'Boyle, D.J.; Van, F.; and Hume, K.I. Effects of alcohol, at two times of day, on EEG-derived indices of physiological arousal. *Electroencephalogr Clin Neurophysiol* 95:97–107, 1995.

Olson, L.G.; King, M.T.; Hensley, M.J.; and Saunders, N.A. A community study of snoring and sleep-disordered breathing. Health outcomes. *Am J Respir Crit Care Med* 152:717–720, 1995.

Palomaki, H. Snoring and the risk of ischemic brain infarction. *Stroke* 22:1021–1025, 1991.

Partinen, M., and Guilleminault, C. Daytime sleepiness and vascular morbidity at seven-year follow-up in obstructive sleep apnea patients. *Chest* 97:27–32, 1990 .

Petrucelli, N.; Roehrs, T.A.; Wittig, R.M.; and Roth, T. The biphasic effects of ethanol on sleep latency. *Sleep Res* 23:75, 1994.

Prinz, P.N.; Roehrs, T.A.; Vitaliano, P.P.; Linnoila, M.; and Weitzmann, E.D. Effect of alcohol on sleep and nighttime plasma growth hormone and cortisol concentrations. *J Clin Endocrinol Metab* 51:759–764, 1980.

Rechtschaffen, A.; Bergmann, B.M.; Everson, C.A.; Kushida, C.A.; and Gilliland, M.A. Sleep deprivation in the rat: X. Integration and discussion of the findings. *Sleep* 12:68–87, 1989.

Robinson, R.W.; White, D.P.; and Zwillich, C.W. Moderate alcohol ingestion increases upper airway resistance in normal subjects. *Am Rev Respir Dis* 132:1238–1241, 1985.

Roehrs, T.; Zwyghuizen-Doorenbos, A.; Timms, V.; Zorick, F.; and Roth, T. Sleep extension, enhanced alertness and the sedating effects of ethanol. *Pharmacol Biochem Behav* 34:321–324, 1989.

Roehrs, T.; Zwyghuizen-Doorenbos, A.; Knox, M.; Moskowitz, H.; and Roth, T. Sedating effects of ethanol and time of drinking. *Alcohol Clin Exp Res* 16:553–557, 1992.

Roehrs, T.; Zwyghuizen-Doorenbos, A.; and Roth, T. Sedative effects and plasma concentrations following single doses of triazolam, diphenhydramine, ethanol and placebo. *Sleep* 16:301–305, 1993.

Roehrs, T.; Beare, D.; Zorick, F.; and Roth, T. Sleepiness and ethanol effects on simulated driving. *Alcohol Clin Exp Res* 18:154–158, 1994*a*.

Roehrs, T.; Claiborue, D.; Knox, M.; and Roth, T. Residual sedating effects of

ethanol. *Alcohol Clin Exp Res* 18:831–834, 1994*b*.

Rouhani, S.; Tran, G.; Leplaideur, F.; Durlach, J.; and Poenaru, S. EEG effects of a single low dose of ethanol on afternoon sleep in the nonalcohol-dependent adult. *Alcohol* 6:87–90, 1989.

Rundell, O.H.; Lester, B.K.; Griffiths, W.J.; and Williams, H.L.; Alcohol and sleep in young adults. *Psychopharmacologia* 26:201–218, 1972.

Schenk, C.H.; Bundlie, S.R.; Patterson, A.L.; and Mahowald, M.W. Rapid eye movement sleep behavior disorder. A treatable parasomnia affecting older adults. *JAMA* 257:1786–1789, 1987.

Scrima, L.; Broudy, M.; Nay, K.N.; and Cohn, M.A. Increased severity of obstructive sleep apnea after bedtime alcohol ingestion: Diagnostic potential and proposed mechanism of action. *Sleep* 5:318–328, 1982.

Scrima, L.; Hartman, P.G.; and Hiller, F.C. Effect of three alcohol doses on breathing during sleep in 30-49 year old nonobese snorers and nonsnorers. *Alcohol Clin Exp Res* 13:420–427, 1989.

Seppala, T.; Partinen, M.; Penttila, A.; Aspholm, R.; Tiainen, E.; and Kaukianen, A. Sudden death and sleeping history among Finnish men. *J Intern Med* 229:23–28, 1991.

Siebler, M.; Daffertshofer, M.; Hennerici, M.; and Freund, H-J. Cerebral blood flow alterations during obstructive sleep apnea syndrome. *Neurology* 40:1461–1462, 1990.

Skoloda, T.E.; Alterman, A.I.; and Gottheil, E. Sleep quality reported by drinking and non-drinking alcoholics. In: *Addiction Research and Treatment: Converging Trends: Proceedings of the First Annual Coatesville-Jefferson Conference on Addiction.* New York: Pergamon Press, 1979. pp. 102–112.

Smirne, S.; Palazzi, S.; Zucconi, M.; Chierchia, S.; and Ferini-Strambi, L. Habitual snoring as a risk factor for acute vascular disease. *Eur Respir J* 6:1357–1361, 1993.

Stone, B.M. Sleep and low doses of alcohol. *Electroencephalogr Clin Neurophysiol* 48:706–709, 1980.

Taasen, V.C.; Block, A.J.; Boysen, P.G.; Wynne, J.W.; White, C.; and Lindsey, S. Alcohol increases sleep apnea and oxygen desaturation in asymptomatic men. *Am J Med* 71:240–245, 1981.

Tachibana, M.; Tanaka, K.; Hishikawa, Y.; and Kaneko, Z. A sleep study of acute psychotic states due to alcohol and meprobamate addiction. *Adv Sleep Res* 2:177–205, 1977.

Thompson, P.M.; Gillin, J.C.; Golshan, S.; and Irwin, M. Polygraphic sleep measures differentiate alcoholics and stimulant abusers during short-term abstinence. *Biol Psychiatry* 38:831–836, 1995.

Van, F.; O'Boyle, D.J.; and Hume, K.I. Effects of alcohol on the sleep-stage structure of a nap in the afternoon. *Biol Psychol* 41:55–59, 1995.

Williams, A.J.; Houston, D.; Finberg, S.; Lam, C.; Kinney, J.L.; and Santiago, S. Sleep apnea syndrome and essential hypertension. *Am J Cardiol* 55:1019–1022, 1985.

Williams, D.L.; MacLean, A.W.; and Cairns, J. Dose-response effects of ethanol on the sleep of young women. *J Stud Alcohol* 44:515–523, 1983.

Williams, H.L., and Rundell, O.H., Jr. Altered sleep physiology in chronic alcoholics: Reversal with abstinence. *Alcohol Clin Exp Res* 5:318–325, 1981.

Young, T.; Palta, M.; Dempsey, J.; Skatrud, J.; Weber, S.; and Badr, S. The occurrence of sleep disordered breathing among middle-aged adults. *N Engl J Med* 328:1230–1235, 1993.

Yules, R.B.; Freedman, D.X.; and Chandler, K.A. The effect of ethyl alcohol on man's electroencephalographic sleep cycle. *Electroencephalogr Clin Neurophysiol* 20:109–111, 1966.

Yules, R.B.; Lippman, M.E.; and Freedman, D.X. Alcohol administration prior to sleep. The effect on EEG sleep stages. *Arch Gen Psychiatry* 16:94–97, 1967.

Zarcone, V.; Schreier, L.; Barchas, J.; Orenberg, E.; and Benson, K. Alcohol, sleep and cerebrospinal fluid changes in alcoholics: Cyclic AMP and biogenic amine metabolites in CSF. *Adv Exp Med Biol* 85A:593–599, 1977.

Zarcone, V.P., Jr.; Schreier, L.; Mitchell, G.; Orenberg, E.; and Barchas, J. Sleep variables, cyclic AMP and biogenic amine metabolites after one day of ethanol ingestion. *J Stud Alcohol* 41:318–324, 1980.

Zwyghuizen-Doorenbos, A.; Roehrs, T.; Lamphere, J.; Zorick, F.; and Roth, T. Increased daytime sleepiness enhances ethanol's sedative effects. *Neuropsychopharmacology* 1:279–286, 1988.

Zwyghuizen-Doorenbos, A.; Roehrs, T.; Timms, V.; and Roth, T. Individual differences in the sedating effects of ethanol. *Alcohol Clin Exp Res* 14:400–404, 1990.

Chapter 17

Alcohol, Aging, and Driving

Patricia F. Waller, Ph.D.

In our society, mobility is the key to access to all that is required to achieve and maintain our full potential—education, employment, health care, and maintenance of ties with family and friends. Mobility is strongly associated with the well-being of the elderly (Carp 1988), and lack of mobility may be related to a variety of problems (e.g., medical, psychological, financial) in this population. In a major study in California, when other important factors were taken into consideration, social isolation was the most powerful predictor of death from all causes over a 9-year period for older persons (Kaplan 1995). Clearly, for most people in our society, transportation is a major factor in access to social contact.

For most elderly persons the driver's license represents independence. Furthermore, the elderly in our society are increasingly dependent on the private vehicle (Rosenbloom 1994) because alternative modes of transportation (e.g., buses, taxis, and rides from family and friends) may not be available or flexible enough to meet their needs.

The research reported in this chapter should be of interest to those who recognize the importance of enabling as many older persons as possible to continue to meet their own transportation needs.

AGING AND DRIVING

Licensure Rate

The age distribution of the population is changing, with older drivers representing the most rapidly growing segment of the driving population. At a somewhat higher rate than the rest of the population, the elderly are increasing their rate of licensure (figure 1) (Massie

P.F. Waller, Ph.D., is director of the Transportation Research Institute, professor in the Department of Health Management and Policy, adjunct professor in the Department of Psychology, and senior research associate in the Alcohol Research Center, University of Michigan, 2901 Baxter Rd., Ann Arbor, MI 48109–2150.

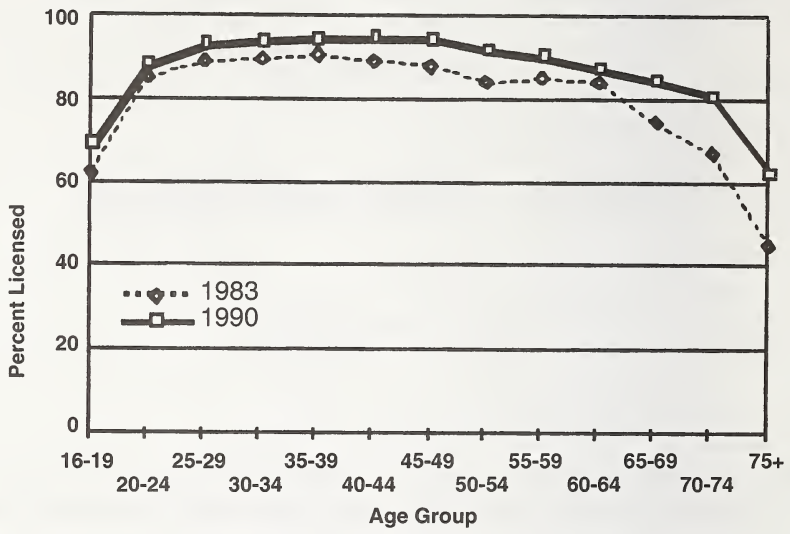

Figure 1. Proportion of population licensed, by age, 1983 vs. 1990, from Nationwide Personal Transportation Surveys. Reprinted from Massie, D.L., and Campbell, K.L. *Analysis of Accident Rates by Age, Gender, and Time of Day Based on the 1990 Nationwide Personal Transportation Survey.* Ann Arbor, MI: University of Michigan Transportation Research Institute, 1993.

and Campbell 1993). The greatest changes are occurring for women. As shown in figure 2, until their midforties women obtain licenses at almost the same rate as men (Massie and Campbell 1993). The lower licensure rates in 1990 for women at the older ages are a cohort effect, but as the current cohort of drivers age, women will continue to hold licenses at almost the same rate as men.

Mileage Driven

The elderly are also increasing their mileage driven. Between 1983 and 1990, women age 75 and over almost doubled their mileage, from an average of 2,889 to 5,411 miles annually. Although men still drive more than women, women are increasing their proportion of the total mileage driven. Figures 3 and 4 illustrate these relationships (Massie and Campbell 1993). Indeed, women account for the major changes in travel behavior over the past 20 years (Pisarski 1992).

AGING AND CRASH RISK

Crash Risk per Licensed Driver Versus Crash Risk per Miles Driven

Older drivers, as a group, have low crash rates based on number of licensed drivers. Even though current older drivers drive more miles than did previous older driver cohorts, their mileage is still much lower than that of younger drivers.

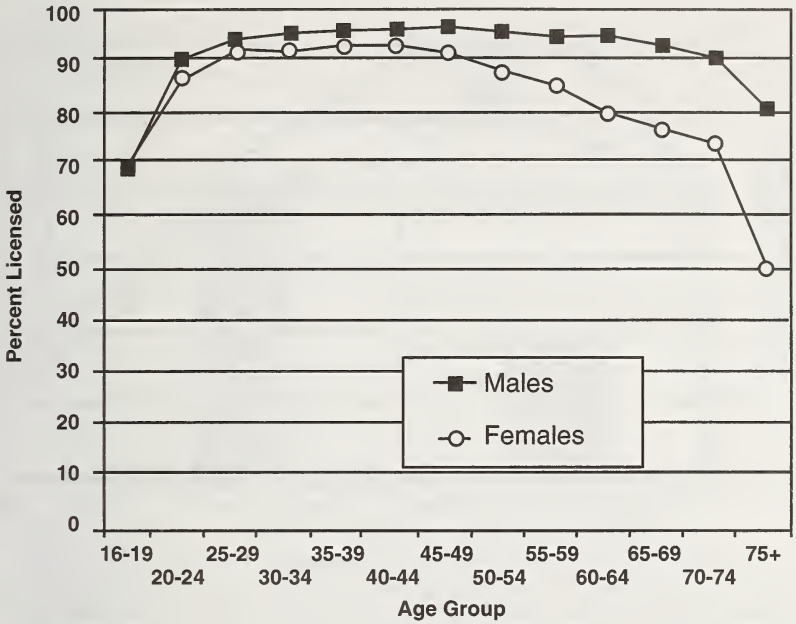

Figure 2. Percent of population holding driver license in 1990, by age and gender, from Nationwide Personal Transportation Surveys. Reprinted from Massie, D.L., and Campbell, K.L. *Analysis of Accident Rates by Age, Gender, and Time of Day Based on the 1990 Nationwide Personal Transportation Survey.* Ann Arbor, MI: University of Michigan Transportation Research Institute, 1993.

Furthermore, older drivers, as a group, tend to restrict themselves, driving only at the safest times and places and when they feel reasonably well. Even so, the data are very clear that with increasing age, the crash risk per mile driven increases (Brainin 1980; Cerrelli 1989; Williams and Carsten 1989). This increase in risk begins in the late fifties and rises at an accelerating rate. Figure 5 illustrates this relationship (Peck and Romanowicz 1993/94). The very real losses in physical and cognitive functioning that accompany increasing age cannot be denied.

This elevated crash risk with increasing age occurs despite the fact that most older drivers restrict their driving. Drivers who must meet a schedule, such as commercial drivers, do not enjoy the luxury of controlling when and where they drive. While the data are limited, it appears that for these drivers the elevation in risk begins earlier and rises more steeply (Hull and Knebel 1968). Figure 6 shows the crash risk per million miles for female school bus drivers (Promisel et al. 1969). The findings are similar for male school bus drivers,

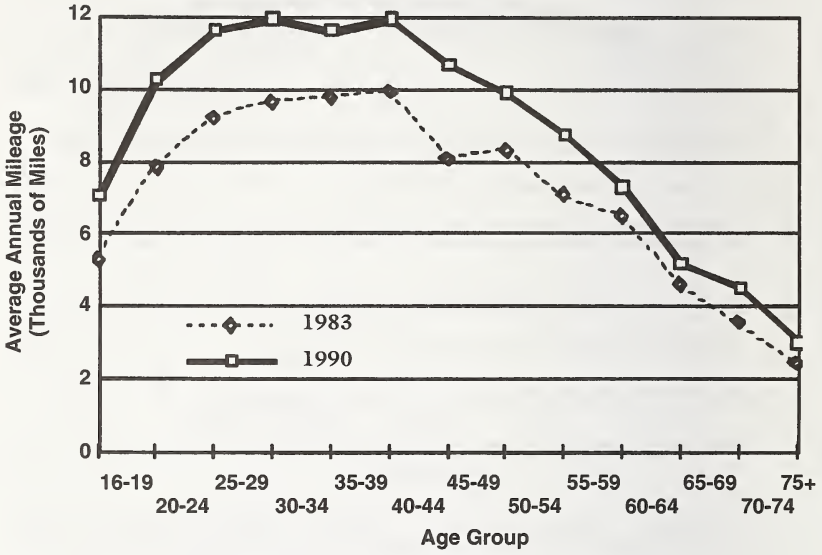

Figure 3. Average annual mileage by driver age, 1983 vs. 1990, from Nationwide Personal Transportation Surveys. Reprinted from Massie, D.L., and Campbell, K.L. *Analysis of Accident Rates by Age, Gender, and Time of Day Based on the 1990 Nationwide Personal Transportation Survey.* Ann Arbor, MI: University of Michigan Transportation Research Institute, 1993.

although they show greater variation at the younger ages.

Risk of Injury

With increasing age, the human body becomes less tolerant to impact. A measure of vehicle crush, the Traffic Accident Damage (TAD) Scale (National Safety Council 1984), has been found to be a strong correlate of driver injury. The scale ranges from 0 (no vehicle damage) to 7 (total crush). When this scale is taken into consideration, older drivers are at much greater risk of fatal injury (figure 7) (Massie and Campbell 1993). This greater vulnerability of older persons to injury from a given impact has been clearly demonstrated (Evans 1988, 1991).

ALCOHOL AND CRASH RISK

The Grand Rapids Study

It is well established that alcohol increases the risk of causing a crash. This relationship was clearly shown in a landmark study conducted in Grand Rapids by Borkenstein and his colleagues (1964) in which breath alcohol measures were obtained from both crash-involved drivers and other drivers at the same times and locations. Figure 8 shows the relationship between blood alcohol concentration

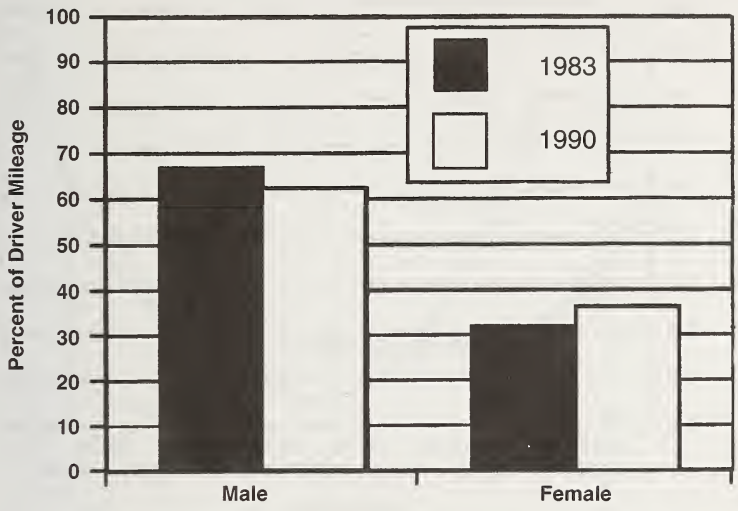

Figure 4. Proportion of annual mileage accumulated by gender, 1983 vs. 1990, from Nationwide Personal Transportation Surveys. Reprinted from Massie, D.L., and Campbell, K.L. *Analysis of Accident Rates by Age, Gender, and Time of Day Based on the 1990 Nationwide Personal Transportation Survey.* Ann Arbor, MI: University of Michigan Transportation Research Institute, 1993.

(BAC) and relative risk of causing a crash.

It may be noted that there is a slight dip in the curve around 0.02 percent BAC. This dip, which has been referred to as the "Grand Rapids Dip," led some to believe that a little alcohol improved driving performance. However, the dip is an artifact of the characteristics of drivers who drink often (but not heavily) and who, for other reasons, are better-than-average drivers. Carefully controlled studies have shown that impairment in performance begins at levels even lower than 0.02 percent BAC (Attwood 1978; Attwood et al. 1980; Moskowitz et al. 1985; Moskowitz and Robinson 1988).

Gender Differences

Data from the Grand Rapids Study showed that women experienced a higher risk of crash at a given BAC than did men (Borkenstein et al. 1964). At the time this relationship was attributed to the relative inexperience of women in driving compared with men. However, other studies have confirmed gender differences in crash risk. In 1972, Carlson reported gender differences in risk of nighttime crashes. More recently, Zador (1991) compared risk of fatal single-vehicle crash (where culpability is generally attributed to the driver) with roadside survey data providing information on alcohol levels in the general driving population. Compared with zero

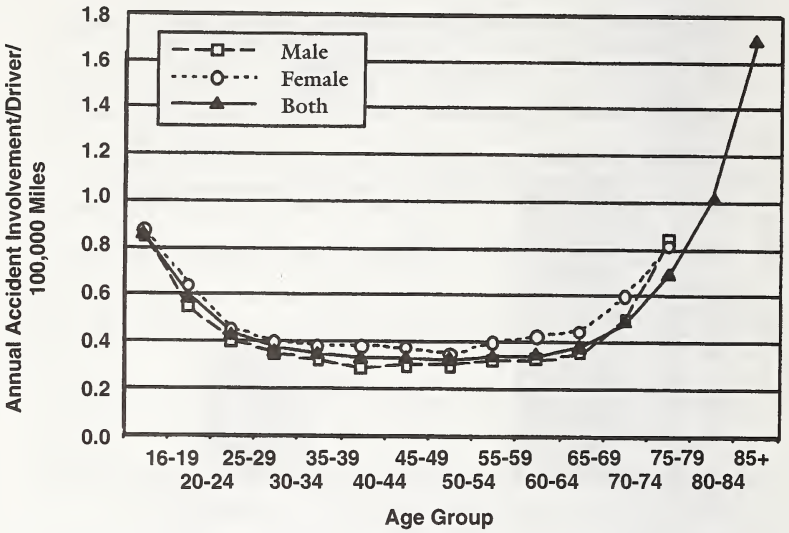

Figure 5. Annual crash rates based on mileage driven, by age and gender. Reprinted from Peck, R., and Romanowicz, P. Teen and senior drivers. *Research Notes* (California Department of Motor Vehicles, Sacramento), Winter 1993/94:3–6.

alcohol, for BACs in the 0.05 to 0.09 percent range (still legal in most states), young men ages 16 through 20 had a relative risk of about 18 of being in a single-vehicle fatal crash; that is, they were 18 times as likely to be so involved. For women the same age, the relative risk was over 54. From ages 21 through 24, the figures were about 12 for men and 35 for women. Above age 24, the corresponding figures were between 8 and 9 for men and over 25 for women. Figure 9 shows these relationships. It is clear that, while relative risk goes up for both men and women and for all ages, the gender differences remain striking.

Other laboratory-based studies, examining behaviors unrelated to driving, have also indicated there may be gender differences in the extent to which alcohol impairs performance (Waller and Blow 1995). One study used simulated traffic signs and measured the ability to detect the presence or absence of a sign. Females were found to be more affected by alcohol than males, and the author attributed the findings to gender-based differential effects of alcohol on vision (Avant 1990).

ALCOHOL, AGING, AND DRIVING PERFORMANCE

Alcohol and Aging

Alcohol use appears to decline with age. Tables 1 and 2 show drinking

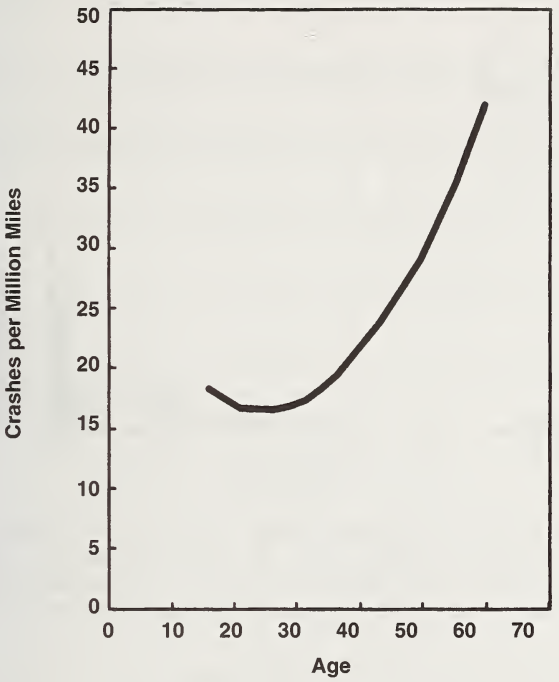

Figure 6. Crash risk per million miles by driver age for female school bus drivers. Reprinted from Promisel, D.M.; Blomberg, R.D.; Nacht, M.L.; and Silver, S. *School Bus Safety—Operator Age in Relation to School Bus Accidents.* Washington, DC: National Highway Safety Bureau, 1969.

categories for men and women in 1983 and 1988. Abstainers included three groups. First were lifetime abstainers, who had consumed fewer than 12 drinks in a lifetime. Second were former drinkers (12 or more drinks in 1 or more years, but no drink in the past year), and third were infrequent drinkers (those who drank less than 0.01 oz daily in the past year). Drinkers were classified into three groups: light drinkers, who consumed about 1–13 drinks per month; moderate drinkers, who consumed about 4–13 drinks per week; and heavy drinkers, who consumed about 2 or more drinks per day or 14 or more drinks per week. In these tables, it should be noted that the percentages for drinking categories are based on the drinking population only. For example, in table 1, in 1988, of the 18- to 29-year-old men who drank, 42 percent were light drinkers (National Institute for Alcohol Abuse and Alcoholism [NIAAA] 1994).

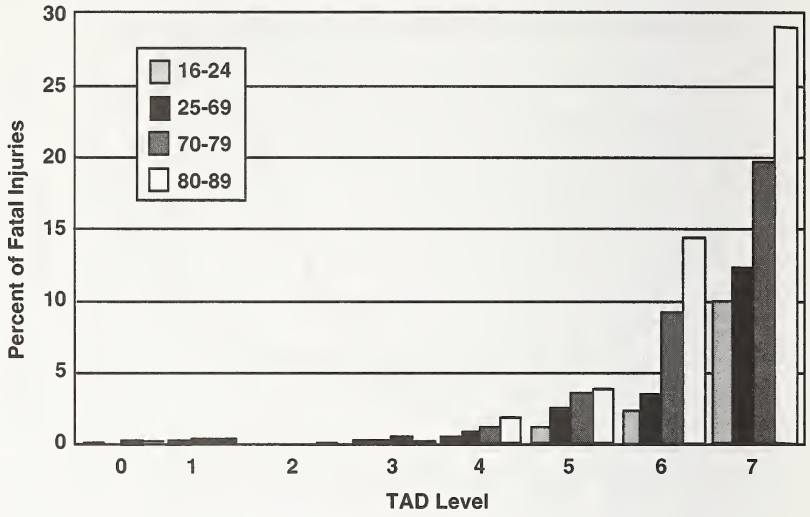

Figure 7. Proportion of fatal injuries by driver age, by crash severity (Traffic Accident Damage [TAD] Scale). Reprinted from Massie, D.L., and Campbell, K.L. *Analysis of Accident Rates by Age, Gender, and Time of Day Based on the 1990 Nationwide Personal Transportation Survey.* Ann Arbor, MI: University of Michigan Transportation Research Institute, 1993.

From 1983 to 1988, the abstinence rates for adult men increased from 28 percent to 32 percent, while for women the increase was from 50 percent to 53 percent. Higher rates were reported for those age 65 and older, with men in this age group increasing from 47 percent to 51 percent, and women from 71 percent to 75 percent. Even so, of those who do report drinking, in 1988 19 percent of the men and 10 percent of the women were heavy drinkers. In fact, for both men and women, when only drinkers were considered, the elderly had among the highest proportions of heavy drinkers. In a meta-analysis of longitudinal studies, there was little evidence that, for those who continued drinking, amount of alcohol consumed per drinking occasion declined. There was even some evidence that frequency of drinking may increase with increasing age (Fillmore et al. 1991).

Aging and Alcohol-Related Fatal Crashes

Alcohol-related fatal crashes have dropped as a proportion of all fatal crashes, and this decrease has occurred for all age groups. Figure 10 shows the percent of fatally injured drivers who tested positive for any alcohol, by driver age, from 1982 through 1994, based on figures from the National Highway Traffic Safety Administration (1995). It can be seen that the older age cohorts have lower percentages of alcohol-related fatal crashes, and, like other age cohorts, their rates have dropped over time.

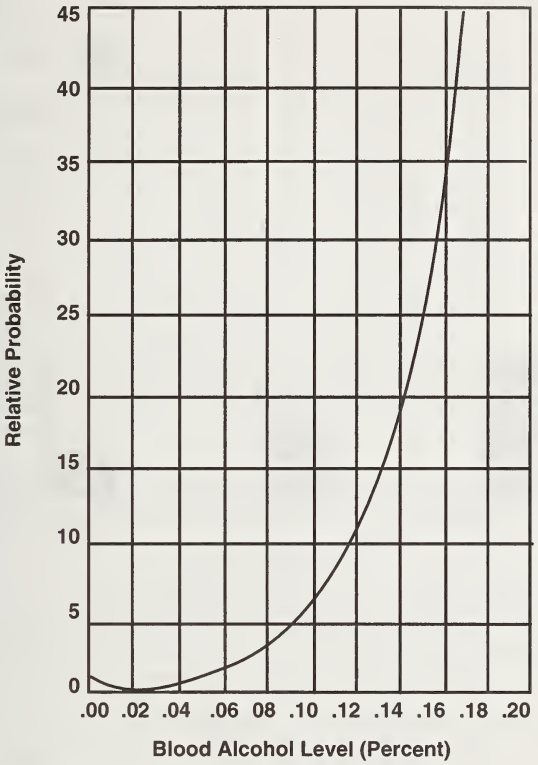

Figure 8. Relative risk of causing a crash as a function of blood alcohol level. Reprinted from Borkenstein, R.F.; Crowther, R.F.; Shumate, R.P.; Ziel, W.B.; and Zylman, R. The role of the drinking driver in traffic accidents (the Grand Rapids Study). *Blutalkohol* 11 (Suppl 1):1–131, 1974.

Alcohol Effects on Older Driver Performance

Alcohol, even at low levels, affects driving performance, but it appears to have greater impairment for older persons. This finding came from the Grand Rapids Study (Borkenstein et al. 1964), but it has not been carefully investigated. Because performance deteriorates with age independent of alcohol, the alcohol effects must be separated from those occurring otherwise.

Older driver crashes are not characterized by alcohol use. Alcohol-related crashes of all levels of severity have dropped for all age groups, including the elderly. Even so, in 1994, almost 11 percent of drivers ages 65–74 in fatal crashes tested positive for alcohol. Almost three-fourths of these, or about 8 percent of fatal crashes for this age group, were at or above 0.10 percent BAC, the legal limit in most states. Some states have implemented even

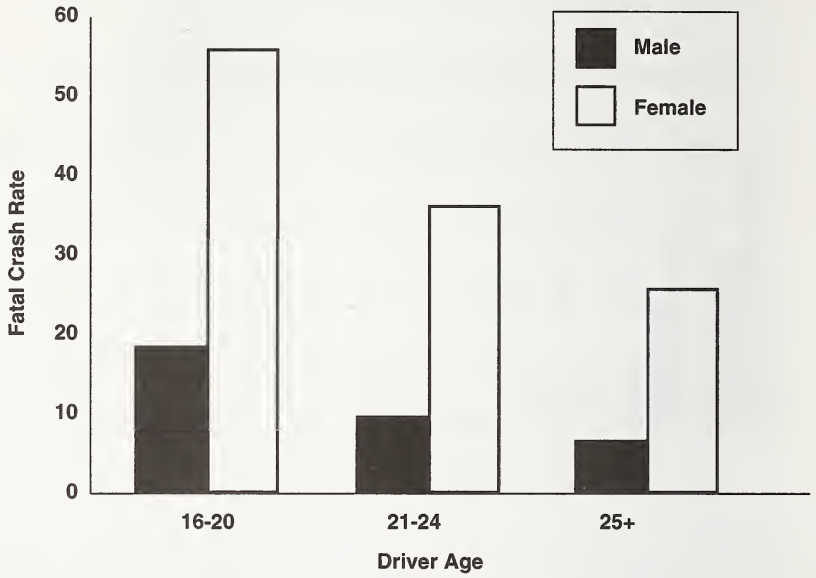

Figure 9. Relative risk of fatal single-vehicle crash at blood alcohol concentration of 0.05 to 0.09 percent compared with zero alcohol. Data from Zador 1991.

lower BAC limits. For drivers age 75 and older, almost 5 percent tested positive for any alcohol, and over three-fifths of these were at or above 0.10 percent BAC (National Highway Traffic Safety Administration 1995).

These proportions are relatively low compared with the driving population as a whole. In 1994, of all male drivers in fatal crashes, 28.5 percent tested positive for any alcohol, compared with 15.2 percent of female drivers in fatal crashes. Corresponding rates for the higher BAC, 0.10 percent or higher, were 21.9 percent for all men and 11.1 percent for all women drivers in fatal crashes (National Highway Traffic Safety Administration 1995). It can be seen that the lower rates of alcohol usage reported for women in the general population are reflected in their lower alcohol-related fatal crash experience.

ALCOHOL AND INJURY

Alcohol has long been associated with injury. It increases injury in at least three ways, namely, by impairing judgment, by impairing psychomotor performance, and by increasing the extent of injury experienced from a given crash.

Judgment

Alcohol may modify the judgment made of a situation, so that one is more likely to engage in high-risk behavior when under the influence of alcohol. There may be gender differences in this phenomenon. When women who

Table 1. Prevalence of Abstinence and Drinking Levels Among Men, by Age Group, 1983 and 1988.

	1983 Drinking Levels (%)				1988 Drinking Levels (%)			
Age	A	L	M	H	A	L	M	H
18–29	22	37	42	21	25*	42	40	18
30–44	22	40	40	21	25*	44	39	17**
45–64	30	41	34	25	36*	45	34	21**
65+	47	45	31	24	51	50	31	19
Total	28	40	38	22	32	44	37	19**

Note: For drinking levels, A = abstainers, L = light drinkers, M = moderate drinkers, and H = heavy drinkers.
*Percentage of abstainers in 1988 is significantly higher ($p < 0.05$) than in 1983.
** Percentage of heavy drinkers is significantly lower ($p < 0.05$) in 1988 than in 1983.
Source: Adapted from National Institute on Alcohol Abuse and Alcoholism. Eighth Special Report to the U.S. Congress on Alcohol and Health. NIH Pub. No. 94–3699. Bethesda. MD: National Institutes of Health, 1994.

Table 2. Prevalence of Abstinence and Drinking Levels Among Women, by Age Group, 1983 and 1988.

	1983 Drinking Levels (%)				1988 Drinking Levels (%)			
Age	A	L	M	H	A	L	M	H
18–29	39	59	33	8	43*	64	30	6**
30–44	43	63	30	8	45	65	29	6**
45–64	54	61	30	10	58*	64	27	9
65+	71	63	27	10	75*	62	29	10
Total	50	61	30	9	53*	64	29	7**

Note: For drinking levels, A = abstainers, L = light drinkers, M = moderate drinkers, and H = heavy drinkers.
*Percentage of abstainers in 1988 is significantly higher ($p < 0.05$) than in 1983.
**Percentage of heavy drinkers is significantly lower ($p < 0.05$) in 1988 than in 1983.
Source: Adapted from National Institute on Alcohol Abuse and Alcoholism. Eighth Special Report to the U.S. Congress on Alcohol and Health. NIH Pub. No. 94–3699. Bethesda, MD: National Institutes of Health, 1994.

drink are compared with men who drink, the women are more likely to judge the risk of crash as higher when alcohol has been consumed. Men, on the other hand, are more likely to believe that alcohol will not affect their performance (Mundt et al. 1992; Agostinelli and Miller 1994). Heavier drinkers of both sexes are even less likely to believe there is a higher risk (Agostinelli and Miller 1994).

Psychomotor Performance

Alcohol affects psychomotor performance, especially when tasks become more complex. After several years of experience, driving becomes highly overlearned and automatic; that is, the driving task does not involve focused concentration. Because of the overlearning, most drunken driving trips are uneventful; that is, they result in neither arrest nor crash. Consequently,

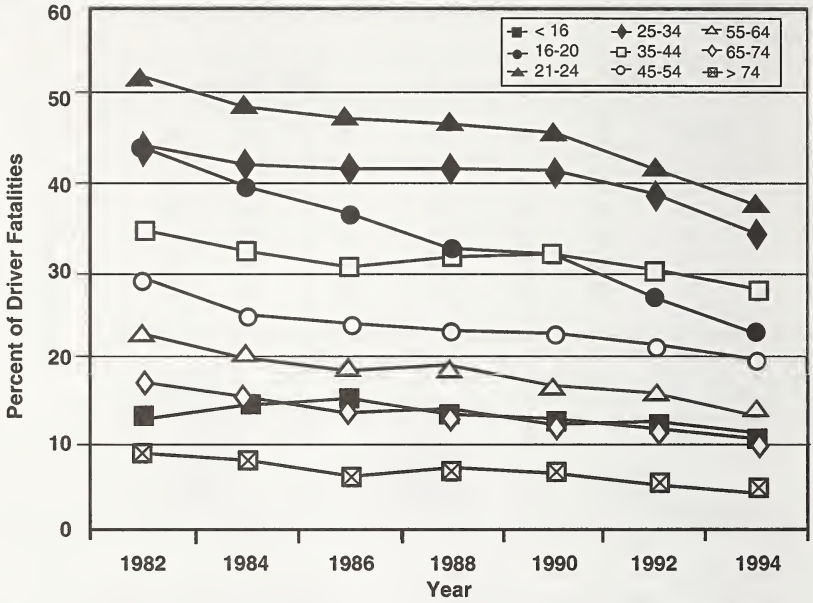

Figure 10. Percent of fatally injured drivers testing positive for alcohol, by driver age. Reprinted from National Highway Traffic Safety Administration. *Traffic Safety Facts 1994.* Washington DC: National Highway Traffic Safety Administration, 1995.

the drivers come to believe that they can "hold their liquor" and that, while others should not drink and drive, they themselves can handle it. However, it is when the unexpected happens, and the task becomes more demanding, that alcohol is more likely to make a difference.

Injury

Once a crash occurs, the presence of alcohol increases the probability of serious or fatal injury. Contrary to folklore, the drunken driver is not more likely to walk away unscathed. When other variables known to be related to driver injury are taken into consideration, the probability of serious or fatal injury increases if the driver has been drinking. While this phenomenon has been clearly demonstrated in well-controlled laboratory studies using animal models (Gettler and Allbritten 1963; DeCrescito et al. 1974; Liedtke and DeMuth 1975; Flamm et al. 1977; Nicholas and DeMuth 1980; Brodner et al. 1981; Anderson 1986; Waller et al. 1986*a*), it is not so readily studied in humans. However, the motor vehicle crash provides a model, albeit somewhat rough, for estimating the physical forces involved in a crash. The overall risk of serious or fatal injury, in a motor vehicle crash of specified dimensions, for the drinking driver is about twofold, but rises to more than fourfold for some types of crashes (Waller et al. 1986*b*).

It is also known that heavy alcohol use may lead to biomedical damage. Women appear to be more vulnerable,

developing liver damage and becoming alcoholic over a shorter period of time and as a result of consuming less alcohol (Blume 1982; Sherlock 1988). Also, heavy chronic use of alcohol may exacerbate osteoporosis, thus increasing the probability of bone fracture from a given impact (Saville 1975; Peng et al. 1982). Whether moderate alcohol use increases or decreases bone strength is controversial, with studies reporting both greater bone damage (Sowers et al. 1985; Stevenson et al. 1989; Hernandez-Avila et al. 1991; Leino et al. 1994) and increased bone mass (Gavaler and Van Thiel 1992; Felson et al. 1995). If alcohol decreases bone mass, it would be expected to increase the likelihood of fractures, and the likelihood of fractures would be greater for older women.

COHORT ISSUES

There are cohort issues that may alter the older driver picture in the future. Older drivers today grew up during Prohibition and the Great Depression, when per capita alcohol consumption was very low. Whatever else Prohibition did, it reduced alcohol-related health problems. The cohort of drivers that will achieve elderly status over the next 20 years may bring very different alcohol-related behaviors, as well as very different driving behaviors.

In the late 1960's and in the 1970's, major social changes occurred. The age of legal majority was lowered from 21 to 18, and most states correspondingly lowered their minimum legal drinking age. The Women's Movement led to major social and economic liberation for women. They entered the labor market in unprecedented numbers, increased their education, postponed marriage, had fewer children and at a later age, and used alcohol in increasing numbers. In an NIAAA report to Congress, it was stated that "drinking patterns of middle age may be maintained into older age to a greater extent than previously appreciated and . . . some of the changes in drinking observed among the elderly may reflect those taking place in society as a whole rather than being an age-specific effect." (NIAAA 1995, p. 23)

In other words, women who grew up at a time when alcohol was not used widely or heavily by women may be less inclined to use it in their older years. In contrast, women who came of age in the 1960's and 1970's may bring with them very different attitudes and behaviors.

Because alcohol appears to differentially increase the crash risk for both women and the elderly, the emerging cohort of elderly drivers may pose very different problems than those we have witnessed in the past. The fact that older persons are more vulnerable to injury from the same physical impact also underscores the increased danger of combining alcohol with highway transportation for this age group.

INTELLIGENT TRANSPORTATION SYSTEMS

Intelligent transportation systems, or ITS, involve the application of scientific and engineering advances in commu-

nications, computer, and information systems to surface transportation. They enable vehicles to communicate with highways (and vice versa) and with other vehicles. A major purpose of ITS is to increase the efficiency of existing facilities so that it will not be necessary to pave additional real estate to build more highways. Other major purposes include facilitating the driving task and improving safety. A population of particular interest and concern is older drivers. On the one hand, ITS may enable more older drivers to continue to meet their transportation needs for a longer period of time. On the other hand, ITS could result in information overload, making the driving task more difficult and decreasing safety.

In 1956 the Federal Government authorized and initiated the interstate highway system, a measure that led to the safest highway system in the world. The authorization for the interstate highway system ended in 1991 and was replaced by legislation initiating ITS. In effect, ITS is the next major era in surface transportation, and decisions and policies currently under way are building the foundation for transportation for the next 30 or 40 years. However, there are many unanswered questions about ITS, especially in relation to older drivers and pedestrians.

A DRIVING SIMULATOR STUDY

One of the important unanswered questions in ITS is how alcohol will affect the ability to use the new technologies, especially alcohol levels below the legal limits for driving. A major technology in ITS is in-vehicle route navigation systems, whereby information is presented on a small screen located to the right front of the driver. Using a driving simulator, one of the studies sponsored by the University of Michigan Alcohol Research Center is investigating the effects of low, but legal, levels of alcohol on driving performance as a function of age and gender. Table 3 illustrates the study design for the first experiment, in which performance is measured as a function of alcohol, age, and gender.

Subjects

Originally there were 108 subjects in the study, but because of equipment failures, only 92 could be included in the analyses of performance on the secondary task. The included subjects

Table 3. Experiment I Design and Sample Size.

	Age		
Gender	Young 30–54	Middle 55–64	Old 65 and Older
Female	17	16	15
Male	17	13	14

were similar to the excluded subjects on age, gender, employment status, and education.

Method

The driving simulator was an interactive PC-based system that automatically recorded speed and speed variance, and lane deviation and variance, among other things. A divided-attention task was incorporated, analogous to the in-vehicle route navigation systems being made available on some new vehicles. Information was presented to the driver in approximately the same location as are route navigation systems. For this study, the secondary task consisted of items from the Stroop test, presented with variable timing. Speed and accuracy were automatically recorded, with reaction times measured to the nearest millisecond.

Alcohol condition consisted of either 0.05 percent BAC or placebo, with subjects counterbalanced for order. Other variables included age and driving difficulty (35 mph vs. 45 mph). All subjects were healthy and used alcohol. After an initial day of alcohol administration and practice, subjects were tested twice, a week apart.

Results

The key findings are summarized as follows.

Alcohol. All subjects responded rapidly. Reaction times were significantly faster under the placebo condition ($p = 0.014$) than under the alcohol condition, but the differences were not large.

Practice. For both men and women, practice made a difference. Practice was by far the most important factor accounting for reaction times ($p = 0.0001$). Taking practice into account, there was a trend for reaction times under the alcohol condition to be slightly longer ($p = 0.084$).

Driving difficulty. Driving difficulty (speed driven) interacted with gender, alcohol, and practice to produce highly significant effects ($p = 0.0004$). However, men and women showed exactly opposite effects in the interaction of practice and alcohol condition, making interpretation difficult.

Alcohol by order. When alcohol was administered the first day, its effects were much more impairing than when it was administered the second day. Practice affected alcohol performance more than placebo performance.

Age. Even though the age groups did not include adolescents or young adults, an age effect was evident ($p = 0.045$). Middle and Old subjects were virtually identical on mean reaction times, while Young subjects responded much more rapidly.

Gender. Gender had no overall effect, but there were gender differences. Practice had a greater effect on the performance of men than of women. On the first test day, women performed better than men. On the second test day, the opposite was true, with men performing more rapidly than women. The change in relative performance was attributable to a much greater change on the part of the men.

This phenomenon held true for all three age groups. For the Young group, the improvement in reaction time for men was 2.74 times that for women. The improvement was 6.3-fold for the Middle group and 1.45-fold for the

Old group. These differences were consistent and striking.

Although men benefited from practice more than women, the differences in performance between the Old and Young subjects were much greater for men than for women. Young men performed much better than Young women, but Old women performed much better than Old men. Men showed much greater differences as a function of age.

These age and gender effects will be investigated further in other measures from the study.

SUMMARY

The following points need to be made in regard to aging, alcohol, and driving:

- The elderly are by far the fastest growing segment of the driving population. Older women in particular are increasing their licensure and their driving.
- After about age 55, the crash risk per mile driven (as opposed to number of licensed drivers) begins to increase, and continues to increase at an accelerating rate. For drivers in their eighties, this risk may exceed that of young beginning drivers.
- The increase in crash risk per mile driven occurs despite the fact that most older drivers try to restrict themselves to the safest times and conditions.
- Drivers who must meet a schedule, such as commercial drivers, cannot exercise such restrictions, and it appears that the elevated risk occurs at earlier ages for them.
- Older drivers (and occupants) are more seriously injured than younger people in a crash of specified dimensions.
- At even very low levels of alcohol, the risk of crash is elevated. This increase in risk rises sharply as BAC increases.
- At every age, women appear to have a much higher crash risk at a given BAC.
- Abstinence rates are increasing and are the highest among the older population. However, of those who do drink, the elderly have among the highest proportions of heavy drinkers.
- Alcohol differentially affects the performance of the elderly, that is, at a given BAC they are more impaired. This difference is also seen in crash experience. Older drivers are at higher risk of crash at a given BAC.
- Alcohol affects injury in at least three ways. First, it impairs judgment, so that one is more likely to be exposed to high-risk situations. Second, it affects psychomotor performance, making it more difficult to respond successfully to a crisis situation. Third, it exacerbates the extent of injury resulting from a given impact.
- Cohort differences may herald a growing problem of alcohol among elderly drivers. It appears that the drinking habits of middle age may persist into older age. The emerging older driver population brings different drinking behaviors, particularly among women. Because women appear to be more impaired by alcohol, and because older persons

appear to be more impaired by alcohol, and because both women and the elderly are a growing presence in the driving population, there is a need to take preventive measures to avoid future problems with alcohol and older drivers.

In addition, preliminary findings from the driving simulator research lead to the following conclusions:

- In ITS, in-vehicle route navigation systems have been introduced to the public in rental cars at airports. Because alcohol was found to be more impairing when the subject is first introduced to the secondary task, and because passengers leaving planes to rent cars often have been drinking, the wisdom of introducing new technology in this manner should be questioned. Because practice is so significant, for at least some ITS technologies it may be appropriate to require short-term practice before a customer is allowed to use the technology. Such practice would be appropriate prior to first use of any new technology, whether the vehicle is rented, leased, or purchased.
- Although driving difficulty was not easy to interpret, it interacted with practice, alcohol, and gender to produce significant differences. Clearly, new technology should be as simple as possible and should be pilot tested on all ages and under alcohol conditions.
- Age effects were strong, with both Middle and Old subjects doing significantly less well than Young subjects. Age interacted with gender to produce findings that suggest that Old men perform more poorly than Old women, but for the Young, the opposite is true. These findings suggest that subpopulations may have greater or lesser difficulty with new technologies.
- The persistent finding that Young men performed better than Young women, but Old women performed better than Old men needs further investigation, with a focus on whether and how alcohol may interact with these gender differences.

ACKNOWLEDGMENT

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REFERENCES

Agostinelli, G., and Miller, W.R. Drinking and thinking: How does a person's drinking affect judgments of prevalence and risk? *J Stud Alcohol* 55:327–337, 1994.

Anderson, T.E. Effects of acute alcohol intoxication on spinal cord vascular injury. *J Neurotrauma* 3:183–192, 1986.

Attwood, D.A. Effects of moderate levels of blood alcohol on responses to information from simulated automobile rear-signal systems. *Accid Anal Prev* 10:11–20, 1978.

Attwood, D.A. Williams, R.D.; and Madill, H.D. Effects of moderate blood alcohol concentrations on closed-course driving performance. *J Stud Alcohol* 41:623–634, 1980.

Avant L.L. Alcohol impairs visual presence/absence detection more for females than for males. *Percept Psychophysiol* 48:285–290, 1990.

Blume, S.B. Alcohol problems in women. *NY State J Med* 82:1222–1224, 1982.

Borkenstein, R.F.; Crowther, R.F.; Shumate, R.P.; Ziel, W.B.; and Zylman, R. The Role of the Drinking Driver in Traffic Accidents (the Grand Rapids Study). Indiana University, Department of Police Administration, 1964. Republished as an article in *Blutalkohol* 11 (Suppl 1):1–131, 1974.

