Psychology

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Aggression

Aggression, in its broadest sense, is behavior, or a disposition, that is forceful, hostile or attacking. It may occur either in retaliation or without provocation.

In narrower definitions that are used in social sciences and behavioral sciences, aggression is a response by an individual that delivers something unpleasant to another person.[1] Some definitions include that the individual must intend to harm another person.[2] Predatory or defensive behavior between members of different species may not be considered aggression in the same sense.

Aggression can take a variety of forms and can be physical or be communicated verbally or non-verbally. Aggression differs from what is commonly called assertiveness, although the terms are often used interchangeably among laypeople, e.g. an aggressive salesperson.[3]

Overview

Two broad categories of aggression are commonly distinguished. One includes affective (emotional) and hostile, reactive, or retaliatory aggression that is a response to provocation, and the other includes instrumental, goal-oriented or predatory, in which aggression is used as a mean to achieve a goal.[4] An example of hostile aggression would be a person who punches someone who insulted him or her. An instrumental form of aggression would be armed robbery. Research on violence from a range of disciplines lend some support to a distinction between affective and predatory aggression. However, some researchers question the usefulness of a hostile vs instrumental distinction in humans, despite its ubiquity in research, because most real-life cases involve mixed motives and interacting causes.

A number of classifications and dimensions of aggression have been suggested. These depend on such things as whether the aggression is verbal or physical; whether or not it involves relational aggression such as covert bullying and social manipulation;[5] whether harm to others is intended or not; whether it is carried out actively or expressed passively; and whether the aggression is aimed directly or indirectly. Classification may also encompass aggression-related emotions (e.g. anger) and mental states (e.g. impulsivity, hostility). Aggression may occur in response to non-social as well as social factors, and can have a close relationship with stress coping style. Aggression may be displayed in order to intimidate.

The operative definition of aggression may be affected by moral or political views. Examples are the axiomatic moral view called the non-aggression principle and the political rules governing the behavior of one country toward another. Likewise in competitive sports, or in the workplace, some forms of aggression may be sanctioned and others not.[6]

Etymology

The term aggression comes from the Latin *aggressio*, meaning attack. The Latin was itself a joining of *ad-* and *gradi-*, which meant step at. The first known use dates back to 1611, in the sense of an unprovoked attack.[7] A psychological sense of "hostile or destructive behavior: dates back to 1912, in an English translation of the writing of Sigmund Freud.[8] Alfred Adler had theorized about an "aggressive drive: in 1908. Child raising experts began to refer to aggression rather than anger from the 1930s.[9]

Ethology

Ethologists study aggression as it relates to the interaction and evolution of animals in natural settings. In such settings aggression can involve bodily contact such as biting, hitting or pushing, but most conflicts are settled by threat displays and intimidating thrusts that cause no physical harm. This form of aggression may include the display of body size, antlers, claws or teeth; stereotyped signals including facial expressions; vocalizations such as bird song; the release of chemicals; and changes in coloration.[10] The term agonistic behaviour is sometimes used to refer to these forms of behavior.

Male elephant seals fighting

Most ethologists believe that aggression confers biological advantages. Aggression may help an animal secure territory, including resources such as food and water. Aggression between males often occurs to secure mating opportunities, and results in selection of the healthier/more vigorous animal. Aggression may also occur for self-protection or to protect offspring. Aggression between groups of animals may also confer advantage; for example, hostile behavior may force a population of animals into a new territory, where the need to adapt to a new environment may lead to an increase in genetic flexibility.[11]

Between species and groups

The most apparent type of interspecific aggression is that observed in the interaction between a predator and its prey. However, according to many researchers, predation is not aggression. A cat does not hiss or arch its back when pursuing a rat, and the active areas in its hypothalamus resemble those that reflect hunger rather than those that reflect aggression.[12] However, others refer to this behavior as predatory aggression, and point out cases that resemble hostile behavior, such as mouse-killing by rats.[13] In aggressive mimicry a predator has the appearance of a harmless organism or object attractive to the prey; when the prey approaches, the predator attacks.

An animal defending against a predator may engage in either "fight or flight" in response to predator attack or threat of attack, depending on its estimate of the predator's strength relative to its own. Alternative defenses include a range of antipredator adaptations, including alarm signals.

Aggression between groups is determined partly by willingness to fight, which depends on a number of factors including numerical advantage, distance from home territories, how often the groups encounter each other, competitive abilities, differences in body size, and whose territory is being invaded. Also, an individual is more likely to become aggressive if other aggressive group members are nearby. One particular phenomenon – the formation of coordinated coalitions that raid neighbouring territories to kill conspecifics – has only been documented in two species in the animal kingdom: 'common' chimpanzees and humans.

Within a group

Aggression between conspecifics in a group typically involves access to resources and breeding opportunities. One of its most common functions is to establish a dominance hierarchy. This occurs in many species by aggressive encounters between contending males when they are first together in a common environment. Usually the more aggessive animals become the more dominant. In test situations, most of the conspecific aggression ceases about 24 hours after the group of animals is brought together. Aggression has been defined from this viewpoint as "behavior which is intended to increase the social dominance of the organism relative to the dominance position of other organisms". Losing confrontations may be called social defeat, and winning or losing is associated with a range of

practical and psychological consequences.

Conflicts between animals occur in many contexts, such as between potential mating partners, between parents and offspring, and between competitors for resources. Group-living animals may dispute over the direction of travel or the allocation of time to joint activities. Various factors limit the escalation of aggression, including communicative displays, conventions and routines. In addition, following aggressive incidents, various forms of conflict resolution have been observed in mammalian species, particularly in gregarious primates. These can mitigate or repair possible adverse consequences, especially for the recipient of aggression who may become vulnerable to attacks by other members of a group. Conciliatory acts vary by species and may involve specific gestures of simply more proximity and interaction between the individuals involved. However, conflicts over food are rarely followed by post conflict reunions, even though they are the most frequent type in foraging primates.

Other questions that have been considered in the study of primate aggression, including in humans, is how aggression affects the organization of a group, what costs are incurred by aggression, and why some primates avoid aggressive behavior.[14] For example, bonobo chimpanzee groups are known for low levels of aggression within a partially matriarchal society. Captive animals including primates may show abnormal levels of social aggression and self-harm that are related to aspects of the physical or social environment; this depends on the species and individual factors such as gender, age and background (e.g. raised wild or captive).

Evolutionary explanations

Like many behaviors, aggression can be examined in terms of its ability to help an animal survive and reproduce, or alternatively to risk survival and reproduction. This cost-benefit analysis can be looked at in terms of evolution. There are profound differences in the extent of acceptance of a biological or evolutionary basis for human aggression, however.

Violence and conflict

Aggression can involve violence that may be adaptive under certain circumstances in terms of natural selection. This is most obviously the case in terms of attacking prey to obtain food, or in anti-predatory defense. It may also be the case in competition between members of the same species or subgroup, if the average reward (e.g. status, access to resources, protection of self or kin) outweighs average costs (e.g. injury, exclusion from the group, death). There are some hypotheses of specific adaptions for violence in humans under certain circumstances, including for homicide, but it is often unclear what behaviors may have been selected for and what may have been a byproduct, as in the case of collective violence.[15][16]

Although aggressive encounters are ubiquitous in the animal kingdom, with often high stakes, most are resolved through posturing, displays and trials of strength. Game theory is used to understand how such behaviors might spread by natural selection within a population, and potentially become 'Evolutionary Stable Strategies'. An initial model of resolution of conflicts is the Hawk-Dove game; others include the Sequential assessment model and the Energetic war of attrition. These try to understand not just one-off encounters but protracted stand-offs, and mainly differ in the criteria by which an individual decides to give up rather than risk loss and harm in physical conflict (such as through estimates of Resource holding potential).[17]

Gender

There are multiple theories that seek to explain findings that males and females of the same species can have differing aggressive behaviors. However the conditions under which women and men differ in aggressiveness are not well understood. In general, sexual dimorphism can be attributed to greater intraspecific competition in one sex, either between rivals for access to mates and/or to be chosen by mates. This may stem from the other gender being constrained by providing greater parental investment, in terms of factors such as gamete production, gestation, lactation, or upbringing of young. Although there is much variation in species generally the more physically

aggressive sex is the male, particularly in mammals. In species where parental care by both sexes is required there tends to be less of a difference. When the female can leave the male to care for the offspring, then females may be the larger and more physically aggressive. Competitiveness despite parental investment has also been observed in some species. A related factor is the rate at which males and females are able to mate again after producing offspring, and the basic principles of sexual selection are also influenced by ecological factors affecting the ways or extent to which one sex can compete for the other. The role of such factors in human evolution is controversial. The pattern of male and female aggression is argued to be consistent with evolved sexually-selected behavioral differences, while alternative or complimentary views emphasize conventional social roles stemming from physical evolved differences. Aggression in women may have evolved to be, on average, less physically dangerous and more covert or indirect.[18] However, there are critiques for using animal behavior to explain human behavior. Especially in the application of evolutionary explanations to contemporary human behavior, including differences between the genders.[19]

In general, much research has suggested that males use more physical aggression than females, while females use more verbal aggression. Children interact with, and are aggressive toward,both same- and other-gender peers. There are more recent findings that show that differences in male and female aggression appear at about two years of age, though the differences in aggression are more consistent in middle-aged children and adolescence.Tremblay, Japel and Pérusse (1999) asserted that physically aggressive behaviors such as kicking, biting and hitting are age-typical expressions of innate and spontaneous reactions to biological drives such as anger, hunger, and affiliation. Girls' relational aggression, meaning non-physical or indirect, tends to increase after age two while physical aggression decreases. There was no significant difference in aggression between males and females before two years of age. A possible explanation for this could be that girls develop language skills more quickly than boys therefore they have better ways of verbalizing their wants and needs. They are more likely to use communication when trying to retrieve a toy with the words "Ask nicely" or "Say please."

Many studies have found differences in the types of aggression used by males and females, at least in children and adolescents. Females between the ages of 10 and 14, around puberty age, show a more extreme rate of relational aggression compared to boys. These findings are true for Western society, but are not true of all cultures. In countries such as Kenya it has been found that young boys and girls have very similar rates of physical aggression. It has been found that girls are more likely than boys to use reactive aggression and then retract, but boys are more likely to increase rather than to retract their aggression after their first reaction. Studies show girls' aggressive tactics included gossip, ostracism, breaking confidences, and criticism of a victim's clothing, appearance, or personality, whereas boys engage in aggression that involves a direct physical and/or verbal assault. This could be due to the fact that girls' frontal lobes develop earlier than boys, allowing them to self-restrain.

One factor that shows insignificant differences between male and female aggression is in sports. In sports, the rate of aggression in both contact and non-contact sports is relatively equal. Since the establishment of Title IX, female sports have increased in competitiveness and importance, which could contribute to the evening of aggression and the "need to win" attitude between both sexes. Among sex differences found in adult sports were that females have a higher scale of indirect hostility while men have a higher scale of assault. Another difference found is that men have up to 20 times higher levels of testosterone than women.

Some studies suggest that romantic involvement in adolescence decreases aggression in males and females, but decreases at a higher rate in females. Females will seem more desirable to their mate if they fit in with society and females that are aggressive do not usually fit well in society, they can often be viewed as antisocial. Female aggression is not considered the norm in society and going against the norm can sometimes prevent one from getting a mate. However, studies have shown that an increasing number of women are getting arrested for domestic violence charges. In many states, women now account for a quarter to a third of all domestic violence arrests, up from less than 10 percent a decade ago. The new statistics reflect a reality documented in research: women are perpetrators as well as victims of family violence. However, another equally possible explanation is a case of improved diagnostics:

it has become more acceptable for men to report female domestic violence to the authorities while at the same time actual female domestic violence has not increased at all. This can be the case when men have become less ashamed of reporting female violence against them, therefore an increasing number of women are arrested, although the actual number of violent women remains the same.

Also, males in competitive sports are often advised by their coaches not to be in intimate relationships based on the premises that they become more docile and less aggressive during an athletic event. The circumstances in which males and females experience aggression are also different. A study showed that social anxiety and stress was positively correlated with aggression in males, meaning as stress and social anxiety increases so does aggression. Furthermore, a male with higher social skills has a lower rate of aggressive behavior than a male with lower social skills. In females, higher rates of aggression were only correlated with higher rates of stress. Other than biological factors that contribute to aggression there are physical factors are well.

Regarding sexual dimorphism, humans fall into an intermediate group with moderate sex differences in body size but relatively large testes. This is a typical pattern of primates where several males and females live together in a group and the male faces an intermediate amount of challenges from other males compared to exclusive polygyny and monogamy but frequent sperm competition.[20]

Evolutionary psychology and sociobiology have also discussed and produced theories for some specific forms of male aggression such as sociobiological theories of rape and theories regarding the Cinderella effect.

Physiology

Brain pathways

Many researchers focus on the brain to explain aggression. Numerous circuits within both neocortical and subcortical structures play a central role in controlling aggressive behavior, depending on the species, and the exact role of pathways may vary depending on the type of trigger or intention.

In mammals, the hypothalamus and periaqueductal gray of the midbrain are critical areas, as shown in studies on cats, rats, and monkeys. These brain areas control the expression of both behavioral and autonomic components of aggression in these species, including vocalization. Electrical stimulation of the hypothalamus causes aggressive behavior and the hypothalamus has receptors that help determine aggression levels based on their interactions with serotonin and vasopressin. These midbrain areas have direct connections with both the brainstem nuclei controlling these functions, and with structures such as the amygdala and prefrontal cortex.

Stimulation of the amygdala results in augmented aggressive behavior in hamsters, while lesions of an evolutionarily homologous area in the lizard greatly reduce competitive drive and aggression (Bauman et al. 2006). In rhesus monkeys, neonatal lesions in the amygdala or hippocampus results in reduced expression of social dominance, related to the regulation of aggression and fear. Several experiments in attack-primed Syrian Golden hamsters, for example, support the claim of circuity within the amygdala being involved in control of aggression. The role of the amygdala is less clear in primates and appears to depend more on situational context, with lesions leading to increases in either social affiliatory or aggressive responses.

The broad area of the cortex known as the prefrontal cortex (PFC) has been implicated in aggression, along with many other functions. such as including inhibition of emotions. Reduced activity of the prefrontal cortex, in particular its medial and orbitofrontal portions, has been associated with violent/antisocial aggression.[21]

The role of the chemicals in the brain, particularly neurotransmitters, in aggression has also been examined. This varies depending on the pathway, the context and other factors such as gender. A deficit in serotonin has been theorized to have a primary role in causing impulsivity and aggression. Nevertheless, low levels of serotonin transmission may explain a vulnerability to impulsiveness, potential aggression, and may have an effect through interactions with other neurochemical systems. These include dopamine systems which are generally associated with

attention and motivation toward rewards, and operate at various levels. Norepinephrine, also known as noradrenaline, may influence aggression responses both directly and indirectly through the hormonal system, the sympathetic nervous system or the central nervous system (including the brain). It appears to have different effects depending on the type of triggering stimulus, for example social isolation/rank versus shock/chemical agitation which appears not to have a linear relationship with aggression. Similarly, GABA, although associated with inhibitory functions at many CNS synapses, sometimes shows a positive correlation with aggression, including when potentiated by alcohol.[22]

The hormonal neuropeptides vasopressin and oxytocin play a key role in complex social behaviours in many mammals such as regulating attachment, social recognition, and aggression. Vasopressin has been implicated in male-typical social behaviors which includes aggression. Oxytocin may have a particular role in regulating female bonds with offspring and mates, including the use of protective aggression. Initial studies in humans suggest some similar effects.

Testosterone

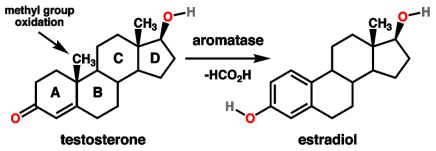
Hormones are chemicals that circulate in the body affecting cells and the nervous system, including the brain. Testosterone is a steroid hormone from the androgen group, which is most linked to the prenatal and postnatal development of the male gender and physique, which in turn has been linked on average to more physical aggression in many species. Testosterone is present to a lesser extent in females, who may be more sensitive to its effects. Animal studies have also indicated a link between incidents of aggression and the individual level of circulating testosterone. However, results in relation to primates, particularly humans, are less clear cut and are at best only suggestive of a positive association in some contexts.[23]

Challenge hypothesis

The challenge hypothesis outlines the dynamic relationship between plasma testosterone levels and aggression in mating contexts in many species. It proposes that testosterone is linked to aggression when it is beneficial for reproduction, such as in mate guarding and preventing the encroachment of intrasexual rivals. The challenge hypothesis predicts that seasonal patterns in testosterone levels in a species are a function of mating system (monogamy versus polygyny), paternal care, and male-male aggression in seasonal breeders. This pattern between testosterone and aggression was first observed in seasonally breeding birds, such as the Song Sparrow, where testosterone levels rise modestly with the onset of the breeding season to support basic reproductive functions. The hypothesis has been subsequently expanded and modified to predict relationships between testosterone and aggression in other species. For example, chimpanzees, which are continuous breeders, show significantly raised testosterone levels and aggressive male-male interactions when receptive and fertile females are present. Currently, no research has specified a relationship between the modified challenge hypothesis and human behavior, or the human nature of concealed ovulation, although some suggest it may apply.

Washington State Song Sparrow

Effects on the nervous system



Testosterone to Estradiol conversion

Another line of research has focused on the proximate effects of circulating testosterone on the nervous system, as mediated by local metabolism within the brain. Testosterone can be metabolized to 17b-estradiol by the enzyme aromatase, or to 5-alpha-dihydrotestosterone (DHT) by 5a-reductase.

Aromatase is highly expressed in regions involved in the regulation of aggressive behavior, such as the amygdala and hypothalamus. In studies using genetic knock-out techniques in inbred mice, male mice that lacked a functional aromatase enzyme displayed a marked reduction in aggression. Long-term treatment with estradiol partially restored aggressive behavior, suggesting that the neural conversion of circulating testosterone to estradiol and its effect on estrogen receptors influences inter-male aggression. In addition, two different estrogen receptors, ERa and ERb, have been identified as having the ability to exert different effects on aggression in mice. However, the effect of estradiol appears to vary depending on the strain of mouse, and in some strains it reduces aggression during long days (16 h of light), while during short days (8 h of light) estradiol rapidly increases aggression.

Another hypothesis is that testosterone influences brain areas that control behavioral reactions. Studies in animal models indicate that aggression is affected by several interconnected cortical and subcortical structures within the so-called social behavior network. A study involving lesions and electrical-chemical stimulation in rodents and cats revealed that such a neural network consists of the medial amygdala, medial hypothalamus and periaqueductal grey (PAG), and it positively modulates reactive aggression. Moreover, a study done in human subjects showed that prefrontal-amygdala connectivity is modulated by endogenous testosterone during social emotional behavior.

In human studies, testosterone-aggression research has also focused on the role of the orbitofrontal cortex (OFC). This brain area is strongly associated with impulse control and self-regulation systems that integrate emotion, motivation, and cognition to guide context-appropriate behavior. Patients with localized lesions to the OFC engage in heightened reactive aggression. Aggressive behavior may be regulated by testosterone via reduced medial OFC engagement following social provocation. When measuring participants' salivary testosterone, higher levels can predict subsequent aggressive behavioral reactions to unfairness faced during a task. Moreover, brain scanning with fMRI shows reduced activity in the medial OFC during such reactions. Such findings may suggest that a specific brain region, the OFC, is a key factor in understanding reactive aggression.

General associations with behavior

Scientists have for a long time been interested in the relationship between testosterone and aggressive behavior. In most species, males are more aggressive than females. Castration of males usually has a pacifying effect on aggressive behavior in males. In humans, males engage in crime and especially violent crime more than females. The involvement in crime usually rises in the early teens to mid teens which happen at the same time as testosterone levels rise. Research on the relationship between testosterone and aggression is difficult since the only reliable measurement of brain testosterone is by a lumbar puncture which is not done for research purposes. Studies therefore have often instead used more unreliable measurements from blood or saliva.[24]

The Handbook of Crime Correlates, a review of crime studies, states most studies support a link between adult criminality and testosterone although the relationship is modest if examined separately for each sex. However, nearly all studies of juvenile delinquency and testosterone are not significant. Most studies have also found testosterone to be associated with behaviors or personality traits linked with criminality such as antisocial behavior and alcoholism. Many studies have also been done on the relationship between more general aggressive behavior/feelings and testosterone. About half the studies have found a relationship and about half no relationship.

Studies of testosterone levels of male athletes before and after a competition revealed that testosterone levels rise shortly before their matches, as if in anticipation of the competition, and are dependent on the outcome of the event:

testosterone levels of winners are high relative to those of losers. No specific response of testosterone levels to competition was observed in female athletes, although a mood difference was noted. In addition, some experiments have failed to find a relationship between testosterone levels and aggression in humans.

The possible correlation between testosterone and aggression could explain the "roid rage" that can result from anabolic steroid use, although an effect of abnormally high levels of steroids does not prove an effect at physiological levels.

Dehydroepiandrosterone

Dehydroepiandrosterone (DHEA) is the most abundant circulating androgen hormone and can be rapidly metabolized within target tissues into potent androgens and estrogens. Gonadal steroids generally regulate aggression during the breeding season, but non-gonadal steroids may regulate aggression during the non-breeding season. Castration of various species in the non-breeding season has no effect on territorial aggression. In several avian studies, circulating DHEA has been found to be elevated in birds during the non-breeding season. These data support the idea that non-breeding birds combine adrenal and/or gonadal DHEA synthesis with neural DHEA metabolism to maintain territorial behavior when gonadal testosterone secretion is low. Similar results have been found in studies involving different strains of rats, mice, and hamsters. DHEA levels also have been studied in humans and may play a role in human aggression. Circulating DHEAS (its sulfated ester) levels rise during adrenarche (~7 years of age) while plasma testosterone levels are relatively low. This implies that aggression in pre-pubertal children with aggressive conduct disorder might be correlated with plasma DHEAS rather than plasma testosterone, suggesting an important link between DHEAS and human aggressive behavior.

Glucocorticoids

Glucocorticoid hormones have an important role in regulating aggressive behavior. In adult rats, acute injections of corticosterone promote aggressive behavior and acute reduction of corticosterone decreases aggression; however, a chronic reduction of corticosterone levels can produce abnormally aggressive behavior. In addition, glucocorticoids affect development of aggression and establishment of social hierarchies. Adult mice with low baseline levels of corticosterone are more likely to become dominant than are mice with high baseline corticosterone levels.

Glucocorticoids are released by the hypothalamic pituitary adrenal (HPA) axis in response to stress, of which cortisol is the most prominent in humans. Results in adults suggest that reduced levels of cortisol, linked to lower fear or a reduced stress response, can be associated with more aggression. However, it may be that proactive aggression is associated with low cortisol levels while reactive aggression may be accompanied by elevated levels. Differences in assessments of cortisol may also explain a diversity of results, particularly in children.

The HPA axis is related to the general fight-or-flight response or acute stress reaction, and the role of catecholamines such as epinephrine, popularly known as adrenaline.

Pheromones

In many animals, aggression can be linked to pheromones released between conspecifics. In mice, major urinary proteins (Mups) have been demonstrated to promote innate aggressive behavior in males. Mups activate olfactory sensory neurons in the vomeronasal organ (VNO), a subsystem of the nose known to detect pheromones via specific sensory receptors, of mice and rats. Pheremones have also been identified in fruit flies, detected by neurons in the antenna, that send a message to the brain eliciting aggression; it has been noted that aggression pheremones have not been identified in humans.[25]

Genetics

In general, differences in a continuous phenotype such as aggression are likely to result from the action of a large number of genes each of small effect, which interact with each other and the environment through development and life.

In a non-mammalian example of genes related to aggression, the fruitless gene in fruit flies is a critical determinant of certain sexually dimorphic behaviors, and its artificial alteration can result in a reversal of stereotypically male and female patterns of aggression in fighting. However, in what was thought to be a relatively clear case, inherent complexities have been reported in deciphering the connections between interacting genes in an environmental context and a social phenotype involving multiple behavioral and sensory interactions with another organism.

