PREFACE

The Child Nutrition and WIC Reauthorization Act of 1989 (Public Law 101-147) required the U.S. Department of Agriculture (USDA) to conduct a review of the nutritional risk criteria used in the Special Supplemental Food Program for Women, Infants and Children (WIC) and the relationship of such criteria to the Program's participant priority system. The legislation specifically directed the Department to consider the preventive nature of the WIC Program and to examine risks to categorically eligible persons, especially pregnant women, from conditions such as homelessness, mental illness, and conditions that pose barriers to the receipt of prenatal care and/or may increase the probability of adverse pregnancy outcomes or other adverse effects on health.

In designing the procedure for completion of the legislatively mandated review, the Department was convinced that its consideration of these important and complex issues would benefit greatly from public participation. Therefore, a Notice was published in the Federal Register on September 14, 1990 which identified the major issues to be addressed by the review and solicited public input on these issues. A copy of the Notice is included with the attached technical papers as background material.

The second phase of the review process involved enlisting independent technical experts to review the comments submitted to USDA in response to the Notice and then to conduct a comprehensive search of the scientific literature available on the issue topics to determine whether a consensus or majority opinion could be established on each one.

The attached technical papers were then developed by a team of professors and graduate students at the University of Arizona's College of Medicine, Department of Family and Community Medicine, under a Cooperative Agreement with USDA's Food and Nutrition Service during the spring and summer of 1991. Drafts of the papers were provided to the National Advisory Council on Maternal, Infant and Fetal Nutrition for discussion at an ad hoc meeting of 12 Council volunteers in June 1991. The papers were then revised, resubmitted to the Department, and used to form the agenda of a full Council meeting in September 1991, along with similar papers developed as part of a separate review addressing the foods prescribed provided to participants by the WIC Program, which was also mandated by Pub. L. 101-147.

The Council's recommendations are included in its 1992 Report to Congress and the President. Copies of the Report are available upon request from the U.S. Department of Agriculture, the Food and Nutrition Services, Supplemental Food Programs Division, 3101 Park Center Drive, Room 540, Alexandria, Virginia 22302, (703) 305-2730.

INTRODUCTION AND OVERVIEW

The purpose of this introduction and overview is to describe the background for and general methodological approach used in the production of an accompanying set of fifteen (15) brief, focused technical papers prepared for the U.S. Department of Agriculture, Food and Nutrition Service, Supplemental Food Programs Branch, under a cooperative agreement with the University of Arizona. The papers were prepared between March and August 1991. Drafts were reviewed by a subcommittee of the National Advisory Committee on Maternal, Infant and Fetal Nutrition (NAC) and by FNS staff, and the final documents were reviewed and discussed by NAC at its meeting in September 1991.

The WIC Program

The Child Nutrition Act of 1966, as amended, established the Special Supplemental Food Program for Women, Infants, and Children (WIC Program) based on growing evidence that nutritional inadequacy is linked to compromised physical and mental development, and that nutritional supplementation could result in positive health improvements when targeted to low-income pregnant and postpartum women and preschool children who are at nutritional risk. The program serves as both an adjunct to health care and an entry into health services for many women and children during critical periods of growth and development. The program provides four types of services to clients, free of direct cost: nutritional and general health assessments; nutrition education; health care and other public assistance referrals, as appropriate; and food benefits, through specified supplemental food packages, which may be provided via vouchers, direct home delivery, or direct distribution. Administered federally by the Food and Nutrition Service (FNS) of the U.S. Department of Agriculture (USDA), the program is managed at the state level by health agencies in the 50 states, Puerto Rico, the Virgin Islands, and the District of Columbia, and by Indian Tribal Organizations which function as state agencies. As of September 1991, the WIC program was serving approximately 5.1 million individuals; about 23% were women, 31% were infants and 46% were preschool children.

Eligibility for the program is based both on income criteria and on nutritional risk criteria. The legislation defines "nutritional risk" as "(A) detrimental or abnormal nutritional conditions detectable by biochemical or anthropometric measures; (B) other documented nutritionally related medical conditions, (C) dietary deficiencies that impair or endanger health, or (D) conditions that predispose persons to inadequate nutritional patterns or nutritionally related medical conditions, including but not limited to alcoholism and drug addiction." These criteria not only assure that the entire program is targeted to those most likely to benefit from it, but also serve as the basis for a priority system which state and local agencies can use to allocate their resources when funding does not permit serving all eligible clients. The priority system currently in use is as follows (Federal Register 55(179): 37683, 9/14/90):

Priority I. Pregnant and breastfeeding women and infants as demonstrated by documented nutritionally related medical conditions.

Priority II. Except those infants in Priority I. Infants up to six months of age born of women who were program participants during pregnancy or who were at nutritional risk during pregnancy due to documented nutritionally related medical conditions.

Priority III. Children at nutritional risk as demonstrated by documented nutritionally related medical conditions.

Priority IV. Pregnant and breastfeeding women and infants at nutritional risk due to an inadequate diet.

Priority V. Children at nutritional risk due to an inadequate diet.

Priority VI. Postpartum women at nutritional risk (state agencies have the option of defining "high-risk" postpartum women and placing them in Priorities III, IV, and/or V).

Priority VII. (State agency option). Previously certified participants who might regress in nutritional status without continued provision of supplemental foods.

In practice, State agencies have had considerable latitude in defining specific criteria within the broad legislative parameters, and the criteria in use vary from state to state. Several agencies, including the General Accounting Office, have expressed some concern about consistency of targeting criteria. The National Advisory Council (NAC) on Maternal, Infant and Fetal Nutrition recommended that FNS issue guidance to state agencies for use in development and evaluation of nutritional risk criteria.

Selection of Issues for the Technical Papers

The current work was undertaken as part of a Congressionally-mandated review of the nutritional risk criteria by the Department of Agriculture.

A notice of the review was published in the Federal Register in September 1991, and public comments solicited. A total of 184 written comments were received, from state and local WIC agencies and personnel, public interest groups, professional organizations, industry, and the general public. The first part of the present scope of work was to review and summarize those comments; that summary was provided to USDA/FNS in March 1991. The summary of comments and input from FNS staff formed the basis for identification of many of the issues for the technical papers. The issues selected represent those which are of concern to commenters and those for which USDA/FNS program staff indicated a need for technical background information. Several major risk factors for malnutrition and/or poor pregnancy outcome were not addressed, since FNS felt that sufficient scientific consensus was already available.

Identification of Relevant Literature

Once the key issues were tentatively identified and the number of papers to be drafted decided, literature searches were undertaken by standard means through computerized searches of the National Library of Medicine database. Unpublished documents and key reports were also obtained when their availability became known, as supplements to the literature accessible through standard searches, directly from the source or author's courtesy. Searches were not limited to U.S. studies, but rather took advantage of the worldwide literature when it could address issues of interest. The literature search was limited to the English-language literature. We utilized the expertise of several consultants who were able to direct us to several important documents which were in press at the time. In addition, USDA/FNS staff were able to locate and share with us several important unpublished reports.

Evaluation of the Quality of Literature Available on Key Issues

There is considerable variability by topic in the amount, recency, and quality of existing scientific information on the issues addressed by the technical papers. Therefore, no hard and fast rule was made about what types of studies would be included in the overall review. On topics for which there was an abundance of relevant literature, recent, peer-reviewed studies with the most appropriate design for investigating the topic were given priority in emphasis. On other topics (e.g., pica in pregnancy) most of the published literature is quite old, some is very anecdotal, and there is a relative lack of recent studies. On still others (e.g., homelessness), scientific interest in the topic is relatively recent and it was necessary to include some information from reports and other unpublished documents rather than to rely entirely on peer-reviewed publications.

Our strategy for coping with this unevenness by topic was simply to be clear within each paper about the quality of literature. Where there are significant methodological problems with a study, they are mentioned. Where the literature is less than ideal to address the topic, this is mentioned. Studies which are central to a given issue are described in some detail, and when comparisons among studies are made the differences in their design are pointed out.

Development of Technical Papers

Each paper was first drafted by the primary author and then reviewed by a faculty team and revised. Revised papers were provided as draft documents to USDA/FNS and to consultants. Several consultants and the USDA/FNS Program Officer for the project traveled to Tucson for a two-day meeting, during which each draft paper was discussed in detail. Following this meeting, the drafts were again revised and made available to a subcommittee of the National Advisory Council on Maternal, Fetal, and Infant Nutrition (NAC). The investigators met with that subcommittee in June 1991 for a full day, and again each paper was discussed in some detail. Following that meeting there was some minor rearrangement of topics. We again asked consultants' advice on specific papers,

updated information on one or two topics on which very recent publications or recommendations had become available, and once again revised the papers.

In each paper, we have attempted to make clear where there is scientific consensus on a given topic; where there is disagreement; where the state of the art is evolving very rapidly; and where the quality of evidence is either poor or difficult to evaluate.

Acknowledgements

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Peter Dallman, MD (University of California, San Francisco)

WIC NUTRITIONAL RISK TECHNICAL PAPERS

#1 Anthropometric Standards for U.S. Children

#2 Anthropometric Assessment of Pregnant Women

#3 Overweight and Obesity in Pregnancy

#4 Overweight and Obesity in Infants and Children

#5 Hematologic Standards for Risk of Iron Deficiency

#6 Age and Primiparity as Risk Factors to Poor Pregnancy Outcome in U.S. Women

#7 Evidence for Effects of Timing of Prenatal Care and Nutritional Supplementation on Pregnancy Outcome

#8 Passive Smoking: What is the Evidence for Health Effects?

#9 Nutritional Risk Implications of Pica in Pregnancy

#10 Intake of Caffeine and Related Compounds: Evidence of Nutritional Risk?

#11 Drug Use and Nutritional Risk in Pregnancy

#12 Homeless Mothers and Children: What is the Evidence for Nutritional Risk?

#13 Appropriate Dietary Assessment Methodology for the WIC Clinic Setting

#14 Pica and Lead Exposure in Infants and Children: Health and Nutritional Risk Implications

Technical Paper 1

Anthropometric Standards for U.S. Children

Technical Paper #1 prepared for the U.S. Department of Agriculture, Food and Nutrition Service, under Cooperative Agreement #58-3198-1-005, "Review of Nutritional Risk Criteria for the WIC Program", with the Department of Family and Community Medicine, University of Arizona, by Gail G. Harrison, PhD and Sahar S. Zaghloul, MB, BCh, MSc. Dr. Harrison is Professor of Family and Community Medicine, College of Medicine; Dr. Zaghloul is Research Assistant, Department of Family and Community Medicine and a doctoral candidate in Nutritional Sciences, University of Arizona, Tucson, Arizona 85724.

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INTRODUCTION

The Special Supplemental Food Program for Women, Infants and Children (WIC) includes anthropometric criteria for defining nutritional risk in children which vary somewhat among geographical state agencies. These criteria are limited to those which can be addressed by measures of height, weight and age. All include low birth weight (<2.5 kg or 5.5 lb) as a criterion; all include some definition of low height-for-age, low weight-for-height, and high weight-for-height for infants and children up to the age of five years. The questions of relevance for the present paper are the following:

a) is 2.5 kg the best cutoff point for identifying nutritional risk in newborns in the U.S.?

b) how does variation in the distribution of anthropometric measures among the different racial and ethnic groups in the U.S. affect the utility of a single reference standard for purposes of identifying nutritionally at-risk children? and

c) what are the consequences of different cutoff points relative to the reference standard for different racial/ethnic groups?

The objective of this paper is to review the information which is available to address these issues for the U.S. population.

THE IDENTIFICATION OF NUTRITIONAL RISK BY WEIGHT AT BIRTH

The internationally accepted cutoff for "low birth weight" is 2500 grams, or 5.5 pounds. Below this weight, infant mortality rises dramatically. However, it is important to appreciate that this may not be the optimal cutoff for identifying the infant whose growth has been constrained by nutritional factors. There are many potential causes for low birth weight, broadly falling into: a) prematurity (birth before 38 weeks of gestation), and b) intrauterine growth retardation (IUGR), which may be caused by a variety of factors including maternal undernutrition and other maternal variables which can constrain delivery of nutrients to the fetus. IUGR is identified by weight for gestational age at birth relative to standard reference data. (The issue of appropriate reference data for identifying IUGR in U.S. infants is of considerable scientific and clinical importance, but will not be addressed in this paper since it cannot be readily assessed within the structure of the WIC program). IUGR can be further divided into proportional vs nonproportional growth retardation - i.e., the fetus who has suffered growth retardation throughout gestation and whose length and weight are proportionately low at birth for gestational age, vs the fetus whose weight is decreased but whose length is relatively less affected, reflecting nutrient deprivation primarily in the last trimester. The distinction can be made on the basis of the ponderal index (Roher's Index), weight/length^3. The former type of IUGR (proportionate) is associated with substantially greater neonatal mortality, particularly for term infants (Balcazar and Haas, 1990, 1991).

Changes in mean birthweight within a population may be important and reflect nutritional risk even if there is no change in the low birthweight rate. The lowest risk of infant mortality is associated with birth weights of 3500-4000 grams, and any increase in the proportion of birthweights below 3500 grams may increase infant mortality (Kramer, 1987). Balcazar and Haas (1991), in a study of 9660 newborns in Mexico City, found that

term infants weighing less than 2900 grams (the 10th percentile of weight for gestational age for term infants based on a composite of several U.S. and European reference standards) had a neonatal mortality risk of 29.0 per 1000, compared to 2.8 per 1000 for term infants whose weight was appropriate for gestational age. Lester et al. (1986) found striking behavioral differences in the immediate postnatal period (18-60 hours after birth) between normal-weight term infants (mean birthweight 3186 grams) and small-for-gestational-age infants (average birthweight 2737 grams) in Puerto Rico; newborns who were small-for-age were less responsive, less able to process visual and auditory stimuli, and exhibited more immature motor development than heavier neonates.

There is also evidence that IUGR resulting in birthweights low for gestational age but above 2500 grams has consequences for neurological and behavioral development and for postnatal growth potential. Kimball et al. (1982) compared the long-term growth and development of three groups of infants: normal birth weight for gestational age (3266 $\pm$ 362 g) and normal ponderal index; low birth weight for gestational age (2600 $\pm$ 276 g) and low ponderal index (IUGR-LPI); and low birth weight (2600 $\pm$ 189 g) and normal ponderal index (IUGR-API). At three years of age, the IUGR-API group remained lighter and shorter than other children, and had smaller head circumference; they were also at greater developmental risk. Long-term effects on postnatal growth and behavioral development were greatest for very low-birthweight ($\leq$1500 g) infants; intermediate for IUGR-API infants; and smallest but still apparent for IUGRD-LIP infants. Largo et al. (1989) investigated the significance of prenatal, perinatal and postnatal variables on neurological, intellectual and language development at age five to seven years. Prematurity had a much larger influence on neurological and intellectual development at age 5-8 years, but children who had been full-term but underweight newborns were more likely to exhibit behavior problems.

In summary, intrauterine growth retardation is often manifested for term infants as birthweight low for gestational age but above the standard 2500-gram cutoff point; it does reflect prenatal undernutrition and there is good evidence that there are long-term effects on development and on physical growth potential.

ANTHROPOMETRIC STANDARDS FOR GROWTH OF INFANTS AND CHILDREN

The most available and well accepted reference standard available for use on a national basis for assessment of anthropometric measures in infants and children is that of the National Center for Health Statistics (Hamill et al., 1979), which has also been adopted for international use (WHO, 1987). The data are based on nationally representative samples of children over age two years, and below two years on the Fels Longitudinal Growth Study data. The latter data are from healthy, Caucasian children in Ohio and thus may not be representative of the growth of other groups of children; the data on older children while nationally representative may be less than optimal for specific subgroups within the population. There are considerable advantages, however, to using a consistent standard for evaluation of growth even though cutoff points may be set differently for different purposes.

Most WIC programs use percentiles of the reference standard to designate risk cut-off points. The scientific literature on nutritional status tends to use Z-scores or standard deviation units, which are more useful in quantifying differences in very malnourished

populations. Figure 1, from Frisancho (1990) shows equivalencies among Z-scores and percentiles.

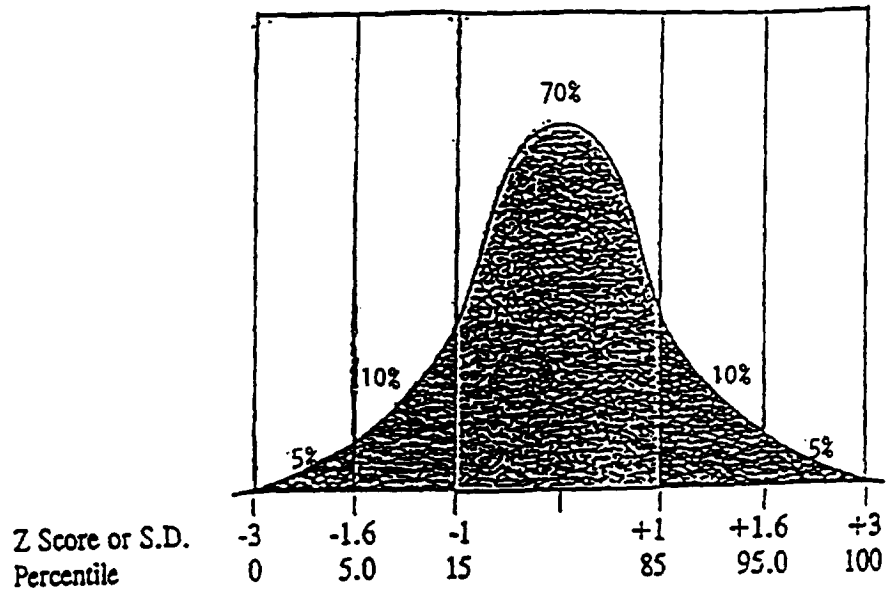


Figure 1 Schematization of the statistical relationship of Z-scores, percentile ranges, and standard deivations. Modified from Frisancho, 1990.

RACIAL/ETHNIC VARIATION IN THE DISTRIBUTION OF ANTHROPOMETRIC INDICES OF GROWTH

For a nationwide program which uses anthropometric measures as evidence of nutritional risk, it is important to appreciate whether the differences among racial and ethnic groups are substantial within the age range of interest and if so, whether they can be assumed to be due to genetic rather than nutritional factors.

Black, White and Hispanic Children

Tables 1-3 (constructed from data in Frisancho, 1990 and NCHS, 1989) summarize available data for nationally representative samples of White and Black children from the second National Health and Nutrition Examination Survey (NHANES II, 1975-80) and, where the numbers are adequate for Mexican-American and Puerto Rican children from the Hispanic Health and Nutrition Examination Survey (HHANES, 1982-84). Shown are means, standard deviations, and percentile distributions for stature (Table 1), weight (Table 2) and body mass index or Quetelet's index (weight/stature^2) (Table 3). While the Body Mass Index is less often used clinically than is weight-for-height as a percent of median expected or as a percentile, it gives essentially the same information. Inspection of these tables will reveal very minimal variation in mean and median values for weight or height among ethnic groups. Only one difference appears to be large enough to be of interest; the fifth percentile for stature is a full two centimeters lower for Black than for White children age one to two years, and persists for boys at the age of 2-2.9 years, but the difference is no longer evident after three years of age.

The numbers of Hispanic children included in the data from which Tables 1-3 were derived are too small to make firm conclusions about weight-for-height distribution; and Native American and Oriental children are not included (NCHS, 1989).

Native American Children

Native American children clearly have an excess of high weight-for-height compared to other groups of children (Harrison and Ritenbaugh, in press; Sugarman et al., 1990; Owen et al., 1979; Peck et al., 1987). The prevalence of adult obesity, and obesity-related diabetes, among some Native American tribes is also extremely high. Sugarman et al. (1990) studied school-aged Navajo children in 1989, twenty years after a survey done in the same area, and documented increases in stature-for-age for both boys and girls, and much greater increases in weight-for-age for both sexes. Thus while the prevalence of obesity seems to be increasing dramatically among Navajo children, there is also evidence of a secular trend in stature in this group. In contrast Owen et al. (1979) in a follow-up study of White Mountain Apache preschool children in 1976, found that compared to a comparably sampled 1969 cohort, there was no increase in height but a significant increase in skinfold thickness. That the relatively high weight-for-height distribution among Native American children is characteristic of very young children is seen in the study of Harrison and White (1981), who analyzed Arizona nutrition surveillance data and found that controlling for birth weight, Native American children had substantially higher weight-for-height in the second year of life than Mexican-American children, who in turn had higher weight-for-height indices than Anglo (non-Hispanic white) children. Length-for-age was not different among the three groups, nor affected by birth weight by age 13-24 months.

Mexican-American Children

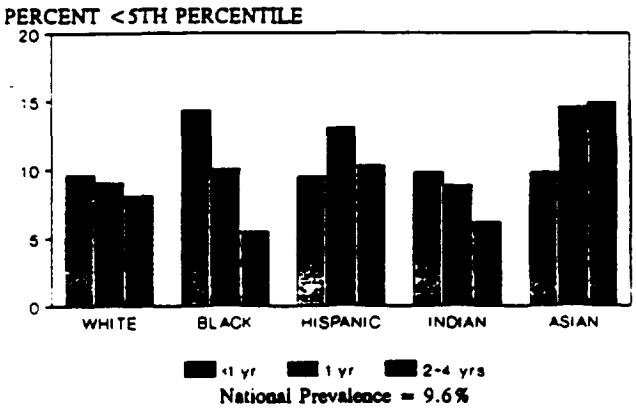
Mexican-American children, who genetically represent a mixture of European White and Native American background, also tend to have relatively short stature and high relative weights (Martorell et al., 1987; Yanochick-Owen and White, 1977; Kautz and Harrison, 1981; Harrison and White, 1981; Dewey et al., 1983; Malina et al., 1983). Overall, greater chest circumference, trunkal skinfolds, and overall fatness seem to describe the Mexican-American child relative to non-Hispanic White children and Black children. In the NHANES I and II data, short stature is related to poverty among Whites, Blacks, and Mexican-Americans; relative weight among Mexican-Americans had no relationship to economic status (Martorell et al., 1987). In the more recent Hispanic HANES survey; neither stature nor relative weight was different between poor and nonpoor Mexican-American children, however (Ryan et al, 1990). Martorell et al. (1989) and Roche (1990) have compared age- and sex-specific weight and stature percentiles for Mexican-American children from the HHANES data with those of White children in the NHANES II data. Until adolescence, there were no significant differences for boys; for girls, the 50th and 95th percentiles for weight were significantly higher for Mexican-Americans.

Asian-American Children

Asian-American children, particularly refugee groups who immigrated from Southeast Asia in the late 1970s and early 1980s, pose particular problems of interpretation and for states in which these subgroups have settled, may raise questions of interpretation of reference

Figure 2

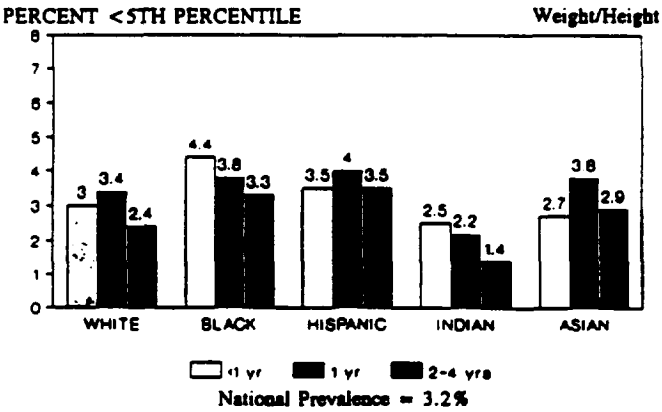
Short Stature by Age/Ethnic Group
1989 Pediatric Nutrition Surveillance
Total States/Territories/Reservations



National Prevalence = 9.6%

Figure 3

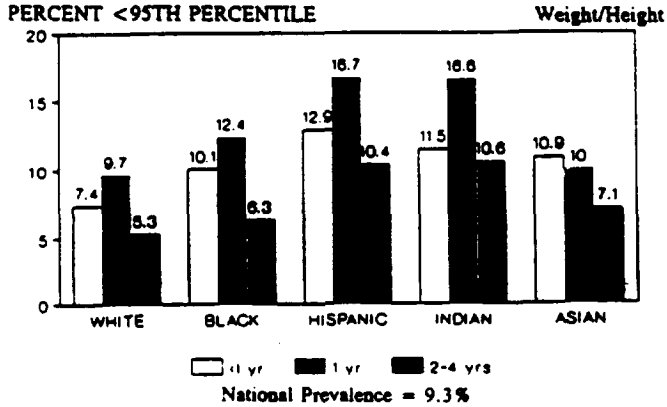
Underweight by Age/Ethnic Group
1989 Pediatric Nutrition Surveillance
Total States/Territories/Reservations



National Prevalence = 3.2%

Figure 4

Overweight by Age/Ethnic Group
1989 Pediatric Nutrition Surveillance
Total States/Territories/Reservations



National Prevalence = 9.3%

Courtesy Division of Nutrition, Centers for Disease Control

standards. Too little data are available currently to make firm conclusions about the normal growth patterns for children belonging to these groups. Early reports of their nutritional status showed high rates of apparent malnutrition, manifested as anemia and stunting (Peck et al., 1981). Recent data from the Centers for Disease Control Pediatric Nutrition Surveillance System (GAO, 1990) show increasing height-for-age and decreasing prevalence of low height-for-age in Asian-American children between 1981 and 1989.

CONSEQUENCES OF PARTICULAR CUTOFF POINTS FOR HEIGHT-FOR-AGE AND WEIGHT-FOR-HEIGHT FOR DIFFERENT RACIAL/ETHNIC GROUPS

Figures 2-4 present data on the prevalence of low height-for-age, low and high weight-for-height, defined at the 5th or 95th percentile, by ethnic group from the Centers for Disease Control Nutrition Surveillance System (1989), for children of the WIC target group age. This database is derived from more than 4000 local clinics including health department clinics, WIC clinics, and Indian Health Service clinics, in 43 geographical units including states, the District of Columbia, Puerto Rico, and Indian tribal units. The relative excess of low height-for-age among White children can be taken as evidence for the low-income nature of the population from which these data were derived. Prevalences of height-for-age below the 5th percentile are highest for Black infants under one year, Hispanic children one to two years, and Asian children older than one year. There is a lower-than-expected prevalence of low weight-for-height in all subgroups. The prevalence of high weight-for-height (above the 95th percentile of the NCHS reference population) is higher among Black, Hispanic, and Native American children than in White or Asian children. None of the ethnic groups, however, shows a prevalence of high weight-for-height so great as to include a large share of the population screened. Very likely the differences seen represent real differences in the prevalence of obesity.

CUTOFF POINTS FOR RISK ASSESSMENT

The reference population is assumed to consist of healthy, normal children whose attained growth represents their genetic potential. If a screened population were exactly the same as the reference population, then, for example, five percent of children would have heights at or below the 5th percentile. If a screened population has an excess of children with height-for-age below a cutoff point (e.g., 10 percent below the 5th percentile), we assume that the difference (5 percent) represents short stature due to environmental causes and is likely to be responsive to nutritional and health intervention. The remaining five percent of children are healthy and normal and will not respond to nutritional intervention. When the ratio of affected to normal children identified is high and the intervention is without risk, we simply accept the false positives (healthy children identified as eligible but who will not respond).

The fifth percentile as a cutoff point for identification of short stature and low weight for height is in common use and has been recommended by a number of experts, albeit arbitrarily (Robbins and Trowbridge, 1984; Zerfas et al., 1977; DHHS, 1981). If a cutoff point is set at a higher level (e.g., the 10th or 25th percentile) it may be possible to identify additional children at nutritional risk, but at the cost of a higher rate of false positives (misclassification of normal children). As the cutoff becomes more liberal, the proportion of healthy children to malnourished ones identified rises. Thus if a screened population includes 30% of children below the 25th percentile for height, we may assume that only

one in six of these will respond to nutritional intervention. The choice of cutoff points for defining risk, therefore, will depend on resources and priorities within the program.

It may not be possible to judge on a cross-sectional basis whether a highly prevalent condition (e.g., short stature, or high weight-for-height) will respond to nutritional intervention. Useful surrogate information, when it is available, includes whether there is an income relationship with the measure and whether there are demonstrable secular trends occurring in the measure. Either situation is a good argument for high prevalence being the result of nutritional etiology. It may be useful to point out that cutoff points for low and high weight-for-height need not be symmetrical, since their use is to identify two quite different conditions.

SUMMARY

There is good reason to consider birthweights above 2500 grams but low for gestational age as a marker for nutritional risk, as they reflect both prenatal undernutrition and postnatal risk for increased mortality and for impaired growth and development. A reasonable cutoff point for full-term infants would be 2900 grams, which reflects approximately the tenth percentile of weight for gestational age at term from a composite of several U.S. and European references standards.

There are demonstrable differences in the distribution of heights and weight-for-height among children of different ethnic and racial groups in the U.S. Among U.S. ethnic groups, there is enough variation to result in the inclusion of large proportions of Asian children as short-for-age relative to national standards and a relatively large proportion of Hispanic and Native American children as heavy-for-height. There is insufficient evidence to suppose that either of these phenomena is not influenced by the nutritional environment, particularly in view of the fact that both phenomena show some evidence of secular trend -- i.e., that height is increasing in some groups of the population and that high weight-for-height is increasing among children in the population as a whole. There is no reason not to use the current national and international reference standard, namely the National Center for Health Statistics reference, for screening in the WIC program in spite of the fact that the data were derived from children who are not representative of the various ethnic groups which now comprise the U.S. population.

Designation of cutoff points for anthropometric measures must consider the resources available and the potential responsiveness of real cases of nutrition-related short stature, low weight-for-height and obesity to the interventions which are provided by the program. More conservative cutoff points, generally speaking, will increase the ratio of cases to false positives and improve targeting while more liberal cutoff points will increase the number of at-risk children served overall. It may be advisable to consider asymmetric cutoff points for low and high weight-for-height, depending on the population served and program resources.

Table 1

MEANS, STANDARD DEVIATIONS, AND PERCENTILES OF STATURE (cm) BY AGE (yrs) FOR MALE AND FEMALE CHILDREN AGES 1-5 YEARS OF DIFFERENT ETHNIC GROUPS

Age (yrs)	Race	Sex	N	Mean	SD	Percentiles								
						5	10	15	25	50	75	85	90	95
1.0-1.9	Black	M	77	82.3	5.4	74.3	76.1	77.5	79.0	81.7	86.0	87.2	89.5	94.0
		F	56	81.0	4.0	73.9	75.7	77.4	78.3	81.2	83.0	84.3	87.0	87.4
	White	M	277	82.5	5.0	75.7	76.8	77.9	79.4	82.2	85.6	87.2	88.0	89.2
		F	264	80.5	4.9	73.0	74.7	75.6	76.9	80.2	83.6	85.8	86.8	88.4
	Mexican American	M	104	82.6	4.6	76.0	76.8	77.4	79.3	82.1	85.3	87.9	88.8	90.1
		F	118	80.7	4.8	74.2	75.1	76.6	77.6	80.4	84.0	85.1	86.5	87.5
	Puerto Rican	M	31	83.2	7.1	-	-	-	77.8	83.6	86.7	-	-	-
		F	31	82.0	6.8	-	-	-	79.1	83.3	86.7	-	-	-
2.0-2.9	Black	M	139	90.7	4.7	83.5	84.8	85.8	87.2	90.1	94.2	96.0	96.8	98.0
		F	118	90.0	5.1	81.5	84.6	85.1	86.5	89.8	93.2	95.0	96.7	97.9
	White	M	504	91.7	4.2	85.8	86.8	87.5	88.9	91.7	94.4	95.8	97.0	98.3
		F	479	90.2	4.4	83.4	84.9	85.7	86.9	90.2	93.0	94.5	95.7	97.4
	Mexican American	M	110	91.5	4.0	85.1	86.1	87.2	88.9	91.7	93.8	95.6	96.6	98.2
		F	116	89.2	3.8	82.7	84.5	85.6	86.9	89.4	91.9	93.4	94.0	94.4
	Puerto Rican	M	34	93.4	5.3	-	-	-	89.1	94.0	96.6	-	-	-
		F	27	88.9	4.1	-	-	-	85.8	88.1	91.6	-	-	-
3.0-3.9	Black	M	151	99.1	5.4	90.2	93.0	94.3	95.8	98.3	102.5	104.5	106.7	108.4
		F	126	98.6	4.7	92.3	93.1	94.2	95.3	98.5	101.0	102.7	103.7	105.7
	White	M	540	99.1	4.5	92.0	94.0	94.8	96.3	98.8	102.0	103.8	104.8	106.3
		F	509	97.5	4.5	90.1	91.6	92.7	94.6	97.4	100.5	102.3	103.4	104.5
	Mexican American	M	131	98.6	4.8	89.7	91.2	93.3	95.6	99.5	101.7	103.6	104.2	105.5
		F	97	97.9	4.6	-	90.0	92.6	94.2	98.4	101.6	102.3	103.6	-
	Puerto Rican	M	38	99.5	4.7	-	-	94.9	95.6	99.0	102.3	104.1	-	-
		F	40	98.0	3.9	-	-	93.2	95.9	97.6	100.6	101.6	-	-
4.0-4.9	Black	M	151	107.1	5.7	98.1	99.6	101.3	103.5	107.7	110.7	112.4	113.7	116.7
		F	147	106.6	5.1	98.4	100.0	101.3	103.1	106.6	109.8	111.9	113.9	115.0
	White	M	547	105.7	4.8	98.3	99.6	100.6	102.6	105.7	108.9	110.5	112.0	113.7
		F	519	104.5	4.8	96.2	98.3	99.4	101.3	104.5	107.7	109.3	110.7	112.2
	Mexican American	M	118	105.3	4.8	97.9	99.3	100.2	101.7	104.9	108.7	111.2	111.8	113.1
		F	96	105.1	4.9	-	98.1	98.6	102.2	105.1	108.6	110.1	110.7	-
	Puerto Rican	M	41	107.3	6.2	-	-	102.7	104.5	107.3	110.1	112.1	-	-
		F	34	106.7	4.7	-	-	-	103.4	106.0	110.2	-	-	-

Data from Frisancho (1990) and NCHS (1989)

Table 2

MEANS, STANDARD DEVIATIONS, AND PERCENTILES OF WEIGHT (kg) BY AGE (yrs) FOR MALE AND FEMALE CHILDREN AGES 1-5 YEARS OF DIFFERENT ETHNIC GROUPS

Age (yrs)	Race	Sex	N	Mean	SD	Percentiles								
						5	10	15	25	50	75	85	90	95
1.0-1.9	Black	M	157	11.8	1.6	9.6	9.9	10.2	10.7	11.6	12.6	13.4	14.4	15.0
		F	134	10.9	1.5	8.6	9.1	9.6	10.0	10.8	11.7	12.3	12.8	13.5
	White	M	508	11.8	1.7	9.5	10.0	10.3	10.7	11.6	12.6	13.1	13.6	14.2
		F	470	10.9	1.4	8.8	9.2	9.5	9.9	10.8	11.8	12.4	12.8	13.4
	Mexican American	M	105	11.8	1.8	9.5	9.8	10.1	10.8	11.5	12.5	13.1	13.5	15.0
		F	119	11.0	1.5	8.8	9.3	9.4	9.8	10.9	12.0	12.7	13.2	13.6
	Puerto Rican	M	34	11.8	2.0	-	-	-	10.6	11.8	12.7	-	-	-
		F	33	11.2	1.6	-	-	-	9.8	11.3	12.3	-	-	-
2.0-2.9	Black	M	142	13.5	1.8	11.1	11.3	11.7	12.1	13.4	14.5	15.6	15.9	16.6
		F	119	13.0	1.8	10.3	10.7	11.2	11.9	12.8	13.9	14.5	15.2	16.2
	White	M	513	13.6	1.7	11.0	11.6	12.0	12.6	13.6	14.6	15.2	15.5	16.6
		F	483	13.0	1.6	10.8	11.2	11.6	12.0	12.8	13.9	14.6	15.0	15.9
	Mexican American	M	111	14.0	1.7	11.3	11.7	12.1	13.0	14.1	15.0	15.5	16.0	17.0
		F	121	13.1	1.7	10.8	11.2	11.6	12.0	12.9	13.8	14.7	15.3	15.7
	Puerto Rican	M	35	14.7	2.1	-	-	12.8	13.1	14.8	15.4	16.0	-	-
		F	27	13.1	2.0	-	-	-	11.9	12.8	14.4	-	-	-
3.0-3.9	Black	M	151	15.7	2.4	12.6	13.1	13.4	14.1	15.4	16.8	17.6	18.6	19.8
		F	128	15.0	2.2	11.8	12.6	13.0	13.7	14.7	16.1	17.2	17.6	18.5
	White	M	541	15.7	2.0	12.9	13.6	13.9	14.4	15.5	16.8	17.5	18.0	18.9
		F	509	15.0	2.0	11.8	12.6	13.0	13.6	14.9	16.2	17.1	17.6	18.5
	Mexican American	M	131	15.8	2.3	12.3	13.0	13.8	14.5	15.4	16.8	17.7	18.3	19.9
		F	97	15.3	2.3	-	12.8	13.1	13.8	15.0	16.8	17.5	17.8	-
	Puerto Rican	M	38	15.5	2.0	-	-	13.3	13.9	15.5	16.7	17.4	-	-
		F	40	15.5	3.3	-	-	13.0	13.7	15.1	16.5	17.1	-	-
4.0-4.9	Black	M	151	17.9	2.4	13.7	14.7	15.4	16.2	17.9	19.5	20.2	20.8	21.7
		F	147	17.7	3.0	13.7	14.3	15.0	16.0	17.2	19.2	20.8	21.2	22.4
	White	M	547	17.7	2.4	14.3	15.1	15.4	16.1	17.5	18.9	19.8	20.5	21.4
		F	523	16.9	2.2	13.7	14.3	14.6	15.3	16.7	18.3	19.0	19.7	20.9
	Mexican American	M	118	17.7	2.1	14.7	14.9	15.4	16.2	17.6	18.9	19.5	20.4	21.7
		F	96	17.7	2.7	-	14.9	15.1	15.8	17.6	19.1	20.4	20.6	-
	Puerto Rican	M	41	19.2	4.1	-	-	16.0	16.4	18.3	21.1	22.0	-	-
		F	34	18.7	3.6	-	-	-	16.1	17.6	20.4	-	-	-

Data from Frisancho (1990) and NCHS (19

Table 3

MEANS, STANDARD DEVIATIONS, AND PERCENTILES OF BODY MASS (w/s²) BY AGE (yrs) FOR MALE AND FEMALE CHILDREN AGES 1-5 YEARS OF DIFFERENT ETHNIC GROUPS

Age (yrs)	Race	Sex	N	Mean	SD	Percentiles								
						5	10	15	25	50	75	85	90	95
1.0-1.9	Black	M	77	17.4	1.8	14.8	15.4	15.8	16.4	17.3	18.2	19.0	19.4	20.2
		F	56	16.8	1.4	14.6	15.2	15.4	15.8	16.5	17.6	18.2	18.5	19.6
	White	M	277	17.3	2.6	15.3	15.6	15.9	16.4	17.1	17.9	18.6	18.9	19.6
		F	264	16.8	1.6	14.3	14.9	15.2	15.8	16.7	17.6	18.2	18.8	19.3
	Mexican American	M	-	-	-	-	-	-	-	-	-	-	-	-
		F	-	-	-	-	-	-	-	-	-	-	-	-
	Puerto Rican	M	-	-	-	-	-	-	-	-	-	-	-	-
		F	-	-	-	-	-	-	-	-	-	-	-	-
2.0-2.9	Black	M	139	16.4	1.4	14.5	14.8	15.0	15.4	16.2	17.2	17.9	18.6	19.0
		F	118	16.1	1.8	13.7	14.1	14.6	14.9	16.1	17.0	17.5	18.1	18.5
	White	M	504	16.2	1.3	14.3	14.5	15.0	15.4	16.2	17.0	17.4	17.7	18.2
		F	479	16.0	1.4	14.1	14.5	14.7	15.1	15.9	16.8	17.3	17.8	18.5
	Mexican American	M	-	16.7	1.4	14.6	15.0	15.3	15.5	16.8	17.6	18.2	18.4	19.4
		F	-	16.3	1.4	14.5	14.8	15.0	15.5	16.2	17.2	17.6	17.8	18.2
	Puerto Rican	M	-	16.9	1.9	-	-	-	15.8	16.6	17.6	-	-	-
		F	-	16.5	1.6	-	-	-	15.4	16.6	17.5	-	-	-
3.0-3.9	Black	M	151	15.9	1.4	13.9	14.6	14.8	15.1	15.7	16.5	16.9	17.2	18.1
		F	126	15.4	1.5	13.2	13.7	14.1	14.4	15.2	16.2	16.8	17.5	17.9
	White	M	540	16.0	1.3	14.2	14.5	14.8	15.2	15.8	16.6	17.1	17.5	18.2
		F	509	15.7	1.3	13.8	14.2	14.5	14.8	15.6	16.4	17.1	17.5	18.0
	Mexican American	M	-	16.2	1.6	14.2	14.5	14.8	15.1	16.0	16.9	17.3	17.8	18.8
		F	-	16.0	1.7	-	14.3	14.5	15.1	15.9	16.8	17.5	17.9	-
	Puerto Rican	M	-	15.9	1.2	-	-	14.6	14.9	15.4	16.1	17.4	-	-
		F	-	16.1	3.6	-	-	14.3	14.6	15.8	16.3	16.8	-	-
4.0-4.9	Black	M	151	15.5	1.3	13.6	13.9	14.3	14.8	15.5	16.3	16.8	17.0	17.4
		F	147	15.5	1.7	13.3	13.6	13.8	14.3	15.4	16.3	17.1	17.7	18.6
	White	M	547	15.8	1.4	14.0	14.4	14.6	14.9	15.6	16.4	16.8	17.2	17.8
		F	519	15.5	1.3	13.7	13.9	14.2	14.6	15.3	16.2	16.7	17.0	17.7
	Mexican American	M	-	15.9	1.1	14.1	14.6	14.8	15.2	15.7	16.6	16.9	17.2	17.8
		F	-	15.9	1.5	-	14.3	14.5	14.8	15.6	16.5	17.3	18.2	-
	Puerto Rican	M	-	16.5	2.1	-	-	14.7	14.9	15.9	17.9	18.9	-	-
		F	-	16.4	2.2	-	-	-	14.9	15.9	17.3	-	-	-

Data from Frisancho (1990) and NCHS (1989)

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Technical Paper 2

Anthropometric Assessment of Pregnant Women

Technical Paper #2 prepared for the U.S. Department of Agriculture, Food and Nutrition Service, under Cooperative Agreement #58-3198-1-005, "Review of Nutritional Risk Criteria for the WIC Program", with the Department of Family and Community Medicine, University of Arizona, by Gail G. Harrison, PhD, Professor of Family and Community Medicine, College of Medicine, University of Arizona, Tucson, AZ 85724.