Brainin, P.A. *Safety and Mobility Issues in Licensing and Education of Older Drivers.* Washington, DC: National Highway Traffic Safety Administration, 1980.

Brodner, R.A.; Van Gilder, J.C.; and Collins, W.F., Jr. Experimental spinal cord trauma: Potentiation by alcohol. *J Trauma* 21:124–129, 1981.

Carlson, W.L. Alcohol usage of the night-time driver. *J Safety Res* 4:12–25, 1972.

Carp, F.M. Significance of mobility for the well-being of the elderly. In: *Transportation in an Aging Society: Improving Mobility and Safety for Older Persons.* Vol. II. Washington, DC: National Research Council, 1988. pp. 1–20.

Cerrelli, E. Older Drivers. *The Age Factor in Traffic Safety.* Washington, DC: National Highway Traffic Safety Administration, 1989.

DeCrescito, F.; Demopoulos, H.B.; Flamm, E.S.; and Ransohoff, J. Ethanol potentiation of traumatic cerebral edema. *Surg Forum* 25:438–440, 1974.

Evans, L. Age and sex effects on severe and fatal injury rates in traffic crashes. In: *FISITA Congress, XXIInd. Automotive Systems Technology: The Future. Vol. I: Technical Papers.* Warrendale, PA: Society of Automotive Engineers, 1988. pp. 1.434–1.442.

Evans, L. *Traffic Safety and the Driver.* New York: Van Nostrand, 1991.

Felson, D.T.; Zhang, Y.; Hannan, M.T.; Kannel, W.B.; and Kiel, D.P. Alcohol intake and bone mineral density in elderly men and women, The Framingham Study. *Am J Epidemiol* 142:485–492, 1995.

Fillmore, K.M.; Hartka, E.; Johnstone, B.M.; Leino, E.V.; Motoyoshi, M.; and Temple, M.T. A meta-analysis of life course variation in drinking: The Collaborative Alcohol-Related Longitudinal Project. *Br J Addict* 86:1221–1268, 1991. Quoted in National Institute on Alcohol Abuse and Alcoholism, *Eighth Special Report to the U.S. Congress on Alcohol and Health* (Bethesda, MD: National Institutes of Health, 1994), p. 23.

Flamm, E.S.; Demopoulos, H.B.; Seligman, M.L.; Tomasula, J.J.; DeCrescito, V.; and Ransohoff, J. Ethanol potentiation of central nervous system trauma. *J Neurosurg* 46:328–335, 1977.

Gavaler, J.S., and Van Thiel, D.H. The association between moderate alcoholic beverage consumption and serum estradiol and testosterone levels in normal postmenopausal women: Relationship to the literature. *Alcohol Clin Exp Res* 16:87–92, 1992.

Gettler, D.T., and Allbritten, F.F., Jr. Effect of alcohol intoxication on the respiratory exchange and mortality rates associated with acute hemorrhage in anesthetized dogs. *Ann Surg* 158:151–158, 1963.

Hernandez-Avila, M.; Colditz, G.A.; Stampfer, M.J.; Rosner, B.; Speizer, F.E.; and Willett, W.C. Caffeine, moderate alcohol intake, and risk of fractures of the

hip and forearm in middle-aged women. *Am J Clin Nutr* 54:157–163, 1991.

Hull, R.W., and Knebel, G.W. *Statistical Summary of School Bus Accident Data.* Washington, DC: Federal Highway Administration, 1968.

Kaplan, G.A. Where do shared pathways lead? Some reflections on a research agenda. *Psychosom Med* 57:208–212, 1995.

Leino, A.; Jarvisalo, J.; Impivaara, O.; and Kaitsaari, M. Ovarian hormone status, life-style factors, and markers of bone metabolism in women aged 50 years. *Calcif Tissue Int* 54:262–267, 1994.

Liedtke, A.J., and DeMuth, W.E. Effects of alcohol on cardiovascular performance after experimental nonpenetrating chest trauma. *Am J Cardiol* 35:243–250, 1975.

Massie, D.L., and Campbell, K.L. *Analysis of Accident Rates by Age, Gender, and Time of Day Based on the 1990 Nationwide Personal Transportation Survey.* Ann Arbor, MI: University of Michigan Transportation Research Institute, 1993.

Moskowitz, H., and Robinson, C.D. *Effects of Low Doses of Alcohol on Driving-Related Skills: A Review of the Evidence.* Washington, DC: National Highway Traffic Safety Administration, 1988.

Moskowitz, H.; Burns, M.M.; and Williams, A.F. Skills performance at low blood alcohol levels. *J Stud Alcohol* 46:482–485, 1985.

Mundt, J.C.; Ross, L.E.; and Harrington, H.E. A modeling analysis of young drivers' judgments of alcohol risk due to alcohol use and other driving conditions. *J Stud Alcohol* 53:239–248, 1992.

National Highway Traffic Safety Administration. *Traffic Safety Facts 1994.* Washington, DC: National Highway Traffic Safety Administration, 1995.

National Institute on Alcohol Abuse and Alcoholism (NIAAA). *Eighth Special Report to the U.S. Congress on Alcohol and Health.* NIH Pub. No. 94–3699. Bethesda, MD: National Institutes of Health, 1994.

National Safety Council. *Vehicle Damage Scale for Traffic Accident Investigators.* 3d ed. Chicago: the Council, 1984.

Nicholas, G.G., and DeMuth, W.E. Blunt cardiac trauma: The effect of alcohol on survival and metabolic function. *J Trauma* 20:58–60, 1980.

Peck, R., and Romanowicz, P. Teen and senior drivers. *Research Notes* (California Department of Motor Vehicles, Sacramento), Winter 1993/94, 3–6.

Peng, T.C.; Garner, S.C.; Frye, G.D.; and Crenshaw, M.A. Evidence of a toxic effect of ethanol on bone in rats. *Alcoholism* 6:96–99, 1982.

Pisarski, A.E. *Travel Behavior Issues in the 90's.* Washington, DC: Federal Highway Administration, 1992.

Promisel, D.M.; Blomberg, R.D.; Nacht, M.L.; and Silver, S. *School Bus Safety—Operator Age in Relation to School Bus Accidents.* Washington, DC: National Highway Safety Bureau, 1969.

Rosenbloom, S. *Travel by Women, 1990 NPTS Subject Area Report.* Tucson, AZ: The Drachman Institute for Land and Regional Development Studies, 1994.

Saville, P.D. Alcohol-related skeletal disorders. *Ann NY Acad Sci* 252:287–291, 1975.

Sherlock S. Liver disease in women. Alcohol, autoimmunity, and gallstones. *West J Med* 149:683–686, 1988.

Sowers, M.F.; Wallace, R.B.; and Lemke, J.H. Correlates of forearm bone mass among women during maximal bone mineralization. *Prev Med* 14:585–596, 1985.

Stevenson, J.C.; Lees, B.; Devenport, M.; Cust, M.P.; and Ganger, K.F. Determinants of bone density in normal women: Risk factors for future osteoporosis? *BMJ* 29:924–928, 1989.

Waller, P.F., and Blow, F.C. Women, alcohol, and driving. In: Galanter, M., ed. Recent Developments in Alcoholism. Vol. 12: *Women and Alcoholism.* New York: Plenum Press, 1995. pp. 103–123.

Waller, P.F.; Hansen, A.R.; Stutts, J.C.; and Popkin, C.L. Alcohol: A potentiating factor in motor vehicle crash injury. In: *Alcohol, Accidents, and Injuries.* Warren, PA: Society for Automotive Engineers, 1986*a*. pp. 53–61.

Waller, P.F.; Stewart, J.R.; Hansen, A.R.; Stutts, J.C.; Popkin, C.L.; and Rodgman, E.A. The potentiating effects of alcohol on driver injury. *JAMA* 256:1461–1466, 1986*b*.

Williams, A.F., and Carsten, O. Driver age and crash involvement. *Am J Public Health* 79:326–327, 1989.

Zador, P.L. Alcohol-related risk of fatal driver injuries in relation to driver age and sex. *J Stud Alcohol* 52:302–310, 1991.

TREATMENT AND PREVENTION

Chapter 18

Alcohol Problems in Health Care Settings: Prevalence, Causal Factors, and Interventions

Wendy L. Adams, M.D., M.P.H.

The last decade has seen several advances in our understanding of the impact of alcohol use on health and the use of health services. One such advance has been clarification of the prevalence of alcohol problems in health care settings. In the 1960's and 1970's, alcoholism was thought by many to be a "self-limiting disease"—people either stopped drinking or died of complications of their alcoholism before reaching old age. In this chapter, I review some of the epidemiologic data that, by showing the substantial public health impact of alcoholism among older people, have changed that view. I also discuss advances in our understanding of how primary care doctors can best address alcohol problems in clinical encounters. A good deal has been learned in recent years about how to use simple screening tools to detect problem drinking among older people in health care settings. In addition, studies have begun to show that physicians using brief counseling strategies in the primary care setting can intervene successfully among problem drinkers.

In cross-sectional population-based studies, the prevalence of alcohol problems declines with increasing age (Cahalan and Cisin 1968; Barnes 1979; Myers et al. 1984). The Epidemiologic Catchment Area study is a good example. In each of its multiple sites, that study showed a clear decline in the prevalence of alcohol abuse and dependence with age (Myers et al. 1984). The few longitudinal studies that have followed people into their late seventies and older generally also have shown a pattern of decline with age, though people tend to maintain their drinking habits into their late sixties and early seventies (Glynn et al. 1985; Stall 1986; Fillmore 1987; Temple and Leino 1989; Adams et al. 1990).

W.L. Adams, M.D., M.P.H., is assistant professor of medicine at the University of Nebraska Medical Center, 600 South 42d St., P.O. Box 985620, Omaha, NE 68198–5620.

The pattern of decline with age seen in the general population does not seem to hold true in health care settings. This point is demonstrated by data from the National Hospital Discharge Survey, which collects data from a national sample of hospitals to determine the frequency of various illnesses. Data from that survey showed that the age group with the highest rate of alcohol-related hospitalizations was the 45- to 64-year-old group. Those age 65 and older had a slightly higher rate than those ages 25–44 (Stinson et al. 1989). One possible interpretation of this finding is that, although there are fewer elderly problem drinkers, they suffer more alcohol-related medical problems than their younger counterparts.

Many studies have now shown that, even though the prevalence of alcohol use disorders and alcohol use itself decline with the increasing age of a population, they are much more common among older people in health care settings than in the general population. In fact, every setting where physicians see elderly patients has been shown to have a relatively high prevalence of heavy drinking and alcohol use disorders.

ALCOHOL PROBLEMS IN THE PRIMARY CARE SETTING

There have been several studies of the prevalence of alcohol problems in primary care settings. In the primary care clinics of the Durham, North Carolina, Veterans Affairs Medical Center, 19 percent of veterans ages 55–64 and 10 percent of those age 65 and older screened positive on the Veterans Alcohol Screening Test (Magruder-Habib et al. 1986). Another study reported on 323 outpatients age 60 and older in a medical school clinic in Virginia. Using the Diagnostic Interview Schedule, researchers there found that 33 percent had a lifetime history of alcohol abuse or dependence and 6 percent had current alcohol problems (Buchsbaum et al. 1992). Jones and colleagues interviewed 154 elderly primary care patients in a teaching hospital clinic in North Carolina. Sixteen percent met DSM-III criteria for lifetime alcohol abuse or dependence, and 4 percent met criteria for current alcohol use disorders (American Psychiatric Association 1980; Jones et al. 1993). In a sample of 241 older-old (75 and older) general practice patients in London, Iliffe and colleagues (1991) found that 3.6 percent of men and 3.2 percent of women reported drinking more than the safety limits of the Royal College of Psychiatrists. Several factors affect the range of prevalence seen in these studies. The age range and gender ratio of a given population are two of the most important predictors of alcohol use and misuse, even within an elderly population. Geographic variation also comes into play, as do religious and ethnic backgrounds, socioeconomic status, education, and health status.

A very large sample of primary care patients was studied at Indiana University (Callahan and Tierney 1995). Of nearly 4,000 people age 60 and older, 10.6 percent were CAGE

positive and reported having used alcohol within the year. This study had enough subjects, and enough African American subjects, to examine age, race, and gender groups. African Americans in that study were somewhat more likely to meet the criteria for alcoholism than European Americans. In general, men were more likely than women to be alcoholic, and the prevalence for both men and women decreased with increasing age. There was an interesting exception, though, seen among men over 75, who had a higher prevalence than those who were slightly younger.

At least one other study found a similar effect among older-old men: the 1-year followup of the Epidemiologic Catchment Area study showed an increased incidence of alcoholism in this group (Eaton et al. 1989). In view of the known high rate of suicide among older men, this finding is of particular interest and concern. Alcohol use among older men deserves further study in a sample of sufficient size to clarify this effect.

The Indiana University study was also able to report on the medical problems of these older primary care patients (Callahan and Tierney 1995). Overall, the most common diagnoses for older people in this clinic were hypertension, arthritis, and diabetes. In a univariate analysis, alcoholics were significantly less likely to present with these illnesses, but were significantly more likely to present with chronic obstructive pulmonary disease (they were also more likely to smoke), injuries, gout, and being underweight. There were, however, differences between the groups that may have had an impact on their medical problems: in addition to being more likely to smoke, the CAGE-positive people were much more likely to be male, African American, and less educated, and were slightly younger than the CAGE-negative people. The study was unable to address the relative importance of these factors in the presenting medical problems.

ALCOHOL PROBLEMS IN THE EMERGENCY DEPARTMENT SETTING

The prevalence of current alcohol problems is considerably higher in the emergency department (ED) setting than in the primary care setting. In 1969, Wechsler and colleagues found that 20 percent of elderly people who came to the ED because of accidents in their homes had positive alcohol breath tests. In 1978, Waller studied elderly people who presented to the ED with falls and found that 13 percent had used alcohol shortly before the fall. Rivara and colleagues (1993), at the University of Washington, studied blood alcohol levels in trauma patients and found that 13 percent of elderly trauma patients who came to the ED had blood alcohol levels over 100 mg/dL.

Two studies have reported on consecutive elderly ED patients with any diagnosis. Tabisz and colleagues (1991) in Winnipeg found a 14 percent prevalence of alcohol problems among such patients. At the University of North Carolina Hospital ED, 14 percent of elderly patients had positive

scores on the CAGE questionnaire and acknowledged current alcohol use (Adams et al. 1992). Interestingly, few of these patients presented with trauma, in contrast to the presentation of younger problem drinkers to EDs. Medical illnesses, particularly gastrointestinal complaints, were the rule. The impact of alcohol on the specific injuries and illnesses of elderly ED patients has been studied very little.

ALCOHOL PROBLEMS IN THE ACUTE CARE HOSPITAL

The prevalence of alcohol problems among elderly hospital inpatients is also quite high. In a study from Johns Hopkins Hospital, Curtis and colleagues (1989) found that 21 percent of medical inpatients over age 60 screened positive for alcoholism on either the CAGE or Short Michigan Alcoholism Screening Test. Patients age 70 and older had a lower prevalence than those ages 60–69: 15 percent versus 26 percent. Ninety-one percent of the patients who screened positive for alcoholism were men, though the sample was predominantly female.

There have been two British studies of alcohol problems among elderly hospital inpatients. In London, Bristow and Clare (1992) found that 9 percent of men and less than 1 percent of women admitted to medical and geriatric services met Royal College of Psychiatrists' criteria for unsafe quantities of alcohol consumption. Another British study reported a 13 percent prevalence of alcohol abuse or dependence among elderly men and 2 percent among elderly women admitted to hospitals (Mangion et al. 1992). Two percent of admissions were clearly alcohol related.

In a study from The Netherlands, Speckens and colleagues (1991) found that 13 percent of men and 7 percent of women admitted to all wards of a general hospital scored positive on the Dutch version of the Munich Alcoholism Test. Alcoholics were more likely than nonalcoholics to have "vague symptoms" as admission diagnoses. They were also more likely to have organic brain syndromes and to use psychotropic medications.

Another approach to studying the epidemiology of alcohol problems among elderly inpatients has been to examine hospital discharge diagnoses. This method allows the researcher access to a larger number of subjects than is feasible in an interview study, but limits the cases to patients whose alcoholism was identified by their physicians, recorded in the medical record, and transcribed to a list of discharge diagnoses. This approach almost certainly results in a marked underestimation of alcohol problems.

The first study to report on alcohol-related hospitalizations using this method was the National Hospital Discharge Survey, mentioned earlier in this chapter. Another report on hospital discharge diagnoses of alcoholism was from a study of 1989 Medicare billing records for the entire United States (Adams et al. 1993). Since 96 percent of people age 65 and older are covered by Part A of Medicare, this method is probably as close as we can get to looking at the entire elderly population

of the United States. The overall prevalence of alcohol-related hospitalizations, at 48 per 10,000 population, was slightly lower than that found by the National Hospital Discharge Survey. The prevalence declined with increasing age and was higher for men than women at every age. The most common alcohol-related diagnosis billed to Medicare, either as a primary or a secondary diagnosis, was alcohol dependence. The second most common was alcoholic liver disease, followed by alcoholic psychosis and alcohol abuse. Alcoholic cardiomyopathy, gastritis, and polyneuropathy occurred at lower levels.

The national database in the Medicare billing records study allowed the researchers to examine geographic variation in alcohol-related hospitalizations. The northern states generally had the highest rates and the lower Midwest the lowest. The state with the lowest prevalence was Arkansas, with 19 cases per 10,000 population, and the state with the highest was Alaska, with 77 cases per 10,000. Rates were higher for men than for women in every state. When compared with the prevalence of hospitalization for myocardial infarction, which is generally recognized as a common and important problem for elderly people, the frequency of alcohol-related hospitalizations was found to be slightly higher.

ALCOHOL AND NURSING HOMES

The effect of alcohol use and alcohol use disorders on nursing homes has been studied very little. The reported prevalence of alcoholism in this setting has a huge range, from 2.8 to 49 percent (Joseph 1995). Studies that have interviewed patients or their proxies show a higher frequency than those that depend on chart review for the diagnosis. As in other settings, younger people and men are more likely to be alcoholic. The high prevalence of alcohol problems in some nursing home populations is of particular interest in view of a recent trend toward using nursing homes for short-term rehabilitative stays. This trend is likely to cause a change in the nursing home population. People recovering from more acute illnesses or injuries may be more likely to be actively alcoholic than the oldest old, chronically ill, and disabled people residing in nursing homes. For patients recovering from alcohol-related illness or injury, the nursing home has potential to be an excellent setting for treatment of alcoholism, though few currently offer treatment programs.

There are now many studies from various health care settings that consistently show a higher prevalence of alcohol problems than in population-based studies. Settings that offer relatively intense levels of care, such as EDs and acute care hospitals, seem to show the highest prevalence.

ALCOHOL AND HEALTH SERVICES USE

There are few studies that address how alcohol use or alcohol use disorders affect health services utilization by el-

derly people. In the Indiana University study, Callahan and Tierney (1995) reported on a 1-year followup of nearly 4,000 patients who had been screened for alcohol use disorders in a primary care clinic. They found that alcoholics were just as likely as the other primary care patients to have standard preventive medicine interventions, such as vaccinations and cancer screening. They were significantly more likely than the nonalcoholics to be hospitalized (21.5 percent vs. 16.9 percent), but not significantly more likely to go to the ED or primary care clinic. They also found that at 2 years, alcoholics were more likely to have died (10.6 percent vs. 6.3 percent). Alcoholism was still a significant predictor of mortality when age, gender, race, education, and smoking were controlled for, though the relative risk was low: 1.39 (CI 1.03–1.88). Alcoholics were much more likely to have died from cancer (51.3 percent vs. 27.9 percent) or cirrhosis (10.3 percent vs. < 1 percent) than nonalcoholics. As noted in the previous discussion of this study, however, alcoholics in this study were more likely to smoke and to be male, African American, and less educated, all of which may have had a bearing on the outcomes listed. These were also people who already had access to health care services: they were selected because they were in a primary care clinic. The generalizability of these findings is therefore limited to similar populations who have access to health care.

Leigh and Fries (1992) studied 1,558 Bank of America retirees in California to determine the effect of their health habits on health services use. In this group of predominantly white, well-educated retirees, alcohol use of more than two drinks per day was a significant predictor of the number of self-reported sick days and of total health care costs in the ensuing 12 months, but not of hospital days or doctor visits. Body mass and cigarette smoking were more powerful predictors of health services use. This study is also generalizable only to populations that have similar composition.

Another study used data from the 1990 National Health Interview Survey to look at health services use (Rice and Duncan 1995). This report selected people who were age 60 and older and who had consumed at least 12 drinks in the year prior (abstainers were excluded). They did a cross-sectional analysis of the association between alcohol use in the 2 weeks prior to the interview and health-related variables. Respondents were queried about their health and health services use during the year prior to the interview. Heavy drinkers (those who consumed two or more standard drinks per day) in that study had slightly fewer physician visits in the year prior to the interview than light and moderate drinkers, though the heavy drinkers reported having slightly more medical conditions. Differences in hospital use were minimal. Since this was a cross-sectional study, inferring cause from these data would be highly inappropriate. If anything, the alcohol use would have to be considered the outcome variable, since the alcohol use occurred after the health services use indicators they

measured. Perhaps the physicians successfully counseled their patients about drinking and they cut down before the survey.

A study from Sweden (Mellstrom et al. 1981) examined men who had been "registered at the Temperance Board" two or more times (probably a measure of quite severe alcohol abuse or dependence). They were found to be more physically and cognitively disabled and more likely to be institutionalized and to die in the 5-year followup period than nonalcoholics.

The Iowa 65+ Rural Health Study also studied men who reported ever having been "heavy drinkers" (definition of the term was not quantified). These self-identified heavy drinkers were more likely to report major illnesses and disability at the baseline interview (they were also more likely to smoke, had lower income, were slightly younger, and had slightly less education than nonheavy drinkers). They also reported more physician visits and hospital stays in a 3-year followup period (Colsher and Wallace 1990).

It seems puzzling that, while virtually all studies of health care populations show higher prevalences of alcohol problems than population-based studies, studies that attempt to show a cause-and-effect relationship between alcohol use and health services use have not shown a clear and consistent effect. There are several possible reasons for this, including the complexity of "alcohol use" as a variable, characteristics of the sample, and causal complexity. The complexity of studying why people use health services has probably been underestimated, as has the complexity of alcohol use as a variable.

Alcohol Use as a Variable

The qualities of alcohol use as a variable present several problems that probably contribute to the difficulty in studying the relationship between alcohol use and health services use. Varying definitions of "heavy drinking," "hazardous drinking," "harmful drinking," "problem drinking," "alcohol abuse," and "alcoholism" have always plagued the literature on this subject. In the studies cited in the preceding section, the definition used does appear to affect whether alcohol use contributes to health services use: studies of more severe problem drinkers seem to show a greater risk of health problems and health services use, as might be expected. Studies that include more moderate heavy drinkers appear to show less effect on health services use. Future research on this subject clearly must examine different levels and patterns of use as well as problems related to drinking.

In addition to the problem of variable definitions, there are various ways of measuring alcohol use. For most studies, self-report is the best measure available. Although self-report is, for the most part, reasonably valid, memory is not perfect, and intentional underreporting does occur at times with people of all ages. Underreporting may be particularly common among individuals with more severe alcohol problems.

In addition to these problems with self-report, memory problems are a major issue in studying older adults. At age 80, the prevalence of demen-

tia is approximately 30 percent; at age 90, it approaches 50 percent. In addition, various questionnaires measure alcohol use and alcohol problems in differing ways and have varying results. Few instruments have been well tested in older populations. The female predominance of this group and other demographic and psychosocial phenomena present challenges in measuring alcohol use. Graham (1986) summarized many of the issues that need to be considered when measuring alcohol use or problems in an older population.

Another difficulty in studying alcohol and health services is that alcohol use is a mutable variable. People change their alcohol use over time. Often, changes in use are related to health problems, complicating the relationship between alcohol and health services use. Heavy and problem drinkers are particularly prone to fluctuations in their use. It is relatively common for alcoholics to abstain for periods of time, sometimes months or even several years. While they are in an abstinent phase, they will not be susceptible to complications of acute heavy drinking, but they may still have chronic complications of their previous alcohol use. Since chronic alcohol use causes different medical complications from short-term alcohol use, this difference has major implications for health services use. Our understanding of the duration of exposure to alcohol that is required to cause various health problems is very limited.

To complicate matters further, alcohol does not have a clear-cut dose-response relationship to its adverse effects the way some toxic substances do. At low and moderate doses, it may be beneficial to health by preventing coronary artery disease (Klatsky et al. 1992, Rimm et al. 1991); at very high doses it is clearly harmful. Probably there is an area of overlap where it is beneficial in preventing coronary heart disease but harmful in causing cancer, cirrhosis, and other complications. If we look at overall health services utilization as an outcome variable, the adverse effect on cancer and cirrhosis may be overwhelmed by the beneficial effect on coronary disease in groups where coronary disease produces a very large proportion of the burden of disease.

Lastly, alcohol use is affected by health status. Elderly people commonly cite health problems as the reason they cut down on alcohol use. If people cut down on their drinking as they begin to develop a health problem, then the remaining drinkers in a cohort become a self-selected healthier group. It then becomes very difficult to discern a relationship between alcohol use and a health problem.

Characteristics of the Sample

The sample selected also will affect the results of a study. The age range, gender ratio, geographic location, religious and ethnic background, health status, and education of the group under study all can have an impact. Age and gender have been strong predictors of alcohol use and alcoholism in most studies. Both variables also affect health and health services use. For instance, older people are more likely to become ill, but less

likely to use or misuse alcohol. However, there is tremendous heterogeneity within the group considered "elderly," and generalizations can be misleading. The age range of the group spans at least 40 years and includes people in their late sixties who are still actively employed and in excellent health as well as people over 100 years of age, who are more likely to be cognitively and physically disabled. Women are less likely to use and misuse alcohol, are less susceptible to some diseases and more susceptible to others, and are somewhat more likely to use health services. Health services use, per capita consumption of alcohol, and some illnesses vary with geographic location. If sampling from a health care setting or a retirement community, a group with features very different from those selected from the general population will be targeted. If nursing home residents are included, yet another picture will emerge. Generalizations about alcohol use among "the elderly" must be made with much caution, considering the variability of this group.

Causal Complexity

Another knotty problem in looking at the relationship between alcohol and health or health services use has to do with the nature of cause-and-effect relationships. Probably no medical or biological phenomenon is the result of a single cause. For instance, alcohol most certainly is a cause of cirrhosis of the liver. The relationship clearly has been established. But heavy drinking is not itself a necessary cause: one can get cirrhosis from an autoimmune or infectious disease without ever consuming any alcohol. Also, alcohol alone is not a sufficient cause of cirrhosis. Although some people get cirrhosis from alcohol intakes as little as two drinks per day, others never get cirrhosis even though they consume 15 drinks per day. There are other factors, such as genetic susceptibility, exposure to hepatitis viruses, and exposure to hepatotoxic drugs, that also affect whether one gets cirrhosis.

Probably there are unknown factors that affect the relationship between alcohol and cirrhosis as well. Rothman (1987) suggested that "For biologic effects, most and sometimes all of the components of a sufficient cause are unknown." To the extent that we know the components of a sufficient cause, we can account for them in the planning and analysis of our studies. Those components of a causal constellation that are unknown or unrecognized, however, generally are not addressed. In a randomized controlled trial, the randomization process decreases the possibility of unknown causes contributing to a spurious effect, but in nonrandomized studies, even this degree of control often is not possible.

In addition, component causes often are not completely independent of one another or of the outcome under study. For instance, in the presence of a hepatotoxic drug, a lower dose of alcohol may cause cirrhosis than would be required in the same individual in the absence of the drug. So even in the case of a clear-cut causal relationship, such as that between

alcohol and cirrhosis, there is considerable complexity. Many of the effects of alcohol on health are less clear-cut than the case of cirrhosis, which may reflect greater complexity in the causal pathway. In studying why people use health services, there are many factors known to be important, such as age, gender, health status, educational level, and socioeconomic status. There are probably many factors that are unknown and difficult to measure, such as personality traits and attitudes toward health care. These factors may also fluctuate over time. For instance, if a doctor is perceived to chastise a patient for drinking behavior, the patient may be less likely to return to the doctor even if the person is quite sick.

As people age, the prevalence of chronic illness increases. In elderly people, therefore, there may be declines in multiple organ systems; changes in daily habits such as smoking, drinking, and eating; shifts in attitudes toward health; and decreased availability of social support. All of these factors, as well as other unknown phenomena, have the potential to affect health-related outcomes, including health services use. The sheer multitude of these factors, along with their complex interactions, can make a clear cause-and-effect relationship very difficult to discern.

A related problem in examining the relationship between alcohol and health or health services use among older people is the large number of comorbidities often exhibited by older people (Fried and Wallace 1992). These co-occurring conditions can affect cause-and-effect relationships in several ways. A comorbidity may affect the risk factor under study. In the case of alcohol, comorbidities may cause decreased alcohol intake in some cases and increased alcohol intake in others. As noted earlier in this chapter, older people often cut down on their alcohol use when they experience a decline in health. On the other hand, people who have depressive symptoms or chronic pain may increase their alcohol use in an attempt to treat these symptoms. Some studies have controlled for the number of comorbidities measured. Since there is probably not a consistent relationship between comorbidities and alcohol use, this approach is likely to be misleading.

Comorbidities may also directly affect the outcome under study. In the case of health care utilization as an outcome, most comorbidities have this potential. When a comorbidity affects the risk factor and is also an independent risk factor for the outcome, the comorbidity becomes a potential confounder of the relationship. If this confound has been taken into account and the comorbidity has been measured well, then a good attempt can be made to control for its impact. However, some diseases are subtle, early in their progression, or undiagnosed. Elderly people are also particularly prone to atypical presentations of disease. If a phenomenon is not a variable in the data set, then of course it cannot be controlled. Comorbidities may also cause other outcomes from the one under study in a way that affects the relationship being studied. For instance, in the course of a study of the relationship between alco-

hol and falls, comorbid hypertension may lead to a stroke, which radically alters the risk of falling.

Consideration of unknown causal components, multiple causal pathways, and comorbidities, as well as their potential for complex interactions with one another, generates a deep respect for the difficulty of modeling health services use in an older population. The heterogeneity of the elderly population and the difficult nature of alcohol as a variable add additional layers of complexity to studying the relationship between alcohol and health services use in older people. Better understanding of this relationship will be extremely valuable for clinicians as well as those who plan for health services, but researchers must carefully consider these complexities in future studies.

THE CLINICAL ENCOUNTER

Important advances have been made in recent years in our understanding of how to detect alcohol problems among older people in clinical settings and also in how physicians may intervene effectively. It is clear that the prevalence of alcohol problems among older people in the primary care setting and the morbidity alcohol can cause make it highly desirable that patients be screened for alcohol problems. A useful screening instrument should be sensitive, so it picks up a large proportion of the people we want to detect, and specific, so it does not falsely label people as problem drinkers when they are not. It should also have good predictive value: the proportion of positive tests that are true positives and the proportion of negative tests that are true negatives should be reasonably high. A useful screening test must be short and easy to use, so that physicians and other people will really use it, and it should detect a range of severity of alcohol problems. This last point is very important and often underappreciated in studying alcohol problems in primary care. Probably the majority of people who have alcohol-related health problems do not meet DSM criteria for alcohol abuse or dependence. If only those individuals with problems severe enough to warrant a diagnosis of an alcohol use disorder are detected, many opportunities to intervene with people who are at risk for health problems from their drinking will be missed.

Unquestionably, the best studied instrument to screen for drinking problems in primary care settings is the CAGE questionnaire. The CAGE has been studied among elderly people in primary care settings and compared to the DSM-III criteria for alcohol abuse or alcoholism. The sensitivity of the CAGE in the primary care setting, using the standard cutoff of two affirmative answers, was 70 percent in one study of older people (Buchsbaum et al. 1991) and 48 percent in another (Jones et al. 1993). Using a cutoff score of one affirmative answer increased the sensitivity of the CAGE, with 86 percent (Buchsbaum et al.) to 88 percent (Jones et al.) of alcoholics detected. Positive predictive values were also considerably greater in both studies when a cutoff score of one was used.

In the primary care setting, a cutoff of one affirmative answer may be the most practical. The consequences of a false positive test in this setting are not grave; in fact, they may lead to a beneficial discussion of which drinking practices are harmful and which are not.

One of the desirable characteristics of a screening test is the ability to detect a range of severity. Whether the CAGE can accomplish this is questionable. Studies generally have concentrated on comparing the CAGE and other screening tests with a DSM diagnosis of alcohol abuse or dependence. Because it has performed quite well and is very easy to use, the CAGE has gained wide acceptance. Often it is the only screening tool used in medical settings. Since alcohol-related medical problems often occur with levels of use that do not meet DSM criteria for an alcohol use disorder, it is important to know if the CAGE can detect a large proportion of individuals who drink heavily enough to increase their risk of medical problems in the absence of an alcohol use disorder.

In the course of a study of physician brief intervention for problem drinkers in the primary care setting, 5,065 primary care patients age 60 and older in physicians' offices in southeastern Wisconsin were screened using both quantity/frequency questions and the CAGE (Adams et al. 1996). Fifteen percent of men and 12 percent of women consumed more than the U. S. Department of Agriculture's (USDA) recommended guidelines of two drinks per day for men and one drink per day for women. Eight percent of men and 2 percent of women reported regularly consuming more than 21 drinks per week. Ten percent of men and 2 percent of women reported consuming more than five drinks per occasion at times. Using the CAGE questionnaire, 9 percent of men and 3 percent of women in this setting screened positive for alcoholism.

The sensitivity of the CAGE was found in this study to be universally poor in detecting heavy drinkers. At a cutoff of two affirmative CAGE answers, only 14 percent of those drinking in excess of the USDA's recommended limits were CAGE positive; since those limits are quite low, that finding was not unexpected. Only 40 percent of people who consumed more than 21 drinks per week were CAGE positive, and only 35 percent of binge drinkers were CAGE positive. Specificities, on the other hand, were quite good; the CAGE rarely misclassified a nonproblem drinker as a problem drinker.

We may conclude that, when used alone as a screening test in a primary care setting, the CAGE will detect people dependent on alcohol quite well. On the other hand, it is not very good at picking up those heavy drinkers who are not dependent, but who are nonetheless at risk for medical complications from their drinking. Including questions on the quantity and frequency of drinking will detect additional heavy drinkers, but further research is needed on screening for this level of medically hazardous drinking.

Another issue important in screening elderly people that has not been addressed in any study of alcohol screening tools is the high prevalence

of cognitive impairment in this age group. It is unlikely that any measure requiring self-report is sufficient to detect problem drinking in those individuals with memory impairment. Since alcohol is one of the most common truly reversible causes of dementia (Clarfield 1988), this issue is of prime importance and requires further study.

One very exciting development in the treatment of alcohol problems in recent years is the use of brief interventions by primary care doctors. In a British study of 909 people who reported drinking more than the Royal College of Physicians recommended limits (Wallace et al. 1988), the subjects were randomized to receive either a brief intervention by their primary care physician or no intervention. The intervention consisted of showing them how their consumption compared with other people's and advising them about potential harmful effects of their own current level of drinking. They were also given a booklet about harmful effects of drinking, were advised not to drink more than a certain quantity and frequency, and were asked to keep a drinking diary. They returned for followup at 4, 7, or 10 months at the discretion of their primary practitioner. At followup visits, they reviewed the drinking diary and were given feedback on the results of blood tests thought to indicate harmful effects of drinking.

After 1 year, the proportion of men who still drank in excess had decreased by 43.7 percent in the case group and 25.5 percent in the control group. In women, the proportion had decreased by 47.7 percent in the case group and 29.2 percent in the control group. They were also able to show small but statistically significant decreases in gamma-glutamyltransferase (GGT) and systolic blood pressure in the case group compared with controls (Wallace et al. 1988).

There have been no reports as yet on the effectiveness of this strategy among older people. However, Fleming, Barry, and Adams have been conducting a study to test the effectiveness of physician brief intervention for older problem drinkers in southeastern Wisconsin. After patients were screened by quantity and frequency measures, those who reported drinking heavily were invited to participate further in the study. Following in-depth interviews to confirm heavy drinking and exclude those who had symptoms or histories of alcohol dependence, 159 people were randomized to intervention or control groups. Preliminary results suggest that there is a substantial and significant difference in alcohol consumption between those who received the intervention and controls after 1 year (Fleming, Barry, and Adams unpublished data). This strategy has great potential to have an impact on alcohol-related problems in the primary care setting.

CONCLUSION

There have been very significant advances in the last 10 years in our understanding of the relationship between alcohol and health care. The prevalence of alcohol problems is clearly higher in health care settings than in the population at large, though

further research is needed to understand the complex relationship between alcohol and health services use. Screening for alcoholism can be effective in the primary care setting, using the very brief and convenient CAGE questionnaire, though additional quantity and frequency questions are needed to detect nondependent heavy drinkers. Lastly, brief intervention appears to be a very promising strategy to decrease problem drinking among elderly people in the primary care setting.

REFERENCES

Adams, W.L.; Garry, P.J.; Rhyne, R.; Hunt, W.C.; and Goodwin, J.S. Alcohol intake in the healthy elderly. *J Am Geriatr Soc* 38:211–216, 1990.

Adams, W.L.; Magruder-Habib, K.; Trued, S.; and Broome, H.L. Alcohol abuse in elderly emergency department patients. *J Am Geriatr Soc* 40:1236–1240, 1992.

Adams, W.L.; Yuan, Z.; Barboriak, J.J.; and Rimm, A.A. Alcohol-related hospitalizations in elderly people: Prevalence and geographic variation in the United States. *JAMA* 270:1222–1225, 1993.

Adams, W.L.; Barry, K.L.; and Fleming, M.F. Screening for problem drinking in older primary care patients. *JAMA* 276:1964–1967, 1996.

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 3d ed. Washington, DC: the Association, 1980.

Barnes, G.M. Alcohol use among older persons: Findings from a western New York State general population survey. *J Am Geriatr Soc* 27:244–250, 1979.

Bristow, M.F., and Clare, A.W. Prevalence and characteristics of at-risk drinkers among elderly acute medical inpatients. *Br J Addict* 87:291–294, 1992.

Buchsbaum, D.G.; Buchanan, R.G.; Lawton, M.J.; and Schnoll, S.H . Alcohol consumption patterns in a primary care population. *Alcohol Alcohol* 26:215–220, 1991.

Buchsbaum, D.G.; Buchanan, R.G.; Welsh, J.; Centor, R.M.; and Schnoll, S.H. Screening for drinking disorders in the elderly using the CAGE questionnaire. *J Am Geriatr Soc* 40:662–665, 1992.

Cahalan, D., and Cisin, A. American drinking practices: Summary of findings from a national probability sample. *Q J Stud Alcohol* 29:139–151, 1968.

Callahan, C.M., and Tierney, W.M. Health services use and mortality among older primary care patients with alcoholism. *J Am Geriatr Soc* 43:1378–1383, 1995.

Clarfield, A.M. The reversible dementias: Do they reverse? *Ann Intern Med* 109: 476–486, 1988.

Colsher, P.L., and Wallace, R.B. Elderly men with histories of heavy drinking: Correlates and consequences. *J Stud Alcohol* 51:528–535, 1990.

Curtis, J.R.; Geller, G.; Stokes, E.J.; et al. Characteristics, diagnosis, and treatment of alcoholism in elderly patients. *J Am Geriatr Soc* 37:310–316, 1989.

Eaton, W.W.; Kramer, M.; Anthony, J.C.; Dryman, A.; Shapiro, S.; and Locke, B.Z. The incidence of specific DIS/DSM-III mental disorders: Data from the NIMH Epidemiologic Catchment Area program. *Acta Psychiatr Scand* 79:163–178, 1989.

Fillmore, K.M. Women's drinking across the adult life course as compared to men's. *Br J Addict* 82:801–811, 1987.

Fried, L.P., and Wallace, R.B. The complexity of chronic illness in the elderly: From clinic to community. In: Wallace, R.B., and Woolson, R.F., eds. *The Epidemiologic Study of the Elderly.* New York: Oxford University Press, 1992.

Glynn, R.L., Bouchard, G.R.; Lo Castro, J.S.; and Laird, N.M. Aging and generational effects on drinking behaviors in men: Results from the Normative Aging Study. *Am J Public Health* 75:1413–1419, 1985.

Graham, K. Identifying and measuring alcohol abuse among the elderly: Serious problems with existing instrumentation. *J Stud Alcohol* 47:322–326, 1986.

Iliffe, S.; Haines, A.; Booroff, A.; Goldenberg, E.; Morgan, P.; and Gallivan, S. Alcohol consumption by elderly people: A general practice survey. *Age Ageing* 20:120–123, 1991.

Jones, T.V.; Lindsey, B.A.; Yount, P.; Soltys, R.; and Farani-Enayat, B. Alcoholism screening questionnaires: Are they valid in elderly medical outpatients? *J Gen Intern Med* 8:674–678, 1993.

Joseph, C. Alcohol and drug misuse in the nursing home. *Int J Addict* 30:1953–1984, 1995.

Klatsky, A.L.; Armstrong, M.A.; and Friedman, G.D. Alcohol and mortality. *Ann Intern Med* 117:646–654, 1992.

Leigh, J.P., and Fries, J.F. Health habits, health care use and costs in a sample of retirees. *Inquiry* 29:44–54, 1992.

Magruder-Habib, K.; Saltz, C.C.; and Barron, P.M. Age-related patterns of alcoholism among veterans in ambulatory care. *Hosp Community Psychiatry* 37:1251–1255, 1986.

Mangion, D.M.; Platt, J.S.; and Syam, V. Alcohol and acute medical admission of elderly people. *Age Ageing* 21:362–367, 1992.

Mellstrom, D.; Rundgren, A.; and Svanborg, A. Previous alcohol consumption and its consequences for ageing, morbidity and mortality in men aged 70–75. *Age Ageing* 10:277–286, 1981.

Myers, J.K.; Weissman, M.M.; Tischler, G.L.; Holzer, C.E.; Leaf, P.J.; Orvaschel, H.; Anthony, J.C.; et al. Six month prevalence of psychiatric disorders in three communities. *Arch Gen Psychiatry* 41:959–967, 1984.

Rice, C., and Duncan, D.F. Alcohol use and reported physician visits in older adults. *Prev Med* 24:229–234,1995.

Rimm, E.B.; Giovannucci, E.L.; Willett, W.C.; Colditz, G.A.; Ascherio, A.; Rosner, B.; and Stampfer, M.J. Prospective study of alcohol consumption and risk of coronary disease in men. *Lancet* 338:464–468, 1991.

Rivara, F.P.; Jurkovich, G.J.; Gurney, J.G.; Seguin, D.; Fligner, C.L.; Ries, R.; Raisys, V.A.; and Copass, M. The magnitude of acute and chronic alcohol abuse in trauma patients. *Arch Surg* 128:907–913, 1993.

Rothman, K.J. Causal inference in epidemiology. In: Rothman, K.J., ed. *Modern Epidemiology.* Boston: Little, Brown, 1987. p.11.

Speckens, A.E.; Heeren, T.J.; and Rooijmans, H.G. Alcohol abuse among elderly patients in a general hospital as identified by the Munich Alcoholism Test. *Acta Psychiatr Scand* 83: 460–462, 1991.

Stall, R. Change and stability in quantity and frequency of alcohol use among aging males: A 19 year follow-up study. *Br J Addict* 84:537–544, 1986.

Stinson, F.S.; Dufour, M.C.; and Bertolucci, D. Alcohol-related morbidity in the aging population. *Alcohol Health Res World* 13:80–87, 1989.

Tabisz, E.; Badger, M.; Meatherall, R.; Jacyk, W.R.; Fuchs, D.; and Grymonpre, R. Identification of chemical abuse in the elderly admitted to emergency. *Clin Gerontologist* 11:27–39, 1991.

Temple, M.T., and Leino, E.V. Long-term outcomes of drinking: A 20-year longitudinal study of men. *Br J Addict* 84:889–899, 1989.

Wallace, P.; Cutler, S.; and Haines, A. Randomised controlled trial of general practitioner intervention in patients with excessive alcohol consumption. *BMJ* 297:663–668, 1988.

Waller, J.A. Falls among the elderly—human and environmental factors. *Accid Anal Prev* 10:21–33, 1978.

Wechsler, H.; Kasey, E.H.; Thum, D.; and Demone, H.W. Alcohol level and home accidents. *Public Health Rep* 84:1043–1050, 1969.

Chapter 19

Older Alcohol Abusers: Recurring Treatment Issues

Larry W. Dupree, Ph.D., and Lawrence Schonfeld, Ph.D.

For approximately the last 15 years, certain questions and issues regarding treatment of older alcohol abusers have surfaced repeatedly when we have made presentations at conferences and seminars and provided consulting services to organizations. Often the questions have come out of the questioner's theoretical bias or preferred way of treating people (of any age) who were abusing alcohol. The questions and issues have centered on the following areas:

- Means to identify older alcohol abusers
- How to encourage voluntary admission
- Instruments useful in defining person-appropriate interventions
- Demonstration of effective programs with older alcohol abusers
- Aids to knowing when discharge is appropriate
- General program issues, which often reflect treatment staff philosophy or bias, often without supportive data (e.g., age-specific vs. age-heterogeneous treatment, preferred treatment format, the necessity of social support network development, how to respond to denial and whether confrontation should be permitted, the necessity for abstinence vs. negotiated drinking goals, how to respond to dually diagnosed older alcohol abusers, and assessment of treatment outcome).

We reviewed the published literature regarding older alcohol abusers for "answers" to these issues, and we present our findings in this chapter.

L.W. Dupree, Ph.D., is acting chair and L. Schonfeld, Ph.D., is professor in the Department of Aging and Mental Health, Louis de la Parte Florida Mental Health Institute, University of South Florida, 13301 Bruce B. Downs Blvd.,Tampa, FL 33612.

We close the chapter with brief sections on treatment approaches that have been validated empirically, a suggested cognitive-behavioral and self-management assessment and intervention model, and general conclusions and recommendations that have empirical support relative to the treatment of older alcohol abusers.

PRETREATMENT SCREENING

Despite recent reports that older alcohol abusers are identified within hospital settings, such as general inpatient admissions and emergency room visits (e.g., Adams and Cox 1997), few older adults enter treatment programs. Recognition of community-residing older alcohol abusers who are not seen in hospital settings is difficult. Research indicates that relatively few physical or medical indicators differentiate older from younger alcohol abusers or from older non-problem drinkers (Hurt et al. 1988). Most nonmedical assessments that rely on screening instruments involving interviews or self-administered questionnaires are inappropriate for older persons because the indicators they use are more common to younger people (e.g., problems at work, family/marital problems, and driving while intoxicated). To address this problem, Graham (1986) suggested adding domains that apply to the elderly such as age-specific criteria for defining "heavy" drinking, housing problems, falls and accidents, poor nutrition, poor activities of daily living, lack of exercise, and social isolation.

Some instruments, such as the Drinking Problems Index (DPI) (Finney et al. 1991) and the Michigan Alcoholism Screening Test—Geriatric Version (MAST-G) (Blow et al. 1992*a*), include items thought to be more common for older adults. Although these measures may be useful for people who admit to alcohol problems (e.g., those in treatment), they may have little screening/identifying value for the vast majority of elderly alcohol abusers who appear at general health and social services organizations but do not self-refer for alcoholism treatment. Also, even if a person is willing to give an accurate report of alcohol consumption, he or she may not be willing to admit to drunkenness, extreme withdrawal symptoms, or psychological dependence. In summary, alcohol screens for older people in general, not in treatment centers or admitting to alcohol problems, have had limited success (Graham 1986; DeHart and Hoffman 1997).

Case Finding

Dupree (1989) compared the effectiveness of three case-finding strategies: a community-based outreach involving staff visits to health centers, a public awareness campaign (intensive media campaign), and a community agency referral network involving various community agencies serving older adults. Results revealed that educating agencies serving older adults produced the greatest number, as well as the most appropriate referrals, of older alcohol abusers. Relatively few referrals resulted from expensive media and public

awareness campaigns, or from on-site staff at public health clinics. Case-finding approaches, as well as techniques for engaging older abusers in treatment once identified, are critical to early intervention and prevention, but have been minimally developed and researched. Many community programs complain of the great difficulty in getting older adults to present themselves for "nonmedical" care.

INTERVIEWING APPROACHES

Professionals who work in the field of addictions often are perplexed when people of any age, who appear to need treatment for substance abuse problems, do not enter treatment. Several reasons may explain the difficulty in convincing older people to enter substance abuse treatment. First, the professional may be incorrect about the existence of the problem, perhaps due to faulty assumptions or inaccurate referral information. Second, the older individual may react to the style or approach taken by the professional. Many professionals have been trained to "confront" alcohol abusers, but this may be counterproductive in some cases, particularly with older people. There is evidence indicating that older alcohol abusers in treatment respond better to supportive rather than confrontive approaches (Kashner et al. 1992). Furthermore, many older individuals are likely to have experienced age-related losses (e.g., death of a spouse, retirement). Using confrontation in such cases may only serve to heighten negative affect such as depression. When confronted or labeled, people who are ambivalent about entering treatment may become more resistant to seeking help; this resistance often leads to a misinterpretation that the person is in denial when he or she may be reacting to a threat to their self-esteem (Miller and Rollnick 1991). Finally, the professional may be guilty of negative attitudes toward older adults (i.e., professional ageism). Ageism subtly affects the screening, diagnosis, and treatment of older adults and may lead to the misinterpretation of behavior problems as age related (Dupree and Patterson 1985; Edelstein et al. 1996).

Rather than confronting older adults, or applying inappropriate stereotypes or biases, several strategies have been suggested to encourage professionals to identify and encourage alcohol abusers to enter treatment. Blake (1990) reported that a major strategy that mental health counselors can use to increase the number of older alcohol abusers in treatment is to be optimistic and to share that optimism (i.e., that older problem drinkers can be helped).