In mice, candidate genes for differentiating aggression between the sexes are the Sry (sex determining region Y) gene, located on the Y chromosome and the Sts (steroid sulfatase) gene. The Sts gene encodes the steroid sulfatase enzyme, which is pivotal in the regulation of neurosteroid biosynthesis. It is expressed in both sexes, is correlated with levels of aggression among male mice, and increases dramatically in females after parturition and during lactation, corresponding to the onset of maternal aggression.

In humans, there is good evidence that the basic human neural architecture underpinning the potential for flexible aggressive responses is influenced by genes as well as environment. In terms of variation between individual people, more than 100 twin and adoption studies studies have been conducted in recent decades examining the genetic basis of aggressive behavior and related constructs such as conduct disorders. According to a meta-analysis published in 2002, approximately 40% of variation between individuals is explained by differences in genes, and 60% by differences in environment (mainly non-shared environmental influences rather than those that would be shared by being raised together). However, such studies have depended on self-report or observation by others including parents, which complicates interpretation of the results. The few laboratory-based analyses have not found significant amounts of individual variation in aggression explicable by genetic variation in the human population. Furthermore, linkage and association studies that seek to identify specific genes, for example that influence neurotransmitter or hormone levels, have generally resulted in contradictory findings characterized by failed attempts at replication. One possible factor is an allele (variant) of the MAO-A gene which, in interaction with certain life events such as childhood maltreatment (which may show a main effect on its own), can influence development of brain regions such as the amygdala and as a result some types of behavioral response may be more likely. The generally unclear picture has been compared to equally difficult findings obtained in regard to other complex behavioral phenotypes.[26]

In humans

Humans share aspects of aggression with non-human animals, and have specific aspects and complexity related to factors such as genetics, early development, social learning and flexibility, culture and morals. Konrad Lorenz stated in his 1963 classic, *On Aggression,* that human behavior is shaped by four main, survival-seeking animal drives. Taken together, these drives—hunger, fear, reproduction, and aggression—achieve natural selection.[27] E.O. Wilson elaborated in *On Human Nature* that aggression is, typically, a means of gaining control over resources. Aggression is, thus, aggravated during times when high population densities generate resource shortages.[28]

Culture

Culture is a factor that plays a role in aggression.

Tribal or band societies existing before or outside of modern states have sometimes been depicted as peaceful 'noble savages' or alternatively as brutish 'beasts'. The Kung Bushmen were described as 'The Harmless People' in a popular work by Elizabeth Marshall Thomas in 1958,[29] while Lawrence Keeley's 1996 War Before Civilization suggested that regular warfare without modern technology was conducted by most groups throughout human history, including most Native American tribes.[30] Studies of hunter-gatherers show a range of different societies. In general, aggression, conflict and violence sometimes occur, but direct confrontation is generally avoided and conflict is socially managed by a variety of verbal and non-verbal methods. Different rates of aggression or violence, currently or in the past, within or between groups, have been linked to the structuring of societies and environmental conditions influencing factors such as resource or property acquisition, land and subsistence techniques, and population change.[31]

Analyzing aggression culturally or politically is complicated by the fact that the label 'aggressive' can itself be used as a way of asserting a judgement from a particular point of view. Whether a coercive or violent method of social control is perceived as aggression – or as legitimate versus illegitimate aggression – depends on the position of the relevant parties in relation to the social order of their culture. This in turn can relate to factors such as: norms for coordinating actions and dividing resources; what is considered self-defense or provocation; attitudes towards 'outsiders', attitudes towards specific groups such as women, the disabled or the lower status; the availability of alternative conflict resolution strategies; trade interdependence and collective security pacts; fears and impulses; and ultimate goals regarding material and social outcomes.[32]

Insignia of the 18th Aggressor Squadron

Cross-cultural research has found differences in attitudes towards aggression in different cultures. In one questionnaire study of university students, in addition to men overall justifying some types of aggression more than women, USA respondents justified defensive physical aggression more readily than Japanese or Spanish respondents, whereas Japanese students preferred direct verbal aggression (but not indirect) more than their American and Spanish counterparts. Within American culture, southern men were shown in a study on university students to be more affected and to respond more aggressively than northerners when randomly insulted after being bumped into, which was theoretically related to a traditional culture of honor in the Southern United States. A similar sociological concept that may be applied in different cultures is 'face'. Other cultural themes sometimes applied to the study of aggression include individualistic versus collectivist styles, which may relate, for example, to whether disputes are responded to with open competition or by accommodating and avoiding conflicts. Other comparisons made in relation to aggression or war include democratic versus authoritarian political systems and egalitarian versus stratified societies. The economic system known as capitalism has been viewed by some as reliant on the leveraging of human competitiveness and aggression in pursuit of resources and trade, which has been considered in both positive and negative terms.[33] Attitudes about the social acceptability of particular acts or targets of aggression are also important factors. This can be highly controversial, as for example in disputes between religions or nation states, for example in regard to the Arab–Israeli conflict.

Media

Some scholars believe that behaviors like aggression may be partially learned by watching and imitating the behavior of others. Some scholars have concluded that media may have some small effects on aggression.[34] There is also research questioning this view.[35] For instance, a recent long-term outcome study of youth found no long-term relationship between playing violent video games and youth violence or bullying.[36] One study suggested there is a smaller effect of violent video games on aggression than has been found with television violence on aggression. This effect is positively associated with type of game violence and negatively associated to time spent playing the games. The author concluded that insufficient evidence exists to link video game violence with aggression. However, another study suggested links to aggressive behavior. One study suggested that adults (i.e. parents) suffering from dissociative symptoms related to post-traumatic stress disorder may be more likely to expose their children to violent programs and video games; links between these issues were also related to poverty.[37]

Fear(survival)-induced pre-emptive aggression

According to philosopher and neuroscientist Nayef Al-Rodhan, "fear(survival)-induced pre-emptive aggression" is a human reaction to injustices that are perceived to threaten survival. It is often the root of the unthinkable brutality and injustice perpetuated by human beings. It may occur at any time, even in situations that appear to be calm and under control. Where there is injustice that is perceived as posing a threat to survival, "fear(survival)-induced pre-emptive aggression" will result in individuals taking whatever action necessary to be free from that threat.

Nayef Al-Rodhan argues that humans' strong tendency towards "fear(survival)-induced pre-emptive aggression" means that situations of anarchy or near anarchy should be prevented at all costs. This is because anarchy provokes fear, which in turn results in aggression, brutality, and injustice. Even in non-anarchic situations, survival instincts and fear can be very powerful forces, and they may be incited instantaneously. "Fear(survival)-induced pre-emptive aggression" is one of the key factors that may push naturally amoral humans to behave in immoral ways.[38] Knowing this, Al-Rodhan maintains that we must prepare for the circumstances that may arise from humans' aggressive behavior. According to Al-Rodhan, the risk of this aggression and its ensuing brutality should be minimized through confidence-building measures and policies that promote inclusiveness and prevent anarchy.[39]

Children

The frequency of physical aggression in humans peaks at around 2–3 years of age. It then declines gradually on average. These observations suggest that physical aggression is not only a learned behavior but that development provides opportunities for the learning and biological development of self-regulation. However, a small subset of children fail to acquire all the necessary self-regulatory abilities and tend to show atypical levels of physical aggression across development. These may be at risk for later violent behavior or, conversely, lack of aggression that may be considered necessary within society. Some findings suggest that early aggression does not necessarily lead to aggression later on, however, although the course through early childhood is an important predictor of outcomes in middle childhood. In addition, physical aggression that continues is likely occurring in the context of family adversity, including socioeconomic factors. Moreover, 'opposition' and 'status violations' in childhood appear to be more strongly linked to social problems in adulthood than simply aggressive antisocial behavior. Social learning through interactions in early childhood has been seen as a building block for levels of aggression which play a crucial role in the development of peer relationships in middle childhood. Overall, an interplay of biological, social and environmental factors can be considered.

What is typically expected of children?

- Young children preparing to enter kindergarten need to develop the socially important skill of being assertive. Examples of assertiveness include asking others for information, initiating conversation, or being able to respond to peer pressure.
- In contrast, some young children use aggressive behavior, such as hitting or biting, as a form of communication.

- Aggressive behavior can impede learning as a skill deficit, while assertive behavior can facilitate learning. However, with young children, aggressive behavior is developmentally appropriate and can lead to opportunities of building conflict resolution and communication skills.
- By school age, children should learn more socially appropriate forms of communicating such as expressing themselves through verbal or written language; if they have not, this behavior may signify a disability or developmental delay

What triggers aggressive behavior in children?

- Physical fear of others
- Family difficulties
- Learning, neurological, or conduct/behavior disorders
- Psychological trauma

Corporal punishment such as spanking increases subsequent aggression in children.

The Bobo doll experiment was conducted by Albert Bandura in 1961. In this work, Bandura found that children exposed to an aggressive adult model acted more aggressively than those who were exposed to a nonaggressive adult model. This experiment suggests that anyone who comes in contact with and interacts with children can have an impact on the way they react and handle situations.

Summary points from recommendations by national associations

- American Academy of Pediatrics (2011): "The best way to prevent aggressive behavior is to give your child a stable, secure home life with firm, loving discipline and full-time supervision during the toddler and preschool years. Everyone who cares for your child should be a good role model and agree on the rules he's expected to observe as well as the response to use if he disobeys."[40]
- National Association of School Psychologists (2008): "Proactive aggression is typically reasoned, unemotional, and focused on acquiring some goal. For example, a bully wants peer approval and victim submission, and gang members want status and control. In contrast, reactive aggression is frequently highly emotional and is often the result of biased or deficient cognitive processing on the part of the student."[41]

Gender

Gender is a factor that plays a role in both human and animal aggression. Males are historically believed to be generally more physically aggressive than females from an early age,[42][43] and men commit the vast majority of murders (Buss 2005). This is one of the most robust and reliable behavioral sex differences, and it has been found across many different age groups and cultures. However, some empirical studies have found the discrepancy in male and female aggression to be more pronounced in childhood and the gender difference in adults to be modest.[44] Still, there is evidence that males are quicker to aggression (Frey et al. 2003) and more likely than females to express their aggression physically. When considering indirect forms of non-violent aggression, such as relational aggression and social rejection, some scientists argue that females can be quite aggressive although female aggression is rarely expressed physically. An exception is intimate partner violence that occurs among couples who are engaged, married, or in some other form of intimate relationship. In such cases women have been found to be more physically aggressive than men, although differences are small, men are less likely to be injured than are women.[45]

Studies show, that females in general have better control over their emotions in comparison to males. Also, males are more likely to retaliate when provoked to gain recognition; females are less likely to retaliate in a violent way because they are shielded by moral sense.[46] Although females are less likely to initiate physical violence, they can express aggression by using a variety of non-physical means. Exactly which method women use to express aggression is something that varies from culture to culture. On Bellona Island, a culture based on male dominance and physical violence, women tend to get into conflicts with other women more frequently than with men. When in conflict with males, instead of using physical means, they make up songs mocking the man, which spread across the island and humiliate him. If a woman wanted to kill a man, she would either convince her male relatives to kill him

or hire an assassin. Although these two methods involve physical violence, both are forms of indirect aggression, since the aggressor herself avoids getting directly involved or putting herself in immediate physical danger.

See also the sections on testosterone and evolutionary explanations for gender differences above.

Situational factors

There has been some links between those prone to violence and their alcohol use. Those who are prone to violence and use alcohol are more likely to carry out violent acts. Alcohol impairs judgment, making people much less cautious than they usually are (MacDonald et al. 1996). It also disrupts the way information is processed (Bushman 1993, 1997; Bushman & Cooper 1990).

Pain and discomfort also increase aggression. Even the simple act of placing one's hands in hot water can cause an aggressive response. Hot temperatures have been implicated as a factor in a number of studies. One study completed in the midst of the civil rights movement found that riots were more likely on hotter days than cooler ones (Carlsmith & Anderson 1979). Students were found to be more aggressive and irritable after taking a test in a hot classroom (Anderson et al. 1996, Rule, et al. 1987). Drivers in cars without air conditioning were also found to be more likely to honk their horns (Kenrick & MacFarlane 1986), which is used as a measure of aggression and has shown links to other factors such as generic symbols of aggression or the visibility of other drivers.

Frustration is another major cause of aggression. The Frustration aggression theory states that aggression increases if a person feels that he or she is being blocked from achieving a goal (Aronson et al. 2005). One study found that the closeness to the goal makes a difference. The study examined people waiting in line and concluded that the 2nd person was more aggressive than the 12th one when someone cut in line (Harris 1974). Unexpected frustration may be another factor. In a separate study to demonstrate how unexpected frustration leads to increased aggression, Kulik & Brown (1979) selected a group of students as volunteers to make calls for charity donations. One group was told that the people they would call would be generous and the collection would be very successful. The other group was given no expectations. The group that expected success was more upset when no one was pledging than the group who did not expect success (everyone actually had horrible success). This research suggests that when an expectation does not materialize (successful collections), unexpected frustration arises which increases aggression.

There is some evidence to suggest that the presence of violent objects such as a gun can trigger aggression. In a study done by Leonard Berkowitz and Anthony Le Page (1967), college students were made angry and then left in the presence of a gun or badminton racket. They were then led to believe they were delivering electric shocks to another student, as in the Milgram experiment. Those who had been in the presence of the gun administered more shocks. It is possible that a violence-related stimulus increases the likelihood of aggressive cognitions by activating the semantic network.

A new proposal links military experience to anger and aggression, developing aggressive reactions and investigating these effects on those possessing the traits of a serial killer. Castle and Hensley state, "The military provides the social context where servicemen learn aggression, violence, and murder." Post-traumatic stress disorder (PTSD) is also a serious issue in the military, also believed to sometimes lead to aggression in soldiers who are suffering from what they witnessed in battle. They come back to the civilian world and may still be haunted by flashbacks and nightmares, causing severe stress. In addition, it has been claimed that in the rare minority who are claimed to be inclined toward serial killing, violent impulses may be reinforced and refined in war, possibly creating more effective murderers.[*citation needed*]

As a positive adaptation theory

Some recent scholarship has questioned traditional psychological conceptualizations of aggression as universally negative. Most traditional psychological definitions of aggression focus on the harm to the recipient of the aggression, implying this is the intent of the aggressor; however this may not always be the case. From this alternate view, although the recipient may or may not be harmed, the perceived intent is to increase the status of the aggressor, not necessarily to harm the recipient. Such scholars contend that traditional definitions of aggression have no validity.[*citation needed*]

From this view, rather than concepts such as assertiveness, aggression, violence and criminal violence existing as distinct constructs, they exist instead along a continuum with moderate levels of aggression being most adaptive. Such scholars do not consider this a trivial difference, noting that many traditional researchers' aggression measurements may measure outcomes lower down in the continuum, at levels which are adaptive, yet they generalize their findings to non-adaptive levels of aggression, thus losing precision.

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American Psychiatric Association

American Psychiatric Association	
Logo of the APA	
Formation	1844
Headquarters	1000 Wilson Boulevard, Suite 1825 Arlington, Virginia, United States
Membership	36,000 members
May 2013–2014 President	Jeffrey Lieberman, M.D.
Website	www.psychiatry.org [1]

The **American Psychiatric Association** (**APA**) is the main professional organization of psychiatrists and trainee psychiatrists in the United States, and the largest psychiatric organization in the world. Its some 36,000 members are mainly American but some are international. The association publishes various journals and pamphlets, as well as the *Diagnostic and Statistical Manual of Mental Disorders* (DSM). The DSM codifies psychiatric conditions and is used worldwide as a key guide for diagnosing disorders.

The organization has its headquarters in Arlington County, Virginia.[2]

History

At a meeting in 1844 in Philadelphia, 13 superintendents and organizers of insane asylums and hospitals formed the Association of Medical Superintendents of American Institutions for the Insane (AMSAII). The group included Thomas Kirkbride, creator of the asylum model which was used throughout the United States. At the meeting they passed the first proposition of the new organization: "It is the unanimous sense of this convention that the attempt to abandon entirely the use of all means of personal restraint is not sanctioned by the true interests of the insane."

The name of the organization was changed in 1892 to The American Medico-Psychological Association to allow assistant physicians working in mental hospitals to become members.

In 1921, the name was changed to the present American Psychiatric Association. The APA emblem, dating to 1890, became more officially adopted from that year. It was a round medallion with a purported facial likeness of Benjamin Rush and 13 stars over his head to represent the 13 founders of the organization. The outer ring contains the words "American Psychiatric Association 1844." Rush's name and an M.D.[3] The Association was Incorporated in the District of Columbia in 1927.

In 1948, APA formed a small task force to create a new standardized psychiatric classification system. This resulted in the 1952 publication of the first DSM. In 1965 a new task force of 10 people developed DSM-II, published in 1968. DSM-III was published in 1980, after a larger process involving some 600 clinicians. The book was now 500 pages long, including many more disorders, and it sold nearly half a million copies. APA published a revised DSM-III-R in 1987 and DSM-IV in 1994, the latter selling nearly a million copies by the end of 2000. DSM-IV-TR with minor revisions was published in 2000. APA is currently developing and consulting on DSM-V, which will be published in May 2013.

In the early 1970s, activists campaigned against the DSM classification of homosexuality as a mental disorder, protesting at APA offices and at annual meetings from 1970 to 1973. In 1973 the Board of Trustees voted to remove homosexuality as a disorder category from the DSM, a decision ratified by a majority (58%) of the general APA membership the following year. A category of "sexual orientation disturbance" was introduced in its place in 1974, and then replaced in the 1980 DSM-III with Ego-dystonic sexual orientation. That was removed in 1987.

In 2002, amidst increasing concern to differentiate themselves from clinical psychologists, the APA assembly membership voted against a proposed name change to the American Psychiatric Medical Association.

Dr. Saul Levin was named on May 15, 2013 as the new chief executive officer and medical director of the APA, making him the first known openly gay person to head the APA.[4]

Organization and membership

APA is led by the President of the American Psychiatric Association and a Board of Trustees with an Executive Committee.

APA reports [5] that its membership is primarily medical specialists who are qualified, or in the process of becoming qualified, as psychiatrists. The basic eligibility requirement is completion of a residency program in psychiatry accredited by the Residency Review Committee for Psychiatry of the Accreditation Council for Graduate Medical Education (ACGME), the Royal College of Physicians and Surgeons of Canada (RCPS(C)), or the American Osteopathic Association (AOA). Applicants for membership must also hold a valid medical license (with the exception of medical students and residents) and provide one reference who is an APA member.

APA holds an annual conference attended by a US and international audience.

APA is made up of some 76 district associations throughout the US.

Theoretical position

The APA reflects and represents mainstream psychiatry in the United States. Reflecting larger trends, the APA members and leaders had been largely psychodynamic in their approaches until recent decades, when the field became more "biopsychosocial."

The DSM is currently intended to be less theoretical than prior editions, having moved away from psychodynamic theories to be more widely accepted, and is proposed to not be committed to a particular theorized etiology for mental disorders. The criteria for many of the mental disorders have been expanded and involve a checklist of so-called 'Feighner Criteria' to try and capture the varying sets of features which would be necessary to diagnose a particular disorder.

Publications and campaigns

APA position statements,[6] Psych.org and practice guidelines[7] and description of its core diagnostic manual the DSM [8] are published.

APA publishes several journals[9] focused on different areas of psychiatry, for example, academic, clinical practice, or news.

APA recently launched a health campaign[10] with a new PR approach.

Top five Choosing Wisely® recommendations

In coordination with the American Board of Internal Medicine, the APA proposes five recommendations for physicians and patients. The list was compiled by members of the Council on Research and Quality Care. The APA places a primary focus on antipsychotic medications due to a rapid increase in sales, from $9.6 billion in 2004 to $18.5 billion in 2011.

1. Don’t prescribe antipsychotic medications to patients for any indication without appropriate initial evaluation and appropriate ongoing monitoring.
2. Don’t routinely prescribe 2 or more antipsychotic medications concurrently.
3. Don’t prescribe antipsychotic medications as a first-line intervention to treat behavioral and psychological symptoms of dementia.

4. Don't routinely prescribe antipsychotic medications as a first-line intervention for insomnia in adults.
5. Don't routinely prescribe antipsychotic medications as a first-line intervention for children or adolescents for any diagnosis other than psychotic disorders.

Notable figures

- Adolf Meyer rose to prominence as the president of the American Psychiatric Association and was one of the most influential figures in psychiatry in the first half of the twentieth century.
- Robert Spitzer was a key figure in the development of later editions of the DSM.
- Donald Ewen Cameron is best known for his MK-ULTRA-related mind-control and behavior modification research for the CIA. Cameron was President of the APA in 1952-1953.
- Current president Jeffrey Lieberman was principal investigator for the NIMH CATIE study.

Drug company ties

In his book *Anatomy of an Epidemic* (2010), Robert Whitaker described the partnership that has developed between the APA and pharmaceutical companies since the 1980s. APA has come to depend on pharmaceutical money. The drug companies endowed continuing education and psychiatric "grand rounds" at hospitals. They funded a political action committee (PAC) in 1982 to lobby Congress. The industry helped to pay for the APA's media training workshops. It was able to turn psychiatrists at top schools into speakers, and although the doctors felt they were independents, they rehearsed their speeches and likely would not be invited back if they discussed drug side effects. "Thought leaders" became the experts quoted in the media. As Marcia Angell wrote in *The New England Journal of Medicine* (2000), "thought leaders" could agree to be listed as an author of ghostwritten articles, and she cites Thomas Bodenheimer and David Rothman who describe the extent of the drug industry's involvement with doctors. *The New York Times* published a summary about antipsychotic medications in October 2010.

In 2008, for the first time, Senator Charles Grassley asked the APA to disclose how much of its annual budget came from drug industry funds. The APA said that industry contributed 28% of its budget ($14 million at that time), mainly through paid advertising in APA journals and funds for continuing medical education.

Controversies

Controversies have related to anti-psychiatry and disability rights campaigners, who regularly protest at American Psychiatric Association offices or meetings. In 1971, members of the Gay Liberation Front organization sabotaged an APA conference in San Francisco. In 2003 activists from MindFreedom International staged a 21-day hunger strike, protesting at a perceived unjustified biomedical focus and challenging APA to provide evidence of the widespread claim that mental disorders are due to chemical imbalances in the brain. APA published a position statement in response and the two organizations exchanged views on the evidence.

There was controversy when it emerged that US psychologists and psychiatrists were helping interrogators in Guantanamo and other US facilities. The American Psychiatric Association released a policy statement that psychiatrists should not take a direct part in interrogation of particular prisoners [11] but could "offer general advice on the possible medical and psychological effects of particular techniques and conditions of interrogation, and on other areas within their professional expertise."

After previous controversy over APA's classification of homosexuality as a mental illness, there is also controversy regarding the remaining category of "sexual disorder not otherwise specified" which can include a state of distress about one's sexual orientation, as well as the diagnosis of "gender identity disorder" or gender dysphoria.[12]

The APA's Standard Diagnostic Manual came under criticism from autism specialists Tony Attwood and Simon Baron-Cohen for proposing the elimination of Asperger's syndrome as a disorder and replacing it with an autism severity scale. Professor Roy Richard Grinker wrote a controversial editorial for the New York Times expressing

support for the proposal.

The APA president in 2005, Steven Sharfstein, caused controversy when, although praising the pharmaceutical industry, he argued that American psychiatry had "allowed the biopsychosocial model to become the bio-bio-bio model" and accepted "kickbacks and bribes" from pharmaceutical companies leading to the over-use of medication and neglect of other approaches.[13] In 2008 APA became a focus of congressional investigations regarding the way that money from the pharmaceutical industry can shape the practices of nonprofit organizations that purport to be independent in their viewpoints and actions. The drug industry accounted in 2006 for about 30 percent of the association's $62.5 million in financing, half through drug advertisements in its journals and meeting exhibits, and the other half sponsoring fellowships, conferences and industry symposiums at its annual meeting. APA is considering its response to increasingly intense scrutiny and questions about conflicts of interest.[14] The APA president of 2009-2010, Alan Schatzberg, has also come under fire after it came to light that he was principal investigator on a federal study into the drug Mifepristone for use as an antidepressant being developed by Corcept Therapeutics, a company Schatzberg had himself set up and in which he had several millions of dollars' worth of stock.[15]

In the 1964 election, *Fact* magazine polled American Psychiatric Association members on whether Barry Goldwater was fit to be president and published "The Unconscious of a Conservative: A Special Issue on the Mind of Barry Goldwater." This led to the banning of diagnosing public figures when you have not performed an examination or been authorized to release information by the patient. This became the Goldwater rule.