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INTRODUCTION

There are two issues in the anthropometric assessment of women relevant to the WIC program, namely, the classification of women as under- or overweight on the basis of non-pregnant or early pregnancy weight and height, and the assessment of the adequacy of weight gain during pregnancy. There is need for attention to both issues, since both prepregnancy weight-for-height and pregnancy weight gain are strongly related to birth weight and other pregnancy outcomes. Women who are underweight prior to pregnancy have increased risk of delivering a low-weight newborn, and women who gain too little weight during pregnancy are likewise at risk. These risks interact, with the underweight woman with low weight gain the most at-risk. The optimal outcome of pregnancy is associated with higher weight gains for women who are initially lean and somewhat lower gains for those who have high relative weights before pregnancy. Women who are markedly overweight prior to pregnancy do best with somewhat lower gains than leaner women; even so they are at increased risk of inadequate weight gains and have more variable pregnancy weight gains than leaner women.

This paper reviews current literature and recommendations for categorizing prepregnancy weight-for-height and for assessing adequacy of pregnancy weight gain. The paper relies heavily on a recent National Academy of Sciences/Institute of Medicine report on *Nutrition in Pregnancy* (IOM, 1990) which provides comprehensive background and recent recommendations on both issues. The IOM report will no doubt influence obstetric practice and public health policies for some time to come; the present paper presents a summary of the relevant sections of that report.

CLASSIFICATION OF RELATIVE WEIGHT ON THE BASIS OF PREPREGNANT STATUS OR IN EARLY PREGNANCY

Classification of prepregnant relative weight-for-height is based on measured height and weight either prior to pregnancy or early in pregnancy. If the first contact with a client occurs past the first trimester, prepregnant weight must be estimated from recall, which should then be evaluated for plausibility. To assess relative weight, it is necessary either to calculate body mass index (BMI) or to compare weight-for-height to a reference standard.

Expression of relative weight as a percentage of a reference population mean or median is the most prevalent method in clinical practice. The calculation is done as follows:

$$\text{Percentage of reference weight} = \frac{\text{observed weight} \times 100}{\text{expected or desirable weight for a given height}}$$

The Metropolitan Life Insurance Company (1959) recommendations are in common clinical use. Other possible reference standards, less commonly used, include the 1983 Metropolitan Life Insurance Company tables (which give slightly heavier recommended weights on average for age than the earlier edition) and the normative data for White and Black women from NHANES II. The life insurance company databases were developed to reflect weights with minimal mortality risk among insured adults and the NHANES data are normative; none of the available standards reflects optimization of any obstetrical outcome.

There is no scientific or statistical basis to recommend any particular reference over another for the present purpose (IOM, 1990).

Body Mass Index (BMI) is a measure of relative weight in more common use in the research literature. It can be calculated either in metric or English units, as follows (IOM, 1990: 66):

$$BMI = wt/ht^2 = kg/m^2 \times 100, \text{ or } lb/in^2 \times 100$$

The metric version is standard in the epidemiological and clinical literature. Calculation of the BMI in English units has the theoretical advantage of being less error-prone in a clinical setting where initial measures are taken in English units, but comparison with standard recommendations requires the metric version. A simple conversion chart is available (IOM, 1990) and is included as Appendix A to this paper.

Recent investigators (Abrams and Parker, 1988; Kleinman, 1990; Naeye, 1990) have used BMI (metric) to describe pregravid weight-for-height, and the recent IOM report (1990) recommends using a metric BMI of 19.8 - 26.0 as the normal range, pending further research (see Table 1). This range includes approximately the 25th - 75th percentiles of prepregnancy weight-for-height of women in the 1980 National Natality Survey (Kleinman, 1990).

Table 1

Categories of Prepregnant Body Mass Index (kg/m^2) Used in Recent Studies of Pregnancy Outcome, and Current Recommendation

Source	Category	BMI Range
Abrams & Parker, 1988	Average	19.2-25.6
	Moderately Overweight	25.6-28.9
	Very Overweight	>28.9
Naeye, 1990	Thin	<20
	Normal	20-24
	Overweight	25-30
	Obese	>30
Kleinman, 1990	Low	<19.8
	Moderate	19.8-26.0
	High	26.1-29.0
	Very High	>29.0
IOM, 1990	Underweight	<19.8
	Normal weight	>19.8-26.0
	Overweight	>26.0-29.0
	Obese	>29.0

Body Mass Index is currently not in common clinical use. A follow-up report to the 1990 NAS/IOM *Nutrition in Pregnancy*, dealing with practical application of the recommendations, is in preparation and will include simple conversion and calculation devices for BMI, which will be field tested over the next year or so.

ASSESSMENT OF THE ADEQUACY OF PREGNANCY WEIGHT GAIN

The major factor affecting the amount of weight gain associated with optimal pregnancy outcome is prepregnancy weight-for-height. Accordingly, recommendations for weight gain should be adjusted for an estimate of pregnancy weight-for height. The current recommendations are summarized in Table 2.

Table 2

Recommended Total Weight Gain Ranges for Pregnant Women[a], by Prepregnancy Body Mass Index (BMI)[b] (from IOM, 1990: p. 10)

Weight-for Height Category	Recommended Total Gain	
	kg	lb
Low (BMI < 19.8)	12.5-18	28-40
Normal (BMI of 19.8 to 26.0)	11.5-16	25-35
High (BMI >26.0 to 29.0)[c]	7-11.5	15-25

[a] Young adolescents and black women should strive for gains at the upper end of the recommended range. Short women (<157 cm or 62 in) should strive for gains at the lower end of the range.

[b] BMI is calculated using metric units.

[c] The recommended target weight gain for obese women (BMI > 29.0) is at least 6.0 kg (15 lb).

A number of pregnancy weight gain grids have been developed. Early weight gain charts (e.g., Thompkins and Wiehl, 1951) were based on normative data from relatively homogeneous samples of healthy women. More recent charts have usually incorporated adjustments for prepregnancy weight-for-height and/or larger or different sets of normative data. The recent IOM report lists no fewer than ten standard grids (IOM, 1990: Table 4-1). The WIC program itself has, throughout the 1980s, contributed to the public and professional awareness of the need for standard weight gain charts for nutritional monitoring during pregnancy. The IOM subcommittee reviewed the weight gain charts used by WIC programs in 21 states (IOM, 1990: 69). They are most often modifications of

Thompkins and Wiehl's (1951) chart, with allowance for prepregnancy weight status. The particular modifications follow no standard format.

The Institute of Medicine Committee has recommended necessary research to further refine pregnancy weight gain recommendations, and in the meantime has published provisional weight gain charts specific to BMI category which are included with this paper as Appendix B. At this time, these must be regarded as the most current and useable recommendations for nutrition screening and monitoring of weight gain in pregnancy. There is no substantial evidence that parity, previous obstetrical history, smoking, use of alcohol or other substances, physical work, or other variables should affect recommended weight gains except insofar as they are associated with differential prepregnant weight-for height. Maternal age and height are of importance only at the very young and very short ends of the distribution, as noted in the footnotes to Table 2 (i.e., young adolescents should strive to gain near the top of the recommended range, and women under 62 inches tall to gain near the bottom of the recommended range). Ethnic background appears to have little effect except perhaps for Black women, who are recommended to strive for slightly higher gains than White women within the appropriate range (see Table 2).

Risk of low weight gain (< 15 pounds) is highest among unmarried women, Black and Hispanic women, cigarette smokers, women with less education, young adolescents, and obese women. These women should receive increased attention in nutrition monitoring and counseling during pregnancy to ensure adequate weight gains.

For purposes of monitoring adequacy of weight gain in pregnancy in the WIC clinic setting, rate of weight gain is more important than total gain. Recommended rates of gain are given for normal weight, underweight, and overweight women by trimester in Appendix A and summarized in Table 3:

Table 3

Recommended Rates of Pregnancy Weight Gain by Trimester

Prepregnancy Body Mass Index	Recommended Gain
19.8-26.0 (Normal Weight)	1.6 kg (3.5 lb) 1st trimester 0.4 kg (1 lb)/week 2nd and 3rd trimesters
< 19.8 (Underweight)	2.3 kg (5 lb) 1st trimester 0.5 kg (1 lb)/week 2nd and 3rd trimesters
26.0-29.0 (Overweight)	0.9 (2 lb) 1st trimester 0.3 kg (0.5-0.75 lb)/week 2nd and 3rd trimesters

USE OF OTHER ANTHROPOMETRIC MEASURES (MID-ARM CIRCUMFERENCE, SKINFOLDS)

While the availability of supplementary anthropometric measures such as skinfold measures may provide more complete information on body composition, helping to differentiate the obese from the muscular woman with high weight-for-height, there is little evidence that the additional information gained is useful for screening or monitoring purposes. Mid-arm circumference has received some attention as a potential monitoring tool during pregnancy, particularly for developing country situations in which scales may not be available and weighing impractical (USAID, 1990). However, weight and height measures offer the best set of information available when they can be used, and the present paper is based on the assumption that weight and height will be available in the WIC clinic setting. If there is significant delay in obtaining weight and height information, and a rough index of risk of underweight is desired, arm circumference might be used. There is some difficulty at this point in establishing agreement on the exact mid-arm point to be used in taking the measurement. There are no relevant data from the U.S. on which to assess the relationship of arm circumference to pregnancy risk, but data from Asian and Latin American populations suggest that a prepregnancy or early pregnancy mid-arm circumference of less than 21-23.5 cm is associated with increased risk of low birth weight and fetal and infant mortality (USAID, 1990).

SUMMARY

The National Academy of Sciences/Institute of Medicine has recently recommended assessment of prepregnant weight-for-height be done on the basis of Body Mass Index, the calculation of which has been simplified by recently published charts. Aids for conversion to use of this index in clinical practice are being developed. Weight gain recommendations for pregnancy are specific for pre-pregnancy weight-for-height, with optimal outcomes associated with higher weight gains for thinner women than for overweight women. Optimal gains for obese women (BMI >29.0) are not entirely clear, but current recommendations are for weight gains of at least 6 kg (15 pounds). Rate of weight gain is potentially of more utility than total pregnancy gain in the WIC clinic setting, and recently published recommendations, specific for pre-pregnancy weight-for-height, are provided. While supplementary anthropometric measures such as skinfold thickness and mid-arm circumference measurements are available, there is little evidence that the additional information gained is useful for screening or monitoring purposes.

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Appendix A

Table for Estimating Body Mass Index (Metric) by Using Either Metric or English Measurements of Prepregnancy Weight and Height; BMI < 19.8 = low; BMI 26.1 - 29.0 = high; BMI > 29.0 = obesity (see shaded area above heavy line).

from: IOM/NAS, 1990

Weight		Height, in. (and cm)																					
		55.9	56.7	57.5	58.3	59.1	59.8	60.6	61.4	62.2	63.0	63.8	64.6	65.4	66.1	66.9	67.7	68.5	69.3	70.1	70.9	71.7	72.4	73.2	74.0
lb	kg	(142)	(144)	(146)	(148)	(150)	(152)	(154)	(156)	(158)	(160)	(162)	(164)	(166)	(168)	(170)	(172)	(174)	(176)	(178)	(180)	(182)	(184)	(186)	(188)
220	100	49.6	48.2	46.9	45.7	44.4	43.3	42.2	41.1	40.1	39.1	38.1	37.2	36.3	35.4	34.6	33.8	33.0	32.3	31.6	30.9	30.2	29.5	28.9	28.3
218	99	49.1	47.7	46.4	45.2	44.0	42.8	41.7	40.7	39.7	38.7	37.7	36.8	35.9	35.1	34.3	33.5	32.7	32.0	31.2	30.6	29.9	29.2	28.6	28.0
216	98	48.6	47.3	46.0	44.7	43.6	42.4	41.3	40.3	39.3	38.3	37.3	36.4	35.6	34.7	33.9	33.1	32.4	31.6	30.9	30.2	29.6	28.9	28.3	27.7
213	97	48.1	46.8	45.5	44.3	43.1	42.0	40.9	39.9	38.9	37.9	37.0	36.1	35.2	34.4	33.6	32.8	32.0	31.3	30.6	29.9	29.3	28.7	28.0	27.4
211	96	47.6	46.3	45.0	43.8	42.7	41.6	40.5	39.4	38.5	37.5	36.6	35.7	34.8	34.0	33.2	32.4	31.7	31.0	30.3	29.6	29.0	28.4	27.7	27.2
209	95	47.1	45.8	44.6	43.4	42.2	41.1	40.1	39.0	38.1	37.1	36.2	35.3	34.5	33.7	32.9	32.1	31.4	30.7	30.0	29.3	28.7	28.1	27.5	26.9
207	94	46.6	45.3	44.1	42.9	41.8	40.7	39.6	38.6	37.7	36.7	35.8	34.9	34.1	33.3	32.5	31.8	31.0	30.3	29.7	29.0	28.4	27.8	27.2	26.6
205	93	46.1	44.8	43.6	42.5	41.3	40.3	39.2	38.2	37.3	36.3	35.4	34.6	33.7	33.0	32.2	31.4	30.7	30.0	29.4	28.7	28.1	27.5	26.9	26.3
202	92	45.6	44.4	43.2	42.0	40.9	39.8	38.8	37.8	36.9	35.9	35.1	34.2	33.4	32.6	31.8	31.1	30.4	29.7	29.0	28.4	27.8	27.2	26.6	26.0
200	91	45.1	43.9	42.7	41.5	40.4	39.4	38.4	37.4	36.5	35.5	34.7	33.8	33.0	32.2	31.5	30.8	30.1	29.4	28.7	28.1	27.5	26.9	26.3	25.7
198	90	44.6	43.4	42.2	41.1	40.0	39.0	37.9	37.0	36.1	35.2	34.3	33.5	32.7	31.9	31.1	30.4	29.7	29.1	28.4	27.8	27.2	26.6	26.0	25.5
196	89	44.1	42.9	41.8	40.6	39.6	38.5	37.5	36.6	35.7	34.8	33.9	33.1	32.3	31.5	30.8	30.1	29.4	28.7	28.1	27.5	26.9	26.3	25.7	25.2
194	88	43.6	42.4	41.3	40.2	39.1	38.1	37.1	36.2	35.3	34.4	33.5	32.7	31.9	31.2	30.4	29.7	29.1	28.4	27.8	27.2	26.6	26.0	25.4	24.9
191	87	43.1	42.0	40.8	39.7	38.7	37.7	36.7	35.7	34.9	34.0	33.2	32.3	31.6	30.8	30.1	29.4	28.7	28.1	27.5	26.9	26.3	25.7	25.1	24.6
189	86	42.7	41.5	40.3	39.3	38.2	37.2	36.3	35.3	34.4	33.6	32.8	32.0	31.2	30.5	29.8	29.1	28.4	27.8	27.1	26.5	26.0	25.4	24.9	24.3
187	85	42.2	41.0	39.9	38.8	37.8	36.8	35.8	34.9	34.0	33.2	32.4	31.6	30.8	30.1	29.4	28.7	28.1	27.4	26.8	26.2	25.7	25.1	24.6	24.0
185	84	41.7	40.5	39.4	38.3	37.3	36.4	35.4	34.5	33.6	32.8	32.0	31.2	30.5	29.8	29.1	28.4	27.7	27.1	26.5	25.9	25.4	24.8	24.3	23.8
183	83	41.2	40.0	38.9	37.9	36.9	35.9	35.0	34.1	33.2	32.4	31.6	30.9	30.1	29.4	28.7	28.1	27.4	26.8	26.2	25.6	25.1	24.5	24.0	23.5
180	82	40.7	39.5	38.5	37.4	36.4	35.5	34.6	33.7	32.8	32.0	31.2	30.5	29.8	29.1	28.4	27.7	27.1	26.5	25.9	25.3	24.8	24.2	23.7	23.2
178	81	40.2	39.1	38.0	37.0	36.0	35.1	34.2	33.3	32.4	31.6	30.9	30.1	29.4	28.7	28.0	27.4	26.8	26.1	25.6	25.0	24.5	23.9	23.4	22.9
176	80	39.7	38.6	37.5	36.5	35.6	34.6	33.7	32.9	32.0	31.3	30.5	29.7	29.0	28.3	27.7	27.0	26.4	25.8	25.2	24.7	24.2	23.6	23.1	22.6
174	79	39.2	38.1	37.1	36.1	35.1	34.2	33.3	32.5	31.6	30.9	30.1	29.4	28.7	28.0	27.3	26.7	26.1	25.5	24.9	24.4	23.8	23.3	22.8	22.4
172	78	38.7	37.6	36.6	35.6	34.7	33.8	32.9	32.1	31.2	30.5	29.7	29.0	28.3	27.6	27.0	26.4	25.8	25.2	24.6	24.1	23.5	23.0	22.5	22.1
169	77	38.2	37.1	36.1	35.2	34.2	33.3	32.5	31.6	30.8	30.1	29.3	28.6	27.9	27.3	26.6	26.0	25.4	24.9	24.3	23.8	23.2	22.7	22.3	21.8
167	76	37.7	36.7	35.7	34.7	33.8	32.9	32.0	31.2	30.4	29.7	29.0	28.3	27.6	26.9	26.3	25.7	25.1	24.5	24.0	23.5	22.9	22.4	22.0	21.5
165	75	37.2	36.2	35.2	34.2	33.3	32.5	31.6	30.8	30.0	29.3	28.6	27.9	27.2	26.6	26.0	25.4	24.8	24.2	23.7	23.1	22.6	22.2	21.7	21.2
163	74	36.7	35.7	34.7	33.8	32.9	32.0	31.2	30.4	29.6	28.9	28.2	27.5	26.9	26.2	25.6	25.0	24.4	23.9	23.4	22.8	22.3	21.9	21.4	20.9
161	73	36.2	35.2	34.2	33.3	32.4	31.6	30.8	30.0	29.2	28.5	27.8	27.1	26.5	25.9	25.3	24.7	24.1	23.6	23.0	22.5	22.0	21.6	21.1	20.7
158	72	35.7	34.7	33.8	32.9	32.0	31.2	30.4	29.6	28.8	28.1	27.4	26.8	26.1	25.5	24.9	24.3	23.8	23.2	22.7	22.2	21.7	21.3	20.8	20.4
156	71	35.2	34.2	33.3	32.4	31.6	30.7	29.9	29.2	28.4	27.7	27.1	26.4	25.8	25.2	24.6	24.0	23.5	22.9	22.4	21.9	21.4	21.0	20.5	20.1
154	70	34.7	33.8	32.8	32.0	31.1	30.3	29.5	28.8	28.0	27.3	26.7	26.0	25.4	24.8	24.2	23.7	23.1	22.6	22.1	21.6	21.1	20.7	20.2	19.8
152	69	34.2	33.3	32.4	31.5	30.7	29.9	29.1	28.4	27.6	27.0	26.3	25.7	25.0	24.4	23.9	23.3	22.8	22.3	21.8	21.3	20.8	20.4	19.9	19.5
150	68	33.7	32.8	31.9	31.0	30.2	29.4	28.7	27.9	27.2	26.6	25.9	25.3	24.7	24.1	23.5	23.0	22.5	22.0	21.5	21.0	20.5	20.1	19.7	19.2
147	67	33.2	32.3	31.4	30.6	29.8	29.0	28.3	27.5	26.8	26.2	25.5	24.9	24.3	23.7	23.2	22.6	22.1	21.6	21.1	20.7	20.2	19.8	19.4	19.0
145	66	32.7	31.8	31.0	30.1	29.3	28.6	27.8	27.1	26.4	25.8	25.1	24.5	24.0	23.4	22.8	22.3	21.8	21.3	20.8	20.4	19.9	19.5	19.1	18.7
143	65	32.2	31.3	30.5	29.7	28.9	28.1	27.4	26.7	26.0	25.4	24.8	24.2	23.6	23.0	22.5	22.0	21.5	21.0	20.5	20.1	19.6	19.2	18.8	18.4
141	64	31.7	30.9	30.0	29.2	28.4	27.7	27.0	26.3	25.6	25.0	24.4	23.8	23.2	22.7	22.1	21.6	21.1	20.7	20.2	19.8	19.3	18.9	18.5	18.1
139	63	31.2	30.4	29.6	28.8	28.0	27.3	26.6	25.9	25.2	24.6	24.0	23.4	22.9	22.3	21.8	21.3	20.8	20.3	19.9	19.4	19.0	18.6	18.2	17.8
136	62	30.7	29.9	29.1	28.3	27.6	26.8	26.1	25.5	24.8	24.2	23.6	23.1	22.5	22.0	21.5	21.0	20.5	20.0	19.6	19.1	18.7	18.3	17.9	17.5
134	61	30.3	29.4	28.6	27.8	27.1	26.4	25.7	25.1	24.4	23.8	23.2	22.7	22.1	21.6	21.1	20.6	20.1	19.7	19.3	18.8	18.4	18.0	17.6	17.3
132	60	29.8	28.9	28.1	27.4	26.7	26.0	25.3	24.7	24.0	23.4	22.9	22.3	21.8	21.3	20.8	20.3	19.8	19.4	18.9	18.5	18.1	17.7	17.3	17.0
130	59	29.3	28.5	27.7	26.9	26.2	25.5	24.9	24.2	23.6	23.0	22.5	21.9	21.4	20.9	20.4	19.9	19.5	19.0	18.6	18.2	17.8	17.4	17.1	16.7
128	58	28.8	28.0	27.2	26.5	25.8	25.1	24.5	23.8	23.2	22.7	22.1	21.6	21.0	20.5	20.1	19.6	19.2	18.7	18.3	17.9	17.5	17.1	16.8	16.4
125	57	28.3	27.5	26.7	26.0	25.3	24.7	24.0	23.4	22.8	22.3	21.7	21.2	20.7	20.2	19.7	19.3	18.8	18.4	18.0	17.6	17.2	16.8	16.5	16.1
123	56	27.8	27.0	26.3	25.6	24.9	24.2	23.6	23.0	22.4	21.9	21.3	20.8	20.3	19.8	19.4	18.9	18.5	18.1	17.7	17.3	16.9	16.5	16.2	15.8
121	55	27.3	26.5	25.8	25.1	24.4	23.8	23.2	22.6	22.0	21.5	21.0	20.4	20.0	19.5	19.0	18.6	18.2	17.8	17.4	17.0	16.6	16.2	15.9	15.6
119	54	26.8	26.0	25.3	24.7	24.0	23.4	22.8	22.2	21.6	21.1	20.6	20.1	19.6	19.1	18.7	18.3	17.8	17.4	17.0	16.7	16.3	15.9	15.6	15.3
117	53	26.3	25.6	24.9	24.2	23.6	22.9	22.3	21.8	21.2	20.7	20.2	19.7	19.2	18.8	18.3	17.9	17.5	17.1	16.7	16.4	16.0	15.7	15.3	15.0
114	52	25.8	25.1	24.4	23.7	23.1	22.5	21.9	21.4	20.8	20.3	19.8	19.3	18.9	18.4	18.0	17.6	17.2	16.8	16.4	16.0	15.7	15.4	15.0	14.7
112	51	25.3	24.6	23.9	23.3	22.7	22.1	21.5	21.0	20.4	19.9	19.4	19.0	18.5	18.1	17.6	17.2	16.8	16.5	16.1	15.7	15.4	15.1	14.7	14.4
110	50	24.8	24.1	23.5	22.8	22.2	21.6	21.1	20.5	20.0	19.5	19.1	18.6	18.1	17.7	17.3	16.9	16.5	16.1	15.8	15.4	15.1	14.8	14.5	14.1
108	49	24.3	23.6	23.0	22.4	21.8	21.2	20.7	20.1	19.6	19.1	18.7	18.2	17.8	17.4	17.0	16.6	16.2	15.8	15.5	15.1	14.8	14.5	14.2	13.9
106	48	23.8	23.1	22.5	21.9	21.3	20.8	20.2	19.7	19.2	18.8	18.3	17.8	17.4	17.0	16.6	16.2	15.9	15.5	15.1	14.8	14.5	14.2	13.9	13.6
103	47	23.3	22.7	22.0	21.5	20.9	20.3	19.8	19.3	18.8	18.4	17.9	17.5	17.1	16.7	16.3	15.9	15.5	15.2	14.8	14.5	14.2	13.9	13.6	13.3
101	46	22.8	22.2	21.6	21.0	20.4	19.9	19.4	18.9	18.4	18.0	17.5	17.1	16.7	16.3	15.9	15.5	15.2	14.9	14.5	14.2	13.9	13.6	13.3	13.0
99	45	22.3	21.7	21.1	20.5	20.0	19.5	19.0	18.5	18.0	17.6	17.1	16.7	16.3	15.9	15.6	15.2	14.9	14.5	14.2	13.9	13.6	13.3	13.0	12.7
97	44	21.8	21.2	20.6	20.1	19.6	19.0	18.6	18.1	17.6	17.2	16.8	16.4	16.0	15.6	15.2	14.9	14.5	14.2	13.9	13.6	13.3	13.0	12.7	12.4
95	43	21.3	20.7	20.2	19.6	19.1	18.6	18.1	17.7	17.2	16.8	16.4	16.0	15.6	15.2	14.9	14.5	14.2	13.9	13.6	13.3	13.0	12.7	12.4	12.2
92	42	20.8	20.3	19.7	19.2	18.7	18.2	17.7	17.3	16.8	16.4	16.0	15.6	15.2	14.9	14.5	14.2	13.9	13.6	13.3	13.0	12.7	12.4	12.1	11.9
90	41	20.3	19.8	19.2	18.7	18.2	17.7	17.3	16.8	16.4	16.0	15.6	15.2	14.9	14.5	14.2	13.9	13.5	13.2	12.9	12.7	12.4	12.1	11.9	11.6
88	40	19.8	19.3	18.8	18.3	17.8	17.3	16.9	16.4	16.0	15.6	15.2	14.9	14.5	14.2	13.8	13.5	13.2	12.9	12.6	12.3	12.1	11.8	11.6	11.3

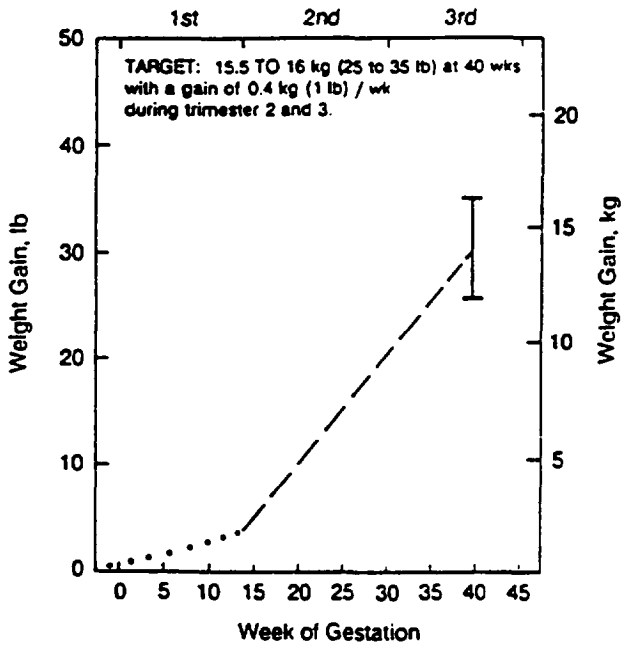
[a] BMI (metric) = (kg/m^2) × 100 ; BMI (English) = (lb/in.^{2}) × 100.
BMI (metric) × 0.142 = BMI (English); BMI (English) × 7 = BMI (metric).

Appendix B

Provisional Weight Gain Charts by Prepregnancy Body Mass Index (BMI)

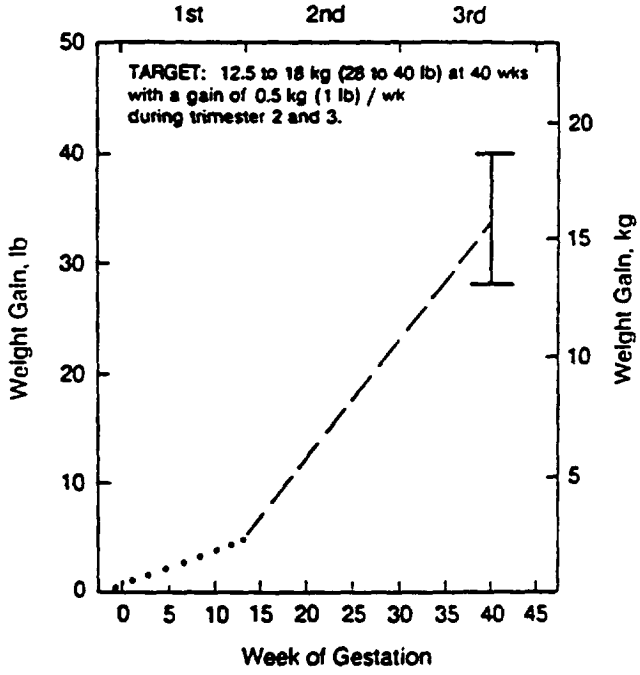
from: IOM/NAS, 1990

A. For Normal Weight Women with BMI of 19.8 to 26.0 (Metric)[a]

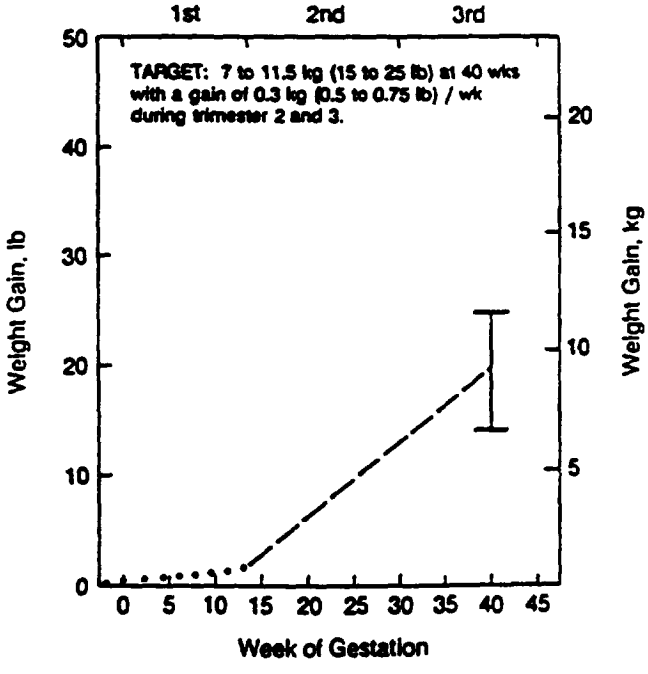


[a]Assumes a 1.6-kg (3.5-lb) gain in first trimester and the remaining gain at a rate of 0.44 kg (0.97 lb) per week.
[b]Assumes a 2.3-kg (5-lb) gain in first trimester and the remaining gain at a rate of 0.49 kg (1.07 lb) per week.
[c]Assumes a 0.9-kg (2-lb) gain in first trimester and the remaining gain at a rate of 0.3 kg (0.67 lb) per week.

B. For Underweight Women with BMI Less Than 19.8 (Metric)[b]



C. For Overweight Women with BMI of >26.0 to 29.0 (Metric)[c]



Technical Paper 3

Overweight and Obesity in Pregnancy

Technical Paper #3 prepared for the U.S. Department of Agriculture, Food and Nutrition Service, under Cooperative Agreement #58-3198-1-005, "Review of the Nutritional Risk Criteria for the WIC Program", with the Department of Family and Community Medicine, University of Arizona, by April H. Dean, MS, RD, and Gail G. Harrison, PhD. Ms. Dean is Research Associate and a doctoral candidate in Nutritional Sciences; Dr. Harrison is Professor of Family and Community Medicine, College of Medicine, University of Arizona, Tucson, AZ 85724.

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INTRODUCTION

Overweight and obesity are highly prevalent among U.S. adults; therefore the implications of these conditions for nutritional risk in pregnancy are of interest in targeting of the WIC program, designed to reach low-income and medically/nutritionally at-risk pregnant women, postpartum women, infants and children. This technical paper will review the available information in the literature and current recommendations to address the following issues:

- What are the risks of overweight and obesity in pregnancy?
- How should overweight and obesity be defined for pregnant women?
- What is the optimal weight gain in pregnancy for the overweight and obese woman?
- Nutritional management of obesity during pregnancy.

DEFINITIONS AND MEASUREMENT

There are a variety of definitions for overweight and obesity available in the literature and in clinical use. Strictly speaking, overweight refers to body weight relative to height which is in excess of that determined to be optimal for a particular set of health outcomes or mortality risk; obesity refers to an excess of body fat, again relative to a standard range determined to be optimal for a defined outcome or risk. Measurement of body fat is much less frequently accomplished in either survey or clinical contexts than are height and weight measures, although skinfold thickness measures are available for large enough reference populations to use them as clinical criteria.

In common clinical and epidemiologic use, relative weight is used as a continuous variable with the term "overweight" used to refer to milder and moderate degrees of excess weight for height and "obesity" used to refer to extremely high weight for height. This convention is probably reasonably valid, since the correlation of relative weight with body fatness is sufficiently high particularly at the extreme ends of the continuum to justify the assumption of excess body fat in the severely overweight individual.

Measures of relative weight in common use include weight as a percent of a standard weight for a given range of height, derived from a reference population, and body mass index (BMI), which is calculated as weight in kilograms divided by height in meters with height raised to an exponent which is variably between 1.5 and 2.0. The calculation is designed to minimize or remove the effect of the fact that height and weight are highly correlated with one another. The Quetelet Index (kg/m^2) is the most commonly used BMI, while the National Center for Health Statistics uses a BMI calculated as $kg/m^{1.5}$ for women.

Evaluation of obesity for the pre-gravid woman is complicated by the fact that none of the available standards are designed to reflect levels of relative weight or fatness at which the risk for complications of pregnancy is elevated. Rather, they are either normative data based on the distribution of weight-for-height or fatness in the U.S. population (e.g., NHANES data), or they are developed to reflect lowest mortality risk among insured adults (i.e., Metropolitan Life Insurance Company standards).

The available literature has not used consistent standards for defining obesity or overweight in pregnant women (Garbaciak et al., 1980; Gross et al., 1980; Abrams and Parker, 1988;

Naeye, 1990). The least interpretable index is used by Gross et al. (1980) of "maximum pregnancy weight" since it does not take into account prepregnant weight vs. weight gain during pregnancy. The two most recent comprehensive studies of pregnancy risk and relative weight have used BMI (Quetelet's Index) criteria (Naeye, 1990 and Abrams and Parker, 1988) with relatively arbitrary but fairly congruent cutoff points.