The "transtheoretical model" offered by Prochaska, DiClemente, and Norcross (1992) provides a more empirical basis for understanding the level of motivation substance abusers have with respect to entering treatment. The model describes five stages of change, through which individuals may "spiral" upward or downward with respect to changing their addictive behavior. Individuals in the earliest stage, the *precontemplation* stage, have no intention of changing behavior

and are unaware or underaware of their problems. Such precontemplators are not open to discussion about their problems and are unlikely to react well to strategies such as consciousness raising, confrontation, or interpretation. At the *contemplation* stage, individuals are aware of the problem and may be thinking about overcoming it, but have not committed to do so. At the *preparation* stage, individuals decide what steps must be taken to overcome the problem and intend to take these steps in the near future. At the *action* stage, individuals begin to modify behavior, experiences, or environment to overcome the problem. Finally, in the *maintenance* stage, individuals work "to prevent relapse and consolidate the gains attained" in the action stage. The professional needs to understand the stage at which the individual presents in order to identify the appropriate strategy for helping the individual accept the need for change and, perhaps, professional assistance.

Miller and Rollnick's *Motivational Interviewing* (1991) provides a framework for understanding how an individual responds to confrontation and labeling by the professional, and also provides a strategy to assist the professional in moving the individual from the precontemplation stage to the contemplation stage or higher. For example, Miller and Rollnick suggested that denial is not a defense mechanism the addict experiences as an inherent part of the disorder, but rather a defensive response of the precontemplator to a therapist's aggressive approach. The precontemplator, who is unaware or ambivalent about the problem, may be forced into a defensive posture if labeled as an alcoholic or addict or accused of being in denial. Because the precontemplator does not agree with and is defensive about the label or accusation, he or she may take a stronger position away from, rather than toward, accepting help for the problem.

Rather than confront, Miller and Rollnick suggested a variety of strategies to help the client move along the stages of change. Confrontation is avoided, and, instead, the emphasis is placed on the client personally providing more and more evidence that the problem exists. By creating cognitive dissonance, the therapist encourages the ambivalent client to acknowledge more and more evidence that problems exist. As the individual expands a personal list of problems, it is the client rather than the therapist who should make statements such as: "This problem is more serious than I realized" (p. 58).

Our experience with older alcohol abusers suggests that such a philosophy works well. In the Gerontology Alcohol Project (GAP) (Dupree et al. 1984), confrontation of clients was not permitted in the case finding/screening stages of the program nor in the active phase of treatment. With respect to screening or identifying potential clients, we approached the issue by assuming that older people were more willing to talk about multiple problems and preferred not to be labeled as alcoholic, especially by staff members who might be much younger (and perhaps less experienced) than themselves. When screening, staff considered

alcohol abuse as a *behavior* with highly individualized, recurring *antecedents.* Therefore, the focus was often on the negative affect and social isolation that preceded drinking. Such problems do not lend themselves well to confrontation, nor do they require labels such as "denial" or "alcoholic." The older adult who can recognize that feeling sad or lonely, bored, or isolated is likely to lead to drinking on that same day may be encouraged to try new approaches to overcoming these emotional states.

ADMISSION TO TREATMENT

Irrespective of age, research suggests that relatively few individuals appropriately referred for treatment actually enter treatment. A number of strategies may be useful in motivating the ambivalent older individual to accept professional help. First, as already noted, while traditional approaches place emphasis on the therapist as the expert, the motivational interviewing approach emphasizes the potential client's role in identifying the evidence or building the case that a substance abuse problem exists. The approach also allows the therapist to help the client "move" from the precontemplation stage to higher stages in the acceptance of change. Second, confrontational approaches should be avoided because they are likely to discourage the ambivalent older individual by forcing him or her into a defensive position (Kashner et al. 1992). Third, staff working with older drinkers not only should be trained in psychopathology (including the addictions), but also should be interested in, and trained to work with, older adults (Flemming et al. 1984, 1986). Finally, offering programs specifically for older adults is more likely to encourage admission, program completion, and more positive treatment outcome (Kofoed et al. 1987; Atkinson 1995).

TREATMENT ISSUES

Once the individual has agreed to enter treatment, the next issue involves the characteristics of the treatment program. In this section we discuss how assessment of individual behavior, coping responses, and alcohol expectancies can help program counselors or therapists plan appropriate interventions.

Assessment and Treatment Planning

Once the older abuser has been admitted, it is necessary to identify the problem areas and the individual's strengths and weaknesses and to develop the short- and long-term goals for treatment. The approach we have employed begins with a structured, behavioral interview to identify the individual's "drinking behavior chain," consisting of each person's antecedents for, and consequences of, drinking. Both the GAP Drinking Profile (Dupree et al. 1984) and the more recently developed Substance Abuse Profile for the Elderly (Schonfeld and Dupree 1996) permit the counselor or therapist to elicit and diagram the older person's drinking or substance use chain (i.e., the situations, thoughts, feelings, cues, urges, and self-statements that

typically lead to the first drink on a drinking day, and the consequences that follow). Intervention techniques can then focus on the personalized drinking behavior chain, and chain-specific coping skills can be introduced and taught to preclude or disrupt its expression. Self-management is the teaching, acquisition, and use of specific coping behaviors for specific individuals regarding their specific antecedents.

By requiring each person to specify the likely triggers for drinking on a given day, and determining the consequences, the professional can also predict what are likely to become the determinants of relapse following completion of treatment and can teach the skills necessary to prevent this behavior from occurring. For older adults, high-risk situations are usually feelings of depression, loneliness, or boredom (Dupree et al. 1984; Schonfeld and Dupree 1991).

An additional value of defining drinking behavior antecedents (as part of the drinking behavior chain) relates to the ability to determine when someone is ready for discharge. Within the self-management model, when an individual has acquired and can successfully enact coping behaviors specific to the antecedents for drinking, plans to gradually wean the person from the program might be considered. Discharge prior to that point of specific skill acquisition is almost certain to result in relapse (and likely very soon). Thus, a person may be considered for discharge when the coping behaviors necessary to precluding or breaking the abuse chain have been acquired.

Coping Style and Alcohol Expectancies

Marlatt and Gordon (1985) investigated determinants of relapse and found that negative emotional states, social pressure, or conflicts with other people often were reported as preceding a relapse. In contrast, few people reported that physical withdrawal, cravings, or positive emotions preceded a relapse. In their relapse prevention model, Marlatt and Gordon proposed that individuals who experience high-risk situations for drinking have a higher probability of drinking when their coping responses are deficient and/or their self-efficacy (the confidence in their ability to cope) is reduced. As a result, they have a higher positive expectancy of the effects of alcohol, which then increases the likelihood of a lapse (slip), followed by an "abstinence violation effect" (i.e., the reactions of cognitive dissonance and self-blame experienced when the typical goal of abstinence is violated, even by one slip), and, lastly, an increased probability for relapse (return to abusive drinking). The availability of a greater variety of coping responses and an increased perception of self-efficacy are hypothesized to reduce the risk of inappropriate alcohol use (Litman et al. 1979; Cronkite and Moos 1980; Finney et al. 1980; Rosenberg 1983; Annis and Davis 1988). In contrast, the person most vulnerable to abuse of alcohol is one whose coping resources are deficient relative to his or her personal high-risk situations.

Moos and colleagues (1990) reported that older adults who relied more on

avoidance (emotional) coping (focusing on avoiding/minimizing unwanted thoughts, feelings, etc.) tended to have more drinking problems and to report more depression and physical symptoms and less self-confidence; whereas older adults manifesting problem-focused coping styles (attempts to resolve the bases of problems and attendant emotions, etc.) had fewer problems. Also, different environmental circumstances, as well as the content of stressful events, suggest that the skills required for successful abstinence or appropriate alcohol use in older adults may differ from those in younger adults (McCrae 1982, 1989).

Alcohol expectancies or the anticipated effect of alcohol use is also an important aspect of the model. Individuals who tend toward heavier alcohol use have been found to expect a significantly greater percentage of positive experiences as a result of use and to display a preponderance of expectations for positive reinforcement from alcohol consumption (Farber et al. 1980). However, even though alcohol expectancies have been demonstrated to be predictors of alcohol use among adolescent and younger adult populations (Brown 1985; Smith et al. 1986; Goldman et al. 1987; Roehling et al. 1987; Christiansen et al. 1989), they have only been explored minimally relative to older adults.

Dupree (1992) identified alcohol expectancies of older adults via an interview process. Older interviewees generated 179 expectancies regarding alcohol use, with 114 not being redundant. On an arbitrary basis for comparison purposes, the 114 expectancies were categorized using the factor scales (subcategories) of the alcohol expectancy questionnaires identified by Brown and colleagues (1980) and Christiansen and colleagues (1982). Brown's six factors were as follows: (1) alcohol is a global positive transforming agent, (2) alcohol enhances social and physical pleasure, (3) alcohol enhances sexual experiences, (4) alcohol increases power and aggression, (5) alcohol increases social assertiveness, and (6) alcohol promotes relaxation and tension reduction. Christiansen's nonoverlapping subscales generated by adolescents included (7) changes in social behavior; (8) improved cognitive and motor abilities, and (9) cognitive and motor impairment. Most of the older adult expectancies were located in factors 1, 5, and 6.

Subsequently Dupree (unpublished data) asked 200 community-residing older adults (ages 51–91; mean = 71.5; median = 72) to respond to a "questionnaire" comprised of expectancy-based statements prompted by the initial older adult alcohol expectancies. Examples of frequently endorsed alcohol expectancy statements were as follows:

- I don't feel ignored when I am drinking. (55 percent men, 46 percent women)
- I feel more comfortable after a few drinks. (56 percent men, 39 percent women)
- For a short time, the alcohol takes over and gives me a sense of well-being. (61 percent men, 55 percent women)

- I drink to feel less lonely. (36 percent men, 36 percent women)
- A few drinks gives me the courage to go on. (36 percent men, 37 percent women)
- I drink to be calmed. (36 percent men, 41 percent women)
- If I drink I feel pretty good. (63 percent men, 42 percent women)
- When I drink it gives me something to do. (42 percent men, 37 percent women)
- I am less bored when I drink. (45 percent men, 44 percent women)
- Drinking helps me pass the time away. (33 percent men, 33 percent women)
- A few drinks helps to soften my grief. (35 percent men, 40 percent women)
- I drink as a method of stress reduction. (40 percent men, 42 percent women)

Thus, preliminary results suggest that older people anticipate positive consequences of alcohol use, primarily in response to alleviating negative emotional states.

If the utility of assessed alcohol expectancies can be demonstrated for older adults as it has been for adolescents and younger adults, specification of alcohol expectancies may be indicative of current abuse, as well as predictive of later abuse, for an older individual and may permit better tailoring of both intervention and prevention efforts. Knowing what an individual expects from alcohol consumption typically specifies the immediate (or short-term) consequences of alcohol use for that individual; consequences that are responsible for sustaining repeated alcohol use unless those consequences are negative. Such information is extremely valuable in planning alcohol interventions because specific deficit areas and specific alcohol expectancies (consequences) can be addressed individually. For example, "drinking makes the future seem brighter" implies negative affect and beliefs, as well as a self-medicating base that is likely to continue. Also, if an older adult's alcohol expectancy is that drinking will lift depression, then a different intervention approach is likely than if the expectancy is that drinking gives the person something to pass the time away. Each expectancy implies an antecedent condition one wants to alter, if only for a little while. Thus, an individual's expectancies of alcohol use may tell us a great deal of that person's state prior to the first drink on a drinking day, motivations for drinking, what coping skills are deficient, why the behavior continues, and where intervention needs to begin (Dupree et al. 1984).

GENERAL PROGRAM ISSUES

Often treatment staff ask questions related more to program and general service delivery issues, as well as treatment philosophy, than to intervention "how-to" specifics. Based upon a review of published literature, we attempt to respond to areas of concern frequently raised.

Age-Specific Treatment

Over the last 10–15 years, there has been an ongoing debate concerning

whether older adults are better off participating in age-specific treatment programs than mixed-age programs where they are "mainstreamed" with younger alcohol or substance abusers (Kofoed et al. 1987). The same debate has occurred within the mental health system, partly in response to the recognition that very few older people use the services of mental health centers. However, when centers offer age-specific programs with staff experienced in aging issues, use by older adults increases (Flemming et al. 1984, 1986; Light et al. 1986; Lebowitz et al. 1987; Lebowitz 1988). Similarly, in the treatment of alcoholism, research suggests that age-specific group treatment may be more effective. In chapter 24, Dr. Atkinson presents an excellent review of the available data regarding the age-specific treatment issue.

Group Treatment

Within the literature regarding treatment of older alcohol abusers, group approaches are emphasized as the basic treatment venue, with individual work rarely used as an adjunct. According to Hinrichsen (1984), "group therapy, whether based on an AA [Alcoholics Anonymous] model or some other type of social support model, appears to be the single most crucial aspect of treatment for elderly people" (p. 35).

Zimberg (1979, 1984, 1985) stated that it is more important to treat the older alcohol abuser through group intervention, which addresses isolation and depression. Socialization and treatment of depression are essential components of his treatment method for the older adult with alcohol problems. He noted that "disulfiram, Alcoholics Anonymous, and referral to alcoholism treatment programs, frequent requirements for the treatment of younger alcoholics, were not necessary for these older patients" (Zimberg 1984, p. 27), although empirical support was not provided. We have emphasized placing attention on the losses experienced by the older adult and teaching self-management behaviors in a group format, with confrontation by staff or other clients within the group being discouraged (Dupree et al. 1984; Schonfeld and Dupree 1995; Dupree and Schonfeld 1996*a*; Schonfeld and Dupree 1997).

Social Support

Kofoed and colleagues (1984, 1987) described the "Class of 45," a veterans outpatient program in Portland, Oregon. This program offered age-specific groups, open-ended discussions, staff experienced in aging, concurrent treatment of mood disorders, counseling for spouses, and monthly social events and activities. Blake (1990) advocated "social therapies," meaning the rebuilding of social networks (e.g., family members, AA, peer groups, and self-help organizations), as well as assistance services provided by community agencies familiar with aging-related problems.

In certain areas of the country, such as states with large retirement areas, it may be a rare event when older drinkers have family with whom they interact and are in close proximity. For example, Dupree and colleagues

(1984) in Florida found that, on average, the total size of the social network for late-onset alcohol abusers was four friends and four family members. However, daily contacts averaged less than one family member or one friend. Half were widowed or divorced; thus, many (67 percent) drank alone. We found similar characteristics in comparing late- and early-onset older alcohol abusers, concluding that through either losses or alienation, the loss of social support was a significant variable contributing to the drinking problem (Schonfeld and Dupree 1991). Thus, family intervention may be difficult, because there may be few significant others in proximity or with the interest to work on the problem.

A component in the GAP (Dupree et al. 1984) was a social support network module. This group intervention was designed to teach clients the communication and interpersonal skills necessary to make new friends and identify and engage in new social activities. Although it was not possible to determine which components of treatment led to positive treatment outcomes, the results indicated a significant increase in the size of the social support network (the number of friends and relatives with whom the person is in contact) at discharge, and again at 12-month followup.

Denial and Confrontation

Confrontation techniques are often used in 12-step programs because of the assumption that many alcoholics experience denial of their drinking problem. However, it is often recommended that confrontation with older alcohol abusers be avoided. Schiff (1988) commented that confrontation does not work with older individuals who need less threatening approaches. Yet, in describing the process of referring older adults for treatment, Hinrichsen (1984) recommended confrontation as a first step, while warning that the "counselor should avoid the label of 'alcoholic,' as well as judgmental, blaming, or punitive statements" (p. 34).

Evidence against using confrontation was offered by Kashner and colleagues (1992), who compared two groups of veterans, ages 45 and older. One group consisted of 65 veterans in a traditional confrontation-oriented treatment program. The second group consisted of 72 veterans in a program emphasizing peer relationships, promoting self-esteem, being "tolerant of patients' failures," and prohibiting confrontation. Results indicated that the nonconfrontation group was twice as likely to report being abstinent at 12-month followup. The results of this study suggest that the supportive approach has positive effects on treatment outcome, while use of confrontation has deleterious effects.

We found that not permitting confrontation facilitated more open discussion between staff and clients, encouraging clients to report "slips" when they occurred (Dupree et al. 1984). Each slip was diagrammed in terms of that person's drinking behavior chain, along with the antecedent conditions and consequences, in order to teach group members how to avoid or manage their high-risk situations.

Regardless of age, anticipated or experienced confrontation may dissuade alcohol abusers from entering into or remaining in treatment (Miller and Rollnick 1991). Staff can learn to be honest with older abusers, and teach group members the same, without being perceived as cruel or "in your face." Perhaps the "confrontation" of Miller and Rollnick, namely the facilitation of client-generated cognitive dissonance, is more effective in the long run, even if slower in developing.

Abstinence

Regarding abstinence as a required treatment goal, Kelly and Remley (1989), representing a social work/counseling perspective, said that "counselors must be guided by their personal convictions, by their clients' wishes, and by the philosophy of the agency they represent when setting treatment goals for elderly alcoholics" (p. 110). They stated that expecting abstinence in all older adults may be unrealistic, but they also suggested that group cohesion is not likely to develop in groups in which members are not working toward the common goal of total sobriety. If abstinence can be required or not according to the specific circumstances of the staff and clients, what does this say about the "therapeutic necessity" of sobriety and abstinence heavily emphasized in various treatment programs? Data supporting both positions are available, and more writers are beginning to view older alcohol abusers as possible exceptions to abstinence as a therapeutic necessity. Like Kelly and Remley, Blake (1990) tempers the need for abstinence as a treatment goal for older problem drinkers. However, selecting a treatment goal that includes continued drinking should be a very careful decision made by both a well-informed counselor and well-informed client. It is recommended that the therapist consider the individual and the precise nature of the drinking problem.

Dual Diagnosis

Little information has been available regarding treatment of the older patient with comorbid mental and substance disorders (often called dually diagnosed), yet the likelihood of older adults within either psychiatric or addictions programs having multiple mental health problems is fairly great. For example, Speer and colleagues (Speer et al. 1991; Speer and Bates 1992) found older dually diagnosed clients to have a higher frequency of diagnoses of personality and affective disorders, but a lower incidence of schizophrenia typically observed in younger patients with comorbidity.

Blow and colleagues (1992*b*), in a nationwide survey of the Veterans Affairs mental health system, found that over 20,000 veterans with a presenting diagnosis of alcoholism sought outpatient mental health services during a 1-month period. Compared with younger adults, older adults had a greater prevalence of organic brain syndrome or dementia, impairment in adaptive functioning (social relations, occupational functioning, and use of leisure time), and major depression. Thus, a comprehensive evaluation of the older abuser (particularly the

older abuser whose alcohol problem is identified to have begun earlier in life) seems warranted. The literature suggests that later-life onset abusers appear to be healthier psychologically than their earlier-life onset counterparts, drinking primarily in response to later-life changes and losses rather than as a result of long-term psychopathology. For a more definitive review of this issue, see chapter 23, in which Dr. Willenbring discusses treatment of comorbid conditions.

Treatment Outcome

"Success" in the treatment of alcoholism may be judged by a number of measures: completion of the program, acquisition of appropriate skills to avoid alcohol abuse, abstinence or modification toward non-problem drinking, and improvement in psychological and social functioning. According to Blake (1990), the variety of drinking behaviors in older adults suggests that a range of counseling goals may be appropriate and that "a broader range of treatment strategies is needed than has characterized traditional alcoholism treatment programs" (p. 354).

Evaluation of treatment for alcoholism often focuses on the presence or absence of drinking. However, if abstinence is the only measure of success, there will be few successes. Also, Atkinson (1995) stated that because problem drinking status in old age is unstable, particularly for late-onset older alcohol abusers, positive treatment outcome may not be indicative of future positive prognosis. He further reported that neither compliance during treatment nor short-term outcome after treatment may be a good indicator of longer term favorable prognosis. Thus, outcome evaluation should take place over a substantial period of time and should consider outcome measures other than abstinence alone.

EMPIRICALLY VALIDATED TREATMENT APPROACHES

Little research-based information is available regarding effective forms of treatment for older alcohol abusers. However, a few studies have reported outcome evaluation data suggesting positive treatment outcomes for older adults; these outcomes are at least as promising as, if not better than, those for younger adults in treatment (Schonfeld and Dupree 1995, 1997). Only one study has used a control group outcome approach with random assignment of subjects to treatment conditions (Kashner et al. 1992), and only two studies have attempted to investigate the need for age-specific treatment programs by comparing treatment outcomes for older adults with those for younger adults (Janik and Dunham 1983; Kofoed et al. 1987). Studies employing behavioral interventions show promise, as indicated by high rates of success after 1 or more years of followup (Schonfeld and Dupree 1995, 1997).

Unfortunately, there has been little evaluation of 12-step programs, or of older adults in 12-step programs, other than a very few studies that include other modalities to constitute a more eclectic approach, making it difficult to determine which component of the

treatment program has the greatest utility. Those few reports on older people in 12-step programs offer only anecdotal remarks such as the experiences were found to be positive, or the older participants enjoyed the lack of profanity and the opportunity to share similar issues (Schiff 1988; Dunlop 1990). However, few studies have evaluated 12-step approaches with the older population (Solomon et al. 1993; Schonfeld and Dupree 1995, 1997). Holder and colleagues (1991) placed AA in the "insufficient evidence of effectiveness" category. Supportive data for other nonbehavioral treatment models for older alcohol abusers also could not be found within the published literature (Schonfeld and Dupree 1995, 1997).

In the category of behavioral interventions with older alcohol abusers, aversion therapy using counterconditioning with emetine-induced nausea has been found to be effective (Wiens et al. 1982–83). However, few programs actually use this technique. There may be substantial medical risks for an older person; reactions to emetine may include hypotension as well as nausea and vomiting. In contrast, cognitive-behavioral and self-management approaches have shown low rates of relapse during followup, without producing medical risks (Dupree et al. 1984; Carstensen et al. 1985; Blow et al. unpublished manuscript 1996).

Carstensen and colleagues (1985) conducted a followup on 16 of 25 men, 65–70 years of age, who completed a behavioral treatment program for alcohol problems 2–4 years earlier. This inpatient treatment program at a veterans hospital included individual counseling, alcohol education, self-management and problem-solving skills, vocational assistance, and marital therapy. Based on followup interviews with the 16 subjects and "significant others," six were drinking abusively (consuming the equivalent of 8 oz of hard liquor per drinking day).

Dupree and colleagues (1984) reporting on a cognitive-behavioral/self-management program, noted that it was effective in preventing relapse among late-onset alcohol abusers. Blow and colleagues (unpublished manuscript 1996) found that 54 percent of their patients reported abstinence from alcohol at 6 months in their cognitive-behavioral, elder-specific, inpatient alcohol treatment program. In addition, psychological distress decreased dramatically between baseline and followup for abstainers, with only a small decline of distress for relapsers.

A Cognitive-Behavioral and Self-Management Model

Our studies at the Florida Mental Health Institute have focused on cognitive-behavioral and self-management interventions for older adults, age 55 and over (Dupree et al. 1984; Schonfeld and Dupree 1991, 1995; Dupree and Schonfeld 1996*a*, 1996*b*; Schonfeld and Dupree 1997). This approach has been widely used throughout the United States in diverse organizations, with diverse age groups and substance users (e.g., criminal justice systems, mental health systems, aging and adult systems, veterans programs, inpatient and outpatient

systems, DUI programs, and independent practice).

The therapeutic aim of a self-management approach is for the client to assume primary responsibility for personal behavior. We have followed three steps in teaching self-management skills to older substance abusers. The first step involves the functional analysis of the older abuser's drinking behavior using a structured interview which incorporates an A-B-C (antecedents-behavior-consequences) paradigm. In this A-B-C approach, the therapist and client identify, often for the first time, the contingencies related to abusive drinking behavior (i.e., the drinking behavior chain) (Miller 1977). The antecedents are the high-risk situations for abuse and potential determinants of relapse after treatment if sufficient coping skills and feelings of personal efficacy are not generalized to the everyday environment. We used the GAP Drinking Profile (Dupree et al. 1984) to identify alcohol use history as well as antecedents and consequences (e.g., thoughts, negative self-statements, situations, activities, feelings, cues, and urges) of alcohol use on "a typical day of drinking." Based on the GAP Drinking Profile, a new instrument, the Substance Abuse Profile for the Elderly, was developed for use with older individuals using alcohol and/or other drugs (Schonfeld and Dupree 1996).

The second stage involves teaching the client to recognize high-risk situations (antecedents) for abusive drinking. In the GAP, we developed an A-B-C treatment group in which the information identified in the GAP Drinking Profile was used in teaching each person to understand his or her drinking behavior chain. Analysis of individual drinking behavior chains provides both insight and instruction in ways to rearrange or control one's personal antecedents of abuse. It also denotes the degree of change necessary to one's current lifestyle, wanting to be only as disruptive as necessary for positive change.

The third, and perhaps most complex, step consists of teaching more effective coping skills specific to the identified antecedents. The absence of relevant coping skills in high-risk situations, in the presence of self-defeating cognitions/misperceptions, often leads to sustained alcohol use or abuse. Skills may be learned through a variety of approaches, such as behavior rehearsal/role playing, cognitive restructuring, problem-solving techniques, homework assignments, and self-monitoring of drinking behavior, cues, or urges. Newly learned self-directed behaviors have the capacity to disrupt, terminate, or preclude the individual's drinking chain. In the GAP, we did not socially reinforce or punish drinking behavior per se, but instead reinforced the older abuser's efforts to control drinking behavior by using learned techniques.

Acquisition and use of these skills very often increase interpersonal effectiveness and interpersonal attractiveness (i.e., the ability to manage one's interpersonal life in a fulfilling and adaptive manner, while also being concerned about the welfare of others), and aid in replenishing diminishing social support networks. Also, self-monitoring and self-management responses decrease the likelihood of abusive drinking, and

are not merely alternatives to drinking. If the person experiences a slip while in treatment, the counselor and client can discuss what skills were not used, and what to do about it should the same circumstances arise again. Finally, interventions can be evaluated and modified as needed, selectively altering the client's lifestyle only to the degree necessary.

Self-control or self-management in this context is not "willpower," but rather is knowledge of what causes personal abusive drinking, what specifically to do about it, and then how to do it. In the GAP, older adults were taught the behaviors necessary for "inoculation" against potential relapse situations and management of a minimum of certain high-frequency, high-risk situations: negative affect (e.g., depression, grief, sadness, loneliness), social pressure, anger/frustration, tension/anxiety, presence of drinking cues, and urges to drink. The self-management model discounts the self-fulfilling prophecy that having one drink will automatically, and inevitably, result in drunkenness, as well as a return to abusive drinking. We emphasize and reinforce self-control with older abusers who are taught that they can stop, refrain from negative (condemning) self-statements, and use the self-management skills they have acquired (Dupree et al. 1984). Thus relapse prevention includes self-monitoring and behavioral assessment; acquisition of relevant coping skills (based on the assessment); and increased self-efficacy and knowledge of the effect of alcohol. It is an inherent, rather than a postdischarge, treatment concern. Relapse prevention begins from the moment of analysis of drinking behavior, and continues right through training specific to individual antecedents.

The value of a self-management approach to older adults, in general, is that they can acquire the knowledge and skills necessary to maintaining themselves even in the presence of recurring aging-related change. Because most abusive drinking patterns have multiple antecedents and may change over time, the training of clients in the analysis of drinking chains and self-management skills allows them to be better prepared for future and evolving high-risk situations. People in transition need "strategies" or coping behaviors generalizable across time, events, and situations. They are prepared to analyze, understand, problem solve, and respond to the factors affecting substance use. Also, acquisition of critical skills enhances both actual and perceived self-control. Within this model, self-control or self-management is defined as the ability to recognize and then manipulate the factors influencing either excessive drinking or problems in living.

LITERATURE-BASED CONCLUSIONS AND RECOMMENDATIONS

It is unfortunate that much of the literature on the older alcohol abuser consists of dated reviews and surveys, as well as speculation and opinions based on previous speculations and opinions. We have little research-based information regarding assessment and effective forms of treatment for older alcohol abusers. Many experts offer recommen-

dations for the treatment of older alcohol abusers without providing evidence supporting those recommendations.

In recent reviews of the literature, we noted six conclusions about treatment of the older alcohol abuser that have empirical support (Schonfeld and Dupree 1995, 1997):

- Emphasize age-specific, group treatment with supportive approaches, avoiding confrontation.
- Focus on negative emotional states such as depression, loneliness, and overcoming losses (e.g., death of a loved one or retirement).
- Rebuild the social support network (i.e., teach the person the skills necessary to rebuild the network).
- Employ staff who are experienced in working with older persons and have interest in doing so.
- Develop linkages with aging services, medical services, and institutional settings, for both referral into treatment and referral out, as well as case management.
- Develop the pace and content of treatment for the older person.

Finally, other than the aversion therapy study (a procedure not typically recommended for older adults), programs emphasizing cognitive-behavioral and self-management interventions with older alcohol abusers are currently the only programs supported by published outcome data.

REFERENCES

Adams, W.L., and Cox, N.S. Epidemiology of problem drinking among elderly people. In: Gurnack, A.M., ed. *Older Adults' Misuse of Alcohol, Medicine, and Other Drugs.* New York: Springer Publishing, 1997. pp. 1–23.

Annis, H.M., and Davis, C.S. Self-efficacy and the prevention of alcoholic relapse: Initital findings from a treatment trial. In: Baker, T.B., and Cannon, D., eds. *Addictive Disorders: Psychological Research on Assessment and Treatment.* New York: Praeger Publishers, 1988. pp. 88–112.

Atkinson, R.M. Treatment programs for aging alcoholics. In: Beresford, T.P., and Gomberg, E.S.L., eds. *Alcohol and Aging.* NY: Oxford University Press, 1995. pp. 186–210.

Blake, R. Mental health counseling and older problem drinkers. *J Ment Health Couns* 12:354–367, 1990.

Blow, F.C.; Brower, K.J.; Schulenberg, J.E.; Demo-Dananberg, L.M.; Young, J.P.; and Beresford, T.P. The Michigan Alcoholism Screening Test—Geriatric Version (MAST-G): A new elderly-specific screening instrument. *Alcohol Clin Exp Res* 16:372, 1992*a*.

Blow, F.C.; Cook, C.A.L.; Booth, B.M.; Falcon, S.P.; and Friedman, M.J. Age-related psychiatric comorbidities and level of functioning in alcoholic veterans seeking outpatient treatment. *Hosp Community Psychiatry* 43:990–995, 1992*b*.

Brown, S.A. Expectancies versus background in the prediction of college drinking patterns. *J Consult Clin Psychol* 53:123–130, 1985.

Brown, S.A.; Goldman, M.S.; Inn, A.; and Anderson, L.R. Expectations of reinforcement from alcohol: Their domain and relation to drinking patterns. *J Consult Clin Psychol* 48:419–426, 1980.

Carstensen, L.L.; Rychtarik, R.G.; and Prue, D.M. Behavioral treatment of the

geriatric alcohol abuser: A long term follow-up study. *Addict Behav* 10:307–311, 1985.

Christiansen, B.A.; Goldman, M.S.; and Inn, A. Development of alcohol-related expectancies in adolescents: Separating pharmacological from social-learning influences. *J Consult Clin Psychol* 50:336–344, 1982.

Christiansen, B.A.; Smith, G.T.; Roehling, P.V.; and Goldman, M.S. Using alcohol expectancies to predict adolescent drinking behavior after one year. *J Consult Clin Psychol* 57:93–99, 1989.

Cronkite, R.C., and Moos, R.H. Determinants of the posttreatment functioning of alcoholic patients: A conceptual framework. *J Consult Clin Psychol* 48:305–316, 1980.

DeHart, S.S., and Hoffman, N.G. Screening and diagnosis: Alcohol use disorders in older adults. In: Gurnack, A.M., ed. *Older Adults' Misuse of Alcohol, Medicine, and Other Drugs.* New York: Springer Publishing, 1997. pp. 25–53.

Dunlop, J. Peer groups support seniors fighting alcohol and drugs. *Aging* 361:28–32, 1990.

Dupree, L.W. Comparison of three case-finding strategies relative to elderly alcohol abusers. *J Appl Gerontol* 8:502–511, 1989.

Dupree, L.W. Alcohol expectancies of older adults: A newer approach to an old problem? Paper presented at the Thirteenth Annual Meeting of the Southern Gerontological Society, Nashville, TN, 1992.

Dupree, L.W., and Patterson, R. Older adults. In: Hersen, M., and Turner, S.M., eds. *Diagnostic Interviewing.* New York: Plenum Press, 1985. pp. 337–359.

Dupree, L.W.; and Schonfeld, L. Substance abuse. In: Hersen, M., and Van Hasselt, V., eds. *Psychological Treatment of Older Adults: An Introductory Text.* New York: Plenum Press, 1996*a*. pp. 281–298.

Dupree, L., and Schonfeld, L. *Substance Abuse Treatment for Older Adults: A Cognitive-Behavioral and Self-Management Approach.* Tampa, FL: Department of Aging and Mental Health, Florida Mental Health Institute, University of South Florida, 1996*b*. [A prepublication manual designed to aid in the development of a comprehensive substance abuse treatment program.]

Dupree, L.W.; Broskowski, H.; and Schonfeld, L. The Gerontology Alcohol Project: A behavioral treatment program for elderly alcohol abusers. *Gerontologist* 24:510–516, 1984.

Edelstein, B.; Staats, N.; Kalish, K.; and Northrop, L. Assessment of older adults. In: Hersen, M., and Van Hasselt, V., eds. *Psychological Treatment of Older Adults: An Introductory Text.* New York: Plenum Press, 1996. pp. 35–68.

Farber, P.D.; Khavari, K.A.; and Douglass, F.M. A factor analytic study of reasons for drinking: Empirical validation of positive and negative reinforcement dimensions. *J Consult Clin Psychol* 48:780–781, 1980.

Finney, J.W.; Moos, R.H.; and Mewborn, C.R. Posttreatment experiences and treatment outcome of alcoholic patients six months and two years after hospitalization. *J Consult Clin Psychol* 48:17–29, 1980.

Finney, J.W.; Moos, R.H.; and Brennan, P.L. The Drinking Problems Index: A measure to assess alcohol-related problems among older adults. *J Subst Abuse* 3:395–404, 1991.

Flemming, A.S.; Buchanan, J.G.; Santos, J.F.; and Rickards, L.D. *Mental Health Services for the Elderly: Report on a Survey of Community Mental Health Centers.*

Vol. I. Washington, DC: The Action Committee To Implement the Mental Health Recommendations of the 1981 White House Conference on Aging, 1984.

Flemming, A.S., Buchanan, J.G., Santos, J.F., and West, P.R. *Mental Health Services for the Elderly: Report on a Survey of Community Mental Health Centers.* Vol. III. Washington, DC: The Action Committee To Implement the Mental Health Recommendations of the 1981 White House Conference on Aging, 1986.

Goldman, M.S.; Brown, S.A.; and Christiansen, B.A. Expectancy theory: Thinking about drinking. In: Blane, H.T., and Leonard, L.E., eds. *Psychological Theories of Drinking and Alcoholism.* New York: Guilford Press, 1987. pp. 181–226.

Graham, K. Identifying and measuring alcohol abuse among the elderly: Serious problems with existing instruments. *J Stud Alcohol* 47:322–326, 1986.

Hinrichsen, J. Toward improving treatment services for alcoholics of advanced age. *Alcohol Health Res World* 8:31–49, 1984.

Holder, H.; Longabaugh, R.; Miller, W.R.; and Rubonis, A.V. The cost effectiveness of treatment for alcoholism: A first approximation. *J Stud Alcohol* 52:517–540, 1991.

Hurt, R.D.; Finlayson, R.E.; Morse, R.M.; and Davis, L.J. Alcoholism in elderly persons: Medical aspects and prognosis of 216 inpatients. *Mayo Clin Proc* 63:753–760, 1988.

Janik, S.W., and Dunham, R.G. A nationwide examination of the need for specific alcoholism treatment programs for the elderly. *J Stud Alcohol* 44:307–317, 1983.

Kashner, T.M.; Rodell, D.E.; Ogden, S.R.; Guggenheim, F.G.; and Karson, C.N. Outcomes and costs of two VA inpatient programs for older alcoholics. *Hosp Community Psychiatry* 43:985–989, 1992.

Kelly, S., and Remley, T.P. Understanding and counseling elderly alcohol abusers. *Am Ment Health Couns Assoc J* 9:105–113, 1989.

Kofoed, L.; Tolson, R.; Atkinson, R.; Toth, R.; and Turner, J. Elderly groups in alcoholism clinic. In: Atkinson, R.M., ed. *Alcohol and Drug Abuse in Old Age.* Washington, DC: American Psychiatric Press Monograph Series, 1984. pp. 35–48.

Kofoed, L.; Tolson, R.; Atkinson, R.M.; Toth, R.; and Turner, J. Treatment compliance of older alcoholics: An elder-specific approach is superior to "mainstreaming." *J Stud Alcohol* 48:47–51, 1987.

Lebowitz, B.D. Correlates of success in community mental health programs for the elderly. *Hosp Community Psychiatry* 39:721–722, 1988.

Lebowitz, B.D.; Light, E.; and Bailey, F. Mental health center services for the elderly: The impact of coordination with area agencies on aging. *Gerontologist* 27:699–702, 1987.

Light, E.; Lebowitz, B.D.; and Bailey, F. CMHCs and elderly services: An analysis of direct and indirect services and service delivery sites. *Community Ment Health J* 22:294–302, 1986.

Litman, G.K.; Eiser, J.; Rawson, N.; and Oppenheim, A. Differences in relapse precipitants and coping behavior between alcoholic relapsers and survivors. *Behav Res Ther* 17:89–94, 1979.

Marlatt, G.A., and Gordon, J.R. *Relapse Prevention: Maintenance Strategies in the Treatment of Addictive Behaviors.* New York: Guilford Press, 1985.

McCrae, R. R. Age differences in the use of coping mechanisms. *J Gerontol* 37:454–460, 1982.

McCrae, R. R. Age differences and changes in the use of coping mechanisms. *J Gerontol Psychol Sci* 44:161–169, 1989.

Miller, P.M. *Behavioral Treatment of Alcoholism.* New York: Pergamon Press, 1977.

Miller, W.R., and Rollnick, S. *Motivational Interviewing: Preparing People To Change Addictive Behavior.* New York: Guilford Press, 1991.

Moos, R.H.; Brennan, P.L.; Fondacaro, M.R.; and Moos, B.S. Approach and avoidance coping responses among older problem and nonproblem drinkers. *Psychol Aging* 5:31–40, 1990.

Prochaska, J.O.; DiClemente, C.C.; and Norcross, J.C. In search of how people change: Applications to addictive behaviors. *Am Psychol* 47:1102–1114, 1992.

Roehling, P.V.; Smith, G.T.; Goldman, M.S.; and Christiansen, B.A. Alcohol expectancies predict adolescent drinking: A three year longitudinal study. Paper presented at the 95th Annual Convention of the American Psychological Association, New York, 1987.

Rosenberg, H. Relapsed versus non-relapsed alcohol abusers: Coping skills, life events, and social support. *Addict Behav* 8:183–186, 1983.

Schiff, S.M. Treatment approaches for older alcoholics. *Generations* 12:41–45, 1988.

Schonfeld, L., and Dupree, L.W. Antecedents of drinking for early- and late-onset elderly alcohol abusers. *J Stud Alcohol* 52:587–591, 1991.

Schonfeld, L., and Dupree, L.W. Treatment approaches for older adults. *Int J Addict* 30:1819–1842, 1995.

Schonfeld, L., and Dupree, L.W. The Substance Abuse Profile for the Elderly (SAPE). In: Dupree, L.W., and Schonfeld, L. *Substance Abuse Treatment for Older Adults: A Cognitive-Behavioral and Self-Management Approach.* Tampa, FL: Department of Aging and Mental Health, Florida Mental Health Institute, University of South Florida, 1996.

Schonfeld, L., and Dupree, L.W. Treatment alternatives and outcomes for the older alcohol abuser. In: Gurnack, A.M., ed., *Older Adults' Misuse of Alcohol, Medicine, and Other Drugs.* New York: Springer Publishing Company, 1997. pp. 112–131.

Smith, G.T.; Roehling, P.V.; Christiansen, B.A.; and Goldman, M.S. Alcohol expectancies predict early adolescent drinking: A longitudinal study. Paper presented at the American Psychological Association, Washington, DC, 1986.

Solomon, K.; Manepalli, J.; Ireland, G.A.; and Mahon, G.M. Alcoholism and prescription drug abuse in the elderly: St. Louis University grand rounds. *J Am Geriatr Soc* 41:57–69, 1993.

Speer, D.C., and Bates, K. Comorbid mental and substance disorders among older psychiatric patients. *J Am Geriatr Soc* 40:886–890, 1992.

Speer, D.C.; O'Sullivan, M.J.; and Schonfeld, L. Dual diagnosis among older adults: A new array of policy and planning problems. *J Ment Health Admin* 18:43–50, 1991.

Wiens, A.N.; Menustik, C.E.; Miller, S.I.; and Schmitz, R.E. Medical-behavioral treatment of the older alcoholic patient. *Am J Drug Alcohol Abuse* 9:461–475, 1982–83.

Zimberg, S. Alcohol and the elderly. In: Petersen, D.M.; Whittington, F.J.; and Payne, B.P., eds. *Drugs and the Elderly:*

Social and Pharmacological Issues. Springfield, IL: Charles C. Thomas, 1979.

Zimberg, S. Diagnosis and management of the elderly alcoholic. In: Atkinson, R.M., ed. *Alcohol and Drug Abuse in Old Age.* Washington, DC.: American Psychiatric Press, 1984. pp. 24–33.

Zimberg, S. Treating the older alcoholic. *Geriatr Med* 4:68–77, 1985.

Chapter 20

Alcohol Withdrawal and Aging

Kirk J. Brower, M.D.

Few studies have specifically investigated the effects of aging on alcohol withdrawal. Nevertheless, the studies to date are remarkably consistent in their main finding: severity of withdrawal increases with age. A review of the existing literature can inform our strategies for treatment and research. The purposes of this chapter are (1) to review the animal and human studies that focus on alcohol withdrawal and aging; (2) to consider the possible mechanisms that may explain age-related differences in alcohol withdrawal; (3) to discuss the treatment implications of these differences; and (4) to suggest directions for future research in this area.

STUDIES OF ALCOHOL WITHDRAWAL AND AGING

Animal Studies

Two studies of rodents, one in mice (Wood et al. 1982) and one in rats (Maier and Pohorecky 1989), are consistent in reporting that the severity of alcohol withdrawal is greater in older than in younger animals. Wood and colleagues compared alcohol withdrawal severity in three age groups of mice (3, 14, and 25 months) and found significantly increased body tremors in the oldest group compared with the youngest group. A similar trend was observed for hypoactivity. (None of the mice suffered convulsions.) The findings were not attributable to age differences in amount of alcohol consumed, blood alcohol levels, or weight loss. Similarly, Maier and Pohorecky demonstrated that older mice (6.5 months old) exhibited greater withdrawal severity than younger mice (3.5 months old) after receiving equivalent amounts and durations of alcohol. (Again, no convulsions were reported.)

Although Maier and Pohorecky also tried to examine the effect of re-

K.J. Brower, M.D., is associate professor of psychiatry at the University of Michigan Medical School and Alcohol Research Center, 400 East Eisenhower Parkway, Suite 2A, Ann Arbor, MI 48108.

peated withdrawals on subsequent withdrawal severity in mice, their study was confounded by age factors. Nevertheless, animal studies offer the advantage of controlling variables such as quantity and duration of alcohol consumption; and the interaction of aging and repeated withdrawals on subsequent withdrawal severity could be easily studied in animals using a design suggested by Maier and Pohorecky (1989).

Human Studies

Five studies to date have specifically examined alcohol withdrawal in elderly persons, although "elderly" was variably defined (table 1). Four of the studies compared an older with a younger group of inpatient alcoholics (Iber 1986; Liskow et al. 1989; Brower et al. 1994; Mudd et al. 1994), and one study compared two outpatient groups of elderly alcoholics, differentiated by age of onset of alcohol problems (Schonfeld and Dupree 1991).

Iber (1986) compared durations of hospitalization for detoxification among male veterans and found that older alcoholics (age 51 and over) needed to stay for twice as many days

Table 1. Human Studies of Alcohol Withdrawal and Aging.

Reference	Sample Characteristics	Treatment Setting
Iber 1986	Older group: ages 51+, all men Younger group: ages 40–50, all men (Sample sizes not reported)	Inpatient detoxification unit in veterans hospital
Liskow et al. 1989	Older group: n = 26, ages 58–77, all men Younger group: n = 24, ages 21–33, all men	Inpatient alcohol treatment unit in veterans hospital
Schonfeld and Dupree 1991	Early-onset group: n = 23, ages 60+, 35% women, onset < 50 yr Late-onset group: n = 23, ages 60+, 35% women, onset ≥ 50 yr	Outpatient day treatment program specialized for elderly "alcohol abusers"
Brower et al. 1994	Older group: n = 48, ages 60+, 38% women Younger group: n = 36, ages 21–35, 25% women	Inpatient and residential alcohol treatment units in community hospital for older and younger groups, respectively
Mudd et al. 1994	Older group: n = 61, ages 60+, 43% women Younger group: n = 57, ages 21–35, 19% women	Inpatient and residential alcohol treatment units in community hospital for older and younger groups, respectively

(11.9 vs. 5.6 days) as younger alcoholics (ages 40–50), most often because of mental impairment or general health problems during the withdrawal period. These findings are consistent with those of a study conducted by our group (Brower et al. 1994); we found that withdrawal symptoms lasted longer (9.0 vs. 6.5 days) and cognitive impairment was more frequent in an elderly group (age 60 and over) than in a younger group (ages 21–35).

Liskow and colleagues (1989) reported that elderly male alcoholics (ages 58–77) had more severe withdrawal symptoms, requiring higher doses of chlordiazepoxide during inpatient detoxification, despite consuming less alcohol prior to treatment than younger alcoholics (ages 21–33). Two possible confounding factors were that the elderly alcoholics as a group had a significantly longer duration of problem drinking (26 vs. 17 years) and more than twice the rate of delirium tremens (DTs) in the past (30.8 percent vs. 12.5 percent) than the younger alcoholics. The authors did report that age remained a significant contributor to withdrawal severity even after covarying for duration of

Results
Longer hospital duration in older vs. younger alcoholics
Greater withdrawal severity and dosage of medication in older vs. younger alcoholics
Higher frequencies of shakes, diaphoresis, and DTs by history in early-onset vs. late-onset elderly alcoholics
Greater frequency and duration of withdrawal symptoms, and different symptom profile in older vs. younger alcoholics
More nursing interventions required for older vs. younger alcoholics

problem drinking. However, the increased rate of DTs in the elderly group's history may have prompted physicians to prescribe increased chlordiazepoxide doses during the study episode; the authors did not statistically adjust for a history of DTs.

In the study that my colleagues and I conducted (Brower et al. 1994), elderly alcoholics undergoing inpatient detoxification were found to have more withdrawal symptoms that lasted longer than a comparably treated group of younger alcoholics (figure 1). Elderly alcoholics were also more susceptible than younger alcoholics to particular types of withdrawal symptoms, such as cognitive impairment (50 percent vs. 8 percent), "weakness" (48 percent vs. 8 percent), and maximum systolic blood pressure (168 mmHg vs. 145 mmHg), whereas younger alcoholics suffered more headaches than older alcoholics (33 percent vs. 4 percent).

The definition of "weakness" in our study included both subjective complaints of weakness and nursing observations that a patient required assistance with ambulation or feeding because of weakness. Although cognitive impairment was associated with an increased rate of cardiac disease in the elderly, it was reversible within the time course of acute withdrawal, suggesting it was not exclusively due to an increased rate of medical comorbidity. Likewise, cognitive impairment and weakness did not correlate with chlordiazepoxide dosage, suggesting they were not side effects of sedative medication. Elderly patients received doses and durations of chlordiazepoxide equivalent to those received by younger patients, in contrast to patients studied by Liskow and colleagues (1989). Nevertheless, it is our contention that equivalent doses in the elderly actually signified greater medication needs than in the younger group, because the elderly respond

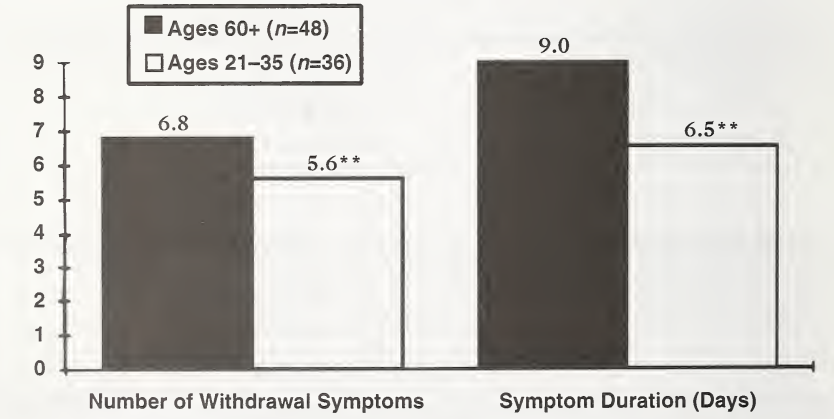

Figure 1. Withdrawal symptoms in older alcoholics compared with younger alcoholics (** $p < 0.02$). Data from Brower et al. 1994.

more strongly to benzodiazepines than younger persons due to age-related pharmacokinetic and pharmacodynamic differences (Closser 1991). We did not control for duration of problem drinking, which was significantly higher in the elderly than in the younger alcoholics, and we were unable to quantify the frequency of previous withdrawal episodes. We were also unable to completely rule out medical causes for the age-related differences in withdrawal phenomena.