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External links

- Official website (http://www.psychiatry.org)

Antisocial personality disorder

Antisocial personality disorder	
Classification and external resources	
ICD-10	F60.2 [1]
ICD-9	301.7 [2]
MedlinePlus	000921 [3]
MeSH	D000987 [4]

Antisocial (or **dissocial**) **personality disorder** is characterized by a pervasive pattern of disregard for, or violation of, the rights of others. There may be an impoverished moral sense or conscience and a history of crime, legal problems, impulsive and aggressive behavior.

Antisocial personality disorder (ASPD) is the name of the disorder as defined in the *Diagnostic and Statistical Manual* (DSM). *Dissocial personality disorder* is the name of a similar or equivalent concept defined in the *International Statistical Classification of Diseases and Related Health Problems* (ICD), where it states that the diagnosis includes antisocial personality disorder. Both manuals have similar but not identical criteria. Both have also stated that their diagnoses have been referred to, or include what is referred to, as psychopathy or sociopathy, though distinctions are sometimes made.[5][6]

Diagnosis

DSM-IV-TR

The APA's Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV-TR), defines antisocial personality disorder (in Axis II Cluster B):

> A) There is a pervasive pattern of disregard for and violation of the rights of others occurring since age 15 years, as indicated by three or more of the following:

1. failure to conform to social norms with respect to lawful behaviors as indicated by repeatedly performing acts that are grounds for arrest;
2. deception, as indicated by repeatedly lying, use of aliases, or conning others for personal profit or pleasure;
3. impulsivity or failure to plan ahead;
4. irritability and aggressiveness, as indicated by repeated physical fights or assaults;
5. reckless disregard for safety of self or others;
6. consistent irresponsibility, as indicated by repeated failure to sustain consistent work behavior or honor financial obligations;
7. lack of remorse, as indicated by being indifferent to or rationalizing having hurt, mistreated, or stolen from another;

> B) The individual is at least age 18 years.
>
> C) There is evidence of conduct disorder with onset before age 15 years.
>
> D) The occurrence of antisocial behavior is not exclusively during the course of schizophrenia or a manic episode.

ASPD falls under the dramatic/erratic cluster of personality disorders.[7] In the DSM-5, the diagnosis antisocial personality disorder is kept, but it is no longer on another axis as the other mental disorders.

ICD-10

The WHO's *International Statistical Classification of Diseases and Related Health Problems*, tenth edition (ICD-10), has a diagnosis called dissocial personality disorder (F60.2 [1]):[8][9]

It is characterized by at least 3 of the following:

1. Callous unconcern for the feelings of others;
2. Gross and persistent attitude of irresponsibility and disregard for social norms, rules, and obligations;
3. Incapacity to maintain enduring relationships, though having no difficulty in establishing them;
4. Very low tolerance to frustration and a low threshold for discharge of aggression, including violence;
5. Incapacity to experience guilt or to profit from experience, particularly punishment;
6. Marked readiness to blame others or to offer plausible rationalizations for the behavior that has brought the person into conflict with society.

The ICD states that this diagnosis includes "amoral, antisocial, asocial, psychopathic, and sociopathic personality". Although the disorder is not synonymous with conduct disorder, presence of conduct disorder during childhood or adolescence may further support the diagnosis of dissocial personality disorder. There may also be persistent irritability as an associated feature.

It is a requirement of the ICD-10 that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

Further considerations

Psychopathy

Psychopathy is commonly defined as a personality disorder characterized partly by antisocial behavior, a diminished capacity for remorse, and poor behavioral controls. Psychopathic traits are assessed using various measurement tools, including Canadian researcher Robert D. Hare's Psychopathy Checklist, Revised (PCL-R).[10] "Psychopathy" is not the official title of any diagnosis in the DSM or ICD.[11][12]

American psychiatrist Hervey Cleckley's work[*citation needed*] on psychopathy formed the basis of the diagnostic criteria for ASPD, and the DSM has stated that ASPD has also been referred to as psychopathy. However, critics have argued that ASPD is not synonymous with psychopathy as the diagnostic criteria are not exactly the same, since criteria relating to personality traits are emphasized relatively less in the former. These differences exist in part because it was believed that such traits were difficult to measure reliably and it was "easier to agree on the behaviors that typify a disorder than on the reasons why they occur".

Although the diagnosis of ASPD covers two to three times as many prisoners as are rated as psychopaths, Robert Hare believes that the PCL-R is better able to predict future criminality, violence, and recidivism than a diagnosis of ASPD. He suggests that there are differences between PCL-R-diagnosed psychopaths and non-psychopaths on "processing and use of linguistic and emotional information", while such differences are potentially smaller between those diagnosed with ASPD and without. Additionally, Hare argued that confusion regarding how to diagnose ASPD, confusion regarding the difference between ASPD and psychopathy, as well as the differing future prognoses regarding recidivism and treatability, may have serious consequences in settings such as court cases where psychopathy is often seen as aggravating the crime.

Nonetheless, psychopathy has been proposed as a specifier under an alternative model for ASPD. In the DSM-5, under "Alternative DSM-5 Model for Personality Disorders", ASPD with psychopathic features is described as characterized by "a lack of anxiety or fear and by a bold interpersonal style that may mask maladaptive behaviors (e.g., fraudulence)." Low levels of withdrawal and high levels of attention-seeking combined with low anxiety demonstrate "social potency" and "stress immunity" in psychopathy.:765 Under the specifier, affective and interpersonal characteristics are comparatively emphasized over behavioral components.

Theodore Millon's subtypes

Theodore Millon suggested five subtypes of ASPD:[13][14]

Subtype	Features
Nomadic (including schizoid and avoidant features)	Feels jinxed, ill-fated, doomed, and cast aside; peripheral, drifters; gypsy-like roamers, vagrants; dropouts and misfits; itinerant vagabonds, tramps, wanderers; impulsively not benign.
Malevolent (including sadistic and paranoid features)	Belligerent, mordant, rancorous, vicious, malignant, brutal, resentful; anticipates betrayal and punishment; desires revenge; truculent, callous, fearless; guiltless.
Covetous (variant of "pure" pattern)	Feels intentionally denied and deprived; rapacious, begrudging, discontentedly yearning; envious, seeks retribution, and avariciously greedy; pleasure more in taking than in having.
Risk-taking (including histrionic features)	Dauntless, venturesome, intrepid, bold, audacious, daring; reckless, foolhardy, impulsive, heedless; unbalanced by hazard; pursues perilous ventures.
Reputation-defending (including narcissistic features)	Needs to be thought of as infallible, unbreakable, invincible, indomitable, formidable, inviolable; intransigent when status is questioned; over-reactive to slights.

Elsewhere, Millon differentiates ten subtypes (partially overlapping with the above) – covetous, risk-taking, malevolent, tyrannical, malignant, unprincipled, disingenuous, spineless, explosive, and abrasive – but specifically stresses that "the number 10 is by no means special ... Taxonomies may be put forward at levels that are more coarse or more fine-grained."[15]

Comorbidity

The following conditions commonly coexist with ASPD:[16]

- Anxiety disorders
- Depressive disorder
- Impulse control disorders
- Substance-related disorders
- Somatization disorder
- Attention deficit hyperactivity disorder
- Borderline personality disorder
- Histrionic personality disorder
- Narcissistic personality disorder
- Sadistic personality disorder

When combined with alcoholism, people may show frontal function deficits on neuropsychological tests greater than those associated with each condition.

Causes and pathophysiology

Personality disorders seem to be caused by a combination of these genetic and environmental influences. Genetically, it is the temperament and the kind of personality a person is born with, and environmentally, it is the way in which a person grows up and the experiences they have had.

Hormones and neurotransmitters

Traumatic events can lead to a disruption of the standard development of the central nervous system, which can generate a release of hormones that can change normal patterns of development. Aggressiveness and impulsivity are among the possible symptoms of ASPD. Testosterone is a hormone that plays an important role in aggressiveness in the brain. For instance, criminals who have committed violent crimes have higher levels of testosterone. The effect of testosterone is counteracted by cortisol which facilitates the cognitive control on impulsive tendencies.

One of the neurotransmitters that have been discussed in individuals with ASPD is serotonin. A meta-analysis of 20 studies found significantly lower 5-HIAA levels (indicating lower serotonin levels), especially in those who are younger than 30 years of age.

J.F.W. Deakin of University of Manchester's Neuroscience and Psychiatry Unit has discussed additional evidence of 5HT's connection with ASPD. Deakin suggests that low cerebrospinal fluid concentrations of 5-HIAA, and hormone responses to 5HT, have displayed that the two main ascending 5HT pathways mediate adaptive responses to post and current conditions. He states that impairments in the posterior 5HT cells can lead to low mood functioning, as seen in patients with ASPD. It is important to note that the dysregulated serotonergic function may not be the sole feature that leads to ASPD but it is an aspect of a multifaceted relationship between biological and psychosocial factors.[*citation needed*]

While it has been shown that lower levels of serotonin may be associated with ASPD, there has also been evidence that decreased serotonin function is highly correlated with impulsiveness and aggression across a number of different experimental paradigms. Impulsivity is not only linked with irregularities in 5HT metabolism but may be the most essential psychopathological aspect linked with such dysfunction. Correspondingly, the DSM classifies "impulsivity or failure to plan ahead" and "irritability and aggressiveness" as two of seven sub-criteria in category A of the diagnostic criteria of ASPD.

Some studies have found a relationship between monoamine oxidase A and antisocial behavior, including conduct disorder and symptoms of adult ASPD, in maltreated children.[*citation needed*]

Limbic neural maldevelopment

Cavum septum pellucidum (CSP) is a marker for limbic neural maldevelopment. One study found that those with CSP had significantly higher levels of antisocial personality, psychopathy, arrests and convictions compared with controls.

Cultural influences

The Socio-cultural perspective of clinical psychology view disorders as being influenced by cultural aspects, since cultural norms differ significantly, mental disorders such as ASPD are viewed differently. Robert D. Hare has suggested that the rise in ASPD that has been reported in the United States may be linked to changes in cultural mores, the latter serving to validate the behavioral tendencies of many individuals with ASPD.[17] While the rise reported may be in part merely a byproduct of the widening use (and abuse) of diagnostic techniques,[18] given Eric Berne's division between individuals with active and latent ASPD – the latter keeping themselves in check by attachment to an external source of control like the law, traditional standards, or religion[19] – it has been plausibly suggested that the erosion of collective standards may indeed serve to release the individual with latent ASPD from their previously prosocial behavior.[20]

There is also a continuous debate as to the extent to which the legal system should be involved in the identification and admittance of patients with preliminary symptoms of ASPD.[21]

Environment

Some studies suggest that the social and home environment has contributed to the development of antisocial behavior.[22] The parents of these children have been shown to display antisocial behavior, which could be adopted by their children.

Head injuries

Researchers have linked physical head injuries with antisocial behavior.[23][24] Since the 1980s, scientists have associated traumatic brain injury, including damage to the prefrontal cortex, with an inability to make morally and socially acceptable decisions. Children with early damage in the prefrontal cortex may never fully develop social or moral reasoning and become "psychopathic individuals ... characterized by high levels of aggression and antisocial behavior performed without guilt or empathy for their victims." Additionally, damage to the amygdala may impair the ability of the prefrontal cortex to interpret feedback from the limbic system, which could result in uninhibited signals that manifest in violent and aggressive behavior.

Treatment

ASPD is considered to be among the most difficult personality disorders to treat.[25][26]Wikipedia:Verifiability Because of their very low or absent capacity for remorse, individuals with ASPD often lack sufficient motivation and fail to see the costs associated with antisocial acts. They may only simulate remorse rather than truly commit to change: they can be seductively charming and dishonest, and may manipulate staff and fellow patients during treatment.[27]Wikipedia:Verifiability Studies have shown that outpatient therapy is not likely to be successful, however the extent to which persons with ASPD are entirely unresponsive to treatment may have been exaggerated.

Those with ASPD may stay in treatment only as required by an external source, such as a parole. Residential programs that provide a carefully controlled environment of structure and supervision along with peer confrontation have been recommended. There has been some research on the treatment of ASPD that indicated positive results for therapeutic interventions. Schema Therapy is also being investigated as a treatment for ASPD. A review by Charles M. Borduin features the strong influence of Multisystemic therapy (MST) that could potentially improve this imperative issue. However this treatment requires complete cooperation and participation of all family members.[28] Some studies have found that the presence of ASPD does not significantly interfere with treatment for other disorders, such as substance abuse, although others have reported contradictory findings.

Therapists of individuals with ASPD may have considerable negative feelings toward clients with extensive histories of aggressive, exploitative, and abusive behaviors. Rather than attempt to develop a sense of conscience in these individuals, therapeutic techniques should be focused on rational and utilitarian arguments against repeating past mistakes. These approaches would focus on the tangible, material value of prosocial behavior.[29]

No medications have been approved by the FDA to treat ASPD, although certain psychiatric medications may alleviate conditions sometimes associated with the disorder and with symptoms such as aggression, including antipsychotic, antidepressant or mood-stabilizing medications.

Prognosis

According to Professor Emily Simonoff, Institute of Psychiatry, "childhood hyperactivity and conduct disorder showed equally strong prediction of antisocial personality disorder (ASPD) and criminality in early and mid-adult life. Lower IQ and reading problems were most prominent in their relationships with childhood and adolescent antisocial behaviour."

Epidemiology

ASPD is seen in 3% to 30% of psychiatric outpatients. The prevalence of the disorder is even higher in selected populations, like prisons, where there is a preponderance of violent offenders.[30] A 2002 literature review of studies on mental disorders in prisoners stated that 47% of male prisoners and 21% of female prisoners had ASPD. Similarly, the prevalence of ASPD is higher among patients in alcohol or other drug (AOD) abuse treatment programs than in the general population (Hare 1983), suggesting a link between ASPD and AOD abuse and dependence.

History

The first version of the DSM in 1952 listed *sociopathic personality disturbance*. Individuals to be placed in this category were said to be "...ill primarily in terms of society and of conformity with the prevailing milieu, and not only in terms of personal discomfort and relations with other individuals". There were four subtypes, referred to as "reactions"; antisocial, dyssocial, sexual and addiction. The antisocial reaction was said to include people who were "always in trouble" and not learning from it, maintaining "no loyalties", frequently callous and lacking responsibility, with an ability to "rationalize" their behavior. The category was described as more specific and limited than the existing concepts of "constitutional psychopathic state" or "psychopathic personality" which had had a very broad meaning; the narrower definition was in line with criteria advanced by Hervey M. Cleckley from 1941, while the term sociopathic had been advanced by George Partridge.

The DSM-II in 1968 rearranged the categories and "antisocial personality" was now listed as one of ten personality disorders but still described similarly, to be applied to individuals who are: "basically unsocialized", in repeated conflicts with society, incapable of significant loyalty, selfish, irresponsible, unable to feel guilt or learn from prior experiences, and who tend to blame others and rationalise. The DSM-II warned that a history of legal or social offenses was not by itself enough to justify the diagnosis, and that a "group delinquent reaction" of childhood or adolescence or "social maladjustment without manifest psychiatric disorder" should be ruled out first. The dyssocial personality type was relegated in the DSM-II to "dyssocial behavior" for individuals who are predatory and follow more or less criminal pursuits, such as racketeers, dishonest gamblers, prostitutes, and dope peddlers. (DSM-I classified this condition as *sociopathic personality disorder, dyssocial type*). It would later resurface as the name of a diagnosis in the ICD manual produced by the WHO, later spelled *dissocial personality disorder* and considered approximately equivalent to the ASPD diagnosis.[31]

The DSM-III in 1980 included the full term *antisocial personality disorder* and, as with other disorders, there was now a full checklist of symptoms focused on observable behaviors to enhance consistency in diagnosis between different psychiatrists ('inter-rater reliability'). The ASPD symptom list was based on the Research Diagnostic Criteria developed from the so-called Feighner Criteria from 1972, and in turn largely credited to influential research by sociologist Lee Robins published in 1966 as "Deviant Children Grown Up".[32] However, Robins has previously clarified that while the new criteria of prior childhood conduct problems came from her work, she and co-researcher psychiatrist Patricia O'Neal got the diagnostic criteria they used from Lee's husband the psychiatrist Eli Robins, one of the authors of the Feighner criteria who had been using them as part of diagnostic interviews.[33]

The DSM-IV maintained the trend for behavioral antisocial symptoms while noting "This pattern has also been referred to as psychopathy, sociopathy, or dyssocial personality disorder" and re-including in the 'Associated

Features' text summary some of the underlying personality traits from the older diagnoses. The DSM-5 has the same diagnosis of *antisocial personality disorder*. *The Pocket Guide to the DSM-5 Diagnostic Exam* suggests that a person with ASPD may present "with psychopathic features" if he or she exhibits "a lack of anxiety or fear and a bold, efficacious interpersonal style".

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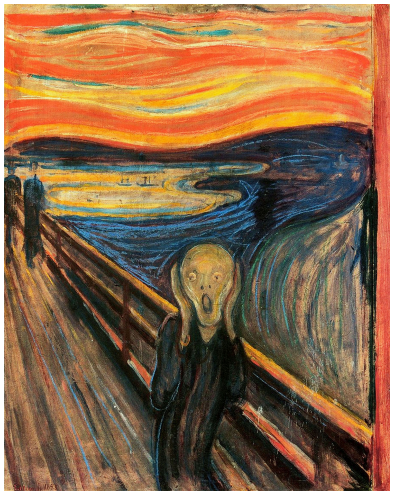
Further reading

- Millon, T.; Davis, R. (1998). "Ten Subtypes of Psychopathy". In Millon, T.; et al. *Psychopathy: Antisocial, Criminal and Violent Behavior*. New York. ISBN 1572303441.

External links

- "Into the Abyss." Article on street crime referencing the roots and consequences of sociopathic behavior (http://www.faculty.missouristate.edu/M/MichaelCarlie/what_I_learned_about/GANGS/WHYFORM/pathological.htm)
- DSM-IV-TR Criteria for Antisocial personality disorder (http://www.behavenet.com/antisocial-personality-disorder#301)
- Psychopathy and Antisocial Personality Disorder: A Case of Diagnostic Confusion (http://www.psychiatrictimes.com/display/article/10168/51816)
- Recent Studies Implicate Slow Monoamine Oxidase Enzyme/High Circulating T3 in Antisocial Behavior/Aggression/Violence 2007 (http://jrc.sagepub.com/cgi/content/abstract/28/2/227)

Anxiety disorder

Anxiety disorder	
Classification and external resources	
 *The Scream* (Norwegian: *Skrik*) an Expressionist painting by Norwegian artist Edvard Munch	
ICD-10	F40 [1]-F42 [2]
ICD-9	300 [3]
DiseasesDB	787 [4]
eMedicine	med/152 [5]
MeSH	D001008 [6]

Anxiety disorders are a group of mental disorders characterized by excessive feelings of anxiety and fear, where anxiety is worry about future events and fear is a reaction to current events. These feelings may cause physical symptoms such as a racing heart and shakiness. There are various forms of anxiety disorders, including generalized anxiety disorder, phobic disorder, and panic disorder. While each has its own characteristics and symptoms, they all include symptoms of anxiety.

Anxiety disorders are partly genetic but may also be due to drug use including alcohol and caffeine. They often occur with other mental disorders, particularly major depressive disorder, bipolar disorder, certain personality disorders, and eating disorders. The term anxiety covers four aspects of experiences that an individual may have: mental apprehension, physical tension, physical symptoms and dissociative anxiety.[7] The emotions present in anxiety disorders range from simple nervousness to bouts of terror. There are other psychiatric and medical problems that may mimic the symptoms of an anxiety disorder, such as hyperthyroidism.

Common treatment options include lifestyle changes, therapy, and medications. Medications are typically recommended only if other measures are not effective. Anxiety disorders occur about twice as often in females as males, and generally begin during childhood. As many as 18% of Americans and 14% of Europeans may be affected by one or more anxiety disorders.

Classification

Generalized anxiety disorder

Generalized anxiety disorder (GAD) is a common, chronic disorder characterized by long-lasting anxiety that is not focused on any one object or situation. Those suffering from generalized anxiety disorder experience non-specific persistent fear and worry, and become overly concerned with everyday matters. According to Schacter, Gilbert, and Wegner's book *Psychology: Second Edition*, generalized anxiety disorder is "characterized by chronic excessive worry accompanied by three or more of the following symptoms: restlessness, fatigue, concentration problems, irritability, muscle tension, and sleep disturbance".[8] Generalized anxiety disorder is the most common anxiety disorder to affect older adults. Anxiety can be a symptom of a medical or substance abuse problem, and medical professionals must be aware of this. A diagnosis of GAD is made when a person has been excessively worried about an everyday problem for six months or more. A person may find that he/she has problems making daily decisions and remembering commitments as a result of lack of concentration/preoccupation with worry.[9] Appearance looks strained, with increased sweating from the hands, feet, and axillae,[*citation needed*] and he/she may be tearful, which can suggest depression.[] Before a diagnosis of anxiety disorder is made, physicians must rule out drug-induced anxiety and other medical causes.[10]

In children GAD may be associated with headaches, restlessness, abdominal pain, and heart palpitations. Typically it begins around 8 to 9 years of age.

Phobias

The single largest category of anxiety disorders is that of phobic disorders, which includes all cases in which fear and anxiety is triggered by a specific stimulus or situation. Between 5% and 12% of the population worldwide suffer from phobic disorders. Sufferers typically anticipate terrifying consequences from encountering the object of their fear, which can be anything from an animal to a location to a bodily fluid to a particular situation. Sufferers understand that their fear is not proportional to the actual potential danger but still are overwhelmed by the fear.[11]

Panic disorder

With panic disorder, a person suffers from brief attacks of intense terror and apprehension, often marked by trembling, shaking, confusion, dizziness, nausea, and/or difficulty breathing. These panic attacks, defined by the APA as fear or discomfort that abruptly arises and peaks in less than ten minutes, can last for several hours. Attacks can be triggered by stress, fear, or even exercise; the specific cause is not always apparent.

In addition to recurrent unexpected panic attacks, a diagnosis of panic disorder requires that said attacks have chronic consequences: either worry over the attacks' potential implications, persistent fear of future attacks, or significant changes in behavior related to the attacks. As such, those suffering from panic disorder experience symptoms even outside specific panic episodes. Often, normal changes in heartbeat are noticed by a panic sufferer, leading them to think something is wrong with their heart or they are about to have another panic attack. In some cases, a heightened awareness (hypervigilance) of body functioning occurs during panic attacks, wherein any perceived physiological change is interpreted as a possible life-threatening illness (i.e., extreme hypochondriasis).

Agoraphobia

Agoraphobia is the specific anxiety about being in a place or situation where escape is difficult or embarrassing or where help may be unavailable.[12] Agoraphobia is strongly linked with panic disorder and is often precipitated by the fear of having a panic attack. A common manifestation involves needing to be in constant view of a door or other escape route. In addition to the fears themselves, the term agoraphobia is often used to refer to avoidance behaviors that sufferers often develop. For example, following a panic attack while driving, someone suffering from agoraphobia may develop anxiety over driving and will therefore avoid driving. These avoidance behaviors can often

have serious consequences.

Social anxiety disorder

Social anxiety disorder (SAD; also known as social phobia) describes an intense fear and avoidance of negative public scrutiny, public embarrassment, humiliation, or social interaction. This fear can be specific to particular social situations (such as public speaking) or, more typically, is experienced in most (or all) social interactions. Social anxiety often manifests specific physical symptoms, including blushing, sweating, and difficulty speaking. As with all phobic disorders, those suffering from social anxiety often will attempt to avoid the source of their anxiety; in the case of social anxiety this is particularly problematic, and in severe cases can lead to complete social isolation.

Obsessive–compulsive disorder

Obsessive–compulsive disorder (OCD) is a type of anxiety disorder primarily characterized by repetitive obsessions (distressing, persistent, and intrusive thoughts or images) and compulsions (urges to perform specific acts or rituals). It affects roughly 3% of the population worldwide. The OCD thought pattern may be likened to superstitions insofar as it involves a belief in a causative relationship where, in reality, one does not exist. Often the process is entirely illogical; for example, the compulsion of walking in a certain pattern may be employed to alleviate the obsession of impending harm. And in many cases, the compulsion is entirely inexplicable, simply an urge to complete a ritual triggered by nervousness.