A recent Institute of Medicine report on Nutrition and Pregnancy (IOM, 199) provides the current recommended criterion for classification of overweight and obesity. Obesity is defined, using the Quetelet method, as a BMI (kg/m^2) greater than 29.0 while overweight is defined as BMI between 26.0 and 29.0. For comparative purposes, weight-for-height at 120 percent of the Metropolitan Life Insurance population corresponds to a BMI Index of 25.6, and 135 percent of standard corresponds to 28.9.

PREVALENCE OF OVERWEIGHT AND OBESITY AMONG WOMEN OF CHILDBEARING AGE

Estimates of the prevalence of overweight and/or obesity vary with the definitional criteria used. If weight-for-height at least 120 percent of life insurance reference tables is used as a criterion for obesity (e.g., Garbaciak et al., 1985), then nearly 20 percent of reproductive-aged women in the U.S. can be considered overweight (Varner, 1985). Using BMI $\geq$85th percentile of national reference data for women in their 20s to define overweight and BMI $\geq$95th percentile to define severe overweight, 27 percent of women in the U.S. are overweight and approximately 11 percent are severely overweight (Najjar, 1987).

All available data indicate that the prevalence of overweight and obesity among women in developed countries is inversely related to income and social class (Stunkard, 1988; Goldblatt et al., 1965). Thus we may expect the prevalence of these conditions to be higher among WIC-eligible women than among women of comparable age in the general population. Stockbauer (1987) reported that 18.1 percent of the Missouri WIC prenatal participants had prepregnancy weights greater than or equal to 20 percent over standard weight for height, compared to 13.5 percent in non-WIC women in the same state. Overweight is also non-randomly distributed in the population with respect to age, education, ethnic group, and region. Data from the NHANES II survey and the Hispanic Health and Nutrition Survey (HHNS, 1982-84) show that among the ethnic groups studied Black women had the high highest prevalence of overweight, followed by Mexican-American and Puerto Rican women; the lowest prevalence was seen in non-Hispanic White women, followed by Cuban women (Life Sciences Research Office, 1989). Mean BMI was higher in both Black and White women with lower educational levels than among more educated women (LSRO, 1989). Gillum (1987), also analyzing NHANES II data, reported that rural and southern Black women aged 25-74 years were more overweight than Black women residing in urban areas and in the northern and western regions. The prevalence of obesity and overweight increases with increasing age among women during their childbearing years (Naeye, 1990).

RISKS OF OBESITY IN PREGNANCY

The physiological stress of obesity is manifested in increased rates of hypertension (NIH, 1979), impaired respiratory function (Ray, 1983), gallbladder disease (Bray, 1985) and diabetes (Topeller et al., 1982). It is not surprising that the added metabolic demands of pregnancy may predispose to various forms of increased risk. Some of the reported

complications of obesity in pregnancy include higher rates of preeclampsia, hypertension, diabetes mellitus, thromboembolic disease, urinary tract infections, Caesarean delivery, therapeutic induction of labor, prolonged second stage of labor, prolonged gestation (>42 weeks), and perinatal mortality. Schramm (1981) reported that overweight (>120 percent ideal weight for height) women in Missouri had increased essential and pregnancy-induced hypertension rates, risk of diabetes, abnormalities of labor, twins, and fetal deaths.

Abrams and Parker (1988) have recently summarized results from their own work and from two other large investigations to determine the relative risks of certain complications of pregnancy due to moderate and severe overweight. They concluded that even moderately overweight women were at higher risk than average for diabetes, pregnancy-induced hypertension, and primary Caesarean deliveries, although severely overweight women bear still higher risks. Very overweight women were 6.5 times more likely to manifest diabetes during pregnancy and 1.9 times as likely to develop pregnancy-induced hypertension than women of average weight. Very overweight women experienced increased risk of 85 percent for perinatal mortality, 60 percent for Caesarean delivery and 42 percent for urinary tract infections; the relative risk for anemia for very overweight women was decreased (0.67) compared to women of average weight.

Early studies (Douglas and Scadron, 1951; Fisher and Frey, 1958) indicated that despite higher risk of complications during pregnancy, perinatal mortality did not increase with maternal obesity. More recent and comprehensive studies, however, contradict this conclusion and indicate substantially increased mortality risk. A report based on data from the National Natality Survey (NNS) (Taffel, 1986) found that fetal death rates were highest among the heaviest women (>72 kg). Garbaciak et al. (1985) investigated the effect of obesity on pregnancy outcome in 9,667 patients, and found significant increases in perinatal mortality among obese and morbidly obese women with antenatal complications; among obese women with and without complications there were increased rates of primary Caesarean delivery and increased mean birth weights compared to nonobese women. Rahaman et al. (1990) reported similar findings in a study examining outcome of pregnancy in 300 obese (BMI>30) women. Perinatal loss was ten times greater among the obese mothers than among nonobese controls, with diabetes, pre-eclampsia and advanced maternal age largely contributing to the difference. These authors also concluded that the obese woman without other risk factors or complications of pregnancy bears no elevation of risk in terms of fetal outcome.

A recent study provides some evidence of a relationship between maternal obesity and decreased viability of infants born pre-term. Lucas and colleagues (1988) reported that maternal fatness was second in importance only to length of gestation in predicting the death of infants born preterm. They also found that premature infants had decreasing birth weight for gestational age with increasing maternal fatness, a finding which is somewhat surprising given the well-established relationship between maternal fatness and increased birth weight and risk of macrosomia in term infants (Calandra et al., 1981).

Naeye (1990) examined 56,857 pregnancies from the National Collaborative Perinatal Study and concluded that perinatal mortality rates progressively increased with increases in maternal pregravid relative body weight. Much of the increase was due to preterm deliveries between 24 and 30 weeks gestation, which were often caused by acute chorioamnionitis. A greater frequency of dizygotic twins was observed with increasing

maternal relative weight, which contributed somewhat to the increase in preterm deliveries. This study confirmed the findings of Lucas et al. (1988) that perinatal mortality rates for pre-term infants born to obese mothers progressively increase with increasing maternal preconceptual weight. The mortality rate for infants born to obese mothers was 121/1000 births compared to infants of thin mothers, who experienced the lowest mortality rate of 37/1000. Major congenital malformations also increased with relative maternal body weight, and these effects persisted after several risk factors were taken into consideration analytically. This same analysis identified a correlation between perinatal mortality risk and low socioeconomic status, which disappeared when relative maternal body weight and other risk factors such as cigarette smoking were taken into account. An observed correlation between perinatal mortality and ethnicity (Black women higher than White) also nearly disappeared when these risk factors were taken into account.

NUTRITIONAL MANAGEMENT OF PREGNANCY IN THE OBESE WOMAN: WEIGHT GAIN AND DIETARY RISK

Several investigators (Abrams and Parker, 1988; Garbaciak et al., 1985; Johnson et al., 1987) have pointed out that because of the greater risk of complications, adequate prenatal care including early screening and treatment for diabetes and hypertension is particularly important for overweight women. The overweight pregnant woman should also routinely be screened for urinary tract infections, since she carries a 9 percent greater risk than the average woman. Accurate establishment of gestational age is more difficult in the obese woman but is particularly important because of increased risk of ineffective labor (Johnson et al., 1987).

Average birth weight tends to be elevated in the offspring of obese women (Rossner and Ohlin, 1990; Garbaciak, 1985; Calandra et al., 1981; Harrison et al., 1981) and the risk of macrosomia (infant birth weight >4000 g) increases, increasing the risk for Caesarean delivery and/or birth trauma. However, one recent study (Rossner and Ohlin, 1990) concluded that increasing birth weight with maternal relative weight only held true up to a maternal BMI of 24 kg/m^2. At higher relative weights, birth weight did not increase with maternal weight.

The risk of low weight gain and even lack of weight gain is elevated in severely obese women, but the relative effect on newborn size is buffered compared to nonobese women (Harrison et al., 1981). An analysis of the 1980 NNS data (Kleinman, 1990) showed mean gestational weight gain decreased as maternal prepregnancy BMI increased, and the variation in gain increased. More than 10 percent of very overweight women lost weight during pregnancy, and approximately one-third had "low" weight gain of 15 pounds or less, a four-fold measure over women with normal prepregnancy BMI.

Dietary patterns and nutritional intake in obese and overweight pregnant women require particular attention, and may have been generally neglected on an invalid assumption that the overweight are adequately nourished. Studies of nonpregnant overweight women indicate a prevalence of dieting of more than 50 percent (Forman et al., 1986); a high risk for dieting among overweight pregnant women is reflected in elevated risk for low or even negative weight gains during pregnancy among the obese (Harrison et al., 1981). Pregnant women who smoke, like nonpregnant adults who smoke, have been shown to have less nutrient dense diets than nonsmokers (Aaronson and Macnee, 1989); thus the overweight

pregnant woman who smokes may represent a particularly at-risk situation in terms of nutritional inadequacy.

There is some evidence (Borberg et al., 1980) that non-diabetic, obese pregnant women receiving dietary advice from a dietitian were able to limit the increase in insulin concentrations of late pregnancy when compared with obese pregnant women not receiving dietary information.

OPTIMAL AND RECOMMENDED WEIGHT GAINS FOR OBESE WOMEN

Naeye (1979) reported improved perinatal outcome in moderately obese individuals (> 135 percent ideal body weight) who limited gestational weight gain to 16 pounds. Winick (1986), in contrast, recommended 20- to 27-pound weight gains for overweight women. Ratner et al. (1991) have recently examined the effects of varying pregnancy weight gain in women with prepregnancy weight greater than 160 percent of ideal, all of whom received nutrition counseling in pregnancy. They found the incidence of primary Caesarean delivery to be greater for obese women who gained more than 24 pounds, and concluded that a gestational weight gain of only 10 pounds in the morbidly obese minimized maternal risks without increasing ketonuria or intrauterine growth retardation. The recent Institute of Medicine report (IOM, 1990) recommends a minimum gain of 6 kilograms for obese women.

SUMMARY

The incidence of obesity and related health problems is increasing in the United States. This is particularly a problem among women and minority populations. Women of low socioeconomic status and lower educational levels are more likely to be obese than those of higher socioeconomic and education levels.

Obesity can be a serious health risk during pregnancy. It is associated with higher rates of preeclampsia, hypertension, diabetes mellitus, urinary tract infection, acute chorioamnionitis, dizygotic twins, major malformations, and Caesarean delivery. Infants born to obese women are more likely to be macrosomic, which increases their likelihood of experiencing birth trauma. Several researchers report increases in perinatal mortality. Preterm infants born to obese mothers often have small birth weights for their gestational ages and are less likely to survive. Fetal death rate is highest among the heaviest women. The risk associated with obesity increases progressively from women who are very thin pregravid to those who are obese. There appears to be no clear threshold, and thus cutoff points of weight-for-height or BMI are arbitrary classifications in this respect.

Adequate and early prenatal care which includes early screening and treatment for diabetes and hypertension are especially important for obese women. Some evidence suggests that education concerning an appropriate, balanced diet during pregnancy can positively affect some of the risk factors of this condition.

Weight gain during pregnancy is generally lower, and is more variable for obese than normal-weight women. Researchers disagree about the appropriate amount of weight an obese woman should gain during pregnancy. While there is some disagreement about the optimal pregnancy weight gain for the obese woman, the current recommendation from a

recent Institute of Medicine study is to encourage weight gains of at least 6 kg (15 pounds).

Little is known about the nutrient intakes of obese women in their childbearing years. There is a great need for further research defining the role of food/nutrient intake in modifying the complications of pregnancy observed in overweight women, and in understanding the dietary behavior of overweight and obese women during pregnancy.

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Technical Paper 4

Overweight and Obesity in Infants and Children

Technical Paper #4 prepared for the U.S. Department of Agriculture under Cooperative Agreement #58-3198-1-005, "Review of the Nutritional Risk Criteria for the WIC Program", with the Department of Family and Community Medicine, University of Arizona, Tucson, AZ 85724 by April H. Dean, MS, RD, Research Associate. Ms. Dean is a doctoral candidate in the program of Nutritional Sciences, University of Arizona.

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INTRODUCTION

The purpose of this technical paper is to review current published information and scientific opinion on several issues related to obesity in early childhood, namely trends in prevalence, consequences and health risks, etiology, and prevention and management strategies.

TRENDS IN PREVALENCE OF OBESITY IN CHILDREN

There is evidence that the problem of pediatric obesity in the United States is increasing at an alarming rate. Depending on the criteria used to define obesity, which at best are arbitrary in children, as many as 25% of children are affected. Gortmaker et al. (1987) have presented convincing evidence for the increasing prevalence. Defining obesity as triceps skinfold thickness greater than the 85th percentile and severe obesity as triceps skinfold greater than the 95th percentile of reference data from the National Health Examination Survey Cycles II and III (1963, 1965-1970), they compared information collected from the NHANES I (1970-1973) and NHANES II (1975-1980). Over the 17-year period, the prevalence of obesity increased by 54% in children aged six to eleven years and by 39% in adolescents. The prevalence of severe obesity increased by 95% in children and 64% in adolescents. Increased prevalence characterized all socioeconomic categories and occurred in Blacks as well as Whites, although obesity was less prevalent among Black children than among Whites throughout the time period.

Analysis of the Hispanic Health and Nutrition Survey (HHANES) data reveals that the increased risk for obesity among Mexican-Americans begins early. Mexican-American children are heavier than non-Hispanic White and Black children at all ages (Martorell et al., 1989). A tendency toward greater relative deposition of fat on the trunk in Mexican-American compared to non-Hispanic White children has been documented in children as young as one year of age (Kautz and Harrison, 1981). Overweight in Native American children has also increased dramatically in the last several years (Sugarman et al., 1990).

CONSEQUENCES OF OBESITY FOR CHILDREN

Metabolic Changes

While the health risks associated with obesity in adults are well documented and include noninsulin dependent diabetes mellitus (Kissebah et al., 1989; Lew and Garfinkel, 1979), several types of cancer (Lew and Garfinkel, 1979), hypertension, hypercholesterolemia, and hypertriglyceridemia (NIH, 1985), such risks are not as extensively documented for children. Severe obesity in both groups is associated with increased risk of atherosclerotic heart disease (Keys, 1980). Rosenbaum and Leibel (1989) have reported that obese children manifest many of the same disturbances as obese adults, including hyperinsulinism, hyperlipidemia, and hypertension (Court et al., 1974; Rosenbaum and Leibel, 1988). Obese children are generally tall for age, mature early (Wolff, 1955) and may have advanced bone age (Garn, 1976). Although there are conflicting reports (Cooper et al., 1990), obese children have been found to be less physically fit than the nonobese (Zanconato et al., 1989). They may have increased risk of respiratory problems (Sommerville et al., 1984).

In an attempt to identify children at greatest risk for future development of cardiovascular disease, a recent report of an expert panel on blood cholesterol levels in children and adolescents by the National Cholesterol Education Program (1991) outlined screening recommendations for children greater than 2 years of age. The panel recommends selective lipoprotein or total cholesterol screening of children with the following risk factors:

1. A family history of premature cardiovascular disease, which was defined as having a parent or grandparent age 55 years or younger who were found to have coronary atherosclerosis diagnosed by coronary arteriography.

2. Parents or grandparents 55 years of age or less who suffered a documented myocardial infarction, angina pectoris, peripheral vascular disease, cerebrovascular disease, or sudden cardiac death.

3. A parent with high blood cholesterol (240 mg/dL or higher).

4. Other risk factors, particularly when familial history is not available. Risk factors include: obesity (weight for height $\geq$ 95th percentile), hypertension, diabetes mellitus and physical inactivity.

Table 1 lists the panel's recommendations concerning classification of total and LDL-cholesterol levels in children at risk.

Table 1

Classification of Total and LDL-Cholesterol Levels
in Children and Adolescents from Families with
Hypercholesterolemia or Premature Cardiovascular Disease

Category	Total Cholesterol	LDL Cholesterol
Acceptable	< 170 mg/dL	< 110 mg/dL
Borderline	170-199 mg/dL	$\geq$ 130 mg/dL
High	$\geq$ 200 mg/dL	$\geq$ 130 mg/dL

The long-term risks of obesity in children have primarily to do with increased risk of remaining obese into adulthood, carrying the metabolic disturbances which predispose to increased risk of premature atherosclerotic heart disease, diabetes, and possibly cancer. The short-term risks of obesity in children are mostly psychosocial.

Risk of Persistence of Obesity

Infant fatness at birth does not predict fatness at age one year (Whitelaw, 1977), and a high level of fatness in infancy is not a strong predictor of obesity in childhood, although

the risk may be slightly higher than random (Peck and Ullrich, 1985). By early childhood, tracking of relative fatness is stronger, and increases with age. Obese children are more likely to become obese adults than are normal-weight or underweight children (Abraham et al., 1971; Rimm and Rimm, 1976). Stark et al. (1981) examined childhood and adult weight and height data from 5,362 children born in 1946 and concluded that the risk of overweight in adulthood was related to the degree of overweight in childhood and was about 40% for overweight seven-year-olds. Mossberg (1989) conducted a 40-year follow up study of obese children and found that while obese children had greater than normal weights as adults, the best predictors for adult obesity were family history of obesity and severe obesity at puberty. Freedman et al. (1987) examined the persistence of obesity and overweight over an eight-year period in the Bogalusa Heart Study. Subjects were children initially aged two to fourteen years. Of the 222 children who had initial triceps skinfold values above the 85th percentile of reference data, 43 percent were obese eight years later. Triceps skinfold relative values tracked most strongly in Black females ($r=0.64$) and somewhat less well for White females ($r=0.45$). The persistence of obesity was increased by initial age (greater than five years) and the severity of early obesity. Sorensen and Sonne-Holm (1988) also reported that severely overweight children are at a much elevated risk for adult obesity. There is a dearth of information on the persistence of obesity in children younger than age approximately three years, but what evidence there is suggests that risk of persistence is lower at younger ages.

Although the obese child has an elevated risk of adult obesity, most obese adults were not obese as children (Rimm and Rimm, 1976; Hartz and Rimm, 1980). That is, the prevalence of obesity in the U.S. population increases steadily with age after adolescence, and most adult obesity is not a consequence of childhood obesity.

Psychosocial Risks

Perhaps the most damaging effects of obesity in children are social and psychological in nature; they may have long-term consequences for self-esteem and social adjustment. The obese child's size may cause adults and other children to expect the child to behave in a manner appropriate for an older child. In addition, obese children experience various forms of discrimination. Children as young as five years old rank obese children as less desirable playmates than disabled or disfigured children (Staffieri, 1987; Richardson et al., 1961). The fat child is often teased or excluded from play (Wolff, 1962). Lack of participation in play activities may further promote obesity by limiting exercise; more important, feelings of inadequacy and poor self-esteem can result (Wadden et al., 1984). While there is debate about the universality of this phenomenon (Kaplan and Wadden, 1986), the psychosocial effects of childhood obesity are certainly damaging to at least some children.

ETIOLOGY OF CHILDHOOD OBESITY

The relative roles of genetic and environmental contributions to obesity in children are as yet undefined. It is well documented that obesity tends to run in families (Garn and Clark, 1976; Garn et al., 1989) and that children are most likely to be obese when both parents are obese, and least likely to develop obesity when both parents are lean. Price et al. (1989) showed that children of obese parents are more likely to develop persistent obesity than other children.

In spite of evidence for a significant genetic component to risk of developing obesity (Price et al., 1989; Stunkard et al., 1990), the increasing prevalence of obesity in the childhood population supports the assumption that environment plays a role (Gortmaker et al., 1987). The two likely candidates for environmental influence, of course, are food intake and physical activity.

Food Intake

Data from the NHANES I and NHANES II surveys show no secular change in mean energy intakes among children (Gortmaker et al., 1990), in spite of an increasing prevalence of obesity during the same period. Several studies have compared food intake of lean and obese subjects. Some have reported greater intakes in the obese (Waxman and Stunkard, 1980); however, most indicate that obese subjects do not eat significantly more than their lean peers (Rolland-Chachera and Bellisle, 1986; Frank et al., 1978). Most studies have been in adults (Keen et al., 1979; McCarty, 1966; Kromhout, 1983; Bingham et al., 1981), and adolescents (Johnson et al., 1956; Stefanick et al., 1959; Hamptom et al., 1967) and a few in infants (Vobecky et al., 1983; Mumford and Morgan, 1982). Vobecky et al. (1983) found that energy intake per kg body weight was lower in infants with relative weights >105% of expected weight than in leaner infants. The overwhelmingly consistent finding across a wide range of ages is fairly large variation in energy intakes within groups of both obese and lean subjects, including preschool children (Huenemann, 1974).

There are only a few studies which report dietary and nutrient intake patterns among obese children except for energy intake. Valoski and Epstein (1990) studied 21 eight-to-twelve-year-old children and found no significant differences between lean and obese children with respect to intakes of calories, iron, vitamin A, vitamin C, thiamin, or riboflavin. Rolland-Chachera and Bellisle (1986) found a significant correlation between degree of adiposity of one-to-three-year-old British children and percent of calories from protein in the diet, but not with percent of calories from fat. Frank et al. (1978) also reported higher protein intakes in a group of 185 ten-year-old children in the Bogalusa Heart Study with high body mass index; when triceps skinfold was the index for obesity, however, saturated fat intake was the only dietary component associated with increasing adiposity. Both Rolland-Chachera and Bellisle (1986) and Whitelaw et al. (1971) have speculated that the increased proportion of obesity among lower social class children in Britain may be related to relatively higher levels of carbohydrate intake.

Energy Expenditure

Lack of physical activity plays an important role in the development of obesity, although variation in physical activity alone does not explain within-sample differences in adiposity among school-aged children (Ross and Gilbert, 1985). Dietz and Gortmaker (1985) report a direct association between television viewing and obesity in children. The genetic component of energy expenditure may be significant as well. Griffiths and Payne (1976) showed that the resting metabolic rate of normal-weight four-year-old children of obese parents was ten percent lower than that of children of nonobese parents, even though the two groups of children had similar heights and weights. Roberts et al. (1988) reported that lower energy expenditure at three months of age was predictive of overweight at age one year.

Other Associated Factors

There are significant regional differences in prevalence of childhood obesity, with the highest rates in the Northeast and the Midwest, and in cities (Dietz and Gortmaker, 1984), independent of race and socioeconomic status. In developed countries including the U.S., women of low socioeconomic status are more likely to be obese than those of higher socioeconomic status (Sobel and Stunkard, 1989); a similar association has been observed in children (Stunkard et al., 1972).

MANAGEMENT OF OBESITY IN CHILDREN

Much uncertainty exists with regard to optimal management of obesity in children. The only major items of consensus are a) that diet regimes should be nutritionally adequate to support normal growth; b) that physical activity is important; and c) that models for management of obesity in adults cannot simply be transferred to children. There is some indication that family-based treatment is most effective, and that effective treatment of obesity in childhood may have lasting effects.

The Committee on Nutrition of the American Academy of Pediatrics (1981) states "caloric restriction to the point of weight loss should not be used for children, whose statural growth and central nervous system development could be impaired by a prolonged catabolic state. Growth of the child's lean mass should be supported, and the child's adipose mass should be held constant." The diet should be adequate in vitamins, minerals, and protein to meet requirements for growth, and growth in stature should be monitored as an indicator of dietary adequacy. The Committee warns against subjecting infants to low-fat milk feedings which may present excessively high renal solute loads. There is also some concern that low-fat or overly dilute feedings may cause a young child to learn behaviors inappropriate for future caloric regulation, such as increased volume ingestion (Committee on Nutrition, 1981).

Successful programs for the treatment of childhood obesity have emphasized appropriate diet, physical activity, self-esteem development, and behavior modification (Jonides, 1990); Epstein and Wing, 1987). Leung and Robow (1990) have pointed out that because of the strong familial link in the development of obesity in children, treatment programs for long term weight loss should involve the entire family. Every member of the family should support the overweight child and adjust eating and behavior habits to conform to the healthier lifestyle. Physical activity and decreasing sedentary time by limiting television viewing are particularly important components of the management of obesity (Gortmaker et al., 1990).

Valoski and Epstein (1990) reported an 18.3 percent average reduction in overweight while linear growth was not affected after six months of a family-based behavioral weight control program for 8-12 year old children. Diet, exercise and behavioral management were included in the program. Nutrition education was a key component of the program and emphasis was placed on nutrient density of foods. The same researchers reported similar success (Epstein et al., 1986) in 1-6 year old children. The diet was modified to increase iron and calcium intakes. Emphasis was placed on the basic four food groups and on making low calorie choices in each food group. A decrease in overweight of 15.4 percent was reported over a ten-week period.

Epstein et al. (1990) have demonstrated that effective treatment of obesity in children can produce effects into adulthood (Stunkard and Berkowitz, 1990). They conducted a ten-year follow up study of 75 obese six-to-twelve-year-old children who were treated by diet, exercise, and behavioral change weekly for eight weeks, followed by six monthly and three long-term follow up meetings. The dietary component emphasize nutritional foods along with elimination of high sugar and high fat foods. Children who participated in the program with their parents showed significantly greater decreases in percent overweight five and ten years later, compared with controls who attended meetings and received the information, but without behavioral change strategies including contracting, self-monitoring, social reinforcement, modeling, and contingency management. Over the long run, decreased intake of high calorie foods was predictive of success. Long-term successful weight loss by obese parents did not occur, but parental support of the child's efforts was critical to long-term success for the child.

Virtually all successful treatment programs for obese children emphasize nutrient dense foods and sensible eating plans including a variety of foods. More nutrient dense foods can replace less nutritious, calorically dense foods in the diet; by so doing, the child improves dietary quality while learning better eating habits.

SUMMARY

The prevalence of obesity among children in the U.S. has increased significantly in the last two decades. The problem is especially prevalent among Mexican-American and Native American children, but the increase in prevalence has affected all ethnic groups and socioeconomic levels. While most obese children do not become obese adults, they do bear an increased risk of adult obesity and its attendant health hazards, and obesity is a risk factor for elevated serum cholesterol even in early childhood.

Low levels of physical activity and family history of obesity seem to be the most important as predictors of obesity in children; overfeeding is much less important as an etiologic factor. Treatment of obesity in childhood requires a long-term approach including careful attention to nutritional quality of the diet in order to support normal growth and development and increased physical activity.

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Hematological Standards for Risk of Iron Deficiency

Technical Paper #5 prepared for the U.S. Department of Agriculture, Food and Nutrition Service, under Cooperative Agreement #58-3198-1-005, "Review of the Nutritional Risk Criteria for the WIC Program", with the Department of Family and Community Medicine, University of Arizona, Tucson, AZ 85724 by Marc J. Morse, MD, and Gail G. Harrison, PhD. At the time of preparation of this paper Dr. Morse was a fourth-year medical student at the University of Arizona. Dr. Harrison is Professor of Family and Community Medicine, College of Medicine, University of Arizona.

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INTRODUCTION

Nutritional anemias are conditions in which the hemoglobin content of the blood is lower than normal due to a deficit in a specific nutrient. The most common of these nutritional anemias is iron deficiency which often is caused by insufficient assimilation of iron from the diet, dilution of body iron by rapid growth, and blood loss (U.S. Department of Health and Human Services, 1988). Because outward manifestations of iron deficiency are rarely obvious, most cases are detected by laboratory tests done at a routine examination. While iron deficiency anemia can occur in all socioeconomic groups it has historically been most common among the poor. As a result, federal programs have paid special attention to iron nutrition. Among these programs is The Special Supplemental Food Program for Women, Infants, and Children (WIC). WIC is targeted to serve those socioeconomically disadvantaged groups at highest risk for iron deficiency namely, infants, children, and women, especially pregnant women. These high risk groups for iron deficiency have been widely studied in the last three decades. This technical paper will briefly review pertinent literature and hematological standards for the above mentioned groups, laboratory tests currently employed in diagnosing iron deficiency anemia, and factors that can cause changes in hematological indices.

INFANTS AND CHILDREN: ETIOLOGY AND PREVALENCE OF IRON DEFICIENCY, AND STANDARD CRITERIA FOR ANEMIA

Infants and children are at high risk for iron deficiency anemia. In infants, the prevalence of iron deficiency is highest between six months, when neonatal iron stores accumulated during fetal life are first likely to become depleted, and two to three years of age (Dallman, Siimes, and Stekel, 1980). During the preschool period, the composition of the diet is often unfavorable for iron absorption. This is particularly true in developing countries, but is also seen in developed countries due to the expense of iron rich foods or dietary practices that exclude them. Availability of dietary iron may be restricted by an excessive use of milk among preschool children which can displace other iron rich foods from the diet and can form insoluble complexes with iron due to high calcium and phosphorus content (Dallman, Siimes, and Stekel, 1980).

The prevalence of iron deficiency anemia among children in the U.S. has declined over the last several years. Evidence for this trend was provided by the second National Health and Nutrition Examination Survey conducted between 1976 and 1980 (Dallman, Yip and Johnson, 1984). From that large sample, only about 6% of infants between 1 and 2 years of age were anemic. As a result of this study, some have suggested that routine screening of hemoglobin or hematocrit in all infants in the United States is no longer necessary and that selective screening on the basis of a history to identify the children at risk be used (Dallman, 1987). Criteria for those children at risk include infants of low socioeconomic background, regular cow's milk started before six months of age, use of formula not fortified with iron, and low birth weight (Dallman, 1987). Pizarro et al. (1991) have recently observed a prevalence of iron deficiency at age nine months in normal infants fed cow's milk formula without added iron of 37.5%, in infants fed human milk 26.5%, and in infants fed iron-fortified cow's milk formula 8%.

Despite the seeming low prevalence from the NHANES II study, pockets of greater prevalence of iron deficiency still exist, since infants living below the poverty line were three times more likely to be iron deficient than those who were not economically disadvantaged (Expert Scientific Working Group, 1984). As recently as 1987, the Centers for Disease Control reported that 15.9% of low income children 6 to 24 months of age were anemic as defined by a hemoglobin concentration less than 11g/dl or a hematocrit less than 33% (Yip, 1989).

The Centers for Disease Control have recently published new reference criteria for use in clinical practice for public health and nutrition programs (Centers for Disease Control, 1989); these values represent the age-specific fifth percentile values for "healthy" persons from NHANES II (Pilch and Senti, 1984; Yip et al., 1984). These values are very close to those recommended by the American Academy of Pediatrics (1985).

1-1.9 years--11.0g/dl hemoglobin, 33% hematocrit
2-4.9 years--11.2g/dl hemoglobin, 34% hematocrit
5-7.9 years--11.4g/dl hemoglobin, 34.5% hematocrit

WOMEN OF CHILDBEARING AGE AND DURING PREGNANCY: ETIOLOGY AND CONSEQUENCES OF IRON DEFICIENCY, AND STANDARD CRITERIA FOR ANEMIA

Women of childbearing age, like infants and children, are also at increased risk for iron deficiency. In the NHANES II study, the percent prevalence of impaired iron status of women aged 15-19 and 20-44 were 7.2% and 6.3% respectively. These values are only slightly less than the 9.3% of children age 1-2 (Expert Scientific Working Group, 1984). Prevalence of anemia in the age 15-17 range was 5.9%, 3.3% for age 18-24, and 5.8% for those women aged 25-44 (Dallman, Yip and Johnson, 1984). The major factors that predispose to iron deficiency anemia in women of this age group are excessive blood loss during menstruation, blood donation, pregnancy, particularly closely spaced pregnancies, and inadequate dietary iron. Demographic risk factors include poverty, low education, high parity, and Black or Hispanic background (LSRO, 1991).

The iron deficiency that develops during pregnancy is caused by the increased requirements for iron to supply the expanding blood volume of the mother as well as the rapidly growing fetus and placenta. As the pregnancy progresses and fetal demands grow, maternal iron stores are drained to keep pace. During the course of gestation, overall iron requirements approach one gram (Bridges, 1990). To compensate for this increased demand, there is an increase in efficiency of dietary iron absorption in pregnant women. However, the increased iron demands are not often met by diet alone; thus current standard recommendation is to prescribe iron supplementation throughout the second and third trimesters of pregnancy (IOM, 1990; LSRO, 1991).

There appears to be increased risk of poor fetal outcome from iron deficient mothers even though studies have shown little difference in hematological values when comparing infants of anemic mothers with non-anemic mothers (Rios et al., 1975; Fenton, Cavill and Fisher, 1977; Dallman, Siimes and Stekel, 1980). Increased incidence of fetal deaths, short gestation lengths, low birth weights, and medical abnormalities at both ends of the hematological distributions for hemoglobin and hematocrit levels have been documented (Garn, Keating and Falkner, 1981). The optimal maternal values for favorable pregnancy

outcome have been suggested to be a hemoglobin of 11-12g/dl and a hematocrit of approximately 33 to 35% (Garn, Keating and Falkner, 1981). Another study found significant increases in prematurity at all hematocrit levels below 38%, but the greatest risk was noted at hematocrit of less than 34% (Lieberman et al., 1988).

In normal pregnancy, hematologic values change substantially (CDC, 1989; IOM, 1990; LSRO, 1991). For women with adequate iron nutriture, hemoglobin and hematocrit values start to decline during the first trimester, reach their nadir at the end of the second trimester, and then rise somewhat in the third trimester (Svanberg et al., 1975; Puolakka et al., 1980; Taylor et al., 1982). These changes reflect the expansion of maternal blood volume which occurs in the 3rd and 4th months of pregnancy. Thus it is essential that standards for hemoglobin and hematocrit be time-dependent.

The Centers for Disease Control in their recommended cutoff levels for anemia used pooled data from four recent European clinical surveys of healthy pregnant women taking iron supplements. The cutoffs are trimester-specific: (Centers for Disease Control, 1989), and have recently been endorsed by two separate bodies of expert opinion (IOM, 1990; LSRO, 1991).

1st trimester--11.0g/dl hemoglobin, 33% hematocrit
2nd trimester--10.5g/dl hemoglobin, 32% hematocrit
3rd trimester--11.0g/dl hemoglobin, 33% hematocrit

AVAILABLE MEASURES OF IRON STATUS

Iron depletion, while thought to develop in three stages, really represents a continuum from normal to severe iron deficiency. These stages theoretically correspond with specific laboratory tests. The first stage consists of depletion of tissue iron stores and is readily detected by the concentration of serum ferritin, which is reflective of iron stores in the liver, spleen and bone marrow. The second stage of iron deficiency consists of a decrease in transport iron, characterized by decreased serum iron and increased total iron-binding capacity. The ratio of these two values gives the transferrin saturation, which will be decreased during the second stage. The third stage occurs when the supply of transport iron decreases to the point of limiting hemoglobin production. During this stage, erythrocyte protoporphyrin is elevated and gradual development of anemia and microcytosis occurs. Only at this stage are hemoglobin and hematocrit values affected. While this is a convenient classification, laboratory tests often fail to conform to the individual stages (Johnson, 1990).

Available laboratory tests which reflect iron status at various stages of depletion, therefore, include serum ferritin; serum iron, serum transferrin, and transferrin saturation; iron binding capacity, free erythrocyte protoporphyrin (FEP); hemoglobin (Hb), hematocrit (Hct), and red cell count and various indices derived from the cell count, such as mean corpuscular volume (MCV). All can be performed on microliter samples of blood (Beaton et al., 1989).

Hemoglobin and Hematocrit

The laboratory tests most commonly used in the diagnosis of iron deficiency are hemoglobin and hematocrit with mean corpuscular volume serving as an ancillary screening method when blood count by electronic counter is performed. Hemoglobin and hematocrit are widely used because they are inexpensive and easily performed. The two indices give approximately the same information over the range of mild anemias; when one rather than the other is used it may be based on available equipment although hemoglobin may be the preferred test because of its apparent greater sensitivity compared to hematocrit (Dallman, Siimes, and Stekel, 1980).

Hemoglobin is a good predictor of response to iron treatment and a good estimate of prevalence of iron deficiency when prevalence is high (Freire, 1989). Analysis of data from the NHANES II using a more strict definition of iron deficiency (the MCV model) showed that hemoglobin was of greatest value as a screen for iron deficiency when the prevalence of iron deficiency in a population was between 5 and 35 percent, a range which encompasses the prevalences for both children and pregnant women of interest to the WIC program. According to this analysis, the sensitivity of Hb for detecting iron deficiency was 37% and specificity was 93% (Binkin and Yip, 1990). The low sensitivity is a disadvantage, and reflects the fact that decreased hemoglobin occurs only after earlier stages of iron deficiency; hemoglobin values may fall substantially and still be in the normal range, while iron deficiency progresses from stage one to stage two (Cook et al., 1986). Nevertheless, hemoglobin will detect severe iron deficiency, at which level functional response to treatment may be expected (LSRO, 1991).

An historical problem with hemoglobin lies in previously derived standard ranges. In the past, these values were obtained from populations that had not been screened to exclude individuals with mild deficiencies of iron or folate or with conditions such as thalassemia. As a result, distributions skewed toward low values occurred (Dallman, Siimes and Stekel, 1980). With the use of the NHANES II data as the reference base, however, this is not a current problem.

Inter-individual variability in hemoglobin levels in iron-replete status is substantial, and poses problems for screening and for longitudinal follow-up, as it produces an expectably rather high rate of misclassification. Various factors which relate to this inter-individual variability are discussed in the following section; Figure 1, from Beaton (1986) shows in graphic form the overlap of distributions of individuals response to iron therapy vs Hb level. The decision as to cutoff points for use in a screening program depends upon the policy decision as to whether it is more important to minimize false negatives (i.e., to miss treating an iron-deficient individual) or to minimize false positives (i.e., to minimize the number of individuals declared eligible for treatment who will not benefit from additional iron).