Mudd and colleagues (1994) described an associated study using the same methodology, with an overlapping patient sample, as that described by Brower and colleagues (1994). They found that older adults required more nursing care during alcohol withdrawal than younger adults, which correlated with the higher number of withdrawal symptoms in the elderly group. Specifically, more than twice as many nursing interventions (24.5 vs. 11.3) were delivered over a longer duration (11.6 vs. 9.4 days) in the elderly compared with the younger group of inpatient alcoholics. The authors did not control for the increased prevalence of medical comorbidity (cardiac and liver disease) in the elderly group, which may have contributed to the increased need for nursing care.

Schonfeld and Dupree (1991) reported that early-onset elderly alcoholics (defined as those who began abusing alcohol before age 50) had higher frequencies of withdrawal symptoms such as shakes (70 percent vs. 20 percent), diaphoresis (68 percent vs. 26 percent), and DTs (26 percent vs. 0 percent) than late-onset alcoholics (those who began abusing alcohol at age 50 or older). However, early-onset alcoholics also drank to intoxication on more days in the past month than did late-onset alcoholics (16 vs. 8 days; $p < 0.03$). Therefore, both duration and frequency of alcoholic drinking likely contribute to withdrawal severity in the elderly, and these factors require consideration in future studies.

Severe Withdrawal Syndromes

Clinical researchers generally distinguish mild to moderate alcohol withdrawal from severe alcohol withdrawal syndromes (Hayashida et al. 1989); the latter are characterized by seizures or DTs. Although the studies comparing older and younger alcoholics summarized in table 1 were consistent in finding greater severity among the elderly within the range of mild to moderate withdrawal syndromes, none of these studies found higher rates of seizures or DTs in elderly alcoholics. It is possible that this finding is a function of treatment setting restrictions among these studies. Alcoholics with severe withdrawal syndromes may more likely be admitted to acute medical and intensive care units than to alcohol-specific treatment units where the studies in table 1 were conducted. The following paragraphs describe studies that have examined the association of age with severe withdrawal syndromes.

DTs. A 1994 study of predictors by Wetterling and colleagues reported similar ages for patients with and

without DTs, whereas Kramp and Hemmingsen (1981) concluded that DTs were more likely in *younger* than older alcoholics. A recent study by Schuckit and colleagues (1995) found that alcoholics with a history of seizures or DTs (n = 211; mean age 40.2 years) were significantly older than alcoholics without severe withdrawal syndromes (n = 1,437; mean age 37.5 years), but age was not significant after controlling for maximum quantity of alcohol consumed, number of prior withdrawal episodes, and medical comorbidity. Although increased age does not appear to be a risk factor for developing DTs after controlling for other factors, age may increase the risk of death when DTs do occur. Feuerlein and Reiser (1986) studied 778 cases of DTs and reported higher mortality rates in alcoholics age 55 and older (27 percent) than in alcoholics under age 55 (7 percent).

Withdrawal Seizures. Evidence is lacking that elderly alcoholics are more prone to withdrawal seizures than younger alcoholics. In the two studies comparing withdrawal severity in younger and older alcoholics (Liskow et al. 1989; Brower et al. 1994), no seizures were reported, which may have reflected the low incidence of withdrawal seizures (1–3 percent) across several studies (Booth and Blow 1993; Morton et al. 1994; Mayo-Smith and Bernard 1995; Schuckit et al. 1995). However, a study of 6,818 alcoholic men undergoing alcohol detoxification also failed to find age-related differences in the occurrence of seizures (Booth and Blow 1993). Similarly, two other studies of treated alcoholics found no significant differences in seizure history as a function of age (Schuckit et al. 1978; Lechtenberg and Worner 1991). By contrast, a recent prospective study (Mayo-Smith and Bernard 1995) on the prevalence of seizures during detoxification of 1,044 patients found a nonsignificant trend for patients with seizures to be older than those without seizures (50.5 vs. 43.6 years). Likewise, a retrospective study (Morton et al. 1994) of 1,695 alcoholic patients found a nonsignificant trend for the seizure group (n = 12) to be older than a comparison group of 28 randomly selected controls (46.8 vs. 38.1 years). Although the prospective and the retrospective studies both reported an age-related, albeit nonsignificant, increase in alcoholic withdrawal seizures, the mean ages of the patient groups with seizures were 15 to 20 years younger than age 65.

On the basis of a single case report involving an 86-year old female alcohol and benzodiazepine "addict," van Sweden and Hoste (1987) suggested that elderly persons might be particularly susceptible to partial complex seizures during substance withdrawal. Whether elderly alcoholics are any more likely than younger alcoholics to suffer partial complex seizures during withdrawal is unknown. Nevertheless, the case serves as a reminder that alcohol withdrawal may be complicated by other drug dependencies in elderly patients.

Methodological Limitations

The studies comparing older and younger alcoholics in table 1 suffer

from sampling bias inherent in using only specialized or single treatment settings. A single treatment unit is likely to minimize age differences due to age-independent, clinical screening criteria for admission. For example, alcoholics with seizures or DTs may be excluded from a particular treatment unit. Furthermore, older alcoholics may be more likely than younger alcoholics to be detoxified in general medical units or intensive care units; whereas younger alcoholics may be more likely to be detoxified in specialized, but less acute, addiction treatment units.

Many of the studies failing to find age-related differences in DTs or seizures did not describe the number of elderly subjects (age 65 and over) in their samples (Morton et al. 1994; Wetterling et al. 1994; Mayo-Smith and Bernard 1995; Schuckit et al. 1995). These studies may not have included enough elderly alcoholics to test the hypothesis that elderly alcoholics have a greater risk of severe withdrawal syndromes than younger alcoholics do.

Another limitation across studies involves the failure to control or adjust for factors that can influence the severity of alcohol withdrawal regardless of age. These factors include quantity, frequency, and duration of alcohol use; severity and typology of dependence; total withdrawal episodes; other substance use; and medical and psychiatric comorbidity. Although each study controlled for one or more of these potential confounds, no study controlled for all of them. Moreover, none of the studies controlled for psychiatric comorbidity, which may increase the severity of withdrawal, at least in younger patients (Johnston et al. 1991).

Some studies utilized retrospective chart reviews or designs or did not use structured, validated instruments for measuring alcohol withdrawal symptoms (Brower et al. 1994; Mudd et al. 1994). However, an optimal rating scale for measuring alcohol withdrawal in the elderly has yet to be established. Although Liskow and colleagues (1989) developed a withdrawal scale that they used in elderly alcoholics, they did not present age-specific validity data. In fact, they reported that withdrawal scores as determined by their scale correlated with medication dosage more significantly in their younger than in their older group of alcoholics.

Finally, all of the studies employed cross-sectional comparisons of older and younger groups. No longitudinal studies of how alcohol withdrawal may change with age have been conducted.

Mechanisms of Increased Withdrawal Severity in Elderly Alcoholics

The reasons for greater withdrawal severity in elderly than in younger alcoholics may include kindling, in which successive withdrawal episodes intensify regardless of age (Brown et al. 1988; Booth and Blow 1993); longer duration of alcoholism; comorbid medical illnesses; and increased physiological sensitivity with aging. By controlling for the first three factors, the animal studies are highly suggestive of increased physiological sensitivity

with aging (Maier and Pohorecky 1989; Wood et al. 1982). Human studies suggest that all of these factors may be operative.

TREATMENT IMPLICATIONS

Intensity of Care and Treatment Setting

Taken together, inpatient studies indicate that compared with younger alcoholics, elderly alcoholics have more severe withdrawal symptoms (Liskow et al. 1989), which last longer (Brower et al. 1994) and result in more nursing interventions (Mudd et al. 1994) and extended hospital stays (Iber 1986). Accordingly, the intensity of care should match the increased withdrawal severity generally observed in the elderly. The American Medical Association (1995, 1996), in guidelines endorsed by the American Society of Addiction Medicine, recommended that detoxification of elderly patients is best done in the hospital, while also recognizing that each case must be treated according to its individual characteristics. Treatment matching according to individual case parameters is essential.

We have successfully detoxified elderly patients with mild to moderate withdrawal in an outpatient setting as part of an intensive day treatment program. Other treatment centers also have successfully applied outpatient detoxification protocols to elderly patients (David Oslin, personal communication, 1996). Anecdotal observations suggest the following to be key ingredients of successful outpatient detoxification in elderly alcoholics: mild to moderate withdrawal symptoms, an absence or low level of medical and psychiatric comorbidity, reliable transportation to and from the clinic with another person, good social support to monitor treatment course at home, the use of structured rating scales to monitor symptoms, and daily clinic visits from 9:00 a.m. to 4:00 p.m. during the workweek as part of an intensive day treatment program. The proportion

Table 2. Benzodiazepine Detoxification of Elderly Alcoholics.

Benzodiazepine Medication	Administration Forms[1]	Elimination and Metabolism
Shorter acting agents		
Lorazepam	PO, IM, IV	Unchanged elimination in elderly persons.
Oxazepam	PO	Minor liver metabolism.
Longer acting agents		
Chlordiazepoxide	PO, IV	Delayed elimination in elderly persons.
Diazepam	PO, IV	Major liver metabolism.

[1] IM = intramuscular; IV = intravenous; PO = oral

of elderly alcoholics who can safely be detoxified in outpatient settings is unknown. It must be emphasized that there are no published studies of outpatient detoxification in the elderly. Therefore, outpatient detoxification of elderly alcoholics cannot be routinely recommended at this time, and the viability of inpatient detoxification units should be protected, especially in an era of increasingly managed care.

Pharmacotherapy

Benzodiazepines are generally considered the treatment of choice for alcohol withdrawal, and the elderly are no exception (American Medical Association 1995). However, there are no comparative pharmacotherapy trials in older alcoholics, so the choice of benzodiazepines remains a matter of each physician's or treatment unit's preference. The major benzodiazepines used to treat alcohol withdrawal in this country are the longer acting agents chlordiazepoxide and diazepam and the shorter acting agents oxazepam and lorazepam (table 2).

Some authors argue that lorazepam and oxazepam are the benzodiazepines of choice for elderly patients (> 60 years old) because, in contrast to chlordiazepoxide and diazepam, they do not undergo extensive liver metabolism and their rates of elimination are essentially unchanged in the elderly (Rosenbloom 1986; Lawlor and Sunderland 1991; Hoey et al. 1994). While it may seem that shorter acting benzodiazepines would lead to less accumulation and thus less cognitive impairment than longer acting benzodiazepines, Ritson and Chick (1986) reported that diazepam was associated with better cognitive functioning than lorazepam in a study of detoxifying alcoholics over a wide age range (20–70 years old), although age-specific analyses were not presented. Whether this finding would hold true for an exclusively elderly population of alcoholics is unknown.

Other authors prefer longer acting benzodiazepines, which allow for steadier and more gradual declines in physiological levels during the taper

Advantages	Disadvantages
Elderly metabolize and eliminate these agents well, so less chance of toxicity due to accumulation.	Abrupt declines in medication levels between doses may possibly result in breakthrough symptoms and seizures. Possibly more cognitive impairment.
Gradual decline in medication levels during taper. Possibly greater seizure prophylaxis and less cognitive impairment.	Delayed elimination may lead to accumulation and sedative toxicity, especially if liver function is impaired.

process, possibly resulting in a lower rate of withdrawal seizures, than observed with shorter acting agents (Mayo-Smith and Bernard 1995). When using longer acting benzodiazepines such as chlordiazepoxide or diazepam, dosage may need to be adjusted downward in elderly relative to younger patients because of slower metabolism and elimination as well as increased cerebral sensitivity (Closser 1991; American Medical Association 1995). However, two studies reported that elderly patients required equivalent or higher doses of chlordiazepoxide compared with younger patients (Liskow et al. 1989; Brower et al. 1994). Therefore, benzodiazepine dosage is not necessarily lower in elderly than younger alcoholics, and the dosage should be titrated carefully to each patient's withdrawal symptoms while monitoring closely for sedative toxicity.

Routes of Administration. Rosenbloom (1986) reported the death of a 65-year-old patient in association with intramuscular chlordiazepoxide for treating alcohol withdrawal. Intramuscular use of chlordiazepoxide and diazepam is always contraindicated due to erratic absorption and the potential for toxicity. Lorazepam is well absorbed when used intramuscularly, and is the preferred agent when intramuscular use is desired (see table 2). Lorazepam, chlordiazepoxide, and diazepam are all available for intravenous use, which is preferred for treating seizures or DTs. Oxazepam is only available in oral form.

Other Agents. Chlorazepate is a benzodiazepine that must be activated in the acidic environment of the stomach before it can exert any systemic therapeutic effect. Because stomach acidity decreases with age (Lawlor and Sunderland 1991), chlorazepate may be unsuitable to treat alcohol withdrawal in the elderly. Case reports of using agents such as phenobarbital (Ives et al. 1991) and clonidine (Ip Yam et al. 1992) to treat elderly cases of alcohol withdrawal have been described with good efficacy and without complications. Nevertheless, there are no controlled studies comparing different agents for detoxifying elderly alcoholics, so the generalizability of these case reports is unknown. Moreover, clonidine cannot be generally recommended because it provides no protection against withdrawal seizures.

DIRECTIONS FOR FUTURE STUDY

The literature reviewed in this chapter points to several areas for further study. Both aging and repeated withdrawal episodes can increase the severity of subsequent alcohol withdrawal episodes. Animal studies could determine the interactive effects of aging and repeated withdrawals on subsequent withdrawal severity using an experimental design outlined by Maier and Pohorecky (1989).

Alcohol withdrawal rating scales that have been developed and validated with younger alcoholics may not optimally apply to older adults. Liskow and colleagues (1989) developed an 18-item rating scale which they used with elderly and younger subjects, but they did not present age-specific validity data. In addition,

a briefer instrument would be desirable, such as the 10-item Revised Clinical Institute Withdrawal Assessment for Alcohol (CIWA-Ar) scale (Sullivan et al. 1989). Validation studies of the CIWA-Ar or similar scales in elderly alcoholics are needed.

The symptom profile of alcohol withdrawal in elderly and younger alcoholics may differ (Brower et al. 1994). Prospective studies could compare further the withdrawal symptom profile in elderly and younger alcoholics using validated, structured rating scales. If the symptom profile of alcohol withdrawal consistently differs in older and younger alcoholics, then treatment would need to be tailored accordingly. Other studies could examine if predictors of withdrawal severity (such as age of onset, consumption variables, comorbidity, and repeated withdrawals) differ in elderly and younger alcoholics. Although cross-sectional studies are easier to conduct, a longitudinal study could determine (a) whether withdrawal symptoms increase in severity as a group of alcoholics age, and (b) the interaction between repeated withdrawal episodes and aging.

Treatment setting is another area of needed investigation. It would be valuable to know what proportion of elderly alcoholics could be detoxified in outpatient settings. While the proportion is likely to be less in elderly alcoholics than in younger alcoholics, this is entirely unstudied in elderly alcoholics. A randomized trial of outpatient versus inpatient detoxification for elderly alcoholics could be modeled after the study by Hayashida and colleagues (1989). In addition, naturalistic studies that target multiple hospital units where elderly alcoholics are likely to be detoxified would help to determine if elderly alcoholics are overrepresented in acute settings where seizures and DTs are most likely to be treated.

Pharmacotherapy trials are needed to compare the longer acting (chlordiazepoxide or diazepam) and shorter acting (lorazepam or oxazepam) benzodiazepines for treating alcohol withdrawal in the elderly. Outcomes of pharmacotherapy trials should include study completion rates, response and duration of global and individual symptom severity, and side effects such as cognitive impairment and oversedation.

CONCLUSIONS

The few studies of alcohol withdrawal in older populations, when compared with younger age groups, have consistently found symptoms of greater intensity and duration. Both animal and human studies provide evidence that duration, frequency, and intensity of moderate withdrawal symptoms increase with age. Increased physiological sensitivity with aging, kindling due to repeated withdrawal episodes, medical comorbidity, and duration of alcohol use are all likely to contribute to the increased severity of moderate withdrawal symptoms in elderly alcoholics. However, evidence is generally lacking that severe withdrawal complications such as seizures or DTs are more common in elderly than in younger alcoholics. Studies are needed that target acute care settings where

elderly alcoholics with severe withdrawal episodes are most likely to be treated.

The profile of withdrawal symptoms may also differ with age: cognitive impairment, weakness, and high blood pressure may be more likely in older than in younger alcoholics. Medical comorbidity might influence the profile of symptoms seen in elderly alcoholics and should be adjusted for in future studies. Rating scales of alcohol withdrawal in the elderly need validation as well. If the findings of differential symptomatology with age are substantiated, then treatments for elderly alcoholics may need to be tailored accordingly.

The intensity of care should generally match the increased withdrawal severity seen in elderly alcoholics. Inpatient detoxification is generally recommended for elderly alcoholics, and elderly alcoholics may require longer hospital stays and more nursing care than younger alcoholics. Although some elderly alcoholics may be detoxified safely in outpatient settings, there are no published studies to date of outpatient detoxification in elderly alcoholics.

Benzodiazepines are currently the treatment of choice for medical detoxification of elderly alcoholics. The dosage of benzodiazepines required to treat alcohol withdrawal is not necessarily lower in elderly than in younger alcoholics. Finally, comparative pharmacotherapy trials are needed to determine whether shorter acting (lorazepam or oxazepam) or longer acting (chlordiazepoxide or diazepam) benzodiazepines provide more optimal treatment while minimizing side effects of cognitive impairment and drowsiness.

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REFERENCES

American Medical Association. *Alcoholism in the Elderly: Diagnosis, Treatment, and Prevention; Guidelines for Primary Care Physicians.* Chicago: the Association, 1995.

American Medical Association Council on Scientific Affairs. Alcoholism in the elderly. *JAMA* 275:797–801, 1996.

Booth, B.M., and Blow, F.C. The kindling hypothesis: Further evidence from a U.S. national study of alcoholic men. *Alcohol Alcohol* 28:593–598, 1993.

Brower, K.J.; Mudd, S.; Blow, F.C.; Young, J.P.; and Hill, E.M. Severity and treatment of alcohol withdrawal in elderly versus younger patients. *Alcohol Clin Exp Res* 18:196–201, 1994.

Brown, M.E.; Anton, R.F.; Malcolm, R.; and Ballenger, J.C. Alcohol detoxification and withdrawal seizures: Clinical support for a kindling hypothesis. *Biol Psychiatry* 23:507–514, 1988.

Closser, M.H. Benzodiazepines and the elderly. *J Subst Abuse Treat* 8:35–41, 1991.

Feuerlein, W., and Reiser, E. Parameters affecting the course and results of delirium tremens treatment. *Acta Psychiatr Scand Suppl* 73(Suppl 329):120–123, 1986.

Hayashida, M.; Alterman, A.I.; McLellan, A.T.; O'Brien, C.P.; Purtill, J.J.; Volpicelli, J.R.; Raphaelson, A.H.; and Hall, C.P. Comparative effectiveness and costs of inpatient and outpatient detoxification of patients with mild-to-moderate

alcohol withdrawal syndrome. *N Engl J Med* 320:358–365, 1989.

Hoey, L.L.; Nahum, A.; and Vance-Bryan, K. A retrospective review and assessment of benzodiazepines in the treatment of alcohol withdrawal in hospitalized patients. *Pharmacotherapy* 14:572–578, 1994.

Iber, F.L. The elderly alcoholic: Experience with Baltimore Veterans and private alcoholism patients over age 60. In: Hutchinson, M.L., and Munro, H.N., eds. *Nutrition and Aging.* Orlando, FL: Academic Press, 1986. pp. 169–178.

Ip Yam, P.C.; Forbes, A.; and Kox, W.J. Clonidine in the treatment of alcohol withdrawal in the intensive care unit. *Br J Anaesth* 68:106–108, 1992.

Ives, T.J.; Mooney, A.J.I.; and Gwyther, R.E. Pharmacokinetic dosing of phenobarbital in the treatment of alcohol withdrawal syndrome. *South Med J* 84(1):18–21, 1991.

Johnston, A.L.; Thevos, A.K.; Randall, C.L.; and Anton, R.F. Increased severity of alcohol withdrawal in inpatient alcoholics with a co-existing anxiety diagnosis. *Br J Addict* 86:719–725, 1991.

Kraemer, K.L.; Mayo-Smith, M.F.; and Caulkins, D.R. Impact of age on the severity, course, and complications of alcohol withdrawal. *Arch Intern Med* 157:2234–2241, 1997.

Kramp, P., and Hemmingsen, R. Age distribution of patients with delirium tremens in Denmark 1836–1978. *Acta Psychiatr Scand* 63:253–261, 1981.

Lawlor, B.A., and Sunderland, T. Use of benzodiazepines in the elderly. In: Roy-Byrne, P.P., and Cowley, D.S., eds. *Benzodiazepines in Clinical Practice: Risks and Benefits.* Washington, DC: American Psychiatric Press, 1991. pp. 213–227.

Lechtenberg, R., and Worner, T.M. Relative kindling effect of detoxification and non-detoxification admissions in alcoholics. *Alcohol Alcohol* 26:221–225, 1991.

Liskow, B.I.; Rinck, C.; Campbell, J.; and DeSouza, C. Alcohol withdrawal in the elderly. *J Stud Alcohol* 50:414–421, 1989.

Maier, D.M., and Pohorecky, L.A. The effect of repeated withdrawal episodes on subsequent withdrawal severity in ethanol-treated rats. *Drug Alcohol Depend* 23:103–110, 1989.

Mayo-Smith, M.F., and Bernard, D. Late-onset seizures in alcohol withdrawal. *Alcohol Clin Exp Res* 19:656–659, 1995.

Morton, A.W.; Laird, L.K.; Crane, D.F.; Partovi, N.; and Frye, L.H. A prediction model for identifying alcohol withdrawal seizures. *Am J Drug Alcohol Abuse* 20:75–86, 1994.

Mudd, S.A.; Boyd, C.J.; Brower, K.J.; Young, J.P.; and Blow, F.C. Alcohol withdrawal and related nursing care in older adults. *J Gerontol Nurs* 20(10):17–26, 1994.

Ritson, B., and Chick, J. Comparison of two benzodiazepines in the treatment of alcohol withdrawal: Effects on symptoms and cognitive recovery. *Drug Alcohol Depend* 18:329–334, 1986.

Rosenbloom, A.J. Optimizing drug treatment of alcohol withdrawal. *Am J Med* 81:901–904, 1986.

Schonfeld, L., and Dupree, L.W. Antecedents of drinking for early- and late-onset elderly alcohol abusers. *J Stud Alcohol* 52:587–592, 1991.

Schuckit, M.A.; Morrissey, E.R.; and O'Leary, M.R. Alcohol problems in elderly men and women. *Addict Dis* 3:405–416, 1978.

Schuckit, M.A.; Tipp, J.E.; Reich, T.; Hesslebrock, V.M.; and Bucholz, K.K.

The histories of withdrawal convulsions and delirium tremens in 1648 alcohol dependent subjects. *J Stud Alcohol* 90:1335–1347, 1995.

Sullivan, J.T.; Sykora, K.; Schneiderman, J.; Naranjo, C.A.; and Sellers, E.M. Assessment of alcohol withdrawal: The Revised Clinical Institute Withdrawal Assessment for Alcohol scale (CIWA-Ar). *Br J Addict* 84:1353–1357, 1989.

van Sweden, B., and Hoste, S. Are complex partial seizures an uncommon withdrawal sign in the elderly? *Eur Neurol* 27:239–244, 1987.

Wetterling, T.; Kanitz, R.D.; Veltrup, C.; and Driessen, M. Clinical predictors of alcohol withdrawal delirium. *Alcohol Clin Exp Res* 18:1100–1102, 1994.

Wood, W.G.; Armbrecht, H.J.; and Wise, R.W. Ethanol intoxication and withdrawal among three age groups of C57BL/6NNIA mice. *Pharmacol Biochem Behav* 17:1037–1041, 1982.

Chapter 21

The Spectrum of Alcohol Interventions for Older Adults

Frederic C. Blow, Ph.D.

Alcohol use disorders are important public health problems in older adults. Heavy alcohol use is associated with a number of adverse health effects in this population. These adverse effects include greater risk for harmful drug interactions, injury, depression, memory problems, liver disease, cardiovascular disease, cognitive changes, and sleep problems (Finch and Barry 1992; Liberto et al. 1992; Gambert and Katsoyannis 1995). There is emerging evidence that problem drinking in late life affects a larger proportion of the elderly population than previously thought (Williams and Debakey 1992; Adams et al. 1996).

DEFINITIONS

To understand the most effective types of alcohol interventions with older adults, definitions of levels of alcohol use and consequences are necessary. The terms presented in this chapter are derived from the clinical and research expertise of professionals in the field.

For the purposes of this chapter, *abstinence* refers to ingesting no alcohol in the previous year. A large percentage of older adults are abstinent, and it is important for clinicians to ascertain why an individual patient is abstinent. Some patients are abstinent because of a previous problem with alcohol, others because of recent illness; still others have lifelong patterns of low-risk use or abstinence. Patients who have a history of alcohol problems may require preventive monitoring to determine if any new stresses could reactivate an old pattern.

Low-risk use is alcohol use that does not lead to problems. Persons in this category can set reasonable limits on alcohol consumption and do not

F.C. Blow, Ph.D., is assistant research scientist and assistant professor at the University of Michigan Alcohol Research Center, 400 East Eisenhower Parkway, Suite A, Ann Arbor, MI 48108.

drink when driving a motor vehicle or boat or when using contraindicated medications. These persons may not need interventions but can benefit from preventive messages (e.g., *Our goal is to prevent additional health problems. Your walking program looks good and you have maintained your weight. Since you have no family history of alcohol or drug problems and are taking no medication to interfere with alcohol, not exceeding a glass of wine two to three times a week should not cause any additional problems for you at this time.* [adapted from Barry 1997]).

Use that increases the chances that a person will develop problems and complications is *at-risk use*. Persons over 65 who drink more than seven drinks per week, or one drink per day, are in the at-risk use category. Although these people do not currently have a health, social, or emotional problem caused by alcohol, they may experience family and social problems, and if this drinking pattern continues over time, health problems could be exacerbated. These older adults can benefit from brief interventions.

Problem use or abuse of alcohol means that a person is drinking at a level that has already resulted in adverse medical, psychological, or social consequences. Potential consequences can include accidents and injuries, medication-interaction problems, and family problems, among others (Barry and Fleming 1994). It is important to note that some older adults who drink even small amounts of alcohol may experience alcohol-related problems. Quantity and frequency of alcohol use may not be the first determinant of the usefulness of intervening. The presence of consequences should drive the need for intervening.

The potential interaction of medication and alcohol is of great concern with this age group. For some clients, any alcohol use at all, combined with the use of specific over-the-counter or prescription medications, can increase problematic consequences. Therefore, alcohol use recommendations for this age group are generally lower than those set for adults under 65 and are usually made on a case-by-case basis.

To determine the prescription and over-the-counter medication use of older adults, the "brown bag" approach is helpful. The practitioner can ask older adults to bring every medication they take in a brown paper bag (i.e., all medications prescribed by a doctor and all medications, vitamins, herbs, and so on that they got at the drugstore and that anyone gave them to try). This will provide an opportunity to better determine potential medication-interaction problems.

Alcohol dependence refers to a medical disorder characterized by loss of control, preoccupation with alcohol, continuing use despite adverse consequences, and suffering from physiological symptoms such as tolerance and withdrawal (American Psychiatric Association 1994).

The spectrum of alcohol interventions for older adults ranges from prevention, education, and brief advice for persons who are abstinent or low-risk drinkers to brief, structured interventions for at-risk or problem drinkers to formalized alcoholism treatment for drinkers who meet criteria for abuse and/or dependence

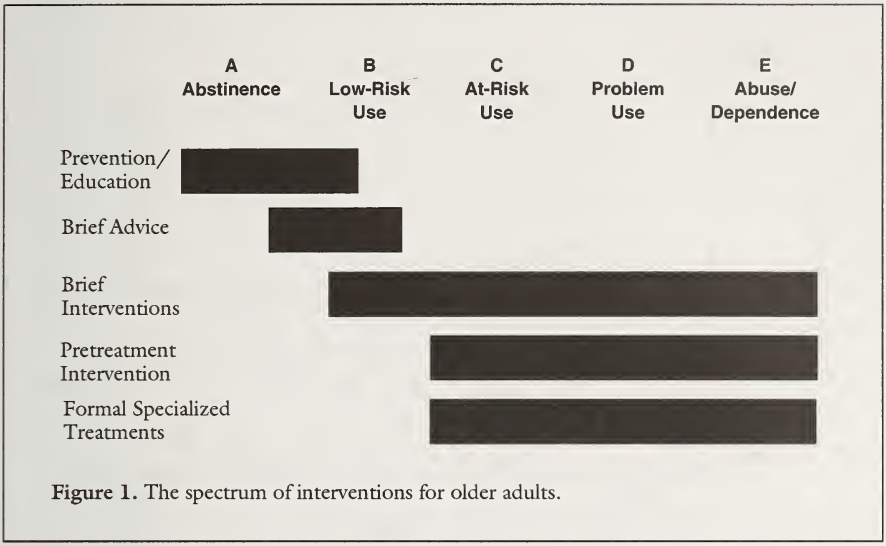

Figure 1. The spectrum of interventions for older adults.

(figure 1). Formalized treatment is generally used with persons who meet criteria for alcohol abuse or dependence and cannot discontinue drinking with a brief intervention protocol. Nonetheless, preintervention strategies are also appropriate for this population.

LIFETIME PATTERNS OF DRINKING

Clinical models of alcoholism and recovery were traditionally thought to follow a natural progression from early signs and symptoms through end-stage disease (Finch and Barry 1992). When patients "hit bottom," they either died or began the long road to recovery. Most persons with alcohol problems do not fit this model. The clinical course of alcohol use in these persons across the lifespan is often marked by periods of abstinence or low-risk use. Figures 2 and 3 illustrate the drinking patterns of persons with low-risk use and problem use, respectively. The various patterns for problem use include the early-onset problem drinker, who is a heavy user of alcohol throughout most of adulthood; the cyclical heavy drinker; and the late-onset problem drinker, who often begins drinking due to stressors of later life (e.g., retirement, death of spouse, diminished physical capacity). Although original clinical estimates of late-onset problems indicated that about one-third of older problem drinkers were in this category, systematic research (Blow et al. unpublished manuscript) is beginning to place the rate at 10 percent or less.

The high prevalence of alcohol use disorders and related problems in older primary care populations, as well as the availability of measures to quickly and easily screen older patients for alcohol use and functional health status, provides a compelling rationale for assessing alcohol use and intervening with problem users in this vulnerable population.

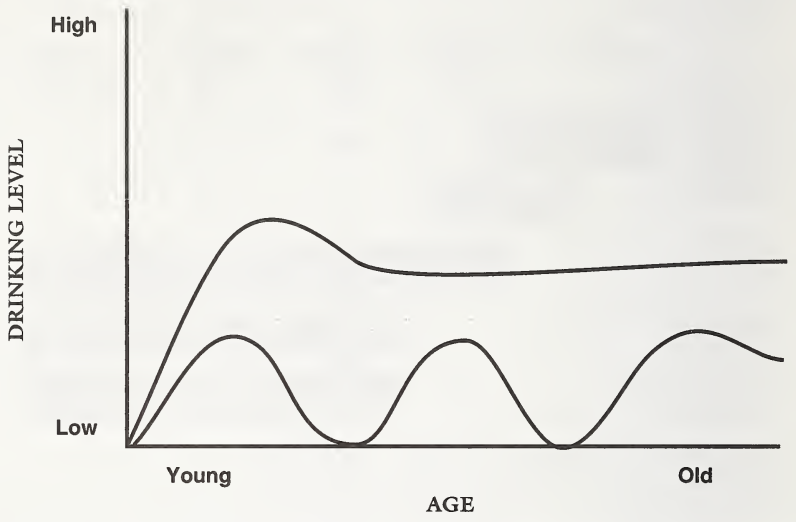

Figure 2. Low-risk use.

EXTENT OF THE PROBLEM

Prevalence estimates for older at-risk and problem drinking using community surveys have ranged from 1 to 15 percent (Schuckit and Pastor 1978; Gurland and Cross 1982; Robins and Regier 1991; Adams et al. 1996). These rates vary widely depending on the definition of alcohol abuse/dependence and the methodology used in obtaining samples. Several researchers have also questioned the accuracy of rates of alcohol problems for older adults because of the use of assessment instruments developed on younger populations.

The elderly seen in medical settings have consistently higher rates of alcohol-related problems (Adams et al. 1993; Dufour and Fuller 1995) than those in the general population. Among clinical populations, however, estimates of alcohol abuse/dependence are substantially higher, because problem drinkers of all ages are more likely to present in health care settings (Beresford 1979; Institute of Medicine 1990). Among elderly patients seeking treatment in hospitals, primary care clinics, and nursing homes for medical or psychiatric problems, rates of concurrent alcoholism have been reported to be between 15 and 58 percent (Gomberg 1980; Schuckit 1982; Beresford et al. 1990; Buchsbaum et al. 1991; Adams et al. 1996).

In a recent study (Beresford and Blow unpublished data), the lifetime prevalence of alcohol dependence among randomly selected hospitalized medical patients was 20.4 percent for those ages 60–69, declining to 13.7 percent among patients ages 70–79 and to 0.0 percent for those age 80

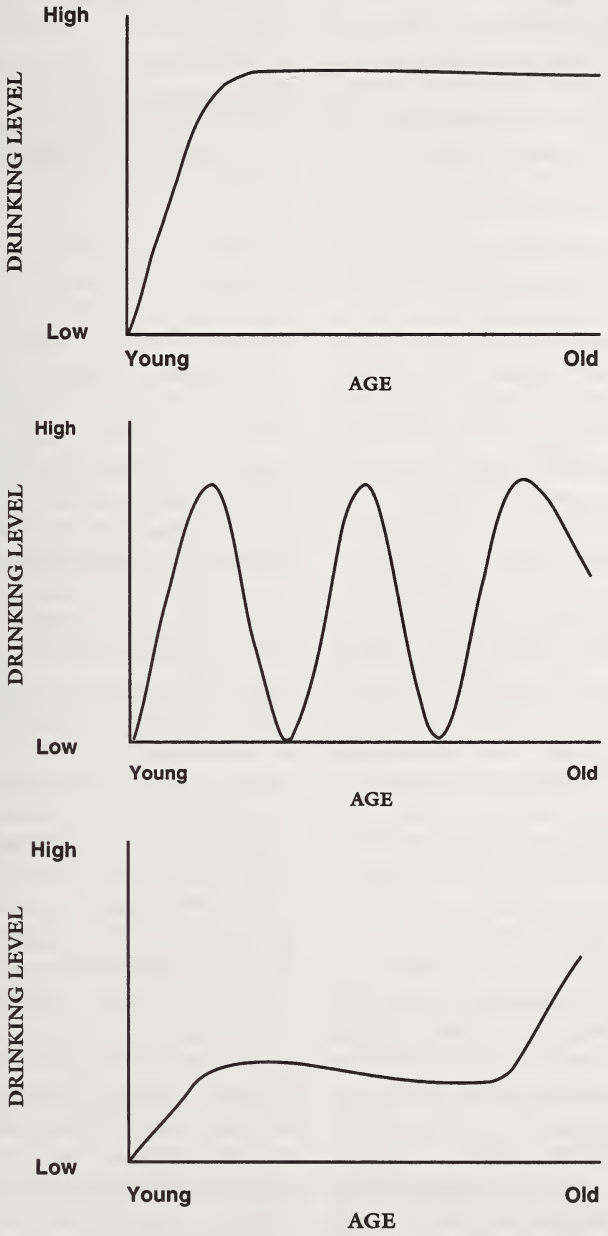

Figure 3 Drinking patterns of persons with problem use. (Top) Early-onset problem use. (Middle) Cyclical heavy use. (Bottom) Late-onset problem use.

and over (Liberto et al. 1992; Gambert and Katsoyannis 1995). The prevalence of alcohol dependence, defined as those patients currently drinking and meeting dependence criteria, was somewhat lower for all patients, with 10.4 percent for those 60–69, 6.8 percent for those 70–79, and 0.0 percent for those 80 and over. Although the rates of alcohol dependence are generally shown to decline with age, in one study of hospital discharge data, the 65-and-older group consistently had the highest proportion (approximately 60 percent) of alcohol-related diagnoses that were not primary diagnoses (Stinson et al. 1988).

In a large primary care study of 5,065 patients age 60 and over, Adams and colleagues (1996) found that 15 percent of the men and 12 percent of the women regularly drank in excess of the limits previously recommended by the National Institute on Alcohol Abuse and Alcoholism (NIAAA), > 7 drinks per week for women and > 14 drinks per week for men; newer NIAAA guidelines (1995) now recommend no more than 1 drink per day for both men and women over age 65. These guidelines are consistent with empirical evidence for risk-free drinking among older adults (Chermack et al. 1996). Eight percent of the men and 2 percent of the women in the primary care sample regularly consumed more than 21 drinks per week. Thus, there is a significant proportion of older adults seeking primary care treatment who are drinking at levels that can cause problems.

Studies of drinking behaviors among patients seeking care in primary care settings have shown that up to 45 percent report a history of excessive or problem use (Coulehan et al. 1987; Buchsbaum et al. 1991). Even lower levels of consumption could result in increased risk of injury or health problems, and these lower but still potentially problem-causing levels may occur in a large proportion of patients coming into contact with health care professionals (Cyr and Wartman 1988; Moore et al. 1989).

Symptoms of harmful drinking often are less visible among older adults, because they can be masked by social, medical, or psychological conditions. In addition, sensitivity to and tolerance of ethanol may be affected by the physiological aging processes (Rosin and Glatt 1971) as well as by health conditions (Baker 1981). Drinking produces higher blood alcohol levels in older adults than in younger persons when comparable amounts of alcohol are consumed, and many problems common among older people, such as chronic illness, poor nutrition, and polypharmacy, may be exacerbated by even small amounts of alcohol (Vestal et al. 1977). What might be considered light or moderate drinking for individuals in their thirties may have untoward health effects in an older person.

Heavy alcohol consumption in this group can be medically, psychologically, and socially hazardous, even if the frequency and amount of consumption do not warrant a formal diagnosis of alcohol abuse or dependence. As the health care system moves to managed care models, the development of strategies to deal with older adults who are at risk because of

their level of alcohol consumption is a growing concern.

RANDOMIZED CONTROLLED BRIEF INTERVENTION STUDIES WITH ADULTS UNDER AGE 65

Targeted early identification and secondary prevention programs for the elderly have been developed and shown to be effective in areas as diverse as cancer, heart disease, smoking, and diet (Blumenthal and Levenson 1987; Davenport and Whittaker 1988; Coambs et al. 1992; LaCroix and Omenn 1992). The success of these programs has sparked interest in the use of brief intervention techniques to decrease alcohol use.

Brief intervention techniques have been used to decrease alcohol use in adolescents, adults under 65 who are nondependent problem drinkers, and, most recently, older adults (Blow et al. unpublished manuscript; Fleming et al. unpublished manuscript). These clinically based interventions include assessment and direct feedback, contracting and goal setting, behavioral modification techniques, and the use of written materials such as self-help manuals. A number of trials, conducted primarily in Europe, have examined the efficacy of brief advice in reducing alcohol use. Although there are several ongoing studies of brief alcohol interventions for older adults, no reports from these studies are in the literature yet.

Kristenson and colleagues (1983) reported the results of a trial conducted in Malmo, Sweden, in the late 1970's. The subjects, advised to reduce their alcohol use in a series of health education visits, subsequently demonstrated significant reductions in gamma-glutamyltransferase levels and health care utilization up to 5 years after the brief interventions. The Medical Research Council trial, conducted in 47 general practitioners' offices in Great Britain (Wallace et al. 1988), found significant reductions in alcohol use by the intervention group compared with the control group 12 months following the intervention. The World Health Organization (WHO) Trial, conducted in 10 countries, found similar differences in alcohol use between the groups (Babor and Grant 1992).

In a brief alcohol intervention trial conducted with 67 community-based primary care physicians (Fleming et al. unpublished manuscript), known as Project TrEAT, 392 heavy drinkers under age 65 received a physician-delivered brief advice protocol (382 controls did not receive the protocol). The main outcomes were alcohol use measures, emergency department visits, and hospital days. There were no significant differences found between groups at baseline on alcohol use, age, socioeconomic status, smoking status, rates of depression or anxiety, frequency of conduct disorders, lifetime drug use, or health care utilization. At the 12-month followup, there was a significant reduction in 7-day alcohol use (t = 4.33, $p < 0.001$), episodes of binge drinking (t = 2.81, $p < 0.001$), and frequency of excessive drinking (t = 4.53, $p < 0.001$) in the experimental group compared with the control group. Chi-square tests of indepen-

dence revealed a significant relationship between group status and lengths of hospitalization over the study period for men ($p < 0.01$). Project TrEAT provided the first direct evidence that physician intervention with at-risk and problem drinkers under 65 decreases alcohol use and health resource utilization in a community-based U.S. health care system.

While the types of brief interventions have varied, the basic design of most studies is a randomized, controlled trial of individuals with hazardous drinking patterns who are assigned either to a brief intervention ranging from 1 to 10 sessions or to one or more control conditions (Kristenson et al. 1983; Chick 1988; Heather et al. 1987; Wallace et al. 1988; Persson and Magnusson 1989; Harris and Miller 1990; Anderson and Scott 1992; Babor and Grant 1992; Fleming et al. unpublished manuscript). The majority of these studies found significantly greater improvements in drinking outcomes for the brief intervention group compared with the control group (significant interaction effects for *time by condition*); however, 10 of the 11 reviewed found significant changes in drinking over time for both the control and brief intervention conditions (significant main effects for *time*).

STRATEGIES TO CHANGE DRINKING BEHAVIOR ACROSS STUDIES

Studies of brief interventions for alcohol problems have employed various approaches to change drinking behaviors. Strategies have ranged from relatively unstructured counseling and feedback to more formal structured therapy and have relied heavily on concepts and techniques from the behavioral self-control training literature (Miller and Munoz 1976; Miller and Taylor 1980; Miller and Rollnick 1991). Drinking goals of brief treatment interventions have been flexible, allowing the individual to choose drinking moderation or abstinence. The goal of brief counseling is to motivate the problem drinker to change his or her behavior, not to assign self-blame.

The characterization of hazardous, harmful, and dependent alcohol consumption patterns was used to distinguish the targets of brief intervention in the WHO study (Babor and Grant 1992). *Hazardous drinking* refers to that level of alcohol consumption or pattern of drinking that is likely to result in harm to the drinker should it persist. *Harmful drinking* is defined as alcohol use that has already resulted in adverse mental or physical effects. *Dependent use* refers to drinking that has resulted in physical, psychological, or social consequences and has been the focus of major diagnostic systems, such as DSM-IV (American Psychiatric Association 1994). Categorizing drinking patterns in this fashion provides both clinicians and researchers with flexible guidelines to identify individuals at risk for alcohol problems who may not meet criteria for alcohol dependence.

Most alcohol brief intervention studies have only included patients "early" in their drinking careers, explicitly excluding dependent drinkers with significant withdrawal symptoms.

The rationale for this practice has been that alcohol-dependent individuals or those affected most severely by alcohol should be referred to formal, specialized alcoholism treatment programs, because their conditions are not likely to be amenable to a low-intensity intervention (Babor et al. 1986; Institute of Medicine 1990). However, only one study to date has addressed the validity of this assumption. Sanchez-Craig and colleagues (1991) found that when comparing the 12-month treatment outcomes of severely dependent and not severely dependent men receiving brief treatment in Toronto and Brazil, there were no significant differences in "successful" outcomes as measured by rates of abstinence or moderate drinking. Further, rates of spontaneous remission of alcoholism suggest that some portion of the most severe alcoholic population will reduce or discontinue their drinking without formal intervention (Institute of Medicine 1990).

Brief intervention studies have been conducted in a wide range of health care settings, ranging from hospitals and primary health care locations (Chick et al. 1988; Wallace et al. 1988; Babor and Grant 1992; Fleming et al. unpublished manuscript) to mental health clinics (Harris and Miller 1990). Individuals recruited from such settings are likely to have some contact with a health care professional over the course of study participation, therefore having potential alcohol-related professional assistance available. Nonetheless, many of these patients would not be identified as having an alcohol problem by their health care provider and would not ordinarily receive any alcohol-specific intervention. The emphasis and short duration of brief intervention strategies have made them an important technique for consideration for use with injured patients in the emergency department who have alcohol problems.

BRIEF INTERVENTIONS FOR OLDER ADULTS

Older adults present unique challenges in applying brief intervention strategies for reducing alcohol consumption. The level of drinking necessary to be considered risky behavior is lower than for younger individuals (Chermack et al. 1996). Intervention strategies need to be nonconfrontational and supportive due to increased shame and guilt experienced by many older problem drinkers. Because of this shame and guilt, older adult problem drinkers find it particularly difficult to identify their own risky drinking. In addition, chronic medical conditions may make it more difficult for clinicians to recognize the role of alcohol in decreases in functioning and quality of life. These issues present barriers to conducting effective brief interventions for this vulnerable population.

Following identification of at-risk or problem drinkers through screening techniques, a semistructured brief intervention can be conducted. The content of the intervention needs to be elder specific and includes the following steps:

1. ***Identify future goals.*** Discuss how the older person would like his

or her life to improve and be different in the future (e.g., *What are some of your goals for the next 3 months to a year regarding your physical and emotional health, activities and hobbies, relationships and social life, and your financial situation and other parts of your life?).* This discussion is important because it helps to set the context for the brief intervention and generally provides increased motivation for the individual to change.

2. ***Provide customized feedback.*** This feedback should be in the form of a health profile on screening questions relating to drinking patterns and other health habits (e.g., smoking and nutrition); (e.g., *You indicated that, on average, you drink alcohol almost every day and drink one to two drinks at a time.*).

3. ***Discuss types of drinkers.*** Explain the type of drinkers in the United States and where the patient's drinking pattern fits into the population norms for their age group (e.g., *National guidelines recommend that men your age drink no more than seven drinks/week: no more than one/day. Your pattern of alcohol use fits into the at-risk drinking category*).

4. ***Introduce the concept of standard drinks.*** Explain that the alcohol content is roughly equivalent for a 12-oz beer, 1.5 oz of distilled spirits, 5 oz of wine, or 4 oz of sherry or liqueur. This concept provides the context for a discussion of sensible drinking limits.

5. ***Discuss reasons for drinking, the pros and cons of drinking, and reasons to cut down or quit drinking.*** This discussion is particularly important because the intervenor needs to understand both the positive and negative role of alcohol in the context of the older patient's life, including coping with loss and loneliness (e.g., *We've spent some time talking before about your sleep problems, your blood pressure problems, the fall you took in the bathroom, and your loneliness since your wife died.*). Some older patients may experience problems in physical, psychological, or social functioning even though they are drinking below cutoff levels (e.g., *Even though your drinking is close to the limit for people your age and you drank at this level for years, I am concerned about some of the health problems you've had and your loneliness.*). Maintaining independence, physical health, and mental capacity can be key motivators in this age group (e.g., *I am concerned that the amount you are drinking could be making some of these problems worse. Our goal is for you to remain as independent as possible and have a good quality of life.*).

6. ***Ask the person to consider changing, quitting, or cutting down on drinking.*** Discuss how changing drinking levels could have important benefits for the individual.

7. ***Suggest sensible drinking limits and strategies for cutting down or quitting.*** Strategies that are useful in this age group include developing social opportunities that do not involve alcohol, getting reacquainted with hobbies and interests from earlier in life, and pursuing volunteer activities, if possible.

8. ***Draw up a drinking agreement in the form of a "prescription."***

Negotiated drinking limits that are signed by the patient and the intervenor are particularly effective in changing drinking patterns.

9. ***Discuss how to cope with risky situations.*** Social isolation, boredom, and negative family interactions can present special problems in this age group, so work with the patient to develop strategies to deal with these problems. Role playing specific stressful situations can be helpful. The role play exercises will vary depending on the patient's particular situation.

10. ***Provide a summary of the session.*** The summary should include the drinking limits, encouragement, discussion of drinking diary cards (calendar) to be completed for the next month, and the recommendation to refer back to the workbook given to the individual during the intervention session. This workbook, which contains materials related to steps 1–9, provides opportunities for the patient and intervenor to complete sections on drinking cues, reasons for drinking, reasons to cut down or quit, the drinking agreement ("prescription"), and drinking diary cards for self-monitoring.

Providers can be trained to administer the intervention protocol through role playing and general skills training techniques in educational programs. The approach to patients is nonconfrontational and generally follows motivational interviewing principles as described by Miller and Rollnick (1991).

Two studies of brief advice for older at-risk and problem drinkers offer the opportunity to determine the effectiveness of two brief intervention protocols with different types of intervenors. The University of Wisconsin study (Fleming et al. unpublished manuscript) used primary care physicians in primary care settings; the University of Michigan study (Blow et al. unpublished manuscript) used nonphysician health care providers in primary care settings.

The University of Wisconsin Study

The recently concluded study by Fleming and colleagues was a randomized controlled brief physician intervention clinical trial for adults age 65 and older in which 24 community-based primary care practices (43 physicians) located in 10 Wisconsin counties took part. Of the 6,073 patients screened for problem drinking, 105 males and 53 females met inclusion criteria ($N = 158$) and were randomized into a control ($n = 71$) or intervention group ($n = 87$); 146 of the subjects in these groups participated in the 12-month followup procedures. The intervention consisted of two 10- to 15-minute, physician-delivered counseling visits that included advice, education, and contracting using a scripted workbook. No significant differences were found between groups at baseline on alcohol use, age, socioeconomic status, smoking status, rates of depression or anxiety, or health care utilization. At the time of the 12-month followup, the experimental group had significantly reduced 7-day alcohol use ($t = 3.77$; $p < 0.001$), episodes of binge drinking ($t = 2.68$; $p < 0.005$), and frequency of excessive drinking ($t = 2.65$; $p < 0.005$) when compared with the control group.