In a slight minority of cases, sufferers of OCD may only experience obsessions, with no overt compulsions; a much smaller number of sufferers experience only compulsions.[13]

Post-traumatic stress disorder

Post-traumatic stress disorder (PTSD) is an anxiety disorder that results from a traumatic experience. Post-traumatic stress can result from an extreme situation, such as combat, natural disaster, rape, hostage situations, child abuse, bullying, or even a serious accident. It can also result from long term (chronic) exposure to a severe stressor, for example soldiers who endure individual battles but cannot cope with continuous combat. Common symptoms include hypervigilance, flashbacks, avoidant behaviors, anxiety, anger and depression. There are a number of treatments that form the basis of the care plan for those suffering with PTSD. Such treatments include cognitive behavioral therapy (CBT), psychotherapy and support from family and friends.

Separation anxiety

Separation anxiety disorder (SepAD) is the feeling of excessive and inappropriate levels of anxiety over being separated from a person or place. Separation anxiety is a normal part of development in babies or children, and it is only when this feeling is excessive or inappropriate that it can be considered a disorder.[14] Separation anxiety disorder affects roughly 7% of adults and 4% of children, but the childhood cases tend to be more severe; in some instances, even a brief separation can produce panic.

Situational anxiety

Situational anxiety is caused by new situations or changing events. It can also be caused by various events that make that particular individual uncomfortable. Its occurrence is very common. Often, an individual will experience panic attacks or extreme anxiety in specific situations. A situation that causes one individual to experience anxiety may not affect another individual, at all. For example, some people become uneasy in crowds or tight spaces, so standing in a tightly packed line, say at the bank or a store register, may cause them to experience extreme anxiety, possibly a panic attack.[15] Others, however, may experience anxiety when major changes in life occur, such as entering college, getting married, having children, etc.

Children

Children may experience anxiety disorders similar to adults.

A common anxiety disorder in children is school phobia, which in some cases can be a type of separation anxiety. Sometimes the anxiety has no obvious cause. In other instances, the child may experience bullying from classmates, or even a teacher. They could also be stressed from the workload they are given. School phobia may also be a form of social phobia, also known as social anxiety. Children with this disorder may avoid speaking in front of their classmates or meeting new people. Typically, social phobia in children is caused by some traumatic event, such as not knowing an answer when called on in class.[16]

The symptoms for both disorders are the same in children as they are in adults. If a child has GAD, they may worry about anything, even if it is seemingly minor. They long for attention, approval, and encouragement from others. The only difference is they are more likely to worry about things that relate to them. Those things may include, grades, bullies, getting hurt, storms, etc. The symptoms of OCD include repetitive and/or compulsive behaviors.[17]

Causes

Drugs

Anxiety and depression can be caused by alcohol abuse, which in most cases improves with prolonged abstinence. Even moderate, sustained alcohol use may increase anxiety levels in some individuals. Caffeine, alcohol and benzodiazepine dependence can worsen or cause anxiety and panic attacks. Anxiety commonly occurs during the acute withdrawal phase of alcohol and can persist for up to 2 years as part of a post-acute withdrawal syndrome, in about a quarter of people recovering from alcoholism. In one study in 1988–1990, illness in approximately half of patients attending mental health services at one British hospital psychiatric clinic, for conditions including anxiety disorders such as panic disorder or social phobia, was determined to be the result of alcohol or benzodiazepine dependence. In these patients, an initial increase in anxiety occurred during the withdrawal period followed by a cessation of their anxiety symptoms.

There is evidence that chronic exposure to organic solvents in the work environment can be associated with anxiety disorders. Painting, varnishing and carpet-laying are some of the jobs in which significant exposure to organic solvents may occur.

Ingestion of caffeine may cause or exacerbate anxiety disorders. A number of clinical studies have shown a positive association between caffeine and anxiogenic effects and/or panic disorder. Those with anxiety can have high caffeine sensitivity.

Stress

Anxiety disorders can arise in response to life stresses such as financial worries or chronic physical illness. Anxiety is also common among older people who have dementia. On the other hand, anxiety disorder is sometimes misdiagnosed among older adults when doctors misinterpret symptoms of a physical ailment (for instance, racing heartbeat due to cardiac arrhythmia) as signs of anxiety.

Genetics

GAD runs in families and is six times more common in the children of someone with the condition.

While anxiety arose as an adaptation, in modern times it is almost always thought of negatively in the context of anxiety disorders. People with these disorders have highly sensitive systems; hence, their systems tend to overreact to seemingly harmless stimuli. Sometimes anxiety disorders occur in those who have had traumatic youths, demonstrating an increased prevalence of anxiety when it appears a child will have a difficult future. In these cases, the disorder arises as a way to predict that the individual's environment will continue to pose threats.

Persistence of anxiety

At a low level, anxiety is not a bad thing. In fact, the hormonal response to anxiety has evolved as a benefit, as it helps humans react to dangers. Researchers in evolutionary medicine believe this adaptation allows humans to realize there is a potential threat and to act accordingly in order to ensure greatest possibility of protection. It has actually been shown that those with low levels of anxiety have a greater risk of death than those with average levels. This is because the absence of fear can lead to injury or death. Additionally, patients with both anxiety and depression were found to have lower morbidity than those with depression alone. The functional significance of the symptoms associated with anxiety includes: greater alertness, quicker preparation for action, and reduced probability of missing threats. In the wild, vulnerable individuals, for example those who are hurt or pregnant, have a lower threshold for anxiety response, making them more alert. This demonstrates a lengthy evolutionary history of the anxiety response.

Evolutionary mismatch

It has been theorized that high rates of anxiety are a reaction to how the social environment has changed from the Paleolithic era. For example, in the Stone Age there was greater skin-to-skin contact and more handling of babies by their mothers, both of which are strategies that reduce anxiety. Additionally, there is greater interaction with strangers in present times as opposed to interactions solely between close-knit tribes. Researchers posit that the lack of constant social interaction, especially in the formative years, is a driving cause of high rates of anxiety.

Many current cases are likely to have resulted from an evolutionary mismatch, which has been specifically been termed a "psychopathogical mismatch." In evolutionary terms, a mismatch occurs when an individual possesses traits that were adapted for an environment that differs from the individual's current environment. For example, even though an anxiety reaction may have been evolved to help with life-threatening situations, for highly sensitized individuals in Westernized cultures simply hearing bad news can elicit a strong reaction in sensitive individuals.

An evolutionary perspective may provide insight into alternatives to current clinical treatment methods for anxiety disorders. Simply knowing some anxiety is beneficial may alleviate some of the panic associated with mild conditions. Some researchers believe that, in theory, anxiety can be mediated by reducing a patient's feeling of vulnerability and then changing their appraisal of the situation.

Mechanisms

Biological

Low levels of GABA, a neurotransmitter that reduces activity in the central nervous system, contribute to anxiety. A number of anxiolytics achieve their effect by modulating the GABA receptors.

Selective serotonin reuptake inhibitors, the drugs most commonly used to treat depression, are frequently considered as a first line treatment for anxiety disorders.

People with obsessive-compulsive disorder (sometimes considered an anxiety disorder), have increased grey matter volumes in bilateral lenticular nuclei, extending to the caudate nuclei, while decreased grey matter volumes in bilateral dorsal medial frontal/anterior cingulate gyri. These findings contrast with those in people with other anxiety disorders, who have decreased (rather than increased) grey matter volumes in bilateral lenticular/caudate nuclei while also decreased grey matter volumes in bilateral dorsal medial frontal/anterior cingulate gyri. Alterations of circadian rhythms associated with obsessive-compulsive disorder have recently come into the focus of research.[18]

Amygdala

The amygdala is central to the processing of fear and anxiety, and its function may be disrupted in anxiety disorders. Sensory information enters the amygdala through the nuclei of the basolateral complex (consisting of lateral, basal, and accessory basal nuclei). The basolateral complex processes sensory-related fear memories and communicates their threat importance to memory and sensory processing elsewhere in the brain, such as the medial prefrontal cortex and sensory cortices.

Another important area is the adjacent central nucleus of the amygdala, which controls species-specific fear responses, via connections to the brainstem, hypothalamus, and cerebellum areas. In those with general anxiety disorder, these connections functionally seem to be less distinct, with greater gray matter in the central nucleus. Another difference is that the amygdala areas have decreased connectivity with the insula and cingulate areas that control general stimulus salience, while having greater connectivity with the parietal cortex and prefrontal cortex circuits that underlie executive functions.

The latter suggests a compensation strategy for dysfunctional amygdala processing of anxiety. Researchers have noted "Amygdalofrontoparietal coupling in generalized anxiety disorder patients may ... reflect the habitual engagement of a cognitive control system to regulate excessive anxiety." This is consistent with cognitive theories that suggest the use in this disorder of attempts to reduce the involvement of emotions with compensatory cognitive strategies.

Clinical and animal studies suggest a correlation between anxiety disorders and difficulty in maintaining balance. A possible mechanism is malfunction in the parabrachial area, a brain structure that, among other functions, coordinates signals from the amygdala with input concerning balance.

Anxiety processing in the basolateral amygdala has been implicated with dendritic arborization of the amygdaloid neurons. SK2 potassium channels mediate inhibitory influence on action potentials and reduce arborization. By overexpressing SK2 in the basolateral amygdala, anxiety in experimental animals can be reduced together with general levels of stress-induced corticosterone secretion.

Prevention

Focus is increasing on prevention of anxiety disorders. There is tentative evidence to support the use of cognitive behavior therapy. As of 2013 there are no effective measures to prevent GAD in adults.

Diagnosis

Anxiety disorders are often severe chronic conditions, which can be present from an early age or begin suddenly after a triggering event. They are prone to flare up at times of high stress and are frequently accompanied by physiological symptoms such as headache, sweating, muscle spasms, tachycardia, palpitations, and hypertension, which in some cases lead to fatigue or exhaustion.

In casual discourse the words "anxiety" and "fear" are often used interchangeably; in clinical usage, they have distinct meanings: "anxiety" is defined as an unpleasant emotional state for which the cause is either not readily identified or perceived to be uncontrollable or unavoidable, whereas "fear" is an emotional and physiological response to a recognized external threat. The term "anxiety disorder" includes fears (phobias) as well as anxieties.

Standardized screening clinical questionnaires such as the Taylor Manifest Anxiety Scale or the Zung Self-Rating Anxiety Scale can be used to detect anxiety symptoms, and suggest the need for a formal diagnostic assessment of anxiety disorder.

Anxiety disorders are often occur along with other mental disorders, in particular depression, which may occur in as many as 60% of people with anxiety disorders. The fact that there is considerable overlap between symptoms of anxiety and depression, and that the same environmental triggers can provoke symptoms in either condition, may help to explain this high rate of comorbidity.

Studies have also indicated that anxiety disorders are more likely among those with family history of anxiety disorders, especially certain types.

Sexual dysfunction often accompanies anxiety disorders, although it is difficult to determine whether anxiety causes the sexual dysfunction or whether they arise from a common cause. The most common manifestations in individuals with anxiety disorder are avoidance of intercourse, premature ejaculation or erectile dysfunction among men and pain during intercourse among women. Sexual dysfunction is particularly common among people affected by panic disorder (who may fear that a panic attack will occur during sexual arousal) and posttraumatic stress disorder.

Treatment

Treatment options available include lifestyle changes, therapy, and medications. Medication are only recommended if other measures are not effective. Stopping smoking has benefits in anxiety as large as or larger than those of medications.

Therapy

Cognitive behavioral therapy (CBT) is effective for anxiety disorders. CBT, as its name suggests, has two main components: cognitive and behavioral. In cases of social anxiety, the cognitive component can help the person question how they can be so sure that others are continually watching and harshly judging him or her. The behavioral component seeks to change people's reactions to anxiety-provoking situations.

CBT appears to be equally effective when carried out via the internet.

As such it serves as a logical extension of cognitive therapy, whereby people are shown proof in the real world that their dysfunctional thought processes are unrealistic. A key element of this component is gradual exposure, in which the patient is confronted by the things they fear in a structured, sensitive manner. Gradual exposure is an inherently unpleasant technique; ideally it involves exposure to a feared social situation that is anxiety provoking but bearable, for as long as possible, two to three times a week. Often, a hierarchy of feared steps is constructed and the patient is exposed to each step sequentially.

The aim is to learn from acting differently and observing reactions. This is intended to be done with support and guidance, and when the therapist and patient feel they are ready. Cognitive-behavioral therapy for social phobia also includes anxiety management training, which may include techniques such as deep breathing and muscle relaxation exercises, which may be practiced 'in-situ'. CBT can also be conducted partly in group sessions, facilitating the sharing of experiences, a sense of acceptance by others and undertaking behavioral challenges in a trusted environment (Heimberg).

Some studies have suggested social skills training can help with social anxiety.[19] However, it is not clear whether specific social skills techniques and training are required, rather than just support with general social functioning and exposure to social situations.[20] Additionally, a recent study has suggested that interpersonal therapy, a form of psychotherapy primarily used to treat depression, may also be effective in the treatment of social phobia.[21]

In social phobia a specific form of short-term CBT, the central component being gradual exposure therapy. Self-help books can contribute to the treatment of people with anxiety disorders.

Medications

Medication are only indicated if other measures have not been found to be effective or a person is not interested in trying them. If medications are used SSRIs are recommended as first-line agents. Benzodiazepines are also sometimes indicated for short-term or "as needed" use. They are usually considered second-line due to disadvantages such as cognitive impairment and risks of dependence. MAOIs such as phenelzine and tranylcypromine are considered an effective treatment and are especially useful in treatment-resistant cases, however, dietary restrictions and medical interactions may limit their use. Pregabalin may be effective. In children and adolescents, when a

medication option is warranted, antidepressants such as SSRIs, SNRIs as well as tricyclic antidepressants can be effective. Buspar is not effective in children and adolescents who have an anxiety disorder.

These medications need to be used with care among older adults, who are more likely to have side effects because of coexisting physical disorders. Adherence problems are more likely among elderly patients, who may have difficulty understanding, seeing, or remembering instructions.

The effectiveness and increased suicide risk of SSRIs has been subject to controversy. General side effects are common and may include headaches, nausea, insomnia, and changes in sexual behavior. Treatment safety during pregnancy has not been established. In late 2004 much media attention was given to a proposed link between SSRI use and suicide. For this reason, the use of SSRIs in pediatric cases of depression is recognized by the Food and Drug Administration as warranting a cautionary statement to the parents of children who may be prescribed SSRIs.[22]

Other drugs

The atypical antipsychotic quetiapine appears effective in generalized anxiety disorder, however rates of adverse effects is greater than that with SSRIs. Evidence for risperidone and olanzapine is not enough to make any comments. For OCD the evidence for risperidone and quetiapine is tentative with insufficient evidence for olanzapine.

Benzodiazepines are an alternative to SSRIs. These drugs are often used for short-term relief of severe, disabling anxiety. Although benzodiazepines are still sometimes prescribed for long-term everyday use, there is concern over the development of drug tolerance, dependency and recreational abuse. It has been recommended that benzodiazepines only be considered for individuals who fail to respond to safer medications. Effects usually begin to appear within minutes or hours. Benzodiazepines are not however, effective in the treatment of children and adolescents who have an anxiety disorder.

Some people with a form of social phobia called performance phobia have been helped by beta-blockers. Taken in low doses, they control the physical manifestation of anxiety and can be taken before a public performance.

Treatment controversy arises because while some studies indicate that a combination of medication and psychotherapy can be more effective than either one alone, others suggest pharmacological interventions are largely palliative, and can actually interfere with the mechanisms of successful therapy. Psychotherapeutic interventions have better long-term efficacy compared to pharmacotherapy.

Caffeine

Caffeine can cause anxiety, along with more minor effects, such as muscle twitchings, hand tremors, and headaches. The best way to prevent caffeinism is to either wean off of caffeine completely or reduce consumption.[23] For some people, anxiety can be reduced by coming off caffeine. Anxiety can temporarily increase during caffeine withdrawal.

Alternative medicine

Regular exercise, and reducing caffeine are often useful in treating anxiety. There is tentative evidence that yoga may be effective. Evidence is insufficient regarding meditation to make any conclusions.

Many other remedies have been used for anxiety disorder. These include kava, where the potential for benefit seems greater than that for harm with short-term use in those with mild to moderate anxiety. The American Academy of Family Physicians (AAFP) recommends use of kava for those with mild to moderate anxiety disorders who are not using alcohol or taking other medicines metabolized by the liver, but who wish to use "natural" remedies. Side effects of kava in the clinical trials were rare and mild.

Inositol has been found to have modest effects in people with panic disorder or obsessive-compulsive disorder. There is insufficient evidence to support the use of St. John's wort, valerian or passionflower.

Children

Several methods of treatment have been found to be effective in treating childhood anxiety disorders. Like adults, children may undergo psychotherapy, cognitive-behavioral therapy, or counseling. They may still be given medication such as SSRIs, but in much smaller doses. However, administering potent medications like antidepressants to children is controversial. As a result, other forms of treatment have become increasingly popular. Family therapy is a form of treatment in which the child meets with a therapist together with the primary guardians and siblings. Each family member may attend individual therapy, but family therapy is typically a form of group therapy. Art and play therapy are also used. Art therapy is most commonly used when the child will not or cannot verbally communicate, due to trauma or a disability in which they are nonverbal. Participating in art activities allows the child to express what they otherwise may not be able to communicate to others.[24] In play therapy, the child is allowed to play however they please as a therapist observes them. The therapist may intercede from time to time with a question, comment, or suggestion. This is often most effective when the family of the child plays a significant role in the treatment.[25]

Prognosis

The prognosis varies on the severity of each case and utilization of treatment for each individual. It is the most common cause of disability in the workplace in the United States.

If these children are left untreated, they face risks such as poor results at school, avoidance of important social activities, and substance abuse. Children who have an anxiety disorder are likely to have other disorders such as depression, eating disorders, attention deficit disorders both hyperactive and inattentive.

Epidemiology

Globally as of 2010 approximately 273 million (4.5% of the population) had an anxiety disorder. It is more common in females (5.2%) than males (2.8%). In Europe, Africa and Asia, lifetime rates of anxiety disorders are between 9 and 16%, and yearly rates are between 4 and 7%. In the United States, the lifetime prevalence of anxiety disorders is about 29% and between 11 and 18% of adults have the condition in a given year.

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External links

- Support Group Providers for Anxiety disorder (http://www.dmoz.org/Health/Mental_Health/Disorders/Anxiety/Social_Anxiety/Support_Groups/) on the Open Directory Project

Attention deficit hyperactivity disorder

Attention deficit hyperactivity disorder	
Classification and external resources	
 Children with ADHD find it more difficult to focus and to complete their schoolwork.	
ICD-10	F90 [1]
ICD-9	314.00 [2], 314.01 [3]
OMIM	143465 [4]
DiseasesDB	6158 [5]
MedlinePlus	001551 [6]
eMedicine	med/3103 [7] ped/177 [8]
MeSH	D001289 [9]

Attention deficit hyperactivity disorder (**ADHD**, similar to **hyperkinetic disorder** in the ICD-10) is a psychiatric disorder of the neurodevelopmental type in which there are significant problems of attention, hyperactivity, or acting impulsively that are not appropriate for a person's age. These symptoms must begin by age six to twelve and be present for more than six months for a diagnosis to be made. In school-aged individuals the lack of focus may result in poor school performance.

Despite being the most commonly studied and diagnosed psychiatric disorder in children and adolescents, the cause in the majority of cases is unknown. It affects about 6–7% of children when diagnosed via the DSM-IV criteria and 1–2% when diagnosed via the ICD-10 criteria.[10] Rates are similar between countries and depend mostly on how it is diagnosed. ADHD is diagnosed approximately three times more in boys than in girls. About 30–50% of people diagnosed in childhood continue to have symptoms into adulthood and between 2–5% of adults have the condition. The condition can be difficult to tell apart from other disorders as well as that of high normal activity.

ADHD management usually involves some combination of counseling, lifestyle changes, and medications. Medications are only recommended as a first-line treatment in children who have severe symptoms and may be considered for those with moderate symptoms who either refuse or fail to improve with counseling.[:p.317] Long term effects of medications are not clear and they are not recommended in preschool-aged children. Adolescents and adults tend to develop coping skills which make up for some or all of their impairments.

ADHD and its diagnosis and treatment have been considered controversial since the 1970s. The controversies have involved clinicians, teachers, policymakers, parents and the media. Topics include ADHD's causes, and the use of stimulant medications in its treatment. Most healthcare providers accept ADHD as a genuine disorder with debate in

the scientific community mainly around how it is diagnosed and treated.

Signs and symptoms

Inattention, hyperactivity (restlessness in adults), disruptive behavior, and impulsivity are common in ADHD. Academic difficulties are frequent as are problems with relationships. The symptoms can be difficult to define as it is hard to draw a line at where normal levels of inattention, hyperactivity, and impulsivity end and significant levels requiring interventions begin.[:p.26]

To be diagnosed per the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-V), symptoms must be observed in two different settings for six months or more and to a degree that is greater than other children of the same age.

Based on the presenting symptom ADHD can be divided into three subtypes—predominantly inattentive, predominantly hyperactive-impulsive, or combined if criteria for both types are met.[:p.4]

An individual with inattention may have some or all of the following symptoms:

- Be easily distracted, miss details, forget things, and frequently switch from one activity to another
- Have difficulty maintaining focus on one task
- Become bored with a task after only a few minutes, unless doing something enjoyable
- Have difficulty focusing attention on organizing and completing a task or learning something new
- Have trouble completing or turning in homework assignments, often losing things (e.g., pencils, toys, assignments) needed to complete tasks or activities
- Not seem to listen when spoken to
- Daydream, become easily confused, and move slowly
- Have difficulty processing information as quickly and accurately as others
- Struggle to follow instructions

An individual with hyperactivity may have some or all of the following symptoms:

- Fidget and squirm in their seats
- Talk nonstop
- Dash around, touching or playing with anything and everything in sight
- Have trouble sitting still during dinner, school, doing homework, and story time
- Be constantly in motion
- Have difficulty doing quiet tasks or activities

Note: These hyperactivity symptoms tend to go away with age and turn into "inner restlessness" in teens and adults with ADHD.

An individual with impulsivity may have some or all of the following symptoms:

- Be very impatient
- Blurt out inappropriate comments, show their emotions without restraint, and act without regard for consequences
- Have difficulty waiting for things they want or waiting their turns in games
- Often interrupts conversations or others' activities

People with ADHD more often have difficulties with social skills, such as social interaction and forming and maintaining friendships. About half of children and adolescents with ADHD experience social rejection by their peers compared to 10–15% of non-ADHD children and adolescents. People with ADHD have attention deficits which cause difficulty processing verbal and nonverbal language which can negatively affect social interaction. They also may drift off during conversations, and miss social cues.

Difficulties managing anger are more common in children with ADHD as are poor handwriting and delays in speech, language and motor development. Although it causes significant impairment, particularly in modern society, many children with ADHD have a good attention span for tasks they find interesting.

Associated disorders

In children ADHD occurs with other disorders about ⅔ of the time. Some of the commonly associated conditions include:

- Learning disabilities have been found to occur in about 20–30% of children with ADHD. Learning disabilities can include developmental speech and language disorders and academic skills disorders. ADHD, however, is not considered a learning disability but it can still significantly impact academic performance.
- Tourette syndrome has been found to occur more commonly in the ADHD population.
- Oppositional defiant disorder (ODD) and conduct disorder (CD), which occur with ADHD in about 50% and 20% of cases respectively. They are characterized by antisocial behaviors such as stubbornness, aggression, frequent temper tantrums, deceitfulness, lying, and stealing. About half of those with hyperactivity and ODD or CD develop antisocial personality disorder in adulthood. Brain imaging supports that conduct disorder and ADHD are separate conditions.
- Primary disorder of vigilance, which is characterized by poor attention and concentration, as well as difficulties staying awake. These children tend to fidget, yawn and stretch and appear to be hyperactive in order to remain alert and active.
- Mood disorders (especially bipolar disorder and major depressive disorder). Boys diagnosed with the combined ADHD subtype are more likely to have a mood disorder. Adults with ADHD sometimes also have bipolar disorder, which requires careful assessment to accurately diagnose and treat both conditions.
- Anxiety disorders have been found to occur more commonly in the ADHD population.
- Obsessive-compulsive disorder (OCD) can co-occur with ADHD and shares many of its characteristics.
- Substance use disorders. Adolescents and adults with ADHD are at increased risk of developing a substance use problem. This is most commonly with alcohol or cannabis. The reason for this may be due to an altered reward pathway in the brains of ADHD individuals. This makes the evaluation and treatment of ADHD more difficult, with serious substance misuse problems usually treated first due to their greater risks.[:p.38]
- Restless legs syndrome has been found to be more common in those with ADHD and is often due to iron deficiency anaemia. However, restless legs can simply be a part of ADHD and requires careful assessment to differentiate between the two disorders.
- Sleep disorders and ADHD commonly co-exist. They can also occur as a side effect of medications used to treat ADHD. In children with ADHD, insomnia is the most common sleep disorder with behavioral therapy the preferred treatment. Problems with sleep initiation are common among individuals with ADHD but often they will be deep sleepers and have significant difficulty getting up in the morning. Melatonin is sometimes used in children who have sleep onset insomnia.