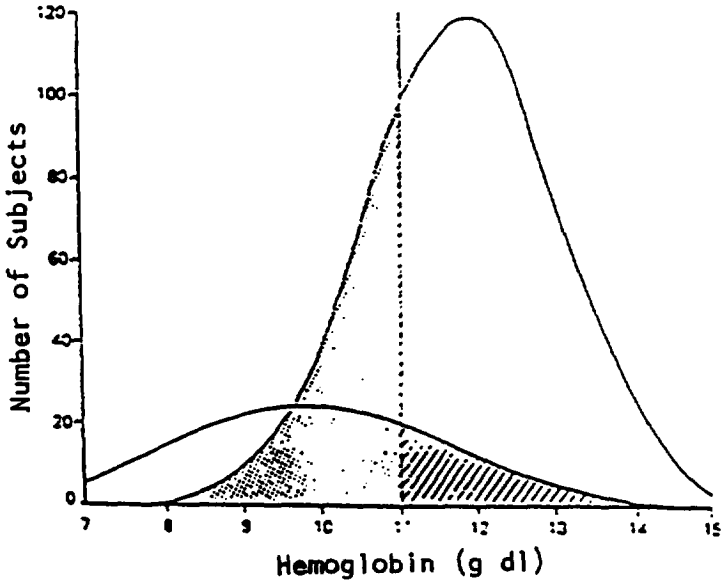

Figure 1. The problem of cutoffs. Portrayed are distributions of levels of hemoglobin among persons known to have adequate intakes of iron (right curve) and among persons who are known to be responsive to iron intake (left curve). Since the two distributions overlap, no single cutoff level can separate adequately from inadequately nourished individuals. The cross-hatched area above 11 g represents persons who are anemic but are classified as normal by the WHO cutoff. The shaded area below 11 g portrays persons classified as anemic by the WHO criterion, but actually not responsive to iron administration.

FACTORS AFFECTING HEMOGLOBIN LEVELS AMONG IRON-SUFFICIENT INDIVIDUALS

The changes in Hb with age in children, which differ between the sexes, present problems for screening in children unless age is taken into account, as a rise in Hb is expected with time (Beaton et al., 1989). Co-occurrence of infectious illness, even in mild form, may produce misclassifications since Hb levels are reduced by inflammation and infection regardless of iron status. There is a well-documented genetic difference in hemoglobin and hematocrit between African-American and Anglo-American adults (Meyers et al., 1979, 1983; Garn et al., 1975). Several studies have shown consistently lower hemoglobin and hematocrit values in African American populations than in Anglo populations. One group (Yip, Schwartz, and Deinard, 1984) found in comparing Anglo, African American and American Indian children that hematocrit was the same in Anglo and American Indian children, but 0.7% lower in African American children. These observations were thought to represent a lowering effect from the subset of the Black population with mild thalassemia. A recent author (Jackson, 1990), after reviewing the literature, found that there is no evidence that mean hemoglobin differences between African Americans and

Anglos is physiologically or pathologically significant. Both of these studies conclude that differences between the races are not significant to warrant separate screening standards.

Altitude is an important factor affecting hemoglobin values. Since most reference values are based on populations living at sea level, corrections for altitude are essential. A long-used approximate rule-of-thumb for correction for altitude based on data from adults is an increase of 4% for each 1000 meters (about 3000 feet) of elevation (Cartwright, 1968). More recently, it has been appreciated that the effect of altitude is not linear but rather logarithmic. Table 1 presents recently-published recommendations for altitude adjustments to hemoglobin and hematocrit values (LSRO, 1991), derived from data from the CDC Pediatric Nutrition Surveillance System on children residing at various altitudes in the mountain states.

Table 1

Recommended Adjustments to Hemoglobin and Hematocrit Values for Altitude

Altitude (feet)	Hemoglobin (g/dL)	Hematocrit (percent)
3000-3999	+0.2	+0.5
4000-4999	+0.3	+1.0
5000-5999	+0.5	+1.5
6000-6999	+0.7	+2.0

Cigarette smoking also has a predictable effect on hemoglobin and hematocrit values (smokers have a higher mean Hb than nonsmokers, and the effect is dose-dependent) (Jackson et al., 1983; LSRO 1991). The Centers for Disease Control have recommended that a correction factor be taken into account in identifying anemia in smokers, as follows: add 0.3 g to Hb standard and 1% to hematocrit standard for smokers; if dose is over two packs/day, add 0.7 g to Hb standard and 1.0% to hematocrit standard (CDC, 1989). The recent LSRO report (1991) recommends the same adjustment for 10-20 cigarettes/day, and 0.5 g Hb and 1.5% Hct for 21-40 cigarettes/day. The adjustment is additive to that for altitude when both are present.

Women taking oral contraceptives have total iron binding capacity values that are substantially elevated and fall into a range that is characteristic of iron deficiency (Yip, Johnson, and Dallman, 1980). Women taking oral contraceptives also show a rise in MCV with increasing age that is not seen in women who do not take birth control pills (Chalmers et al., 1979).

A final factor commonly associated with abnormal hematological values is alcohol related. Most cases of drug induced hematosuppression are traceable to acute or chronic alcoholism (Jandl, 1987). The pancytopenia of prolonged ingestion is not usually fatally progressive,

but the displacement of food by the alcohol can ultimately cause multiple nutritional deficiencies. The most common of these deficiencies is folate, but can include other nutritional elements such as iron. The anemia of alcoholism is usually self-limited unless accompanied by bleeding or folate deficiency. The uncomplicated anemia of alcoholic hematosuppression usually does not lower the hemoglobin concentration below 7 to 8 g/dl. Alcohol also has a direct suppressive effect upon red cell production that is reversible on abstinence (Jandl, 1987).

EVIDENCE FOR EFFECTIVENESS OF SCREENING

The relationship of WIC benefits to hematological indices has been examined in several studies with mixed results. Unfortunately, many studies are flawed by design problems, most notably lack of control groups (Rush et al., 1988). The studies that did use control groups came to differing conclusions. One group found no difference in hemoglobin or hematocrit levels between infants who were enrolled in WIC and control subjects from health department clinics in two Maryland counties without WIC programs (Paige cited in Rush, 1988). Another study found greatly reduced levels of anemia comparing a community both before and after WIC (Vazquez-Seoane, 1985). One study found higher hemoglobin and hematocrit levels among pregnant WIC recipients than among pregnant control subjects (Kennedy and Gershoff, 1982), while another group found no association between WIC benefits and hematological values in several health department clinics in Appalachian counties (Collins et al., 1981). Recently, one group (Smith et al., 1986), in a retrospective study, assessed the effectiveness of individual counseling, group nutrition classes and provision of WIC food vouchers on an experimental group of anemic children, while a control group of anemic children was not given WIC vouchers or nutrition education. The result was increased hemoglobin levels for both groups, but a greater measurable increase in the experimental group that was statistically significant. Probably, the best conclusions can be drawn from the linked results reported by the Centers for Disease Control (Centers for Disease Control, 1978). A decrease in the proportion of children with low hematocrit or hemoglobin concentrations after the first follow-up visit seemed to represent beneficial results from participation in WIC.

CHOICE OF SCREENING TESTS FOR IRON DEFICIENCY

Hemoglobin and/or hematocrit have the advantages of logistic and cost feasibility for large screening programs. Further, they detect a level of iron deficiency which represents greatest benefit from intervention, namely severe enough to cause anemia.

Given the preventive objectives of the WIC program and the steadily decreasing prevalence of iron deficiency anemia in the U.S. population, it may be useful to consider alternative strategies, including the selective use of serum ferritin or erythrocyte protoporphyrin (EP). Two recent papers (Jackson, 1990; Romslo, 1990) review the available indices of iron status and recommended that if only one measure of iron nutritional status is obtained, it should probably be plasma ferritin since it detects earlier deficits in iron stores and has been shown to be the best predictor of hematocrit values. This held true for all groups sampled, including pregnant women. The cost of serum ferritin is currently too high to make it reasonable for large-scale screening, and in addition reliable screening requires a repeated measure. EP has the advantage of screening for lead exposure as well as iron deficiency,

and may be an attractive alternative in some locations where lead exposure is a problem for children. The state of the art in iron deficiency is evolving rapidly, and the issue may bear revisiting within a few years. For now, the recommendation of expert panels (e.g., LSRO, 1991) is that hemoglobin represents that best available and cost-effective measure for screening and that cutoff points cited in this paper are based on sound scientific evidence.

SUMMARY

The prevalence of iron deficiency anemia in infants and children is still high among those living in households with incomes below the poverty line. In infants, the deficiency is greatest between 6 months and 2-3 years of age. CDC has recently published age-specific reference criteria for use in clinical practice for public health and nutrition programs which correspond closely to those recommended by the American Academy of Pediatrics. Increased iron demands, the rapid depletion of existing iron stores, and increased risk of poor pregnancy outcomes also combine to place pregnant women at risk for iron deficiency. CDC's recommended cutoff levels for anemia in pregnant women are time-dependent based on trimester. Several factors have been proven to affect hemoglobin levels among iron-sufficient individuals, including infectious illnesses, racial/ethnic background, altitude, cigarette smoking, alcohol consumption and use of oral contraceptives.

There are a number of laboratory tests available for measurement of iron status. Hemoglobin and hematocrit tests are used most frequently because they are inexpensive and easily performed. The FEP test has also been popular because it detects not only iron deficieny but lead-exposure levels as well. Some of the more sensitive blood tests (e.g, serum ferratin) are prohibitively expensive for widespread use in clinical settings.

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Age and Primiparity as Risk Factors for Poor Pregnancy Outcome Among U.S. Women

Technical Paper #6 prepared for the U.S. Department of Agriculture, Food and Nutrition Service, under Cooperative Agreement #58-3198-1-005, "Review of Nutritional Risk Criteria for the WIC Program", with the Department of Family and Community Medicine, University of Arizona, by Osman M. Galal, MD, PhD. Dr. Galal was formerly Research Professor of Family and Community Medicine, College of Medicine, University of Arizona, Tucson, AZ 85724, and is now Professor of Community Health Sciences, School of Public Health, University of California at Los Angeles, Los Angeles, CA 90024.

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INTRODUCTION

The present document will address three main issues, namely:

1. To what extent is young maternal age a nutritional risk factor in the U.S.?
2. To what extent is older maternal age a nutritional risk factor in the U.S.?
3. To what extent is primiparity an indendent risk factor?

It will also discuss the utility of targeting the integrated intervention package which WIC represents towards younger and older pregnant women.

It has been regarded that mothers at the youngest and oldest ends of the range of reproductive life are considered high-risk from an obstetrical point of view (Hansen, 1986). Older textbooks routinely sated that the risk of low birth weight was a U-shaped function of maternal age (LaGuardia, 1989). Recent data indicate that this rule of thumb is at least an oversimplification.

Young adolescent mothers have particularly high nutritional needs, and are more likely to be ill prepared socially, economically, physically and emotionally to cope with the demands of pregnancy and parenthood than are somewhat older women. The special needs of adolescent mothers have been well recognized by health professionals. For example, the American Dietetic Association in 1988 published a position paper on nutritional risk for pregnant adolescents, which stated that "any pregnant adolescent is at nutritional risk..." and concluded that pregnant adolescents need nutrition intervention throughout the duration of their pregnancies (ADA, 1989). Another example is the recent monograph for health care practitioners by Story (1990).

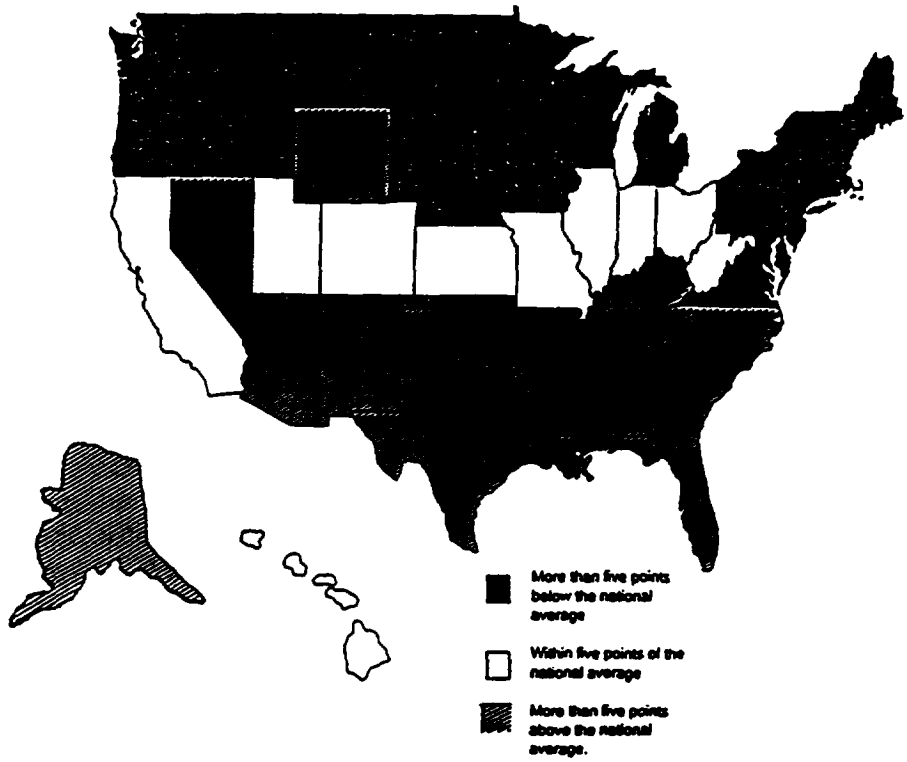
Women at the older end of the age range, variably defined as over 35 or over 40 years, enter pregnancy with quite different potential risks. These derive from age-related changes in the vascular system resulting in a higher incidence of pregnancy-induced hypertension; essential hypertension; greater pre-pregnant relative weight compared to younger women; greater incidence of diabetes; and more variable weight gains in pregnancy. Most of the literature on this subject has focused on the older primigravida. Since a 1958 statement of the American College of Gynecologists and Obstetricians the woman experiencing her first pregnancy at age 35 or later has been termed an "elderly primigravida" (Morrison, 1975). In fact, there is essentially no basis for any particular cutoff point, since the problems which advancing maternal age may pose during pregnancy are probably more or less steadily progressive with age and rather highly variable from person to person (Kane, 1967).

RECENT TRENDS IN BIRTH RATES BY AGE AND PARITY

In the U.S., approximately 13% of infants are born to adolescent mothers. Actual rates of pregnancy are difficult to estimate because of inadequate statistics on abortion and miscarriage among adolescents, but it is likely that the infants carried to term in this age group represent about one-half of the pregnancies. It has been estimated that 26% of black teens and 7% of white teens will have carried a pregnancy to term before age 18 years (Story, 1990). Mott (1986) reported that 26% of women who gave birth to their first child at the age of 16 had a second child within 24 months. Birth rates among women

aged 15-19 for the U.S. exceed rates for Canada and for western European countries by 2-4 fold. There is major regional variation in birthrates among adolescent women, with southern and some western states having the highest rates (see Figure 1, from Story, 1990).

Figure 1
Estimated adolescent birthrates per 1,000 women 15-19, 1985.



The recent decline in birthrates seen in the U.S. population has been much less among teenage than among older women, and there is some indication that the birthrate to very young adolescents (<16 years) may be rising (Story, 1990: 2). Thus the proportion of adolescent women in the general obstetrical population has increased recently and continues to do so in this country, particularly in public health care services.

Meanwhile, changes in demographic and work patterns for women in the nation over the last two decades have resulted in the postponement of childbearing for a significant number of women into their mid-30s and occasionally later. These effects were first documented in the late 1970s; 1976 and 1977 were the first years in which national statistics showed increases in the rate of first births to women aged 30-39, followed by 9% and 10% rises in 1978 and 1979 in this age group (Weigley, 1983). Between 1960 and 1985 there was a three-fold increase in first births to women aged 30 years and older among both blacks and whites, in spite of the fact that the proportion of total births to women in this age group was lower in 1985 than in 1960 (Institute of Medicine, 1990: 45).

The age distribution of pregnant women over the last several decades has changed less than has parity, reflecting the overall decline in birthrates in the population. In 1960,

approximately 25% of births to white mothers and half of births to black mothers were to mothers of "high parity" (3+ for mothers under age 20 and 4+ for mothers 20 and over); in 1985 these figures were 9% and 15% respectively (Institute of Medicine, 1990: 44-45).

Thus the last several decades have seen an increase in the proportion of primiparae, particularly but not limited to those at the older end of the reproductive age range; a decrease in the proportion of high-parity women; and an increasing proportion of young adolescents among pregnant women. Low birthweight rates in the U.S. have remained high through the years with a slow decrease but with increasing rates of prematurity (McCormick, 1991).

LOW BIRTH WEIGHT RISK, INFANT MORTALITY AND MORBIDITY AMONG YOUNG AND VERY YOUNG MOTHERS

Cutoff points to define "young" vary among investigators, making direct comparisons across different analyses somewhat difficult. The term "teen-aged mother" is somewhat misleading, since girls as young as 10 years may experience pregnancy and since by age 18 years (and perhaps 16) biological maturity is such as to place a young woman in a biological risk category not different from the somewhat older woman. "Adolescent mother" is similarly inadequate as a label since adolescence refers to a socioculturally defined phase of the life cycle the beginning and end of which varies among cultures and subcultures, and which is not limited to or entirely coincident with pubertal development in the biological sense. Thus it is not surprising that state and geographical agency WIC criteria also vary depending on local perceptions of risk and the local age distribution of pregnancies among young women. From a biological point of view, the significant variable is gynecological age, or elapsed time since menarche; the young mother who is less than two years past menarche can be regarded as less mature, still in her own growth period, and more nutritionally at risk than the mother who is more than two years post-menarche.

There is no doubt that very young mothers are at relatively high risk for the delivery of low-birth weight babies (Institute of Medicine, 1990; Taffel, 1986; Naeye, 1981a; McCormick et al., 1984) and for elevated rates of neonatal and post-neonatal mortality (McCormick et al., 1984; Taffel, 1986). Taffel (1986) presented data from the 1980 National Natality Survey and the 1980 National Fetal Mortality Survey, showing that 9.1% of live births to mothers under age 20 years weighed less than 2500 grams, compared to 6.7% for the overall population, 6.8 percent for women aged 20-24 years and 5.8 percent for women aged 25-29 years. This elevation of risk of low birth weight held across all categories of gestational weight gain except 22-25 pounds, and was particularly marked among women with low pregnancy weight gains. Among women gaining less than 16 pounds during pregnancy, 19.2% of babies born to mothers under 20 years of age were low birth weight, compared to 13.4% of babies whose mothers were 22-24 years old and 11.2% whose mothers were aged 25-29 years.

Exploration of the factors which predispose younger mothers to elevated risk of delivering low birth weight infants requires separation of the effects of gynecological age per se from effects of inadequate prenatal care, socioeconomic status, and nutrition including pre-pregnant relative weight, weight gain in pregnancy, and dietary quality. The available data do not allow a complete picture to be drawn at this point in time, but important recent data

and analyses point to the importance of adequate prenatal care, nutritional support, and social support in improving outcome of pregnancy for young mothers.

Slap and Schwartz (1989) in a study of 577 medical records of women aged 19 and younger who delivered infants at the University of Pennsylvania hospital between July 1982 and December 1983, showed that within the adolescent age group (19.4% of the entire study population), inadequate prenatal care and adverse obstetrical history were the most important predictors of low birth weight. A similar study reported by the Institute of Medicine (1985) showed a decline of 30 percent in low birth weight incidence if high-quality prenatal care (defined by the number of visits to a general or specialized obstetric clinic) was received by adolescent mothers. Other studies (Sexton and Hebel, 1984) have identified genitourinary infection and smoking as major risk factors for low birth weight in adolescent mothers, both of which are amenable to change with adequate prenatal care.

The Institute of Medicine's recent (1990) report on *Nutrition and Pregnancy* concludes on the basis of recent data (Scholl et al., 1984, 1988; Duenhoelter et al., 1975; Horon et al., 1983) that the relationship of gestational weight gain to fetal growth is similar in adolescents and older women once confounding factors including pre-pregnancy relative weight are controlled (IOM, 1990: 185). In fact, however, analysis of national data show that teenage women are more likely to have inadequate (< 16 pound) pregnancy weight gains than women aged 20-35 despite their lower average pre-pregnancy weight-for-height (Taffel, 1986). This situation may vary considerably by the environmental and social context, as shown by the fact that an analysis of weight gain among White Vermont women in the late 1960s and early 1970s showed higher weight gains for the youngest age group (12-17 years) and lower gains for the oldest group (25-32 years) relative to intermediate ages (Ancri et al., 1977).

A recent study by Gale et al. (1989) is of particular interest since it addresses risk for young mothers in a total social and cultural context. They studied 421 primiparous women aged 15-19 years who lived in a Jewish settlement in Israel and delivered in a general hospital. The sample included two groups. One hundred ninety mothers belonged to an ultra-orthodox sect of the Jewish community, in which early marriage and childbearing are the norm. All were married, and lived in an environment which provided extensive social, family and economic support to them during their pregnancies. The comparison group included 231 young mothers who were predominantly from a lower social class background, the majority unmarried, and not necessarily supported by their families. Outcomes of pregnancy, including neonatal outcomes and complications of delivery, were not different between the young mothers in the study group and women aged 21-24; in the comparison group, however, mothers under 19 years of age experienced elevated rates of low birth weight.

The results of this study would tend to argue that much of the increased risk for low birth weight associated with young maternal age in the U.S. population may be in fact mediated by poor prenatal care, poor diet, and/or poor psychosocial environment and support systems. Unmarried status is most common in the U.S. among the youngest mothers, and is associated with elevated risk of low pregnancy weight gain (Taffel, 1986), and adolescent mothers are at particular risk for inadequate or no health insurance (Story, 1990). There is little evidence with which to address the relative risk of parity vs age in young adolescents, but what there is suggests that multiparity, not primiparity, is an additional risk factor for the young mother (< 16 years) if she is underweight (Naeye, 1986b).

Risk of infant (neonatal and postneonatal) mortality for offspring of young mothers has been addressed in national data from the 1960s (Niswander and Gordon, 1972) and 1980 (Taffel, 1986) and in regional data from the 1970s (McCormick et al., 1984). Pre-term deliveries, stillbirths, and neonatal

deaths are more common among young adolescent mothers (Niswander and Gordon, 1972). There is evidence that the effect is mediated by weight gain, with a pronounced decline in risk of fetal death as pregnancy weight gain increases and an increased risk of low weight gain in young mothers (Taffel, 1986). McCormick et al. (1984) compared two groups of mothers: those < 17 years of age and those 18-19 who were parity 2+, with other mothers in four areas of the U.S. They found rates of neonatal mortality more than 150% higher in the former group, largely due to the elevated proportion of low birth weight infants born to these mothers. Post-neonatal infant mortality rates were also elevated, from causes consistent with socioeconomic disadvantage.

NUTRITIONAL NEEDS AND GROWTH AMONG YOUNG AND VERY YOUNG MOTHERS

In spite of the conventional wisdom that the young mother and her fetus compete for nutrients and therefore have very high combined needs (Washington, 1977), there is considerable controversy over the issue. Most of the evidence, as mentioned above, points to equivalent birth outcomes for young mothers whose pregnancy weight gains and prenatal care are adequate and who live in equally supportive social and economic environments to older women.

The question of whether the gynecologically immature young mother (less than two years post-menarche) may pose a different biological dilemma from the slightly older mother remains open. Frisancho et al. (1984) has pointed out that the smaller placental weights typical of very young mothers result in a lower contribution of placental weight to birth weight; this observation argues in favor of maternal competition for nutrients. Naeye (1981a) analyzed data on young Black mothers from the National Collaborative Perinatal Project, and found that nonsmoking, normal- or underweight girls had lower weight newborns at age 10-16 years than did women aged 17-19 even controlling for prepregnant weight for height, weight gain in pregnancy, and parity. The Collaborative Perinatal Study, which collected data on cigarette smoking, did not provide data on drug or alcohol use so these are uncontrolled in this analysis.

The recent IOM (1990) report on Nutrition in Pregnancy cites two other studies relevant to the issue of whether young adolescents have different requirements in pregnancy than older ones. Haiek and Lederman (1989) showed 200-400g differences in birth weight at term for infants born to mothers < 15 years of age compared to 19-30-year-old mothers, stratified by relative weight at full term, except for adolescents who achieved 140% of their prepregnant relative weight. Potentially confounding cigarette, drug and alcohol use were not controlled. The other relevant study is that of Frisancho et al. (1985) of poor young mothers in urban Peru. An approximate 200g deficit in birth weight was found after controlling for gestational weight gain in term infants of mothers < 15 years old compared to those 17-25 years. These results were not controlled, however, for parity or within-sample socioeconomic variability.

Pattern of weight gain, in young mothers as in general, remains less investigated and less well understood. One recent study (Hediger et al., 1989a, 1989b) addressed the question of first- vs second-semester weight gain in teenaged New Jersey adolescents, and found that inadequate early pregnancy gain (< 4.3 kg by 20 weeks) was associated with increased risk of intrauterine growth retardation even when second-semester weight brought total gain within the recommended range.

It remains controversial whether young women who have not attained their full linear growth continue to grow during and after pregnancy. In adolescents who do not become pregnant, growth in stature typically continues for a year or more following menarche. The endocrine environment of pregnacy stimulates epiphyseal closure of the long bones (Thompson, 1976) and therefore might be expected to

put an end to further growth in stature. However, two studies exist to indicate that some growth may continue in the young adolescent during pregnancy and postpartum., although it may be less than if the pregnancy had not occurred. Garn et al. (1984) analyzed data from the Collaborative Perinatal Project in which some adolescents were followed through two and a few through three pregnancies; they found maternal growth less than expected for the postmenarcheal period, but still detectable. Scholl et al. (1988) used measurement of leg length, a relatively sensitive measurement of linear growth, and found that younger adolescents (12-15 years) exhibited greater growth increments during their first pregnancy than older adolescents (15-18 years) in their second pregnancy, and both groups exhibited some growth compared to more mature nonpregnant women (18-29 years).

Several authors (Garn et al., 1984; Frisancho et al., 1983) have recommended greater weight gains for adolescent mothers than for older women. The recent IOM report does not set a separate goal but rather mentions that "young adolescents and Black women should strive for gains at the upper end of the recommended range" (IOM 1990: 10), the ranges being specified on the basis of pre-pregnant weight-for-height.

DIETARY PATTERNS IN YOUNG PREGNANT MOTHERS

There is little information on nutrient intake of pregnant adolescents, and that which is available indicates that their diets are not much different from those of nonpregnant teenagers (King et al., 1972). Nutrients most likely to be in short supply include vitamin A, calcium, and iron (Hamptom et al., 1967), which are the target nutrients in the WIC food package. Thus the WIC food package is ideally suited to improve dietary quality in this group. Loris et al. (1985) found relatively adequate diets among 145 pregnant women ages 13 to 19 attending a teen obstetric clinic in Davis, California. Endres et al. (1985) reviewed ten studies of dietary intake which included pregnant adolescents, and found that compared to older women they tended to consume more snack foods and less fluid milk.

Concern over weight control, and dieting to control weight, is highly prevalent among adolescent girls and may manifest itself as an attempt to avoid gaining weight in the teenager who is pregnant. Given the recommendation that the very young woman gain more weight than the older woman for optimal prognosis, support of unrestricted weight gain is a priority for nutrition education.

Relatively few teenage mothers elect to breastfeed their infants compared to older women. The available information indicates that about 50% of teen mothers plan to breastfeed, but only about 20% actually do so (Worthington-Roberts et al., 1981). Thus support for breastfeeding should be an important component of nutrition education programs for this group.

AGE VS PRIMIPARITY AS CONTRIBUTORS TO RISK FOR YOUNG MOTHERS

With the exception of increased risk for pre-eclampsia, the pregnancy risks often attributed to primiparity are actually more directly the result of young maternal age, which is more likely in primigravidas. Pre-eclampsia is mainly a disease of primigravidas, although there is some evidence that there may be elevated risk in the first pregnancy with a new partner in multigravidas, suggesting a possible immunological role in etiology (Naeye, personal communication). Other factors associated with primigravidity tend to be age-mediated. Primigravidas are more likely to be very young (less than two years post-menarche) and thus competitors with their own fetuses for nutrients; very young mothers are less likely to be married, to be in stable, supportive social situations, and to have accumulated significant nutrient stores than are slightly older women.

THE OLDER GRAVIDA

From a biological point of view, the risks associated with advanced maternal age have to do primarily with uterine and utero-placental blood flow. Uterine arteries age more rapidly than arteries in most other organs; as a result they are less elastic in older women and may not permit increased blood flow when fetal needs increase. The process of uterine aging is enhanced by multiple pregnancies. The result is increased risk of stillbirth and neonatal deaths for a wide range of seemingly unrelated disorders, particularly in late gestation, for women who are >35 years of age. In addition, older gravidas are at higher risk for fetuses with congenital malformations and for dizygotic twins (Naeye, personal communication).

There is little information available about nutritional status of the older pregnant woman. Body weight does increase with age in the general adult population, as does the incidence of hypertension, diabetes, and other conditions for which obesity is an exacerbating factor. Counseling non-gravid women of childbearing age to attain or maintain ideal body weight before conception might conceivably reduce risks in pregnancy. The older gravida who has experienced nutritional depletion is also at high risk unless pregnancy weight gain is adequate. The woman over age 35 who weighs less than she weighed pior to subsequent pregnancies represents a high-risk individual from a nutritional point of view. There is no evidence available that older pregnant women have any particular risk of poor diet compared to younger ones; Endres (1985) in a review of data on older pregnant women in the WIC program, found dietary intakes which were more nutrient dense for older (>35 years) women than for adolescents.

SUMMARY

Trends in childbearing in the U.S. are resulting in a higher proportion of young adolescents among pregnant women, a higher portion of first births to older (over age 30) women, a decrease in high parity mothers and an increased proportion of primiparae of all ages. Youth is a much stronger risk for low birth weight than is primiparity; indeed, the 18- or 19-year old multiparae is at as much risk as the younger primipara and more than her age-matched primipara. The prevalence of pregnancy among very young women is strongly related to race and to region, with Black women and women in the South and some areas of the West at higher risk.

Most of the available recent data indicates that when weight gains are adequate and other potentially confounding variables are controlled, birth outcomes are similar for adolescents as for somewhat older women. However, in the U.S. adolescent pregnant women are at increased risk for inadequate weight gains; they are also more likely to be unmarried, to be less educated, and to be at economic disadvantage compared to older women. All of this predisposes not only to increased risk of low birth weight, but also to increased rates of infant (both neonatal and postneonatal) mortality for adolescent mothers. There is some controversy over whether the very young mother (less than two years post-menarche) is able to maintain her own growth during pregnancy and postpartum, given adequate nutritional support. There is no evidence that diets of pregnant teenage women are different from those of nonpregnant teenagers; compared to diets of older pregnant women they tend to be lower in milk products and higher in snack foods.

The older pregnant mother (usually defined as >35 years) enters pregnancy with increased risk of vascular insufficiency resulting in slow fetal growth and prolonged gestation; of pregnancy-induced or pre-existing hypertension; of obesity and diabetes. Early identification and appropriate management of these risk factors can potentially result in improved pregnancy outcome.

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Evidence for Effects of Timing of Prenatal Care and Nutritional Supplementation on Pregnancy Outcome

Technical Paper #7 prepared for the U.S. Department of Agriculture, Food and Nutrition Service, under Cooperative Agreement #58-3198-1-005, "Review of the Nutritional Risk Criteria for the WIC Program", by Amr S. Soliman, MB, BCh, MSc, Gail G. Harrison, PhD, and Osman M. Galal, MD, PhD. Dr. Soliman is Research Assistant, Department of Family and Community Medicine, and a PhD candidate in the Program in Nutritional Sciences, University of Arizona. Dr. Harrison is Professor of Family and Community Medicine, College of Medicine, University of Arizona, Tucson, AZ 85724. Dr. Galal is formerly Research Professor in the Department of Family and Community Medicine, University of Arizona and currently Professor of Community Health Sciences, School of Public Health, University of California, Los Angeles 90024.

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INTRODUCTION

There is now substantial evidence from several recent studies (Devaney et al., 1990; New York State Health Department, 1990) that both prenatal care and prenatal participation in the WIC Program lead to improved birth outcomes and are associated with substantial savings in postpartum health care costs. However, there aren't any definitive studies of the effects of timing of prenatal care or prenatal WIC participation or of the determinants of prenatal care or prenatal WIC enrollment. Several studies have attempted to measure an effect of duration of participation in WIC, and have concluded that early enrollment in WIC is predictive of improved birth outcomes, but the results cannot be considered conclusive because of methodological problems.

The purpose of the present brief technical paper is to review the evidence for effects of timing of prenatal care and nutritional supplementation on pregnancy outcome. We will focus on: a) prevalence and risk factors for inadequate prenatal care; b) participation in prenatal WIC and its determinants; c) the relationship between WIC participation and prenatal care; d) evidence of WIC's effect on pregnancy outcome; and e) weight gain patterns during pregnancy and evidence for an effect of timing of nutritional supplementation on pregnancy outcome.

PREVALENCE AND RISK FACTORS FOR INADEQUATE PRENATAL CARE

Inadequate prenatal care is defined as beginning later than the fifth month of pregnancy or, if earlier, including fewer than half the number of visits recommended by the American College of Obstetricians and Gynecologists (Witwer, 1990). In 1988, about three-quarters of pregnant women in the U.S. received prenatal care beginning in the first trimester, but about 25% received no or late prenatal care (NCHS, 1990). There is considerable evidence that among U.S. women lack of prenatal care is strongly associated with increased maternal and fetal morbidity (Institute of Medicine, 1988, 1990; Moore et al., 1986).

Documented risk factors for late and/or inadequate prenatal care include maternal age (less than 20 and more than 40 years), ethnicity (Blacks and Hispanics are at increased risk) and education (risk increased for women with less than a high school education) (Curry, 1990). Rates of late or no prenatal care among Blacks in 1980 and 1987 were 8.8 percent and 11 percent respectively, compared with 4.3 percent and 5 percent for Whites (NCHS, 1990). Analysis of data from the National Natality Survey showed that 9.5% of teenage mothers postponed prenatal care until the third trimester of pregnancy or received no prenatal care at all, in comparison to half that proportion in older women from the same population (Singh et al., 1985). McDonald et al. (1988) reported that low income, longer travel time, receiving Medicaid benefits, and rural residence were predictors of insufficient prenatal care and that older maternal and paternal age, private insurance, planned pregnancy, and more years of father's education were significant predictors of adequate utilization of prenatal care during pregnancy. A recent Institute of Medicine report (1990) summarizes the causes of rising rates of no or late prenatal care as including increasing rates of poverty, unemployment, socioeconomic instability and increased numbers of single mothers in the population (IOM, 1990).

PARTICIPATION IN PRENATAL WIC AND ITS DETERMINANTS

In Fiscal Year 1989, WIC provided benefits to approximately 590,000 pregnant women, who represented about 85% of all eligible pregnant women. However, data suggest that coverage may be lower for certain subgroups of pregnant women. Devaney et al. (1990) reported that in 1987-88, 48% to 73% of all Medicaid births in the five states studied were to WIC participants. At the levels of poverty needed to qualify for Medicaid assistance in the five study states in 1987-88 (33% to 88% of the federal poverty line), it seems reasonable to assume that virtually all of the women in the sample would have been eligible for WIC.

The Food and Nutrition Service does not routinely collect information on pregnant WIC participants broken down by trimester of enrollment, and there is very little research on factors that predict the decision to enroll in prenatal WIC, or the timing of that decision. Risk factors for lack of adequate prenatal care reviewed earlier included maternal characteristics such as age, race, education, and low income, in addition to distance from clinic, Medicaid eligibility, and rural residence. Some of these same factors may also predict lack of participation or late enrollment in the WIC program, although data from two studies suggests that maternal characteristics are not a significant determinant.

Ku (1989) used data from the 1984 Study of WIC Participant and Program Characteristics to examine the effect of maternal characteristics and local clinic operational factors on early enrollment in WIC. Individual factors included participation in WIC in a prior pregnancy, race, age at WIC entry, income as a percent of the Federal poverty level, household size, and WIC priority level. Local agency characteristics included availability of prenatal and other health services at the same site, outreach policies for pregnant women, years of local WIC operation, and local agency size. The only significant predictor of early enrollment was prior participation in the WIC program (adjusted odds ratio 2.1, $p < .001$) (Ku, 1989).

Haddad and Willis (1983) used data from the Massachusetts WIC Evaluation, augmented by a survey of WIC sites in Massachusetts to investigate the probability of early enrollment in WIC, barriers to early enrollment, and effectiveness of WIC outreach efforts. Local program characteristics investigated included length of clinic operation, sponsoring agency, type of food delivery system, and media outreach. Maternal characteristics included marital status, age, parity , race, education, and previous abortion. Barriers to participation included hours of clinic operation, availability of child care, and staff language capabilities. The authors found that length of program operation, use of a retail food delivery system, and parity had statistically significant effects on the likelihood of early enrollment.

WIC PARTICIPATION AND PRENATAL CARE

Most of the literature on the effects of prenatal care on pregnancy outcome treats prenatal care as a single unit, in spite of the fact that preventive prenatal care consists of a number of different components which may vary individually in presence/absence and in quality. These include screening for various medical conditions and risk factors, appropriate testing and genetic counseling, explanation of risks, monitoring of weight gain and fetal growth, management of minor and major complications of pregnancy, planning for management of

labor and delivery, nutrition education, advice on cigarette smoking, drugs, alcohol intake, sexual activity, exercise and other behaviors, education and planning for parenting, infant feeding, and family adjustment. The WIC program includes in its intervention package some of the components of good prenatal care, namely nutrition education, monitoring of weight gain, and advice on some important lifestyle factors. In addition, it also provides referrals to prenatal care and other health care services. In areas where obstetrical services are limited and there is a substantial waiting period for enrollment in prenatal care, the WIC program may serve as the only readily accessible pregnancy support during the first trimester or longer.

In a cross-sectional study of Medicaid-eligible women in five states, Devaney et al. (1990) found that in all five states studied, WIC participants were more likely to have received any prenatal care than nonparticipants, and were also more likely to have received adequate prenatal care. The reasons for this could not be determined from the data.

EFFECT OF WIC PARTICIPATION ON PREGNANCY OUTCOME

Several studies have shown higher birthweights, improved anthropometric measurements, decreased fetal deaths, and/or decreased Medicaid expenses related to participation in WIC during pregnancy (Kennedy et al., 1982; Schramm, 1986; Devaney et al., 1990). Devaney et al. (1990) found average increases in birthweight of 51 to 117 grams, increases in gestational age of .2 to .8 weeks, decreases in the incidence of low birthweight ranging from 2.2% to 5.1%, and decreases in the incidence of preterm births ranging from 2.3% to 6.3%.

Devaney et al. (1990) also evaluated the impact of prenatal WIC participation on Medicaid costs during the first 60 days postpartum in five states. They found that both prenatal care and WIC participation had substantial, independent positive effects on Medicaid costs in each of the five states. For every dollar spent on WIC, states saved from $1.77 to $3.13 in Medicaid costs for newborns and their mothers during the first 60 days postpartum. The investigators used a modified Kessner Index to account for the adequacy of prenatal care which takes into account both the number of visits as well as the trimester in which prenatal care began. Adequate prenatal care for a full-term birth was defined as a minimum of nine visits beginning in the first trimester. The receipt of an adequate or intermediate level of prenatal care was associated with savings $267 to $1005 per mother/infant pair, separate from, and additive to, the effect of prenatal WIC participation. Savings attributable to WIC participation ranged from $277 to $598.