The University of Michigan Study

Unique aspects of the University of Michigan study include the oversampling of African American older adults, the use of multidimensional outcomes, and computerized feedback to the participants. Older patients in primary care clinics in southeastern Michigan and northwest Ohio (Toledo area) were asked to complete a health survey while they waited for their primary care physician appointments. Patients who agreed to participate were told that they might be called for future studies; however, they were not sensitized to issues of alcohol use because alcohol questions were embedded among questions regarding health, smoking, and exercise. The survey required about 15 minutes to complete. Patients identified as at-risk/hazardous drinkers were invited to participate in a clinical trial of an elder-specific brief intervention. The clinical trial portion of the study is expected to run through spring of 1999.

The primary care clinics in this study included single physician offices, multiphysician health maintenance organization clinics, and outpatient general medicine clinics at Veterans Administration (VA) medical centers. At all sites except the VA clinics, participants were given a ticket for a bimonthly drawing for $100 as an incentive to complete the survey. The majority of the surveys (85 percent) were self-administered in the waiting rooms. The remaining 15 percent of the sample were interviewed by a research assistant. Interviews were conducted at the participant's request, typically for reasons of visual difficulties, or physical disabilities that precluded writing. Interviews typically took place in waiting rooms using response cards to preserve the participants' confidentiality. Persons who could not read were given the interview in oral format in a room away from the clinic waiting area. The response rate was 70 percent.

Participant Demographics

A total of 4,772 older adults were screened in primary care settings between February 1995 and February 1997. Forty-five percent (n = 2,122) were women. The mean age of participants was 68.6 (SD = 7.4), with a range from 55 to 95 years of age. Eighty-two percent were Caucasian, 15 percent were African American, and 3 percent were other races. Sixty-four percent were married, 18 percent widowed, 11 percent divorced, 3 percent never married, 2 percent separated, and 2 percent had live-in partners. Forty-two percent had completed high school, 29 percent had completed less than high school, and 29 percent had completed at least some college.

Measures

The study's health survey included the Medical Outcomes Study Short Form, 36-item General Health Survey (MOS SF-36); health habits questions including alcohol use in the past 3 months; and the Short Michigan Alcoholism Screening Test—Geriatric Version (SMAST-G) and the CAGE for the last year.

The MOS SF-36 is an interview that was originally developed for use with adults as a 20-item scale for the

Medical Outcomes Study (Stewart et al. 1988; Tarlov et al. 1989; Ware and Sherbourne 1992) from more detailed measures utilized in the Rand Health Experiment. It was subsequently expanded to 36 items. As currently defined, the SF-36 measures eight domains: general health, physical functioning, physical role functioning, pain, vitality, mental health functioning, emotional role functioning, and social functioning. The SF-36 has published norms for these various subscales (McHorney et al. 1991). Likert-scaled items assessed health status. Negatively keyed items were recoded. Within each domain, scores were summed, and the total was then expressed as a percentage of the maximum possible. Higher scores indicated better functioning. The SF-36 subscales were the dependent variables of interest in the current study.

Questions about alcohol use included lifetime use, past year use, and reasons for quitting (if currently abstinent). Three alcohol consumption questions were asked for the previous 3-month period, covering average frequency, average quantity, and binge frequency. The number of drinking days per week was multiplied by the reported number of drinks per drinking day to form the alcohol use measure (number of standard drinks per week). Cutoff levels for hazardous drinking were at least nine drinks per week for women and at least 12 drinks per week for men. These cutoff levels were chosen to be lower than those recommended by Sanchez-Craig and colleagues (1995) for younger samples due to age-related changes in alcohol sensitivity.

The frequency of binge drinking was an additional criterion for classifying participants as high-risk drinkers. Binge drinking was defined as drinking four or more standard drinks on a drinking occasion. To meet the criterion for binge drinking frequency in this study, a participant had to report drinking four or more drinks at a time at least twice in the past 3 months; this criterion is lower than that recommended for younger adults.

The SMAST-G is the short form of the Michigan Alcoholism Screening Test—Geriatric Version (MAST-G), and was developed from the Michigan Alcoholism Screening Test (MAST) as a screen for alcohol problems among elderly patients (Blow et al. 1992). Consisting of 24 elder-specific items, the MAST-G has been shown to have high sensitivity and specificity in screening for alcohol problems in a wide range of elderly problem drinkers, ranging from hazardous and harmful use to dependence. The SMAST-G consists of the 10 items from the original version that showed the best sensitivity and specificity. A sample item is: "In the last year, have you usually taken a drink to relax or calm your nerves?" A score of 2 or more (i.e., two "yes" responses) indicates probable alcohol problems. The SMAST-G questions asked about participants' experiences within the last year.

The CAGE (Mayfield et al. 1974) is the most widely used alcohol problem screening test in clinical practice. It contains four items regarding alcohol use: wanting to *C*ut down, feeling *A*nnoyed that people criticized one's drinking, feeling *G*uilty about others

criticizing drinking, and having an *E*yeopener in the morning to get rid of a hangover. Two positive responses are considered a positive screen and indicate that further assessment is warranted. The sensitivity and specificity of the CAGE varies from 60 to 90 percent and from 40 to 90 percent, respectively (Mayfield et al. 1974; Ewing 1984). For this study, participants were asked to respond to questions according to their experiences within the last year. CAGE alcohol items were embedded along with CAGE-like items about exercise, smoking, and weight (Fleming and Barry 1991). For example, participants were asked consecutively if, in the last year, they felt guilty about their smoking, lack of exercise, weight, or alcohol use.

Preliminary Screening Results

Sixty-six percent of the older adults in the study to date were abstainers, 28 percent were low-risk drinkers, and 6 percent were at-risk drinkers or heavy drinkers. Significant gender differences were found, with 8.6 percent of men but only 2.6 percent of women identified as at-risk drinkers. A multivariate analysis of variance procedure (MANOVA), controlling for race and gender, found main effects of drinking status on general health, physical functioning, physical role functioning, pain, vitality, mental health functioning, emotional role functioning, and social functioning. Low-risk drinkers scored better than abstainers on all measures, and better than at-risk drinkers on all measures except physical role functioning, vitality, and social functioning (table 1). There were main effects of race on general health and social functioning. Older adults who are at risk for alcohol problems may not present with poor physical health functioning but seem to present with lower mental health functioning. Further dissemination of screening data results is under way. The brief alcohol intervention trial is in year 4 of 5 years.

TREATMENT OUTCOMES FOR OLDER ADULTS

Although alcoholism is a significant and growing health problem in the United States (Gomberg 1980; American Medical Association, Council on Scientific Affairs 1996), there have been few systematic studies of alcoholism treatment outcome among older adults (Atkinson 1995). The study of treatment outcomes for older adults who meet criteria for alcohol abuse/dependence has become a critical issue because of their unique needs for targeted treatment intervention. Because traditional residential alcoholism treatment programs generally provide services to few older adults, sample size issues have been a barrier to studying treatment outcomes for elderly alcoholics in most settings. The development of elder-specific alcoholism treatment programs in recent years has provided sufficiently large numbers of older alcoholics to facilitate studies of this special population (Atkinson 1995).

A remaining limitation with this population is the lack of longitudinal studies of treatment outcomes.

Table 1. Mean SF-36 Scale Scores by Drinking Risk Group in Primary Care Settings.

SF-36 Subscale	Lifetime Abstainers (n = 557) Mean (SD)	Other Abstainers (n = 2,587) Mean (SD)	Low-Risk Drinkers (n = 1,343) Mean (SD)	At-Risk Drinkers (n = 139) Mean (SD)	Heavy Drinkers (n = 146) Mean (SD)
General health	58.91 (21.98)	53.26 (22.16)	61.54 (20.74)	57.83 (20.07)	57.43 (20.46)
Physical functioning	62.97 (30.75)	59.22 (29.59)	69.70 (26.74)	66.09 (28.63)	62.65 (27.76)
Physical role functioning	55.10 (41.43)	48.18 (42.01)	59.34 (40.34)	65.61 (39.19)	57.54 (40.54)
Pain	60.68 (28.11)	57.06 (27.65)	66.04 (26.07)	64.01 (24.06)	60.76 (27.81)
Vitality	56.04 (24.16)	51.33 (22.76)	56.86 (21.24)	57.87 (19.78)	55.76 (19.89)
Mental health functioning	75.76 (18.65)	71.96 (19.09)	76.64 (15.66)	73.25 (19.51)	71.36 (20.47)
Emotional role functioning	77.44 (34.81)	74.15 (37.20)	82.72 (31.00)	80.17 (33.76)	75.86 (36.07)
Social functioning	81.50 (24.99)	78.71 (25.53)	85.62 (21.23)	85.75 (20.90)	81.86 (23.80)

Previous research on elderly alcoholism treatment can be categorized into two broad categories, treatment compliance studies and prospective studies of treatment outcomes.

Treatment Compliance Studies

Most treatment outcome research for elderly alcoholics has focused on compliance with treatment program expectations, in particular the patient's fulfillment of prescribed treatment activities and goals, including drinking behavior (Atkinson 1995). Results from compliance studies have shown that age-specific programming improved treatment completion and resulted in higher rates of attendance at group meetings compared with mixed-age treatment (Kofoed et al. 1987). In addition, older alcoholics were significantly more likely to complete treatment than younger patients (Wiens et al. 1982–83). Atkinson and colleagues (1993) found that the proportion of older male alcoholics completing treatment was twice that of younger men.

Age of onset of alcohol problems has been a major focus of research for elderly treatment compliance studies. In a study by Schonfeld and Dupree (1991) using a matched-pairs, post

hoc design, rates of completion of 6-month day treatment for 23 older men and women alcoholics (age 55 and older) whose problem drinking began before age 50 (early onset) were compared with completion rates for 23 who began problem drinking after age 50 (late onset). Those classified as late-onset alcoholics were significantly more likely to complete treatment, although in a subsequent report including the larger sample of 148 from which these patients were selected, there was no difference in completion rate based on age of onset (Schonfeld and Dupree 1991).

In another study of 132 male alcoholic veterans 60 years of age and older, the sample was divided into early-onset (age 40 and younger, n = 50), midlife onset (ages 41–59, n = 62), and late-onset (age 60 and older, n = 20) subgroups (Atkinson 1990). Age of onset was related to program completion and to weekly meeting attendance, with the late-onset group showing the best compliance. However, a subsequent analysis of 128 older men in alcoholism treatment (age 55 and older) found that drinking relapses during treatment were unrelated to age of onset (Atkinson et al. 1993). Furthermore, onset age did not contribute significantly to variance in program completion, but was related to meeting attendance rate (Atkinson et al. 1993). Thus, studies on the effect of age of onset on treatment compliance have yielded mixed results.

In a study of treatment matching, Rice and colleagues (1993) compared drinking outcomes for randomly assigned men and women alcoholics 3 months after beginning one of three mixed-age outpatient treatment conditions scheduled to last for 4 months. The sample included 53 patients ages 18–29, 134 patients ages 30–49, and 42 patients age 50 years and older. There were no main effects of age or treatment condition on treatment compliance. However, there were significant age x group x treatment condition effects. For older patients, the number of days abstinent was greatest and the number of heavy drinking days fewest among those treated in an individual-focused rather than in a group condition. This study suggested that elderly alcoholics may respond better to individual-focused interventions, rather than traditional mixed-age group-oriented treatment.

There remain major limitations in the treatment compliance literature, including lack of drinking outcome data, failure to report on treatment dropouts, and variations in definitions of treatment completion. Few carefully controlled, prospective treatment outcome studies including sufficiently large numbers of older alcoholics have been conducted to address the methodological limitations of prior work.

Prospective Studies of Treatment Outcomes

There are few prospective treatment outcome studies reported in the literature, in part due to the complexity of studying older alcoholics in treatment, and in part due to difficulties in following them after completion of treatment. Thus, sample sizes tend to be too small to provide definitive results.

A notable exception is a study of 137 male veterans with alcohol problems (ages 45–59 years, $n = 64$; ages 60–69 years, $n = 62$; age 70 and older, $n = 11$) who were randomly assigned after detoxification to age-specific treatment or standard mixed-age treatment (Kashner et al. 1992). Outcomes at 6 months and 1 year showed that elder-specific program patients were 2.9 times more likely at 6 months and 2.1 times more likely at 1 year to report abstinence compared with mixed-age group patients. Treatment groups, however, could not be compared at baseline because baseline alcohol consumption and alcohol severity data were not included in the study.

Physical and Psychiatric Comorbidity

Several factors have been associated with treatment outcome in previous studies of younger adults that are relevant for the elderly, including psychiatric comorbidities and physical and emotional health functioning. Both physical comorbidity and psychiatric comorbidity are common among older alcoholics seeking treatment (Finlayson et al. 1988; Hurt et al. 1988; Blow et al. 1992; Moos et al. 1993; Tyas and Rush 1994). These comorbidities might have positive as well as negative impacts on treatment outcomes in this vulnerable population (Atkinson 1995). For example, poor general health functioning could improve drinking outcomes because drinking may become incompatible or unpleasant for those with significant physical problems. Conversely, poor general health functioning may force some patients to discontinue treatment prematurely, reducing treatment compliance (Atkinson 1995).

Limitations of Previous Treatment Outcome Research

While the examination of factors related to completion of programming is important for the identification of patient characteristics for those who will remain in treatment, existing studies have an inherent selectivity bias and provide no information on treatment dropouts or on short- or long-term treatment outcomes. Other issues with sampling may also limit the generalizability of previous studies. For example, the majority of reports on elderly alcoholism treatment outcome have included only men. Furthermore, age cutoffs for inclusion in studies have varied widely: some studies have included non-elderly persons in the "older" category, with several studies including persons as young as age 45.

In addition to sampling issues, there are limitations related to assessment techniques and approaches. The majority of studies have utilized relatively unstructured techniques for assessing alcohol-related symptoms and consequences of drinking behavior. Also, the manner in which outcomes have been assessed has been narrow in focus. Most studies have dichotomized treatment outcome (abstention vs. relapse) based solely on drinking behavior. Given evidence from numerous studies that heavy or binge drinking is more strongly related to alcohol consequences than is average alcohol

consumption (Anda et al. 1988; Kranzler et al. 1990; Chermack et al. 1996), it is possible that there are important differences in outcome for nonabstinent individuals, depending on whether their reuse of alcohol following treatment involves binge drinking. For these reasons researchers have suggested that nonabstinent drinking outcomes should be categorized along dimensions such as whether drinkers ever drink to the point of intoxication (Heather and Tebbutt 1989). Finally, previous studies have not addressed other relevant domains that may be positively affected by treatment, such as physical and mental health status and psychological distress.

Recognizing that elderly individuals have been underrepresented in standard alcoholism treatment programs (Booth et al. 1992; Higuchi and Kono 1994), as well as in treatment outcome studies (Atkinson 1995), a report published by the Institute of Medicine (1990) called for specific studies focusing on the long-term impact of treatment and on factors associated with more successful treatment outcomes in the elderly. A just completed study at the University of Michigan conducted by my research group (Blow et al. unpublished manuscript) was designed to address these issues. This study is described in the following section.

University of Michigan Treatment Outcome Study

We conducted a study to determine treatment outcomes for older adults receiving specialized elder-specific inpatient alcoholism treatment. Using a prospective, longitudinal study design, the purpose of this study was to determine treatment outcome among older adults seeking treatment in an inpatient elder-specific alcoholism treatment program. To address limitations of previous work, this study utilized validated techniques to assess baseline alcohol symptoms and psychiatric comorbidity, age of onset of alcohol problems, pre- and posttreatment drinking patterns, physical and emotional health functioning, and psychological distress. The study also examined a range of different drinking outcomes, including abstinence, non–binge drinking, and binge drinking. Finally, multiple outcome measures were included in addition to reported drinking behavior, such as measures of reported physical and emotional health and psychological distress.

Adults over the age of 55 in the treatment program ($N = 90$) were interviewed. Structured interviews detailing drinking histories, psychiatric measures, and health functioning were conducted at baseline and by telephone 6 months after discharge. The physical health functioning of the sample was similar to that reported by seriously medically ill inpatients in other studies, while psychosocial functioning (as assessed by the social functioning and emotional role functioning subscales) was significantly worse (table 2). Nearly one-third of the sample had one or more comorbid psychiatric disorders, with anxiety disorders and major depression most common.

Participants who completed the 6-month followup assessment ($n = 65$) were classified into three outcome groups: abstainers ($n = 35$), non–

Table 2. Mean SF-36 Scores Among Older Alcoholics in Treatment Compared With Previously Reported Means for Seriously Medically Ill Patients.

SF-36 Subscale	Older Alcoholics	Seriously Medically Ill Patients[1]
Physical functioning	59.1	57.4
Social functioning*	50.6	80.0
Physical role functioning	31.7	43.9
Emotional role functioning*	43.2	76.2
Mental health*	59.8	77.6
Vitality	45.5	47.8
Pain	57.4	65.1
General health perceptions	55.4	49.1

[1] Data from McHorney et al. 1991.
* $p < 0.01$.

binge drinkers (who never exceeded four drinks on any drinking day during the followup period; $n = 12$), and binge drinkers (who had 1 or more days in which they consumed five or more drinks; $n = 18$). There were 25 non-completers, who did not complete the 6-month followup assessment. These groups did not differ significantly on demographic variables, pretreatment drinking patterns and symptoms, age of onset of alcohol problems, psychiatric comorbidity, or length of stay in treatment. General health improved between baseline and followup for all groups. Psychological distress decreased significantly between baseline and followup for abstainers and non–binge drinkers. However, binge drinkers did not show a decline in psychological distress, and they were significantly more distressed at 6-month followup than both the abstainers and the non–binge drinkers.

Results suggest that a large percentage of older adults who receive elder-specific treatment attain abstinent outcomes, and that both abstainers and individuals who refrain from binge drinking exhibit marked reductions in psychological distress. Psychiatric comorbidity was not associated with drinking outcome. Future research in this area should focus on further delineating risk factors for drinking relapse, including a better understanding of specific treatment component needs for older adults with alcohol problems.

CONCLUSIONS

The availability of a range of prevention and intervention strategies for older adults—from prevention, education, and brief advice for persons who are abstinent or low-risk drinkers to brief, structured interventions and formalized treatment for older persons with alcohol abuse/dependence—provides the tools for health care providers to work with older adults across a spectrum of drinking patterns. The report by the Institute of Medicine (1990) found that most alcohol-related problems occur in nondependent

drinkers. To implement alcohol prevention and brief intervention strategies in clinical practice will require the development of systematized protocols that provide easy service delivery. The need to implement effective strategies with a variety of older drinkers who are at risk for more serious health, social, and emotional problems is high, both from a public health perspective and from a clinical perspective. With changes in the health care system to managed models of care, the time is right to move forward into a comprehensive system of alcohol interventions with older adults, the fastest growing segment of the U.S. population and one of the most vulnerable.

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REFERENCES

Adams, W.L.; Yuan, Z.; Barboriak, J.J.; and Rimm, A.A. Alcohol-related hospitalizations of elderly people: Prevalence and geographic variation in the United States. *JAMA* 270:1222–1225, 1993.

Adams, W.L.; Barry, K.L.; and Fleming, M.F. Screening for problem drinking in older primary care patients. *JAMA* 276:1964–1967, 1996.

American Medical Association, Council on Scientific Affairs. Alcoholism in the elderly. *JAMA* 275:797–801, 1996.

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 4th ed. Washington, DC: the Association, 1994.

Anda, R.R.; Williamson, D.F.; and Remington, P.L. Alcohol and fatal injuries among U.S. adults: Findings from the NHANES I Epidemiologic Follow-Up Study. *JAMA* 260:2529–2532, 1988.

Anderson, P., and Scott, E. The effect of general practitioners' advice to heavy drinking men. *Br J Addict* 87:891–900, 1992.

Atkinson, R.M. Aging and alcohol use disorders: Diagnostic issues in the elderly. *Int Psychogeriatr* 2:55–72, 1990.

Atkinson, R.M. Treatment programs for aging alcoholics. In: Beresford, T.P., and Gomberg, E.S.L., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 186–210.

Atkinson, R.M.; Tolson, R.L.; and Turner, J.A. Factors affecting outpatient treatment compliance of older male problem drinkers. *J Stud Alcohol* 54:102–106, 1993.

Babor, T.F., and Grant, M., eds. *Project on Identification and Management of Alcohol-Related Problems. Report on Phase II: A Randomized Clinical Trial of Brief Interventions in Primary Health Care.* Geneva: World Health Organization, 1992.

Babor, T.F.; Ritson, E.B.; and Hodgson, R.J. Alcohol-related problems in the primary health care setting: A review of early intervention strategies. *Br J Addict* 81:23–46, 1986.

Baker, S.L. Substance abuse disorders in aging veterans. In: Gottheil, E.; McLellan, A.T.; and Druley, K.A., eds. *Matching Patient Needs and Treatment Methods in Alcoholism and Drug Abuse.* Springfield, IL: Charles C. Thomas, 1981.

Barry, K.L. Alcohol and drug abuse. In: Mengel, M., and Hellerman, W., eds. *Fundamentals of Clinical Practice: A Textbook on the Patient, Doctor, and Society.* New York: Plenum Press, 1997. pp. 335–357.

Barry, K.L., and Fleming, M.F. The family physician. *Alcohol Health Res World* 18:105–109, 1994.

Beresford, T.P. Alcoholism consultation and general hospital psychiatry. *Gen Hosp Psychiatry* 1:293–300, 1979.

Beresford, T.P.; Blow, F.C.; Hill, E.; Singer. K.; and Lucey, M.R. Comparison of CAGE questionnaire and computer-assisted laboratory profiles in screening for covert alcoholism. *Lancet* 336:482–485, 1990.

Blow, F.C.; Brower, K.J.; Schulenberg, J.E.; Demo-Dananberg, L.M.; Young, J.P.; and Beresford, T.P. The Michigan Alcoholism Screening Test—Geriatric Version (MAST-G): A new elderly-specific screening instrument. *Alcohol Clin Exp Res* 16:372, 1992.

Blumenthal, J.A., and Levenson, R.M. Behavioral approaches to secondary prevention of coronary heart disease. *Circulation* 76:I130–1137, 1987.

Booth, B.M.; Blow, F.C.; Cook, C.A.L.; Bunn, J.Y.; and Fortney, J.C. Age and ethnicity among hospitalized alcoholics: A nationwide survey. *Alcohol Clin Exp Res* 16:1029–1034, 1992.

Buchsbaum, D.G.; Buchanan, R.G.; Centor, R.M.; Schnoll, S.H.; and Lawton, M.J. Screening for alcohol abuse using CAGE scores and likelihood ratios. *Ann Intern Med* 115:774–777, 1991.

Chermack, S.T.; Blow, F.C.; Hill, E.M.; and Mudd, S.A. The relationship between alcohol symptoms and consumption among older drinkers. *Alcohol Clin Exp Res* 20: 1153–1158, 1996.

Chick, J. Early intervention for hazardous drinking in the general hospital setting. *Aust Drug Alcohol Rev* 7:339–343, 1988.

Coambs, R.B.; Li, S.; and Kozlowski, L.T. Age interacts with heaviness of smoking in predicting success in cessation of smoking. *Am J Epidemiol* 135:240–246, 1992.

Coulehan, J.L.; Zettler-Segal, M.; Block, M.; McClelland, M.; and Schulberg, H.C. Recognition of alcoholism and substance abuse in primary care patients. *Arch Intern Med* 147:349–352, 1987.

Cyr, M.G., and Wartman, S.A. The effectiveness of routine screening questions in the detection of alcoholism. *JAMA* 259: 51–54, 1988.

Davenport, J. and Whittaker, K. Secondary prevention in elderly survivors of heart attacks. *Am Fam Physician* 38:216–224, 1988.

Dufour, M., and Fuller, R.K. Alcohol in the elderly. *Annu Rev Med* 46:123–132, 1995.

Ewing, J.A. Detecting alcoholism: The CAGE questionnaire. *JAMA* 252:1905–1907, 1984.

Finch, J., and Barry, K.L. Substance use in older adults. In: Fleming, M.F., and Barry, K.L., eds. *Addictive Disorders.* Mosby Yearbook, 1992. pp. 270–286.

Finlayson, R.E.; Hurt, R.D.; Davis, L.J.; and Morse, R.M. Alcoholism in elderly persons: A study of the psychiatric and psychosocial features of 216 inpatients. *Mayo Clinic Proc* 63:761–768, 1988.

Fleming, M.F., and Barry, K.L. A three-sample test of an alcohol screening questionnaire. *Alcohol Alcohol* 26:81–91, 1991.

Fleming, M.; Barry, K.; Manwell, L.; Johnson., K.; and London, R. A trial of early alcohol treatment (Project TrEAT): A randomized trial of brief physician

advice in community-based primary care practices. *JAMA* 277:1039–1045, 1997. (With editorial p. 1045).

Gambert, S.R., and Katsoyannis, K.K. Alcohol-related medical disorders of older heavy drinkers. In: Beresford, T., and Gomberg, E.S.L., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 70–81.

Gomberg, E.S.L. *Drinking and Problem Drinking Among the Elderly.* Ann Arbor: University of Michigan Institute of Gerontology, 1980.

Gurland, B.J., and Cross, P.S. Epidemiology of psychopathology in old age: Some implications for clinical services. *Psychiatr Clin North Am* 5:11–26, 1982.

Harris, K.B., and Miller, W.R. Behavioural self-control training for problem drinkers: Components of efficacy. *Psychol Addict Behav* 4:90–92, 1990.

Heather, N., and Tebbutt, J. Definition of non-abstinent and abstinent categories in alcoholism treatment outcome classifications: A review and proposal. *Drug Alcohol Depend* 24:83–93, 1989.

Heather, N.; Campion, P.D.; Neville, R.G.; and MacCabe, D. Evaluation of a controlled drinking minimal intervention for problem drinkers in general practice (the DRAMS scheme). *J R Coll Gen Pract* 37:358–363, 1987.

Higuchi, S., and Kono, H. Early diagnosis and treatment of alcoholism: The Japanese experience. *Alcohol Alcohol* 29:263–273, 1994.

Hurt, R.D.; Finlayson, R.E.; Morse, R.M.; and Davis, L.J. Alcoholism in elderly persons: Medical aspects and prognosis of 216 inpatients. *Mayo Clin Proc* 63:753–760, 1988.

Institute of Medicine. *Broadening the Base of Treatment for Alcohol Problems.* Washington, DC: National Academy Press, 1990.

Kashner, T.M.; Rodell, D.E.; Ogden, S.R.; Guggenheim, F.G.; and Karson, C.N. Outcomes and costs of two VA inpatient treatment programs for older alcoholic patients. *Hosp Community Psychiatry* 43:985–998, 1992.

Kofoed, L.L.; Tolson, R.L.; Atkinson, R.M.; Toth, R.L.; and Turner, J.A. Treatment compliance of older alcoholics: An elder-specific approach is superior to "mainstreaming." *J Stud Alcohol* 48:47–51, 1987.

Kranzler, H.R.; Babor, T.F.; and Lauerman, K.J. Problems associated with average alcohol consumption and frequency of intoxication in a medical population. *Alcohol Clin Exp Res* 14:119–126, 1990.

Kristenson, H.; Ohlin, H.; Hulten-Nosslin, M.; Trell, E.; and Hood, B. Identification and intervention of heavy drinking in middle-aged men: Results and follow-up of 24–60 months of long-term study with randomized controls. *Alcohol Clin Exp Res* 7:203–209, 1983.

LaCroix, A.Z., and Omenn, G.S. Older adults and smoking. *Clin Geriatr Med* 8:69–87, 1992.

Liberto, J.G.; Oslin, D.W.; and Ruskin, P.E. Alcoholism in older persons: A review of the literature. *Hosp Community Psychiatry* 43:975–984, 1992.

Mayfield, D.; McLeod, G.; and Hall, P. The CAGE questionnaire: Validation of a new alcoholism screening instrument. *Am J Psychiatry* 131:1121–1128, 1974.

McHorney, C.A.; Ware, J.E.; and Raczek, A.E. The MOS 36-Item Short-Form Health Survey (SF-36): II. Psychometric

and clinical tests of validity of measuring physical and mental health constructs. *Med Care* 31:247–263, 1991.

Miller, W.R., and Munoz, R.F. *How To Control Your Drinking.* Englewood Cliffs, NJ: Prentice-Hall, 1976.

Miller, W.R., and Rollnick, S. *Motivational Interviewing.* New York: Guilford Press, 1991.

Miller, W.R., and Taylor, C.A. Relative effectiveness of bibliotherapy, individual and group self-control training in the treatment of problem drinkers. *Addict Behav* 5:13–24, 1980.

Moore, R.D.; Bone, L.R.; Geller, G.; Mamon, J.A.; Stokes, E.J.; and Levine, D.M. Prevalence, detection, and treatment of alcoholism in hospitalized patients. *JAMA* 261:403–407, 1989.

Moos, R.H.; Mertens, J.R.; and Brennan, P.L. Patterns of diagnosis and treatment among late-middle-aged and older substance abuse patients. *J Stud Alcohol* 54:479–487, 1993.

National Institute on Alcohol Abuse and Alcoholism (NIAAA). *The Physicians' Guide to Helping Patients With Alcohol Problems.* NIH Pub. No. 95–3769. Bethesda, MD: National Institutes of Health, 1995.

Persson, J., and Magnusson, P.H. Early intervention in patients with excessive consumption of alcohol: A controlled study. *Alcohol* 6:403–408, 1989.

Rice, C.; Longabaugh, R.; Beattie, M.; and Noel, N. Age group differences in response to treatment for problematic alcohol use. *Addiction* 88:1369–1375, 1993.

Robins, L.N., and Regier, D.A. *Psychiatric Disorders in America: The Epidemiologic Catchment Area Study.* New York: Free Press, 1991.

Rosin, A.J., and Glatt, M.M. Alcohol excess in the elderly. *Q J Stud Alcohol* 32:53–59, 1971.

Sanchez-Craig, M.; Neumann, B.; Souza-Formigoni, M.; and Rieck, L. Brief treatment for alcohol dependence: Level of dependence and treatment outcome. *Alcohol Alcohol* Suppl 1:515–518, 1991.

Sanchez-Craig, M.; Wilkinson, D.A.; and Davila, R. Empirically based guidelines for moderate drinking: 1-year results from three studies with problem drinkers. *Am J Public Health* 85:823–828, 1995.

Schonfeld, L., and Dupree, L.W. Antecedents of drinking for early- and late-onset elderly alcohol abusers. *J Stud Alcohol* 52:587–592, 1991.

Schuckit, M.A. A clinical review of alcohol, alcoholism, and the elderly patient. *J Clin Psychiatry* 43:396–399, 1982.

Schuckit, M.A., and Pastor, P.A., Jr. The elderly as a unique population: Alcoholism. *Alcohol Clin Exp Res* 2:31–38, 1978.

Scott, E., and Anderson, P. Randomized controlled trial of general practitioner intervention in women with excessive alcohol consumption. *Drug Alcohol Rev* 10:313–321, 1990.

Stewart, A.L.; Hays, R.D.; and Ware, J.E., Jr. The MOS Short-Form General Health Survey: Reliability and validity in a patient population. *Med Care* 26:724–735, 1988.

Stinson, F.S.; Dufour, M.C.; and Bertolucci, D. Alcohol-related morbidity in the aging population. *Alcohol Health Res World* 13:80–87, 1988.

Tarlov, A.R.; Ware, J.E., Jr; Greenfield, S.; Nelson, E.C.; Perrin, E.; and Zubkoff,

M. The Medical Outcomes Study: An application of methods for monitoring the results of medical care. *JAMA* 262:925–930, 1989.

Tyas, S.L., and Rush, B.R. Trends in the characteristics of clients in alcohol/drug treatment services in Ontario. *Can J Public Health* 85:13–16, 1994.

Vestal, R.E.; McGuire, E.A.; Tobin, J.D.; Andreas, R.; Norris, A.H.; and Mezey, E. Aging and ethanol metabolism. *Clin Pharmacol Ther* 231:343–354, 1977.

Wallace, P.; Cutler, S.; and Haines, A. Randomized controlled trial of general practitioner intervention in patients with excessive alcohol consumption. *BMJ* 297:663–668, 1988.

Ware, J.E. Jr., and Sherbourne, C.D. The MOS 36-Item Short-Form Health Survey (SF-36): I. Conceptual framework and item selection. *Med Care* 30:473–481, 1992.

Wiens, A.N.; Menustik, C.E.; Miller, S.I.; and Schmitz, R..E. Medical-behavioral treatment of the older alcoholic patient. *Am J Drug Alcohol Abuse* 9:461–475, 1982–83.

Williams, G.D., and Debakey, S.F. Changes in levels of alcohol consumption: United States, 1983 to 1988. *Br J Addict* 87:643–648, 1992.

Chapter 22

Natural Recovery Over the Lifespan

Mark B. Sobell, Ph.D., Linda C. Sobell, Ph.D.,
John C. Cunningham, Ph.D., and Sangeeta Agrawal, M.S.

Natural recovery from alcohol problems refers to the process of recovering without formal treatment or self-help groups. Several years ago we examined the phenomenon of natural recovery across the lifespan in individuals who had recovered from alcohol problems (Sobell et al. 1993). The recovered subjects were divided into three cohorts, ages 20–35, 36–50, and ≥ 51 years. The major finding for the oldest group was that their drinking problem began in middle age, with a mean age of onset of 42 years. The mean age of onset for the 20–35 group was 22, and for the 36–50 group was 28. Although consistent with recent research about middle- and late-age onset of drinking problems (Atkinson 1994; Brennan and Moos 1996), this finding was at variance with clinical opinion. For example, the DSM-III-R (American Psychiatric Association 1987) stated: "In males, symptoms of Alcohol Dependence or Abuse rarely occur for the first time after age 45" (p. 174). The other age differences among the naturally recovered subjects primarily involved the younger group being different from the two older groups and are not relevant to the present discussion.

THE FOSTERING SELF-CHANGE STUDY

Goals and Methods

As an extension of research on natural recoveries, we are conducting a project

M.B. Sobell, Ph.D., and L.C. Sobell, Ph.D., are professors at the Center for Psychological Studies, Nova Southeastern University, 3301 College Ave., Fort Lauderdale, FL 33314; they are also senior scientists with the Clinical Research and Treatment Institute, Addiction Research Foundation, 33 Russell St., Toronto, Ontario, Canada M5S 2S1. J.C. Cunningham, Ph.D., is a scientist with the Clinical Research and Treatment Institute, Addiction Research Foundation, and is assistant professor in the Department of Behavioural Sciences, University of Toronto, Toronto, Ontario, Canada. S. Agrawal, M.S., is a senior research assistant with the Clinical Research and Treatment Institute, Addiction Research Foundation.

aimed at promoting self-recoveries in the community (Sobell et al. 1996). This project, which is being conducted in Toronto, is designed to encourage people with drinking problems to attempt self-change and to increase the likelihood of their succeeding. Such individuals cannot have had previous help or treatment for their alcohol problem. They are being solicited through advertisements asking for individuals who want to change their drinking on their own using free self-help materials that will be mailed to them. Recruitment began in late 1995 and had continued for 8 months when this chapter was prepared. Thus, only subject characteristics and drinking-related history variables for subjects recruited during that period are presented in this chapter.

To be eligible for the project, respondents have to be of legal drinking age (19 in the province of Ontario). Respondents are first screened by telephone for a history of previous treatment or formal help for alcohol problems. Excluding people with a history of prior treatment is intended to ensure that more severely dependent alcohol abusers are excluded from this test of a brief intervention. On the other hand, we want to ensure that all subjects either have an identifiable alcohol problem or are consuming alcohol at hazardous levels. Therefore, only those respondents who report drinking on average more than 12 standard drinks per week or having consumed at least 5 standard drinks on at least 5 days in the past year are eligible for the project. One standard drink was defined as 12 oz of regular (5 percent alcohol) beer, 5 oz of 12 percent alcohol table wine, or 1.5 oz of spirits; each contains 0.6 oz of pure ethanol.

Respondents who meet the initial study criteria are sent a consent form and several questionnaires assessing their drinking and related behaviors. These questionnaires include a Timeline Followback calendar for the past 12 months (Sobell and Sobell 1992), the Alcohol Use Disorders Identification Test (AUDIT) assessing severity of dependence (Saunders et al. 1993), and a brief questionnaire that collects demographic and drinking history data. To assess respondents' motivation to change, the questionnaires include the following two questions: "At this moment, how important is it that you change your current drinking?" and "At this moment, how confident are you that you will change your current drinking?" Finally, respondents are asked to complete the Brief Situational Confidence Profile, a variation of the Situational Confidence Questionnaire (Annis and Graham 1988), in which respondents indicate their self-confidence to resist urges to drink heavily in eight potential high-risk situations.

The assessment materials are mailed to respondents with instructions that completing the materials will help them better evaluate their current drinking. Respondents are asked to sign the consent form and return it with the completed assessment materials in a preaddressed, stamped envelope. Upon returning the consent form and questionnaires,

subjects who meet the study criteria are randomly assigned to one of two groups: Fostering Self-Change (FSC) or Educational Materials Control (EMC). The FSC group receives a motivational intervention using advice feedback materials (Sobell et al. 1996). Based on their assessment responses, each respondent in the FSC group is sent a 6-page personalized advice feedback summary titled "Where Does Your Drinking Fit In?" (see Appendix A in Sobell et al. 1996). The materials consist of (1) a summary of their drinking as recorded in the Timeline Followback calendar, including an estimate of the amount of money they have spent on drinking and the amount of alcohol-derived calories they consumed over the past year; (2) charts relating their drinking level to national normative data and to the risk of various health consequences; (3) their reported consequences of drinking; (4) their self-confidence that they can avoid heavy drinking in various types of situations; and (5) a decisional balance exercise they are asked to complete to help them evaluate the pros and cons of changing or continuing their current drinking. Respondents in the EMC group do not receive the personalized feedback materials but instead are given two informational pamphlets that discuss the nature of alcohol abuse and provide general advice on how to deal with alcohol problems.

At the end of 1 year, respondents will be sent a second set of questionnaires to complete, from which they will again receive feedback. At that time respondents in the EMC group will also receive personalized feedback materials similar to what the FSC group received at the start of the study.

To address questions about the validity of alcohol abusers' self-reports, a random sample of 10 percent of the subjects in each group will be offered $100 for an in-person interview and provision of a collateral informant who will be asked independently to provide information about the respondent's drinking and related behavior in the past year. Using a portable breath tester, this subsample of respondents also will be breath tested before their interview.

A surprising aspect of the Fostering Self-Change study is that the average age of respondents has been older than is typical for treatment outcome studies. To date, the median and mean ages of the 1,017 total respondents who reported their age are 47.0 years and 48.0 years, respectively. In this chapter we examine data from the 291 respondents who were eligible for the study and completed and returned their assessment questionnaires. The median and mean ages for this group are 47.0 and 48.0 years, respectively. This group's range in ages provides an opportunity to explore differences in age-related variables.

Two types of data are available for analysis. First, we examine drinking and related variables with respect to respondents' current ages. The age categories used are ≤ 34, 35–50, and ≥ 51 years. Second, a subset of individuals with late-onset alcohol problems are compared with respondents whose problems had an earlier onset.

Table 1. Characteristics of All Respondents, Grouped by Age at Time of Study.

Variable	Age Group A:≤ 34[a]	B: 35–50[b]	C:≥51[c]	*p*	Source of Difference
Mean age (years)	30.5	42.8	60.3	n.a.	
% Male	61.3	59.7	68.1	< 0.048	n.d.[d]
Mean days drinking/week	4.1	4.9	5.6	< 0.001	C > B > A
Mean drinks/drinking day	7.3	6.3	5.4	< 0.001	A > B, C
Mean most drinks in a day, past year	14.3	11.6	9.5	< 0.001	A > B > C
Mean no. days ≥ 5 drinks, past year	148.8	176.8	175.2	0.176	

Note: n.a. = not applicable.
[a] n for age = 131; n's for other variables = 110–112.
[b] n for age = 436; n's for other variables = 380–385.
[c] n for age = 396; n's for other variables = 305–313.
[d] χ^2, source of difference not determinable.

Because the screening questions did not inquire about age of onset, differences between late- and early-onset individuals are only possible for respondents who returned their assessment materials.

Findings for All Respondents

Age differences in respondents' drinking characteristics are shown in table 1. Of the 810 respondents who provided information on their drinking, the oldest age group reported drinking significantly more frequently than the two younger groups. In terms of their single greatest drinking day ("most drinks in a day") in the past year, however, the older group drank significantly less than the other two groups, and the youngest group reported consuming significantly more drinks per drinking day than the other two groups. There also was a significant difference for gender, which appeared to indicate that the oldest group had more men than the younger groups.

Findings for Study Subjects

Table 2 presents results for the 291 respondents to date who have returned all of the questionnaires and are in the study. The analyses showed that the oldest group reported significantly more drinking days than the younger groups. There were no significant differences between groups in mean number of drinks consumed per drinking day. Several other age differences were also found. Although the oldest subjects reported having had a drinking problem for significantly longer than the younger groups, their problem appeared to be less severe as reflected by their scores on the AUDIT and the number of drinking-related consequences they reported. The youngest respondents reported significantly more hospitalizations and arrests than the other two age groups. Consistent with having less

Table 2. Characteristics of Study Participants, Grouped by Age at Time of Study.

Variable	Age Group (*n* per variable) A: ≤ 34 (40–41)	B: 35–50 (135–141)	C: ≥ 51 (105–109)	*p*	Source of Difference
Mean age (years)	31.5	43.4	60.3	n.a.	
% Male	65.9	58.9	72.5	n.s.	
Mean years problem drinking	7.2	10.4	14.2	< 0.001	C > B, A
% Married/partner	39.0	60.3	63.3	0.022	χ^2, n.d.[a]
% Employed full-time	73.2	71.6	39.4	< 0.001	χ^2, n.d.[a]
% White (ethnicity)	92.7	95.7	92.7	n.s.	
% University educated	15.0	37.0	25.7	0.015	χ^2, n.d.[a]
% Problem severity: Major	53.7	51.8	40.7	n.s.	
% Intend reduce/quit in next 2 weeks	70.7	72.3	66.1	n.s.	
% No problem in 12 months	53.7	46.8	55.0	n.s.	
Timeline % drinking days	66.5	74.7	86.8	< 0.001	C > A, B
Timeline mean drinks/drinking day	5.7	5.9	5.8	n.s.	
Mean AUDIT score	22.9	21.1	17.8	< 0.001	A, B > C
Mean no. alcohol-related consequences	4.2	4.2	2.7	< 0.001	A, B > C
Mean no. alcohol-related hospitalizations	1.0	0.1	0.0	0.023	A > B, C
Mean no. alcohol-related arrests	1.6	0.4	0.1	< 0.001	A > B, C
Mean SCQ score (self-efficacy)	45.1	45.2	56.2	< 0.001	C > A, B
Mean motivational level	7.0	7.2	6.7	0.035	B > C
Mean importance of changing	76.3	72.3	65.8	0.006	A, B > C

Note: n.a. = not applicable; n.s. = not significant; SCQ = Situational Confidence Questionnaire.
[a] χ^2, source of difference not determinable.

severe problems, subjects in the oldest group also had a significantly lower rating of the importance of changing than did subjects in the two younger groups.

Comparison of Late- Versus Early-Onset Problem Drinkers

An alternative to analyzing data by subjects' age at study entry is to compare subjects in terms of whether their drinking problem had a late versus early onset. Several studies of older problem drinkers have found differences among those individuals related to whether their problem drinking was a recent development or a long-standing problem (Atkinson et al. 1985; Atkinson et al. 1990; Brennan and Moos 1990, 1991; Atkinson 1994; Beresford 1995). *Late onset* has been defined in various ways. Although Atkinson (1994) prefers a definition of onset after 60 years of age, others have defined it as onset after the age of 50 (see, e.g., Brennan and Moos 1991). For the present analyses, the latter criterion will be used because it allows our findings to be compared with those of Brennan and Moos. Despite using a different age criterion, however,

Atkinson's findings have relevance to the present study. In his review of the literature he suggested that late-onset problem drinkers tend to have less severe problems and to have less impaired functioning than early-onset problem drinkers. He also speculated that late-onset elderly problem drinkers are likely to have more social and economic resources and are likely to seek out treatment at private facilities. Given these findings, a high representation of late-onset individuals in naturally recovered samples or among people attempting self-change might be expected.

Brennan and Moos (1995) found that late-onset problem drinkers (onset ≥ 51 years) drank less, had fewer drinking-related problems, were less depressed and anxious, and were more likely to receive support from their children and friends than early-onset problem drinkers. They also found a higher proportion of women among the late-onset group (39 percent vs. 22 percent of the early-onset group). Few of the problem drinkers they identified among a population of 55- to 65-year-olds reported having sought help in the past year.

To compare late-onset with early-onset problem drinkers in our study, it was necessary to restrict the analyses to individuals ≥ 51 years of age. This is because only those individuals had the opportunity to fulfill the late-onset criterion. Such a restriction resulted in 38 respondents in the late-onset group and 71 in the early-onset group. Among respondents age 51 or over, 34.9 percent had developed their drinking problem after age 51. Because this sample consists of people who answered media solicitations, the sample sizes may not reflect the relative frequency of late- and early-onset drinking problems in the general population. The current late-onset sample, however, indicates that late-onset drinking problems may affect a substantial number of persons.

Table 3 presents comparisons of late- and early-onset subjects across several variables. Gender was analyzed and the difference between the groups was not significant. This finding is inconsistent with that of Brennan and Moos, who found women to be more prevalent in their late-onset group. To further explore this finding, we examined the gender distribution among individuals 51 years of age or older in three treatment studies conducted at the Addiction Research Foundation. For the combined studies ($N = 68$), gender differences between early-onset and late-onset cases were not significant but the direction of the means was reversed: 20.5 percent of the early-onset group (8/39) were female versus 27.6 percent of the late-onset group (8/29).

The difference in age between the early-onset and late-onset groups was expected because of the age criteria for the analysis: late-onset respondents had to be at least 51 years old to be in that group and had to have had their drinking problem develop at age 51 or older; early-onset respondents only had to be at least age 51 when they entered the study, but their problem could have begun many years earlier. These criteria would tend to weight the late-onset group toward

Table 3. Characteristics of Participants Age ≥ 51 Years, by Age of Onset of Drinking Problem; Late Onset = ≥ 51 Years.

Variable	Early Onset (n = 71)	Late Onset (n = 38)	p
Mean age (years)	57.2	66.2	n.a.
% Male	67.6	81.6	n.s.
Mean years problem drinking	18.8	5.5	0.001
% Married/partner	60.6	68.4	n.s.
% Employed full-time	47.9	23.7	0.014
% White (ethnicity)	94.4	89.5	n.s.
% University educated	31.9	13.9	0.045
% Problem severity: Major	53.5	16.2	< 0.001
% Intend reduce/quit in next 2 weeks	74.6	50.0	0.010
% No problem in 12 months	43.7	76.3	0.001
Timeline % drinking days	84.7	90.7	n.s.
Timeline no. of drinks/drinking day	6.1	5.1	n.s.
Mean AUDIT score	19.1	15.4	0.003
Mean no. alcohol-related consequences	3.0	2.0	0.003
Mean no. alcohol-related hospitalizations	0.0	0.1	n.s.
Mean no. alcohol-related arrests	0.2	0.1	n.s.
Mean SCQ score (self-efficacy)	53.7	60.9	n.s.
Mean motivational level	7.1	6.0	< 0.001
Mean importance of changing	70.4	57.2	0.003

Note: n.a. = not applicable; n.s. = not significant; SCQ = Situational Confidence Questionnaire.

having a greater number of older individuals. Similarly, the difference in mean years of drinking problem likely reflects limitations imposed by our categorization criteria. Because the drinking problem of late-onset respondents could not have begun until age 51, it would be expected that they would report a shorter mean problem drinking history than the early-onset respondents.

The difference in employment status, with half as many late-onset as early-onset individuals being employed full-time, may reflect greater retirement rates in the late-onset group. However, it is also possible that lack of a full-time job poses a higher risk for the onset of problem drinking. The present data do not shed light on this issue.

Significantly fewer of the late-onset group than of the early-onset group had a university education, but the meaning of this finding is unclear. It may reflect a greater number of individuals going to college in recent years compared with a decade or two ago.

Other findings in table 3 suggest that late-onset respondents had a less severe drinking problem, as indicated by subjective ratings of problem severity, lower AUDIT scores, and fewer alcohol-related consequences. While reported drinking levels did not differ significantly between the

groups, the early-onset group had a higher level of motivation to resolve their problem and significantly more of them said they were planning to quit or reduce their drinking in the 2 weeks following their completing the study questionnaires. However, significantly more late-onset respondents believed they would be problem-free in 1 year. The lessened urgency to deal with their problem exhibited by late-onset respondents, as well as their greater confidence in resolving their problem, may be related to their problem being less severe.

CONCLUSIONS

Analyzing data by dividing a sample into cases with earlier and later onset is likely to yield a late onset-group with lower problem severity because the late-onset group will have a somewhat shorter problem history. This methodological issue is important when interpreting study results. From a clinical standpoint, however, what is important is that a substantial number of individuals develop drinking problems later in life, and that their problems often are less serious than those of individuals with longer drinking histories.

Atkinson and colleagues (1990) reported that late-onset problem drinkers were less likely to have sought help than early-onset, which might be explained by the differences in problem severity. Further, Brennan and Moos (1991) reported that relatively few older individuals with alcohol problems seek treatment. If such individuals find self-change to be an attractive option, this might explain the unexpectedly high average age of respondents in the Fostering Self-Change study. Another implication of less problem severity is that late-onset individuals may be good candidates for brief interventions.

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REFERENCES

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 3d ed., rev. Washington, DC: the Association, 1987.

Annis, H.M., and Graham, J.M. *Situational Confidence Questionnaire (SCQ 39): User's Guide.* Toronto: Addiction Research Foundation, 1988.