There is an association with persistent bed wetting, language delay, and developmental coordination disorder (DCD), with about half of people with DCD having ADHD. The language delay in people with ADHD can include problems with auditory processing disorders such as short-term auditory memory weakness, difficulty following instructions, slow speed of processing written and spoken language, difficulties listening in distracting environments e.g. the classroom, and weakness in reading comprehension.[11]

Cause

The cause of most cases of ADHD is unknown; however, it is believed to involve interactions between genetic and environmental factors. Certain cases are related to previous infection of or trauma to the brain.

Genetics

Twin studies indicate that the disorder is often inherited from one's parents with genetics determining about 75% of cases. Genetic factors are also believed to be involved in determining whether or not ADHD persists into adulthood.

Typically a number of genes are involved, many of which directly affect dopamine neurotransmission. Those involved with dopamine include: DAT1, DRD4, DRD5, TAAR1, MAOA, COMT, and DBH. Other genes associated with ADHD include: 5HTT, HTR1B, SNAP25, GRIN2A, ADRA2A, TPH2, and BDNF. A common variant of a gene called LPHN3 is estimated to be responsible for about 9% of cases and when this gene is present, people are particularly responsive to stimulant medication.

Natural selection may have favored the traits of ADHD as, at least individually, they may have provided a survival advantage, becoming dysfunctional only when combined. Additionally, some women may be more attracted to males who are risk takers, increasing the frequency of genes that predispose to ADHD in the gene pool. As it is more common in children of anxious or stressed mothers, some argue that ADHD is an adaptation that helps children face a stressful or dangerous environment with, for example, increased impulsivity and exploratory behavior.

Hyperactivity might have been beneficial, from an evolutionary perspective, in situations involving risk, competition, or unpredictable behavior (i.e. exploring new areas or finding new food sources). In these situations, ADHD could have been beneficial to society as a whole even while being harmful to the individual. Additionally, in certain environments it may have offered advantages to the individuals themselves, such as quicker response to predators or superior hunting skills.

Environment

Environmental factors are believed to play a lesser role. Alcohol intake during pregnancy can cause fetal alcohol spectrum disorder which can include symptoms similar to ADHD. Exposure to tobacco smoke during pregnancy can cause problems with central nervous system development and can increase the risk of ADHD. Many children exposed to tobacco do not develop ADHD or only have mild symptoms which do not reach the threshold for a diagnosis. A combination of a genetic predisposition with tobacco exposure may explain why some children exposed during pregnancy may develop ADHD and others do not. Children exposed to lead, even low levels, or polychlorinated biphenyls may develop problems which resemble ADHD and fulfill the diagnosis. Exposure to the organophosphate insecticides chlorpyrifos and dialkyl phosphate is associated with an increased risk; however, the evidence is not conclusive.

Very low birth weight, premature birth and early adversity also increase the risk as do infections during pregnancy, at birth, and in early childhood. These infections include among others: various viruses (measles, varicella, rubella, enterovirus 71) and streptococcal bacterial infection. At least 30% of children with a traumatic brain injury later develop ADHD and about 5% of cases are due to brain damage.

A small number of children may react negatively to food dyes or preservatives. It is possible that certain food coloring may act as a trigger in those who are genetically predisposed. The United Kingdom and European Union have put in place regulatory measures based on these concerns. Dietary sugar and the artificial sweetener aspartame appears to have little to no effect, except possibly in children under six years of age where sugar may increase inattention.

Society

The diagnosis of ADHD can represent family dysfunction or a poor educational system rather than an individual problem. Some cases may be explained by increasing academic expectations, with a diagnosis being a method for parents in some countries to get extra financial and educational support for their child. The youngest children in a class have been found to be more likely to be diagnosed as having ADHD possibly due to their being developmentally behind their older classmates. Behavior typical of ADHD occur more commonly in children who have experienced violence and emotional abuse.

Per social construction theory it is societies that determine the boundary between normal and abnormal behavior. Members of society: including physicians, parents, and teachers determine which diagnostic criteria are used and, thus, the number of people affected. This leads to the current situation were the DSM-IV arriving at levels of ADHD three to four times higher than those obtained with the ICD 10. Thomas Szasz, a supporter of this theory, has argued that ADHD was "invented and not discovered."

Pathophysiology

Brain structure

The pathophysiology of ADHD is unclear with there being a number of competing explanations. In children with ADHD there is a general reduction of brain volume, with a proportionally greater decrease in the volume in the left-sided prefrontal cortex. The brain pathways connecting the prefrontal cortex and the striatum also appears to be involved. This suggest that inattention, hyperactivity, and impulsivity may reflect frontal lobe dysfunction, with addition brain regions such as the cerebellum also being implicated. Other brain systems related to attention have also been found to differ between people with and without ADHD.

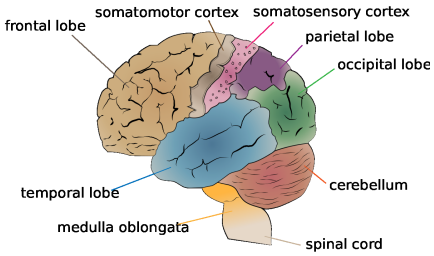


Diagram of the human brain

Neurotransmitters

Previously it was thought that the elevated number of dopamine transporters in people with ADHD was part of the pathophysiology but it appears that the elevated numbers are due to adaptation to exposure to stimulants. People with ADHD may have a low arousal threshold and compensate for this with increased stimuli, which in turn results in disruption of attention and increases hyperactive behavior. The reason for this is due to abnormalities in how the dopamine system responds to stimulation. There may additionally be abnormalities in the adrenergic, serotoninergic and cholinergic or nicotinergic pathways. Glutaminergic neurotransmission seems to be also involved.

Executive function

One theory of suggests that the symptoms arise from a difficulty in executive functions. Executive functions refers to a number of mental processes that are required to regulate, control, and manage daily life tasks. Some of these impairments include: problems with organizational skills, time keeping, excessive procrastination, concentration problems, processing speed, regulating emotions, using working memory and short-term memory problems. People usually have decent long-term memory. The criteria for an executive function deficit are met in 30–50% of children and adolescents with ADHD. One study found that 80% of individuals with ADHD were impaired in at least one EF task, compared to 50% for individuals without ADHD. Due to the rates of brain maturation and the increasing demands for executive control as a person gets older ADHD impairments may not fully manifest themselves until adolescence or even early adulthood.

Diagnosis

ADHD is diagnosed by an assessment of a person's childhood behavioral and mental development, including ruling out the effects of drugs, medications and other medical or psychiatric problems as explanations for the symptoms.[:p.19–27] It often takes into account feedback from parents and teachers with most diagnoses begun after a teacher raises concerns. It may be viewed as the extreme end of one or more continuous human traits found in all people.[:p.130] Whether someone responds to medications does not confirm or rule out the diagnosis. As imaging studies of the brain do not give consistent results between individuals, they are only used for research purposes and not diagnosis.

In North America, the DSM-IV or DSM-V criteria are often used for diagnosis, while European countries usually use the ICD-10. With the DSM-IV criteria a diagnosis of ADHD is 3–4 times more likely than with the ICD-10 criteria. It is classified as a psychiatric disorder of the neurodevelopmental disorder type. Additionally it is classified as a disruptive behavior disorder along with oppositional defiant disorder, conduct disorder and antisocial personality disorder. A diagnosis does not imply a neurological disorder.

Associated conditions that should be screened for include anxiety, depression, oppositional defiant disorder, conduct disorder, and learning and language disorders. Other conditions that should be considered are other neurodevelopmental disorders, tics, and sleep apnea.

Diagnostic and Statistical Manual

As with many other psychiatric disorders, formal diagnosis is made by a qualified professional based on a set number of criteria. In the United States these criteria are defined by the American Psychiatric Association in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM). Based on the DSM-criteria, there are three sub types of ADHD:

1. ADHD predominantly inattentive type (ADHD-PI) presents with symptoms including being easily distracted, forgetful, daydreaming, disorganization, poor concentration, and difficulty completing tasks. Often people refer to ADHD-PI as "attention deficit disorder" (ADD), however, the latter has not been officially accepted since the 1994 revision of the DSM.
2. ADHD, predominantly hyperactive-impulsive type presents with excessive fidgetiness and restlessness, hyperactivity, difficulty waiting and remaining seated, immature behavior; destructive behaviors may also be present.
3. ADHD, combined type is a combination of the two other subtypes.

This subdivision is based on presence of at least six out of nine long-term (lasting at least six months) symptoms of inattention, hyperactivity–impulsivity, or both. To be considered, the symptoms must have appeared by the age of six to twelve and occur in more than one environment (e.g. at home and at school or work). The signs must be not appropriate for a child of that age and there must be evidence that it is causing social, school or work related problems.

Most children with ADHD have the combined type. Children with the inattention subtype are less likely to act out or have difficulties getting along with other children. They may sit quietly, but without paying attention resulting in the child difficulties being overlooked.

International Classification of Diseases

In the tenth edition of the *International Statistical Classification of Diseases and Related Health Problems* (ICD-10) the signs of ADHD are given the name "hyperkinetic disorders". When a conduct disorder (as defined by ICD-10) is present, the condition is referred to as *hyperkinetic conduct disorder*. Otherwise the disorder is classified as *disturbance of activity and attention*, *other hyperkinetic disorders* or *hyperkinetic disorders, unspecified*. The latter is sometimes referred to as, *hyperkinetic syndrome*.

Adults

Adults with ADHD are diagnosed under the same criteria, including that their signs must have been present by the age of six to twelve. Questioning parents or guardians as to how the person behaved and developed as a child may form part of the assessment; a family history of ADHD also adds weight to a diagnosis. While the core symptoms of ADHD are similar in children and adults they often present differently in adults than in children, for example excessive physical activity seen in children may present as feelings of restlessness and constant mental activity in adults.

Differential

ADHD symptoms which maybe related to other disorders		
Depression	**Anxiety disorder**	**Mania**
• Feelings of guilt, hopelessness, low self-esteem, or unhappiness • Loss of interest in hobbies, regular activities, sex, or work • Fatigue • Too little, poor, or excessive sleep • Difficulty paying attention • Changes in appetite • Irritability • Low tolerance for stress • Thoughts of suicide • Unexplained pain	• Worry or a persistent feeling of anxiety • Irritability • Inability to relax • Being hyperalert • Tires easily • Low tolerance for stress • Difficulty paying attention	• Excessive happiness • Hyperactivity • Racing thoughts • Aggression • Excessive talking • Grandiose delusions • Decreased need for sleep • Inappropriate social behavior • Difficulty paying attention

Symptoms of ADHD such as low mood and poor self-image, mood swings, and irritability can be confused with dysthymia, cyclothymia or bipolar disorder as well as with borderline personality disorder. Some of the symptoms that are due to anxiety disorders, antisocial personality disorder, developmental disabilities or mental retardation or the effects of substance abuse such as intoxication and withdrawal can overlap with some ADHD. These disorders can also sometimes occur along with ADHD. Medical conditions which can cause ADHD type symptoms include: hyperthyroidism, seizure disorder, lead toxicity, hearing deficits, hepatic disease, sleep apnea, drug interactions, and head injury.

Primary sleep disorders may affect attention and behavior and the symptoms of ADHD may affect sleep. It is thus recommended that children with ADHD be regularly assessed for sleep problems. Sleepiness in children may result in symptoms ranging from the classic ones of yawning and rubbing the eyes, to impulsivity, hyperactivity, aggressiveness, mood swing and inattentiveness. Obstructive sleep apnea, can also cause ADHD type symptoms.

Management

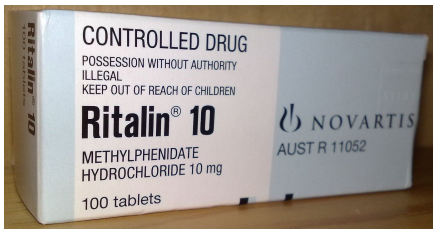
The management of ADHD typically involves counseling or medications either alone or in combination. While treatment may improve long term outcomes it does not get rid of negative outcomes entirely. Medications used include stimulants, atomoxetine, alpha-adrenergic agonists and sometimes antidepressants. They have at least some effect in about 80% of people. Dietary modifications may also be of benefit with evidence supporting free fatty acids and reduced exposure to food coloring. Removing other foods from the diet is not currently supported by the evidence.

Psychosocial

There is good evidence for the use of behavioral therapies in ADHD and they are the recommended first line treatment in those who have mild symptoms or are preschool-aged. Psychological therapies used include: psychoeducational input, behavior therapy, cognitive behavioral therapy (CBT), interpersonal psychotherapy, family therapy, school-based interventions, social skills training, parent management training, and neurofeedback. Parent training and education have been found to have short-term benefits. There is little high quality research on the effectiveness of family therapy for ADHD, but the evidence that exists shows that it's similar to community care and better than a placebo. Several ADHD specific support groups exist as informational sources and may help families cope with ADHD.

Training in social skills, behavioral modification and medication may have some limited beneficial effects. The most important factor in reducing later psychological problems, such as major depression, criminality, school failure, and substance use disorders is formation of friendships with people who are not involved in delinquent activities.

Medication



methylphenidate (Ritalin) 10 mg tablets

Stimulant medications are the pharmaceutical treatment of choice and improve symptoms, at least in the short term. There are a number of non-stimulant medications, such as atomoxetine, bupropion, guanfacine and clonidine that may be used as alternatives. There are no good studies comparing the various medications; however, they appear more or less equal with respect to side effects. Stimulants but not atomoxetine appear to improve academic performance. There is little evidence on their effects on social behaviors. Medications are not recommended for preschool children, as the long-term effects in this age group are not known. The long-term effects of stimulants generally are unclear with one study finding benefit, another finding no benefit and a third finding evidence of harm. Their long term use does; however, appear to normalize brain structure. Atomoxetine, due to its lack of abuse potential, may be preferred in those who are at risk of abusing stimulant medication. Guidelines on when to use medications vary by country, with the United Kingdom's National Institute of Clinical Excellence recommending use only in severe cases, while most United States guidelines recommend medications in nearly all cases.

While stimulants and atomoxetine are usually safe, there are side-effects and contraindications to their use. Stimulants may result in psychosis or mania; however, this is relatively uncommon. Regular monitoring has been recommended in those on long term treatment. Stimulant therapy should be stopped from time to time to assess for continuing need for medication. Stimulant medications have the potential for abuse and dependence and while people with ADHD have an increased risk of substance abuse, the use of stimulants generally appears to either reduce this risk or have no effect on it. The safety of these medication in pregnancy is unclear.

Zinc deficiency has been associated with inattentive symptoms and there is evidence that zinc supplementation can benefit children with ADHD who have low zinc levels. Iron, magnesium and iodine may also have an effect on ADHD symptoms. There is evidence of a modest benefit of omega 3 supplementation, but it is not recommended in place of traditional medication.

Prognosis

An 8-year follow up of children diagnosed with ADHD (combined type) found that they often have difficulties in adolescence, regardless of treatment or lack thereof. In the US, less than 5% of individuals with ADHD get a college degree, compared to 28% of the general population aged 25 years and older. The proportion of children meeting criteria for ADHD drops by about half in the three years following the diagnosis and this occurs regardless of treatments used. ADHD persists into adulthood in about 30–50% of cases. Those affected are likely to develop coping mechanisms as they mature, thus compensating for their previous symptoms.

Epidemiology

ADHD is estimated to affect about 6–7% of people aged 18 and under when diagnosed via the DSM-IV criteria. When diagnosed via the ICD-10 criteria rates in this age group are estimated at 1–2%. Children in North America appear to have a higher rate of ADHD than children in Africa and the Middle East; this is believed to be due to differing methods of diagnosis rather than a difference in underlying frequency. If the same diagnostic methods are used rates are more or less the same between countries. It is diagnosed approximately three times more often in boys than in girls. This difference between sexes may reflect either a difference in susceptibility or that females with ADHD are less likely to be diagnosed than males.

Rates of diagnosis and treatment have increased in both the United Kingdom and the United States since the 1970s. This is believed to be primarily due to changes in how the condition is diagnosed and how readily people are willing to treat it with medications rather than a true change in how common the condition is. It is believed that changes to the diagnostic criteria in 2013 with the release of the DSM V will increase the percentage of people with ADHD especially among adults.

History

Hyperactivity has long been part of the human condition. Sir Alexander Crichton describes "mental restlessness" in his book *An inquiry into the nature and origin of mental derangement* written in 1798. ADHD was first clearly described by George Still in 1902. The terminology used to describe the condition has changed over time and has included: in the DSM-I (1952) "minimal brain dysfunction", in the DSM-II (1968) "hyperkinetic reaction of childhood", in the DSM-III (1980) "attention-deficit disorder (ADD) with or without hyperactivity". In 1987 this was changed to ADHD in the DSM-III-R and the DSM-IV in 1994 split the diagnosis into three subtypes, ADHD inattentive type, ADHD hyperactive-impulsive type and ADHD combined type. These terms were kept in the DSM-V in 2013. Other terms have included "minimal brain damage" used in the 1930s.

The use of stimulants to treat ADHD was first described in 1937. In the 1930s, Benzedrine became the first amphetamine medication approved for use in the United States. Methylphenidate was introduced in the 1950s, and enantiopure dextroamphetamine in the 1970s.

Society and culture

Controversies

ADHD and its diagnosis and treatment have been considered controversial since the 1970s. The controversies have involved clinicians, teachers, policymakers, parents and the media. Positions regarding ADHD range from believing it is simply the far end of a normal range of behavior[:p.23] to considering that it is the result of an underlying genetic condition. Other areas of controversy include the use of stimulant medications and specifically their use in children, as well as the method of diagnosis and the possibility of overdiagnosis. The National Institute for Clinical Excellence, while acknowledging the controversy, states that the current treatments and methods of diagnosis are based on the dominant view of the academic literature.[:p.133]

With widely differing rates of diagnosis across countries, states within countries, races, and ethnicities, some suspect factors other than the presence of the symptoms of ADHD are playing a role in diagnosis. Some sociologists consider ADHD to be an example of the medicalization of deviant behavior, or in other words, the turning of the previously non medical issue of school performance into a medical one. Most healthcare providers accept ADHD as a genuine disorder, at least in the small number of people with severe symptoms. Among healthcare providers the debate mainly centers around diagnosis and treatment in the much larger number of people with less severe symptoms.

As of 2009[12], 8% of all United States Major League Baseball players had been diagnosed with ADHD, making the disorder common among this population. The increase coincided with the League's 2006 ban on stimulants, which has raised concern that some players are mimicking or falsifying the symptoms or history of ADHD to get around the ban on the use of stimulants in sport.

Media commentary

A number of notable individuals have given controversial statements regarding ADHD. Tom Cruise has referred to the medications Ritalin and Adderall as "street drugs". Ushma S. Neill criticized this view, stating that the doses of stimulants used in the treatment of ADHD do not cause behavioral addiction and that there is some evidence of a reduced risk of later substance addiction in children treated with stimulants. In England, Susan Greenfield spoke out publicly in 2007 in the House of Lords about the need for a wide-ranging inquiry into the dramatic increase in the diagnosis of ADHD in the UK and possible causes. Her comments followed a BBC Panorama program that highlighted research that suggested medications are no better than other forms of therapy in the long term. In 2010, the BBC Trust criticized the 2007 BBC Panorama program for summarizing the research as showing "no demonstrable improvement in children's behavior after staying on ADHD medication for three years" when in actuality "the study found that medication did offer a significant improvement over time" although the long-term benefits of medication were found to be "no better than children who were treated with behavior therapy."

Special populations

Adults

It is estimated that between 2–5% of adults have ADHD. Around half of children with ADHD continue to have ADHD as adults. Of those who continue to have symptoms approximately 25% have the full disorder and 75% partially 'grow out' of it. Most adults remain untreated. Many have a disorganized life and use non-prescribed drugs and alcohol as a coping mechanism. Other problems may include relationship and job difficulties, and an increased risk of criminal activities. Associated mental health problems include: depression, anxiety disorder, and learning disabilities.

Some ADHD symptoms in adults differ from those seen in children. While children with ADHD may climb and run about excessively, adults may experience an inability to relax or talk excessively in social situations. Adults with ADHD may start relationships impulsively, display sensation-seeking behavior, and be short-tempered. Addictive behavior such as substance abuse and gambling are common. The DSM-IV criteria have been criticized for not being appropriate for adults; those who present differently may lead to the claim that they outgrew the diagnosis.

Children with high IQ scores

The diagnosis of ADHD and the significance of its impact on children with a high intelligence quotient (IQ) is controversial. Most studies have found similar impairments regardless of IQ, with higher rates of repeating grades and having social difficulties. Additionally, more than half of people with high IQ and ADHD experience major depressive disorder or oppositional defiant disorder at some point in their lives. Generalised anxiety disorder, separation anxiety disorder and social phobia are also more common. There is some evidence that individuals with

high IQ and ADHD have a lowered risk of substance abuse and anti-social behavior compared to children with low and average IQ and ADHD. Children and adolescents with high IQ can have their level of intelligence mismeasured during a standard evaluation and may require more comprehensive testing.

Research

The QEEG, a type of EEG, is being studied to help with the diagnosis of ADHD. It usefulness for this reason is not very clear. There are concerns that it is not a sufficiently specific test for ADHD. In the United States the Food and Drug Administration has approved a machine for this indication.

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External links

- Attention deficit hyperactivity disorder (http://www.dmoz.org/Mental_Health/Disorders/Neurodevelopmental/ADD_and_ADHD/) on the Open Directory Project
- National Institute of Mental Health on ADHD (http://www.nimh.nih.gov/topics/topic-page-adhd.shtml)
- New Zealand MOH Guidelines for the Assessment and Treatment of Attention-Deficit/Hyperactivity Disorder (http://www.health.govt.nz/publication/new-zealand-guidelines-assessment-and-treatment-attention-deficit-hyperactivity-disorder)

Bipolar disorder

Bipolar disorder	
Classification and external resources	
 Bipolar disorder is characterized by transitions between depression and mania	
ICD-10	F31 [1]
ICD-9	296.0 [2], 296.1 [3], 296.4 [4], 296.5 [5], 296.6 [6], 296.7 [7], 296.8 [8]
OMIM	125480 [9] 309200 [10]
DiseasesDB	7812 [11]
MedlinePlus	000926 [12]
eMedicine	med/229 [13]
MeSH	D001714 [14]

Bipolar disorder (also known as **bipolar affective disorder**, **manic-depressive disorder**, or **manic depression**) is a mental illness typically classified as a mood disorder. It is characterized by episodes of an elevated or agitated mood known as mania, usually alternating with episodes of depression. These episodes can impair the individual's ability to function in ordinary life. About 3% of people have bipolar disorder worldwide, a proportion consistent for both men and women and across racial and ethnic groups. The cause is not clearly understood, but both genetic and environmental risk factors are believed to play a role. Treatment commonly includes mood stabilizing medication and psychotherapy.

There are widespread problems with social stigma, stereotypes, and prejudice against individuals with bipolar disorder.

Signs and symptoms

Mania is the defining feature of bipolar disorder, and can occur with different levels of severity. With milder levels of mania, known as hypomania, individuals appear energetic, excitable, and may be highly productive. As mania worsens, individuals begin to exhibit erratic and impulsive behavior, often making poor decisions due to unrealistic ideas about the future, and sleep very little. At the most severe level, manic individuals can experience very distorted beliefs about the world known as psychosis. A depressive episode commonly follows an episode of mania. The biological mechanisms responsible for switching from a manic or hypomanic episode to a depressive episode or vice

versa remain poorly understood.