TIMING OF WIC ENROLLMENT AND PREGNANCY OUTCOME

Given the different components of prenatal care that WIC provides, and the consistently positive effect of WIC participation on birth outcomes, it seems logical to assume that the longer a woman receives such benefits, the greater her chances of an improved outcome relative to a woman who receives benefits for a shorter period of time. In other words, it seems likely that WIC has a "dose-response" effect. However, isolating such an effect is methodologically difficult because the duration of participation in WIC is inevitably confounded with the duration of gestation. In other words, women with longer gestations have more time in which to participate in WIC, but the longer gestation may itself be an

effect of WIC participation. The inclusion of both gestational age and WIC participation as predictors in regression models does not solve the problem because gestational age is itself a birth outcome, and its inclusion as a predictor could lead to biased estimates.

Several studies have investigated dose-response effects of WIC, and have concluded that longer or more intensive participation in the program is associated improved birth outcomes (Edozien et al., 1979; Kotelchuck et al., 1984; Rush et al., 1988). However, these findings must be interpreted with caution in light of the difficulty noted above.

Edozien et al. (1979) studied the effect of duration of WIC supplementation the during pregnancy on the weight gain, birthweight, and duration of gestation; WIC participation for more than 3 months was associated with increased birthweights (average 68 g for 3 to 6 months supplementation and 136 g for more than 6 months supplementation). There was also a minor positive effect of participation for more than six months on duration of gestation.

Kotelchuck et al. (1984) studied birth outcomes among prenatal WIC participants in Massachusetts. In addition to a reduction in the incidence of low birthweight and infant mortality, and an increase in the use of prenatal care among WIC participants, the authors found that birthweights of infants born to WIC participants are proportional to the duration of WIC benefits; that is, the earlier the beginning of prenatal WIC participation the heavier the newborn.

Rush et al. (1988) conducted a longitudinal study of pregnant women using a nationally representative sample of WIC participants and controls. Outcomes investigated included nutrient intake and weight gain in pregnancy, birthweight, length, head circumference, gestational age, and fetal death. With respect to a dose-response effect, the authors found no significant association but a consistent "predicted direction" between early WIC enrollment and longer pregnancy, increase in head circumference, and increase in newborn length.

EFFECTS OF TIMING OF NUTRITIONAL INTERVENTION ON PREGNANCY OUTCOME

Although it has not been possible to estimate the effect of duration of WIC participation directly, some support for the existence of such an effect can be derived from current knowledge about patterns of weight gain patterns during pregnancy, and from the considerable body of literature on nutritional supplementation during pregnancy in different populations.

Weight Gain Patterns

Weight gain during pregnancy is one of the most important independent predictors of birthweight, and because it is modifiable, the study of weight gain patterns offers an opportunity to explore the effects, if any, of timing and duration of intervention. Current recommendations for weight gain in pregnancy vary by prepregnant weight-for-height status, ranging from 16 to 40 pounds with thinner women recommend to gain more weight and heavier women less. Even obese women should have weight gains of at least 16 pounds (Institute of Medicine, 1990). In practice, total weight gain is highly variable with those at highest risk of low weight gain including adolescents and overweight women. In

the WIC program setting, rate of gain is a more accessible index than total gain, and the same report has provided guidelines for adequacy of rate of gain adjusted for prepregnant weight-for height (IOM, 1990).

The first trimester has been generally neglected except from the standpoint of avoidance of teratogenic substances, and often when prenatal care services are limited the first trimester may not receive any medical supervision. Yet weight gain in the first trimester is an important predictor of adequacy of the plasma volume expansion which occurs late in the first trimester (Hytten, 1980). In primigravidas the nonpregnant plasma volume of about 2600 ml expands by approximately an additional 1250 ml, and the expansion may be as much as 1500 ml in multigravidas (Letsky, 1980). Inadequacy of this plasma volume expansion is associated with poor formation of the uteroplacental vascular bed and lowered blood pressure, and is a very poor prognostic factor for pregnancy outcome; risk factors for poor plasma volume expansion included pre-pregnancy underweight and low first trimester gain (Hytten, 1980; Briend, 1985; Naeye, in press).

Calorie Intake and Nutrient Supplementation

Adequate food intake is of obvious importance during pregnancy. Calorie intake is correlated with weight gain in the first trimester (Papoz et al., 1982), and nutritional needs become obviously elevated after the first trimester. A number of investigators have attempted to influence pregnancy outcome by experimental supplementation of pregnant women with additional calories, nutrients or both during pregnancy (Lechtig et al., 1975; Mora et al., 1979; Rush et al., 1980; Prentice et al., 1983; Kardjati et al., 1988, 1990). The outcome variables most consistently investigated, and the studies that have looked at them are displayed in the following table.

Caloric Intake and Nutrient Supplementation Studies		
Infants		
Outcomes	**Number of Studies**	**Authors**
Birthweight	11	Mora et al. (1979); Osofsky (1975); Lechtig et al. (1975), (1979); Rush et al. (1980); McDonald et al. (1981); Viegas et al. (1982); Prentice et al. (1983); Watney and Alton (1986); Rush et al. (1988); Kardjati et al. (1988)
Incidence of prematurity	2	Viegas et al. (1982); Campbell (1983)
Head circumference	4	Osofsky (1975); McDonald et al. (1981); Prentice et al. (1983); Campbell (1983)
Gestational age	4	Mora et al. (1979); Rush et al.(1980); Prentice et al. (1983); Campbell (1983)
Perinatal mortality	2	Lechtig et al. (1975); Rush et al. (1980)
Mothers		
Outcomes	**Number of Studies**	**Authors**
Weight gain during pregnancy	5	Lechtig et al. (1975); Mora et al. (1979); Rush et al. (1980); McDonald et al. (1981); Kardjati et al. (1990)
Changes in body mass index	1	Kardjati et al. (1990)
Lactation performance	2	McDonald et al. (1981); Prentice et al. (1980)
Changes in maternal weight and skinfold thickness	2	McDonald et al. (1981); Prentice et al. (1980)

The results of a number of dietary supplementation studies are summarized in Appendix 1. They have varied in terms of type of supplement, timing and duration of supplementation, and nutritional status of the population as well as in the outcome variables measured. A few studies involved supplementation during most of the pregnancy (Lechtig et al., 1975; McDonald et al., 1981) while others began in the third trimester (Mora et al., 1979; Rush

et al., 1980; Osofsky, 1975; Watney and Alton, 1986) and still others in the second trimester (Prentice et al., 1983) or the second and third trimesters (Viegas et al., 1982). In general, supplementation studies in malnourished populations have shown positive effects on average birthweights (Mora et al., 1979; Lechtig et al., 1979; McDonald et al., 1981; Prentice et al., 1983) with differences averaging from 89 to 186 g. Reduction in the low birthweight rate was demonstrated in studies from Guatemala (Lechtig et al., 1975) and the Gambia (Prentice et al., 1983). Studies in U.S. and other populations, even those deemed to be "at risk" or low income, have shown ambiguous effects or even decreases in average birthweights (Osofsky, 1975; Rush et al., 1980; Viegas et al., 1982; Watney and Alton, 1986; Rush, 1988). This seemingly contradictory finding suggests that there are other factors that influence birthweight in these populations that are not responsive to nutritional supplementation.

Studies which have measured the effect of supplementation on maternal weight gain or skinfold thickness have shown no significant positive effects except for Mora et al. (1979) and Rush et al. 1980), which found positive effects of supplementation, particularly when it started before 15 weeks of gestation. Two studies of supplementation during the interconceptional period (McDonald et al., 1981; Prentice et al., 1983) found positive effects on mean birthweight but no significant effects on maternal weight gain. There is evidence that in malnourished populations energy intake is associated with higher pregnancy weight gains (Lechtig et al., 1975; Campbell et al., 1981).

SUMMARY

There is now substantial evidence that both prenatal care and prenatal WIC participation lead to improved birth outcomes. However, according to NCHS, only three quarters of pregnant U.S. women received adequate prenatal care in 1988. There was considerable variation in the receipt of prenatal care by maternal age, race, education, income, and residence. An estimated 85% of all eligible pregnant women received WIC benefits in Fiscal Year 1989, but data from a study of the Medicaid-eligible population in five states in 1987 suggests that coverage for this subgroup may have been lower. This same study suggests that women who participated in WIC were also more likely to have received adequate prenatal care.

There is some research that addresses the issue of timing of enrollment in WIC and the effect of duration of participation, but these studies cannot provide conclusive evidence of an effect of timing because there are methodological problems that make reliable estimation difficult.

Some evidence for an effect of timing can be obtained from studies of weight gain in pregnancy, and from studies of nutritional supplementation during pregnancy. Supplementation studies in malnourished populations have consistently found measurable increases in mean birthweight, while those in more heterogeneous populations or in populations in which undernutrition is not common have resulted in mixed outcomes. Most supplementation studies have utilized supplements in the latter half of pregnancy. The first trimester has been generally ignored, but there is evidence that weight gain in the first trimester may be important in relation to the adequacy of expansion of maternal plasma volume, which takes place in the third and fourth months of pregnancy, and which is related to a variety of pregnancy outcomes. The role of nutritional status and dietary

quality in the first trimester in relation to plasma volume expansion is not understood but is a critical area for further research.

Taken together, these findings suggest that nutritional supplementation during pregnancy, and early prenatal care, some elements of which are routinely provided by WIC, are important in assuring better pregnancy outcomes. National statistics on prenatal care suggest that a substantial number of women currently are not receiving adequate prenatal care. Those at highest risk for inadequate prenatal care include very young, low educated, and Black or Hispanic women.

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APPENDIX: STUDIES of DIETARY SUPPLEMENTATION and OUTCOME of PREGNANCY

		Description and Results			
Author's Name and Year Published	Population and Design	Time of Supplementation	Supplement	Impact on Newborn	Impact on Mother
Osofesky, 1975	Prospective study of 240 low-income pregnant women in Philadelphia.	Third trimester.	High protein-mineral supplement containing 6.5g protein/100 kcal. Total intake of 80.3g protein/d reported. Controls consumed 71.8g protein/d.	Significant decrease in mean birth weight of supplemented group ($p<0.05$). 3005g of supplemented and 3119g of controls. Significant decrease in length and head circumference of supplemented groups in comparison with control ($p<0.05$).	Not measured.
Mora et al., 1979	Prospective study random allocation of 456 Colombian women at risk of malnutrition.	Third trimester until term.	Foodstuffs for the entire family including dry milk, bread and vegetable oil provided 34.8g protein/d and 856 kcal/d/individual. Intake of females after supplementation was 55.4g protein/d vs 34.9g before, and 1611 kcal vs 1766 kcal before and after supplementation respectively.	Supplemented mothers had 89g higher male birth weight. Supplemented group's mean birth weight = 3040g. Unsupple-mented group's mean birth weight + 2951g ($p<0.05$). No significant difference in the proportion LBW among supplemented. Supplemented group had 11% and controls had 8.7% incidences of LBW. Among unsupplemented group, birth weight was not statistically different for both sexes. Mean birth weight of the total sample did not differ between groups.	Significant increase in weight gain during pregnancy in supplemented mothers only with male deliveries ($p<0.05$).

	Description and Results				
Author's Name and Year Published	Population and Design	Time of Supplementation	Supplement	Impact on Newborn	Impact on Mother
Lechtig et al., 1975, 1979	Randomized trial of 405 rural pregnant women in four villages in Guatemala.	Starting at first trimester until term.	163 kcal and 11g protein/180ml for two villages. The other two villages got 59 kcal but no protein/180ml in cool drink. Supplement was offered ad libitum.	20,000 kcal was the median caloric supplement during pregnancy. 3105g was the mean BW for the above 20,000 kcal group, and 2994g was the mean for the <20,000 kcal group. Neither the calorie nor the protein-calorie supplement had a significant effect on birth weight.	The higher the calorie intake from supplement, the lesser the proportion of low weight gain ($p<0.05$) during the last two trimesters, irrespective of the type of supplement used.
McDonald et al., 1981	Randomized controlled double-blind trial of 294 women in Taiwan.	Three weeks after delivery of the first study, infant continued through lactation and pregnancy and lactation of the second study infant.	Group A: 12.5 oz. can of liquid supplement for each woman provided 800 kcal and 40g of protein/d, while Group B received 80 kcal. Home food provided 36.7g protein and 1133 kcal/d for Group A and 1202 kcal/d for Group B.	Mean birth weight of the second infant was 161.4g more than that of the first infant, among male infants of Group A only ($p<0.05$). Number of LBW infants or incidence of fetal deaths showed no statistical difference between groups. No difference in infant length or head circumference.	No statistical differences in maternal weight gain during pregnancy.

	Description and Results				
Author's Name and Year Published	Population and Design	Time of Supplementation	Supplement	Impact on Newborn	Impact on Mother
Viegas et al., 1982	Randomized trial of 152 Asian women nutritionally at high risk in Birmingham.	Started giving the supplements at 18th-20th week of gestation through the 38th week (i.e. second and third trimesters).	Treatment groups got either: a) 273 kcal/d + 26g protein/d + 30mg vit C + 3mg Fe (first supplement); or b) 273 kcal/d + 30mg vit C + 3mg Fe (second supplement). Control group got 3mg Fe/d + 30mg vit C/d.	Birth weight was not statistically different among the three groups.	Mothers in the protein and energy supplement group gained significantly more weight and fat than the other two groups between the 18th and 28th weeks.
Rush et al., 1980	Randomized trial of 770 New York Black women weighing <140 lbs at conception and having at least one of the following conditions: Low weight gain - weight <110 lbs at conception-past Low birth weight (LBW) and/or consumption of <50g protein during the last 24 hours.	Beginning at 30 weeks of gestation until term.	Daily beverages to three groups: complement group got 6g protein/322 kcal in 8 oz. can. Supplement group got 40g protein/470 kcal in 8 oz. beverage daily. Control group got routine minerals and vitamins.	Mean birth weights (BW) for the supplement, complement and control groups were 2938g, 3011g and 2970g respectively. None was significantly different from the other groups. Significant increase in the duration of gestation in complement group to 11.7% in comparison to 16.3% in controls (non-significant).	No difference between complement and control groups in total weight gain or average weight gain during the study. Among women recruited before 15 weeks of gestation, weight gain during pregnancy higher in supplement group ($p < 0.05$).

	Description and Results				
Author's Name and Year Published	Population and Design	Time of Supplementation	Supplement	Impact on Newborn	Impact on Mother
Bhatngar et al., 1983	Prospective study of 131 pregnant women served by health center as supplement group, and 163 other pregnant women not served by the health center as a control group in Delhi, India. Average usual diet for both groups provided 1200 kcal/d.	From the 24th week of gestation until term.	Local recipe providing 16g of protein and 300 kcal/d for the supplemented group only.	Supplemented group had lower proportion of LBW than the control group (21.7% to 34.7% respectively) ($p<0.05$). Infant weight of the supplemented group higher than those of the control group at each month up to the age of three months.	Supplemented group gained 4.28 ± 0.85 kg in comparison to 3.57 ± 0.85 kg of the control group during the period 24-36 weeks of gestation ($p<0.001$).
Prentice et al., 1983	Prospective study of all pregnant women in a Gambian village comparing all births during two years of supplementation to outcomes of previous four years.	As soon as pregnancy was diagnosed, 16 weeks was the average gestational age at admission. Supplementation continued until term.	Groundnut-based biscuits and vitamin-fortified tea drink for six days/wk provided 950 kcal during the dry season and 1100 kcal/d during the wet season.	Mean birth weight increased by 186g. Six-fold reduction in the proportion of LBW (28.2% to 4.7%) in wet season. LBW was reduced from 11.7% to 8% in dry season ($p>0.05$). Mean birth weights of supplemented and unsupplemented groups in wet season were 3030g and 2844g respectively. There was a 186g increase in supplemented group ($p<0.01$). No statistical differences in birth weights between groups in dry season.	Not measured.

	Description and Results				
Author's Name and Year Published	Population and Design	Time of Supplementation	Supplement	Impact on Newborn	Impact on Mother
Karkjati et al., 1988, 1990	Controlled randomized trial of 542 nutritionally at-risk women recruited during the first or second trimester.	Supplementation started at 26th week and continued throughout the 28th week of gestation.	Women were given either high or low energy supplement. Dry powder in warm water provided 465 kcal and 7.1g protein for the height supplement group and 52 kcal and 6.2g protein for the low supplement group. Supplement was provided daily for both groups. Home diet provided 1500 ± 499 kcal and 41 ± 13g protein before the study. During the study, home diet provided 1514-1717 kcal, 4.44-44.2g protein depending upon the compliance.	Mean birth weight of the total group was 100g higher than the baseline mean birth weight (2933g vs 2835g) and LBW (<2500g) was reduced from 12.2% before the study to 9.5% after supplementation in all groups combined. Neither change in birth weight nor LBW rate were significant ($p>0.05$). The highest increase in birth weight was observed in the groups of highest intake of supplement and lowest home dietary intake.	No statistical difference in third trimester weight gain. Sum of skinfold thickness, BMI and four-week postpartum weight gain.

	Description and Results				
Author's Name and Year Published	Population and Design	Time of Supplementation	Supplement	Impact on Newborn	Impact on Mother
Watney and Alton, 1986	Randomized trial of 156 women (78 White and 78 Asian) gained inadequate increase in skinfold thickness during the second trimester of pregnancy as an index of malnutrition in Bromwich, England.	Third trimester until term.	Milk-based supplement with the same nutritional contents of the protein energy supplement of Viegas et al., 1982. The supplement was given to half the women in each group.	Supplemented women had lighter newborns.	Not measured.

Passive Smoking: What is the Evidence for Health Effects?

Technical Paper #8 prepared for the U.S. Department of Agriculture, Food and Nutrition Service, under Cooperative Agreement #58-3198-1-005, "Review of Nutritional Risk Criteria for the WIC Program", with the Department of Family and Community Medicine, University of Arizona, Tucson, AZ 85724 by Awal Dad Khan, MS, and Gail G. Harrison, PhD. Mr. Khan is Research Specialist in the Department of Family and Community Medicine and a doctoral candidate in Nutritional Sciences; Dr. Harrison is Professor of Family and Community Medicine, College of Medicine, University of Arizona, Tucson, AZ 85724.

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INTRODUCTION

Cigarette smoking constitutes the most important single preventable cause of premature death and disability for the U.S. population (DHHS, 1986). The adverse effects of cigarette smoking on the smoker have been thoroughly documented, and the concept that smoking is hazardous to health is now generally accepted by the public. Cigarette smoking has strong epidemiological associations with cancers of the lung and other respiratory tract cancers, coronary heart disease and myocardial infarction, peripheral vascular disease, and stroke. Maternal smoking during pregnancy contributes to perinatal mortality, intrauterine growth retardation, preterm delivery and low birth weight. In addition, deficits in school performance and development also have been associated with maternal smoking during pregnancy (Naeye and Peters, 1984; Sexton et al., 1990). In the last several years there has been growing concern about the health effects of cigarette smoking on nonsmokers who are exposed to the noxious products of the cigarette smoke of others ("passive smoking") (Huber, 1975). The available data on health effects of passive smoking is growing rapidly, showing in general that the dangers are real and significant, and parallel those of direct smoke. To date, most of the available research and commentary on the subject has focused on the health effects for nonsmokers, although passive smoking is potentially an issue for smokers and nonsmokers alike.

DEFINITIONS

The literature on smoking provides several terms to differentiate exposures by source. "Mainstream smoke" is the smoke which is directly inhaled by the smoker, filtered by the lungs and then exhaled into the environment (Johnson et al., 1990; Spitzer et al., 1990). "Sidestream smoke" is generated from the burning end of a tobacco product during the smoldering between puffs and is a mixture of irritating gases and tar particles; the particles in sidestream smoke are of smaller diameter than those in mainstream smoke and are more likely to deposit in the most distant alveoli of the lung (Klus and Kuhn, 1982). "Environmental tobacco smoke" is a combination of sidestream and the mainstream smoke exhaled by smokers that contaminates the air surrounding a smoker (Spitzer et al., 1990; Johnson et al., 1990). "Passive smoking" is generally considered to be the inhalation of environmental tobacco smoke produced by someone else's voluntary smoking. Some authors limit the definition to indoor environments (Cummings et al., 1989), and some have broadened the definition to include exposure of a fetus to smoke products and metabolites by way of active or passive maternal smoking (Spitzer et al., 1990).

Passive smoking involves breathing both sidestream smoke and exhaled mainstream smoke (Davis et al., 1989); environmental tobacco smoke in the immediate vicinity of a smoker contains about 85 percent sidestream smoke and 15 percent mainstream smoke (Byrd et al., 1989). Environmental smoke is about 2.5 times higher in carbon monoxide concentration than mainstream smoke (American Academy of Pediatrics, 1982).

PREVALENCE OF PASSIVE SMOKING

The prevalence of ongoing, prolonged exposure to passive smoke among nonsmokers is a function of two major factors: the prevalence of active smokers in the surrounding population and public and private policies which allow or prohibit active smoking in common areas such as public buildings and conveyances and the workplace. Although the

prevalence of mainstream smoking in the general U.S. population has declined over the past two decades, the decline has been slower for women than for men (CDC, 1989; USDHHS, 1989). The prevalence of smoking is higher among Blacks, among blue-collar workers and among less educated adults than in the overall population. Among women during pregnancy, there has also been a decline in smoking rates of about 0.5% per year over the last decade (Stockbauer and Land, 1991), with the decrease accounted for primarily by Black women, teenagers and better educated women; smoking rates among White pregnant women aged 20-29 years have actually increased somewhat. In 1987, twenty-six percent women of aged 18 to 24 years and 31 percent of those 25 to 44 years smoked. Thirty-one percent of women with less than a high school education smoked as compared with 15 percent of those with college education (Shoenborn and Boyd, 1989). Data from 26 states in the Behavioral Risk Factor Surveillance System in 1985 and 1986 showed that approximately one-fifth of U.S. women smoke throughout pregnancy. (Williamson et al., 1989).

There are few reliable estimates of the prevalence of passive smoke exposure; however, the American Academy of Pediatrics has estimated that 9 to 12 million children under the age of five years are exposed to smoke-contaminated environments at home. A recent study of non-smoking high school students estimated that 68 percent either resided with one or more smokers at home or were exposed to one or more friends' smoking environment for more than two hours/week (Tsimoyianis et al., 1987). The National Center for Health Statistics reported that in 1988 about one-half of all U.S. children under five years of age had been exposed to environmental cigarette smoke via prenatal maternal smoking and or sidestream smoke from household members (Overpeck and Moss, 1991). Twenty-one percent were exposed only after birth and 28 percent were exposed both prenatally and from subsequent household exposure. Low income and low maternal education were associated with higher exposure rates. Black children had higher exposure (60 percent) than Whites (49 percent) or Hispanics (44 percent). Only six percent of the children lived in households where smoking cessation was reported in the period since the baby was child was born.

MEASUREMENT OF EXPOSURE

Most of the literature on health effects of passive smoking has relied on report by the subject and/or parents about the number of active smokers in the environment. The presence and levels of cotinine, a metabolic by-product of nicotine, and thiocyanate in serum and urine have also been used to quantify exposure, and several studies now exist to indicate good agreement between severity of exposure judged by self- or parental reports and biological evidence of level of exposure for adolescents (Moskowitz et al., 1990) and for infants (Greenberg et al., 1989; Chilmonczyk et al., 1990). Chilmonczyk et al. (1990) collected household smoking information from 518 mothers of six- to eight-week-old infants and measured the infants' urine cotinine at their first well-baby checkup. Rates of urinary cotinine over 10 μg/L were 8% in 305 households in which no smokers were reported; 44% in 96 households where a member other than the mother smoked; 91% in 43 households where only the mother smoked; and 96% in 74 households where the mother and another household member smoked. A Chinese study (Chen et al., 1990) has documented that serum thiocyanate in infants corresponds to the number of cigarettes smoked collectively by family members per day, in a dose-response fashion. In general, the risks associated with passive and active smoking represent linear functions of exposure,

with no evidence of a threshold effect, i.e., all smoking can be expected to have an effect which corresponds directly to quantity and concentration of the smoke.

HEALTH CONSEQUENCES OF PASSIVE SMOKING

Acute Effects

Passive smoking has been reported to produce a variety effects reflecting acute irritation, including eye irritation, nasal discharge, headache, cough, sore throat, wheezing, hoarseness, nausea and dizziness when exposure is severe over even short periods of time (Weber, 1984; Speer, 1968; Horsfield, 1984; Mattson et al., 1989).

Respiratory Infections

There is abundant evidence from well-designed studies, including three large prospective cohort studies, that children who are heavily exposed to cigarette smoking have more upper and lower respiratory tract illnesses, and more severe respiratory illnesses and hospitalizations for bronchitis and pneumonia, than other children (Fergusson et al., 1980, 1981; Rantakallio, 1978; Byrd et al., 1989; Harlap and Davis, 1974; Chen et al., 1986; Charlton, 1984; Pedreira et al., 1985; Bland et al., 1978; Ware et al., 1984; American Academy of Pediatrics, 1982). One study estimated that an increase in mothers' smoking of five cigarettes per day on average would result in an annual increase of 2.5 to 3.5 incidents of lower respiratory illness per 100 children at risk (Fergusson and Horwood, 1985).

Several studies have controlled for effects of social class and of housing (Harlap and Davis, 1974; Chen et al., 1986). Persistent middle ear infections have also been linked to passive smoke (Kraemer et al., 1983; Stahlberg et al., 1986; Byrd et al., 1989; Black, 1985). Black (1985) estimated that 10 to 36 percent of chronic middle-ear effusions in children could be attributable to smoking exposure. Some studies have reported number of restricted-activity and bed-disability days per year increased for children in proportion to the number of adult smokers in the household (Ostro, 1989; Bonham and Wilson, 1981; Rantakallio, 1978).

Asthma

A strong direct relationship has also been documented between passive smoking and childhood asthma (Weitzman et al., 1990a and 1990b; Murray and Morrison, 1989; Evans et al., 1987; Burchfield et al., 1986; Horwood et al., 1985; Gortmaker et al., 1982; Dahms et al., 1981). Two studies of respiratory function in children support these findings. A cross-sectional study of fourth-grade students showed that boys of smoking parents had a risk of "increased airway responsiveness" four times that of boys with non-smoking parents, but girls showed a nonsignificant association. Boys whose parents smoked also showed increased reactivity to allergens as assessed by the skin prick test index (Martinez et al., 1988). The authors concluded that passive smoking, by increasing the frequency of bronchial hyperresponsiveness and of atopy, might increase the risk of asthma particularly in boys. In the other study, Tsimoyianis et al. (1987) measured the relationship between passive smoking, pulmonary function, and respiratory symptoms in 193 teenaged high-school athletes. They found a four-fold increase in prevalence of low forced expiratory

flow and of cough in athletes exposed to passive smoking compared to athletes not exposed (13.6% vs 3.31%). Boys were more frequently exposed to passive smoking than girls, but the effects were more pronounced in girls.

Risk Factors for Coronary Vascular Disease

In a study of passive smoke exposure among 216 preadolescent twin pairs from the Medical College of Virginia Twin Study (Moskowitz et al., 1990), children who were exposed to passive smoking (105 twin pairs) compared to those who were not exposed (111 pairs) had higher serum thiocyanate levels, higher levels of total serum cholesterol and lower levels of high density lipoprotein (HDL) cholesterol, after adjusting for age, weight, height and sex.

Effects on Lactation

Breast-fed infants whose mothers smoke may be at particular risk for exposure to smoke products since they inhale environmental smoke and also ingest smoke-related chemicals through breast milk (Labrecque et al., 1989). Whether there is any tendency toward lower rates or duration of breastfeeding or reduced milk production in smoking mothers is not clearly understood. In a mailed questionnaire survey of women at their four months postpartum in Norway, more smokers than nonsmokers reported stopping breastfeeding early because of "too little milk" ($p<.05$). The prevalence of breastfeeding at three months was 90 percent among nonsmokers and 65 percent among smokers ($p<.05$) (Matheson and Rivrud, 1989). Decreased basal prolactin values among smokers compared to nonsmokers during lactation has been reported (Andersen et al., 1982), and there are limited animal data suggesting a suppressive effect of nicotine on prolactin levels in rats (Hamosh et al., 1979).

Effects on the Fetus

Negative effects on birth weight, lean body mass of the newborn, and elevation of perinatal mortality risk is well documented among infants of mothers who actively smoke cigarettes (USDHHS, 1980, 1986; USDHEW, 1979; Abel, 1980; Naeye, 1981; Harrison et al., 1983). The mechanism is that smoking causes fetal hypoxia which arises as a consequence of increased carboxyhemoglobin levels, attenuated blood oxygen unloading, and vasoconstriction affecting maternal blood supply to the placenta (Longo, 1982; Abel, 1980). In addition, women who smoke tend to be lighter and to gain less weight in pregnancy (IOM, 1990). Fetal oxygen deprivation and nutritional factors result in growth retardation, with an average weight reduction in the newborns of smokers of 200 grams, ranging from 40 to more than 400 g in a dose-response fashion, independent of socioeconomic status and maternal age (Abel, 1980). Evidence supporting a causal link is supported by randomized clinical trials (MacArthur et al., 1987; Sexton and Habel, 1984). The effect on fetal growth is primarily on lean body mass, with adipose tissue relatively spared (Harrison et al., 1983).

There is much less documentation of the effects of passive smoking, but the evidence to date is consistent with similar effects as in active maternal smoking. Yerushalmy (1962) and Macmahon et al. (1966) some time ago reported a relationship between father's smoking and low birth weight risk, and to point out a heightened risk when both parents

smoked over maternal smoking alone. Animal studies in which pregnant mice or rats have been exposed to passive cigarette smoke have since showed an association with decreased birth weight (Bassi et al., 1984; Reznik, 1980; Haworth and Ford, 1972). One cohort study in the U.S. which measured the actual maternal exposure to cigarette smoke (Martin and Bracken, 1986) found a positive association between passive smoking and risk of low birth weight; the relative risk of low birth weight for mothers exposed to passive smoke for at least two hours a day compared to others was 2.17 (95 percent confidence interval 1.05-4.50) after adjusting for maternal age, gestational length, race, and parity. A similar finding was reported by Rubin et al. (1986) from their case-control study of Danish mothers whose husbands smoked. Whether smoking was examined as a continuous or a discrete variable, the effect of father's smoking on birth weight was significant. On average, birth weight was reduced by 120 g per pack of cigarettes (or cigar, or pipe equivalent) smoked by the father per day. In a recent British study (Mathai et al., 1990), maternal smoking was associated with a decrease in birth weight of 12 g per cigarette smoked a day and 25 g per microgram of cotinine/mg of urinary creatinine. Mothers who did not smoke actively but were exposed to smoke passively during pregnancy experienced no significant reduction in birthweight. In a prospective matched-pair study of German women and their infants, newborns not exposed to smoke prenatally weighed 400 g more on the average than newborns of smoking mothers, and 200 g more than newborns whose fathers smoked (Bickenbach et al., 1987).

A recent follow-up study of 714 three-year old children of women who had quit smoking during pregnancy and a comparison group where mothers continued to smoke (Fox et al., 1990) found that children of women who quit smoking during their pregnancy were taller and heavier than those whose mothers smoked throughout pregnancy. Deficits in weight were barely significant ($0.05 < p < 0.10$) but deficits in height were statistically significant ($p < .001$) even after adjustment was made for maternal and household variables and maternal postpartum smoking status.

Other anthropometric measurements of the newborn, such as length and head circumference, have been even less studied. Several studies have documented shorter length of newborns whose mothers actively smoke during pregnancy, with an average difference of approximately one cm (Bealac-Baillargeon, 1987; Nilsen et al., 1984), and the retardation in stature has been shown to persist to age seven years (Naeye, 1981). Rona et al. (1985) reported that children exposed to twenty cigarettes per day in the home were about 0.30 cm shorter at birth than children of non-smokers regardless of maternal smoking status during pregnancy (Rona et al., 1985). Bickenbach et al. (1987) found nonsignificant effects on newborn length and no effect on head circumference from passive smoke exposure during pregnancy.

Effects on Nutrient Intake and Tissue Levels

There is considerable evidence that active smoking increases metabolic rate (thus increasing energy requirements) and interferes with several metabolic pathways resulting in elevated needs for ascorbic acid and perhaps vitamin B12, folate, and zinc. To date, there is no evidence available to support or deny that exposure to passive cigarette smoke has similar effects. There are conflicting data on the relationship of maternal smoking to iron status in the newborn (IOM, 1990; Garn et al., 1978).

EFFECTS OF INTERVENTION FOR PREGNANT WOMEN

In spite of the long-appreciated direct links between cigarette smoking and adverse pregnancy outcomes, there have been relatively few smoking cessation programs reported specifically for pregnant women. It has been suggested that the elimination of smoking during pregnancy could reduce postnatal morbidity by about five percent (CDC, 1990; IOM, 1985). WIC-eligible women may be at higher risk for smoking than the general population; analysis of birth certificates of WIC participants for Missouri in 1982 indicated that 45 percent of pregnant WIC participants were smokers, compared to 30 percent among all Missouri women in that year (Land and Stockbauer, 1986). Data on 127,512 pregnant women in several states' WIC programs from 1979 through 1985 suggested that smokers had 2.5 times more low birth weight infants and very few participants stopped smoking after enrollment in the smoking cessation intervention programs sponsored by the WIC clinics (Neiburg et al., 1986).

Sexton (1991) had recently provided a review of smoking interventions during pregnancy. She cites six randomized clinical trials, two in Britain and four in the U.S., which reported quit rates (Baric et al., 1976; Donovan, 1977; Sexton and Hebel, 1984; Windsor et al., 1985; Secker-Walker et al., 1986; MacAuthur et al., 1987). There were significant methodological differences among the trials, including type and length of intervention, initial enrollment criteria,and method for assessing smoking status at the end of pregnancy. With one exception, all reported quit rates in the treatment group of nine to fourteen percent, approximately five percent higher than in control groups. The trial of Sexton and Hebel (1984) reported higher quit rates in both groups, but included women who had quit recently based on high risk of recidivism.

Other recent studies report some success with smoking cessation programs for pregnant women receiving prenatal care in a Health Maintenance Organization (Ershoff et al., 1989, 1990). Mayer et al. (1990) have reported the results of a randomized clinical trial in a WIC clinic setting in Grand Rapids, Michigan. Pregnant smokers attending the clinic were randomly assigned to a Usual Care (UC) group or a Multiple Component (MC) intervention group; the MC group received a twenty-minute, one-to-one counseling session focusing on risk information and behavior change strategies. The MC group had a higher quit rate (11 vs 3 percent for the UC group) assessed in the last month of pregnancy.

SUMMARY

There is now considerable evidence that passive smoking (exposure to the contaminants in the air generated by active smokers in the near environment) is associated with increased risk of respiratory illness and of severe respiratory illness in children. There is also abundant evidence of acute irritant effects of passive smoking on all ages, particularly from exposure at high levels over short periods of time such as in airplanes and other closed spaces. There is some preliminary evidence in humans, supported by animal model studies, that passive smoking in pregnancy can affect birth weight of the newborn.

Although the prevalence of cigarette smoking has been decreasing in the U.S. over the last two decades, this decrease has been uneven in the population. Among White women aged 20-29 years, the prevalence of smoking has actually increased. It is estimated that one-fifth of U.S. women smoke throughout their pregnancies. The fetus as "passive smoker"

when the mother is an active smoker has been thoroughly documented to be at risk for decreased birth weight, decreased birth length, impaired postnatal growth, and increased perinatal mortality. Smoking cessation programs have seldom focused on pregnant women, but have been shown in several instances to be more effective than no intervention.

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Technical Paper 9

Nutritional Risk Implications of Pica in Pregnancy

Technical Paper #9 prepared for the U.S. Department of Agriculture, Food and Nutrition Service, under Cooperative Agreement #58-3198-1-005, "Review of Nutritional Risk for the WIC Program", by Awal Dad Khan, MS, Research Specialist, Department of Family and Community Medicine, College of Medicine, University of Arizona, Tucson, AZ 85724. Mr. Khan is a doctoral candidate in the Nutritional Sciences Program, University of Arizona.

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INTRODUCTION

Pica is the persistent, compulsive ingestion of any substance, whether a common dietary item or a non-food item (Danford, 1982; Lackey, 1984; Shapiro and Linas, 1985). Pica may be limited to a single substance or may be generalized to include more than one food or non-food item (McLaughlin, 1987). Pica may be classified broadly into four categories based on the substance(s) ingested: a) geophagia, or eating of clay and earth; b) amylophagia, or eating of starch and/or starch paste; c) pagophagia, or ice-eating; and d) ingestion of miscellaneous materials, among which have been reported ash, carbon, chalk, magnesium carbonate, metal polish, plaster, shoe polish, soot, wax, and other items.

Pica is known in many parts of the world and occurs among men, women and children; of interest here is its prevalence among some U.S. subpopulations, where it is particularly associated with pregnancy. The most commonly reported types of pica in pregnancy are geophagia and amylophagia (Danford, 1982; Gelfand et al., 1975; NRC, 1982; Talkington et al., 1970; Keith et al., 1968; Keith et al., 1969b). Etiology is clearly culturally based and influenced, although there are suggestions that nutrient deficiencies may play a part in at least some cases. The purpose of this technical paper is to review the available literature pertaining to:

a) the prevalence of pica during pregnancy and risk factors for the practice, and
b) the potential health and nutritional consequences of the practice.

Much of the literature on pica among pregnant women in the U.S. is old and is largely anecdotal or case-study based. There is a striking lack of recent studies relating to either the prevalence or the consequences of pica during pregnancy.

PREVALENCE AND RISK FACTORS FOR PICA DURING PREGNANCY

Information on the prevalence of pica during pregnancy is somewhat sparse and inconsistent, and much of the relevant literature is quite old, in spite of the obvious nutritional implications of the practice. Definitions have not been uniform, and the practice is no doubt underreported by subjects since medical disapproval of pica is well known.