Atkinson, R.M. Late onset problem drinking in older adults. *Adv Alcohol* 9:321–326, 1994.

Atkinson, R.M.; Turner, J.A.; Kofoed, L.L.; and Tolson, R.L. Early versus late onset alcoholism in older persons: Preliminary findings. *Alcohol Clin Exp Res* 9:513–515, 1985.

Atkinson, R.; Tolson, R.L.; and Turner, J.A. Late versus early onset problem drinking in older men. *Alcohol Clin Exp Res* 14:574–579, 1990.

Beresford, T.P. Alcohol and aging: Looking ahead. In: Beresford, T., and Gomberg, E., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 327–336.

Brennan, P.L., and Moos, R.H. Life stressors, social resources, and late-life

problem drinking. *Psychol Aging* 5:491–501, 1990.

Brennan, P.L., and Moos, R.F. Functioning, life context, and help-seeking among late-onset problem drinkers: Comparison with nonproblem and early-onset problem drinkers. *Br J Addict* 86:1139–1150, 1991.

Brennan, P.L., and Moos, R.H. Life context, coping responses, and adaptive outcomes: A stress and coping perspective on late-life problem drinking. In: Beresford, T., and Gomberg, E., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 230–248.

Brennan, P.L., and Moos, R.H. Late-life problem drinking: Personal and environmental risk factors for 4-year functioning outcomes and treatment seeking. *J Subst Abuse* 8:167–180, 1996.

Saunders, J.B.; Aasland, O.G.; Babor, T.F.; De La Fuente, J.R.; and Grant, M. Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption—II. *Addict* 88:791–804, 1993.

Sobell, L.C., and Sobell, M.B. Timeline Follow-Back: A technique for assessing self-reported alcohol consumption. In: Litten, R.Z., and Allen, J., eds. *Measuring Alcohol Consumption: Psychosocial and Biological Methods.* Totowa, NJ: Humana Press, 1992. pp. 41–72.

Sobell, L.C.; Cunningham, J.A.; Sobell, M.B.; and Toneatto, T. A life span perspective on natural recovery (self-change) from alcohol problems. In: Baer, J.S.; Marlatt, G.A.; and McMahon, R.J., eds. *Addictive Behaviors Across the Lifespan: Prevention, Treatment, and Policy Issues.* Beverly Hills, CA: Sage, 1993. pp. 34–66.

Sobell, L.C.; Cunningham, J.A.; Sobell, M.B.; Agrawal, S.; Gavin, D.R.; Leo, G.I.; and Singh, K.N. Fostering self-change among problem drinkers: A proactive community intervention. *Addict Behav* 21:817–833, 1996.

Chapter 23

Treatment of Comorbidity in Older Adults With Alcohol Problems

Mark L. Willenbring, M.D.

Scientific inquiry typically requires isolation of certain characteristics from surrounding or coexisting phenomena. Isolation or categorization allows for a deeper understanding of the phenomenon in question. In medical research, for example, disorders are often isolated from one another in order to better understand their characteristics and response to treatment. Our institutions reflect these separations. Professions are organized around studying or treating different disorders or sets of disorders or dysfunctions. The same is true of professional departments, systems and institutions of care, and funding streams.

From a clinical standpoint, however, neither we nor our disorders come in such neat packages. Patients do not read textbooks before coming to physicians or other professional caregivers, and thus they present in clinical settings with all sorts of messy combinations of problems. When they have at least two or more major conditions, the term *comorbidity* is used to describe this coexistence of major categories of problems.

This chapter is an exploration of major types of comorbidity among older adults with alcohol problems. After briefly examining the nature of comorbidity and the challenges that comorbidity presents for the development of treatment models, I discuss comorbidity and treatment as they relate specifically to older adults.

MORBIDITY, COMORBIDITY, AND ALCOHOL PROBLEMS

The word *morbid* derives from the Latin word *morbus,* meaning disease.

M.L. Willenbring, M.D., is associate professor in the Department of Psychiatry, University of Minnesota, Minneapolis; and assistant director of the Psychiatry Service, VA Medical Center (116A), One Veterans Drive, Minneapolis, MN 55417.

Its synonyms are diseased, sick, abnormal, or pathological (*Webster's New Twentieth Century Dictionary, 2d ed.*). This is obviously a broad definition that might cover a considerable range of human problems. *Comorbidity* is the simultaneous presence of two or more morbid conditions. As we shall see, comorbidity is the rule, not the exception. Most people with significant dysfunctions have other coexisting or complicating problems, whether or not they seek professional care. The presence of multiple disorders seems to be most common among those with the most severe disorders (Kessler et al. 1994; Howard et al. 1996). The order of onset for different disorders is often not clear, since it is usually determined retrospectively. Different disorders may occur in sequence or simultaneously.

Etiology is even more problematic, since order of onset does not presume causality, or even association. Very little is known about the truly complex situations that routinely present themselves in clinical practice, since even studies of the treatment of comorbid conditions usually focus on specific well-defined types, such as coexisting mental and substance use disorders. Comorbidity is not confined to medical disorders. For example, homelessness, demoralization, despair, poverty, and social isolation are all important factors that must be addressed in treatment situations, but they are not medical diseases. Comorbidity, like the individual disorders that constitute it, is variable in type and severity, across both individuals and time.

Functional deficiencies of many types may co-occur with problematic alcohol use. Broadly, they may be divided into physical, psychiatric, substance use, social, and spiritual areas, but the number of potential combinations of disorders is large. The focus of most studies or descriptions has been on the most severe forms of comorbidity, and those that present in clinical situations. However, human problems vary considerably in severity. Variability is particularly important to consider from the standpoint of treatment planning, since relative severity may be an important determinant of where services are sought and delivered, and overall severity may determine whether services are sought or delivered at all. Therefore, it is important to consider how morbidity and comorbidity are defined and the implications these definitions have for what services are made available and how they are administered. Finally, comorbidity is considered a challenge not only because of its inherent complexity and potential severity, but also because of the characteristics of the service system, which is not configured in a way that facilitates treatment of complex problems (Ridgely et al. 1990; Willenbring 1994; Osher 1996).

COMORBIDITY AND AGING

Before examining comorbidity specifically as it relates to aging, it is important to consider whether old age itself should be considered a comorbid condition when alcohol problems are present. The

question arises because of the increasing emphasis on elder-specific treatment for alcohol problems in recent years. Some of the modifications in elder-specific treatment relate to the increased probability of clinically significant comorbidity in the elderly (Atkinson 1995; Finlayson 1995; see also chapter 24). For example, there is an increased emphasis on identifying and addressing the various problems that occur with greater frequency in elderly problem drinkers, including a wide variety of medical, psychiatric, and social problems. However, other elder-specific treatment modifications seem to address other differences in age cohorts, such as comfort discussing personal problems in a group context, that are not dysfunctional but may influence treatment receptivity (Atkinson 1995). Old age, then, appears be accompanied by an increased risk of comorbidity, but it is not itself a comorbid condition.

COEXISTING SUBSTANCE USE

Use, abuse, and dependence of both alcohol and drugs are present in a smaller proportion of older adults when compared with younger cohorts (Bucholz et al. 1995). However, a cohort effect exists, where younger cohorts drink and use drugs at higher rates than older cohorts throughout the lifespan (Closser and Kosten 1992; Bucholz et al. 1995). Thus, although alcohol and drug abuse and dependence are less common in older groups now, the prevalence can be expected to rise as younger cohorts age.

Nicotine dependence is the most common coexisting substance use disorder, with large proportions of heavy drinkers also smoking heavily (Carmody et al. 1985; Bien and Burge 1990). Comorbid nicotine dependence contributes substantially to the excess mortality reported in alcoholics (Hurt et al. 1996). It is unclear what role heavy drinking and smoking may play in relapse, but there is some evidence suggesting that relapse to one may increase relapse to the other, or that continued smoking may result in more drinking relapse (Zimmerman et al. 1990; Gulliver et al. 1995). How and when to treat nicotine dependence that is comorbid with alcohol dependence is not clear at this time (Bobo and Gilchrist 1983; Joseph et al. 1993; Knapp et al. 1993; Hurt et al. 1995). Treatment of comorbid nicotine dependence is an area urgently requiring further research.

Prescription drug abuse and dependence are more likely to occur in older adults than in younger persons (Jinks and Raschko 1990; Dufour et al. 1992; Finlayson 1994), presumably because older adults are more likely to have drugs prescribed for them or to have conditions such as chronic pain or insomnia that place them at risk for developing drug dependence. However, serious problems exist concerning the definition of abuse or dependence of prescription drugs, especially when there is a clear medical indication for the drug. There is no consensus concerning the appropriate use of opioid analgesics, anxiolytics, or hypnotics for chronic conditions. Although tolerance and physical dependence are common, development of an abuse or depen-

dence syndrome is unusual, except in persons with current or past alcohol or drug problems (Marks 1978; Baldessarini 1996). The appropriate use of potentially addictive drugs in persons with a past history of drug or alcohol abuse is even more problematic, with little to guide the clinician. However, it would be inappropriate to completely preclude their use given the lack of alternatives, especially for chronic pain.

Illicit drug users appear to be continuing their use patterns as they age (Robins and Regier 1991; Kessler et al. 1994). A generation of opiate addicts receiving opioid agonist substitution therapy are now reaching older age and are developing the various problems that accompany aging. Opiate addicts frequently have coexisting alcohol problems, cocaine dependence, chronic hepatitis C, or AIDS. Use and abuse of marijuana, cocaine, stimulants, and hallucinogens, while uncommon in elders now, can be expected to rise in prevalence as current users age.

MEDICAL, PSYCHIATRIC, AND SOCIAL COMORBIDITIES

The rates of many common disabling medical problems, such as arthritis, cancer, cardiovascular disease, hypertension and stroke, and diabetes, rise with age. However, certain disorders or problems appear on an anecdotal basis to be more commonly associated with heavy drinking. Chronic pain is a particularly difficult problem in heavy drinkers, because they experience alcohol as giving them more relief than prescribed medications. It is likely that most people with coexisting chronic pain and heavy drinking were heavy drinkers prior to the onset of the pain (i.e., primary), but the pain may exacerbate alcohol abuse or dependence and will make recovery more difficult. Insomnia, a common problem in the aging, poses some similar difficulties, although it may be somewhat more likely that heavy drinking occurs secondary to insomnia. There is a dearth of empirical data concerning these problems. More research is needed on the relationship between alcohol use and chronic pain or insomnia, and treatment of alcohol problems in people with these comorbid conditions.

Heavy drinking increases the risk of numerous severe medical problems, most commonly liver cirrhosis, gastrointestinal bleeding, chronic pancreatitis (with severe chronic pain), cardiomyopathy and heart failure, cardiac arrhythmias, hypertension, myopathy, and malnutrition (Gambert and Katsoyannis 1995). Combined nicotine and alcohol use results in synergistic increases in cancers, especially of the gastrointestinal tract and lung (Schmidt and Popham 1981; McCoy and Napier 1986). Heavy drinkers are at greater risk as well for falling and trauma, including fractures and head trauma. Many medically ill elderly heavy drinkers will thus present to primary and specialty medical settings, not to specialty addiction services.

Cognitive disorders, such as delirium, amnestic disorders, and dementia, are common presentations in

heavy drinkers in both medical and psychiatric settings. As for other psychiatric disorders, the National Comorbidity Survey (NCS) revealed that the only psychiatric disorder associated with alcohol abuse was social phobia, but alcohol dependence and drug dependence were associated with increases in affective disorders (especially mania), all anxiety disorders, and antisocial personality or adult antisocial behavior (Kessler et al. 1994). Although this survey only included subjects up to age 54, its findings are generally consistent with other studies such as the National Institute of Mental Health Epidemiologic Catchment Area (ECA) study (Robins and Regier 1991). An important finding from the NCS was that comorbidity was concentrated: more than half of all lifetime disorders occurred in roughly one-sixth of the population that had three or more disorders (Kessler et al. 1994). Both the ECA and the NCS found that a minority of those with mental and substance use disorders sought and received professional help. This was true even in those with multiple, disabling disorders (Kessler et al. 1994; Howard et al. 1996). The NCS also found that persons with psychiatric and substance use disorder comorbidity who did obtain service did so in a variety of settings, including medical, specialty mental health, and specialty addiction treatment. These data must be considered when developing intervention strategies for comorbid conditions.

One type of comorbidity that has received considerable attention is the coexistence of severe and persistent mental disorders, usually psychotic disorders or severe bipolar affective disorder, and substance use disorders. Although definitive studies have not been done, the evidence to date suggests that integrating community support programs and substance abuse treatment that focuses on improving motivation to change is a promising strategy (Drake et al. 1996). However, most studies have been done among younger individuals, and it is not clear how applicable their findings are to older adults. Anecdotal evidence suggests that as such severely impaired persons age, they are more likely to reside in institutional settings, so community support programs would not be useful to them.

Not only individuals but families are involved, and as people age, family dynamics assume greater importance. People with alcohol problems frequently have severe disruptions of family life. Often, this leads to estrangement through separation, divorce, or lack of contact with children. Family members who are dependent upon the affected person may find it difficult to maintain normal intimacy, falling instead into a defensive pattern of enmeshment. This may particularly occur in older women who are financially dependent upon an alcoholic spouse. Many interventions that are appropriate in younger groups, such as "tough love" or advising the spouse to leave, may not be appropriate in aging couples. Adult offspring may themselves abuse alcohol, and they can be important supporters of continuing pathological drinking in aging parents. Abusive

behavior, including violence, may occur, with drinkers being either perpetrators or victims of abuse.

In treatment, these family dynamics must be addressed, because they may determine the success of efforts to achieve abstinence. As heavy drinkers become more infirm, family members are often put in the difficult position of assuming greater responsibility for someone with whom they may have had a stable if dysfunctional adjustment that now must be changed. Intense feelings from past experiences may be engendered in all participants.

In addition to defined psychiatric disorders, there are several social disabilities or problems that must be considered as potential comorbid conditions. Chief among these is social isolation, which is not only a risk factor for development or worsening of heavy drinking, but may significantly impede recovery. It is more difficult for older persons to find social outlets, and heavy drinkers often have poor social skills. Bereavement, grief, and unresolved losses are common challenges faced by older persons. These common problems are exacerbated in older heavy drinkers, who may have many painful consequences of heavy drinking to face. Examples include ruined marriages, estranged children, lost employment opportunities, poor health, and premature retirement and disability.

Elders everywhere face barriers in getting places, with transportation frequently cited as the most important overall concern. These concerns are magnified in heavy drinkers, many of whom have lost licenses due to intoxicated driving. Affordable housing that does not promote social isolation yet is affordable and acceptable to the client may be difficult to find, but it is crucial to recovery. Homelessness is highly associated with problematic substance use. Although homelessness has assumed much broader proportions recently than in the past, older single heavy-drinking males—the classic skid row alcoholics—continue to represent a significant proportion of homeless persons (Cohen and Sokolovsky 1989). Homelessness is a problem that is not confined to urban areas (First et al. 1994). Income maintenance for poor persons has become more difficult to obtain, with many localities eliminating welfare eligibility for any males or for females without children. Anecdotally, the elimination of federal Supplemental Security Income (SSI) benefits for substance use disorders is already driving many people out of housing and into the streets and shelters.

For those individuals who are more severely disabled by physical or mental disorders, long-term care is becoming increasingly difficult to obtain. State mental hospitals and veterans hospitals are no longer available for long-term care of mentally ill persons in most states. Many long-term care facilities will not accept people with active substance use disorders. There is a lack of information and research into the proper community care for such persons.

There is a group of age-appropriate tasks, often considered as developmental or spiritual in nature, that should be

taken into account when providing services for older heavy drinkers. Reviewing one's life, with its successes and failures, and developing some sense of meaning, place, order, or context for it, is a developmental task for all older persons (Colarusso and Nemiroff 1981). This may be more problematic for heavy drinkers because of the failures and losses engendered by alcohol abuse. Anxiety about facing these issues may make recovery more difficult, and the depth of guilt and shame encountered during the process may provoke relapse. At the same time, there is an even greater need to achieve a sense of forgiveness and peace, which are commonly considered spiritual concerns (Vogel 1995). Although spiritual problems are not generally thought of as a comorbidity, failure to address them may impede recovery from drinking problems. No matter what their personal beliefs, professionals need to address spiritual matters with their clients in terms their clients understand. Spirituality is exceedingly difficult to study, since by its very nature it is not understandable in a logical way and is difficult or impossible to describe. Nevertheless, research concerning spirituality is much needed.

BARRIERS TO ADDRESSING COMORBIDITY

With the daunting variety of potential problems that may be comorbid with alcohol use disorders, how can the treatment system respond effectively? In this section, I examine barriers to addressing comorbidity in the current system; in the next section I offer some suggestions for how the system might respond more effectively.

Fragmentation of care into specific services that are not effectively coordinated is the major problem in addressing any complex combination of problems. Many factors contribute to fragmented care. Narrow treatment ideologies frequently fail to consider or adjust for coexisting problems. Many programs have historically assumed, based more upon folklore than science, that addressing the alcohol or drug problems would take care of comorbid problems. Treatment program staff, rather than adapting treatment models, have frequently excluded people who do not fit their model or who cannot participate in it due to comorbidities.

Professional training and development, as well as the administrative structure of service organizations, contribute to disorder-specific, "single-track" treatment systems. Most professionals are trained to provide their specific function or service, without taking responsibility for the overall outcome. This results in missed problems, redundant care, inappropriate care, poor outcome, and dissatisfaction with treatment.

The development of single-track treatment was reinforced by the development of addiction treatment outside of the mainstream of other professional care, which led to separate funding streams and administrative systems. Financial and administrative barriers to the care of mentally ill substance users have been addressed elsewhere in greater detail (Ridgely et al. 1990; Osher 1996). However, it is important

to note that these problems remain largely unaddressed.

Alcohol and drug treatment programs have responded to the problems of single-track systems by developing new programs for people with specific types of comorbidity, or by adding new services to existing programs. Elder-specific services have become more common in alcohol and drug treatment. Typically, elder-specific treatment involves a combination of attention to cohort differences and attention to the comorbidities that occur more frequently in elders. Examples of cohort differences that are often considered in elder-specific care include the lower prevalence of illicit drug abuse in current elders, and values concerning sharing personal faults in a group setting. Modifications consistent with cohort difference are elder-specific groups and less emphasis on confrontation, especially within groups, along with more individual counseling. Attention to comorbid problems includes a closer liaison with medical providers and more attention to problems such as cognitive decline, hearing or vision loss, physical handicaps, and transportation (Atkinson 1984).

Although elder-specific tracks within existing alcohol and drug programs are a positive step (albeit one with limited empirical support at this time), they are at best a partial and limited solution to the larger problem of the treatment of complex problems. In order to participate in such programs, clients need to be able to participate in a demanding schedule of individual and group counseling, education, and related activities. Many individuals with serious comorbidities continue to be excluded from such programs. Furthermore, such programs only treat a limited spectrum of alcohol problems, neglecting both milder problems that do not require intensive interventions and more severe and chronic problems that require long-term management. Finally, no matter how good an alcohol program is, it will not help those who do not seek care there. The majority of persons with substance use disorders never receive any professional care, even when comorbid conditions are present (Kessler et al. 1994). If we wish to reach a greater proportion of individuals with alcohol use disorders, we must take a broader approach than simply altering alcohol-specific treatment. This is especially true when serious comorbidity complicates the alcohol use disorder, since comorbidity affects how and where an individual may present for help.

DEVELOPING INTERVENTION STRATEGIES FOR COMORBIDITY

The first step in designing intervention strategies that will more effectively address comorbidity is to define target populations more precisely. When two or more disorders are present, especially when they are severe, this is not an easy task. Since most systems are designed to treat one primary type of disorder, the first question that often arises is, What is the primary problem? In this case, pri-

mary problem means the most important or prominent problem, or perhaps the one needing intervention most urgently. In many ways, it makes sense to think of triaging clients toward the system that deals best with the problem that most clearly defines the situation. This works well especially when one problem is clearly more severe than the comorbid problems. For example, someone with severe alcohol dependence and mild major depression may be treated in a specialty alcohol treatment program, while a person with a severe myocardial infarction and mild alcohol dependence should be treated in a primary or specialty medical setting.

However, where there is not clarity about which problem is primary, service providers may disagree about which problem should be addressed first. For example, in a person who has chronic nonterminal pain that requires opiates, and who also has alcohol dependence and depression, it is not unusual to have the pain program reject the client because of active drinking, the alcohol program to reject the client because of the continuing need for opiates, and the mental health provider to reject the client because of both. Where do such people belong? Who will take care of them? Who decides and coordinates treatments from different systems?

Mental health and alcohol treatment providers are not alone in having this mindset; it is as likely to occur with medical and social service providers. Perhaps the only providers that cannot reject such patients are primary care medical providers, who may often feel stuck with an impossible situation for which they are getting little help from their specialty medical colleagues or from mental or addiction treatment providers. Chronic pancreatitis presents a good example. Many such patients continue to drink, at least sporadically and sometimes frequently. Conventional alcohol treatment is often refused or ineffective. Opiates may be required for pain control, either intermittently or continuously. However, there are no established treatment guidelines for the management of such patients. Primary care providers may be caught between alcohol treatment providers who tell them they are enabling if they continue to see the patient, a patient who is angry and in distress, a confused family, and gastroenterologists who insist that opiates never be prescribed for pancreatitis in patients who drink.

Location of presentation may be an important factor in developing intervention strategies. The factors that contribute to the decision to seek care, and where to seek it, are not well delineated, especially in complex conditions. It is known that even when there are multiple disorders, a variety of treatment sectors may be chosen. For example, in the NCS, even people with severe multiple psychiatric disorders presented in medical, mental health, and specialty alcohol treatment sectors (Kessler et al. 1994). Of course, most people, even those with multiple severe disorders, did not seek professional care at all, suggesting that other sectors, such as churches, social service agencies, and community outreach agencies, should

be considered for case finding and service delivery. More research is needed on the factors contributing to consumers' decisions in this regard, and how they reflect other attitudes and beliefs that might influence receptivity to different types of interventions.

Location of service delivery may also be an important factor in developing intervention strategies. Traditional alcohol treatment has made little use of public health nurses, long-term care providers, social service agency providers, or medical care providers as primary providers of alcohol interventions. However, it makes sense at times to deliver care in one of these service sectors, either because of client preference or because a particular provider is best able to coordinate or deliver care.

Although models for brief interventions in mild alcohol use disorders are now widely available (if not widely practiced) (Bien et al. 1995), there are no extant models for the primary care management of more severe substance use disorders, especially those accompanied by severe medical morbidity. There is evidence that persons presenting for care in medical settings differ substantially from those who present to specialty alcohol treatment and may require different types of interventions (Willenbring et al. 1994). It is likely that those presenting in other sectors, such as specialty mental health and social service agencies, also differ substantially from those presenting in medical settings and thus require different types of interventions.

It is important for alcohol treatment providers and planners to consider focusing more on providing interventions for the entire population of persons covered by a particular health plan, or in a particular catchment area, rather than treating only those who present themselves to specialty alcohol treatment. In order to do this, consideration must be given to the prevalence and incidence of alcohol use disorders among the at-risk population, the variation in severity and pattern of disorders and comorbid conditions, and the characteristics of the continuum of care available.

Developing intervention strategies using this perspective is quite different from developing a specialty alcohol treatment program, even one for a specific combination of comorbidity such as coexisting addictive and mental disorders. A population perspective requires creative use of many types of interventions, especially long-term, low-intensity, or intermittent ones, delivered by a wide variety of providers in a wide variety of locations. Whereas specialty alcohol treatment is typically oriented toward individuals who are ready to change, interventions in many other settings need to be focused on case finding, raising awareness, and improving motivation for positive change. Incorporating consumer preferences is essential, since these preferences will generally determine whether, where, and when they present for care and what services they will make use of. With this in mind, let us examine where interventions could take place, and what kind of problems could best be managed in different sectors of care. The sectors considered here are medical care, specialty mental

health treatment, specialty alcohol and drug treatment, long-term care, and community care.

Medical Care

Target populations most likely to present in medical care settings are (1) those on opposite ends of the spectrum of severity of alcohol problems and (2) drinkers who happen to have more severe medical disorders, whether related to drinking or not. In addition, primary care settings are appropriate for certain types of interventions, such as management of acute toxic and withdrawal states. Some patient populations, such as those with oropharyngeal tumors, may present to specialty care settings and should receive most of their care there, including alcohol interventions. Specialty care settings should not be ignored in targeting and developing such interventions.

One challenge for developing interventions in primary care settings is the wide variety of problems encountered, the need for different interventions for these different types of problems, and the paucity of models to treat most of them. Most research and teaching in this regard have focused on screening and identification and brief interventions (Bien et al. 1995). Brief interventions are generally oriented toward either seeking reduction in use among nondependent heavy drinkers or identifying and referring persons with alcohol abuse or dependence to specialty alcohol treatment. Many heavy drinkers encountered in medical settings, however, may not respond to either approach. The biggest problem has been the lack of models for alcohol-dependent persons who either refuse referral to specialty programs or do not respond to specialty treatment. Research done in our center has suggested that patients presenting in medical settings tend to be older and have more family and health concerns than those presenting to specialty alcohol treatment. In contrast, those presenting to specialty treatment have more social problems and antisocial personality characteristics (Willenbring et al. 1994). We have also shown that a treatment model using principles of management common in the treatment of chronic medical disorders and delivered in a primary care setting is well accepted by patients and other staff and may result in reduced 2-year mortality in severely medically ill alcoholics (Willenbring et al. 1995).

When developing an intervention, it is important to keep consumer beliefs and preferences in mind. For example, when a nurse-based brief intervention program that would operate adjunctively to a general medical clinic was being developed, we surveyed physicians about their major concerns and how we could address them. We found, much to our surprise, that physicians were much more concerned about tobacco use than about alcohol use in their patients. Until we received that result, we were not planning on including smoking cessation in our program. However, we did include it, and it has turned out to be the most frequent source of referrals. Furthermore, it has turned out that dealing with heavy smokers

has been the most effective way to identify heavy drinkers. Physicians appear to be more comfortable with identifying and referring smokers and are motivated to do so; thus, given the high correlation between smoking and heavy drinking, we have been able to identify and address the heavy drinking as well.

Specialty Mental Health and Addiction Treatment

Persons with moderate to severe mental disorders, with or without comorbid substance use disorders, require management in specialty mental health settings. General mental health settings should be adequate to provide brief or even sustained psychotherapeutic interventions for mild to moderate substance use disorders occurring in conjunction with mental disorders. However, staff must become proficient in integrating alcohol treatment interventions into mental health care. Research is needed to examine the effectiveness of brief interventions for mild substance problems in mental health settings. Because of its roots in psychotherapy, motivational interviewing techniques may be more easily adapted by mental health professionals than other models of alcohol treatment (Miller and Rollnick 1991).

Research on the effectiveness of treatment for coexisting mental and addictive disorders has focused primarily on persons with severe and persistent mental disorders, especially psychosis. Although there is limited empirical support at this time, most experts advocate integrating treatments for these two conditions (Drake et al. 1996). Such "dual diagnosis" programs may be located either in specialty mental health or specialty alcohol and drug treatment settings. Where it should be located depends on local administrative, funding, and personnel factors.

Elderly persons with moderate to severe alcohol and drug problems, and who have no or only mild comorbid conditions, will optimally be treated in specialty alcohol treatment programs. This general principle may need to be modified depending upon the motivation of the patient and the characteristics of the program. Most treatment programs are oriented to treat individuals who are in an action stage of change (Prochaska and DiClemente 1984). That is, the programs work best in helping someone make a change once they have already decided they need to. More research is needed on how best to help those who are in a more conflicted state regarding change. Specialty programs may wish to incorporate motivational enhancement, or motivational enhancement strategies may be used in other settings, such as primary medical care or mental health care, to increase the rate of successful referral to specialty alcohol treatment.

Consumer preference should be considered in deciding where and how interventions should be implemented. For example, many consumers with serious mental illnesses may prefer to receive treatment within the setting used to treat the primary disorder. Furthermore, consideration must be given to how specialty alcohol treatment programs are

designed and whether they are responsive to the needs of persons with serious mental illness.

Long-Term Care

It is important to consider how to manage drinking and alcohol use disorders in settings that are not usually considered as locations for alcohol treatment. For the elderly, long-term care settings are one particularly important example. A history of problem drinking has been found to be common in some nursing home settings (Joseph et al. 1995*a*, 1995*b*). It is well known, though not well documented, that chronic heavy drinking often leads to functional deficits that may result in the need for long-term care. In fact, severe functional deficits constitute the comorbid condition that, along with heavy drinking, requires long-term care. Obviously, the range of potential deficits is large, and includes moderate to severe dementia and amnestic disorders, chronic obstructive pulmonary disease, liver failure, and other chronic, disabling conditions.

There are many questions concerning the management of drinking among residents of long-term care facilities. Should residents be allowed to drink? Should they be allowed to keep or consume alcohol on site? Should the facility serve alcoholic beverages in their dining or recreational facilities? There is evidence that use of alcohol in social situations is beneficial overall for nursing home residents (Mishara et al. 1975). Should policies be different in different types of facilities, such as board and lodging, board and care (intermediate care), and skilled nursing facilities? Should policies be different for residents with different types of medical, mental, functional, or substance problems?

Another policy question is how dysfunctional drinking by residents of long-term care should be managed. Many facilities probably manage this primarily by excluding anyone with a recent history of problem drinking. Exclusion is likely to diminish but not eliminate the occurrence of problem use by residents. At least some facilities specialize in accepting alcohol-dependent persons, but it is not known what their drinking policies are or how effective they are. There are also board and lodging facilities (often called extended care facilities or halfway houses) that are considered part of the spectrum of care within specialty alcohol treatment. Drinking policies in these facilities range from requiring abstinence and immediately discharging those who drink, to allowing some drinking off site, to allowing drinking in general, as long as it is not too disruptive. It is unlikely that one policy will work for all or even most facilities. Characteristics of the setting, the population served, and the mission of the organization must be taken in account. Although some research has been done on management of alcoholics in nursing homes (Linn et al. 1972; Chien et al. 1973), there are no published studies that suggest which policies might be most effective.

In addition to policy questions concerning drinking behavior, research is needed regarding the utility of program interventions (counseling, skills training, support groups) in

long-term care settings. Many settings now include some programming, but there are no data available evaluating its effectiveness.

Consumer preference should be considered in determining policies. In one study of older homeless public inebriates, we found a preference by consumers for facilities that were close to other services, allowed some drinking off site, and served good food (Willenbring unpublished data). In another study of consumer preference regarding drinking policy within a nursing home setting that had a significant proportion of chronic inebriates, most residents of the facility endorsed a policy of allowing drinking on the grounds, but not in the facility, because they felt that it was safer for drinking residents to remain on the grounds than to be forced to go off site to drink. In addition, residents wanted to be sure that any behavior that would threaten others or that would seriously disrupt the milieu would not be tolerated. These opinions were endorsed by a majority of both drinking and nondrinking residents (Willenbring et al. unpublished data).

Community Care

The community is another setting where many heavy drinkers receive a considerable amount of care that is not usually considered alcohol treatment. However, functional deficits and moderate to severe medical and psychiatric comorbidity that is not so severe as to require long-term institutional care often require community care. This is increasingly so given the pressure to reduce costs.

Models for the treatment of alcohol problems in community settings have not even been articulated, let alone tested for effectiveness. Public health nurses, home health aides, and social services agency staff members frequently encounter heavy drinking and its sequelae in the home, and undoubtedly respond as best they can. Family members are also called upon to provide care and support for ailing heavy drinkers, and they may be an effective resource for informal interventions. Family members require support and guidance on how to respond to an aging parent or sibling with increasingly severe functional deficits. Admonitions to "let go" do not work as well when the affected family member is becomingly increasingly incompetent and unable to care for himself or herself.

Management of assets is another area needing investigation. As functional deficits worsen, it often becomes necessary for someone else to manage assets. There are many questions as to how to do this most effectively. For example, is it best to focus only on provision of basic necessities and to allow the drinker to decide how to use the remaining funds, even if that is to purchase alcoholic beverages? Should such discretionary funds be restricted, doled out in small amounts, or totally eliminated? How does asset management fit in with the relationship between asset manager and asset owner? Is the asset manager acting simply as an extension of the asset owner, or should paternalistic, and therefore coercive, approaches be used? For example, Spicer and col-

leagues (1994) found that overly coercive approaches resulted in a loss of relationship between asset managers (representative payees) and homeless inebriate clients. Representative payees were most effective when there was some correspondence of goals between case manager and client.

Another area needing further investigation is in specific public policy measures that may differently affect older heavy drinkers. Price and availability of alcoholic beverages may affect older drinkers and those with severe comorbidity more or less than other members of society. For example, with the lower income of elderly persons, price increases may result in greater reductions of use. The same may be true of grocery store as opposed to liquor store sales. Fletcher and colleagues (1996) found that home delivery of alcohol was used primarily by heavy drinkers in urban areas, and that older, medically ill alcoholics did not differ from younger alcoholics seeking treatment in their use of home delivery services. Home delivery may be a particularly useful area to explore, since many older alcoholics are unable to drive, and may even be so sick as to be unable to walk to a liquor store, yet can receive delivery in their homes. Also, since the interaction between vendor and customer is less likely to be publicly witnessed, vendors may have less compunction about selling alcohol to someone obviously ill or intoxicated (Fletcher et al. 1996).

ETHICAL CONCERNS

One final area needing further inquiry is that of the morality or ethics of certain aspects of policy and treatment in the care of the elderly, especially those with severe comorbid conditions. Competence is frequently, if not usually, compromised, especially during times of intoxication, withdrawal, and acute medical and psychiatric conditions. However, it is not clear that this is adequately considered when seeking informed consent for treatment interventions. It often appears that someone is considered competent as long as they go along with recommendations by professional staff and family, and the issue of incompetence is only raised when the individual refuses. Elderly persons with compromised cognitive and physical function are vulnerable to coercion and abuse, and this problem may be markedly increased when heavy drinking is part of the picture. Careful ethical analysis and research into this problem are sorely needed.

SUMMARY

Heavy-drinking elderly persons with moderate to severe comorbid functional, psychiatric, and medical comorbidities constitute a complex and difficult group to help. Conventional treatment programs are usually not well equipped to handle more than mild comorbidity. Treatment models themselves tend to be time limited and focused on complete rehabilitation, and thus do not address the complexity and chronicity of severe comorbidity. A broader concept of "treatment" may be helpful in approaching persons with multiple complex problems. Research is needed in all aspects of institutional, programmatic, community, public

health, informal (e.g., family), and ethical aspects of care for these populations.

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REFERENCES

Atkinson, R.M. Substance use and abuse in late life. In: Atkinson, R.M., ed. *Alcohol and Drug Abuse in Old Age.* Washington, DC: American Psychiatric Press, 1984.

Atkinson, R.M. Treatment programs for aging alcoholics. In: Beresford, T., and Gomberg, E., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 186–210.

Baldessarini, R.J. Drugs and the treatment of psychiatric disorders: Psychosis and anxiety. In: Hardman, J.G.; Limbird, L.E.; Molinoff, P.M.; and Ruddon, R.W., eds. *The Pharmacological Basis of Therapeutics.* New York: McGraw-Hill, 1996. pp. 399–430.

Bien, T.H., and Burge, R. Smoking and drinking: A review of the literature. *Int J Addict* 25:1429–1454, 1990.

Bien, T.H.; Miller, W.R.; and Tonigan, J.S. Brief interventions for alcohol problems: A review. *Addiction* 90:1118–1121, 1995.

Bobo, J.K., and Gilchrist, L.D. Urging the alcoholic client to quit smoking cigarettes. *Addict Behav* 8:297–305, 1983.

Bucholz, K.K.; Sheline, Y.I.; and Helzer, J.E. The epidemiology of alcohol use, problems, and dependence in elders: A review. In: Beresford, T., and Gomberg, E., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 19–41.

Carmody, T.P.; Brischetto, C.S.; Matarazzo, J.D.; O'Donnell, R.P.; and Connor, W.E. Co-occurrent use of cigarettes, alcohol, and coffee in healthy, community-living men and women. *Health Psychol* 4:323–335, 1985.

Chien, C.-P.; Stotsky, B.A.; and Cole, J.O. Psychiatric treatment for nursing home patients: Drug, alcohol and milieu. *Am J Psychiatry* 130:543–548, 1973.

Closser, M. H., and Kosten, T.R. Alcohol and cocaine abuse. A comparison of epidemiology and clinical characteristics. *Recent Dev Alcohol* 10:115–128, 1992.

Cohen, C.I., and Sokolovsky, J. *Old Men of the Bowery: Strategies for Survival Among the Homeless.* New York: Guilford Press, 1989.

Colarusso, C.A., and Nemiroff, R.A. *Adult Development.* New York: Plenum Press, 1981.

Drake, R.E.; Mueser, K.T.; Clark, R.E.; and Wallach, M.A. The course, treatment, and outcome of substance disorder in persons with severe mental illness. *Am J Orthopsychiatry* 66:42–51, 1996.

Dufour, M.C.; Archer, L.; and Gordis, E. Alcohol and the elderly. *Clin Geriatr Med* 8:127–141, 1992.

Finlayson, R.E. Prescription drug dependence in the elderly population: Demographic and clinical features of 100 inpatients. *Mayo Clin Proc* 69:1137–1145, 1994.

Finlayson, R.E. Comorbidity in elderly alcoholics. In: Beresford, T.P., and Gomberg, E., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 56–69.

First, R.J.; Rife, J.C.; and Toomey, B.G. Homelessness in rural areas: Causes, patterns, and trends. *Soc Work* 39:97–108, 1994.

Fletcher, L.A.; Nugent, S.; Ahern, S.; and Willenbring, M.L. The use of alcohol home delivery services by male problem drinkers: A preliminary report. *J Subst Abuse* 8:251–261, 1996.

Gambert, S.R., and Katsoyannis, K.K. Alcohol-related medical disorders of older heavy drinkers. In: Beresford, T., and Gomberg, E., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 70–81.

Gulliver, S.B.; Rohsenow, D.J.; Colby, S.M.; Dey, A.N.; Abrams, D.B.; Niaura, R.S.; and Monti, P.M. Interrelationship of smoking and alcohol dependence, use and urges to use. *J Stud Alcohol* 56:202–206, 1995.

Howard, K.I.; Cornille, T.A.; Lyons, J.S.; Vessey, J.T.; Lueger, R.J.; and Saunders, S.M. Patterns of mental health service utilization. *Arch Gen Psychiatry* 53:696–703, 1996.

Hurt, R.D.; Croghan, I.T.; Offord, K.P.; Eberman, K.M.; and Morse, R.M. Attitudes toward nicotine dependence among chemical dependency unit staff—before and after a smoking cessation trial. *J Subst Abuse Treat* 12:247–252, 1995.

Hurt, R.D.; Offord, K.P.; Croghan, I.T.; Gomez-Dahl, L.; Kottke, T.E.; Morse, R.M.; and Melton, L.J. Mortality following inpatient addictions treatment. *JAMA* 275:1097–1103, 1996.

Jinks, M.J., and Raschko, R.R. A profile of alcohol and prescription drug abuse in a high-risk community-based elderly population. *DICP* 24:971–975, 1990.

Joseph, A.M.; Nichol, K.L.; and Anderson, H. Effect of treatment for nicotine dependence on alcohol and drug treatment outcomes. *Addict Behav* 18:635–644, 1993.

Joseph, C.L.; Atkinson, R.M.; and Ganzini, L. Problem drinking among residents of a VA nursing home. *Int J Geriatr Psychiatry* 10:243–248, 1995*a*.

Joseph, C.L.; Ganzini, L.; and Atkinson, R.M. Screening for alcohol use disorders in the nursing home. *J Am Geriatr Soc* 43:1–6, 1995.

Kessler, R.C.; McGonagle, K.A.; Shao, S.; Nelson, C.B.; Hughes, M.; Eshleman, S.; Wittchen, H.-U.; and Kendler, K.S. Lifetime and 12-month prevalence of DMS-III-R psychiatric disorders in the United States: Results from the the National Comorbidity Survey. *Arch Gen Psychiatry* 51:8–19, 1994.

Knapp, J.M.; Rosheim, C.L.; Meister, E.A.; and Kottke, T.E. Managing tobacco dependence in chemical dependency treatment facilities: A survey of current attitudes and policies. *J Addict Dis* 12:89–104, 1993.

Linn, M.W.; Linn, B.S.; and Greenwald, S.R. The alcoholic patient in the nursing home. *Aging Hum Dev* 2:273–277, 1972.

Marks, J. *The Benzodiazepines: Use, Overuse, Abuse.* Lancaster, England: MTP Press, 1978.

McCoy, D.G., and Napier, K. Alcohol and tobacco consumption as risk factors for cancer. *Alcohol Health Res World* 10:28–33, 1986.

Miller, W.R., and Rollnick, S. *Motivational Interviewing: Preparing People To Change Addictive Behavior.* New York: Guilford Press, 1991.

Mishara, B.L.; Kastenbaum, R.; Baker, F.; and Patterson, R.D. Alcohol effects in old age: An experimental investigation. *Soc Sci Med* 9:535–547, 1975.

Osher, F.C. A vision for the future: Toward a service system responsive to those with co-occurring addictive and mental disorders. *Am J Orthopsychiatry* 66:71–76, 1996.

Prochaska, J.O., and DiClemente, C.C. *The Transtheoretical Approach: Crossing Traditional Boundaries of Therapy.* Homewood, IL: Dow Jones/Irwin, 1984.

Ridgely, S.M.; Goldman, H.; and Willenbring, M.L. Barriers to the care of the dually-diagnosed: Organizational and financing issues. *Schizophr Bull* 16:123–132, 1990.

Robins, L., and Regier, D., eds. *Psychiatric Disorders in America: The Epidemiologic Catchment Area Study.* New York: Free Press, 1991.

Schmidt, W., and Popham, R.E. Role of drinking and smoking in mortality from cancer and other causes in male alcoholics. *Cancer* 47:1031–1041, 1981.

Spicer, P.; Willenbring, M.L.; Miller, F.; and Raymond, E. Ethnographic evaluation and the experience of asset management for homeless public inebriates. *Pract Anthropol* 16:23–26, 1994.

Vogel, L.J. Spiritual development in later life. In: Kimble, M.A.; McFadden, S.H.; Ellor, J.W.; and Seeber, J.J., eds. *Aging, Spirituality, and Religion.* Minneapolis, MN: Fortress Press, 1995. pp. 74–86.

Willenbring, M.L. Case management applications in substance use disorders. *J Case Manage* 3:150–157, 1994.

Willenbring, M.L.; Johnson, S.B.; and Tan, E. Characteristics of male medical patients referred for alcoholism treatment. *J Subst Abuse Treat* 11:259–265, 1994.

Willenbring, M.L.; Olson, D.H.; and Bielinski, J. Integrated outpatient treatment for medically ill alcoholic men: Results from a quasi-experimental study. *J Stud Alcohol* 56:337–343, 1995.

Zimmerman, R.S.; Warheit, G.J.; Ulbrich, P.M.; and Auth, J.B. The relationship between alcohol use and attempts and success at smoking cessation. *Addict Behav* 15:197–207, 1990.

Chapter 24

Age-Specific Treatment for Older Adult Alcoholics

Roland M. Atkinson, M.D.

This chapter is not about which forms of intervention (e.g., inpatient, outpatient, pharmacotherapy, Alcoholics Anonymous [AA]) are best for older alcoholics. What little is known about this question I have reviewed elsewhere (Atkinson 1995). Instead I want to address the question of whether it is advantageous to segregate older adults in an age-specific treatment program or track, no matter what form or location of treatment is selected. Let's begin with the punch lines.

At this time there is very little systematic evidence, based on studies of treatment outcome, to support the notion that it is better to treat older adult alcoholics with their agemates, rather than mixed together with younger adults. Indeed, a number of studies demonstrate that older adults have outcomes following alcohol treatment in mixed-age settings that are as good as or better than those of younger adults. On the other hand, there are age-specific issues that often need to be addressed to help assure an adequate outcome for older alcoholics. These issues, a number of which are listed in table 1, may be given short shrift in programs that treat mainly younger adults or that lack

Table 1. Age-Specific Issues.

- Loss (people, vocation, status)
- Social isolation and loneliness
- Major financial problems, poverty
- Dislocation of habitat
- Family conflict and estrangement
- Burden of time management ("boredom")
- Complex medical problems
- Sensory deficits
- Reduced mobility
- Cognitive impairment, loss
- Impaired self-care
- Loss of youthful appearance
- Reduced self-regard, demoralization

R.M. Atkinson, M.D., is professor and head of the Division of Geriatric Psychiatry (OP02), Oregon Health Sciences University, 3181 SW Sam Jackson Park Rd., Portland OR 97201.

Table 2. Age Effects on Outcome of Treatment for Alcoholism in Mixed-Age Programs.

Setting	No. of Studies	No. of Older Patients	Outcome: Older vs. Younger Patients No. of Studies		
			Worse	Same	Better
Inpatient	9	592	0	5	4
Outpatient	2	3,213	0	1	1

Note: Data for inpatient studies are from Glatt 1956; Ellis and Krupinski 1964; Myerson and Mayer 1966; Bateman and Petersen 1971; Blaney et al. 1975; Schuckit 1977; Wiens et al. 1982–83; Fitzgerald and Mulford 1992; Moos et al. 1994. Data from outpatient studies are from Ritson 1968; Janik and Dunham 1983. Cutoff ages for defining the "older" group vary by study and range from 45 to 65 years.

staff appropriately trained in gerontology. Because of this, most professionals who have worked with older alcoholics advocate age-specific treatment whenever this can be arranged. And the few studies comparing age-specific versus mixed-age treatment do all demonstrate superior compliance or outcome using an age-specific approach.

THE CASE AGAINST AGE-SPECIFIC TREATMENT

Three main points are usually argued by those who oppose age-specific treatment. First and most important, studies that have compared the outcomes of patients in mixed-age programs show that older patients tend to do as well as, or even better than, younger patients. Second, older patients may benefit from transgenerational contacts with younger patients. Third, establishing a separate track for older adults may be too costly, especially for smaller programs. I will expand on each of these points in the following paragraphs.

1. Older patients do well in mixed-age alcohol treatment programs.

Table 2 summarizes the results of my meta-analysis of the 11 best reports on mixed-age treatment programs in the literature from North America, the United Kingdom, and Australia, spanning the past four decades. These results support the argument that older patients do well in mixed-age programs (see Atkinson 1995, pp. 192–196, for a more extensive presentation of these data). The studies cited in table 2 vary in the cutoff age used to define "older" (from age 45 to age 65) and tend to be flawed by inclusion of relatively small numbers of older patients, inadequate and inconsistent outcome criteria, and fairly brief timeframes for followup. Nevertheless, the overall pattern of outcomes is clear enough with respect to age: supporters of mixed-age treatment are justified in contending that treatment in a mixed-age setting does *not* confer some special disadvantage on the older client. Programs unable to mount a special track for older clients need not fear that these clients are necessarily being deprived of the chance for an adequate outcome. On the other hand, because of the flaws in these studies, there is little basis for complacency that mixed-age treatment affords the *optimal* arrangements for the aging patient. Put another way,

these studies do not and cannot answer the question whether outcome of the older patients would have been even better had they been treated in an age-specific track.

2. Older patients benefit from transgenerational contacts with younger patients.

In the larger community some older adults resent segregation with agemates in retirement centers or may simply prefer the company of younger adults. It may also be possible for some older persons to resolve conflicts with younger relatives with the help of therapeutic contacts with younger patients in treatment settings. If there is a choice of entry to an age-specific or mixed-age track, such preferences can and should be honored. But my colleagues and I (Kofoed et al. 1987) have reported on the opposite phenomenon, in which older alcoholic clients "voted with their feet" by dropping out of a mixed-age, 1-year outpatient group counseling program. Arranging age-specific groups nearly doubled the length of older clients' retention in treatment, reduced premature discharges (dropouts, discharges for repeated drinking lapses), and increased somewhat the rate of attendance at required meetings (table 3).

Agemates enhance the opportunity for social peer bonding, shared reminiscence, and life review in group settings. Older adults in mixed-age therapy groups not infrequently voice complaints about the pace and the "psychologizing" that goes on, and they also tend not to tolerate the use of profanity or stories of antisocial behavior related to substance abuse told by younger participants in mixed-age treatment groups and AA meetings.

3. Establishing a separate track for older adults is too costly.

Except in very large programs, it very well may be too costly to create a separate program for inpatient detoxification or residential treatment for older adults. Detoxification of older patients with significant medical comorbidities may instead require the use of a consulting geriatrician or placement in a medical inpatient unit. But even within

Table 3. Outpatient Treatment Compliance: Age-Specific Versus Mixed-Age Conditions.

Condition (*n*)	Treatment Duration	Attendance Rate	Irregular Discharge Rate[1]
Mixed-age (24)	5.0 months	68%	67%
Age-specific (25)	9.1 months	81%	20%
p (2-tail, *t* test)	< 0.01	n.s.	< 0.01

Note: Compliance was measured in terms of attendance at weekly meetings expected for 1 year. Data from Kofoed et al. 1987. n.s. = not significant.

[1] Patient determined (dropouts) + staff determined (discharge for repeated drinking lapses).

a smaller mixed-age residential program or outpatient program, it should be possible to select or designate at least one staff member for special training and expertise in working with older adults. If only one or two older adults at a time are enrolled in the program, individual case management by this designated staff member is probably the best approach to assure that age-specific themes are addressed. One or more special group activities led by this person and others can be activated whenever several older clients are concurrently enrolled. Having a flexible definition of "older" (e.g., age threshold at 60 or even 55) may increase membership to a critical mass for productive social bonding and group discussion. Larger outpatient clinics can and should be able to offer age-specific tracks at little added cost. Likewise, except in small communities, it is feasible for local AA leadership to foster the development of age-specific AA chapters. Attitudes of AA leaders on this point vary considerably, however, from one locale to another. The question of purported extra cost or effort often is instead one of program philosophy and, unfortunately, "ageism."