Manic episodes

Mania is a distinct period of at least one week of elevated or irritable mood, which can take the form of euphoria, and exhibit three or more of the following behaviors (four if irritable): speak in a rapid, uninterruptible manner, are easily distracted, have racing thoughts, display an increase in goal-oriented activities or feel agitated, or exhibit behaviors characterized as impulsive or high-risk such as hypersexuality or excessive money spending. To meet the definition for a manic episode, these behaviors must impair the individual's ability to socialize or work. If untreated, a manic episode usually lasts three to six months.

People with mania may also experience a decreased need for sleep, speak excessively in addition to speaking rapidly, and may have impaired judgment. Manic individuals often have issues with substance abuse due to a combination of thrill-seeking and poor judgment. At more extreme levels, a person in a manic state can experience psychosis, or a break with reality, a state in which thinking is affected along with mood. They may feel out of control or unstoppable, or as if they have been "chosen" and are on a special mission, or have other grandiose or delusional ideas. Approximately 50% of those with bipolar disorder experience delusions or hallucinations. This may lead to violent behaviors and hospitalization in an inpatient psychiatric hospital. The severity of manic symptoms can be measured by rating scales such as the Young Mania Rating Scale.

The onset of a manic (or depressive) episode is often foreshadowed by sleep disturbances. Mood changes, psychomotor and appetite changes, and an increase in anxiety can also occur up to three weeks before a manic episode develops.

Hypomanic episodes

Hypomania is a milder form of mania defined as at least four days of the same criteria as mania, but does not cause a significant decrease in the individual's ability to socialize or work, lacks psychotic features (i.e., delusions or hallucinations), and does not require psychiatric hospitalization. Overall functioning may actually increase during episodes of hypomania and is thought to serve as a defense mechanism against depression. Hypomanic episodes rarely progress to true manic episodes. Some hypomanic people show increased creativity while others are irritable or demonstrate poor judgment. Many experience hypersexuality. Hypomanic people generally have increased energy and increased activity levels.

Hypomania may feel good to the person who experiences it. Thus, even when family and friends recognize mood swings, the individual often will deny that anything is wrong. What might be called a "hypomanic event", if not accompanied by depressive episodes, is often not deemed as problematic, unless the mood changes are uncontrollable, volatile or mercurial. Most commonly, symptoms continue for a few weeks to a few months.

Depressive episodes

Signs and symptoms of the depressive phase of bipolar disorder include persistent feelings of sadness, anxiety, guilt, anger, isolation, or hopelessness; disturbances in sleep and appetite; fatigue and loss of interest in usually enjoyable activities; problems concentrating; loneliness, self-loathing, apathy or indifference; depersonalization; loss of interest in sexual activity; shyness or social anxiety; irritability, chronic pain (with or without a known cause); lack of motivation; and morbid suicidal thoughts. In severe cases, the individual may become psychotic, a condition also known as severe bipolar depression with psychotic features. These symptoms include delusions or, less commonly, hallucinations, which are usually unpleasant. A major depressive episode persists for at least two weeks, and may continue for over six months if left untreated.

The earlier the age of onset, the more likely the first few episodes are to be depressive. Because a bipolar diagnosis requires a manic or hypomanic episode, many patients are initially diagnosed and treated as having major depression.

Mixed affective episodes

In the context of bipolar disorder, a mixed state is a condition during which symptoms of both mania and depression occur at the same time. Individuals experiencing a mixed state may have manic symptoms such as grandiose thoughts while at the same time experiencing depressive symptoms such as excessive guilt or feeling suicidal. Mixed states are considered to be high-risk for suicidal behavior since depressive emotions such as hopelessness are often paired with mood swings or difficulties with impulse control. Anxiety disorder occurs more frequently as a comorbidity in mixed bipolar episodes than in non mixed bipolar depression or mania. Substance abuse (including alcohol) also follows this trend.

Associated features

Associated features are clinical phenomena that often accompany the disorder but are not part of the diagnostic criteria. In adults with the condition, bipolar disorder is often accompanied by changes in cognitive processes and abilities. These include reduced attentional and executive capabilities and impaired memory. How the individual processes the world also depends on the phase of the disorder, with differential characteristics between the manic, hypomanic and depressive states. Some studies have found a significant association between bipolar disorder and creativity. Those with bipolar disorder may have difficulty in maintaining relationships.[15] There are several common childhood precursors seen in children who later receive a diagnosis of bipolar disorder; these disorders include mood abnormalities, full major depressive episodes, and attention deficit hyperactivity disorder (ADHD).

Comorbid conditions

The diagnosis of bipolar disorder can be complicated by coexisting (comorbid) psychiatric conditions including the following: obsessive-compulsive disorder, substance abuse, eating disorders, attention deficit hyperactivity disorder, social phobia, premenstrual syndrome (including premenstrual dysphoric disorder), or panic disorder. A careful longitudinal analysis of symptoms and episodes, enriched if possible by discussions with friends and family members, is crucial to establishing a treatment plan where these comorbidities exist.

Causes

The causes of bipolar disorder likely vary between individuals. Genetic influences are believed to account for 60-80% of the risk of developing the disorder indicating a strong hereditary component. The overall heritability of the bipolar spectrum has been estimated at 0.71. Twin studies have been limited by relatively small sample sizes but have indicated a substantial genetic contribution, as well as environmental influence. For bipolar disorder type I, the (probandwise) concordance rates in modern studies have been consistently estimated at around 40% in identical twins (same genes), compared to about 5% in fraternal twins. A combination of bipolar I, II and cyclothymia produced concordance rates of 42% vs 11%, with a relatively lower ratio for bipolar II that likely reflects heterogeneity. There is overlap with unipolar depression and if this is also counted in the co-twin the concordance with bipolar disorder rises to 67% in monozygotic twins and 19% in dizygotic. The relatively low concordance between dizygotic twins brought up together suggests that shared family environmental effects are limited, although the ability to detect them has been limited by small sample sizes.

Genetic

Genetic studies have suggested that many chromosomal regions and candidate genes are related to bipolar disorder susceptibility with each gene exerting a mild to moderate effect. The risk of bipolar disorder is nearly ten-fold higher in first degree-relatives of those affected with bipolar disorder when compared to the general population; similarly, the risk of major depressive disorder is three times higher in relatives of those with bipolar disorder when compared to the general population.

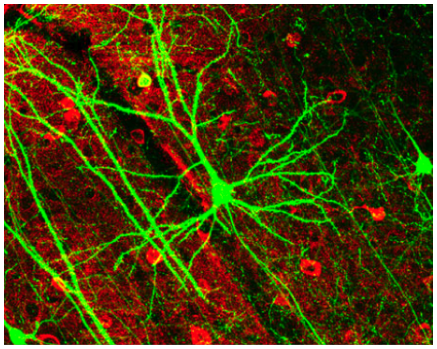
Although the first genetic linkage finding for mania was in 1969, the linkage studies have been inconsistent. The largest and most recent genome-wide association study failed to find any particular locus that exerts a large effect reinforcing the idea that no single gene is responsible for bipolar disorder in most cases.

Findings point strongly to heterogeneity, with different genes being implicated in different families. Robust and replicable genome-wide significant associations showed several common single nucleotide polymorphisms, including variants within the genes CACNA1C, ODZ4, and NCAN.

Advanced paternal age has been linked to a somewhat increased chance of bipolar disorder in offspring, consistent with a hypothesis of increased new genetic mutations.

Physiological

Abnormalities in the structure and/or function of certain brain circuits could underlie bipolar. Meta-analyses of structural MRI studies in bipolar disorder report an increase in the volume of the lateral ventricles, globus pallidus and increase in the rates of deep white matter hyperintensities. Functional MRI findings suggest that abnormal modulation between ventral prefrontal and limbic regions, especially the amygdala, are likely contribute to poor emotional regulation and mood symptoms.



Brain imaging studies have revealed differences in the volume of various brain regions between BD patients and healthy control subjects

According to the "kindling" hypothesis, when people who are genetically predisposed toward bipolar disorder experience stressful events, the stress threshold at which mood changes occur becomes progressively lower, until the episodes eventually start (and recur) spontaneously. There is evidence of hypothalamic-pituitary-adrenal axis (HPA axis) abnormalities in bipolar disorder due to stress.

Other brain components which have been proposed to play a role are the mitochondria and a sodium ATPase pump.[16] Alterations to these components are believed to cause cyclical periods of poor neuron firing (depression) and hypersensitive neuron firing (mania). Circadian rhythms and melatonin activity also seem to be altered.

Environmental

Evidence suggests that environmental factors play a significant role in the development and course of bipolar disorder, and that individual psychosocial variables may interact with genetic dispositions. There is fairly consistent evidence from prospective studies that recent life events and interpersonal relationships contribute to the likelihood of onsets and recurrences of bipolar mood episodes, as they do for onsets and recurrences of unipolar depression. There have been repeated findings that between a third and a half of adults diagnosed with bipolar disorder report traumatic/abusive experiences in childhood, which is associated on average with earlier onset, a worse course, and more co-occurring disorders such as PTSD. The total number of reported stressful events in childhood is higher in those with an adult diagnosis of bipolar spectrum disorder compared to those without, particularly events stemming from a harsh environment rather than from the child's own behavior.

Neurological

Less commonly bipolar disorder or a bipolar-like disorder may occur as a result of or in association with a neurological condition or injury. Such conditions and injuries include (but are not limited to) stroke, traumatic brain injury, HIV infection, multiple sclerosis, porphyria and rarely temporal lobe epilepsy.[17]

Neuroendocrinological

Dopamine, a known neurotransmitter responsible for mood cycling, has been shown to have increased transmission during the manic phase. The dopamine hypothesis states that the increase in dopamine results in secondary homeostatic down regulation of key systems and receptors such as an increase in dopamine mediated G protein-coupled receptors. This results in decreased dopamine transmission characteristic of the depressive phase. The depressive phase ends with homeostatic up regulation potentially restarting the cycle over again.

Two additional neurotransmitters, gamma-Aminobutyric acid (GABA) and glutamate, have been found to cause elevated mood states. Glutamate is significantly increased within the left dorsolateral prefrontal cortex during the manic phase of bipolar disorder, and returns to normal levels once the phase is over. GABA is found in higher concentrations in people with bipolar disorder, overall leading to a decrease in GABA (B) receptors. The increase in GABA is possibly caused by a disturbance in early development causing a disturbance of cell migration and the formation of normal lamination, the layering of brain structures commonly associated with the cerebral cortex.

Evolutionary

Because bipolar disorder affects an individual's ability to function in society and has a high morbidity rate, evolutionary theory would suggest that the genes responsible would have been naturally selected against, effectively culling the disorder. Yet there continue to be high rates of bipolar disorder in many populations, suggesting an evolutionary benefit to the genes responsible.

Proponents of evolutionary medicine hypothesize that the genes that cause severe bipolar disorder when inherited in large doses may increase fitness when inherited in small doses.[18] High rates of bipolar disorder throughout history suggest that the ability to switch between depressive and manic moods conveyed some evolutionary advantage on ancestral humans. Theories put forward to explain the evolutionary advantages of major depressive disorder may also explain the adaptiveness of the depressive episodes of bipolar disorder. For example, in individuals under increased stress, depressive mood may serve as a defensive strategy that causes the individual to retreat from the external stressor, increase sleep, and preserve resources and energy for better times. Additionally, manic moods may convey advantage in some situations. Creativity, confidence, and high energy have all been linked to mania and hypomania.[19][20] The ability to utilize mild manic symptoms to be more productive and think more creatively during stress-free times may have increased the fitness of ancestral humans. Being able to employ both hypomania and mild depression convey advantages that benefit individuals in a variable environment. However, if the genes enabling the manipulation of both of these moods are over activated, the manic and/or severe depressive moods of full bipolar disorder may be triggered instead.

Evolutionary biologists have hypothesized that bipolar disorder could have come from an adaptation to extreme climactic conditions in the northern temperate zone during the Pleistocene. The Evolutionary Origin of Bipolar Disorder (EOBD) hypothesis states that during the short summers of extreme climactic zones, hypomania would be adaptive, allowing the completion of many tasks necessary for survival within a short period of time. During long winters the lethargy, hypersomnia, lack of interest in social activities, and overeating of depression would be adaptive to group cohesion and survival.[21] Evidence for the EOBD hypothesis include an association between bipolar disorder and a cold-adapted build, correlation between seasonality and mood changes in those with bipolar disorder, and low rates of bipolar disorder in African Americans. The EOBD hypothesis suggests that in the absence of the extreme climactic conditions that fostered the success of bipolar disorder genes, many bipolar disorder behaviors are maladaptive and can often severely impair normal functioning.

Prevention

Prevention of bipolar has focused on stress (such as childhood adversity or highly conflictual families) which, although not a diagnostically specific causal agent for bipolar, does place genetically and biologically vulnerable individuals at risk for a more pernicious course of illness. There has been debate regarding the causal relationship between usage of cannabis and bipolar disorder.

Diagnosis

Bipolar disorder often goes unrecognized and is commonly diagnosed during adolescence or early adulthood. Diagnosis is based on the self-reported experiences of an individual as well as abnormalities in behavior reported by family members, friends or co-workers, followed by secondary signs observed by a psychiatrist, nurse, social worker, clinical psychologist or other clinician in a clinical assessment. There are lists of criteria for someone to be so diagnosed. These depend on both the presence and duration of certain signs and symptoms. Assessment is usually done on an outpatient basis; admission to an inpatient facility is considered if there is a risk to oneself or others. The most widely used criteria for diagnosing bipolar disorder are from the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders, the current version being DSM-IV-TR, and the World Health Organization's International Statistical Classification of Diseases and Related Health Problems, currently the ICD-10. The latter criteria are typically used in Europe and other regions while the DSM criteria are used in the USA and other regions, as well as prevailing in research studies. The DSM-V, published in 2013, included further and more accurate sub-typing.

An initial assessment may include a physical exam by a physician. Although there are no biological tests which confirm bipolar disorder, tests may be carried out to exclude medical illnesses such as hypothyroidism or hyperthyroidism, metabolic disturbance, a chronic disease, or an infection such as syphilis or HIV. An EEG may be used to exclude a seizure disorder such as epilepsy, and a CT scan of the head may be used to exclude brain lesions. Investigations are not generally repeated for relapse unless there is a specific *medical* indication. There is no specific medical test to diagnose bipolar disorder.

Several rating scales for the screening and evaluation of bipolar disorder exist, such as the Bipolar spectrum diagnostic scale. The use of evaluation scales can not substitute a full clinical interview but they serve to systematize the recollection of symptoms. On the other hand, instruments for the screening of bipolar disorder have low sensitivityWikipedia:Please clarify and limited diagnostic validity.

Bipolar spectrum

Bipolar spectrum disorders include the following four disorders: bipolar I disorder, bipolar II disorder, cyclothymic disorder, and bipolar disorder not otherwise specified. These disorders typically also involve depressive symptoms or episodes that alternate with the elevated mood states, or with mixed episodes that feature symptoms of both. The concept of the bipolar spectrum is similar to that of Emil Kraepelin's original concept of manic depressive illness.[22] Currently, manic depressive illness is usually referred to as bipolar disorder or simply bipolar. A simple nomenclature system was introduced in 1978 to classify more easily individuals' affectedness within the spectrum.

Points on the spectrum using this nomenclature are denoted using the following codes:

- *M*—severe mania
- *D*—severe depression (unipolar depression)
- *m*—less severe mania (hypomania)
- *d*—less severe depression

Thus, *mD* represents a case with hypomania and major depression. A further distinction is sometimes made in the ordering of the letters, to represent the order of the episodes, where the patient's normal state is euthymic, interrupted by episodes of mania followed by depression (*MD*) or vice versa (*DM*).

Employing this schema, major depression would be denoted *D*. Unipolar mania (*M*) is, depending on the authority cited, either very rare,[23] or nonexistent with such cases actually being *Md*.

Unipolar hypomania (*m*) without accompanying depression has been noted in the medical literature.[24] There is speculation as to whether this condition may occur with greater frequency in the general, untreated population; successful social function of these potentially high-achieving individuals may lead to being labeled as normal, rather than as individuals with substantial dysregulation.

Criteria and subtypes

There is no clear consensus as to how many types of bipolar disorder exist. In DSM-IV-TR and ICD-10, bipolar disorder is conceptualized as a spectrum of disorders occurring on a continuum. The DSM-IV-TR lists three specific subtypes and one for non-specified:[25]

Bipolar I disorder: One or more manic episodes. Subcategories specify whether there has been more than one episode, and the type of the most recent episode. While depressive episodes are common in bipolar disorder, they are unnecessary to make the diagnosis.

Bipolar II disorder: No manic episodes, but one or more hypomanic episodes and one or more major depressive episode. Hypomanic episodes do not go to the full extremes of mania (*i.e.*, do not usually cause severe social or occupational impairment, and are without psychosis), and this can make bipolar II more difficult to diagnose, since the hypomanic episodes may simply appear as a period of successful high productivity and is reported less frequently than a distressing, crippling depression.

Cyclothymia: A history of hypomanic episodes with periods of depression that do not meet criteria for major depressive episodes. There is a low-grade cycling of mood which appears to the observer as a personality trait, and interferes with functioning.

Bipolar disorder NOS (not otherwise specified): This is a catchall category, diagnosed when the disorder does not fall within a specific subtype. Bipolar NOS can still significantly impair and adversely affect the quality of life of the patient.

The bipolar I and II categories have specifiers that indicate the presentation and course of the disorder. For example, the "with full interepisode recovery" specifier applies if there was full remission between the two most recent episodes.

Rapid cycling

Most people who meet criteria for bipolar disorder experience a number of episodes, on average 0.4 to 0.7 per year, lasting three to six months. *Rapid cycling*, however, is a course specifier that may be applied to any of the above subtypes. It is defined as having four or more mood disturbance episodes within a one year span and is found in a significant proportion of individuals with bipolar disorder. These episodes are separated from each other by a remission (partial or full) for at least two months or a switch in mood polarity (i.e., from a depressive episode to a manic episode or vice versa). The definition of rapid cycling most frequently cited in the literature (including the DSM) is that of Dunner and Fieve: at least four major depressive, manic, hypomanic or mixed episodes are required to have occurred during a 12-month period. Ultra-rapid (days) and ultra-ultra rapid or ultradian (within a day) cycling have also been described. The literature examining the pharmacological treatment of rapid cycling is sparse and there is no clear consensus with respect to its optimal pharmacological management.

Differential diagnosis

There are several other mental disorders which may involve similar symptoms to bipolar disorder. These include schizophrenia, major depressive disorder, attention deficit hyperactivity disorder (ADHD), and some personality disorders, such as borderline personality disorder.

It has been noted that the bipolar disorder diagnosis is officially characterized in historical terms such that, technically, anyone with a history of (hypo)mania and depression has bipolar disorder whatever their current or future functioning and vulnerability. This has been described as "an ethical and methodological issue", as it means no one can be considered as being recovered (only "in remission") from bipolar disorder according to the official criteria. This is considered especially problematic given that brief hypomanic episodes are widespread among people generally and not necessarily associated with dysfunction.

Management

There are a number of pharmacological and psychotherapeutic techniques used to treat bipolar disorder. Individuals may use self-help and pursue recovery.

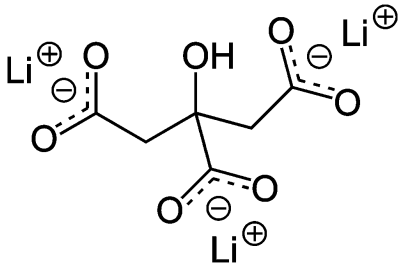
Hospitalization may be required especially with the manic episodes present in bipolar I. This can be voluntary or (if mental health legislation allows and varying state-to-state regulations in the USA) involuntary (called civil or involuntary commitment). Long-term inpatient stays are now less common due to deinstitutionalization, although these can still occur. Following (or in lieu of) a hospital admission, support services available can include drop-in centers, visits from members of a community mental health team or Assertive Community Treatment team, supported employment and patient-led support groups, intensive outpatient programs. These are sometimes referred to partial-inpatient programs.

Light therapy is one of several approaches to treating bipolar disorder. No one method is universally successful and most persons suffering from the illness need several forms of support.

Psychosocial

Psychotherapy is aimed at alleviating core symptoms, recognizing episode triggers, reducing negative expressed emotion in relationships, recognizing prodromal symptoms before full-blown recurrence, and, practicing the factors that lead to maintenance of remission.[26][27][28] Cognitive behavioural therapy, family-focused therapy, and psychoeducation have the most evidence for efficacy in regard to relapse prevention, while interpersonal and social rhythm therapy and cognitive-behavioural therapy appear the most effective in regard to residual depressive symptoms. Most studies have been based only on bipolar I, however, and treatment during the acute phase can be a particular challenge. Some clinicians emphasize the need to talk with individuals experiencing mania, to develop a therapeutic alliance in support of recovery.

Medication



Lithium is one of many treatments for bipolar disorder.

A number of medications are used to treat bipolar disorder. The medication with the best evidence is lithium, which is effective in treating acute manic episodes, and preventing relapses, more so for manic than for depressive episodes. Lithium reduces the risk of suicide, self-harm, and death in people with bipolar disorder.

Four anticonvulsants are used in the treatment of bipolar disorder. Carbamazepine effectively treats manic episodes, with some evidence it has greater benefit in rapid-cycling bipolar disorder, or those with more psychotic symptoms or a more schizoaffective clinical picture. It is less effective in preventing relapse than lithium. Carbamazepine became a popular treatment option for bipolar in the late 1980s and early 1990s, but was displaced by sodium valproate in the 1990s,[*citation needed*] which has become a commonly prescribed treatment, and is effective in treating manic episodes. Lamotrigine has some efficacy in treating bipolar depression, and this benefit is greatest in more severe depression. It has also been shown to have some benefit in preventing further episodes, though there are concerns about the studies done, and is of no benefit in rapid cycling disorder. The effectiveness of topiramate is unknown. Depending on the severity of the case, anticonvulsants may be used in combination with lithium or on their own.[29]

Antipsychotic medications are effective for short-term treatment of bipolar manic episodes and appear to be superior to lithium and anticonvulsants for this purpose. However, other medications such as lithium are preferred for long-term use. Olanzapine is effective in preventing relapses, although the evidence is not as solid as for lithium. Antidepressants have not been found to be of any benefit over that found with mood stabilizers.

Short courses of benzodiazepines may be used in addition to other medications until mood stabilizing become effective.

Alternative medicine

The addition of omega 3 fatty acids may have beneficial effects on depressive symptoms, although studies have been scarce and of variable quality.

Prognosis

For many individuals with bipolar disorder a good prognosis results from good treatment, which, in turn, results from an accurate diagnosis. Of the various forms of bipolar disorder, rapid cycling bipolar disorder is associated with the worst prognosis. Because bipolar disorder can have a high rate of both under-diagnosis and misdiagnosis, it is often difficult for individuals with the condition to receive timely and competent treatment.

Bipolar disorder can be a severely disabling medical condition. However, many individuals with bipolar disorder can live full and satisfying lives. Quite often, medication is needed to enable this. Persons with bipolar disorder may have periods of normal or near normal functioning between episodes.[30]

Functioning

Functioning in bipolar I and II varies over time along a spectrum from good to fair to poor. During periods of major depression or mania (in BPI), functioning was on average poor, with depression being more persistently associated with disability than mania. Functioning between episodes was on average good — more or less normal. Subthreshold symptoms were generally still substantially impairing, however, except for hypomania (below or above threshold) which was associated with improved functioning.

Another study confirmed the seriousness of the disorder as "the standardized all-cause mortality ratio among patients with bipolar disorder is increased approximately two-fold." Bipolar disorder is currently regarded "as possibly the most costly category of mental disorders in the United States." Episodes of abnormality are associated with distress and disruption, and an elevated risk of suicide, especially during depressive episodes.