Pica among pregnant women has been consistently reported as most common in the rural South, where it is deeply ingrained in the traditions of the black population (Vermeer and Frate, 1975, 1979) and among black women in cities of both the South and the North among recent migrants from rural and/or southern areas (Maravilla and Berk, 1978). It also occurs, but with less frequency, among rural and urban and Hispanic women. There is considerable evidence that historically the practice was imported as a culturally-established behavior from West Africa, where dirt- or clay-eating is an established tradition in some areas, to the U.S. at the time of the slave trade. Geophagia during pregnancy became firmly established as a part of southern black culture in the U.S., later diffusing to southern whites and to the cities into which both black and white southern families migrated (Hunter, 1973; Vermeer and Frate, 1975). Amylophagia has not been reported in the Hispanic populations, and it is unclear whether geophagia in that group, which takes on a more structured and overtly religious form, diffused from other U.S. cultural groups or arose through a different historical route.

Among subgroups particularly at risk, prevalence rates among pregnant women have been reported to range up to 75 percent (Danford, 1982; Horner et al., 1991), indicating that in some subpopulations the practice must be looked upon as normative rather than deviant. While pica has sometimes been reported in the literature as an eating disorder, it is so common and institutionalized in some groups that it is difficult to regard as disordered behavior (Ferguson, 1989). There is evidence that the practice is becoming less common (or less commonly reported), but it is still widespread in some regions at least through the mid-1980s.

Table 1 summarizes reported prevalence of geophagia and/or amylophagia among pregnant women in studies from 1950 through 1987. Most (16 of 20 studies) were cross-sectional prevalence studies; three were historical cohort studies and one was a prospective cohort study.

Table 1

Prevalence of Pica (clay, dirt, starch eating) During Pregnancy

References (year)	Region	Study Design*	Population Race/Residence	Prevalence (Percent)
Ferguson & Keaton (1950)	Mississippi	CS	Black, Rural	68
			White, Rural	17
Posner et al. (1957)	Harlem, NY	CS	Black, Urban	18
Edwards et al. (1954)	Tuskegee, AL	HC	Black, Rural	50-75
Edwards et al. (1959)	Tuskegee, AL	CS	Black, Rural	27-49
Payton et al. (1960)	Tennessee	PC	Black, NA	18
Dunston (1961)	New York	HC	Black, Urban	43
Sage (1962)	Pennsylvania	CS	Black, Urban	40
Edwards et al. (1964)	Tuskegee, AL	HC	Black, Rural	na
O'Rourke et al. (1967)	Augusta, GA	CS	Black, Rural	55
Keith et al. (1968)	Chicago, IL	CS	Black, Urban	38
			White, Urban	11
Keith et al. (1969a)	Chicago, IL	CS	Black, Urban	26
			White, Urban	0
Bruhn & Pangborn (1971)	California	CS	Spanish American	38
			White	19
Rogers (1972)	Alabama	CS	Black, Rural	45
Bronstein & Dollar (1974)	Augusta, GA	CS	Black, Rural	18
			Black, Urban	14
Mansfield (1977)	Pitt County, NC	CS	Black, Urban	19
			White, Urban	0
Lackey (1978)	Tennessee	CS	Black, NA	54
			White, NA	27
Vermeer & Frate (1975)	Mississippi	CS	Black, Rural	28
Vermeer & Frate (1979)	Holmes County, MS	CS	Black, Rural	47
Butler (1982)	Pitt County, NC	CS	Black, Urban	35
			White, Urban	6

Cited in Horner et al. (1987)	Pitt County, NC	CS
	Black, Urban	12
	White, Urban	0

* CS: Cross-Sectional; HC: Historical Cohort; PC: Prospective Cohort; NA: Not Available

Horner et al. (1991), in the only very recent treatment of the subject in the literature which we were able to identify, have contributed an analytical review in which they have estimated the relative strength of race, rural/urban residence, and chronological time in predicting the likelihood of pica during pregnancy. They used appropriate statistical methodology to estimate odds ratios controlling for confounding variables, and concluded that race and rural residence are both major predictors, that the prevalence of (reported) pica is decreasing over time, and that southern vs northern residence is a significant but relatively weak predictor of risk. Their analysis showed that black women were more than four times as likely as white women to report pica; that rural residents were more than twice as likely as urban residents; and that residents of the South were slightly more likely than northern residents to report pica in pregnancy. Table 2, from their review, shows a moderate decline in reported pica by decade, from more than one-third of all women studied in high-risk areas in the 1950s and 1960s to about one-quarter in more recent studies.

Table 2

Prevalence of Pica During Pregnancy by Ten-Year Period*

Period	Prevalence (Percent)	95% Confidence Intervals
1950-1959	38.3	(34.6, 41.8)
1960-1969	33.7	(31.9, 35.9)
1970-1979	26.5	(23.1, 30.0)
1980-1987	28.0	(22.4, 34.6)

* Source: Horner et al. (1991)

Besides ethnicity and residence, a variety of other risk factors for pica have been mentioned in the literature, including socioeconomic status, age, psychosocial and family stress, religious beliefs, family history including practice of pica by mother and grandmothers (Furuset, 1973; Mansfield, 1977), and nutritional deficiencies particularly of iron, zinc, and calcium (Sayetta, 1986). While there is a documented association of pica with mineral deficiencies, especially iron, it is not at all clear whether the deficiency precedes or is consequent upon the practice.

The most frequently reported reasons for pica are obviously cultural. For geophagia they include taste, perceived protective effects on the fetus, and relief of various conditions during pregnancy including nausea, dry mouth, and nervous tension (Lackey, 1984).

Motivations for amylophagia which have been reported include helping to produce a healthy baby, to make the baby's skin lighter, and to facilitate easy delivery (Lackey, 1984). Starch has also been broadly used in folk medical systems to treat intestinal distress (Lackey, 1984).

HEALTH AND NUTRITIONAL EFFECTS OF PICA IN PREGNANCY

Medical Complications

There have been rare reports of medical complications arising from pica, including one maternal death (Key et al., 1982), gastric obstruction (Allan and Woodruff, 1963), complicated delivery (Gudsdon and Tunca, 1974) and dysfunctional labor due to fecal impaction (Holt and Hendricks, 1969).

Toxemia of Pregnancy

Several studies have related pica to increased risk of toxemia of pregnancy; however, they have not been controlled for other potentially contributory variables. O'Rourke et al. (1967) found toxemia to be twice as prevalent in mothers who reported practicing pica as in those who did not. Much earlier, Ferguson and Keaton (1950) had reported elevated incidence of toxemia among clay-eating pregnant women in rural southern Mississippi compared to women who did not report pica. Two studies conducted in the 1960s found similar prevalence of toxemia in clay- and starch-eating pregnant women (Edwards et al., 1964; Keith et al., 1968); one of them (Edward et al., 1964) documented hypertension and edema to be twice as prevalent in clay-eating pregnant women as in those who did not report pica. None of these studies controlled for parity or other variables which probably influence risk of toxemia, the etiology of which is still unknown. Thus we may only conclude that pica may be associated with increased risk of toxemia, but that there is no evidence on which to base cause-and-effect conclusions.

Outcome of Pregnancy

The relationship, if any, between pica and pregnancy outcome has not been well documented, although several authors have suggested an association between the prevalence of pica in pregnant black women and their poorer birth outcomes relative to white women (Furuset, 1973; Mansfield, 1977). Edwards et al. (1964) found that there was no difference in birth weight, length, head or chest circumference, or gestational age in infants born to mothers who reported eating clay or cornstarch during pregnancy and those who did not. They did, however, find an elevated risk of the newborn being rated "poor" or "very poor" on the hospital's routine rating procedure (unspecified), and higher percentages of stillbirths and early neonatal deaths among infants of mothers who reported pica.

Nutritional Consequences of Pica

There are a number of possible mechanisms by which pica might interfere with adequate nutriture, including the following:

1. The ingested materials may provide calories without other essential nutrients (consumption of one pound of starch a day, an amount not unusual in reported studies, provides 1590 kcal, composed of 86 percent carbohydrate and 14 percent water, and no significant amounts of other nutrients).

2. Any material consumed in large quantities, even if non-caloric, may displace the consumption of essential nutrients and energy from food sources. Reported consumption of clay, for example, has ranged from 120 to 650 g/day.

3. The material may serve as a mineral exchanger, resulting in malabsorption of needed nutrients.

4. The ingested materials may contain toxic substances.

There has been little study of the overall diet and nutritional status of women who practice pica. Edwards et al. (1964) studied a group of pregnant black women at a prenatal clinic in Alabama, and found geophagia to be significantly associated with low intakes of iron-rich foods, thiamine and niacin. Intakes of protein, calcium and iron were quite low in the study population as a whole and not appreciably different in women with and without pica. Although their methodology did not allow distinguishing energy intakes between women who did and did not report practicing pica, they concluded that omission of clay or cornstarch from the diet appeared to influence appetite for food, as reflected in lower within-subject calorie intakes from food on days on which these substances were consumed.

Interactions of pica with nutritional status have been reported primarily in relation to minerals, particularly iron. Zinc (Ronaghly and Halsted, 1975; Danford et al., 1982) and magnesium (Danford et al., 1982) status have been reported to be compromised in the presence of significant pica in case reports. Iron status, however, has been more extensively studied.

All three common forms of pica (geophagia, amylophagia, and pagophagia) have been associated with iron deficiency and anemia (Ferguson and Keaton, 1950; Anziulewicz et al., 1959; Dunston, 1961; Edwards et al., 1964; O'Rourke et al., 1967; Keith et al., 1968, 1969a; Reynolds et al., 1968; Coltman, 1969; Campbell and Davidson, 1970; Bronstein and Dollar, 1974). O'Rourke et al. (1967) found average hemoglobin values in pregnant women in Georgia to be significantly lower in practicers of pica than in controls. Several years later, Bronstein et al. (1974) in the same Georgia community found more evidence of iron depletion among pregnant women practicing pica than among those not reporting pica. Among 27 subjects reporting pica, 55.5 percent had low serum iron levels, compared to 13.6 percent of 22 controls. Keith et al. (1968) studied laundry starch eaters, and found only minor differences in iron status (average hemoglobin level of 9.6 g/dl, compared to 10.2 g/dl for a control group not reporting pica). In another study by the same author, differences were also marginal (Keith et al., 1969b), with differences in hemoglobin, hematocrit, and mean corpuscular volume of less than five percent. The author of these studies theorizes that ingestion of laundry starch contributes to anemia by displacement of iron-rich foods from the diet, a hypothesis favored over any specific mechanism for interfering with iron bioavailability also by other authors (Sage, 1962). Blum et al. (1968) concluded that iron absorption was reduced more than 50 percent

when iron therapy and laundry starch eating were combined. Talkington et al. (1970) found that iron absorption and utilization were not affected by clay or starch in the presence or absence of a standard dose of ferrous iron; they also concluded that if starch contributes to iron deficiency anemia it does so by displacement of iron-containing foods in the diet.

Clay contains substances which chelate ionized minerals and interfere with both iron and zinc absorption (Halsted, 1968; Crosby, 1971; Arcasoy et al., 1978; Ferguson, 1989; Wintrobe et al., 1981). Minnick et al. (1968) found that ingestion of Turkish clay decreased absorption of a test dose of iron by 25 percent. Patterson and Staszak (1977) fed pregnant rats clay and found significant reductions in hemoglobin and hematocrit, and in birth weight of the pups. Magnesium carbonate, a less commonly-reported pica substance, also interferes with iron absorption and has produced severe anemia in at least one case study (Leming et al., 1981). Minnick et al. (1968) demonstrated that in vivo, labeled iron was absorbed less efficiently when in the presence of clay.

The relationship between iron therapy and pica has eluded clarification (Ansell and Wheby, 1972; Coltman, 1969). Pagophagia (ice-eating and craving for ice) has been variously reported to subside after treatment with iron (Reynolds et al., 1968) and to abate with medical attention before any influence of iron therapy on iron status (Coltman, 1969). Mengel et al. (1964) reported that geophagia ceased with iron therapy. To our knowledge, no studies on the relationship between iron status and pica have been published since routine obstetrical practice has incorporated iron supplementation for all pregnant women receiving prenatal care.

The effects of nutrition education on pica and on dietary quality among women with pica have virtually not been studied. One recent study of low-income pregnant adolescents enrolled in a "Teen Pregnancy Service" in Milwaukee, Wisconsin in 1987-88 (Schnech et al., 1990) documented pica in about 10 percent of participants but did not attempt to explore the effects of the program on the practice.

SUMMARY

Pica in pregnant women, most commonly the regular ingestion of cornstarch or earth, is apparently decreasing in prevalence but remains common in the rural south and among recent migrants to cities in several areas. It is more common among black women than other ethnic groups, and is probably underreported. Both geophagia and amylophagia are clearly associated with iron deficiency, although cause and effect are unclear. The most supportable explanation is displacement of iron-containing foods from the diet (particularly for starch, which provides energy) along with some negative influence on bioavailability of nonheme iron (particularly for clay). Pica may be regarded as a risk factor for poor dietary quality in general; although the evidence to support this statement is thin, there is no evidence to the contrary and on the grounds of caloric and volume displacement alone it seems a reasonable conclusion. Nutrition education is potentially effective in improving dietary quality for mothers who practice pica.

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Technical Paper 10

Intake of Caffeine and Related Compounds: Evidence of Nutritional Risk in Pregnancy

Technical Paper #10 prepared for the U.S. Department of Agriculture, Food and Nutrition Service, under Cooperative Agreement #58-3198-1-005, "Review of Nutritional Risk Criteria for the WIC Program", by Laura Kettel Khan, MIM, Senior Research Specialist, Department of Family and Community Medicine, College of Medicine, University of Arizona, Tucson, AZ 85724. Ms. Kettel Khan is a doctoral candidate in Nutritional Sciences at the University of Arizona.

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INTRODUCTION

The purpose of this technical paper is to review the literature relevant to the question "Does intake of caffeine or caffeinated beverages have health and nutritional consequences for the WIC target population?" Specifically the paper addresses this issue for pregnant and lactating women.

PREVALENCE OF CAFFEINE USE

Caffeine (1,23,7 trimethylxanthine) is a plant alkaloid that is related to uric acid, adenine and guanine which are all structurally dioxypurines (Weathersbee et al., 1977). Caffeine is listed in the Code of Federal Regulations as a multipurpose food substance that is generally recognized as safe (GRAS). Caffeine is found in coffee, tea, cocoa, cola beverages, solid milk chocolate, and over the counter medications (cold tablets, allergy and analgesic preparations, appetite suppressants, and stimulants) (Bunker et al., 1979; Dalvi, 1986; Watkinson et al., 1985). Tea and cocoa also contain other methylxanthines, theophylline and theobromine respectively. Dietary caffeine is overwhelmingly derived from beverages. Chocolate and other confectioneries contain only a few milligrams per 100 grams, while a single cup of coffee or tea contains approximately 80 milligrams of caffeine depending on the strength of the infusion and the size of the cup. Soft drinks contain lower but variable amounts of caffeine, averaging about 0.01 percent or 45 milligrams per 12 ounces.

More than eighty percent of U.S. adults consume caffeine in some form, with several million kilograms of caffeine being consumed annually in the United States (Graham, 1978). However, the distribution of habitual caffeine consumption within the consuming population is not well documented. The mean consumption of caffeine for individuals over 18 years of age in the United States is estimated to be 2.6 milligrams per kilogram per day with ten percent of the population consuming more than 5.4 milligrams per kilogram per day (one cup of coffee provides about 1 mg/kg body weight for the average size adult) (Burg, 1977).

Caffeine is a readily absorbed and freely diffusible substance. However, when taken in the usual manner in the diet, peak levels following a given intake are much lower and less predictable than might be expected. Axelrod and Reichenthal found that the peak serum levels of caffeine attained by normal consumers may range from 5 to 6 milligrams per liter maintained over a few hours per day (Axelrod et al., 1953). The metabolism of caffeine can be influenced by pregnancy, oral contraceptive use, diet, smoking, age, gender, ethnic origin, and health status (Kalow, 1985; Yesair et al., 1984).

During pregnancy, some women develop a temporary "loss of taste" for caffeine and tend to reduce their intake (Aldridge et al., 1979; Rosenburg et al., 1982; Watkinson et al., 1985). Estimated average intakes during pregnancy for U.S. women range from 99 to 270 milligrams per day (Beaulac-Baillargeon et al., 1986; Graham, 1978; Srisuphan et al., 1986; Tebbutt et al., 1984). Estimates of the prevalence of use of caffeine during pregnancy range from 69 to 79 percent (Graham, 1978; Resch et al., 1985; Srisuphan et al., 1986) to 90 to 98 percent (Beaulac-Baillargeon et al., 1986; Hill, 1973; Watkinson et al., 1985). Heavier caffeine consumption has been found to be related to less formal education (Watkinson et al., 1985), greater pre-pregnancy weight (Linn et al., 1982; Watkinson et al., 1985), older maternal age (Linn et al., 1982), greater gravidity and parity (Watkinson et al.,

1985), alcohol usage (Watkinson et al., 1985), and cigarette smoking (Beaulac-Baillargeon et al., 1986; Istvan et al., 1984; Linn et al., 1982; Srisuphan et al., 1986; Watkinson et al., 1985).

HEALTH AND NUTRITIONAL EFFECTS OF CAFFEINE AND CAFFEINATED BEVERAGES

Acute effects of caffeine and other methylxanthines include: catecholamine release, increased free fatty acid mobilization, stimulation of the central nervous system increased gastric acid secretion, increased heart rate and basal metabolic rate, altered blood pressure and blood vessel diameter, diuresis and relaxation of smooth muscle (Brooten et al., 1983; James et al., 1985; Temples et al., 1985). Consumption of 500 to 750 milligrams per day of caffeine can also result in restlessness, anxiety, irritability, agitation, muscle tremor, sensory disturbances, heart palpitations, nausea, vomiting and diarrhea (Josephson et al., 1976; Victor et al., 1981). The biological effects of limited dietary intakes of caffeine are slight and not easy to detect; the most important are behavioral which are undramatic and subtle.

Habitual caffeine use has been hypothesized to be related to a variety of health problems, including pancreatic cancer, diabetes, peptic ulcer, fibrocystic breast disease and elevated plasma cholesterol and cardiovascular disease (MacMahon et al., 1981; Cheraskin et al., 1968; Raebel et al., 1984; Bergman et al., 1987; LaCroix et al., 1986; Yano et al., 1987). These linkages are controversial and not of sufficient strength to warrant recommending changes in caffeine consumption for the population as a whole.

Information about the nutritional status of pregnant women who consume caffeine is limited, and has been recently reviewed in *Nutrition in Pregnancy* (Institute of Medicine, 1990). Watkinson and Fried (1985) reported that women who consumed more than 300 milligrams of caffeine a day during pregnancy tended to be lower in weight for height and to consume less energy, protein, calcium, vitamin A, thiamin, riboflavin, and vitamin C than women who consumed 300 milligrams per day or less. The comparison was based on only a small number of heavy caffeine users and the differences were not statistically significant. In a prospective study of pregnant women in Costa Rica (Munoz et al., 1988), consumption of three or more cups of coffee per day was significantly associated with lower maternal and neonatal hemoglobin and hematocrit levels. Prenatal supplementation with iron did not appear to prevent the decrease in hematologic values. The authors note that in this population coffee consumption may encourage calcium intake since it is commonly diluted with large quantities of milk.

Intake of coffee and tea may have effects on nutrient bioavailability due to compounds other than caffeine, namely tannins and tannates. Increases in urinary calcium excretion (Massey and Hollingber, 1988a,b), decreased urinary thiamin excretion (Lewis and Inoue, 1981), and decreased absorption of zinc and iron (Morck et al., 1983; Pecoud et al., 1975) have been reported. Iron absorption from bread was reduced to one third and from a vegetable soup to one fourth when served with tea compared with water (Disler et al., 1975a). In a Western-type breakfast, iron absorption was reduced about 60 percent by tea (Rossander et al., 1979). In a study on the effect of various drinks on the absorption of non-heme iron from a hamburger meal, tea reduced the absorption by 64 percent and coffee by 39 percent (Morck et al., 1983). The role of tannin in tea in inhibiting non-heme

iron absorption has been attributed to the formation of insoluble iron tannates formed within the gastrointestinal lumen which renders the iron unavailable for absorption (Conrad et al., 1962; Disler et al., 1975a). It is possible that the inhibiting effect of coffee on iron absorption is also due to tannates. It has been suggested that tannins may be partly responsible for the low bioavailability of iron in many vegetable foods (Disler et al., 1975b).

PRENATAL EXPOSURE TO CAFFEINE

Caffeine is lipolytic and therefore readily crosses biologic membranes, including the placenta (Arnaud et al., 1983; Goldstein et al., 1962; Horning et al., 1973). It then equilibrates rapidly between the maternal plasma and the fetus (Axelrod et al., 1953). Caffeine is actively secreted in uterine secretions, and substantial quantities have been documented in the amniotic fluid, the umbilical cord blood, and the urine and plasma of neonates (Sieber et al., 1971; Sommer et al., 1975; Cook et al., 1976; Parsons et al., 1976; Dumas et al., 1982; Horning et al., 1973). Caffeine passes readily to the fetus, but the fetus cannot metabolize caffeine effectively, nor can the infant do so until several months after birth (Aldridge et al., 1979). Maternal consumption of two cups of coffee significantly increases maternal epinephrine concentrations and temporarily decreases intervillous placental blood flow (Kirkinen et al., 1983). Maternal consumption of regular coffee was associated with a twofold increased in the incidence of fetal breathing activity and a significant fall in baseline fetal heart rate; decaffeinated coffee also increased the incidence of fetal breathing activity and produced a slight reduction in fetal heart rate (Salvador et al., 1989). McGowan and colleagues (1987) compared fetal breathing rates and body movement incidences between two caffeine consumption groups; high (greater than 500 mg/day) and low (less than 250 mg/day). Fetal breathing rates and body movement incidences were similar in both groups before and after administration of 200 mg caffeine. Fetal breathing movement incidence decreased significantly in the low consumption group but was sustained at baseline levels in the high consumption group.

The U.S. Food and Drug Administration issued a warning in 1980 advising pregnant women to avoid or limit caffeine intake based largely on teratogenesis in rodents. This recommendation is extremely conservative, given the conceptual and biological leaps which would be required to extrapolate the data to the human situation (Goyan, 1980). Rodents are generally much more susceptible to teratogenic influences than are primates, and there are differences between rodents and humans in the metabolism of caffeine (Arnaud et al., 1983; Gilbert et al., 1986; Latini et al., 1981; Yesair et al., 1984). Further, the dosage level necessary to induce malformations in rodents is far more than can be achieved by usual human intake (Bergman et al., 1987; Wilson et al., 1984). In order to ingest the equivalent of 100 milligram per kilogram per day of caffeine (the level necessary to induce malformations in pregnant rats), a 60 kg human would need to consume approximately 50 to 70 cups of coffee per day, or at least 20 cups per day if corrections are made for "metabolic body weight" (Weathersbee et al., 1977).

Few carefully controlled studies on the role of caffeine in human pregnancy outcome have been conducted, and almost all of the available data for humans are based on retrospective questionnaire or interview methods. Some studies have focused on a single source such as coffee while ignoring tea, cola beverages, and over-the-counter drugs. The existing human studies provide incomplete and conflicting results with regard to the effect of caffeine consumed during pregnancy on the risk of birth defects, spontaneous abortion, pre-term

delivery and low birth weight (Brooten et al., 1983; Ernster et al., 1984; James et al., 1985; Leviton, 1984; Morris et al., 1981; Weathersbee et al., 1979; Weathersbee et al., 1977).

Caffeine consumption has been associated with an increased incidence of spontaneous abortion in some studies (Mau et al., 1984; Srisuphan et al., 1986; Weathersbee et al., 1977), but not in others (Linn et al., 1982; Watkinson et al., 1985). Decreases in infant body weight have been reported with maternal caffeine use of greater than 300 milligram per day (Kuzma et al., 1981; Mau et al., 1974; Watkinson et al., 1985; Caan et al., 1989; Martin et al., 1987). Several studies have looked for associations between congenital malformations and maternal caffeine use and none have been found (Berkowitz et al., 1982; Kurppa et al., 1983; Kuzma et al., 1981; Linn et al., 1982; Rosenberg et al., 1982) except by Borlee et al. (1978). No documentation is available on infant and child growth patterns after prenatal exposure to caffeine.

A recent prospective cohort study which investigated the relationship between caffeine use (coffee, tea, soft drinks, drugs) and late spontaneous abortion has reported a positive association (Srisuphan et al., 1986). In a sample of 3135 subjects the unadjusted relative risk for late first or second trimester spontaneous abortion in moderate to heavy caffeine users (> 150 milligrams daily) was 1.69 (95% CI (confidence interval) 1.04-2.71). When the possible confounding factors of gestational age at interview, maternal age, prior gynecological surgery, religion and history of spontaneous abortion were controlled for, the adjusted relative risk was relatively unchanged at 1.73. Light caffeine use (1 to 150 milligrams daily) was associated with an increased risk of spontaneous abortion only among women who had a spontaneous abortion in their previous pregnancy.

A significant association between coffee consumption and low birth weight (< 2500 grams) but not with length of gestation has been reported in a German study (Mau et al., 1974). In this prospective cohort study 5200 women from 20 maternity departments were interviewed during the first three months of pregnancy about their coffee intake and other habits such as alcohol consumption and smoking. Using 'none', 'seldom' or 'frequent' as categories, 4.7 percent of women who consumed no coffee had low birth weight babies compared to 6.4 percent who seldom drank coffee and 7.5 percent who were frequent coffee drinkers. In a prospective study of 3,891 antenatal patients at Yale-New Haven Hospital between 1980 and 1982, 76.7 percent consumed caffeine from coffee, tea, colas, and drugs (Martin et al., 1987). When comparison was made with women who had no caffeine exposure, the relative risks of low birth weight after adjustment for confounding factors were 1.4 (95% CI 0.7 -3.0) for 1 to 150 milligrams of caffeine daily; 2.3 (95% CI 1.1-5.2) for 151 to 300 milligram; and 4.6 (95% CI 2.0-10.5) for over 300 milligrams. Decreases in mean birth weight were 6, 31 and 105 grams, respectively.

A case-control study examining the effect of first trimester maternal caffeine consumption and risk for low birth weight was performed in the Kaiser Permanente Medical Care Program (Caan et al., 1989). Heavy consumption (> 3 serving/day) of coffee, cola and greater than 300 milligrams per day of caffeine from all sources was associated with a marginally increased risk of low birth weight. The association between maternal caffeine consumption and low birth weight, intrauterine growth retardation, and prematurity, adjusting for multiple confounding factors was examined in a case-control study of 1,230 women (Fenster et al., 1991). Heavy caffeine consumption (>300 mg/day) was

associated with intrauterine growth retardation [under odds ratio 3.86 (95% CI = 1.80, 8.40), after adjustment 2.90 (95% CI = 1.23, 6.87)]. Caffeine consumption was unrelated to preterm delivery.

Weathersbee and his colleagues conducted a retrospective study based on questionnaire returns from 489 predominantly Mormon households (Weathersbee et al., 1977). Spontaneous abortion, stillbirth and premature delivery were increased among 16 women who consumed greater than 600 milligrams of caffeine per day. A high rate of complications was also reported for the pregnancies of women who consumed less than 400 milligrams. A cause and effect relationship between high levels of caffeine consumption and poor pregnancy outcome cannot be drawn from this study due to poor study design and unaccounted confounding variables.

Linn et al. interviewed 12,205 women during the first trimester of pregnancy and then analyzed their medical records following delivery (Linn et al., 1982). No associations were found between consumption of four or more cups of coffee per day and spontaneous abortion, low birth weight, short gestation or congenital malformations. Watkinson and Fried examined retrospective data collected from 286 women participating in the Ottawa Prenatal Prospective Study (Watkinson et al., 1985). When caffeine was considered as a continuous variable, no significant relationships were found between pre-pregnancy and pregnancy maternal caffeine use and birth weight and length, head circumference, ponderal index, Apgar scores, length of labor or gestation. Caffeine consumption of greater than 300 milligrams per day was associated with a lowered birth weight and ponderal index and a smaller head circumference, but not with an increased incidence of miscarriage, premature birth, Caesarean section or breech birth. Borlee et al. reported that that self-reported consumption of coffee was significantly higher in a group of 202 mothers of newborn children with birth defects than in a control group of 175 mothers of normal children; however, they did not control for alcohol use or cigarette smoking.

TRANSMISSION OF CAFFEINE TO THE NURSING INFANT

Given the high prevalence of caffeine use in the U.S. population, it is important to determine the possible effects of early caffeine intake through breast milk (Berlin et al., 1984). Caffeine passes into breast milk but does not freely diffuse. The caffeine level in breast milk approximates one percent that in the mother's plasma (Lawrence, 1980). Caffeine is also less bound by constituents of breast milk relative to those in serum (Tyrala et al., 1979). Aranda and his colleagues found maternal plasma caffeine concentration to be highly correlated with breast milk concentration but not with the infant's plasma concentration, presumably because of a concomitant increase in the rate of caffeine elimination with increasing age (Aranda et al., 1980). Although the diffusion rate of caffeine into breast milk is minimal, consumption of approximately 600 milligrams per day of caffeine has been reported to cause wakefulness and irritability in the sensitive nursing infant (Lawrence, 1980). No ill effects in infants have been reported with maximum intakes 200 to 336 milligrams per day of caffeine by their nursing mother (Berlin et al., 1984; Lawrence, 1980). Infants metabolize caffeine very slowly and could theoretically accumulate a significant level of caffeine in relation to weight.

SUMMARY

At this time the existing literature provides incomplete and conflicting evidence as to the effects of caffeine on pregnancy outcomes. Because of the difficulties in interpreting many of the existing studies it would seen premature to make definitive statements. However, the five most carefully designed studies (Watkinson et al., 1985; Srisuphan et al., 1986; Martin et al., 1987; Caan et al., 1989; Fenster et al., 1991) do indicate an association with both spontaneous abortion and low birth weight and on this basis women should probably be advised to limit their caffeine consumption during pregnancy. A similar recommendation for nursing mothers would seem appropriate based on the inability of the young infant to rapidly metabolize the compound. There is insufficient evidence to support recommendations to completely avoid intake of caffeine or related compounds during either pregnancy or lactation. There is no particular evidence that heavy caffeine use prior to pregnancy predisposes to nutritional risk during pregnancy. In 1980, the U.S. Food and Drug Administration (FDA) recommended that the most prudent action for pregnant women and those who may become pregnant was to use caffeine-containing products sparingly or avoid them all together (U.S. Food and Drug Administration, 1980). The FDA subcommittee concluded that although it appears reasonable to curtail caffeine intake during pregnancy, the data are inadequate to make a specific recommendation.

There is little available information on the effects of caffeine and related compounds on maternal and infant nutrition, although it is clear that the tannin and tannates present in tea and coffee have a negative effect on the bioavailability of nonheme iron when consumed in the same meal.

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Technical Paper 11

Drug Use and Nutritional Risk in Pregnancy

Technical Paper #11 prepared for the U.S. Department of Agriculture, Food and Nutrition Service, under Cooperative Agreement #58-3198-1-005, "Review of the Nutritional Risk Criteria for the WIC Program", by Laura Kettel Khan, MIM, Senior Research Specialist, Department of Family and Community Medicine, College of Medicine, University of Arizona, Tucson, AZ 85724. Ms. Kettel Khan is a doctoral candidate in the Nutritional Sciences, University of Arizona.

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INTRODUCTION

The purpose of this technical paper is to review the available information to address the issue "What are the health and nutritional risks associated with illicit drug use in the WIC target population?" We will review information on the prevalence of drug use, particularly among women of childbearing age, and the very limited data available on nutritional status and diet of drug users. Finally, we present a brief summary, as an Appendix, of the effects of prenatal exposure to specific drugs.

PREVALENCE OF DRUG USE IN AMERICA

As drug use has risen in America in the last 10 years, so has concern about the health and safety of child-bearing age women (15 to 44 years) and children who are born into and raised in addicted households. There are approximately 10 million adult alcoholics, 500,000 heroin addicts, and between 5 and 8 million regular cocaine users in the United States (Black et al., 1980 and Chasnoff, 1988). Between 20 percent and 50 percent of the total population is at risk of serious dysfunction from the misuse of drugs, according to a study by juvenile and family court judges (Thomas, 1989). Most drug users are in their child-bearing years (Dixon, 1989). More than 300,000 infants are born annually to American women using crack cocaine and 10,000 infants to women using opiates (Chasnoff, 1989b and Hans, 1989).

Drug-use patterns have changed recently within the United States in several respects (Chasnoff, 1987a). More women of child-bearing age use drugs, particularly cocaine. Until recently figures showed that fewer women than men abused drugs. A recent Justice Department study, however, indicated that women arrested in 14 cities were as likely or more likely than men to abuse heroin, cocaine, and amphetamines (U.S. Department of Justice, 1989). In addition, adolescent cocaine abuse is a serious contributor to the total drug problem (Smith, 1986).

National statistics for 1985 indicate that 8 to 14 percent of women of child-bearing age are addicted to drugs. In 1988, the National Institute on Drug Abuse (NIDA) estimated that of the 56 million American women between 15 and 44 years of age, 15 percent were currently substance abusers. Estimates are that 2 to 3 percent are dependent on mood-altering drugs, 2 to 3 percent are cocaine dependent, 0.5 percent are opiate dependent, and 5 to 8 percent are alcohol dependent (U.S. Congress, House Select Committee on Children, Youth and Families, 1986). Although statistical data are insufficient, there are indications that approximately 1 in 10 infants may be exposed to illicit drugs during pregnancy. The National Institute on Drug Abuse 1988 National Household Survey revealed that 8.8 percent of women of child-bearing age admitted to having used an illicit drug in the month before questioning (National Institute of Drug Abuse, 1989). A recent survey of 36 private and public hospitals showed that approximately 11 percent of women delivering in these hospitals had used illegal drugs at some time during their pregnancies (Chasnoff, 1989a). Depending on the population and how carefully health care providers look for this problem, illegal substance abuse in pregnancy is detected in 0.4 to 37 percent of women (Chasnoff, 1989a).

As drug use by women of child-bearing age has escalated in the last decade, the number of infants being admitted to special-care nurseries for complications caused by their intra-

uterine exposure to alcohol and other drugs has increased in parallel (Chasnoff, 1989a). It is also important to consider that drug-exposed infants often go unrecognized and are discharged from the newborn nursery to homes where they are at increased risk for a complex of medical and social problems.

The popularity of drugs is not limited to any particular ethnic category or socio-economic class. White middle-class women are as likely as Black or poor women to abuse drugs during pregnancy, according to one recent study (Chasnoff, 1989b). A preliminary study in Pinellas County, Florida demonstrated that cocaine and marijuana use during pregnancy were almost randomly distributed across racial and socio-economic lines. A urine drug screen was positive, most commonly for marijuana or cocaine, in 16.3 percent of 380 women from public clinics and 13.1 percent of 335 women in the care of private obstetricians (Chasnoff, 1990). Multiple drug abuse is also common in women; in one survey, multisubstance abuse in drug-dependent mothers occurred in 30 percent of cocaine users, 45 percent of methadone users, and 66 percent of heroin users (Noble, 1988).

HEALTH AND NUTRITION EFFECTS OF DRUG USE

Delineating the effects of individual drugs is problematic for many reasons. Drugs cross the placenta and can cause adverse effects on the developing fetus. Street drugs may be diluted with other drugs or substances of unknown toxicity and maternal use of multiple drugs, alcohol and cigarettes is common. The effects of drugs may be compounded by factors such as poor nutrition, exposure to infectious diseases, lack of medical care, and inadequate child-rearing practices.

The basic tenets of maternal-fetal physiology and pharmacology hold for all drugs and are worthy of review. The following information is a summary of Dattel's overview (1990). Drugs, particularly, illicit drugs, tend to be of low molecular weight, passing freely from the maternal compartment through the placental and into the fetal compartment. The pharmacokinetics of all drugs are altered during pregnancy, and these alterations can have either protective or detrimental effects for mother, fetus, or both. In general, there is reduced maternal concentration of drug late in gestation due to an increase in maternal blood volume (and therefore the volume of distribution), and an increase in total clearance from increased renal (but not hepatic) perfusion. Additionally, the feto-placental unit is a part of this increase in total volume, and therefore, a reduction in maternal drug concentration reflects, in part, fetal exposure.

Placental transfer of low molecular weight substances is primarily by simple diffusion. In late gestation, transport increases due to both physical and chemical parameters such as blood flow and acid-base changes in fetal circulation. Most drugs (such as antibiotics, barbiturates, ethanol, meperidine, and local anesthetics) cross the placenta within minutes. Drug concentrations in the fetus are usually 50 to 100 percent of maternal levels, and for some substances, fetal concentrations are higher. The rules of pharmacology hold for the fetus as well, and transfer from fetal to maternal compartments also exists.

Prenatal Exposure to Drugs

The following is a brief summary of the effects of prenatal exposure to illicit drugs. A brief discussion of the adverse effects of individual illicit drugs on pregnancy and the neonate as

well as on the infant and lactation is in the Appendix. For a more complete description of the known effects of drug and alcohol exposure on the fetus, see the recent review and bibliography by Chasnoff, in *Pediatric Clinics of North America,* December 1989, and the entire volume 562 of the *Annals of the New York Academy of Sciences.*

The effects of drugs on the developing fetus follow basic principles of embryology. As major organogenesis occurs from three to ten weeks after conception, major malformation or spontaneous abortion will be found with fetal drug exposure at this time. Drugs account for 4 to 5 percent of fetal malformations (Bleger, 1970). An increased spontaneous abortion rate has been reported with cocaine abuse (Chasnoff et al., 1985), but many other substances including heroin have not been shown to have major teratogenic effects from first trimester exposure (Rayburn et al., 1986). Other physiological effects including intrauterine growth retardation (IUGR) will occur with exposure after ten weeks. The fetal sympathetic system (α and β) is present in ileum, carotid artery, and aortic arch sinuses from early in the second trimester, and has been shown to be responsive to catecholamines (Rayburn et al., 1986). Theoretically, this may explain some of the effects of abused stimulants. In addition, prolonged exposure to barbiturates, opiates, or alcohol stimulates the glucuronyl transferase mechanism of the fetal liver and allows increased conjugation of circulating bilirubin that may be an adaptation for neonatal life. Finally, all substances can potentially have neonatal effects, such as withdrawal and permanent neurobehavioral alterations. No documentation is available on the effects of prenatal drug exposure on infant or child growth patterns.

Two thirds of American women breast-feed at hospital discharge (Hoegerman et al., 1990). Because of the prevalence of drug abuse in our society, some women nurse despite continued drug ingestion. Drugs of abuse that are contraindicated during breast-feeding include amphetamines, cocaine, heroin, marijuana, nicotine, and phencyclidine (American Academy of Pediatrics, Committee on Drugs, 1989; Chancy, 1988; and Chasnoff, 1987b).