THE CASE FOR AGE-SPECIFIC TREATMENT

There are several interrelated reasons to recommend age-specific treatment. As discussed in the following paragraphs, these reasons are related to special staffing needs and the "climate" of treatment for older clients, policy and procedural considerations, and the outcome of treatment.

1. Staffing needs for older clients are special.

Older adult alcoholics may not be willing to identify themselves publicly as alcoholic or problem drinkers, and they often tend to respond negatively to the confrontational approaches frequently favored by many chemical dependency counselors. Staff who work with older clients must also be alert to subtle clues to intercurrent medical, psychiatric, and social problems, expressions that may be unique to older persons. Accordingly, the requirements for effective counseling include patience; tolerance of a low-keyed, even oblique approach to discussing emotionally loaded themes and relationships; a sense of accomplishment in seeing clients make small gains in problem solving, life satisfaction, and social bonding; and sufficient grounding in the medical comorbidities and social problems of older persons to notice early warning signs of unwanted change and report them.

2. The "climate" of treatment often needs to be tailored to older clients.

If it takes special skills to be the ideal counselor to older alcoholics, this is because the themes, pace, and general atmosphere of treatment need to be modified for productive engagement of older clientele. Adequate respect for the courtesies favored by older persons is not always forthcoming in "mainstream" programs (calling older people new to the program by their first names is a small but telling example). Group therapy with older persons takes on special characteristics (Finkel 1990; Leszcz 1996). A group

for older adult alcoholics must above all offer emotional support, enhanced self-esteem, and hope to its members. Humor and lighthearted reminiscence are often valuable. The pace may be very slow, with a need for repetition of key points in discussion.

Cognitive-behavioral strategies seem to work especially well with older adult alcoholics (Dupree et al. 1984; see also chapter 19 in this monograph), especially when there is an emphasis on increasing self-efficacy (Rice et al. 1993). The cognitive-behavioral method arranges important information very explicitly in small units, and each participant proceeds at whatever pace is needed to assure assimilation of the material. Assimilation is measured via discussion and completion of "homework" before the work moves on to a new theme. Contrast this with the variable clarity, subtlety, and pace of most spontaneously proceeding "talk therapy" or AA "story telling."

The physical and logistical arrangements must also be tailored for older clientele. Issues of transportation, wheelchair access, and scheduling of daytime activities are illustrative.

3. Special policy and procedural considerations may apply to older patients.

Management of drinking lapses. Older alcoholics do not necessarily view drinking slips with the same degree of gravity as the staff or even younger patients. Greater tolerance of drinking lapses thus may be necessary in work with older clients, as long as the pattern is one of gradually decreasing severity, duration, and frequency of the drinking lapses. My group's experience indicates that a doctrinaire approach, seen in some mixed-age programs that banish clients for drinking lapses, will assuredly drive older alcoholics away.

Family participation in care. Although important in any setting, family involvement may be critically important in successful engagement and treatment of older alcoholics. The program must be flexible in engaging the key person in the social network, who may often be a caregiving daughter (Dunlop et al. 1982; Dunlop 1990), nonmarried cohabitant, or close friend. Participation of a spouse in the treatment program has been demonstrated to affect program compliance in married older alcoholics (Atkinson et al. 1993).

Duration of participation in the program. If older clients often do not fully identify themselves as alcoholic, it follows that they may need the continuing external support of the program for a longer interval to assure continued sobriety. Mixed-age programs tend to rely on AA affiliation to provide enduring support for sobriety, but every age-specific outpatient program I have reviewed practices an arrangement of open-ended participation at the program site. Typically patients "graduate" from a phase labeled "active treatment" into an "alumni" group that may be led by a professional or securely sober peer leader. In our program, among older adults completing a full year of treatment, 75 percent chose voluntarily to continue in an alumni group. If nothing else, this reflects the strength of social

bonding that can occur in an age-specific program. Where age-specific AA chapters exist, AA can be an excellent alternative for open-ended support for the older alcoholic.

4. Elder-specific treatment may produce superior results.

Only three studies have compared the fate of older patients assigned either to age-specific or mixed-age treatment. These studies, described extensively elsewhere (Atkinson 1995, pp. 197–204), are limited by the small numbers of patients studied (total in the three studies of 111 in age-specific conditions vs. 106 in mixed-age conditions) and the setting of Veterans Affairs (VA) programs. Only one of these studies actually examined treatment outcomes (Kashner et al. 1992), and it is the only study that randomized patients to the respective treatment conditions. The other studies looked at treatment compliance—the extent to which patients remained in the program.

This empirical base is indeed very slim pickings, but the pattern of results in these studies, summarized in table 4, is clear: all point to better compliance or outcome for patients treated in the age-specific condition. In the randomized inpatient study, patients treated in the age-specific alcohol treatment unit (ATU), where many of the age-related issues mentioned earlier were addressed, were 2.9 times more likely at 6-month followup, and 2.1 times more likely at 1 year, to report abstinence than their counterparts who received usual treatment in a mixed-age ATU (Kashner et al. 1992). Moreover, these differences increased with patient age: patients who were ages 50, 55, 60, and 70 years old were, respectively, 0.5, 1, 1.6, and 5.1 times more likely to abstain from drinking following age-specific treatment than after the mixed-age treatment. That is, age 60 appeared to be the best cutoff for enhancing outcome through assignment to an age-specific program. Patient care costs were slightly lower (2.5 percent lower) in the age-specific program.

Table 4. Age-Specific Versus Mixed-Age Treatment for Older Alcoholics.

Study	Setting	*N's**	Age Cutoff	Type of Study	Results: Age-Specific
Thomas- Knight 1978	IP ATU	14/17	55	compliance	superior
Kofoed et al. 1987	outpatient alcohol clinic	25/24	55	compliance	superior
Kashner et al. 1992	IP ATU	72/65	45	1-year outcome	superior (age 60 and older)

Note: IP ATU = inpatient alcohol treatment unit.
* First number for age-specific condition. Second number for mixed-age condition.

BARRIERS TO ESTABLISHING AGE-SPECIFIC TREATMENT

The barriers to establishing age-specific treatment include difficulty in obtaining staff with the appropriate training and characteristics, the burdens caused by having to deal with comorbidities, the stresses of working with elderly clients, funding problems, and variable referral volume.

1. Staffing: Who has the "right stuff"?

Above all, high levels of compassion for and interest in older people are indispensable characteristics for staff who work with older alcoholics. The ideal professional should also have training and expertise in three disciplines: chemical dependency, gerontology, and mental health. People with such a rich and varied background are scarce and may command higher salaries or fees. At the least, in a sustained program for older patients, a nurse needs to be available to manage and broker needs for medical assessment and care.

2. Comorbidities can impose program burdens.

Most substance abuse programs attempt to limit costly medical services. However, a program that wishes to hang out a shingle inviting referrals of older adults must be prepared for clients who will need thorough, not perfunctory, medical and psychiatric screening and in some cases creation of linkages to medical care providers, requiring a case management strategy. In-home services may be needed for initial intervention or followup at times of reduced functional status. These activities impose added costs and required levels of staff expertise.

3. Stresses of work with elderly clients can be difficult for patients and staff.

When our group retrained alcoholism counselors, who were accustomed to working with younger adults, to work with older adults, these staff reported the stressfulness of witnessing their clients growing ill from progressive medical comorbidities and of clients' deaths. In our age-specific program 5 percent of clients died during the index year of treatment, a very high proportion compared with younger treatment cohorts (Atkinson et al. 1993), and other patients were forced to drop out as functional impairments increased. In group-based treatment, losses affect not only staff but other patients as well.

4. Program funding can be precarious.

Threats to funding. We have seen good age-specific programs undone because of financial problems. Medicare will pay for alcoholism-related treatment services but at rates low enough to discourage a team-oriented model that incorporates adequate social, medical, and psychiatric services within the program. Nor will Medicare typically pay for intensive day treatment fees as such, or for long-term outpatient visits. The required 50 percent outpatient copays may be prohibitive for many persons lacking supplemental "medigap" in-

surance coverage. State and local publicly (Medicaid) funded programs are crucial for patients under age 65 who are uninsured, but it can be difficult to interest state and county officials in funding programs for a special population that is often perceived as small. The current emphasis across the entire national health care landscape on primary care, with a corresponding aversion to highly specialized care, militates against support for special population-based treatment.

Funding strategies for the late 1990's. Three strategies have aided age-specific alcohol treatment programs financially: splitting off separately billable services, negotiating payment for service packages, and using volunteer staff. It is now common practice in many chemical dependency programs to separate psychiatric and medical professional services from the program, instead of arranging referral-based participation of these providers, who then bill for services independently of the program. The second strategy is one that is becoming increasingly important as Medicare programs are restructured into managed care organizations. With this strategy, the treatment program negotiates directly with the state or regional Medicare manager for what amounts to a lump sum to provide an agreed-upon package of services to the elderly alcoholic. The Medicare manager may be supportive because of the argument that by providing relatively cheap outpatient alcoholism treatment now, more expensive care later (i.e., care for medical problems that are caused or made worse by continued heavy drinking) can be reduced. Recall Adams and colleagues' finding that Medicare paid nearly one-quarter of a billion dollars for alcohol-related acute inpatient admissions in 1989 alone (Adams et al. 1993; see also chapter 18 in this monograph). Usually the basis of the negotiated arrangement is a relatively generously funded amount that will cover intensive outpatient services for up to a few months. Funds thus generated can be sufficient to cover the costs of counselor time and overhead for later, less intensive outpatient visits, especially if the program can also create and sustain an adequate volunteer program to supplement paid staff, which is the third strategy of importance in maintaining financial solvency. Establishing an effective volunteer program requires a substantial investment by the treatment program in leadership, screening, training, supervision, and incentives for volunteers.

5. Referral volume is often variable.

Besides funding, the other major threat to maintaining an age-specific program is variable or insufficient volume of referrals for treatment. Reluctance of patients and family, the tendency of health and social services professionals to miss the diagnosis or fail to initiate a referral, costs and other barriers to access all contribute to this problem. To overcome these barriers, one effective strategy is to link or affiliate the program with an integrated health care system. Another strategy is to designate program staff specifically for roles in health and social services provider education, clinical

consultation, and outreach. Having a liaison staff member who can go into any clinic, to the bedside on any ward, or to the home of any prospective patient creates countless opportunities for case finding and intervention to facilitate patients moving into treatment. These two strategies are often linked in practice.

RECOMMENDATIONS

First, there is obviously a need for further studies comparing age-specific treatment with mixed-age treatment. The focus of such studies should be in the outpatient setting in programs that serve women as well as men. The treatment studies funded by the National Institute on Alcohol Abuse and Alcoholism (NIAAA) generally have included few older patients (Project MATCH Research Group 1997). NIAAA should encourage a relatively modest multisite outpatient study comparing age-specific with usual (mixed-age) treatment of older alcoholics.

Second, age-specific community outreach services for older alcoholics need further development. I have in mind programs aimed at older alcoholics who are shut away in single-room occupancies and retirement centers, and who cannot or will not attend an alcohol treatment program. Some of these, like the COPA Program in Toronto (Graham et al. 1995), the Spokane "Gatekeepers" aging services program (Jinks and Raschko 1990), and the North of Market Program in San Francisco (Fredriksen 1992), have been remarkably effective in reaching, in particular, socially and physically isolated elderly women alcoholics. These programs need to be sustained and extended to other communities, preferably linked to activities of the Area Agencies on Aging as well as to alcoholism treatment services.

Third, brief intervention and prevention efforts may also need an age-specific orientation to be effective. There is evidence that successful appeals to older adults to stop smoking differ from appeals that work with younger adults (Orleans et al. 1994). The same may be true for influencing older "at-risk" drinkers to reduce their alcohol consumption. Data bearing on this question may be forthcoming from age-specific brief intervention studies in Michigan by Blow and colleagues (see chapter 21).

Fourth, new models for funding under Medicare may need encouragement from NIAAA and the Center for Substance Abuse Treatment (CSAT). An advocacy role with the Health Care Financing Administration should be considered by NIAAA and/or CSAT to help promote adequate funding for tested models of age-specific treatment for alcohol problems.

EPILOGUE: THE UNMEASURED BENEFITS OF AN NIAAA DEMONSTRATION PROJECT

I want to conclude by telling a story about the widespread, long-lasting but undocumented positive consequences of a federal grant. It is the

sort of story that is seldom told, because of the short-term nature of grants and government-influenced timeframes in general. It should be required reading for anyone—agency and congressional staff, journalists, peer reviewers—who has any say about the role of the federal government in promoting health and social programs for the public good.

In the mid-1970's, after a call for proposals, NIAAA funded a 3-year demonstration project on age-specific case finding and treatment for older alcoholics in Clark County, WA, part of the greater Portland (OR)–Vancouver (WA) metropolitan area that straddles the Columbia River. The program, Senior Alcohol Services (SAS), got underway in 1978. In an innovative manner, it provided outreach and case finding services, and outpatient treatment, to older alcoholics and their family caregivers, and it created successful links to a hospital-based inpatient chemical dependency unit and to AA. After the project grant ran out, temporary funds to sustain the program were obtained from the State of Washington, and then the program died, in about 1984. SAS made little splash nationally, although brief reports of its work were published, shortly before its death, by the program's leaders (Dunlop et al. 1982) and by NIAAA (Williams 1983). This could have been the end of the story, but it wasn't.

As it happened, SAS was located only a few blocks from the chemical dependency program at the Vancouver Division of the Portland VA Medical Center, a geographic arrangement that had favored the VA staff's habit of sending older veterans seeking care for alcohol problems to SAS rather than serving them at the VA, a glaring instance of ageism. But one VA chemical dependency counselor, an older, recovering alcoholic himself, became interested in gerontology and older clients and sought out guidance from SAS staff in order (hopefully) to initiate an age-specific outpatient group treatment track at the VA. His efforts received an unexpected boost when SAS, faced with dwindling financial support, was forced to refuse care to VA-eligible clients beginning in 1982. This led to the establishment of the age-specific Portland VA outpatient program in that year, a program which has run to the present day, beginning its 16th year recently (Atkinson et al. in press). This program, with which I have been associated as research director since its inception, has treated over 500 patients and has served as an important site for systematic published clinical studies of factors affecting treatment compliance as well as the phenomenon of late-onset alcoholism.

Meanwhile, one of the original SAS leaders went on to develop the first community hospital–based age-specific alcohol treatment program in Portland in 1988 (Dunlop 1990). Another local hospital in the same system was moved by this example to initiate a similar program and hired as its first director a social worker who had become interested in age-specific treatment while working at the

Portland VA chemical dependency unit and observing its successful program for older veterans. Leaders of all these programs, as well as others whose level of awareness had been raised by contacts with them, organized in the mid-1980's to formulate a community-wide agenda for responding to alcohol problems in older adults in Portland (Dunlop et al. 1990). Their efforts eventually attracted a sizable grant from a private charitable foundation in order to pursue educational projects for geriatric health and social service providers and for the public on alcohol and aging, establish an information and referral system, and sponsor annual conferences bringing in national authorities to speak and conduct workshops (Project DARE—Drug and Alcohol Resources for the Elderly). Several other community- and hospital-based age-specific treatment programs in the area were developed in response to these efforts. Strides were made in establishing age-specific AA chapters as well. SAS had originally negotiated with local AA leaders to begin an age-specific chapter in Vancouver that has operated continuously to the present. At least two other such chapters have also emerged in Portland.

It is, of course, impossible to know how many of the activities described here would have developed if SAS had never existed. On the other hand, the direct links between SAS and the VA, two community hospital–based programs, and the foundation-sponsored Project DARE, are indisputable. And few communities have witnessed the proliferation of interest and programs for aging alcoholics seen in the Portland area. None of this, obviously, could have been incorporated in the "final" report SAS made to NIAAA, probably in 1981 or 1982. But to me it is abundantly clear that this demonstration project did just what such a project should ideally do: it demonstrated in a fashion that influenced an entire urban metropolitan community. By investing taxpayer dollars in SAS, NIAAA leveraged its resources to impressive advantage to benefit older problem drinkers, their families, and the community, for nearly 20 years (and counting).

REFERENCES

Adams, W.L.; Yuan, Z.; Barboriak, J.J.; and Rimm, A.A. Alcohol-related hospitalizations of elderly people: Prevalence and geographic variation in the United States. *JAMA* 270:1222–1225, 1993.

Atkinson, R.M. Treatment programs for aging alcoholics. In: Beresford, T., and Gomberg, E., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 186–210.

Atkinson, R.M.; Tolson, R.L.; and Turner, J.A. Factors affecting outpatient treatment compliance of older male problem drinkers. *J Stud Alcohol* 54:102–106, 1993.

Atkinson, R.M.; Turner, J.A.; and Tolson, R.L. Treatment of older adult problem drinkers: Lessons learned from the "Class of '45." *J Ment Health Aging,* in press.

Bateman, N.I., and Petersen, D.M. Variables related to outcome of treatment for hospitalized alcoholics. *Int J Addict* 6:215–224, 1971.

Blaney, R.; Radford, I.S.; and MacKenzie, G. A Belfast study of the prediction of

outcome in the treatment of alcoholism. *Br J Addict* 70:41–50, 1975.

Dunlop, J. Peer groups support seniors fighting alcohol and drugs. *Aging* 361:28–32, 1990.

Dunlop, J.; Skorney, B.; and Hamilton, J. Group treatment for elderly alcoholics and their families. *Soc Work Groups* 5:87–92, 1982.

Dunlop, J.; Manghelli, D.; and Tolson, R. Older problem drinkers: A community treatment continuum. *Aging* 361:33–37, 1990.

Dupree, L.W.; Broskowski, H.; and Schonfeld, L. The Gerontology Alcohol Project: A behavioral treatment program for elderly alcohol abusers. *Gerontologist* 24:510–516, 1984.

Ellis, A.S., and Krupinski, J. The evaluation of a treatment programme for alcoholics: A follow-up study. *Med J Aust* 1:8–13, 1964.

Finkel, S.I. Group psychotherapy with older people. *Hosp Community Psychiatry* 41:1189–1191, 1990.

Fitzgerald, J.L., and Mulford, H.A. Elderly vs. younger problem drinker "treatment" and recovery experiences. *Br J Addict* 87:1281–1291, 1992.

Fredriksen, K.I. North of Market: Older women's alcohol outreach program. *Gerontologist* 32:270–272, 1992.

Glatt, M.M. Treatment results in an English mental hospital alcoholic unit. *Acta Psychiatr Scand* 37:143–168, 1956.

Graham, K.; Saunders, S.J.; Flower, M.C.; Timney, C.B.; White-Campbell, M.; and Pietropaolo, A.Z. *Addictions Treatment for Older Adults. Evaluation of an Innovative Client-Centered Approach.* New York: Haworth Press, 1995.

Janik, S.W., and Dunham, R. G. A nationwide examination of the need for specific alcoholism treatment programs for the elderly. *J Stud Alcohol* 44:307–317, 1983.

Jinks, M., and Raschko, R. A profile of alcohol and prescription drug abuse in a high-risk community-based elderly population. *Ann Pharmacother* 24:971–975, 1990.

Kashner, T.M.; Rodell, D.E.; Ogden, S.R.; Guggenheim, F.G.; and Karson, C.N. Outcomes and costs of two VA inpatient treatment programs for older alcoholic patients. *Hosp Community Psychiatry* 43:985–989, 1992.

Kofoed, L.L.; Tolson, R.L.; Atkinson, R.M.; Toth, R.L.; and Turner, J.A. Treatment compliance of older alcoholics: An elder-specific approach is superior to "mainstreaming." *J Stud Alcohol* 48:47–51, 1987; correction 48:183, 1987.

Leszcz, M. Group psychotherapy. In: Sadavoy, J.; Lazarus, L.W.; Jarvik, L.F.; and Grossberg, G.T., eds. *Comprehensive Review of Geriatric Psychiatry—II.* 2d ed. Washington, DC: American Psychiatric Press, 1996. pp. 851–879.

Moos, R.H.; Brennan, P.L.; and Mertens, J.R. Diagnostic subgroups and predictors of one-year readmission among late-middle-age and older substance abuse patients. *J Stud Alcohol* 55:173–183, 1994.

Myerson, D.J., and Mayer, J. Origins, treatment and destiny of Skid-Row alcoholic men. *N Engl J Med* 275:419–425, 1966.

Orleans, C.T.; Jepson, C.; Resch, N.; and Rimer, B.K. Quitting motives and barriers among older smokers. *Cancer* 74:2055–2061, 1994.

Project MATCH Research Group. Matching alcoholism treatments to client heterogeneity: Project MATCH posttreatment drinking outcomes. *J Stud Alcohol* 58:7–29, 1997.

Rice, C.; Longabaugh, R.; Beattie, M.; and Noel, N. Age group differences in response to treatment for problematic alcohol involvement. *Addiction* 88:1369–1375, 1993.

Ritson, B. The prognosis of alcohol addicts treated by a specialized unit. *Br J Psychiatry* 114:1019–1029, 1968.

Schuckit, M.A. Geriatric alcoholism and drug abuse. *Gerontologist* 17:168–174, 1977.

Thomas-Knight, R. Treating alcoholism among the aged: The effectiveness of a special treatment program for older problem drinkers. *Dissertation Abstr Int B* 39:3009, order no. 7823210, 1978.

Wiens, A. N.; Menustik, C.E.; Miller, S.I.; and Schmitz, R.E. Medical-behavioral treatment of the older alcoholic patient. *Am J Drug Alcohol Abuse* 9:461–475, 1982–83.

Williams, M. Senior program stresses peer, family involvement. *NIAAA Information and Feature Service*, No. 106:1, 1983.

Chapter 25

Commentary on Prevention of Alcohol Problems in the Elderly

Gayle M. Boyd, Ph.D.

This monograph addresses important issues related to the nature, prevalence, etiology, and treatment of alcohol problems among older adults. Noticeably absent, however, is a comprehensive review of prevention research directed toward older adults. It is absent because, unfortunately, the literature does not exist. In this chapter I will discuss some theoretical issues that should be considered in developing a prevention research agenda and suggest some directions for future activity in this area.

Currently in the United States the fastest growing segment of the population is persons age 65 and over. Between 1980 and 1990 this population increased by 21.6 percent, compared with an increase of only 8.5 percent in the population under 65 (U.S. Bureau of the Census 1992). By the year 2030, when the "baby boomer" generation born following World War II is past retirement age, there will be approximately 65 million persons over the age of 65, comprising about 22 percent of the total population (Spencer 1989). For comparison, in 1990 this age group made up only 12 percent of the population. Given the size of the projected population and some evidence that this generation may drink more than their predecessors, alcohol-related problems among persons over the age of 65 will be an increasingly important public health issue.

The older population is at risk for a variety of alcohol-related problems, which have been described in several chapters in this monograph. Some effects of alcohol mimic changes that normally occur with aging, and its use

G.M. Boyd, Ph.D., is a health scientist administrator in the Prevention Research Branch of the Division of Clinical and Prevention Research at the National Institute on Alcohol Abuse and Alcoholism, 6000 Executive Blvd., Bethesda, MD 20892. She serves as program director for research grants on the prevention of alcohol problems in youth and in older adults.

may produce additive deleterious effects. In addition to adverse effects on the liver, heart, gastrointestinal tract, and immune system and increased risk for some cancers, alcohol use has been associated with disturbances of sleep, cognitive and motor impairment, falls and other types of accidents, motor vehicle crashes, violence, depression, personal neglect, inability to carry out responsibilities, and disruption of personal relationships (National Institute on Alcohol Abuse and Alcoholism 1990, 1994). Adverse consequences of particular concern due to the numbers of persons at risk include interactions with medications, depression, accidents, and motor vehicle crashes. Older persons appear to be more sensitive to alcohol, both pharmacologically and behaviorally (see chapter 6), and this increases their risk for alcohol-related motor vehicle crashes (see chapter 17). However, moderate use is not necessarily contraindicated for healthy elderly persons, and some studies have found evidence of positive effects (Goodwin et al. 1987; Dufour 1992, 1996).

The appropriate goal for prevention efforts is to reduce the incidence of alcohol-related problems among older adults. It should be noted that many of the problems described in this monograph may occur at very low levels of drinking in susceptible individuals. Therefore, as with the younger population, prevention objectives are not limited to reducing alcohol abuse and dependence, but should encompass a full array of problems that may be associated with various patterns of consumption.

However, relatively little is known about the true prevalence of alcohol-related problems in the older adult population. As discussed in chapters 2, 19, and 21, characteristics of this population make collection of epidemiologic data especially challenging. Most notable of these characteristics is heterogeneity. There are great disparities among older individuals in health status and cognitive and physical functioning, in addition to demographic factors; and patterns of alcohol use, including abstinence, are not distributed evenly. To further complicate the scene, demographic patterns are rapidly changing with the influx of immigrant groups and increased longevity. Estimates of alcohol consumption based on the general population over a specified age are not especially useful in assessing the extent to which alcohol problems are present, designing interventions, or projecting future needs.

Instead of estimates that are of limited utility, we need risk profiles for specific subpopulations that explore the full range of alcohol problems that may be experienced. Little is known about the underlying factors that promote abstinence or light to moderate drinking in these populations or changes in drinking. We need to learn the extent to which older populations are experiencing significant problems associated with alcohol, the nature of those problems, how they arise and are maintained, and what incentives and barriers to change are present.

There are a variety of potential lifetime patterns of alcohol use involving increased, decreased, cyclical, or unchanging consumption with age.

Three patterns among the elderly are of special public health concern: (1) the continuation of a lifetime pattern of alcohol abuse or alcoholism into old age (early-onset alcohol abuse); (2) the initiation of alcohol abuse in old age without a prior history of alcohol problems (late-onset alcohol abuse); and (3) drinking at a level not classified as abusive by existing criteria, but that places the individual at risk for alcohol-related problems due to health conditions, medication interactions, decreased tolerance, impaired motor skills, or other circumstances. The last two patterns are most relevant for prevention research.

Because alcohol problems among the elderly are forecast to be an increasingly important public health problem and available resources are finite, it is important that prevention research efforts be directed in an efficient manner to increase the body of information that is most needed for the development of effective interventions. The experience of prevention programs directed toward younger populations, including those addressing health problems that are not alcohol related, may be useful in guiding these efforts.

In 1989 the Institute of Medicine published *Prevention and Treatment of Alcohol Problems: Research Opportunities,* which urged adoption of a public health model of alcohol-related problems. In such a model health problems are seen to arise from complex interactions of factors from three domains: individual biological and psychological characteristics, knowledge, beliefs, and experience (host); the social and physical context within which drinking occurs (environment); and alcohol itself (agent). Logically, of course, preventive intervention efforts might operate within these or otherwise-defined domains and target a variety of factors and processes that contribute to the presence of alcohol problems within a population.

The growing literature on the etiology of alcohol problems in the young provides strong support for the importance of considering multiple domains of influence. Complex developmental models have emerged in which risk is seen to increase and decrease over time with the changing interplay of underlying contributing factors related to individual biological and psychological makeup, developmental stage, family and peer influences, sociocultural context, and societal norms (Zucker et al. 1994). In chapter 1 of this monograph, Zucker suggests that alcohol use in later life stages might be described by similar models in which a developmental dimension captures the biological, cognitive, economic, and social changes that typically accompany the aging process.

Prevention interventions for other age groups have been directed toward individuals (education, skills development, alternate behaviors), families (parenting practices), peers (social norms), organizations (organization norms through intervention delivery and policy change), and the larger social environment (social norms and policies, e.g., availability and sanctions). While the content of prevention programs for older adults will differ substantially from those for younger groups, the potential utility of strategies that include

more than individual-focused education should at least be considered.

Treatments necessarily focus on individuals, and case finding—locating and recruiting abusive and dependent drinkers—naturally becomes a major research and intervention focus. Prevention, however, is directed toward populations with the intent of reducing their risk for alcohol-related problems, and the identification of appropriate target populations is integral to intervention development. A 1994 Institute of Medicine report, *Reducing Risks for Mental Disorders: Frontiers for Preventive Intervention Research,* recasts the traditional public health classification system of primary, secondary, and tertiary prevention to accommodate behavioral disorders, including drinking. The terms *universal, selective,* and *indicated,* which were previously introduced by Gordon (1983), are proposed in this report to describe three population-defined levels of preventive intervention. Universal prevention is directed toward the general population regardless of individual risk, selective prevention is targeted to specific high-risk groups or individuals known to have high imminent or lifetime risk, and indicated interventions are directed toward individuals who have begun to manifest some early signs or symptoms of incipient alcohol problems.

The Institute of Medicine report also notes that public health requires public allocation of resources, and costs must be weighed against potential benefit. Cost-effectiveness can be maintained when resource allocations increase, on a per capita basis, if the target population is circumscribed and known to be at greater risk. Therefore, interventions generally follow a gradient of increasing intensity, from universal to selective and indicated prevention programs (Institute of Medicine 1994).

In chapter 21 of this monograph, Blow incorporates this natural continuum of intervention in his spectrum of interventions for older adults. The spectrum ranges from prevention/education activities and brief advice for nondrinkers and low-risk drinkers to formal treatment programs for heavy and dependent drinkers. The interventions that he suggests for the first two groups are logical choices that are fairly low in intensity and cost. The more thorny questions about how to access the target populations, the optimal modality for materials (e.g., written or audiovisual), appropriate content, and delivery systems await research.

There are myriad prevention-related research questions that could be addressed were time and money unlimited. But since those luxuries are not available, resources need to be allocated in ways that will advance the field most rapidly. Holder and colleagues (1995) discussed the potential value of heuristic models for prevention research for use in assessing the state of knowledge in a field and identifying the most promising areas for future research. They recommended use of a phases model that would incorporate the logical sequence by which research progresses from basic to applied questions, similar to the models developed for prevention research in cancer (Greenwald and Caban 1986), heart

disease (National Heart, Lung, and Blood Institute 1987), and mental disorders (Institute of Medicine 1994). Although a formal phases model for alcohol prevention research has not yet been developed, it would necessarily include an ideal progression from basic research through preintervention research, efficacy testing, effectiveness testing, and demonstrations. That is, the problem being addressed is first identified within the general population or special populations; causes, mechanisms, and consequences are studied; the feasibility of particular intervention strategies are explored; intervention programs are developed and tested; and effective interventions are disseminated. Each phase builds upon those that precede it.

However, as with other populations in which prevention efforts are needed, program planners do not necessarily have the luxury of awaiting the development of research-based and well-tested interventions. For example, recent concern about alcohol problems on college campuses has impelled many college administrations to adopt stricter policies regarding alcohol use. However, these have been instituted without benefit of foundational research; and there has been insufficient evaluation to determine which, if any, are effective (Commission on Substance Abuse at Colleges and Universities 1994). Such program-driven interventions, as opposed to research-driven or investigator-initiated interventions, can provide opportunities for research through rigorous evaluation of program effects, as well as processes. The inclusion of so-called natural experiments that take advantage of "spontaneous" intervention activities or environmental changes is an important feature of the phases model called for by Holder and colleagues (1995). In fact, this is often the only way that policy effects, such as those produced by changes in alcohol taxes or penalties associated with drinking and driving, can be studied. Natural experiments can also stretch scarce research dollars because the intervention itself need not be funded.

As problem drinking among older adults becomes a more pressing public health concern, there will be increased prevention efforts by public and private program planners. It is important that the research community act to develop a solid body of knowledge on which such programs can be based. But it is also important to recognize opportunities for efficient advancement through research phases (shortcuts) that may be presented by "naturally" occurring interventions. For example, the American Association of Retired Persons (AARP) has developed educational video and written materials on alcohol abuse (available from AARP, Program Resources Department, P.O. Box 51040, Station R, Washington, DC 20091). Although these materials were based on the best available information and formative research was carried out to assure acceptability to the target audience, there has been no efficacy or effectiveness evaluation. Given the excellent distribution network available to AARP, these materials have the potential of reaching a wide audience; and research on their efficacy or effectiveness could lead to a significant public health benefit.

There are virtually no randomized controlled intervention studies for the prevention of alcohol problems among the elderly. Nor does there appear to be adequate foundational or basic research to support full-scale intervention trials at this time. Additional preintervention research is clearly needed, but it is essential that it address the research questions most directly related to the development of future interventions.

The need for specific population profiles has already been described, and a number of potential risk and protective factors for experiencing alcohol problems in later life have been identified in this monograph. It is important that population profiles and explorations of processes and factors underlying risk maintain a prevention perspective. That is, emphasis should be placed on identifying *modifiable* factors that account for sufficient variance to merit intervention, markers that can readily identify individuals or groups at risk, the acceptability of particular intervention strategies, appropriate modalities for providing information, need for tailoring messages for particular populations, and channels of access through which populations can be reached.

The question of population access has considerable practical significance for both research and intervention delivery; and, whenever possible, information should be collected on frequency and level of contact with specific organizations and systems that could be used for intervention delivery. It may sometimes be appropriate to define target populations according to the potential means of intervention delivery. The most obvious of these is the health care delivery system, including hospital acute care and emergency departments, outpatient facilities, and convalescent hospitals with short-term stay patients. Age-segregated living facilities offer another potential means of reaching older populations, including residents in retirement communities, subsidized housing, congregate living facilities, retirement hotels, and residential nursing homes. Older adults might also be reached through social or cultural institutions with which they come in contact, such as senior citizen centers and churches; through government offices such as departments of motor vehicles, Social Security, and Medicare; and through service outreach organizations like Meals on Wheels. Commercially obtained age-specific mailing lists might also be useful in accessing some groups, especially well-educated and active individuals not living in age-segregated housing.

Intervention messages will, naturally, be tailored according to population characteristics, level of risk for specific alcohol problems, and delivery channel. Since the intended outcome is to influence in a constructive way individual decisions about whether, when, where, and how much to drink, it is important to understand key components of the decision process: How much is alcohol valued? What benefits are associated with alcohol? What role does it play in the individual's social and emotional life? What perceived "costs" are associated with drinking? What perceived "costs" would be associated with changes in drinking prac-

tices? To what extent is drinking determined by social influences (peer pressure)? As with younger groups, information provided to affect a decision must be relevant to that process as perceived by the decisionmaker (Fischhoff and Quadrel 1995). Some of the methods developed to uncover critical cognitions that underlie drinking decisions by adolescents and young adults, such as expectancy research (Fromme et al. 1986; Goldman 1994; Leigh and Stacy 1994), may be useful for studying older populations.

Although methodologies may be borrowed from alcohol prevention research with other age groups, their applicability to older adults must be evaluated. Research and intervention methods, including recruitment strategies, intervention delivery systems, modalities used to convey information, data collection instruments, and evaluation measures, may require extensive development and feasibility testing. Feasibility field tests may also be needed to determine intervention acceptability, assess potential efficacy, and estimate projected costs.

Environmental and policy interventions have been found to reduce alcohol-related problems in the general population. For example, increases in the minimum legal drinking age have been associated with reductions in alcohol-related traffic fatalities among underage drivers (Toomey et al. 1996). Higher alcohol prices have been associated with decreases in alcohol consumption and related problems, although responsiveness to price changes is not constant across different segments of the population (Kenkel and Manning 1996). The physical and policy context in which drinking by the elderly takes place should be examined with the intent of identifying possible age-specific or general opportunities for intervention. For instance: alcohol use policies in age-segregated living facilities may influence misuse by residents; features of labeling and dispensing of medications may affect the incidence of adverse interactions with alcohol; and age-specific drink-driving laws, similar to the zero-tolerance law for youth, might reduce crashes among older adults. And, as indicated earlier, naturally occurring policy changes may provide opportunities for research by evaluating their effects on drinking and drinking-related problems among older adults.

All prevention efforts should be rooted in a firm respect for the intended recipients. Interventions delivered to "captive" audiences, and especially policy interventions, can be somewhat coercive. Issues of individual autonomy, as well as individual health and public good, must be carefully weighed. Unlike underage drinking, alcohol use by adults is perfectly legal. Further, older individuals have a lifetime of decisionmaking experience, and in the absence of cognitive impairment, are capable of evaluating the relative risks and benefits of moderate alcohol use when provided with adequate information and viable alternatives. It should also be recognized that older individuals make lifestyle choices from the vantage point of late life, and this may affect the relative subjective value of potential decision outcomes. This special perspective should be acknowl-

edged and respected in the development and evaluation of interventions, whether environmental, information based, or motivational. On the other hand, age should not be an excuse for disregarding the presence of preventable morbidity. Rather, it should challenge researchers, service providers, and social planners to give more careful attention to identifying or creating viable alternatives to problem drinking *within* the context of life stage.

REFERENCES

Commission on Substance Abuse at Colleges and Universities. *Rethinking Rites of Passage: Substance Abuse on America's Campuses.* New York: Center on Addiction and Substance Abuse at Columbia University, June 1994.

Dufour, M.C. Risks and benefits of alcohol use over the life span. *Alcohol Health Res World* 20(3):145–151, 1996.

Dufour, M.C.; Archer, L.; and Gordis, E. Alcohol and the elderly. *Clin Geriatr Med* 8(1):127–141, 1992.

Fischhoff, B., and Quadrel, M.J. Adolescent alcohol decisions. In: Boyd, G.M.; Howard, J.; and Zucker, R.A., eds. *Alcohol Problems Among Adolescents: Current Directions in Prevention Research.* Hillsdale, NJ: Lawrence Erlbaum Associates, 1995. pp. 59–84.

Fromme, K.; Kivlahan, D.R.; and Marlatt, G.A. Alcohol expectancies, risk identification, and secondary prevention with problem drinkers. *Adv Behav Res Ther* 8:237–251, 1986.

Goldman, M.S. The alcohol expectancy concept: Applications to assessment, prevention, and treatment of alcohol abuse. *Appl Prev Psychol* 3:131–144, 1994.

Goodwin, J.S.; Sanchez, C.J.; Thomas, P.; Hunt, C.; Garry, P.J.; and Goodwin, J.M. Alcohol intake in a healthy elderly population. *Am J Public Health* 77(2):173–177, 1987.

Gordon, R. An operational classification of disease prevention. *Public Health Rep* 98:107–109, 1983.

Greenwald, P., and Caban, C.E. A strategy for cancer prevention and control research. *Bull World Health Organ* 64:73–78, 1986.

Holder, H.; Boyd, G.; Howard, J.; Flay, B.; Voas, R.; and Grossman, M. Alcohol-problem prevention policy: The need for a phases research model. *J Public Health Policy* 16(3):324–346, 1995.

Institute of Medicine. *Prevention and Treatment of Alcohol Problems: Research Opportunities.* Washington, DC: National Academy Press, 1989.

Institute of Medicine. *Reducing Risks for Mental Disorders: Frontiers for Preventive Intervention Research.* Washington, DC: National Academy Press, 1994.

Kenkel, D., and Manning, W. Perspectives on alcohol taxation. *Alcohol Health Res World* 20(4):230–238, 1996.

Leigh, B.C., and Stacy, A.W. Self-generated alcohol outcome expectancies in four samples of drinkers. *Addict Res* 1(4):335–348, 1994.

National Heart, Lung, and Blood Institute. *Guidelines for Demonstration and Education Research Grants.* Bethesda, MD: U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, 1987.

National Institute on Alcohol Abuse and Alcoholism. *Seventh Special Report to the U.S. Congress on Alcohol and Health.*

DHHS Pub. No. (ADM) 90–1656. Washington, DC: U.S. Government Printing Office, 1990.

National Institute on Alcohol Abuse and Alcoholism. *Eighth Special Report to the U.S. Congress on Alcohol and Health.* NIH Pub. No. 94–3699. Bethesda, MD: National Institutes of Health, 1994.

Spencer, G. *Projections of the Population of the United States, by Age, Sex, and Race: 1988 to 2080.* U.S. Bureau of the Census 1989 Current Population Reports Series P-25, No. 1018. Washington, DC: U.S. Government Printing Office, 1989.

Toomey, T.L.; Rosenfeld, C.; and Wagenaar, A.C. The minimum legal drinking age: History, effectiveness, and ongoing debate. *Alcohol Health Res World* 20(4):213–218, 1996.

U.S. Bureau of the Census. *Statistical Abstract of the United States 1992.* 112th ed. Washington, DC: U.S. Government Printing Office, 1992.

Zucker, R.; Boyd, G.; and Howard, J., eds. *The Development of Alcohol Problems: Exploring the Biopsychosocial Matrix of Risk.* National Institute on Alcohol Abuse and Alcoholism Research Monograph 26. NIH Pub. No. 94–3495. Bethesda, MD: National Institutes of Health, 1994.

SUMMARY OF RESEARCH ISSUES AND PRIORITIES

Chapter 26

Research Issues and Priorities

Edith S. Lisansky Gomberg, Ph.D., Andrea M. Hegedus, Ph.D., and Robert A. Zucker, Ph.D.

CORE RESEARCH PROBLEMS

Basic Definitions

A fundamental problem that leads all too frequently to an ambiguous and confusing literature is the lack of agreement on definition of terms. For example, "stress" may be defined by a list of negative life events, by subjective experience, or by exposure to an objectively described stimulus that is known to produce changes in autonomic system activity (e.g., the surprise presentation of a loud noise). This problem is not found in alcohol studies alone, but is common whenever the complexity of the phenomena necessitates some agreement on the concepts under study. In this context, how do we define "aging," "old age," and "the elderly"?

The definition of aging varies widely, perhaps because there is a good deal of heterogeneity in the aging process. Such heterogeneity has a longer time to play itself out as the population matures, so older people are likely to be more varied and heterogeneous than are other age groups. Thus, individual differences are more significant here than for younger populations.

At what point in the life course does a middle-aged person become an older person, or, to put it another

E.S.L. Gomberg, Ph.D., is professor of psychology in the Department of Psychiatry and Alcohol Research Center and adjunct professor of psychology in the School of Social Work, University of Michigan, 400 East Eisenhower Parkway, Suite 2A, Ann Arbor, MI 48108–3318. A.M. Hegedus, Ph.D., is administrative director at the University of Michigan Alcohol Research Center and assistant research scientist in the Department of Psychiatry, University of Michigan. R.A. Zucker, Ph.D., is professor of psychology in the Departments of Psychiatry and Psychology, University of Michigan, and is director of the University of Michigan Alcohol Research Center.

way, when is old age? The life stage chart presented by Zucker in chapter 1 offers one way to differentiate between midlife and later life. Other age categories are used in the application of governmental programs. For example, the social security systems of Western Europe and the United States are set up for people 65 and older, with some gender and national variation. This age was chosen by Bismarck in the 19th century, in devising one of the earliest social security systems. In the United States, the Federal Government tends to accept 65 as the cutoff point (e.g., National Institute on Alcohol Abuse and Alcoholism [NIAAA] 1994). Some census data analyses are based on age 60 as the cutoff point. This issue has been addressed by demographers. For example, Myers (1990), in discussing the dimensions of aging, commented:

> Another consideration that adds further complexity concerns the various definitions that may be used for delineating old age and thereby, the aged population. The demographer commonly uses a fixed age such as 60 or 65 as a boundary point, because it facilitates standardized analyses and is grounded in legal and conventional practice (e.g., in Social Security provisions, retirement decisions, etc.) It is interesting to note, however, that the practice of designating the age of 60 or the age of 65 may vary even among different agencies within the same parent organizations. (pp. 21–22)

Webster's Third New International Dictionary of the English Language Unabridged offers the following definitions of "elderly": "1: somewhat old: rather advanced in years: past middle age...2:... characteristic of one past the prime of life...." These definitions do not mention specific ages and so are not useful for research purposes. To study any issue in gerontology, we must agree on cutoff points for age groups so that research reports are comparable. From a review of research reports in two 1997 issues of the *Journal of Gerontology (Series B. Psychological Sciences and Social Sciences)*, we found that the ages of subjects varied from a mean age of 68 to a mean age of over 86 (table 1). The psychological studies were laboratory studies of multiplication, levels of learning, and so on. The social science studies included theory, policy, mortality statistics and other epidemiologic research. There were substantial age differences in subjects selected for experimental, clinical, and epidemiologic research. Experimental and epidemiologic studies are limited only by the existence or availability of older subjects. Clinical research is limited to those elderly persons who present with particular symptoms or syndromes; in alcohol studies, for example, the number of alcohol abusers and alcohol-dependent individuals drops sharply as people age, so research tends to be directed toward the earlier years of elderly status.

Early on, Neugarten (1968) distinguished between the "young-old"—those persons ages 60 through 74—and the "old-old"—those age 75 and over.

Table 1. Age Sampling of Research Reports in Two 1997 Issues of the *Journal of Gerontology (Series B)*.

Issue	Age Range	Mean Age	
March 1997			
Allen et al.	62–82	72.45	
Fisk et al.	63–82	70.51	
Pratt et al.	60–83	70.9	
Morrow et al.	—	68	
Lawrence et al.	60–75+	70.7	African-American
		69.2	Puerto Rican
		71.5	Caucasian
May 1997			
Gall et al.	61–75	69	
Tesch-Romer	51–87	71.2	
Johansson et al.	"oldest old"	86.85	
Mirowsky	50 +	—	
Resnick et al.	—	82.1	
Bernard et al.	65–85+	—	

The "oldest old" or "frail elderly" usually refers to people in their eighties or older. A question has been raised in the United States about redefining "middle age" as lasting from ages 55 to 75 (Arden 1987); it had previously been defined as lasting up to age 65. The "aged population" is defined culturally and politically and, to some extent, individually, but for research, the age of older subjects will vary with the questions raised, the methods used, and the availability of willing subjects. For biological and social scientists, comparison of results is only possible if each researcher defines the age parameters for his or her study using categories that have been agreed upon within the research community.

A final note on the older population: lay and popular definitions of the elderly as "senior citizens" are almost always younger than the social science definition. The American Association of Retired Persons, for example, admits to membership people who are 50 years of age and older. Merchandisers who offer senior discounts are likely to offer them at 62 or 60 or even 55. The message seems to be that where there is an economic motive for recruiting older persons, the definition of "elderly" is likely to move downward so as to be more inclusive.

Alcohol Use and Abuse Among the Elderly: Special Issues

Life-Course Variations in Drinking

Two questions concerning drinking patterns need to be posed. First, are the quantity and frequency of drinking different among the elderly? Second, what is the direction of change—that is, do they drink more, drink less, or become abstinent? It appears that the drinking patterns of middle age tend

to be carried on as people grow older, and it is now believed that some of the reported changes in drinking among older people may be part of a society-wide decrease in patterns of consumption (NIAAA 1994). The NIAAA report also raises the question of whether younger people who now drink more heavily than their parents and grandparents will carry those consumption levels into old age. In chapter 1, Zucker presents evidence for age trajectory differences among those now in middle adulthood and argues that increases in both consumption levels and drinking problems are forthcoming in the next generation. These conclusions about age trajectory differences are generally supported by cross-national, meta-analytic studies (Johnstone et al. 1996).

Gender and Racial/Ethnic Variability

All reports about alcohol use and abuse must take into consideration gender and racial/ethnic variations. Consistently—even in younger age groups—women begin drinking somewhat later and drink less than men do; they approach parity during high school and college years, but as young adults the differences in consumption reappear. Racial/ethnic differences are seen in the age trajectory of alcohol-related problems; for example, African Americans develop alcohol abuse later in life than whites (Caetano 1984; Herd 1988, 1990; see also chapter 4 in this monograph). The black/white differences in the age trajectory of problem drinking are also present in the Epidemiologic Catchment Area studies (Robins 1989). Caetano (1988) has reported on Hispanic patterns of alcohol consumption, and in chapter 1 Zucker notes the major differences in age trajectory of alcohol problems between the Hispanic, black, and white populations. Since drinking behaviors are also related to education, socioeconomic status, and other social variables, the complexity of alcohol-related behaviors of older people as it relates to these demographic variables cannot be exaggerated.

Reasons for Changes in Drinking Patterns With Aging

Many hypotheses have been offered to explain why there is a drop in the percentage of older persons who drink alcohol (Gomberg 1982). It is only recently that some information has been gathered (Gomberg et al. 1997). The responses of a sample interviewed in primary care physicians' offices showed that those who were former light drinkers and those who were former heavy drinkers had different reasons for stopping drinking. Health concerns and dislike of taste/effects ranked highest for former light drinkers, whereas former heavy drinkers described interventions, both formal and informal, as the primary reason for stopping. In an expanding population of older persons, it would be useful to know the relationship between health status, medication use, and decisions to drink.

Sensible Drinking

Defining the sensible use of alcohol is not a problem unique to older persons. In all studies that involve alco-

holic beverages, investigators define "moderate," "heavy," and "problematic" use. There has been a good deal written about "hazardous drinking," alcohol abuse and dependence, and alcoholism; concern about these definitions goes back many decades. In 1951 the World Health Organization Expert Committee on Mental Health presented definitions, but, as Adams points out in chapter 18, "varying definitions... have always plagued the literature." Even a term that seems as obvious as "binge" turns out to have different meanings for laypersons and the technical literature (Beglin and Fairburn 1992); the authors urged that the term "should be clearly defined by clinical practice" (p. 124).

When the two areas, alcohol and gerontology, converge, several questions can be raised. What is sensible drinking for older persons (Dufour et al. 1992)? Is heavier drinking more than 1 drink per day for older persons? How do we screen and find older alcohol abusers and what are "the unique problems" presented by the elderly heavy drinker (*Alcohol Abuse and Misuse Among the Elderly* 1992)? What are the risk factors? What are the treatments specifically suited for elderly alcohol abusers? Many of these questions were raised by Graham (1986) in her critique of studies of older alcohol abusers and are addressed in this monograph by both Adams (chapter 18) and Blow (chapter 21). Both contributors note the diversity of problem outcomes among the elderly, including some initially paradoxical ones. For example, as Adams notes, the relationship between problem drinking and health status among older persons is confounded by the fact that the cohort association tends to decrease with increasing age as drinking becomes restricted or eliminated by those with health problems.