Recovery and recurrence

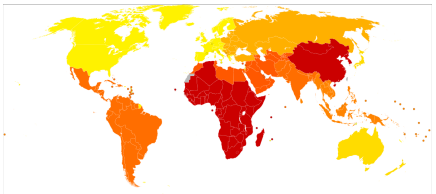
A naturalistic study from first admission for mania or mixed episode (representing the hospitalized and therefore most severe cases) found that 50% achieved syndromal recovery (no longer meeting criteria for the diagnosis) within six weeks and 98% within two years. Within two years, 72% achieved symptomatic recovery (no symptoms at all) and 43% achieved functional recovery (regaining of prior occupational and residential status). However, 40% went on to experience a new episode of mania or depression within 2 years of syndromal recovery, and 19% switched phases without recovery.

Symptoms preceding a relapse (prodromal), specially those related to mania, can be reliably identified by people with bipolar disorder. There have been intents to teach patients coping strategies when noticing such symptoms with encouraging results.

Suicide

Bipolar disorder can cause suicidal ideation that leads to suicidal attempts. Individuals whose bipolar disorder begins with a depressive or mixed affective episode seem to have a poorer prognosis and an increased risk of suicide. One out of two people with bipolar disorder attempt at least once during their lifetime and many attempts are successfully completed. The annual average suicide rate is 0.4%, which is 10-20 times that of the general population.[31] The standardized mortality ratio from suicide in bipolar disorder is between 18 and 25. The lifetime risk of suicide has been estimated to be as high as 20% in those with bipolar disorder.

Epidemiology



Burden of bipolar disorder around the world: disability-adjusted life years per 100,000 inhabitants in 2002.

Bipolar disorder is the sixth leading cause of disability worldwide and has a lifetime prevalence of about 3% in the general population. However, a reanalysis of data from the National Epidemiological Catchment Area survey in the United States suggested that 0.8% of the population experience a manic episode at least once (the diagnostic threshold for bipolar I) and a further 0.5% have a hypomanic episode (the diagnostic threshold for bipolar II or cyclothymia). Including sub-threshold diagnostic criteria, such as one or two symptoms over a short time-period, an additional 5.1% of the population, adding up to a total of 6.4%, were classified as having a bipolar spectrum disorder. A more recent analysis of data from a second US National Comorbidity Survey found that 1% met lifetime prevalence criteria for bipolar I, 1.1% for bipolar II, and 2.4% for subthreshold symptoms. There are conceptual and methodological limitations and variations in the findings. Prevalence studies of bipolar disorder are typically carried out by lay interviewers who follow fully structured/fixed interview schemes; responses to single items from such interviews may suffer limited validity. In addition, diagnoses (and therefore estimates of prevalence) vary depending on whether a categorical or spectrum approach is used. This consideration has led to concerns about the potential for both underdiagnosis and overdiagnosis.

The incidence of bipolar disorder is similar in men and women as well as across different cultures and ethnic groups. A 2000 study by the World Health Organization found that prevalence and incidence of bipolar disorder are very similar across the world. Age-standardized prevalence per 100,000 ranged from 421.0 in South Asia to 481.7 in

Africa and Europe for men and from 450.3 in Africa and Europe to 491.6 in Oceania for women. However, severity may differ widely across the globe. Disability-adjusted life year rates, for example, appear to be higher in developing countries, where medical coverage may be poorer and medication less available.

Within the United States, African and European Americans have similar rates of bipolar disorder, while Asian Americans have lower rates.

Late adolescence and early adulthood are peak years for the onset of bipolar disorder.[32] One study also found that in 10% of bipolar cases, the onset of mania had happened after the patient had turned 50.

History

German psychologist Emil Kraeplin first distinguished between manic–depressive illness and "dementia praecox" (now known as schizophrenia) in the late 19th century

Variations in moods and energy levels have been observed as part of the human experience since throughout history. The words "melancholia" (an old word for depression) and "mania" originated in Ancient Greek. The word melancholia is derived from *melas*/μελας, meaning "black", and *chole*/χολη, meaning "bile" or "gall",[33] indicative of the term's origins in pre-Hippocratic humoral theories. Within the humoral theories, mania was viewed as arising from an excess of yellow bile, or a mixture of black and yellow bile. The linguistic origins of mania, however, are not so clear-cut. Several etymologies are proposed by the Roman physician Caelius Aurelianus, including the Greek word *ania*, meaning "to produce great mental anguish", and *manos*, meaning "relaxed" or "loose", which would contextually approximate to an excessive relaxing of the mind or soul. There are at least five other candidates, and part of the confusion surrounding the exact etymology of the word mania is its varied usage in the pre-Hippocratic poetry and mythologies.

In the early 1800s, French psychiatrist Jean-Étienne Dominique Esquirol's lypemania, one of his affective monomanias, was the first elaboration on what was to become modern depression.[34] The basis of the current conceptualisation of manic–depressive illness can be traced back to the 1850s; on January 31, 1854, Jules Baillarger described to the French Imperial Academy of Medicine a biphasic mental illness causing recurrent oscillations between mania and depression, which he termed *folie à double forme* ("dual-form insanity"). Two weeks later, on February 14, 1854, Jean-Pierre Falret presented a description to the Academy on what was essentially the same disorder, and designated *folie circulaire* ("circular insanity") by him.

These concepts were developed by the German psychiatrist Emil Kraepelin (1856–1926), who, using Kahlbaum's concept of cyclothymia,[35] categorized and studied the natural course of untreated bipolar patients. He coined the term *manic depressive psychosis*, after noting that periods of acute illness, manic or depressive, were generally punctuated by relatively symptom-free intervals where the patient was able to function normally.[36]

The term "manic–depressive *reaction*" appeared in the first American Psychiatric Association Diagnostic Manual in 1952, influenced by the legacy of Adolf Meyer who had introduced the paradigm illness as a reaction of biogenetic factors to psychological and social influences.[37] Subclassification of bipolar disorder was first proposed by German psychiatrist Karl Leonhard in 1957; he was also the first to introduce the terms *bipolar* (for those with mania) and *unipolar* (for those with depressive episodes only).[38]

Society and culture

There are widespread problems with social stigma, stereotypes, and prejudice against individuals with a diagnosis of bipolar disorder.

Singer Rosemary Clooney's public revelation of bipolar disorder in 1977 made her an early celebrity spokeswoman for mental illness

Kay Redfield Jamison, a clinical psychologist and Professor of Psychiatry at the Johns Hopkins University School of Medicine, profiled her own bipolar disorder in her memoir *An Unquiet Mind* (1995).[39] In his autobiography *Manicdotes: There's Madness in His Method* (2008) Chris Joseph describes his struggle between the creative dynamism which allowed the creation of his multi-million pound advertising agency Hook Advertising, and the money-squandering dark despair of his bipolar illness.[40]

Several dramatic works have portrayed characters with traits suggestive of the diagnosis that has been the subject of discussion by psychiatrists and film experts alike. A notable example is *Mr. Jones* (1993), in which Mr. Jones (Richard Gere) swings from a manic episode into a depressive phase and back again, spending time in a psychiatric hospital and displaying many of the features of the syndrome.[41] In *The Mosquito Coast* (1986), Allie Fox (Harrison Ford) displays some features including recklessness, grandiosity, increased goal-directed activity and mood lability, as well as some paranoia.[42] Psychiatrists have suggested that Willy Loman, the main character in Arthur Miller's classic play *Death of a Salesman*, suffers from bipolar disorder, though that specific term for the condition did not exist when the play was written.

TV specials, for example the BBC's *The Secret Life of the Manic Depressive*, MTV's *True Life: I'm Bipolar*, talk shows, and public radio shows, and the greater willingness of public figures to discuss their own bipolar disorder, have focused on psychiatric conditions, thereby, raising public awareness.

On April 7, 2009, the nighttime drama *90210* on the CW network, aired a special episode where the character Silver was diagnosed with bipolar disorder. Stacey Slater, a character from the BBC soap EastEnders, has been diagnosed with the disorder. The storyline was developed as part of the BBC's Headroom campaign. The Channel 4 soap *Brookside* had earlier featured a story about bipolar disorder when the character Jimmy Corkhill was diagnosed with the condition.

Specific populations

Children

In the 1920s, Emil Kraepelin noted that manic episodes are rare before puberty. In general, bipolar disorder in children was not recognized in the first half of the twentieth century. This issue diminished with an increased following of the DSM criteria in the last part of the twentieth century.

Lithium is the only medication approved for treating mania in children by the FDA

While in adults the course of bipolar disorder is characterized by discrete episodes of depression and mania with no clear symptomatology between them, in children and adolescents very fast mood changes or even chronic symptoms are the norm. In pediatric bipolar disorder is commonly characterized by outbursts of anger,

irritability and psychosis, rather than euphoric mania, which is more likely to be seen in adults. Early onset bipolar disorder is more likely to manifest as depression rather than mania or hypomania.

The diagnosis of childhood bipolar disorder is controversial, although it is not under discussion that the typical symptoms of bipolar disorder have negative consequences for minors suffering them. The debate is mainly centered on whether what is called bipolar disorder in children refers to the same disorder as when diagnosing adults, and the related question of whether the criteria for diagnosis for adults are useful and accurate when applied to children. Regarding diagnosis of children, some experts recommend following the DSM criteria. Others believe that these criteria do not correctly separate children with bipolar disorder from other problems such as ADHD, and emphasize fast mood cycles. Still others argue that what accurately differentiates children with bipolar disorder is irritability. The practice parameters of the AACAP encourage the first strategy. American children and adolescents diagnosed with bipolar disorder in community hospitals increased 4-fold reaching rates of up to 40% in 10 years around the beginning of the 21st century, while in outpatient clinics it doubled reaching 6%. Studies using DSM criteria show that up to 1% of youth may have bipolar disorder.

Treatment involves medication and psychotherapy. Drug prescription usually consists in mood stabilizers and atypical antipsychotics. Among the former, lithium is the only compound approved by the FDA for children. Psychological treatment combines normally education on the disease, group therapy and cognitive behavioral therapy. Chronic medication is often needed.

Current research directions for bipolar disorder in children include optimizing treatments, increasing the knowledge of the genetic and neurobiological basis of the pediatric disorder and improving diagnostic criteria. The DSM-V has proposed a new diagnosis which is considered to cover some presentations currently thought of as childhood-onset bipolar.

Elderly

There is a relative lack of knowledge about bipolar disorder in late life. There is evidence that it becomes less prevalent with age but nevertheless accounts for a similar percentage of psychiatric admissions; that older bipolar patients had first experienced symptoms at a later age; that later onset of mania is associated with more neurologic impairment; that substance abuse is considerably less common in older groups; and that there is probably a greater degree of variation in presentation and course, for instance individuals may develop new-onset mania associated with vascular changes, or become manic only after recurrent depressive episodes, or may have been diagnosed with bipolar disorder at an early age and still meet criteria. There is also some weak evidence that mania is less intense and there is a higher prevalence of mixed episodes, although there may be a reduced response to treatment. Overall, there are likely more similarities than differences from younger adults. In the elderly, recognition and treatment of bipolar disorder may be complicated by the presence of dementia or the side effects of medications being taken for other conditions.

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[35] Millon 1996, p. 290.
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[37] Goodwin & Jamison 2007, Chapter 1.
[38] Goodwin & Jamison 2007, Introduction.
[39] Jamison 1995.
[40] Joseph 2008.
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External links

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- NICE Bipolar Disorder clinical guidelines (http://www.nice.org.uk/Guidance/CG38) from the U.K. National Institute for Health and Clinical Excellence website

Borderline personality disorder

Borderline personality disorder	
Classification and external resources	
ICD-10	F60.3 [1]
ICD-9	301.83 [2]
MedlinePlus	000935 [3]
eMedicine	article/913575 [4]
MeSH	D001883 [5]

Borderline personality disorder (**BPD**) (called **emotionally unstable personality disorder, emotional intensity disorder, borderline type** in the ICD-10) is a cluster-B personality disorder whose essential features are a pattern of marked impulsivity and instability of affects, interpersonal relationships, and self image. The pattern is present by early adulthood and occurs across a variety of situations and contexts.

Other symptoms usually include intense fears of abandonment and intense anger and irritability, the reason for which others have difficulty understanding. People with BPD often engage in idealization and devaluation of others, alternating between high positive regard and great disappointment. Self-harm and suicidal behavior are common.

This disorder is recognized by the Diagnostic and Statistical Manual of Mental Disorders. Because a personality disorder is a pervasive, enduring and inflexible pattern of maladaptive inner experience and pathological behavior, there is a general reluctance to diagnose personality disorders before adolescence or early adulthood. Some emphasize, however, that without early treatment, symptoms may worsen.

There is an ongoing debate about the terminology of this disorder, especially the word "borderline". The ICD-10 manual refers to this disorder as *Emotionally unstable personality disorder* and has similar diagnostic criteria. There is related concern that the diagnosis of BPD stigmatizes people with BPD and supports discriminatory practices, because it suggests that the personality of the individual is flawed. In the DSM-5, the name of the disorder remains the same.

Signs and symptoms

The most distinguishing symptoms of BPD are marked sensitivity to rejection and thinking about and feeling afraid of possible abandonment. Overall, the features of BPD include unusually intense sensitivity in relationships with others, difficulty regulating emotions, and impulsivity. Other symptoms can include feeling unsure of one's personal identity and values, having paranoid thoughts when feeling stressed, and severe dissociation.

Emotions

People with BPD feel emotions more easily, more deeply, and for longer than others do. Emotions may repeatedly resurge and persist a long time. Consequently, it can take longer than normal for people with BPD to return to a stable emotional baseline following an intense emotional experience.

In Marsha Linehan's view, the sensitivity, intensity, and duration with which people with BPD feel emotions have both positive and negative effects. People with BPD are often exceptionally idealistic, joyful, and loving. However, they can feel overwhelmed by negative emotions, experiencing intense grief instead of sadness, shame and humiliation instead of mild embarrassment, rage instead of annoyance, and panic instead of nervousness. People

with BPD are especially sensitive to feelings of rejection, isolation, and perceived failure. Before learning other coping mechanisms, their efforts to manage or escape from their intense negative emotions can lead to self-injury or suicidal behavior. They are often aware of the intensity of their negative emotional reactions and, since they cannot regulate them, they shut them down entirely. This can be harmful to people with BPD, as negative emotions alert people to the presence of a problematic situation and move them to address it.

While people with BPD feel joy intensely, they are especially prone to dysphoria, or feelings of mental and emotional distress. Zanarini et al. recognize four categories of dysphoria that are typical of this condition: extreme emotions; destructiveness or self-destructiveness; feeling fragmented or lacking identity; and feelings of victimization. Within these categories, a BPD diagnosis is strongly associated with a combination of three specific states: 1) feeling betrayed, 2) "feeling like hurting myself", and 3) feeling out of control. Since there is great variety in the types of dysphoria experienced by people with BPD, the amplitude of the distress is a helpful indicator of borderline personality disorder.

In addition to intense emotions, people with BPD experience emotional lability, or changeability. Although the term suggests rapid changes between depression and elation, the mood swings in people with this condition actually occur more frequently between anger and anxiety, and between depression and anxiety.

Behavior

Impulsive behaviors are common, including: substance or alcohol abuse, eating disorders, unprotected sex or indiscriminate sex with multiple partners, reckless spending and reckless driving. Impulsive behaviors can also include quitting jobs or relationships, running away, and self-injury.

People with BPD act impulsively because it gives them immediate relief from their emotional pain. However, in the long term, people with BPD suffer increased pain from the shame and guilt that follow such actions. A cycle often begins in which people with BPD feel emotional pain, engage in impulsive behaviors to relieve that pain, feel shame and guilt over their actions, feel emotional pain from the shame and guilt, and then experience stronger urges to engage in impulsive behaviors to relieve the new pain. As time goes on, impulsive behaviors can become an automatic response to emotional pain.

Self-harm and suicidal behavior

Self-harming or suicidal behavior is one of the core diagnostic criteria in the DSM IV-TR. Management of and recovery from this behavior can be complex and challenging. The suicide rate among patients with BPD is 8 to 10 percent.

Self-injury is common, and can take place with or without suicidal intent. The reported reasons for non-suicidal self-injury (NSSI) differ from the reasons for suicide attempts. Reasons for NSSI include expressing anger, self-punishment, generating normal feelings (often in response to dissociation), and distracting oneself from emotional pain or difficult circumstances. In contrast, suicide attempts typically reflect a belief that others will be better off following the suicide. Both suicidal and non-suicidal self-injury are a response to feeling negative emotions.

Sexual abuse can be a particular trigger for suicidal behavior in adolescents with BPD tendencies.Wikipedia:Manual of Style/Dates and numbers

Interpersonal relationships

People with BPD can be very sensitive to the way others treat them, feeling intense joy and gratitude at perceived expressions of kindness, and intense sadness or anger at perceived criticism or hurtfulness. Their feelings about others often shift from positive to negative after a disappointment, a perceived threat of losing someone, or a perceived loss of esteem in the eyes of someone they value. This phenomenon, sometimes called splitting or black-and-white thinking, includes a shift from idealizing others (feeling admiration and love) to devaluing them (feeling anger or dislike). Combined with mood disturbances, idealization and devaluation can undermine relationships with family, friends, and co-workers. Self-image can also change rapidly from positive to negative.

While strongly desiring intimacy, people with BPD tend toward insecure, avoidant or ambivalent, or fearfully preoccupied attachment patterns in relationships, and they often view the world as dangerous and malevolent. BPD is linked to increased levels of chronic stress and conflict in romantic relationships, decreased satisfaction of romantic partners, abuse and unwanted pregnancy. However, these factors appear to be linked to personality disorders in general.

Manipulation to obtain nurturance is considered to be a common feature of BPD by many who treat the disorder, as well as by the DSM-IV. However, some mental health professionals caution that an overemphasis on, and an overly broad definition of, manipulation can lead to misunderstanding and prejudicial treatment of people with BPD within the health care system. (See Manipulative behavior and Stigma under Controversies.)

Sense of self

People with BPD tend to have trouble seeing a clear picture of their identity. In particular, they tend to have a hard time knowing what they value and enjoy. They are often unsure about their long-term goals for relationships and jobs. This difficulty with knowing who they are and what they value can cause people with BPD to experience feeling "empty" and "lost".

Cognitions

The often intense emotions experienced by people with BPD can make it difficult for them to control the focus of their attention—to concentrate. In addition, people with BPD may tend to dissociate, which can be thought of as an intense form of "zoning out". Dissociation often occurs in response to experiencing a painful event (or experiencing something that triggers the memory of a painful event). It involves the mind automatically redirecting attention away from that event, presumably to protect against experiencing intense emotion and unwanted behavioral impulses that such emotion might otherwise trigger. Although the mind's habit of blocking out intense painful emotions may provide temporary relief, it can also have the unwanted side effect of blocking or blunting the experience of ordinary emotions, reducing the access of people with BPD to the information contained in those emotions which helps guide effective decision-making in daily life. Sometimes it is possible for another person to tell when someone with BPD is dissociating, because their facial or vocal expressions may become flat or expressionless, or they may appear to be distracted; at other times, dissociation may be barely noticeable.

Diagnosis

Personality disorders
Cluster A (odd)
• Paranoid • Schizoid • Schizotypal
Cluster B (dramatic)
• Antisocial • Borderline • Histrionic • Narcissistic
Cluster C (anxious)
• Avoidant • Dependent • Obsessive–compulsive
Not specified
• Depressive • Passive-aggressive • Sadistic • Self-defeating • Psychopathy
• v • t • e [6]

Diagnosis of borderline personality disorder is based on a clinical assessment by a qualified mental health professional. The best method is to present the criteria of the disorder to patients and to ask them if they feel that these characteristics accurately describe them. Actively involving patients with BPD in determining their diagnosis can help them become more willing to accept it. Although some clinicians prefer not to tell patients with BPD what their diagnosis is, either from concern about the stigma attached to this condition or because BPD used to be considered untreatable, it is usually helpful for patients with BPD to know their diagnosis. This helps them know that others have had similar experiences and can point them toward effective treatments.

In general, the psychological evaluation includes asking the client about the beginning and severity of symptoms, as well as other questions about how symptoms impact the client's quality of life. Issues of particular note are suicidal ideations, experiences with self-harm, and thoughts about harming others. Diagnosis is based both on the client's report of his or her symptoms and on the clinician's own observations. Additional tests for BPD can include a physical exam and laboratory tests to rule out other possible triggers for symptoms, such as thyroid conditions or substance abuse.

Diagnostic and Statistical Manual

The Diagnostic and Statistical Manual of Mental Disorders fifth edition (DSM-5) has removed the multiaxial system. Consequently, all disorders, including personality disorders, are listed in Section II of the manual. A person must meet 5 of 9 criteria to receive a diagnosis of borderline personality disorder. The DSM-5 defines the main features of BPD as a pervasive pattern of instability in interpersonal relationships, self image, and affects, as well as markedly impulsive behavior.

In addition, the DSM-5 proposes alternative diagnostic criteria for Borderline personality disorder in section III, "Alternative DSM-5 Model for Personality Disorders." These alternative criteria are based on trait research and

include specifying at least four of seven maladaptive traits.

According to Marsha Linehan, many mental health professionals find it challenging to diagnose BPD using the DSM criteria, since these criteria describe such a wide variety of behaviors. To address this issue, Linehan has grouped the symptoms of BPD under five main areas of dysregulation: emotions, behavior, interpersonal relationships, sense of self, and cognition.

International Classification of Disease

The World Health Organization's ICD-10 defines a disorder that is conceptually similar to borderline personality disorder, called *(F 60.3* [1]*) Emotionally unstable personality disorder*. Its two subtypes are described below.[7]

F60.30 Impulsive type

At least three of the following must be present, one of which must be (2):

1. marked tendency to act unexpectedly and without consideration of the consequences;
2. marked tendency to engage in quarrelsome behavior and to have conflicts with others, especially when impulsive acts are thwarted or criticized;
3. liability to outbursts of anger or violence, with inability to control the resulting behavioral explosions;
4. difficulty in maintaining any course of action that offers no immediate reward;
5. unstable and capricious (impulsive, whimsical) mood.

F60.31 Borderline type

At least three of the symptoms mentioned in *F60.30 Impulsive type* must be present [see above], with at least two of the following in addition:

1. disturbances in and uncertainty about self-image, aims, and internal preferences;
2. liability to become involved in intense and unstable relationships, often leading to emotional crisis;
3. excessive efforts to avoid abandonment;
4. recurrent threats or acts of self-harm;
5. chronic feelings of emptiness.
6. demonstrates impulsive behavior, e.g., speeding, substance abuse

The ICD-10 also describes some general criteria that define what is considered a Personality disorder.

Millon's subtypes

Theodore Millon has proposed four subtypes of BPD. He suggests that an individual diagnosed with BPD may exhibit none, one, or more of the following:

Subtype	Features
Discouraged (including avoidant features)	Pliant, submissive, loyal, humble; feels vulnerable and in constant jeopardy; feels hopeless, depressed, helpless, and powerless.
Petulant (including negativistic features)	Negativistic, impatient, restless, as well as stubborn defiant, sullen, pessimistic, and resentful; easily slighted and quickly disillusioned.
Impulsive (including histrionic or antisocial features)	Capricious, superficial, flighty, distractible, frenetic, and seductive; fearing loss, becomes agitated, and gloomy and irritable; potentially suicidal.
Self-destructive (including depressive or masochistic features)	Inward-turning, intropunitively angry; conforming, deferential, and ingratiating behaviors have deteriorated; increasingly high-strung and moody; possible suicide.

Family members

People with BPD are prone to feeling angry at members of their family and alienated from them. On their part, family members often feel angry and helpless at how their BPD family members relate to them.[8]

A study in 2003 found that family members' experiences of burden, emotional distress, and hostility toward people with BPD were actually worse when they had greater knowledge about BPD. These findings may indicate a need to investigate the quality and accuracy of the information received by family members.

Parents of adults with BPD are often both over-involved and under-involved in family interactions. In romantic relationships, BPD is linked to increased levels of chronic stress and conflict, decreased satisfaction of romantic partners, abuse, and unwanted pregnancy. However, these links may apply to personality disorders in general.

Adolescence

Onset of symptoms typically occurs during adolescence or young adulthood, although symptoms suggestive of this disorder can sometimes be observed in children. Symptoms among adolescents that predict the development of BPD in adulthood may include problems with body-image, extreme sensitivity to rejection, behavioral problems, non-suicidal self-injury, attempts to find exclusive relationships, and severe shame.[] Many adolescents experience these symptoms without going on to develop BPD, but those who experience them are 9 times as likely as their peers to develop BPD. They are also more likely to develop other forms of long-term social disabilities.