Nutritional Effects of Drug Use

There are few data on the nutritional status of pregnant drug users (Institute of Medicine, 1990). There are a few studies done for marijuana and cocaine use, but other factors, such as cigarette smoking, alcohol consumption, and other drug abuse were not controlled for in all the studies and may confound some of the reported adverse effects. Abel (1971) reported that marijuana stimulates the appetite, but other studies provide conflicting results regarding pregnant women's dietary intake and nutritional status. In a case control study, marijuana users consumed significantly more calories and protein and gained slightly more weight during the pregnancy than controls (O'Connell and Fried, 1984). Another study reported that women who used marijuana weighed slightly less before pregnancy and gained significantly less weight during the pregnancy (Zuckerman et al., 1989). A third study found no consistent relationships between weight gain and the frequency of marijuana use during pregnancy (Linn et al., 1983). As a vasoconstrictor, cocaine may lead to fetal hypoxia (Woods et al., 1987) and reduce the nutrient supply to the fetus. Inadequate maternal diet may result from cocaine since it acts as an appetite suppressant (Cregler and Mark, 1986; Gawin and Ellinwood, 1988 and Resnick et al., 1977) and thereby contribute to fetal growth retardation. In two studies, pregnant women who used cocaine weighed significantly less before pregnancy, had lower hematocrit levels, and

gained slightly less weight during pregnancy (Frank et al., 1988 and Zuckerman et al., 1989).

INTERVENTIONS AND HEALTH CARE

Nutritional Supplementation

The use of specific nutrients to modify the withdrawal process or to stimulate recovery in the addict has not been well studied to date (Mohs, et al., 1990). With specialized supplements with high levels of several vitamins, minerals, D-phenylalanine, L-phenylalanine, L-trypotophan, and L-glutamine one group of investigators reports benefits in increasing compliance and in reducing hunger for drugs during withdrawal (Blum et al., 1988a and Blum et al., 1988b). The use of nutritional support in the treatment of drug abuse has been suggested, such as nutritional changes during the withdrawal or detoxification stage of drug cessation treatment and for renutrition to enhance withdrawal from alcohol and cocaine (Blum et al., 1988a and Blum et al., 1988b). In these two studies, dietary supplementation (specifically tyrosine and tryptophane) helped restore brain chemicals modified by cocaine or alcohol.

Treatment Programs for Women

The National Institute on Drug Abuse established its Program for Women's Concerns in 1974. In 1976 a national conference on women's issues was held focusing on the identification of the treatment needs of women and the types of programs that would have a positive impact on female drug abusers. Public Law 94-371 was passed in 1976 granting priority consideration for the funding of women's treatment and prevention programs (Beschner and Thompson, 1981).

A surprisingly small number of programs address special needs for women. Across the United States, in one study, 35 programs were identified as offering special services for women in the areas of health, child care, vocational counseling, and employment (Stevens et al., 1989). Of these programs, 25 were further investigated, in which a total of 547 women were involved in treatment. Seven of these 25 programs were residential drug-free programs serving only women. These programs serving both men and women numbered three with a total of 42 women being serviced. The remainder of the 547 women were involved in women-only outpatient, men and women outpatient, and men and women outpatient methadone treatment. Furthermore, Reed, Beschner and Mondanaro (1982) indicate that women have traditionally been underserved and are at a disadvantage in most treatment programs as they are viewed more negatively than addicted men and their special biological and social needs may be ignored and misunderstood.

It is difficult to get an accurate picture of female drug abusers' needs as treatment statistics are a primary source for identifying problem areas. Women seek out and enter treatment programs less often than men (Gutierres, Jonathan and Rhoades, 1981); reasons include social expectations and pressures, lack of adequate treatment facilities for women, and lack of facilities for the children of women in treatment. Oftentimes, women are required to place their children in state foster homes so that they can enter a residential treatment facility (Stevens et al., 1989).

In order to address the special needs of the female drug abuser adjustments have been made within programs such as Amity, Inc. The programmatic changes at Amity have had significantly affected the treatment outcomes for the women (Stevens et al., 1989). In particular, the female/male client ratio, the presence of child care, the regularity of women's groups, and the availability of source material relating to a variety of women's issues have been cited by the clients themselves as significant factors increasing their success in treatment.

SUMMARY

Drug abuse is not solely a minority and an inner-city problem; all socio-economic and demographic strata have been affected. The mother's use of drugs, poor prenatal care, poor maternal nutrition and health and a poor home environment put infants and children at risk. The number of women who abuse drugs during pregnancy is unknown, but as many as 375,000 infants may be affected annually (National Institute of Drug Abuse, 1989). Although some substances appear to result in less damage than others, it is impossible to differentiate individual effects because of the common use of multiple substances. The potential for synergy among substances with similar physiologic effects is an additional factor. The effectiveness of intervention and societal support has been demonstrated by a variety of investigators (Bromwich, 1977; Hayden, 1985; and Harel et al., 1985; Stevens, et al., 1989).

There are few data on the nutritional status of pregnant drug users, nor is it well known what effects drug exposure may have on specific nutrients. The immediate health and nutritional perinatal effects of maternal substance abuse are often obvious, but the long-term effects on infants' and children's health, growth and development are as yet poorly understood.

There is a growing concern over the scarcity of treatment programs which address special needs for drug-abusive women. Women have been found to enter treatment programs less frequently than men, for a variety of reasons. Significant factors enhancing the effectiveness of such programs include the presence of child care, the regularity of women's groups, and the availability of source material on various women's issues.

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Adverse Effects of Illicit Drugs

Cocaine

Pregnancy and the Neonate. Cocaine causes a 10-fold increased rate of hemorrhage or placental abruption, a 23 to 38 percent rate of spontaneous abortion, and increased rates of premature labor, precipitous delivery, fetal distress, and meconium staining. Cocaine-exposed infants are more often intrauterine growth retarded, with head circumferences small and out of proportion to other body measurements. Cocaine-induced vasospasm may be responsible for the increased rate of genitourinary, cardiac, and central nervous system (CNS) anomalies reported in these infants. The same mechanism is postulated for perinatal cerebral infarcts, atresia of the bowel, and necrotizing enterocolitis reported in full-term cocaine-exposed infants (Chasnoff, 1988 and Dixon, 1989). Dixon reports that more than one third of cocaine-exposed neonates had structural abnormalities of the brain on ultrasound or computed tomographic scan (Dixon, 1989).

Neonates exposed to cocaine in utero may appear normal or may show various neurobehavioral abnormalities. These include increased muscle tone, tremors, persistence of primitive reflexes, frequent startling, deficient movement patterns, poor state control, irregular sleeping patterns, poor feeding, and impairment in visual processing. Some show poor social interaction, with gaze aversion and difficulty in being consoled (Chasnoff, 1988; Dixon, 1989 and Lewis et al., 1989). Cocaine-exposed infants can go through what appears to be late-onset withdrawal at 2 to 8 weeks of age. They become irritable, hypertonic, slightly febrile, sleep poorly, and do not tolerate change (Dixon, 1989).

Infants and Toddlers. Information is beginning to emerge from follow-up studies on older infants and toddlers who were exposed to cocaine in utero. Dixon reports that three fourths of the children exposed to cocaine or methamphetamine have developmental quotients of less than 100 during the first year, with the greatest delays in fine motor and visual co-ordination (Dixon, 1989). Extreme hyperactivity may cause caretakers and physicians to consider treatment with stimulants, even for very young children. Infants who appeared normal as neonates have later developed severe neurologic deficits including hemiparesis and parkinsonian dystonia (Dixon, 1989). The risk of sudden infant death (SIDS) in infants exposed to cocaine or opiates in utero appears to be 2 to 20 fold higher than baseline rates (Bauchner et al., 1988 and Ward, 1989). It has not been determined if this is due only to the effects of the drugs or also to environmental factors in addicted households, such as an increased risk of smothering or 'lying over' the infant when parents are intoxicated or 'high'.

Older cocaine-exposed infants show abnormal muscle tone, intentional tremors, and tremors at rest through the first year of life. Many have persistent irritability, asymmetric muscle tone, fisting past the age of 6 months, increased extensor tone with arching, difficulty bringing arms to midline, difficulty sitting balanced, and limited interaction with people and objects (Lewis et al., 1989). A group of cocaine-exposed infants raised in adoptive homes from birth also show neurobehavioral deficits, indicating as Dixon postulates, significant structural central nervous system damage has occurred in utero. (Bays, 1990 - personal communication with I.J. Chasnoff and S.J. Budden). A 1987 study indicates that abnormalities in T-cell function can persist through the first year of life in

infants exposed to intravenous heroin, cocaine, or methamphetamines in utero. This suggests that drugs have a toxic effect on the developing immune system (Culver et al., 1987).

Methamphetamine

Pregnancy and the Neonate. Methamphetamine enhances presynaptic release of norepinephrine and pharmacologically is very similar in its effect to cocaine. Methamphetamine is metabolized to amphetamine and can be detected in adult urine for up to 48 hours after its use. Amphetamine is generally used via IV administration and is part of a polydrug abuse syndrome often coupled with alcohol. In the 1950s, oral amphetamine was prescribed to reduce maternal weight gain in pregnancy with no reported fetal effects (Dixon, 1989). However, experience since the 1970s has indicated both IUGR and neurobehavioral changes in the fetus (Little et al., 1988). In humans and animals, amphetamine raises both systolic and diastolic blood pressures. Pulse pressure is also increased due to β_1 adrenergic receptor effect. The response of the human uterus is generally an increase in tone, probably through greater β_1 than β_2 stimulation. Prenatal exposure to methamphetamine produces perinatal effects similar to those observed with cocaine including increased incidence of pre-term labor, hemorrhage, placental abruption, fetal distress, and intrauterine growth retardation with disproportionate decrease in head circumference. Although methamphetamine-exposed infants may exhibit symptoms typical of opiate withdrawal, some full-term neonates are excessively sleepy, rarely cry, and feed so poorly as to require gavage feeding. They may appear clinically to be blind or deaf, even through tests of visual and auditory function are normal. A third of full-term methamphetamine-exposed neonates in a series of 74 had evidence of brain hemorrhage, cavitation, or infarction (Dixon, 1989).

Marijuana (Tetrahydrocannabinol)

Pregnancy and the Neonate. Approximately 6 million women of child-bearing age are current users of marijuana (Adams et al., 1989). Cannabis is an ancient drug made from the flower tops of hemp plants. Most commonly it is called hashish or marijuana. It is the 1-Δ^9-tetrahydrocannabinol (THC) isomer that is believed responsible for the psychologic effects of marijuana. Other than the effects on the CNS, the physiologic effects are primarily on the cardiovascular system. With a few exceptions, most studies indicate that mothers who report smoking marijuana more than once a month during pregnancy have a significantly higher risk of delivering a pre-term, low birth-weight or small-for-gestational-age infant. Marijuana appears to have an effect in addition to the adverse effect of cigarette smoking alone. Hatch and Bracken report that the effect of marijuana use was stronger than that of smoking 20 or more cigarettes daily (Hatch et al., 1986).

Fried has written a comprehensive review on the consequences of prenatal exposure to marijuana (Fried, 1989). He indicates that 20 percent of pregnant women in his Ottawa study group used marijuana before pregnancy, and 10 percent used it while pregnant. Women who were heavy marijuana users reduced their use of alcohol and cigarettes more than their use of marijuana during pregnancy and returned to pre-pregnancy levels of marijuana use within one year after delivery. Prenatal marijuana exposure had no apparent

effect on miscarriage rates, meconium staining, Apgar scores, birth complications, or overt physical anomalies. Several minor anomalies were found in the newborns including marked epicanthal folds and ocular hypertelorism. Other studies have found that women smoking marijuana through pregnancy are five times more likely to have infants with anomalies typical of the fetal alcohol syndrome (Fried, 1989).

Infants and Toddlers. Fried and his colleagues followed marijuana-exposed infants to age 24 months 715 (Fried, 1989). They observed neurobehavioral signs in neonates consistent with those seen in withdrawal from opiates but milder in degree. Signs included increased fine tremors, startling, and a
trend toward increased irritability. Also seen were effects consistent with retardation of maturation of the visual system: poor habituation to visual stimuli, lack of optical blink, and strabismus. Follow-up at 12 to 14 months of age failed to reveal adverse effects attributable to marijuana exposure in any area tested. Fried speculates that either the adverse effects of marijuana are transitory, or are subtle and harder to detect in older children. A recent study identifies a 10 fold increased risk of acute nonlymphoblastic leukemia in children whose mothers used marijuana just before and during pregnancy (Robison et al., 1989).

Opiates

Pregnancy and the Neonate. As many as 10,000 children per year are born to women using opiates (Hans, 1989). The acute effects on the neonate of intrauterine exposure to opiates were described almost 100 years ago (Wilson, 1989). Opiate-exposed infants have a higher incidence of fetal distress, intrauterine growth-retardation, and persistently small head circumference even when prenatal care has been adequate (Hans, 1989). For days to months after birth infants may exhibit signs of opiate withdrawal. These include tremors, hypertonia, poor sleeping and feeding patterns, impaired interactive abilities, diarrhea, fever, tachypnea, tachycardia, high-pitched crying, sweating, and seizures. Enrolling the opiate-abusing mother in a methadone maintenance program during pregnancy is recommended to lessen the adverse effects on the fetus of unpredictable doses and withdrawal periods characteristic of illicit opiate use. After birth, however, withdrawal from methadone can be longer and more severe than withdrawal from heroin (Chasnoff, 1988 and Wilson, 1989).

Infants and Toddlers. Most studies have followed opiate-exposed children only to age two or five years; the longest follow-up study is on children age ten years. The adverse effects of environmental conditions are difficult to factor out. For example, children not exposed to opiates in utero but raised by addicted fathers or in a drug milieu have a high incidence of slow mental development, conduct disorders, and school problems (Wilson, 1989). Almost every study indicates that opiate-exposed infants and children show deficits when compared with drug-free controls not including compounding variables. Observed deficits include impaired motor performance and cognition, poor organization and perception, lack of concentration, hyperactivity, impulsiveness, aggressiveness, lack of inhibition, and poor visual-motor coordination. A poor environment can worsen the damage of opiates, whereas enrolling pregnant addicts in a methadone maintenance program tied to comprehensive medical and psychiatric services may have an ameliorating effect (Hans, 1989; Kaltenbach et al., 1989; and Wilson, 1989).

Phencyclidine (PCP)

Abuse of PCP, an inexpensive hallucinogen and mood-altering drug, has been variable over time and geographic locality. No urines were positive for PCP in arrestees from Birmingham, Dallas, or Portland, Oregon, in the last quarter of 1988, whereas in Washington, D.C., 27 percent of female arrestees and 29 percent of male arrestees tested positive for PCP (U.S. Department of Justice, 1989). The few published reports on effects of prenatal exposure to PCP describe neonates with nonspecific abnormal neurobehavioral findings rather than frank withdrawal. Infants are characterized as jittery, hyperreactive, irritable, hypertonic, and difficult to console, with coarse flapping movements of the hands and wide-eyed blank stares. No long-term studies on large numbers of PCP-exposed infants have been reported (Fico et al., 1989).

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Technical Paper 12

Homeless Mothers and Children: What is the Evidence for Nutritional Risk?

Technical Paper #12 prepared for the U.S. Department of Agriculture, Food and Nutrition Service, under Cooperative Agreement #58-3198-1-005, "Review of Nutritional Risk Criteria for the WIC Program", by Awal Dad Khan, MS, Research Specialist, Department of Family and Community Medicine, College of Medicine, University of Arizona, Tucson, AZ 85724. Mr. Khan is a doctoral candidate in the Program in Nutritional Sciences, University of Arizona.

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INTRODUCTION

Homelessness is not a new issue, but the plight of the homeless in the U.S. and some other industrialized countries has captured public attention in the last several years as the number and nature of the homeless have changed. Homelessness is variably defined as a housing problem, an employment problem, a problem brought on by deinstitutionalization of mentally ill persons, a symptom of the breakdown of family traditions and/or of an inadequate social welfare system, or any combination of these factors (Rossi and Wright, 1987). The common theme in the recent literature is a move away from the traditional view of the middle-aged male derelict, drunk and down on his luck, as the stereotype of the homeless individual (Shulsinger, 1990). Today's homeless population contains a sizeable fraction of women and children (Lam, 1987; Rossi, 1989). Indeed women, children and youth now comprise approximately one-third of the homeless population in America (Wright, 1988; Breakey, 1989; Bassuk and Rosenberg, 1990).

The purpose of this paper is to review: a) definitions, prevalence and trends in homelessness particularly in relation to women and children, and b) the available evidence associating homelessness with nutritional risk.

DEFINITIONS

Several agencies have provided definitions which may be used in counting the number of homeless persons. The National Governors' Association defined the homeless person as "an undomiciled person who is unable to secure permanent and stable housing without any special assistance", and the U.S. General Accounting Office defines the homeless as those persons who lack resources and community ties necessary to provide their own adequate shelter (American Academy of Pediatrics, 1988). The legislative definition of a homeless individual, from the Hunger Prevention Act of 1988 (PL 100-435), is a woman, infant or child who lacks a fixed and regular nighttime residence or whose primary nighttime residence is: a supervised publicly or privately operated shelter (including a welfare hotel, a congregate shelter, or a shelter for victims of domestic violence) designated to provide temporary living accommodation; an institution that provides temporary residence for individuals intended to be institutionalized; a temporary accommodation in the residence of another individual; or a public or private place not designed for, or ordinarily used as, a sleeping accommodation for human beings (Federal Register, Vol. 54, No. 239, page 51294, 1989). These definitions apply as well to homeless families, and cover a broad spectrum of housing or shelter conditions, making it relatively difficult, in the context of the WIC Program, to determine an otherwise eligible applicant's "homeless" status.

There are debates over whether simple lack of shelter is by itself sufficient to render an individual homeless; advocacy groups in Great Britain have introduced the concept of "houselessness" distinguished from "homelessness" (Bailey, 1977). While houselessness implies absence of physical residence, the term homelessness is reserved for a condition of more generalized deprivation (Bachrach, 1987). Several writers agree that for a person to be classified as homeless, his or her lack of physical residence must occur under conditions of social isolation or disaffiliation (Larew, 1980; Segal and Baumohl, 1985). Complicating the definitional dilemma is the fact that the homeless include, besides single adults and families, adolescents on their own, and increasing numbers of the "new homeless", economically displaced individuals who have lost their jobs, exhausted their resources, and

recently entered into the ranks of the homeless and consider the condition to be temporary. Regardless of the definitional framework, it is clear that the homeless population is heterogeneous and includes many subgroups.

PREVALENCE AND TRENDS IN HOMELESSNESS

Estimates of the number of people who lacked access to conventional dwelling or residence in 1987 ranged from 350,000 to more than three million (Wright and Lam, 1987). Table 1 shows estimates of the size of the national homeless population for various years between 1982 and 1988 derived from several sources. The four estimates reported by USHUD were based on the following approaches: a) shelter population and local area street counts, b) extrapolation from estimates in 60 metropolitan areas obtained in more than 500 local interviews, c) extrapolation of estimates from a national sample of 125 shelter operators, and d) extrapolation from highest published estimates.

Table 1

National Estimates of the Homeless Population*

Source & Year	Estimate
Hombs and Snyder (1982)	2,200,000
U.S. Department of Housing and Urban Development	
1. 1984a	192,000
2. 1984b	254,000
3. 1984c	353,000
4. 1984d	586,000
Freeman and Hall (1986)	287,000
Tucker (1987)	700,000
Alliance Housing Council (1988)	1.3-2.0 million

* Adapted from Institute of Medicine (1988).

Whatever the absolute numbers, there is consensus that the number of homeless has increased appreciably in recent years (Rossi et al., 1987). The U.S. Conference of Mayors in 1987 estimated that U.S. cities had experienced on average a 20 percent annual increase over several years in the number of people needing shelter, and that one-fourth of this need could not be met with existing emergency shelters (U.S. Conference of Mayors,

1987). An average annual increment of 20 percent would suggest that more than three million people will be homeless at the end of the year 1991.

People in all regions of the country and of every age and varied ethnic background are to be found among the homeless of America, as are pregnant women and mothers with infants and young children (Bassuk, 1986) and unhoused and inadequately served chronically mentally ill individuals (Bachrach, 1984). Surveys conducted in major cities across the U.S. in the 1980s (Los Angeles, San Francisco, Portland, Dallas, Phoenix, St. Louis, Chicago, and New York City) have reported homeless populations that are younger, better educated, and disproportionately nonwhite compared to previous generations of homeless (Robertson et al., 1985; Ropers, 1985; Ropers and Robertson, 1985). Stephens et al. (1991) provide a demographic profile of the homeless, using HRSA-HCH (Health Care for the Homeless Program of the Health Resources Administration) calendar year 1989 data as: 47 percent non-Hispanic White, 36 percent Black, 11 percent Hispanic, 16 percent of other ethnic groups; 21 percent children, 20 percent women ages 15-44 years, of which 12 percent were pregnant and 27 percent of those pregnant were under age 20.

The fastest growing segment of the homeless population are families with children, with a majority headed by single women (Bassuk, 1984; Bassuk et al., 1986; U.S. Congress, 1986). In a fairly recent survey of 29 American cities, it was estimated that 28 percent of the homeless were families with children (U.S. Conference of Mayors, 1987). In King County, Washington, a study of 82 homeless families found half headed by a single woman with at least one child under 18 years; slightly over one-third consisted of two adults with children (Miller and Lin, 1988). In New York City in the mid-1980s, homeless families comprised 35 to 50 percent of the homeless population (Bassuk et al., 1986; New York Coalition for the Homeless, 1986) and in Los Angeles, approximately 40 percent (U.S. Conference of Mayors, 1984, 1986). The Children's Defense Fund (1987) reported increases in two-parent homeless families in Detroit, Denver, Louisville, St. Paul, Charleston, and other cities. In Philadelphia, Trenton, and Yonkers, about half of the homeless population were members of families with children. The national increase in family homelessness is primarily a result of two converging economic trends; rapidly decreasing availability of affordable housing in major cities and increase in the number of families with below-poverty level incomes. There are more two-parent homeless families in the western part of the country than in the east (Bassuk et al., 1986; McChesney, 1986; Dumpson, 1987), perhaps reflecting migration of recently unemployed heads of households with their families westward in search of better employment opportunities. Two-parent homeless families are also more prevalent in rural than in urban areas. The rural unemployment rate has exceeded that in cities in recent years, a reversal of historical trends (Institute of Medicine, 1988).

RISK FACTORS FOR HOMELESSNESS AMONG MOTHERS AND THEIR CHILDREN

Just as there are many routes to homelessness, there are a variety of risk factors including poverty, unemployment and underemployment, domestic violence, natural disaster, residence in an area in which inexpensive housing is not available, lack of security, gentrification of neighborhoods, and changes in public welfare programs (Austerberry and Watson, 1983; Harrington, 1984; Bassuk et al., 1986; Bachrach, 1987; Ropers and Boyers, 1987; Bassuk and Rosenberg, 1988; Edelman and Minhaly, 1989; Patterson and

Roderick, 1990; Goering et al., 1990; Sclar, 1990; Wood et al., 1990). Lack of education does not appear to be a powerful risk factor (Ropers and Boyer, 1987).

Wood et al. (1990) studied 196 homeless with 194 housed poor families in Los Angeles, to gain an understanding of the nature of events that precipitate family homelessness. Three-fourths of both groups had incomes below the poverty level, and both groups spent an average of two-thirds of their income on housing. Characteristics which were reported at higher rates in the homeless as compared to the housed poor families included spouse abuse (35 vs 16 percent), drug use (43 vs 30 percent) and child abuse (28 vs 10 percent). Adults in the homeless group were more likely than those in the housed poor group to have grown up in households with alcohol- or drug-abusing parents, and to report histories of childhood abuse and/or foster care in their own childhoods. Homeless families tended to report long histories of housing instability, with an average of 3.7 moves in the year prior to becoming homeless. The distribution of precipitating events was quite different for two-parent compared to single-parent homeless families, with economic and housing problems cited as the precipitating event by 72 percent of two-parent families and only 48 percent of single-parent families, while family problems were cited more often by single-parent families (31 percent compared to 16 percent of two-parent families). The prominent role of major family disruptions or child abuse during their own childhoods as characteristics of currently homeless adults in families is supported by a study of 82 families sheltered in Massachusetts family shelters in 1985 (Bassuk and Rubin, 1987). This study also found long histories of residential instability, with an average of four residential moves in the year prior to the current homelessness. The picture which emerges is one in which family breakdown in the context of inadequate economic resources and unavailability of inexpensive housing pushes families who have been precariously housed into homelessness (Hopper and Hamberg, 1984).

There is evidence from several studies that pregnant and recently delivered women, particularly young women, are disproportionately represented among the homeless. Weitzman (1989) compared 704 homeless families on public assistance programs in New York City with 524 families on public assistance who had housing; the homeless group was considerably younger than the housed (44 percent under age 25, compared to 17 percent among housed individuals, and only 4 percent over age 40 compared to 26 percent in this age group among the housed families). Thirty-five percent of the homeless women were pregnant at the time of the interview, compared with 6 percent of the housed women; 26 percent of the homeless had given birth in the previous year compared to 11 percent of the housed. The author did not attempt to age-control the rates reported, but even if the primary effect is one of age distribution the fact remains that pregnant women are represented at an enhanced rate among the homeless. Having had at least one child before age 18 was significantly associated with homelessness in this study, but current family size was not. Weitzman also inquired as to the frequency of women voluntarily or involuntarily "giving up" children during difficult periods; two percent of the homeless women in that study indicated that a minor child had left their households during the previous year.

HEALTH AND NUTRITIONAL CONSEQUENCES OF HOMELESSNESS: WOMEN

That homelessness is associated with health-related problems is intuitively obvious but poorly documented. Studies have suggested that homeless persons with limited financial resources are at risk for protein-energy malnutrition (Buff et al., 1980). However, the

degree of risk and the prevalence of malnutrition remain to be quantified. Very little information is available on the health and nutritional status of homeless mothers or even of homeless women, in spite of the fact that women constitute approximately 18 to 20 percent of the homeless population in various databases (City of Boston Emergency Shelter Commission, 1983; Roth et al., 1985; Brickner, 1985). Gelberg and Linn (1989) surveyed the literature on homeless adults and found 23 papers which provided relevant data on physical health status, but none which reported findings separated by age or sex. Winkleby (1990) compared risk factors for ill health between homeless and housed adults over 19 years who attended a free meal program in northern California, finding that the homeless poor had significantly less health insurance coverage, less utilization of preventive health services, and higher rates of cigarette smoking than the non-homeless sample. Ropers and Boyer (1987) surveyed 269 homeless men and women in 1983-84 in Los Angeles County; 45 percent indicated that their health had worsened after they no longer had a place to live. Twenty-five percent of 145 mothers living in Philadelphia shelters reported having chronic illnesses (including hypertension, diabetes, renal disease and cardiovascular disorders) which interfered with their daily activities (Parker et al., 1991).

Chavkin et al. (1987) were the first to report higher rates of low birth weight infants born to homeless women, comparing mothers living in the welfare hotels in New York City with the city's housed poor (18 vs 8.5 percent). This risk paralleled the rates of inadequate prenatal care (39.7 vs 14.5 percent). Homeless mothers were also more likely than the housed poor to have experienced the death of a child within the first year of life; the infant mortality rate in the shelters has been reported at 24.9 per 1000 (Chavkin et al., 1987; Weinreb, 1988).

In Britain, the London Food Commission Maternity Alliance Shelter (1988) reported that a high proportion of homeless women were late starters and poor attenders at prenatal clinics, and had higher than expected antenatal complications including anemia and infections. Another London study compared pregnancy outcomes of homeless and housed poor women, and found elevated rates of both low birth weight and pre-term delivery among homeless women (Paterson and Roderick, 1990). Contrary to a previous report by Drennan and Stearn (1986) which reported that a quarter of infants of homeless mothers in the same population were of low birth weight, Paterson and Roderick (1990) found no difference in low birth weight rates between homeless and housed poor women. Both studies reported higher rates for complications of pregnancy among the homeless, and higher rates of positive history for stillbirth and neonatal death. These British study populations were of mixed ethnicity, including Asian as well as European women.

Inadequate diet has been mentioned as a factor affecting the health of the homeless (Institute of Medicine, 1988; Nichols et al., 1986), but information on diets and nutritional status of homeless women is very limited. Wright et al. (1987) reported a higher rate of nutritional deficiencies among homeless women than men. Bunston and Breton (1990) studied eating patterns and problems of 84 women living in hostels and drop-in centers in Toronto and found that the average number of servings in each of the four food groups was below the Canadian Food Guide recommendations. About ninety-six percent of the women ate less than the recommended number of servings of three food groups. Only 13.1 percent of the women reported either the recommended number of servings in three or four food groups. Analysis of predisposing factors to dietary inadequacy pointed to lack of money as the overwhelming determinant, and to an important role of social agencies in

providing an adequate diet. Nearly two-thirds of the women ate their lunches provided either by hostels or drop-in centers. Further, a large majority (86 percent) reported eating "junk food" (undefined), accounting for 28 percent of their total food intake. Inadequate dietary intake did not correlate in this study with age, education, or length of time homeless. However, it was related to the quantity of food consumed, the amount of "junk food" consumed, the percentage of economic subsidy by social agencies, and the use of hotels. The authors speculated that the longer a woman had been homeless, the more likely she would know where food was available which in turn would influence her diet and nutritional adequacy.

HEALTH AND NUTRITIONAL CONSEQUENCES OF HOMELESSNESS: CHILDREN

There is considerable evidence of increased risk of infectious, traumatic, and emotional insults to the homeless child, and evidence of nutritional risk is beginning to be documented.

Homeless children have high rates of delayed immunization. In a hospital-based study of 265 children under five years of age living in welfare hotels in New York City, 27 percent were either unimmunized or significantly delayed in their immunization schedule, compared with eight percent of a control group of poor but domiciled children (Alperstein et al., 1988; Alperstein and Arnstein, 1988). Compared to the general U.S. pediatric population, homeless children of all ages in Seattle were twice as likely to lack measles immunization (21 vs 9 percent) and to never have had a tuberculosis skin test (48 vs 27 percent) (Miller and Lin, 1988). Homeless children appeared to overutilize emergency department services, underutilize preventive health services and have far fewer dental visits than the general pediatric population (Miller and Lin, 1988). In another New York City study, Acker et al. (1987) compared 98 children under age 12 who were living in welfare hotels with 253 domiciled poor children from the Bellevue Hospital pediatric outpatient clinics. In this group the rate of immunization delay was even higher, at 49 percent. Wright (1990) reported that 2.2 percent of homeless children who received care during the first year of the National Health Care for the Homeless Program were diagnosed as anemic which is twice the rate reported by Miller and Lin (1988) for children in sheltered homeless families.

Risk of infectious disease, particularly those whose spread is exacerbated by unsanitary conditions, is a concern in crowded temporary shelters (Bass et al., 1990). Diseases for which increased risk is present in this environment include conjunctivitis, ringworm, and diarrhea (Philadelphia Citizens for Children and Youth, 1988). One group of investigators has reported that homeless children are twice as likely to suffer from various chronic physical disorders as compared with ambulatory children in the general population. The authors also reported more illness such as anemia, malnutrition and refractory asthma among homeless children (Wright and Weber-Burdin, 1987). Wright (1990) compared health problems of homeless children seen in the National Health Care for the Homeless Program with those of children included in the National Ambulatory Medical Care survey. About seven percent of homeless children had scabies and lice infestation compared with 0.2 percent of the National Ambulatory Medical Care survey children, upper respiratory infections were about twice as common, and skin disorders four times as common. Similar patterns have been observed among homeless teenagers as well (Yates et al., 1988).

Studies which have included measures of growth and/or anemia show general lack of evidence of chronic malnutrition but, increasingly, indications of current nutritional inadequacy. Since available data are limited to children living in shelters of various types, we may conclude that the situation of unsheltered children may be worse. The New York study of Alperstein et al. (1988) showed no differences in prevalence of height-for-age below the fifth percentile among welfare hotel residents vs poor domiciled children (7.7 vs 7.5 percent) and only a small difference in weight-for-age below the fifth percentile (8.7 vs 6.4 percent). A study of 213 sheltered children under the age of five living in emergency shelters in Boston found the prevalence of low (below the fifth percentile) height-for-age, weight-for-age, and weight-for-height to be 7.7, 7.6 and 2.9 percent respectively (Lewis and Meyers, 1989). Miller and Lin (1988) studied the health status of 158 sheltered homeless children aged 17 days to 17 years in Seattle; less than ten percent were short for age and 35 percent had weights-for-height over the 95th percentile. In the New York City study of Acker et al. (1987), there was a significant difference between welfare hotel resident children and poor but domiciled children in the distributions of height- and weight-for age, with decreased linear growth evident in the homeless children. For children aged six months to two years, the homeless were significantly at higher risk for iron deficiency as measured by elevated free erythrocyte protoporphyrin. Specific studies on dietary intake or nutritional deficiency disorders among homeless persons, whether child or adult, are relatively rare. It has been noted that the menu used by the city of New York in its shelters for homeless persons, supplied at least one-third of the daily requirement for all known nutrients (Winiek, 1985).

In addition to high risk for physical health problems, homeless children suffer from increased rates of emotional and developmental problems. Bassuk et al. (1986, 1987, 1988) have described developmental, emotional and learning problems among homeless children residing in family shelters in Massachusetts. They report 47 percent of preschoolers manifested serious developmental delay in at least one of the areas they tested (language skills, gross motor skills, fine motor coordination, and personal/social development). One-third manifested developmental lags in more than two areas. Among school-aged children, depression, anxiety, and learning difficulties were commonly reported. Forty-three percent had already failed to complete a grade and 25 percent were in special classes. A failing rate of about 30 percent in school among children in Los Angeles homeless families was reported (Wood, 1989). Other studies have documented erratic school attendance as a risk among homeless school-aged children (Institute of Medicine, 1988). A study of school-aged sheltered homeless children in New York City found an average of two or more years behind age-appropriate grade levels in reading and mathematics, frequent discipline problems, and frequent reports of having experienced and/or witnessed physical violence (Kronenfeld et al., 1980), and several Massachusetts studies have supported the conclusion that significant developmental lags, depression, anxiety and learning difficulties are to be found at high rates among homeless children (Bassuk et al., 1986; Bassuk, 1986; Bass et al., 1990).

The generalized nature of risks to health and well-being of inadequate shelter, forced mobility, and food insecurity has been discussed by several authors (Drennan and Stearn, 1986; Morton, 1990). Sclar (1990) has referred to the "children's game of musical chairs, with n players and n-1 chairs", in which the winner is the last one among the continuing players who is favored by luck as the others drop out. He suggests that infant mortality, AIDS, drug abuse and other social ills linked to homelessness will not be amenable to

interventions which treat each as discrete entities, but rather that a comprehensive approach including decent housing and transportation, good nutrition, and rational health planning is required.

UTILIZATION OF WIC AND OTHER SUPPLEMENTAL PROGRAMS BY HOMELESS MOTHERS AND CHILDREN

Limited evidence suggests that rates of participation in WIC may be lower among the homeless than among other eligible women and children. Although homeless families in some studies include an overrepresentation of families who have been on welfare entitlement programs for an extended period of time (Bassuk et al., 1986), WIC participation may be more difficult because of lack of secure personal storage for refrigerated items and lack of access to cooking facilities (Philadelphia Citizens for Children and Youth, 1988). Weitzman (1989) found that 44 percent of sheltered homeless pregnant and new mothers indicated that they received WIC benefits, compared to 60 percent in a housed control group. Offering nutrition education and health care referrals within the setting of shelters might help increase participation. The participant families primarily relied on canned foods, dry cereals and other non-perishable food items for nourishment (Gallagher, 1986). The author suggested that lack of refrigeration, cooking facilities or storage facilities is particularly critical for mothers with infants and young children.

SUMMARY

Families, and therefore women and children, constitute the fastest growing segment of the homeless population nationwide. Interpersonal and domestic violence is more likely to play a precipitating role in the advent of homelessness for families headed by single women, whereas unemployment and economic problems are more likely to be precipitating events for two-parent families. While there is a dearth of comprehensive and well controlled studies of the health and nutritional status of the homeless, the evidence which is accumulating indicates a clear increase in risk of health problems, low utilization of preventive health services including prenatal care and immunization, and developmental and learning problems in children of all ages.

There is also evidence of increased risk of malnutrition among homeless children and increased risk of low birth weight for homeless women, but this is highly correlated with the general, and attendant lack of prenatal care among pregnant homeless women. Inadequate dietary patterns relate directly to issues of poverty, access to health care services and other public assistance programs such as Food Stamps and AFDC. What little information is available points to qualitative nutritional inadequacy, and/or to nutritional adequacy over the short term based on assistance provided by temporary shelters. All of the available literature on health and nutritional status is based on sheltered homeless families; we may assume that the nutritional and health situation is less good for those not housed even temporarily. However, in the context of the WIC Program, it is difficult even to define who may be homeless because the scope of the legislative definition is so broad.

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Technical Paper 13

Appropriate Dietary Assessment Methodology for the WIC Clinic Setting

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INTRODUCTION

Nutritional assessment is the interpretation of information gathered through dietary, biochemical, anthropometric, clinical and/or sociodemographic studies. Such information is then used to describe the health status of individuals or populations as influenced by their intake and utilization of nutrients. Nutritional assessment can take one of several formats: surveys, surveillance, or screening (Gibson, 1990). *Nutrition surveys* provide baseline data to determine a population's overall nutritional status; the format is a cross-sectional survey which may include a comprehensive or limited set of data collected. In general, surveys are likely to underestimate the prevalence of acute and severe undernutrition because they are least likely to include in the sampling frame the most at-risk segments of the population, unless specifically designed to oversample these subgroups. They are generally well able to describe population subgroups at risk for chronic and moderate malnutrition. The data from nutrition surveys are often used to allocate resources to at-risk populations and to design policies to improve the overall nutritional status of populations. *Nutrition surveillance* provides continuous monitoring of the nutritional status of specific population groups. Surveillance systems provide for periodic measurement either of the same individuals or like individuals over an extended period of time. Surveillance systems can provide data to identify causes of malnutrition and to evaluate change in nutritional status over time. When surveillance occurs for the individual it is called *monitoring.* Finally, *nutrition screening* is used to identify malnourished or at-risk individuals who require intervention. The data collected for an individual are compared to predetermined risk levels to determine eligibility for intervention.