More generally, it needs to be underscored that the diversity of outcomes affected by alcohol use is potentially larger as one becomes older, and definition of a boundary for abusive drinking needs to be differentially adjusted as a function of the research question and/or the clinical problem. Although the NIAAA (1995) "one drink a day" guideline is an excellent rule, there are circumstances involving medication interaction where even this figure is problematic, and there are likely other circumstances where the figure might be relaxed a bit. In this regard, Blow's notion of an armamentarium of treatments for the spectrum of alcohol use needs to be complemented with the notion of a spectrum of limits for problem use that vary both with age and with the medical issue that is co-occurring.

Early Onset and Later Onset

As early as 1964, one can find clinical accounts that differentiated earlier from later onset problem drinkers (i.e., Droller 1964). Later investigators (e.g., Rosin and Glatt 1971; Schuckit 1977) began to recognize the utility of a developmentally based distinction as a differentiator of both process and outcome for older drinkers. The *early onset/late onset* terminology has been applied to alco-

holics (Schuckit 1977) and problem drinkers (Brennan and Moos 1991). The age cutoff varies, but as in Schuckit's study, the distinction is usually made between older long-term alcoholics or problem drinkers whose alcohol problems started in their twenties or early adult years and older alcoholics or problem drinkers whose alcohol problems began late in life.

The question is, how do we define "late in life"? If we define late onset as 40 and older (as many have done), these elderly alcoholics may have had a 20-year history of problem drinking, even at age 60. The applicability of the distinction between early and late onset among older alcoholics is clearly seen in a report on Skid Row alcoholics:

> The men generally had been abusing alcohol for long periods of time. The moderate, heavy, or spree drinkers reported having begun such patterns on the average of 25 years ago. Noteworthy was that one fifth of these drinkers had begun such patterns relatively late in life (i.e., past 50). (Cohen and Sokolovsky 1989, p. 184)

But for other populations, the distinction becomes blurred. Atkinson (1987), in a literature review, commented that onset after 40 was most commonly cited as late onset. Rice and Longabaugh (1995) also used that cutoff point. However, Schonfeld and Dupree (1991) used 50 and older as the marker of late onset. In addition, as Atkinson and his colleagues noted, "data from elderly clinical samples demonstrate that onset of initial drinking problems at or after age 60 is common, occurring in 29% to 68% of cases in three recent series" (Atkinson et al. 1992). The early versus late onset distinction also reflects an important gender difference among older alcohol abusers (Gomberg 1995): a study of male and female elderly alcoholics in treatment showed 70 percent of the men reporting early onset (before age 30) compared with 17 percent of the older women. When asked about onset at age 40 or older, 12 percent of the men and 71 percent of the women responded affirmatively.

It appears that within the late onset category (i.e., symptomatology onset at age 40 and older) those alcoholics or problem drinkers who present with a 20- to 30-year history should be differentiated from those whose problems began more recently. This suggests that three categories of onset could be defined in future work, to more closely match the developmental literature of "younger" adult drinking, to more carefully differentiate between elderly drinkers whose symptomatology has been of relatively shorter versus longer duration, and to take better account of the life-cycle differences upon which the symptomatology is superimposed:

- early adult onset: onset up through age 35
- middle adult onset: onset between ages 36 and 59
- older adult onset: onset at age 60 or older

Even this categorization scheme ignores the younger adult literature, which has commonly used age 25 as a cutoff (cf. Irwin et al. 1990), but we believe it is a developmentally more

appropriate category system than the one that sets "late" problems at the halfway point of the life cycle.

The methodological issue of the accuracy of retrospectively determined symptoms is not a simple one and has been virtually unaddressed in this literature on early versus late onset. This is an important concern, given that characterizations of course are ostensibly being described here, yet they are to a degree heavily dependent upon the respondent's ability (and willingness) to recall past behavior. Given the cognitive deficits known to occur with aging (Craik 1986), the accuracy of these categorization schemas remains untested. We return to this issue in the next section.

DESIGN AND METHOD ISSUES

The biggest problem facing both biomedical and psychosocial researchers is the sizable heterogeneity of the aging population. In a classic article on human aging written a decade ago, Rowe and Kahn commented on this issue: "Research in aging has emphasized average age related losses and neglected the substantial heterogeneity of older persons" (Rowe and Kahn 1987, p. 143).

Originally, research on alcohol and aging was done with clinical samples (Gomberg 1980); older groups were well represented in hospitals and clinics. With the developing methodological sophistication gained by epidemiologic study of entire populations, an expanded knowledge base has developed that sometimes has challenged the generalizability of clinical studies and has expanded our horizons about the nature of the aging hypotheses to be explored (Baltes 1997). For example, the deterioration of cognitive processes among the elderly was a widely held belief, but recent work in this area has suggested that some functions are more affected by aging than others (Park et al. 1996). In a review of aging and cognition, Moscovitch and Winocur (1992) combined contemporary cognitive theory and neuropsychology in their discussion of memory problems, set shifting, and problem solving. In chapter 6 of this monograph, Kalant discusses effects of age × alcohol interactions on attention, central information processing, learning and memory, and other components of psychomotor performance. Nixon (chapter 12) points out that even when impairment occurs, it is not of uniform severity among the elderly. Research is needed on those functions that remain relatively unimpaired or that even improve. These issues are inextricably linked with the study of alcohol and aging effects.

Another important issue has to do with data collection. Some studies of elderly drinking behavior deal with changes over time (e.g., Dunham 1981), but even those that deal with current drinking still require history taking to establish subject categorizations. All such nonlongitudinal survey research by definition deals with retrospective recall, and there is an inherent potential for errors in such data collection. However, as pointed out by Herzog in chapter 2 of this monograph, these errors appear to be no worse for

older respondents than for younger ones. Still, the need for prospective studies is great because no general knowledge base yet exists that will reliably identify the intervals and the content base within which we can be secure that recalled data are undistorted.

SELECTED CONTENT FOCI

Research on Alcohol Abuse and Dependence (Alcoholism)

Withdrawal

Reports agree that alcohol withdrawal is more severe among older patients, with symptoms of greater severity and duration (Liskow et al. 1989; Brower et al. 1994). However, it is not well understood how withdrawal differs among the elderly and how the varying severity relates to alcohol dependence. In chapter 20, Brower provides a review of the literature and discusses explanatory mechanisms and treatment implications. His discussion highlights a number of anomalies and still-unanswered questions: If withdrawal is more severe, why are seizures or DTs no more common in elderly compared with younger alcoholics? Given the generally increased withdrawal severity found among the elderly, is outpatient detoxification still a therapeutic option for some? If so, what are the criteria? To what degree are shorter versus longer acting benzodiazepines more optimal in the treatment of withdrawal, given the likely interaction with elderly cognitive impairment? Research to define the parameters in all of these areas is needed.

Lifetime Drinking Trajectories and the Understanding of Stability and Change

There may be useful strategies for the typing or classification of elderly problem drinkers that do not involve psychiatric comorbidity (see chapter 23); one obvious candidate involves distinctions in lifetime history of alcohol use and abuse. The issue of onset has already been discussed in this chapter, and questions about the roles of genetics, stress, partner drinking, and other factors have been raised throughout this monograph. But heavy drinkers do not necessarily drink heavily or problematically throughout their lifetimes, and it would be valuable to know more about intermittent bouts of heavy or problematic drinking. This knowledge might permit us to identify warning signals to use in elderly prevention programs; it also would focus attention on an issue that is not yet well understood, concerning the possible instability of the alcohol-related diagnosis across long spans of time (Zucker et al. 1997).

Longitudinal studies of alcohol use into the older years have been rare. Vaillant (1996) reported a study of male alcohol abusers followed from age 40 to 60–70 years. Two groups were studied: a college sample and a core city sample. For both groups, relapse was rare after 5 years of abstinence, but controlled drinking without relapse was unlikely after the same period of abstinence. Onset of drinking problems was earlier for the core city men, and although they were more likely to

become alcohol dependent, they were twice as likely to achieve stable abstinence. Alcohol abuse in both groups continued for decades with neither remission or progression.

What is needed is more longitudinal study of respondents who drink within normal limits to address the question of age changes in drinking behaviors. A number of existing national databases (e.g., Monitoring the Future, Project AHEAD) are valuable resources within which to explore such issues, but researchers have not yet taken advantage of these resources.

Comorbidity and Classification

The relationship between psychiatric comorbidity and classification of alcohol disorders has been extensively explored among younger age groups, but it is not known whether the same patterns of comorbidity exist among older problem drinkers. One study of the Epidemiologic Catchment Area data by Mellow and colleagues (unpublished manuscript) indicates that, not surprisingly, the prevalence of type II, or antisocial, alcoholics drops very substantially among elderly alcoholic men, and this type is virtually nonexistent among alcoholic women. This work is only the tip of the iceberg and needs to be substantially expanded. In contrast to the decreased prevalence of type II alcoholism, suicide rates rise among older men; the motives and situations that drive this depression-related phenomenon also need to be understood.

Given the prominence of medical disorders among the elderly, a reasonable hypothesis is that some medical disorders will actually influence the presentation and clinical course of alcohol disorders in a synergistic manner. Therefore, when studying comorbidity in relation to alcohol disorders in the elderly, it seems logical that researchers should focus on the presence or absence of medical disease as well as psychiatric symptomatology.

Treatment

Despite a fair amount of publication on the topic, much research still needs to be done on treatment issues. Should benzodiazepines be used? Antabuse? Elder-specific treatment groups? Alcoholics Anonymous? Some examples of work on diagnosis and treatment practices with older patients can be found in papers by Curtis and colleagues (1989), Fitzgerald and Mulford (1992), and Moos and colleagues (1993) and in a book by Graham and colleagues (1995). This monograph contains a section on treatment and prevention, with chapters on recurring treatment issues, brief intervention, natural recovery, treatment of comorbidity, and the question of age-specific treatment (chapters 18–24). Many significant research questions remain, and, despite the equivocal nature of the recent Project MATCH findings (Project MATCH Research Group 1997), the issue of what kinds of elderly patients do best with what kinds of treatments is among the most important. In particular, the degree to which elder-specific treatments are warranted, and the condi-

tions under which this might be true, is an essential question that needs focused study.

Case-Finding Issues

It is noted frequently that elderly alcoholic patients are "significantly less likely to be diagnosed" (Curtis et al. 1989, p. 310) by physicians and hospitals. Although emergency department staff and legal authorities probably encounter a number of elderly problem drinkers, few are sensitized to or trained to detect alcohol-related problems. The American Medical Association has been attempting to alert general practitioners to the problem of underdiagnosis and the need to be more aware of alcohol-related problems. It is relevant to note that when the University of Michigan Alcohol Research Center sought problem drinkers in treatment, the cutoff age was reduced to 55 because of the difficulty in finding cases. It has been hypothesized that the older generation feels more shame about admitting to a drinking problem, but the issue is more complex than that. Finding problem drinkers, whether in treatment or not (most likely in hospitals), is a solvable task, which to a degree harkens back to the definitional problem of specifying what is and is not elderly problem drinking; this area needs more attention.

Prevention Issues

As observed by Boyd in chapter 25, there are virtually no randomized controlled intervention studies for elderly alcohol abuse prevention. The issue of strategies in prevention characterizes the whole age range. The likelihood that strategies will be effective here will require attention to the specific issues to be addressed among the elderly, as well as the utilization of already proven techniques from younger subpopulations. The effectiveness of prevention program tailoring will depend on the acquisition of more knowledge about the range of drinking/problem drinking of older people, the contexts where it is likely to be more or less problematic (e.g., when driving, in interaction with specific medical disorders, or with specific medication regimens), and the setting where it is likely to be most cost-effective to implement.

Biomedical Research

Genetics

McClearn (chapter 5) provides a comprehensive discussion of genetics, aging, and alcohol use, and Bucholz and colleagues (chapter 3) present new Australian twin data that likewise address heritability issues. McClearn's review points to a genetic role, not only in abusive use of alcohol, but also in alcohol consumption. His conclusions: between one-third and two-thirds of population variability in quantity and frequency of alcohol use is probably heritable, and there is a decline in heritability of alcohol use as people grow older. McClearn's conclusions are supported by the results for women in the Bucholz et al. study but are at apparent odds with the Australian data for men, for whom the proportion of additive genetic variance increased with increasing age. These discrepant findings ultimately need to be reconciled.

The study by Bucholz and colleagues is a contribution to an increasing body of developmental genetic studies (e.g., Elkins et al. 1997) that are beginning to specify both the age-stage and environment-specific parameters under which genetic mediation of drinking (and abuse) takes place.

Effects of Alcohol on Aging Organisms

The effects of alcohol on older persons have been summarized previously (Gomberg 1990; Dufour et al. 1992). The same alcohol doses produce higher blood alcohol concentration in older persons than in younger persons. The evidence for age differences in alcohol metabolism, absorption, and elimination is not clear, although investigators describe older organisms as "more sensitive" to alcohol effects. Distinctions need to be made in the physiological effects of alcohol on normal, moderate, and heavy consumers of alcohol. There is an extensive literature on cerebral and neurological impairments among alcoholics (see chapters 7–10).

Of some interest are several reports of the role of first-pass metabolism and gastric alcohol dehydrogenase activity in the effects of alcohol. Although Frezza and colleagues (1990) reported gender differences, a more recent report indicates that among older subjects first-pass metabolism is low for both men and women and the gender difference does not appear (Pozzato et al. 1995). The reduction in gender gap is possibly due to effects of aging on metabolism.

One final point needs to be made regarding the effects of alcohol on older persons. The neuropathological literature has typically regarded impairments of severe and chronic alcoholism to be a product of severe and long-term alcohol use. Recent work by the Gilman group suggests that the sites of impairment may themselves have been differentially vulnerable in alcoholics, perhaps because of genetic differences in site affinity for ethanol. This work indicates a possible interaction between ethanol as a traumatic agent and diathesis variability of the host. Insofar as this work can be confirmed it suggests a more interactive model of insult, even for the neuropathology of elderly alcoholics.

Alcohol-Drug-Medication Interactions

The scientific evidence for the interaction between alcohol and prescription drugs and alcohol and over-the-counter drugs has been summarized by Dufour, Archer, and Gordis (1992), and the social importance of this problem has aroused enough interest that the American Association of Retired Persons and the Hazelden Foundation have combined to produce a warning pamphlet (Alcohol, Medications and Older Adults 1995). Older persons are more likely to be prescribed multiple medications, and if they mix alcohol with medications, this is a potential area of significant danger. In addition, the consequences of psychoactive drug use combined with alcoholic beverages are of special importance for women, who are bigger users of prescribed psychoactive drugs. More generally, considering the increased use of

medications among older subgroups, understanding these interactions is a research priority of the first order. Because such research presents many methodological problems, the area has been relatively neglected, yet it is one with obvious and very important clinical implications.

Medical Consequences of Moderate and Heavy Drinking

The medical consequences of heavy drinking have been summarized previously by Dufour, Archer, and Gordis (1992); they discussed effects on digestion, heart function, and pain relief. In this monograph, data on the medical consequences of heavy drinking are presented by Finlayson and Hurt in chapter 11 and by Adams in chapter 18. Several of the chapters in the Biological Mechanisms section of this volume deal with neuropathology and central nervous system effects from alcoholism.

It has, of course, long been known that heavy drinking has detrimental medical consequences. What is still open for debate, however, is the question of long-range effects of more moderate drinking. Klatsky and colleagues (1990) studied the risk of cardiovascular mortality in current alcohol drinkers, ex-drinkers, and nondrinkers. More recently, Hanna and colleagues (1997) reported on the relationship between drinking and heart disease morbidity from the National Health Interview Survey. Their findings, which are consistent with many other studies, suggest that protection from heart disease occurs only at lower levels of drinking.

Adams and her colleagues (1993) studied Medicare records to ascertain alcohol-related hospitalizations of elderly people and reported such hospitalizations as "common," with rates similar to those for myocardial infarction. Although the highest rate of hospitalization was in the 45- to 64-year-old group, the rate for the 65-and-over group ranked second. We are currently exploring, at the University of Michigan Alcohol Research Center, the relationship between current health status and a lifetime history of moderate drinking, heavy drinking, or abstinence. There is much work yet to be done in this area.

Interactions of Alcohol and Nicotine

Alcohol and tobacco most often are used at the same time; drinkers are more likely to be smokers, and there is reason to believe that some of the consequences of alcohol intake are linked to smoking. This problem has been addressed in an NIAAA Research Monograph (Fertig and Allen 1995). We are exploring the question of the role of smoking in the health of elderly persons (Gomberg et al. 1997): those with a past history of heavy drinking report significantly more smoking and significantly poorer health than those with lifetime moderate drinking or abstinence. This relationship between drinking and smoking should be further explored in research on the health of the elderly.

Behavioral/ Psychological Research

Demographics Relating to Elder Alcohol Use and Abuse

The question of which elderly subgroups continue to drink moderately,

continue to drink heavily, or remain abstinent has been studied by epidemiologists. Starting with Cahalan, Cisin, and Crossley's (1969) report of American drinking practices, which showed 47 percent of those 60 and older to be abstainers and 6 percent of respondents in that age group to be heavy drinkers, there have been many reports on the demographics of elderly alcohol use, and these studies have become increasingly sophisticated. Chapter 3 in this monograph presents alcohol consumption research data from both cross-sectional and longitudinal research. As in all epidemiologic studies reported in the literature, there are more abstainers among elderly women and more heavy drinkers among elderly men.

The relationship of drinking status to employment, marital status, residence, and other variables among older persons needs further exploration. A report about older women (Wilsnack et al. 1995), for example, showed higher education or income related to the greater likelihood of alcohol use (as it is in all age groups of women). On older women's primary relationships, they commented:

> There has been remarkably little research on how older women drinkers relate to partners in ongoing relationships, and very little is known about problems in these relationships. (p. 279)

Behavioral Effects of Alcohol on Younger Versus Older Subjects

The literature on behavioral effects seems to be concentrated largely on animal subjects. When older and younger individuals are compared, it is mainly elderly versus younger problem drinkers (e.g., Fitzgerald and Mulford 1992). The *American Journal of Public Health* issue of June 1993 contains no less than eight articles on effects of alcohol, but they are, again, devoted to problem drinking, abuse, and dependence. Although these studies are important, there also needs to be a research focus on the effects of light to moderate drinking. Some investigators, interested in cognitive changes, have developed a hypothesis about "premature aging" among heavy drinkers (Tarter 1995). Studies in cognitive effects of alcohol seem to be one avenue of research that would stimulate study of age differences in behavioral effects.

Cognitive Studies

In spite of the many chapters devoted to cognition in the volume by Birren and Schaie (1990), there was no mention at all about the effects of alcohol or any other drug on elderly cognition. Schaie (1990) emphasized individual patterns of change and antecedent variables to account for the sizable individual differences in cognitive abilities. Salthouse (1990) was concerned with "cognitive competence," success in functioning with the environment—surely a real issue relating to alcohol effects. More needs to be known about the cognitive effects of heavy drinking, moderate drinking, and social drinking and how they may interact with age-related changes in specific cognitive processing functions (Park et al.

1996). Some of these questions are raised by Nixon in chapter 12.

Effects of Alcohol on Sleep and Driving

Aldrich reports on effects of alcohol on sleep in chapter 16, and Waller reports on the relationship between alcohol and driving in chapter 17. These issues are particularly relevant to older persons because of the widespread complaint of insomnia among the elderly, because of the overrepresentation of sleep disturbances among alcoholics, and because changes (decrements) in the driving performance of aging drivers have major public health implications. Both of these content areas have been relatively unexplored, and both of these research groups have uncovered major new findings. This work needs to be continued and expanded.

Stress, Coping, and Adaptation, and Their Relationship to Alcohol Use

Stress has been a popular concept with social scientists. Despite the fact that stress must be conceptualized as an aspect of the environment, as a subjective response, or as a combination of both, this differentiation is often ignored in the extensive literature. Current developments in research on stress and alcohol were presented at a symposium of the Research Society on Alcoholism in 1995 (Ragland and Ames 1996), and the results are not reassuring. Linkages between "stress" (job satisfaction, negative life events) and alcohol use and abuse were weak or nonexistent. One report (Hill et al. 1994) showed a link between childhood exposure to alcohol problems and physical abuse and later years of heavy alcohol use.

Stress, in one guise or another, has played a role in several explanatory theories about the etiology of alcohol use and abuse, including tension reduction theory (Cappell and Greeley 1987) and stress-dampening theory (Sher 1987). More recent literature on earlier life indicates a substantial, albeit complex set of relationships. Despite the promise in this literature on younger people, studies of the relationship between stress and alcohol use and abuse among older adults have not been productive. In one study of the everyday, moderate use of alcohol and other psychoactive drugs in relation to stress and coping of older adults (Huffine et al. 1989), no relationship was found between use of drugs and alcohol and the way subjects "appraised and coped with stressful encounters" (p. 101). Welte's summary of the stress literature in chapter 13 of this monograph also indicates that clinical research among older adults has yielded ambiguous results, and the epidemiologic data do not support a relationship between stress and alcohol use. The search for stress variables as related to late-life alcoholism may need to return to an early classification of elderly alcoholics as "survivors" (early onset), those with intermittent episodes of problem drinking, and the "reactive problem drinkers" who are responding to "the stresses and losses of aging" (Gomberg 1980 [p. 12], 1982 [p. 275]).

More generally, this research arena has not yet developed the definitions or the methods that support a linkage between the stresses of aging and late-life alcoholism. There continues to be a simplistic assumption that stress in the older years is somewhat related to elderly alcohol abuse, particularly in later onset and recent-onset drinkers. To study this relationship effectively, it is essential to study the situations and events perceived as stressful by the elderly, and then to examine the coping mechanisms, including the use of alcohol as a means to deal with the perceived stress. As noted in a comprehensive critical review of stress and coping among older adults by Lazarus and DeLongis (1983), "many of the age-related changes in sources of stress and positive feeling result, in part, from a shift in how events are appraised by the person rather than solely from changing circumstances" (p. 248).

Special Issues Relating to Alcohol Abuse in the Elderly

Elder Abuse and Neglect

The issue of elder abuse and neglect has been raised frequently in recent Gerontological Society meetings. Although there is a sizable professional literature on the subject, the linkage between alcohol and elder abuse and neglect is only occasionally mentioned in these writings. Two papers that do address this linkage are by Anetzberger and colleagues (1994), who report on alcoholism per se and elder abuse, and by Hwalek and colleagues (1996), who report on the association of elder abuse and "substance abuse" in an Illinois program. The latter report does not specify which substances were involved, but, considering the age group of both abuser and victim, it is likely to have been alcohol. It should be noted that the older person, the "victim," was reported to be a substance abuser in 9 percent of cases.

As the elderly population increases, as the resource structure for caregiving becomes more taxed, and as the proportion of frail elderly increases in this population, it can be anticipated that this issue will become greater in the population at large. A substantial research agenda is thus warranted, to define the parameters of situations and individuals at highest risk for such assault and to establish effective guidelines for prevention, early intervention, and remediation of this trauma.

Homelessness

As the problem of homelessness has expanded, attention given by alcohol researchers to the older Skid Row alcoholic has diminished. It is true that the population characterized as homeless is more diverse than it once was; however, the homeless, street alcoholic still exists (Cohen and Sokolovsky 1989; Rubington 1995). Cohen and Sokolovsky (1989) estimated that 75 to 80 percent of the men living in Bowery flophouses whom they studied were over age 50. This marginal subpopulation is one with high psychiatric comorbidity and, almost by definition, is significantly disadvantaged in social resources. Thus, an understanding of the role that alcohol may play in sus-

taining a homeless adaptation, as well as contributing to entry into it, remains a significant social as well as scientific concern. Here, as with many other problems of the elderly, focused minilongitudinal study designs are needed to establish order of precedence of such effects.

Underrepresentation of Older Persons With Alcohol Problems in Treatment Settings

It has been well documented that older persons are underrepresented in their use of community mental health resources. It is, therefore, not surprising that older persons are underrepresented in substance abuse treatment facilities even when counted against their relatively smaller numbers. This underrepresentation is, of course, in sharp contrast to the increased use of medical services by older persons. The stigma of presenting with an alcohol problem has been offered as a frequent explanation for this phenomenon, and some clinicians suggest that older people should not be confronted directly with their alcohol or drug problem.

The 1994 NIAAA report to Congress suggested a number of explanations for the underrepresentation of older persons in alcohol treatment settings, including detection problems relating to the physiological effects of alcohol being mistakenly attributed to aging, chronic disease, or memory difficulties. The report indicated the need for different criteria for defining problem drinking in older persons. The relatively small number of older adults presenting at alcoholism treatment facilities (Rush and Timney 1985) continues to be observed in newer studies (e.g., Rice and Duncan 1995), and clinicians' misconceptions about ease of detection also continue. Rice and Duncan hypothesized that alcohol consumption would be positively related to health service use among older adults, but their results showed "harmful" use of alcohol in older adults to be negatively associated with physician visits, even after adjusting for respondent gender and health status measures (Rice and Duncan 1995). The conclusion: problematic use of alcohol is going undetected and undiagnosed. In an effort to address this problem, in 1995 the American Medical Association issued guidelines for primary care physicians, entitled *Alcoholism in the Elderly: Diagnosis, Treatment, Prevention.*

Sociological/ Anthropological Research

Family Cohesiveness

Although there are current changes in the family patterns of Americans related to immigration, fertility, and geographic mobility, it is still estimated that more than a third of older Americans will, at some point, live with an adult child. Such living arrangements constitute one aspect of the diversity and heterogeneity of family relationships (Bengtson et al. 1990). Family relationships and patterns will vary with age, marital status, childbearing, socioeconomic status, and ethnicity as well as other factors. Older white males have the highest probability of being married and elderly, black women the least. Black elderly are more likely to be single, separated, or divorced than white elderly.

Cohesive family patterns distinguish white Americans from racial/ethnic minorities, which show, in comparison to the general population, larger household sizes, more intergenerational households, more frequent family contact, more filial piety, and more family support exchanges (Jackson et al. 1990). How do these patterns relate to problem drinking in the elderly? It is a reasonable hypothesis that family patterns will be more fractured in the case of early-onset problem drinkers than in the case of those whose heavy/problem drinking began later in life. However, to our knowledge, no one has investigated this issue. There has been some comparison of marital status of male/female and black/white elderly alcoholics (Gomberg 1995; Gomberg and Nelson 1995). Related, perhaps, to earlier and later onset drinking, more male patients were separated or divorced, while more female patients were widowed. There were no significant differences between black/white patients in marital status. Considering the role of the family in the development and treatment of alcohol problems, this is an area of study that merits more research attention than it has received.

Adaptation to Changing Role and Status

This issue of adaptation to changing role and status has appeared in the research on elderly problem drinking, with some interest in the role of retirement and widowhood and its relationship to alcohol use and abuse. Results on retirement are mixed; those treatment facilities that are used by higher income persons do report some relationship between retirement and increases in drinking, but generally blue-collar workers seem to drink less when no longer working. This area merits more study. Results on widowhood are also mixed, as reported by Wilsnack and her colleagues (1995). Some studies show widows drinking less than older married women; in general, few measures of alcohol abuse seem related to the marital status of older women. The pattern called "alcoholism à deux" (i.e., wife drinking with husband) is probably more frequent, but that observation, too, awaits verification.

Social Networks and Social Supports

The association between lack of social relationships and increased morbidity/mortality is "fairly well established" (Antonucci 1990), although it has not been explored with elderly problem drinkers. The assumption that elderly heavy drinkers or problem drinkers are isolates, which is based on the image of the Skid Row alcoholic, is probably not true. There are many groups, particularly men, who drink together in public and/or in private places. Women, younger and older, are less likely to drink in groups in public places.

One obvious social network is Alcoholics Anonymous. In our current study of an older population interviewed in physicians' offices (Gomberg et al. 1997), almost 20 percent of current abstainers identify themselves as formerly heavy drinkers or alcoholics; of these almost 90 percent attribute the cessation of their problem drinking to

Alcoholics Anonymous. The fellowship of Alcoholics Anonymous has made a particular appeal to older alcohol abusers, to the extent of publishing, with the Hazelden Foundation, *Letters to an Elderly Alcoholic* (Carle 1980). There are research questions, not only about Alcoholics Anonymous participation, but also about social networks in general: Are disrupted social networks a cause or an effect of hazardous drinking, or both?

Drinking in Retirement Communities

Although there is anecdotal evidence of increased drinking in some retirement communities, there is limited research in the area. Alexander and Duff (1988) reported on drinking in retirement communities, and this report has had some impact on gerontologists interested in elderly drinking. It would be useful to know more about the nature of the retirement communities that facilitate increased drinking; it would help make prevention and intervention efforts more effective.

Cross-Cultural Comparison: Permission To Drink With Aging

In a report on alcohol use and abuse among Chinese Americans (Yu et al. 1989), the authors described two categories of persons depicted as drinkers in Chinese classical literature and in daily life: the "literati or gentry class" and "the elderly, for whom drinking is tolerated ostensibly for health reasons" (p. 337). Although the elderly Chinese Americans interviewed reported that they flushed and showed other physical reactions, they continued to drink.

A World Health Organization study of drinking norms in Scotland, Mexico, and Zambia asked respondents about permission to drink at various ages (Roizen 1981). The highest percentage of respondents of all three nationalities gave permission for men age 40: 99 percent in Scotland, 83 percent in Mexico, and 88 percent in Zambia. For men age 60, 97 percent of Scots, 63 percent of Mexicans, and 77 percent of Zambians gave permission to drink. For women age 60, 60 percent of Scots, 61 percent of Mexicans, and 65 percent of Zambians gave permission to drink. It remains for cross-cultural and cross-national surveys to study which countries lessen the sanctions for elderly drinking and which countries view permission to drink as a reward of aging.

Government Policies Regarding the Elderly

Government policy is a well-developed field of interest in gerontology, and there are a number of questions relevant to alcohol and aging. For example, to what extent do driving-while-intoxicated drivers fall into the 60-and-older age group? What are the implications of age-related, accelerated decrements in driving performance with alcohol use for driving-while-impaired standards? Of Medicare's financial problems? Of research on age-related diseases (e.g., Alzheimer's)? Of the need for health care among the aging veteran population and the aging incarcerated population? And so on.

Stigmatization and Spoiled Identity

Although this is a largely theoretical area, stigmatization and spoiled identity may be factors in elderly alcohol abuse. The United States, with its emphasis

on youth culture, is perceived to be oriented negatively toward the elderly. Although this view may be exaggerated, it is true that our society has not defined useful social roles for elderly men and women. In most agricultural societies, men do not retire until they can no longer work; older women in many societies are assigned caretaking of the children while younger women work. Patterns of work, retirement, leisure, and time to fill merit study to examine the relationship of these variables to alcohol use and abuse among older people.

Role of Religion and Spirituality

When elderly respondents were queried about their reasons for current abstinence (Gomberg et al. 1997), religious objections ranked third, after health reasons and dislike of the effects. A difference was reported between black and white elderly alcoholics in strength of religious beliefs (Gomberg and Nelson 1995). Asked about the importance of religious or spiritual beliefs in their everyday lives, 74 percent of the black men said that it was very important, compared with 44 percent of the white men. Substance abuse therapists are currently paying a good deal of attention to the role of belief systems and spirituality among their clients, and it seems an important area for further exploration.

CONCLUSION

Since gerontology is a relatively new area of interest, developing as it has in response to the growing proportion of older people in the population, there are a plethora of unanswered questions. For those clinicians and researchers interested in the entire lifespan, for those interested in biomedical or psychological or social issues, there are questions everywhere. That is both a frustration and an incredible challenge.

The conference reported in this monograph shows major gains over what was known in 1983. We know more about the effects of alcohol on the aging individual, including the effects on the brain and on interpersonal interactions. Perhaps most important, the definitions of aging and the models of process now used have changed, and these new or revised definitions and models inform a new generation of research designs and analytic questions. Models linking neurobiology and behavior are increasingly being developed, and the two-way interaction between the environment as a structure and the brain as a structure, and their mutual dependency and interrelationship, is beginning to be documented. But there are many questions yet to be answered, and we have a long way to go.

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REFERENCES

Adams, W.L.; Yuan, Z.; Barboriak, J.J.; and Rimm, A.A. Alcohol-related hospitalizations of elderly people. *JAMA* 270:1221–1226, 1993.

Alcohol Abuse and Misuse Among the Elderly. Hearing Before the Select Committee on Aging of the House of Representatives, 102nd Congress. U.S. Government Printing Office, 1992. (testimony of Enoch Gordis)

Alcohol, Medications and Older Adults. How To Get Help [Pamphlet]. American Association of Retired Persons/Hazelden, 1995.

Alexander, F., and Duff, R.W. Drinking in retirement communities. *Generations* XII:58–61, 1988.

Allen, P.A.; Smith, A.F.; Jerge, K.A.; and Vires-Collins, H. Age differences in mental multiplication. *J Gerontol* 52B:P81–P90, 1997.

American Medical Association Department of Geriatric Health. *Alcoholism in the Elderly: Diagnosis, Treatment, Prevention. Guidelines for Primary Care Physicians.* Chicago: the Association, 1995.

Anetzberger, G.J.; Korbin, J.E.; and Austin, C. Alcoholism and elder abuse. *J Interpersonal Violence* 9:184–193, 1994.

Antonucci, T.C. Social supports and social relationships. In: Binstock, R.H., and George, L.K., eds. *Handbook of Aging and the Social Sciences.* San Diego, CA: Academic Press, 1990. pp. 112–129.

Arden, E. Ready, willing, able and retired. *Michigan Alumnus* 93:32–34, 1987.

Atkinson, R.M. Alcohol problems of the elderly. *Alcohol Alcohol* 22:415–417, 1987.

Atkinson, R.M.; Ganzini, L.; and Bernstein, M.J. Alcohol and substance-use disorders in the elderly. In: *Handbook of Mental Health and Aging.* 2d ed. San Diego, CA: Academic Press, 1992. pp. 515–555.

Baltes, P.B. On the incomplete architecture of human ontogeny: Selection, optimization, and compensation as foundation of developmental theory. *Am Psychol* 52:366–380, 1997.

Beglin, S.J., and Fairburn, C.G. What is meant by the term "binge"? *Am J Psychiatry* 149:123–124, 1992.

Bengtson, V.; Rosenthal, C; and Burton, L. Families and aging: Diversity and heterogeneity. In: Binstock, R.H., and George, L.K., eds. *Handbook of Aging and the Social Sciences.* San Diego, CA: Academic Press, 1990. pp. 263–287.

Bernard, S.L.; Kincade, J.E.; Konrad, R.R.; Arcury, T.A.; Rabiner, D.J.; Woomert, A.; DeFriese, G.H.; and Ory, M.G. Predicting mortality from community surveys of older adults: The importance of self-rated functional ability. *J Gerontol* 52B:S155–S163, 1997.

Birren, J.E., and Schaie, K.W. *Handbook of the Psychology of Aging.* 3d ed. San Diego, CA: Academic Press, 1990.

Brennan, P.L., and Moos, R.H. Functioning, life context, and help-seeking among late-onset problem drinkers: Comparisons with nonproblem and early-onset problem drinkers. *Br J Addict* 86:1139–1150, 1991.

Brower, K.J.; Mudd, S.; Blow, F.C.; Young, J.P.; and Hill, E.M. Severity and treatment of alcohol withdrawal in elderly versus younger patients. *Alcohol Clin Exp Res* 18:196–201, 1994.

Caetano, R. Ethnicity and drinking in Northern California: A comparison among whites, blacks and Hispanics. *Alcohol Alcohol* 19:31–44, 1984.

Caetano, R. Alcohol use among Hispanic groups in the United States. *Am J Drug Alcohol Abuse* 14:293–308, 1988.

Cahalan, D.; Cisin, I.H.; and Crossley, H.M. *American Drinking Practices.* Monograph 6. New Brunswick, NJ: Rutgers Center of Alcohol Studies Publications Division, 1969.

Cappell, H., and Greeley, J. Alcohol and tension reduction: An update on research and theory. In: Blane, H.T., and Leonard, K.B., eds. *Psychological Theories of Drinking and Alcoholism.* New York: Guilford Press, 1987.

Carle, C.E. *Letters to an Elderly Alcoholic.* Center City, MN: Hazelden Foundation, 1980.

Cohen, C.I., and Sokolovsky, J. *Old Men of the Bowery: Strategies for Survival Among the Homeless.* New York: Guilford Press, 1989.

Craik, F.I.M. A functional account of age differences in memory. In: Klix, F., and Hagendorf, H., eds. *Human Memory and Cognitive Capabilities.* Amsterdam: North-Holland, 1986. pp. 409–422.

Curtis, J.R.; Geller, G.; Stokes, E.J.; Levine, D.M.; and Moore, R.D. Characteristics, diagnosis and treatment of alcoholism in elderly patients. *J Am Geriatr Soc* 37:310–316, 1989.

Droller, H. Some aspects of alcoholism in the elderly. *Lancet* 13:137–139, 1964.

Dufour, M.C.; Archer, L.; and Gordis, E. Alcohol and the elderly. *Clin Geriatr Med* 8:127–141, 1992.

Dunham, R.G. Aging and changing patterns of alcohol use. *J Psychoactive Drugs* 13:143–149, 1981.

Effects of Alcohol. *Am J Public Health* 83:811–861, 1993.

Elkins, I.J.; McGue, M.; and Iacono, W. Genetic and environmental influences on parent-son relationships: Evidence for increasing genetic influence during adolescence. *Dev Psychol* 33:351–363, 1997.

Fertig, J.B., and Allen, J.P., eds. *Alcohol and Tobacco: From Basic Science to Clinical Practice.* National Institute on Alcohol Abuse and Alcoholism Research Monograph 30. NIH Pub. No. 95–3931. Bethesda, MD: National Institutes of Health, 1995.

Fisk, A.D.; Rogers, W.A.; Cooper, B.P.; and Gilbert, D.K. Automatic category search and its transfer: Aging, type of search and level of learning. *J Gerontol* 52B:P91–P102, 1997.

Fitzgerald, J.L., and Mulford, H.A. Elderly vs. younger problem drinker "treatment" and recovery experiences. *Br J Addict* 87:1281–1291, 1992.

Frezza, M.; DiPadova, C.; Pozzato, G.; Terpin, M.; Baraona, E.; and Lieber, C.S. High blood alcohol levels in women. *N Engl J Med* 322:95–99, 1990.

Gall, T.L.; Evans, D.R.; and Howard, J. The retirement adjustment process: Changes in the well-being of male retirees across time. *J Gerontol* 522: P110–P117, 1997.

Gomberg, E.S.L. *Drinking and Problem Drinking Among the Elderly.* Ann Arbor: University of Michigan Institute of Gerontology, 1980.

Gomberg, E.S.L. Drugs, alcohol, and aging. In: Kozlowski, L.T.; Annis, H.M.; Cappell, H.D.; Glaser, F.B.; Goodstadt, M.S.; Israel, Y.; Kalant, H.; Sellers, E.M.; and Vingilis, E.R., eds. *Research Advances in Alcohol and Drug Problems.* Vol. 10. New York: Plenum Press, 1990. pp. 171–213.

Gomberg, E.S.L. Older women and alcohol use and abuse. In: Galanter, M., ed. *Recent Developments in Alcoholism.* Vol. 12. New York: Plenum Press, 1995. pp. 61–79.

Gomberg, E.S.L., and Nelson, B.W. Black and white older men: Alcohol use and abuse. In: Beresford, T.P., and Gomberg, E.S.L., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 307–323.

Gomberg, E.S.L.; Walton, M.A.; Bandekar, R.; Coyne, J.M.; and Blow, F.C. Alcohol and elderly health: Gender differences for lifetime abstainers versus former drinkers. *Alcohol Clin Exp Res* 21:24A, 1997 (abstract).

Graham, K. Identifying and measuring alcohol abuse among the elderly: Serious problems with existing instrumentation. *J Stud Alcohol* 47:322–326, 1986.

Graham, K.; Saunders, S.J.; Flower, M.C.; Timnery, C.B.; White-Campbell, M; and Pietropaolo, A.Z. *Addictions Treatment for Older Adults.* New York, Haworth Press, 1995.

Hanna, E.Z.; Chou, S.P.; and Grant, B.F. The relationship between drinking and heart disease morbidity in the United States: Results from the National Health Interview Survey. *Alcohol Clin Exp Res* 21:111–118, 1997.

Herd, D. Drinking by black and white women: Results from a national survey. *Soc Problems* 35:493–505, 1988.

Herd, D. Subgroup differences in drinking patterns among black and white men: Results from a national survey. *J Stud Alcohol* 51:221–232, 1990.

Hill, E.M.; Blow, F.C.; Young, J.P.; and Singer, K.M. Family history of alcoholism and childhood adversity: Joint effects on alcohol consumption and dependence. *Alcohol Clin Exp Res* 18:1083–1090, 1994.

Huffine, C.L.; Folkman, S.; and Lazarus, R.S. Psychoactive drugs, alcohol, and stress and coping processes in older adults. *Am J Drug Alcohol Abuse* 15:101–113, 1989.

Hwalek, M.A.; Neale, A.V.; Goodrich, C.S.; and Quinn, K. The association of elder abuse and substance abuse in the Illinois elder abuse system. *Gerontologist* 36:694–700, 1996.

Irwin, M.; Schuckit, M.; and Smith, T. Clinical importance of age at onset in type 1 and type 2 primary alcoholics. *Arch Gen Psychiatry* 47:320–324, 1990.

Jackson, J.; Antonucci, T.C.; and Gibson, R.C. Cultural, racial, and ethnic minority influences on aging. In: Birren, J.E., and Schaie, K.W., eds. *Handbook of the Psychology of Aging.* 3d ed. San Diego, CA: Academic Press, 1990. pp. 103–123.

Johansson, B.; Allen-Burge, R.; and Zarit, S.H. Self-reports on memory functioning in a longitudinal study of the oldest old: Relation to current, prospective, and retrospective performance. *J Gerontol* 52B:P139–P146, 1997.

Johnstone, B.M.; Leino, V.; Ager, C.R.; Ferrer, H.: and Fillmore, K.M. Determinants of life-course variation in the freqency of alcohol consumption: Meta-analysis of studies from the Collaborative Alcohol-Related Longitudinal Project. *J Stud Alcohol* 57:494–506, 1996.

Klatsky, A.L.; Armstrong, M.A.; and Friedman, G.D. Risk of cardiovascular mortality in alcohol drinkers, ex-drinkers and nondrinkers. *Am J of Cardiol* 66:1247–1242, 1990.

Lazarus, R.S., and DeLongis, A. Psychological stress and coping in aging. *Am Psychol* 38:245–254, 1983.

Liskow, B.I.; Rinck, C.; Campbell, J.; and DeSouza, C. Alcohol withdrawal in the elderly. *J Stud Alcohol* 50:414–421, 1989.

Lawrence, R.H.; Tennstedt, S.L.; and Almy, S.L. Subject-caregiver response comparability on global health and functional status measures for African-American, Puerto Rican, and Caucasian elders and their primary caregivers. *J Gerontol* 52B:S103–S111, 1997.

Mirowsky, J. Age, subjective life expectancy, and the sense of control: The horizon hypothesis. *J Gerontol* 52B: S125–S134, 1997.

Moos, R.H.; Mertens, J.R.; and Brennan, P.L. Patterns of diagnosis and treatment among late-middle-aged and older substance abuse patients. *J Stud Alcohol* 54:479–487, 1993.

Morrow, D.G.; Stine-Morrow, E.A.L.; Leirer, V.O.; Andrassy, J.M.; and Kahn, J. The role of reader age and focus of attention in creating situation models from narratives. *J Gerontol* 52B: P73–P80, 1997.

Moscovitch, M., and Winokur, G. The neuropsychology of memory and aging. In Craik, F.I.M., and Salthouse, T.A., eds. *The Handbook of Aging and Cognition.* Hillsdale, NJ: Lawrence Erlbaum Associates, 1992. pp. 315–372.

Myers, G.C. Demography of aging. In: Binstock, R.H., and George, L.K., eds. *Handbook of Aging and the Social Sciences.* San Diego, CA: Academic Press, 1990. pp. 19–44.

National Institute on Alcohol Abuse and Alcoholism. *Eighth Special Report to the U.S. Congress on Alcohol and Health.* NIH Pub. No. 94–3699. Bethesda, MD: National Institutes of Health, 1994. pp. 23–24.

National Institute on Alcohol Abuse and Alcoholism. *The Physician's Guide to Helping Patients With Alcohol Problems.* NIH Pub. No. 95–3769. Bethesda, MD: National Institutes of Health, 1995.

Neugarten, B.L. The awareness of middle aging. In: Neugarten, B.L., ed. *Middle Age and Aging.* Chicago: University of Chicago Press, 1968. pp. 93–98.

Park, D.C.; Smith, A. D.; Lautenschlager,G.; and Earles, J.L. Mediators of long-term memory performance across the life span. *Psychol Aging* 11:621–637, 1996.

Pozzato, G.; Moretti, M.; Franzin, F.; Croce, L.S.; Lacchin, T.; Benedetti, G.; Sablich, R.; Stebel, M.; and Campanacci, L. Ethanol metabolism and aging: The role of "first pass metabolism" and gastric alcohol dehydrogenase activity. J Gerontol 50A:B135–B141, 1995.

Pratt, J.; Abrams, R.A.; and Chasteen, A.L. Initiation and inhibition of saccadic eye movements in younger and older adults: An analysis of the gap effect. *J Gerontol* 52B:P103–P107, 1997.

Project MATCH Research Group. Matching alcoholism treatments to client heterogeneity: Project MATCH posttreatment drinking outcomes. *J Stud Alcohol* 38(6):7–29, 1997.

Ragland D.R., and Ames, G.M. Symposium: Current developments in the study of stress and alcohol consumption. *Alcohol Clin Exp Res* 20:51A–53A, 1996.

Resnick, H.E.; Fries, B.E.; and Verbrugge, L.M. Windows to their world: The effect of sensory impairments on social engagement and activity time in nursing home residents. *J Gerontol* 52B:S135–S144, 1997.

Rice, C., and Duncan, D.F. Alcohol use and reported physician visits in older adults. *Prev Med* 24:229–234, 1995.

Rice, C., and Longabaugh, R. Early versus late onset among older adults: Correlates of alcohol use prior to treatment for alcohol dependence. Poster presented at the meeting of the Research Society on Alcoholism, Steamboat Springs, CO, 1995.

Robins, L.N. Alcohol use in blacks and whites as indicated in the Epidemiological Catchment Area Program. In: Spiegler, D.; Tate, D.; Aitken, S.; and Christian, C., eds. *Alcohol Use Among U.S. Ethnic Minorities.* National Institute on Alcohol Abuse and Alcoholism Research Monograph 18. DHHS Pub. No. (ADM) 89–1435. Washington, DC: U.S. Government Printing Office, 1989. pp. 63–74.

Roizen, R. *The World Health Organization Study of Community Responses to Alcohol-Related Problems.* Annex 41, Final Report of Phase 1 of the WHO Study. Geneva: World Health Organization, October 21, 1981.

Rosin, A.J., and Glatt, M.M. Alcohol excess in the elderly. *Q J Stud Alcohol* 32:53–59, 1971.

Rowe, J.W., and Kahn, R.L. Human aging: Usual and successful. *Science* 237:143–149, 1987.

Rubington, E. Elderly homeless alcoholic careers. In: Beresford, T.P., and Gomberg, E.S.L., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 293–306.

Rush, B.R., and Timney, C. *Treatment Services for Alcohol and Drug Abuse in Ontario. Results of a Provincial Survey—1983.* Toronto: Addiction Research Foundation, 1985.

Salthouse, T.A. Cognitive competence and expertise in aging. In: Birren, J.E., and Schaie, K.W., eds. *Handbook of the Psychology of Aging.* 3d ed. San Diego, CA: Academic Press, 1990. pp. 311–319.

Schaie, K.W. Intellectual development in adulthood. In: Birren, J.E., and Schaie, K.W., eds. *Handbook of the Psychology of Aging.* 3d ed. San Diego, CA: Academic Press, 1990. pp. 291–310.

Sher, K.J. Stress response dampening. In: Blane, H.T., and Leonard, K.B., eds. *Psychological Theories of Drinking and Alcoholism.* New York: Guilford Press, 1987.

Schonfeld, L., and Dupree, L.W. Antecedents of drinking for early- and late-onset elderly alcohol abusers. *J Stud Alcohol* 52:587–592, 1991.

Schuckit, M.A. Geriatric alcoholism and drug abuse. *Gerontologist* 17:168–174, 1977.

Tarter, R.E. Cognition, aging and alcohol. In: Beresford, T., and Gomberg, E., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 82–98.

Tesch-Romer, C. Psychological effects of hearing aid use in older adults. *J Gerontol* 52B:P127–P138, 1997.

Vaillant, G.E. A long-term follow-up of male alcohol abuse. *Arch Gen Psychiatry* 53:243–249, 1996.

Wilsnack, S.; Vogeltanz, N.; Diers, L.; and Wilsnack, R. Drinking and problem drinking in older women. In: Beresford, T.P., and Gomberg, E.S.L., eds. *Alcohol and Aging.* New York: Oxford University Press, 1995. pp. 263–292.

World Health Organization Expert Committee on Mental Health. Report of the First Session of the Alcoholism Subcommittee. *World Health Organ Tech Rep Ser.* No. 42, 1951.

Yu, E.S.H.; Liu, W.T.; Xia, Z.; and Zhang, M. Alcohol use, abuse and alcoholism among Chinese Americans: A review of the epidemiological data. In: Spiegler, D.; Tate, D.; Aitken, S.; and Christian, C., eds. *Alcohol Use Among U.S. Ethnic Minorities.* National Institute on Alcohol Abuse and Alcoholism Research Monograph 18. DHHS Pub. No. (ADM) 89–1435. Washington, DC: U.S. Government Printing Office, 1989. pp. 329–342.

Zucker, R.A., Davies, W.H., Kincaid, S.B., Fitzgerald, H.E., Reider, E.E., and Bingham, D.R. Conceptualizing and scaling the developmental structure of behavior disorder: The lifetime alcohol problems score as an example. *Dev Psychopathol* 9:453–471, 1997.

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