For all of these systems, the triangulation of dietary, clinical, anthropometric, laboratory and sociodemographic data is essential to derive a full picture of nutritional status or risk. Dietary assessment, in all cases, provides information which relates to the first stage of a potential nutritional deficiency (Gibson, 1990).

The Special Supplemental Food Program for Women, Infants, and Children (WIC) utilizes nutritional assessment in the format of *screening* to determine eligibility for intervention, with the goal of targeting scarce resources to achieve the maximum benefit in terms of improvement of nutrition-related outcomes for the population. In some states, the data derived from nutrition screening is then further utilized as part of a *surveillance* system to track program-related changes in nutritional status. This technical paper focuses on the dietary assessment methodologies appropriate in the clinic setting typical of the WIC program, specifically for low-income women and children.

The data derived from the dietary assessment 1) identify the first stage of a potential nutritional deficiency; and 2) provide the necessary information to direct effective intervention: nutrition care planning, nutrition education and monitoring participant progress and behavioral change.

DIETARY METHODOLOGIES FOR ASSESSING INDIVIDUAL FOOD AND/OR NUTRIENT INTAKES

Marr (1971) categorized the various methods for collecting dietary data from individuals into three types: (1) present intake by recording; (2) past intakes recalled; and (3) short-cut

methods for epidemiological studies. Edozien and Bazzarre (1978) modified Marr's classification into two categories: (1) dietary records and (2) dietary recalls.

Dietary Records

Dietary or food records may be collected by weighing actual foods consumed, recording estimated food quantities, or recording qualitative data by means of menu or food frequency formats.

Weighed food records require that the subject, parents or caretaker weigh and record all food and beverages consumed for a specified period of time. Samples of foods consumed may be saved for nutrient analysis, or nutrient composition may be determined using food composition data. While precise, this method is expensive and requires an extraordinary level of subject time and participation; it may also bias intake away from usual behavior. *Estimated food records* are essentially the same format but portion sizes are estimated using household measures. Again, the subject must be literate, cooperative, and accurate in estimating quantities, and able to take the time required to produce accurate data. *Food frequency records* or *menu records* are similar except that quantitative data are omitted and only the number of portions consumed is recorded.

Dietary records are appropriate for some clinical settings in which the individual client/patient is to be seen several times, a record of actual behavior is desirable, and the records is to be used as the basis for education and behavior change. This is typically the situation for a patient who must adopt a prescribed diet such as for diabetes, hypertension, etc. For screening, the purpose of which is to identify high-risk individuals, records are neither practical nor desirable, because of the record-keeping burden placed on the subject and the error produced by inaccuracies in estimating portion sizes. Several studies have documented that intakes of individuals during a period of record-keeping do not represent their usual level of food consumption reported throughout the year (Kim et al., 1984; Burke and Pao, 1976).

Dietary Recalls

Dietary recalls may be used to quantify the usual amounts and types of foods consumed over a long period of time (dietary history); to quantify the type and amount of foods consumed during a specific time in the past (typically the previous 24 hours); or to estimate the frequency of use for specified foods over a long period of time (food frequency). The first of these uses, the dietary history, was developed first by Burke (1947); it is a comprehensive assessment for the individual, very labor- and time-intensive and not generally appropriate for screening purposes. The second two types of recalls, 24-hour recall and recall of usual food intake by food frequency, are of more relevance in the WIC clinic setting, and will be considered in more detail.

The twenty-four hour recall of food intake is a very commonly used tool in dietary assessment. It may be completed by the participant using a paper-and-pencil questionnaire or at an interview conducted by a trained professional or paraprofessional investigator. Typically the previous 24 hours, or a 24-hour period from "waking up yesterday until waking up today" is covered. The foods recalled are quantified by household measures. The interview format presumably provides more complete information than a self-

administered questionnaire; however, it has the potential disadvantage of bias if the subject is influenced to idealize the food intake for the sake of perceived expectations of the interviewer. The self-administered questionnaire, however, is less costly to administer. In either case, no special knowledge is required of the subject and the time involved is minimal. The major limitations of this method are a) variation in subjects' memory of foods consumed and ability to accurately quantify portion sizes; and b) the fact that any 24-hour period is unlikely to be representative of usual, long-term intake. The greatest value of the 24-hour recall is in its use as a nutrition education tool in helping the client to identify inadequate or inappropriate food intakes.

Food frequency questionnaires, or recall of frequency of use for specified foods, imposes the least respondent burden of all dietary assessment methods in common use (Gibson, 1990 and Edozien and Bazzarre, 1978). A self-completed questionnaire or short interview by a trained interviewer is utilized to derive data on the frequency of usual consumption of specified food items. The questionnaire or interview includes a list of foods and a set of frequency-of-use response categories. The list of foods may be extensive or focused on particular foods of interest. The food frequency questionnaire can identify food patterns associated with inadequate intakes of nutrients, and can provide semiquantitative data if subjects are asked to quantify usual portion sizes. The major advantages of this approach are a) ability to estimate long-term, usual intake patterns; and b) low cost and ease of administration. Disadvantages include variability in subjects' ability to remember and estimate foods and frequency of consumption and the need to devise specifically adapted questionnaires for local dietary patterns and/or nutrients of interest.

Other Innovations

A variety of innovative methods have been attempted to improve the accuracy and efficiency of dietary assessment in populations; a particularly relevant one is the telephone survey. Krantzler et al. (1982) used telephone interviews to collect 24-hour recalls and seven-day diet records; they found that telephone-administered interviews were quite feasible and have promise for other applications. Photography and videotape provide other possible avenues for recording food intake, but are expensive and not practical for clinic settings (Bird and Elwood, 1983).

CONVERTING DIETARY INTAKE DATA TO ESTIMATES OF NUTRIENT INTAKE

Quantitative food consumption data obtained by various methods can be converted into energy and nutrient intake estimates through the use of food composition data available through published tables and in the form of various computerized databases. The most commonly used and comprehensive food composition database in the U.S. is USDA Handbook No. 8, continuously updated and based on the U.S. Nutrient Data Bank maintained by the Nutrient Data Research Group of the USDA's Consumer Nutrition Center (Rizek et al., 1981). The micro-computer, now commonplace in the health care setting, has made conversion of food intake data to nutrient intake estimates possible on-site in clinic settings using nutrient database software. A variety of programs is available, at costs of ten to several thousand dollars (Snetselaar, 1989). Decisions as to which program to purchase should involve not only cost but comprehensiveness of the database, ease of use, and recency of update.

Food composition data, like other information, is subject to random and systematic error, most of which is unknown (NRC, 1986). The most important consideration for dietary assessment purposes is that food composition data represent the best available estimate of nutrient actually present in representative sample of the food item; they do not take into account bioavailability.

In the busy clinic setting, constraints of staff time, subject time, and cost affect the use of dietary intake data. If calculations are done by hand or even by microcomputer, it may make sense to limit the effort to nutrients in which there is particular interest (e.g., iron, calcium, protein). The simplest qualitative interpretation involves no nutrient conversion at all, but rather comparison of food frequency or 24-hour recall data with recommended amounts of food groups. This is a simple and practical approach to identifying possible areas of dietary weakness, but has severe limitations if the diet has limited variety, relies on unconventional foods or has special culturally-based components which do not translate easily into food groupings.

SOURCES OF ERROR IN DIETARY ASSESSMENT

Both random and systematic errors occur related to the ability of data collected to represent actual intake. Gibson (1990) identifies several major sources of error in the collection and recording of dietary assessment data. They are:

Respondent bias. Respondents may over- or under-estimate basic facts such as income and age; likewise, they may consciously or unconsciously over-report the consumption of acceptable or desirable foods (e.g., vegetables and fruits), or under-report "bad" foods (e.g., fast foods, snack foods) and the use of tobacco and alcohol. Salvini et al. (1989) found that observed differences in mean daily food intake quantities between a dietary record and amounts calculated from a dietary questionnaire suggested that the questionnaire tended to over-represent socially desirable foods. On the other hand, recording methods may result in changed behavior as well (Sempos, 1985).

Respondent inaccuracy in estimating food quantities may result in the so-called "flat slope syndrome," i.e., a tendency to overestimate low intakes and underestimate high ones. In general, accuracy in portion size estimation can be enhanced through appropriate training of interviewers and use of food models and other aids to portion size estimation.

Respondent memory limitation may result in the unintentional omission or addition of foods to the data. Memory can be distorted because of incongruence of certain aspects of the actual event with the subject's image of the situation, including the suppression of those aspects of the eating event which are less socially acceptable (Edozien and Bazzarre, 1978). Krall et al. (1988) have reviewed factors which affect ability to accurately recall and report past dietary intake; intelligence, age, mood, attention, salience of the information, and frequency of exposure to a stimulus. These have been demonstrated to impact memory. Appropriate design of food frequency questionnaires can be explored to format natural eating and purchasing habits in order to aid memory.

Interviewer bias may occur if different interviewers probe for information to different degrees, intentionally omit certain inquiries, and/or record responses incorrectly. Several studies have been conducted of interviewer error and bias in large multi-center studies

which standardized training and interview protocols. Schectman et al. (1990) has analyzed data from 24-hour recalls and food frequencies from more than 10,000 subjects under conditions of good interviewer training and quality control, and found unavoidable error. Beaton (1979) examined the National Heart, Lung and Blood Institute Lipid Research Clinics' dietary data with regard to sources of variance, and found no significant interviewer effects or training effects in 24-hour recalls, indicating that interviewer-associated error can be minimized by training and precise interview protocols. The Bogolusa Heart Study data focus on 24-hour recalls from children and thus are of particular interest. In comparing data from the same 24-hour period among different interviewers, Frank et al. (1984) found only 76% agreement on recorded food names, and 87% agreement on the assignment of food identification codes. The differences were greatest in quantifying liquids, meats and sweets. Training and quality control procedures are the best approaches to minimizing interviewer error and bias.

Coding and computation error may occur when portion sizes are converted from household measures into weights, or when food items are incorrectly coded. Gross errors can be reduced if standard rules are established to deal with incomplete or ambiguous food descriptions (Anderson, 1986) and large comprehensive databases are used (Dwyer and Suitor, 1984). Again, training, quality control procedures, and human or computer-based checks on logic can minimize errors.

VALIDITY AND RELIABILITY ISSUES IN DIETARY ASSESSMENT TOOLS

There are a variety of complex methodological problems in the choice of appropriate dietary instruments and in interpretation of the data derived from them. There is no ideal measure for assessing the validity of any method of determining dietary intake (Willett et al., 1983). However, there are issues which can be considered in selecting or developing tools and in the use of the data which they produce.

Reliability or reproducibility is the degree to which a method gives similar results when used repeatedly in the same situation. Conventionally, reliability is measured using a test-retest design followed by an assessment of the extent of agreement between estimates of nutrient intake collected on the two separate occasions. Reliability is not expected to be high between 24-hour recalls, since actual intake is not exceedingly similar from day to day for most individuals; it is expected to be better with food frequency instruments.

Validity is the degree to which an assessment tool measures that which it is intended to measure. Since establishment of absolute validity is usually not possible, validity is usually assessed by evaluating the "test" methodology against another "reference" method whose reliability and ability to measure similar parameters over the same time period is better understood. Validity over the short-run (ability of a 24-hour recall to measure foods actually consumed, for example) is minimally affected by age except for children and the elderly, not appreciably affected by gender, and not subject to major training effects in most circumstances unless recall or recording days are consecutive. It is affected by the types of error detailed in the preceding section, relating to interviewer and respondent biases. Validity in the long run (i.e., ability of an instrument to accurately reflect usual intake) is affected primarily by intra-subject variation in intake. No literature is available on how socioeconomic status affects the validity and reliability of the dietary assessment tools, especially the 24-hour recall or food frequency. This issue may be important enough

for future research to determine the relationship between socioeconomic status and the reliability and validity of dietary assessment methodologies.

INTRA-SUBJECT VARIABILITY IN FOOD INTAKE: IMPLICATIONS FOR ASSESSMENT OF DIETARY INTAKE

Within-person variation in food intake which is characteristic of day-to-day variation and day-of-the-week, seasonal, and other periodic variation (such as that generated by periodicity of income) is the single major factor affecting the ability to infer usual intake from dietary assessment data (NAS, 1986; Anderson, 1986; Beaton et al., 1979). The ratio of intra-individual to inter-individual variance in dietary intake data collected on a 24-hour recall basis has a very wide range depending on the nutrient(s) of interest and the population being studied, but is uniformly large enough to make a single 24-hour recall a very inaccurate method for assessing individual intake.

Day-of-the-week effects have been studied in several databases. Beaton (1979) found that the weekend-weekday variation was different for men and women; van Staveren et al. (1982) observed that both men and women had lower intakes of dietary fiber on weekends than weekdays. Gibson et al. (1985) noted no weekend effect on intake of cholesterol, vitamin A or sodium. Weekend effects on alcohol consumption are well documented. Most investigators recommend proportionately representing all days of the week, or at least weekend/weekday variation, in population studies to avoid day-of-the-week biases (Beaton et al., 1979; Sempos et al., 1985). For the individual, it is evident that a single 24-hour recall can represent only weekday or weekend patterns. *Seasonal effects on food intake* may show considerable regional variation and may be significant for vitamin A, vitamin C, iron and fat (van Staveren et al., 1986; Gibson, 1990). *Day-to-day variation in intake* depends not only on individual characteristics but on the distribution of the particular nutrient of interest in foods; nutrients which are widely distributed in foods such as protein, energy, and iron will show less day-to-day variation than those which are concentrated in relatively few foods such as vitamin A and cholesterol.

In general, food frequency methods for assessment of dietary intake provide more stable estimates of usual intake than does one or even several 24-recalls. The number of 24-hour recalls which is required to achieve stable estimates of nutrient intakes is too large to be practical in a clinic setting.

FACTORS TO CONSIDER IN SELECTING THE APPROPRIATE DIETARY TOOLS

There is no ideal method for assessing food or nutrient intakes. The choice of method depends primarily on the purpose of the investigation, and is constrained by available personnel, financial and other resources, and respondent burden. All methods contain inherent potential for substantial random and systematic error. However, the fact remains that self-reported information on diet is the best available source of information on the earliest antecedent of nutritional deficiency, namely dietary inadequacy.

When the purpose of dietary assessment is screening, estimating usual intakes and deficits in usual intakes is the primary goal. In that case, food frequency instruments provide the most stable and valid estimates of the available and practical approaches. In devising or adapting a specific questionnaire to a local population, such characteristics as literacy,

interest, motivation, proportion of meals eaten away from home, eating environment, should be considered as well as resources available for analysis of the data. The dietary assessment instrument and its protocol must be standardized and pretested for validity and reliability. More detailed information is usually associated with higher cost, respondent burden, and needs for interviewer training. When the purpose of the dietary assessment is nutrition education, the food frequency questionnaire may also be used but there are potential advantages to other methods including the 24-hour or -day recall, including focusing of the attention of the client on actual current behavior.

INTERPRETATION OF NUTRIENT INTAKE DATA IN TERMS OF NUTRITIONAL RISK

Dietary assessment data are primarily used to obtain presumptive evidence of dietary inadequacies or excesses in individuals. Dietary data alone are not sufficient to infer nutritional inadequacy on an individual basis (Christakis, 1973). At best, given information on the distribution of intakes in a population and on the distribution of requirements for the nutrient in the same population, statistical risk of deficiency can be inferred. Beaton (1972) was the first to use a probability assessment approach to predict the prevalence of inadequate iron intakes in a population. Similar applications have been made for assessments of the prevalence of inadequate intakes of protein, vitamin A, vitamin C, thiamin, riboflavin and calcium (Anderson et al., 1981; NRC, 1986). On an individual level, this approach yields an estimate of probability that a given level of nutrient intake does not meet the individual's requirement. In the absence of information on that individual's actual requirement, inference cannot be made about the severity of deficiency.

Comparison of estimated nutrient intakes to the Recommended Dietary Allowances is a usual method for estimating risk of inadequacy. It should be realized that failure to meet the RDA for any given nutrient is not an indication of deficiency. The RDA for energy is set at an average requirement level. For an individual the best indication of adequacy of intake is weight maintenance (for an adult) or growth rate or weight gain at the expected rate (for children and pregnant women). For other nutrients, RDAs are set at variable levels substantially higher than average requirements. As intake drops below the RDA, risk of inadequacy rises. There is no rational basis for any particular cutoff point in terms of percent of the RDA for intake, but a general rule is that the more conservative (lower) the cutoff the higher the risk of inadequacy; also, the greater the number of nutrients which are scored low relative to the RDA, the greater the risk of inadequacy in dietary intake.

These limitations should not be interpreted to mean that dietary assessment has no uses. As a food-based source of information, assessment of dietary intake can provide a powerful tool for nutrition education. In addition, when used in concert with other indicators for risk of nutritional inadequacy such as anthropometry, clinical and sociodemographic risk factors and laboratory measures of nutritional status, dietary information can provide confirmatory evidence for dietary etiology of nutritional problems.

SUMMARY

In the clinic setting, nutrition screening usually includes dietary intake assessment as well as biochemical, clinical and sociodemographic assessments to identify individuals at risk of nutrient inadequacies or excesses, or who are poorly nourished. When data are being collected to determine eligibility for WIC, dietary assessment tools should identify the first stage of risk for deficiency; therefore, they should provide as accurate as possible estimates of usual food intake. When dietary data are collected for other purposes, such as nutrition education, selection of an appropriate tool may be more influenced by other considerations such as immediacy or currency of information reflected. No dietary assessment tool can provide absolute indications of adequate or inadequate nutritional status. This is true of the 24-hour recall and the food frequency methodologies that are frequently used in the WIC clinic setting. These factors directly affect the validity and reliability of dietary assessment methodologies. The intra-subject or within-person variation in food intake is the single major factor affecting the ability to infer usual intake from dietary assessment data. No current research literature is available on the impact of socioeconomic status on the validity or reliability of dietary assessment tools.

Understanding the limitations of the various tools and the constraints imposed by available resources and respondent characteristics, the local program can make an appropriate choice of instrument for dietary assessment. The current data indicate that the semiquantitative food frequency instruments, even when self-administered, provide the most stable and valid estimates of the available and practical approaches for the clinic setting. The food frequency may require modification for children and other cultures since the food frequency generally reflects American adult food groupings. The value of data derived from any tool will depend on well-trained staff, on well implemented quality control procedures, on nutrient conversion procedures and databases which are up to date and accurate, and on reasoned interpretation.

The choice of or the development of an appropriate, valid and reliable tool that can be utilized to quickly collect and quantify the individual's dietary patterns and hunger status is crucial. Such an instrument would reflect dietary patterns and risk factors which are more sensitive indicators of dietary inadequacy than are indices of nutritional status that reflect longer time periods.

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Technical Paper 14

Pica and Lead Exposure in Infants and Children: Health and Nutritional Risk Implications

Technical Paper #14 prepared for the U.S. Department of Agriculture, Food and Nutrition Service, under Cooperative Agreement #58-3198-1-005, "Review of Nutritional Risk Criteria for the WIC Program", by Awal Dad Khan, MS, Research Specialist, Department of Family and Community Medicine, University of Arizona, Tucson, Arizona 85724. Mr. Khan is a doctoral candidate in the Nutritional Sciences Program, University of Arizona.

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INTRODUCTION

Pica, the repeated ingestion of non-food substances, has frequently been implicated in the etiology of lead toxicity in young children. Lead poisoning in preschool children due to chronic ingestion of paint chips peeling from the walls of older inner city housing has been well documented in the United States (Needleman, 1988a; Landrigan et al., 1987). In many cases, the ingestion of lead paint chips (plumbophagia) is simply the result of normal mouthing behavior of young children. Lead paint is common in older housing, it is often highly concentrated and it can be easily accessible to children both as paint chips and as dust. Soil and dust that contain lead are also important sources of lead exposure. Children appear to obtain lead from dust and soil as a result of their normal exploratory behavior (Roels et al., 1976; Sayre et al., 1974; Barltrop, 1966). Paint chips containing lead are considered to be the major source for children with extremely high blood lead levels, while dust is a source for the more common, moderately elevated lead doses (Landrigan et al., 1987). Increasingly, lead poisoning has been reported when families move into cities as "urban homesteaders" and the children are inadvertently exposed to chips or dust from lead based paint as houses are rehabilitated. Clusters of lead based paint poisoning have resulted from demographic shifts within cities, when families with young children have moved into neighborhoods with deteriorating older housing. Urban-dwelling children can also be exposed to chips or dust from lead based paint produced during the de-leading of exterior painted steel structures, such as bridges and expressways (Landrigan et al., 1982).

PREVALENCE OF PICA IN CHILDREN

Children, particularly those in the 15-30 month age group who are in an active stage of oral development and engage in mouthing ingest a wide variety of foreign materials (Maravilla and Berk, 1978; Barltrop, 1966). Pica is commonly seen in young children and is not regarded as pathological (McLoughlin, 1987). Pica is thought to be underreported because its practices may be assumed to be a normal habit among young children. A National Research Council report in the mid-1970s estimated that approximately 50 percent of children between the age of one and three years indulged in pica (NAS/NRC, 1976). Danford (1982), in a review on pica and nutrition, summarized chronological prevalence data on pica practices, generated through cross-sectional studies, cross checking that at least through the 1970s the practice was still widespread with estimates of prevalence ranging from 10 to 55 percent (see Table 1).

Table 1

Prevalence of Pica

References (Year)	Region	Population (Percent)	Prevalence
Cooper (1957)	Baltimore, Maryland	Black, > 6 months	27
		White, > 6 months	17
Millican et al. (1962)	Washington D.C.	Black, 1-6 years	32
		White, 1-6 years	10
Lourie et al. (1963)	Washington D.C.	Children, low income	55
		Children, high income	30
Barltrop (1966)	Boston (interview)	Children, 1-6 years	15
	Boston (mail)	Children, 1-6 years	20
Bruhn & Pangborn (1971)	California	Spanish-American children	32
Vermeer & Frate (1979)	Mississippi	Children, 1-4 years	16

Adapted from Danford, 1982.

Robischon (1971) reported the prevalence of pica as high as 50 percent in black children and as 34 percent in white children, based on mothers' reports of children ingesting selected substances, including matches, paper and ashes. She also observed behavior in a sample of 90 black well children aged between 19 to 24 months and reported a pica prevalence of 37 percent. Vermeer and Frate (1979) reported geophagia prevalence of 16 percent among children in a southern rural black community. Geophagia was evident among children of both sexes aged approximately from one through four years; at older ages this practice was associated almost exclusively with females.

The most authoritative estimate is that between three and 12 million American children have neurotoxic levels of lead in their blood (Mushak and Chrochetti, 1988; APHA, 1991). Among preschoolers, more than three million suffer from lead poisoning from paint. The Centers for Disease Control estimates that 12 million children under seven years of age are at high risk. The Department of Health and Human Services has mentioned that as many as three to four million children under six years of age are at risk, of whom 17 percent may have blood levels high enough to cause mental and behavioral problems and other adverse health effects (APHA, 1991).

RISK OF LEAD POISONING

Pica has been demonstrated to increase an individual's lead burden in an environment with lead contamination (Annest et al., 1983; Charney et al., 1980; McKusker, 1979; Lin-Fu, 1973). A nationwide survey conducted from 1976-1980 reported that children from all geographic areas and socioeconomic groups are at risk of lead poisoning from lead paint (Mahaffey et al., 1982). Nationally, 17 percent of children in urban areas are estimated to

have blood lead levels greater than 15 μg/dl, a level associated with adverse medical effects (Agency for Toxic Substances and Disease Registry, 1988). In Wisconsin, 36,000 children are estimated to be at risk for lead poisoning based on three risk factors, namely, age under seven years, residence in housing built before 1950, and annual family income of below $15,000 (Schirmer et al., 1991).

Older data from the 1960s-1970s show the persistence of the problem. Klein (1974) estimated that five to ten percent of children between one and five years of age had lead poisoning, including more than 30 percent of children with pica. One hundred thirty six children (117 black and 19 white) were reported to the Cleveland Board of Health by a Cleveland, Ohio hospital as cases of lead poisoning during the period 1963-69. The prevalence of lead pica was higher among black children of southern origin than among whites (Chatterjee and Gettman, 1972). Children between 10 months and 12 years of age in New York City screened for lead poisoning had a prevalence of mouthing foreign substances of 34 percent for paper, 12 percent for ashes, and four percent for dirt, while 17 percent were exposed to paint and plaster. Only two variables, age under three at screening and a history of exposure to lead-containing paint or plaster, showed a clear and consistent relationship to elevated blood lead levels (McKusker, 1979).

Lead poisoning has been raised as a problem for children living in temporary shelters in New York City. One recent study reported elevated blood lead levels in 3.8 percent for homeless children compared with 1.7 percent for domiciled children (Alperstein et al., 1988). Although the prevalence of elevated lead blood levels was twice as great in the homeless children as in the domiciled children, the prevalence of 3.8 percent is quite low when compared with the 18.6 percent reported for black children of parents earning less than $6,000 per year in the early 1980s (Mahaffey et al., 1982). However, from the same study, the percentage of children with elevated blood lead levels of parents earning more than $15,000 per year was only 0.7 percent. Twelve percent of 76 children under age of 12 years living in Philadelphia homeless shelters has abnormally high free erythrocyte protoporphyrin levels, of which 11 percent had been diagnosed as having lead poisoning in the previous year (Parker et al., 1991).

Data from the Hispanic Health and Nutrition Examination Survey have been analyzed to compare blood lead levels in Mexican American, Puerto Rican, and Cuban children (Carter-Pokras et al., 1990). Puerto Rican children had the highest mean blood lead levels and percent with elevated blood lead, followed by Mexican American and Cuban children. For both Puerto Rican and Mexican American children, younger age was associated with higher risk of having elevated blood lead levels. Mexican American children whose families had incomes below poverty level had higher mean blood lead levels than Mexican American children from higher income families.

PREDISPOSING FACTORS FOR PICA-ASSOCIATED LEAD TOXICITY

Several predisposing factors for pica have been identified and are summarized in Table 2. These predisposing factors have not yet all been established through case-control studies and several rest on only indirect evidence.

Table 2

Risk Factors for Pica and Lead Poisoning

** Nutritional status
* Poor nutrition/health
* Early feeding problems
* Dietary iron deficiency
* Zinc deficiency
* Calcium deficiency
* Magnesium deficiency
* Metabolic disease
** Sociodemographic
* Age (preschoolers and schoolers)
* Socioeconomic status
* Ethnicity
* Inadequate and older housing
* Poor parental supervision
* Parental education
** Cultural/Psychological
* Positive family history of pica
* Psychosocial and family stress
* Inadequate emotional support
** Non-Nutritional
* Mobility
* Mouthing
* Disrupted oral needs

Lead absorption is higher in children than in adults (USEPA, 1986). Both animal and human studies have shown that deficiencies in iron, calcium and zinc all result in increased gastrointestinal absorption of lead (Mahaffey, 1981; Snowdon and Sanderson, 1974; Snowdon, 1977). Lead toxicity can increase twenty-fold with severe calcium deficiency; as the amount of $CaCl_2$ increases, lead absorption decreases (Zeigler et al., 1978). High-calcium diets will decrease absorption of both zinc and iodine as well as lead. Of particular concern is the interaction of lead with iron deficiency, because the prevalence of iron deficiency in infancy and early childhood is significant (Nathan and Oski, 1981). Iron deficiency even in the absence of anemia, appears to be the single most important predisposing factor for increased absorption of lead (Clark et at., 1988).

HEALTH AND NUTRITIONAL CONSEQUENCES OF PICA AND LEAD EXPOSURE

Pica is associated with malnutrition in children (Gelfand et al., 1975) particularly iron deficiency anemia; there are also controversial reports that iron deficiency may be a cause

of pica during childhood. Geophagia has been associated with low hemoglobin levels, low serum iron, hypokalemia, low serum ascorbic acid, normal serum protein, low albumin, high incidence of respiratory illness, and normal anthropometric measurements including height and weight (Lanzkowsky, 1959; Gutelius et al., 1962; Mohan et at., 1968). Arcasoy et al. (1978) in a study in Turkey found decreased iron and zinc absorption associated with geophagia (clay eating) among Turkish children. The duration of geophagia ranged from four to 15 years. All children but one had severe growth retardation (below the 3rd percentile in height) and seven showed hypogonadism. The authors speculated that inadequate diet and geophagia were the main causes of iron deficiency.

Several case studies support the notion of pica resulting from nutrient deficiencies in young children (Tenenbein, 1990; Pynoos et al., 1978). In two boys under six years of age with Bartter's syndrome, a potassium wasting disease, a craving for potatoes was reported (Pynoos et al., 1978). Karayalcin and Lanzkowsky (1976) reported that a boy who consumed Comet cleanser had zinc deficiency which was responsive to treatment with oral zinc sulfate. A patient with pica for metal had low hair- and serum-zinc levels and exhibited poor growth and appetite. The patient recovered following zinc therapy (Hambidge and Silverman, 1973).

The health consequences of lead exposure have been well documented in the literature. Lead affects virtually every system in the body, and is particularly harmful to the developing brain and nervous system, red blood production, renal and reproductive system, the kidney and the bone marrow (Schirmer et al., 1991; Mahaffey, 1981). Severe lead poisoning may terminate in coma, convulsions or death (Lin-Fu, 1980), and even mild exposures have measurable adverse effect on cognitive development such as decreased intelligence, developmental delays, behavioral disturbances.

Effects on Nutrient Intake and Absorption

The information on dietary patterns and food intake associated with pica during childhood is limited and not recent. Most of the available reports are anecdotal. Gutelius et al. (1962) found that children with pica consumed less meat and milk, fewer vitamin-C rich foods and a smaller variety and quantity of foods than children without pica. Sand eating has been encountered in children with excessive milk intake and refusal of solid food intake (Catzel, 1963). There is a clear need for better and more comprehensive studies to investigate the nutritional consequences of pica and to elucidate the etiology of pica during childhood.

Nutritional factors can influence susceptibility to lead toxicity. Studies in animal models (Quaterman and Morrison, 1975; Snowdon, 1977; Snowdon and Sanderson, 1974) suggest that inadequate intakes of both calcium and iron may potentiate the toxic effects of lead poisoning. Low calcium intake increases lead absorption, because of competition between calcium and lead absorptive intestinal receptors. Johnson and Tenuta (1979) found an inverse correlation between blood lead levels and calcium and milk intakes in children age one to six years. An inverse relationship between blood lead levels and vitamin D intake, serum calcium levels, 25-hydroxyvitamin D (25[OH]D) levels, and 1,25 dihydroxyvitamin D ($1,25[OH]_2D$) levels has also been reported (Sorrell et al., 1977; Rosen et al., 1980). The relationship between iron deficiency and lead absorption was studied in 191 children with elevated erythrocyte protoporphyrin (Yip et al., 1981). Of the children

with elevated blood lead levels ($\geq$30 μg/dl), 30 percent were anemic and 86 percent were iron deficient. In the highest lead risk classification, 43 percent were anemic and all were iron deficient. The existence and severity of iron deficiency or anemia increased with the severity of lead toxicity.

A recent case-control study (Laraque et al., 1990) reported calcium status in relation to blood lead levels and behavior of 64 black children aged 18 to 47 months (36 boys and 28 girls). A pica score, reflecting ingestion of non-food substances, a mouthing habit score, and a general behavioral score were used to estimate various behaviors. Both pica and paint chip scores were significantly higher in cases (blood lead level $\geq$1.45 μmol/L) than controls (blood lead level $<$1.45 μmol/L). Unlike previous studies, the present study did not demonstrate any difference on four calcium intake measures between cases and controls. Likewise calcium intake was not associated with pica scores. A history of pica, especially for paint chips, was the only significant differentiating factor between the groups.

Association with Iron Deficiency

Numerous investigators have reported the prevalence of microcytic anemia among children with lead poisoning (Cohen and Ahrens, 1959; Smith, 1964; Greengard, 1966). However, iron deficiency is also prevalent among children at risk for lead intoxication. Clark et al. (1988) have addressed the question of whether the anemia in children with lead poisoning is caused by iron deficiency, lead poisoning, or an interaction between the two, in a multivariate analysis in which various iron status measures and blood lead levels were used to predict hemoglobin and mean corpuscular volume (MCV) in children admitted to hospital for treatment of lead poisoning. The best models for both hemoglobin and MCV prediction include only age and transferrin saturation; lead level did not add to predictive power. The majority of lead-poisoned children who had adequate iron stores also had normal hemoglobin concentrations and MCV values. Interaction terms were not significant. The authors concluded that the anemia often seen with lead poisoning results from co-occurring iron deficiency, rather than from lead.

Effects on the Fetus

A few studies have suggested an association between maternal lead exposure and retarded fetal growth. Glasgow researchers found that maternal blood lead levels were inversely related to duration of gestation (Moore et al., 1982). Another study found that women with blood lead levels $>$14 μg/dl were four times likely to have preterm babies than those with blood lead level $<$8 μg/dl (McMichael et al., 1986). Needleman (1988b) evaluated the relationship of maternal blood lead to a number of outcomes in 5,000 newborns. Serum lead concentrations were low in the entire sample so that levels of exposure were defined at three levels: low as $\leq$3 μg/dl, moderate as 6 to 7 μg/dl and high as $\geq$10 μg/dl. There was no association between lead levels and birth weight, jaundice or major malformations when the analysis was controlled for race, socioeconomic status, maternal illness and substance abuse. Lead was, however, significantly associated with minor malformations and the risk was dose-dependent.

Effects on Growth in Infancy and Childhood

Schwartz et al. (1986), using data of 2965 children between six months and seven years of age from the National Health and Nutrition Examination Survey (NHANES), found that blood lead levels were a highly significant predictor of height, weight and chest circumference. The relationships persisted when other variables were controlled.

Effects on Cognitive Performance

Higher dropout rates from school, poor performance on ability and attainment tests, increased absenteeism and poor hand/eye coordination have been documented among school children who were exposed to lead in early childhood (Luckardt and Tucker, 1990). Similar findings were found among 855 British school children aged six to nine years (Fulton et al., 1987).

Bellinger et al. (1989) conducted a prospective cohort study to assess the association between early development and low-level prenatal and postnatal lead exposure. The Bayley Scales of Infant Development were administered between six and 24 months. The Mental Development Index declined with increasing concentration of lead in blood. Vulnerability to low-level lead toxicity appeared to be a joint function of socioeconomic status and age at exposure.

Elevated blood lead levels have been correlated with deficits in psychological and classroom performance in children six to seven years of age (Needleman et al., 1979). When reevaluated as young adults, impairment in neurobehavioral function was still found and was significantly related to lead content of teeth shed at the ages of six and seven (Needleman et al., 1990). The authors concluded that childhood lead exposure is associated with deficits in central nervous system functioning in young adulthood. Moreover, a recent meta-analysis of 24 studies of childhood exposure to lead in relation to IQ establishes a strong link between low-dose lead exposure and intellectual deficits in children (Needleman and Gastsobis, 1990). Whether the link is a causal one still remains to be solved. Bellinger et al. (1986) reported that asymptomatic school-age children with high blood lead levels scored lower on the Wechsler IQ scales, on a number of measures of auditory and language function, and on a measure of attention. Teachers who did not know the children's lead levels reported that those with higher lead levels were more destructive, disorganized, hyperactive and impulsive.

METHODS OF SCREENING FOR LEAD EXPOSURE

Available methods for screening for lead exposure or toxicity include determination of blood lead level, erythrocyte protoporphyrin (EP) levels or both. The Centers for Disease Control (CDC) (1985) has suggested yearly or semi-yearly screening for children one to five years of age in high-risk settings or with significant predisposing factors. The CDC also suggests that children under three years of age with normal lead and EP levels be tested every six months; if one test produces an abnormal value, then a repeat test is suggested. The EP determination provides a sensitive and inexpensive screening tool for both increased lead absorption and iron deficiency (Nathan and Oski, 1981). Elevation in EP levels can reflect early iron deficiency before anemia becomes clinically evident (Yip et al., 1983).

Both capillary tubes and filter paper have been used for obtaining screening samples. Capillary tubes are more cumbersome but have the advantage of providing sufficient blood for concomitant lead determination if the EP level is elevated. Filter paper sampling provides ease of collection and transport, but the accuracy of analyses based on this is not yet established. To guide the interpretation of screening results, the CDC developed a series of guidelines for each of the two analytical techniques for determining the EP, namely extraction of protoporphyrin for erythrocytes and subsequent measurement in a fluorimeter and direct fluorimetry of a thin layer of RBCs (hematofluorometer). The extraction method is preferred because of its greater reproducibility, particularly at lower concentrations of erythrocyte protoporphyrin (Landrigan et al., 1987). The guidelines are summarized in Tables 3 and 4.

Table 3

Zinc Protoporphyrin by Hematofluorometer: Risk Classification of Asymptomatic Children for Priority Medical Evaluation

Blood Lead (μg/dl)	Erythrocyte Protoporphyrin (μg/dl)			
	<35	35-74	75-174	>175
Not done	I	+	+	+
<24	I	Ia	Ia	*
25-49	Ib	II	III	III
50-69	$	III	III	IV
>70	$	$	IV	IV

Table 4

Erythrocyte Protoporphyrin by Extraction: Risk Classification of Asymptomatic Children for Priority Medical Evaluation

Blood Lead (μg/dl)	Erythrocyte Protoporphyrin (μg/dl)			
	<35	35-109	110-249	>250
Not done	I	+	+	+
<24	I	Ia	Ia	*
25-49	Ib	II	III	III
50-69	$	III	III	IV
>70	$	$	IV	IV

The risk classifications are defined as:

- \+ Blood lead test needed to estimate risk
- * Erythropoietic protoporphyria. Iron deficiency may cause elevated EP levels up to 300 μg/dl, but this is rare
- $ In practice this combination of results is not generally observed; if it is observed, immediately retest with whole blood
- Ia,b Children with any symptoms compatible with lead toxicity
- II Children younger than 36 months of age
- III Children whose blood lead and EP levels place them in the upper part of a particular class
- IV Children whose siblings are in a higher class

These classifications refer to the interpretation of screening results, but the final diagnosis and disposition rest on a more complete medical and laboratory examination of the child.

CONCLUSIONS

Pica during childhood is a natural form of exploratory behavior which occurs through hand-to- mouth exploration. When it occurs in a lead contaminated environment, it is a potent risk factor for lead poisoning. Childhood lead intoxication remains one of the most important contemporary public health problems in the United States. There are many indications that pica poses health and nutritional risks for infants and children and is associated with malnutrition, socioeconomic status, and living in deteriorated housing. There is evidence that low intake of calcium, zinc and/or iron may potentiate lead toxicity. The apparent irreversibility of neurobehavioral consequences of lead exposure intensifies the need for concern over exposure of infants and children to lead.

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