Clinicians are discouraged from diagnosing anyone with BPD before the age of 18, due to the normal ups and downs of adolescence and a still-developing personality. However, BPD can sometimes be diagnosed before age 18, in which case the features must have been present and consistent for at least 1 year.

A BPD diagnosis in adolescence might predict that the disorder will continue into adulthood. Among adolescents who warrant a BPD diagnosis, there appears to be one group in which the disorder remains stable over time, and another group in which the individuals move in and out of the diagnosis. Earlier diagnoses may be helpful in creating a more effective treatment plan for the adolescent. Family therapy is considered a helpful component of treatment for adolescents with BPD.

Differential diagnosis and comorbidity

Lifetime comorbid (co-occurring) conditions are common in BPD. Compared to those diagnosed with other personality disorders, people with BPD showed a higher rate of also meeting criteria for

- mood disorders, including major depression and bipolar disorder
- anxiety disorders, including panic disorder, social anxiety disorder, and post-traumatic stress disorder (PTSD)
- other personality disorders
- substance abuse
- eating disorders, including anorexia nervosa and bulimia
- attention deficit hyperactivity disorderWikipedia:No original research#Primary, secondary and tertiary sources
- somatoform disorders
- dissociative disorders

Comorbid Axis I disorders

Axis I diagnosis	Overall (%)	Male (%)	Female (%)
Mood disorders	**75.0**	**68.7**	**80.2**
Major depressive disorder	32.1	27.2	36.1
Dysthymia	_9.7	_7.1	11.9
Bipolar I disorder	31.8	30.6	32.7
Bipolar II disorder	_7.7	_6.7	_8.5
Anxiety disorders	**74.2**	**66.1**	**81.1**
Panic disorder with agoraphobia	11.5	_7.7	14.6
Panic disorder without agoraphobia	18.8	16.2	20.9
Social phobia	29.3	25.2	32.7
Specific phobia	37.5	26.6	46.6
PTSD	39.2	29.5	47.2
Generalized anxiety disorder	35.1	27.3	41.6
Obsessive-compulsive disorder**	15.6	---	---
Substance use disorders	**72.9**	**80.9**	**66.2**
Any alcohol use disorder	57.3	71.2	45.6
Any drug use disorder	36.2	44.0	29.8
Eating disorders**	**53.0**	**20.5**	**62.2**
Anorexia nervosa**	20.8	_7 *	25 *
Bulimia nervosa**	25.6	10 *	30 *
Eating disorder not otherwise specified**	26.1	10.8	30.4
Somatoform disorders**	**10.3**	**10 ***	**10 ***
Somatization disorder**	_4.2	---	---
Hypochondriasis**	_4.7	---	---
Somatoform pain disorder**	_4.2	---	---
Psychotic disorders**	**1.3**	**_1 ***	**_1 ***
* Approximate values ** Values from 1998 study --- Value not provided by study			

|+Gender differences in Axis I lifetime comorbid diagnosis, 2008 and 1998

A 2008 study found that at some point in their lives, 75 percent of people with BPD meet criteria for mood disorders, especially major depression and Bipolar I, and nearly 75 percent meet criteria for an anxiety disorder. Nearly 73 percent meet criteria for substance abuse or dependency, and about 40 percent for PTSD. It is noteworthy that less than half of the participants with BPD in this study presented with PTSD, a prevalence similar to that reported in an earlier study. The finding that less than half of patients with BPD experience PTSD during their lives challenges the theory that BPD and PTSD are the same disorder.

There are marked gender differences in the types of co-morbid conditions a person with BPD is likely to have-- a higher percentage of males with BPD meet criteria for substance-use disorders, while a higher percentage of females with BPD meet criteria for PTSD and eating disorders. In one study, 38% of participants with BPD met the criteria for a diagnosis of ADHD. In another study, 6 of 41 participants (15%) met the criteria for an autism spectrum disorder (a subgroup that had significantly more frequent suicide attempts).

Regardless that it is an infradiagnosed disorder, a few studies have shown that the "lower expressions" of it might lead to wrong diagnoses. The many and shifting Axis I disorders in people with BPD can sometimes cause clinicians to miss the presence of the underlying personality disorder. However, since a complex pattern of Axis I diagnoses has been found to strongly predict the presence of BPD, clinicians can use the feature of a complex pattern of comorbidity as a clue that BPD might be present.

Mood disorders

Many people with borderline personality disorder also have mood disorders, such as major depressive disorder or a bipolar disorder. Some characteristics of BPD are similar to those of mood disorders, which can complicate the diagnosis. It is especially common for people to be misdiagnosed with bipolar disorder when they have borderline personality disorder, or vice versa. For someone with bipolar disorder, behavior suggestive of BPD might appear while the client is experiencing an episode of major depression or mania, only to disappear once the client's mood has stabilized. For this reason, it is ideal to wait until the client's mood has stabilized before attempting to make a diagnosis.

At face value, the affective lability of BPD and the rapid mood cycling of bipolar disorders can seem very similar. It can be difficult even for experienced clinicians, if they are unfamiliar with BPD, to differentiate between the mood swings of these two conditions. However, there are some clear differences.

First, the mood swings of BPD and bipolar disorder tend to have different durations. In some people with bipolar disorder, episodes of depression or mania last for at least two weeks at a time, which is much longer than moods last in people with BPD. Even among those who experience bipolar disorder with more rapid mood shifts, their moods usually last for days, while the moods of people with BPD can change in minutes or hours. So while euphoria and impulsivity in someone with BPD might resemble a manic episode, the experience would be too brief to qualify as a manic episode.

Second, the moods of bipolar disorder do not respond to changes in the environment, while the moods of BPD do respond to changes in the environment. That is, a positive event would not lift the depressed mood caused by bipolar disorder, but a positive event would potentially lift the depressed mood of someone with BPD. Similarly, a negative event would not dampen the euphoria caused by bipolar disorder, but a negative event would dampen the euphoria of someone with borderline personality disorder.

Third, when people with BPD experience euphoria, it is usually without the racing thoughts and decreased need for sleep that are typical of hypomania. And severe, high levels of sleep disturbance are rarely a symptom of BPD, whereas they are a common symptom of bipolar disorders (along with appetite disturbance).

Because the two conditions have a number of similar symptoms, BPD was once considered to be a mild form of bipolar disorder, or to exist on the bipolar spectrum. However, this would require that the underlying mechanism causing these symptoms be the same for both conditions. Differences in phenomenology, family history, longitudinal course, and responses to treatment indicate that this is not the case. Researchers have found "only a modest association" between bipolar disorder and borderline personality disorder, with "a strong spectrum relationship with [BPD and] bipolar disorder extremely unlikely." Benazzi et al. suggest that the DSM-IV BPD diagnosis combines two unrelated characteristics: an affective instability dimension related to Bipolar-II, and an impulsivity dimension not related to Bipolar-II.

Premenstrual dysphoric disorder

Premenstrual dysphoric disorder (PMDD) occurs in 3–8 percent of women. Symptoms begin 5–11 days before a woman's period and cease a few days after it begins. Symptoms may include: marked mood swings, irritability, depressed mood, feeling hopeless or suicidal, a subjective sense of being overwhelmed or out of control, anxiety, binge eating, difficulty concentrating, and substantial impairment of interpersonal relationships. Women with PMDD typically begin to experience symptoms in their early twenties, although many do not seek treatment until their early

thirties. Although some of the symptoms of PMDD and BPD are similar, they are different disorders. They are distinguishable by the timing and duration of symptoms, which are markedly different: the symptoms of PMDD occur only during the luteal phase of a woman's menstrual cycle, whereas BPD symptoms occur persistently at all stages of the menstrual cycle. In addition, the symptoms of PMDD do not include impulsivity.

Comorbid Axis II disorders

Axis II diagnosis	Overall (%)	Male (%)	Female (%)
Any Cluster A	**50.4**	**49.5**	**51.1**
Paranoid	21.3	16.5	25.4
Schizoid	12.4	11.1	13.5
Schizotypal	36.7	38.9	34.9
Any Other Cluster B	**49.2**	**57.8**	**42.1**
Antisocial	13.7	19.4	_9.0
Histrionic	10.3	10.3	10.3
Narcissistic	38.9	47.0	32.2
Any Cluster C	**29.9**	**27.0**	**32.3**
Avoidant	13.4	10.8	15.6
Dependent	_3.1	_2.6	_3.5
Obsessive-compulsive	22.7	21.7	23.6

|+Percentage of people with BPD and a lifetime comorbid Axis II diagnosis, 2008 More than two-thirds of people diagnosed with BPD also meet the criteria for another Axis II personality disorder at some point in their lives. (In a 2008 study, the rate was 73.9 percent.) Cluster A disorders, which include paranoid, schizoid, and schizotypal, are the most common, with a prevalence of 50.4 percent in people with BPD. The second most common are another Cluster B disorder, which include antisocial, histrionic, and narcissistic. These have an overall prevalence of 49.2 percent in people with BPD, with narcissistic being the most common, at 38.9 percent; antisocial the second most common, at 13.7 percent; and histrionic the least common, at 10.3 percent. The least common are Cluster C disorders, which include avoidant, dependent, and obsessive-compulsive, and have a prevalence of 29.9 percent in people with BPD. The percentages for specific comorbid Axis II disorders can be found in the table to the right.

Causes

As is the case with other mental disorders, the causes of BPD are complex and not fully agreed upon. Evidence suggests that BPD and post-traumatic stress disorder (PTSD) may be related in some way. Most researchers agree that a history of childhood trauma can be a contributing factor, but less attention has historically been paid to investigating the causal roles played by congenital brain abnormalities, genetics, neurobiological factors, and environmental factors other than trauma.

Genetics

The heritability of BPD is estimated to be 65%. That is, 65 percent of the variability in symptoms among different individuals with BPD can be explained by genetic differences. (Note that this is different from saying that 65 percent of BPD is "caused" by genes.) Twin studies may overestimate the effect of genes on variability in personality disorders due to the complicating factor of a shared family environment.

Twin, sibling and other family studies indicate partial heritability for impulsive aggression, but studies of serotonin-related genes have suggested only modest contributions to behavior.

Brain abnormalities

Hippocampus

The hippocampus tends to be smaller in people with BPD, as it is in people with post-traumatic stress disorder (PTSD). However, in BPD, unlike PTSD, the amygdala also tends to be smaller.

Amygdala

The amygdala is smaller and more active in people with BPD. Decreased amygdala volume has also been found in people with obsessive-compulsive disorder. One study has found unusually strong activity in the left amygdalas of people with BPD when they experience and view displays of negative emotions. Since the amygdala is a major structure involved in generating negative emotions, this unusually strong activity may explain the unusual strength and longevity of fear, sadness, anger, and shame experienced by people with BPD, as well as their heightened sensitivity to displays of these emotions in others.

Prefrontal cortex

The prefrontal cortex tends to be less active in people with BPD, especially when recalling memories of abandonment. This relative inactivity occurs in the right anterior cingulate (areas 24 and 32). Given its role in regulating emotional arousal, the relative inactivity of the prefrontal cortex might explain the difficulties people with BPD experience in regulating their emotions and responses to stress.

Hypothalamic-pituitary-adrenal axis

The hypothalamic-pituitary-adrenal axis (HPA axis) regulates cortisol production, which is released in response to stress. Cortisol production tends to be elevated in people with BPD, indicating a hyperactive HPA axis in these individuals. This causes them to experience a greater biological stress response, which might explain their greater vulnerability to irritability. Since traumatic events can increase cortisol production and HPA axis activity, one possibility is that the prevalence of higher than average activity in the HPA axis of people with BPD may simply be a reflection of the higher than average prevalence of traumatic childhood and maturational events among people with BPD. Another possibility is that, by heightening their sensitivity to stressful events, increased cortisol production may predispose those with BPD to experience stressful childhood and maturational events as traumatic.

Increased cortisol production is also associated with an increased risk of suicidal behavior.

Neurobiological factors

Estrogen

Individual differences in women's estrogen cycles may be related to the expression of BPD symptoms in female patients. A 2003 study found that women's BPD symptoms were predicted by changes in estrogen levels throughout their menstrual cycles, an effect that remained significant when the results were controlled for a general increase in negative affect.

Symptoms experienced due to disturbed levels of estrogen are often misdiagnosed as BPD, like extreme mood swings and depression. As endometriosis is an estrogen responsive disease, severe PMS and PMDD symptoms are observed, that are both physical and psychological in nature. Hormone-responsive mood disorders also known as reproductive depression are seen to cease only after menopause or hysterectomy. Psychotic episodes treated with estrogen in women with BPD show considerable improvement but must not be prescribed to those with endometriosis as it worsens their endocrine condition. Mood stabilizing drugs used for bipolar disorder do not help patients with disturbed estrogen levels. A correct diagnosis between endocrine disorder and psychiatric disorder must be made.[*citation needed*]

Adverse childhood experiences

There is a strong correlation between child abuse, especially child sexual abuse, and development of BPD. Many individuals with BPD report a history of abuse and neglect as young children. Patients with BPD have been found to be significantly more likely to report having been verbally, emotionally, physically or sexually abused by caregivers of either gender. They also report a high incidence of incest and loss of caregivers in early childhood.

Individuals with BPD were also likely to report having caregivers of all sexes deny the validity of their thoughts and feelings. Caregivers were also reported to have failed to provide needed protection and to have neglected their child's physical care. Parents of all sexes were typically reported to have withdrawn from the child emotionally, and to have treated the child inconsistently. Additionally, women with BPD who reported a previous history of neglect by a female caregiver and abuse by a male caregiver were significantly more likely to report experiencing sexual abuse by a non-caregiver.

It has been suggested that children who experience chronic early maltreatment and attachment difficulties may go on to develop borderline personality disorder.

However, none of these studies provide evidence that childhood trauma necessarily causes or contributes to causing BPD. Rather, both the trauma and the BPD could be caused by a third factor. For example, it could be that many caregivers who tend to expose children to traumatic experiences do so partly because of their own heritable personality disorders, the genetic predisposition for which they may pass on to their children, who develop BPD as a result of that predisposition and other factors, and not as a result of prior mistreatment.[9]

Other developmental factors

The intensity and reactivity of a person's negative affectivity, or tendency to feel negative emotions, predicts BPD symptoms more strongly than does childhood sexual abuse. This finding, differences in brain structure (see Brain abnormalities), and the fact that some patients with BPD do not report a traumatic history, suggest that BPD is distinct from the post-traumatic stress disorder that frequently accompanies it. Thus researchers examine developmental causes in addition to childhood trauma.

Newer research published in January 2013, from Dr Anthony Ruocco at the University of Toronto, has highlighted two patterns of brain activity that may underlie the dysregulation of emotion indicated in this disorder; there has been described increased activity in the brain circuits responsible for the experience of heightened negative emotions, coupled with reduced activation of the brain circuits that normally regulate or suppress these generated negative emotions. These two neural networks are seen to be dysfunctionally operative in the frontolimbic regions

but the specific regions vary widely in individuals, which calls for the analysis of more neuroimaging studies. Also, differing from earlier studies, sufferers of BPD showed less activation in the amygdala in situations of increased negative emotionality than the control group. Dr. John Krystal, Editor of Biological Psychiatry added that: "This new report adds to the impression that people with borderline personality disorder are 'set-up' by their brains to have stormy emotional lives, although not necessarily unhappy or unproductive lives," [10]

Writing in the psychoanalytic tradition, Otto Kernberg argues that a child's failure to achieve the developmental task of psychic clarification of self and other and failure to overcome splitting might increase the risk of developing a borderline personality.

A child's inability to tolerate delayed gratification at age 4 does not predict later development of BPD.

Mediating and moderating factors

Executive function

While high rejection sensitivity is associated with stronger symptoms of borderline personality disorder, executive function appears to mediate the relationship between rejection sensitivity and BPD symptoms. That is, a group of cognitive processes that include planning, working memory, attention, and problem-solving might be the mechanism through which rejection sensitivity impacts BPD symptoms. A 2008 study found that the relationship between a person's rejection sensitivity and BPD symptoms was stronger when executive function was lower, and that the relationship was weaker when executive function was higher. This suggests that high executive function might help protect people with high rejection sensitivity against symptoms of BPD.

A 2012 study found that problems in working memory might contribute to greater impulsivity in people with BPD.

Family environment

Family environment mediates the effect of child sexual abuse on the development of BPD. An unstable family environment predicts the development of the disorder, while a stable family environment predicts a lower risk. One possible explanation is that a stable environment buffers against its development.

Self-complexity

Self-complexity, or considering one's self to have many different characteristics, appears to moderate the relationship between Actual-Ideal self-discrepancy and the development of BPD symptoms. That is, for individuals who believe that their actual characteristics do not match the characteristics that they hope to acquire, high self-complexity reduces the impact of their conflicted self-image on BPD symptoms. However, self-complexity does not moderate the relationship between Actual-Ought self-discrepancy and the development of BPD symptoms. That is, for individuals who believe that their actual characteristics do not match the characteristics that they should already have, high self-complexity does not reduce the impact of their conflicted self-image on BPD symptoms. The protective role of self-complexity in Actual-Ideal self-discrepancy, but not in Actual-Ought self-discrepancy, suggests that the impact of conflicted or unstable self-image in BPD depends on whether the individual views self in terms of characteristics that she hopes to acquire, or in terms of characteristics that she should already have.

Thought suppression

A 2005 study found that thought suppression, or conscious attempts to avoid thinking certain thoughts, mediates the relationship between emotional vulnerability and BPD symptoms. A later study found that the relationship between emotional vulnerability and BPD symptoms is not necessarily mediated by thought suppression. However, this study did find that thought suppression mediates the relationship between an invalidating environment and BPD symptoms.

Management

Psychotherapy is the primary treatment for borderline personality disorder. Treatments should be based on the needs of the individual, rather than upon the general diagnosis of BPD. Medications are useful for treating comorbid disorders, such as depression and anxiety. Short-term hospitalization has not been found to be more effective than community care for improving outcomes or long-term prevention of suicidal behavior in those with BPD.

Psychotherapy

Long-term psychotherapy is currently the treatment of choice for BPD. There are five such treatments available: mentalization-based treatment (MBT), transference-focused psychotherapy, dialectical behavior therapy (DBT), general psychiatric management, and schema-focused therapy. While DBT is the therapy that has been studied the most, empirical research has shown that all of these treatments are effective for treating BPD, except for schema-focused therapy.[] Long-term therapy of any kind, including schema-focused therapy, is better than no treatment, especially in reducing urges to self-injure.

Mentalization-based therapy and transference-focused psychotherapy are based on psychodynamic principles, while dialectical behavior therapy is based on cognitive-behavioral principles and mindfulness. General psychiatric management combines the core principles from each of these treatments, and it is considered easier to learn and less intensive. Randomized controlled trials have shown that DBT and MBT are the most effective, and the two share many similarities. Researchers are interested in developing shorter versions of these therapies to increase accessibility, to relieve the financial burden on patients, and to relieve the resource burden on treatment providers.

From a psychodynamic perspective, a special problem of psychotherapy with people with BPD is intense projection. It requires the psychotherapist to be flexible in considering negative attributions by the patient rather than quickly interpreting the projection.

Medications

A 2010 review by the Cochrane collaboration found that no medications show promise for "the core BPD symptoms of chronic feelings of emptiness, identity disturbance and abandonment." However, the authors found that some medications may impact isolated symptoms associated with BPD or the symptoms of comorbid conditions.

Of the typical antipsychotics studied in relation to BPD, haloperidol may reduce anger, and flupenthixol may reduce the likelihood of suicidal behavior. Among the atypical antipsychotics, aripiprazole may reduce interpersonal problems, impulsivity, anger, psychotic paranoid symptoms, depression, anxiety, and general psychiatric pathology. Olanzapine may decrease affective instability, anger, psychotic paranoid symptoms, and anxiety, but a placebo had a greater ameliorative impact on suicidal ideation than olanzapine did. The effect of Ziprasidone was not significant.

Of the mood stabilizers studied, valproate semisodium may ameliorate depression, interpersonal problems, and anger. Lamotrigine may reduce impulsivity and anger; topiramate may ameliorate interpersonal problems, impulsivity, anxiety, anger and general psychiatric pathology. The effect of carbamazepine was not significant. Of the antidepressants, amitriptyline may reduce depression, but mianserin, fluoxetine, fluvoxamine and phenelzine sulfate showed no effect. Omega-3 fatty acid may ameliorate suicidality and improve depression. As of 2010, trials with these medications had not been replicated, and the effect of long-term use had not been assessed.

Because of weak evidence and the potential for serious side effects from some of these medications, the UK National Institute for Health and Clinical Excellence (NICE) 2009 clinical guideline for the treatment and management of BPD recommends: "Drug treatment should not be used specifically for borderline personality disorder or for the individual symptoms or behavior associated with the disorder." However, "drug treatment may be considered in the overall treatment of comorbid conditions." They suggest a "review of the treatment of people with borderline personality disorder who do not have a diagnosed comorbid mental or physical illness and who are currently being prescribed drugs, with the aim of reducing and stopping unnecessary drug treatment."

Services

Individuals with BPD sometimes use mental health services extensively. They accounted for about 20 percent of psychiatric hospitalizations in one survey. The majority of patients with BPD continue to use outpatient treatment in a sustained manner for several years, but the number using the more restrictive and costly forms of treatment, such as inpatient admission, declines with time. Experience of services varies. Assessing suicide risk can be a challenge for clinicians, and patients themselves tend to underestimate the lethality of self-injurious behaviors. People with BPD typically have a chronically elevated risk of suicide much above that of the general population and a history of multiple attempts when in crisis. Approximately half the individuals who commit suicide meet criteria for a personality disorder. Borderline personality disorder remains the most commonly associated personality disorder with suicide.

Prognosis

With treatment, the majority of people with BPD can find relief from distressing symptoms and achieve remission, defined as a consistent relief from symptoms for at least two years. A longitudinal study tracking the symptoms of people with BPD found that 34.5% achieved remission within two years from the beginning of the study. Within four years, 49.4% had achieved remission, and within six years, 68.6% had achieved remission. By the end of the study, 73.5% of participants were found to be in remission. Moreover, of those who achieved recovery from symptoms, only 5.9% experienced recurrences. A later study found that ten years from baseline (during a hospitalization), 86% of patients had sustained and stable recovery from symptoms.

Thus contrary to popular belief, recovery from BPD is not only possible but common, even for those with the most severe symptoms. However, it is important to note that these high rates of relief from distressing symptoms have only been observed among those who receive treatment of some kind.

Patient personality can play an important role during the therapeutic process, leading to better clinical outcomes. Recent research has shown that BPD patients with higher levels of trait agreeableness undergoing Dialectical Behavior Therapy (DBT) exhibited better clinical outcomes than other patients either low in Agreeableness or not being treated with DBT. This association was mediated through the strength of a working alliance between patient and therapist; that is, more Agreeable patients developed stronger working alliances with their therapists which in turn led to better clinical outcomes.

In addition to recovering from distressing symptoms, people with BPD also achieve high levels of psychosocial functioning. A longitudinal study tracking the social and work abilities of participants with BPD found that six years after diagnosis, 56% of participants had good function in work and social environments, compared to 26% of participants when they were first diagnosed. Vocational achievement was generally more limited, even compared to those with other personality disorders. However, those whose symptoms had remitted were significantly more likely to have good relationships with a romantic partner and at least one parent, good performance at work and school, a sustained work and school history, and good psychosocial functioning overall.

Epidemiology

The prevalence of BPD was initially estimated to be 1 to 2 percent of the general population and to occur three times more often in women than in men. However, the lifetime prevalence of BPD in a 2008 study was found to be 5.9% of the general population, occurring in 5.6% of men and 6.2% of women. The difference in rates between men and women in this study was not found to be statistically significant.

Borderline personality disorder is estimated to contribute to 20 percent of psychiatric hospitalizations, and to occur among 10 percent of outpatients.

29.5 percent of new inmates in Iowa fit a diagnosis of borderline personality disorder in 2007, and the overall prevalence of BPD in the U.S. prison population is thought to be 17 percent. These high numbers may be related to the high frequency of substance abuse and substance use disorders among people with BPD, which is estimated at 38 percent.

History

The coexistence of intense, divergent moods within an individual was recognized by Homer, Hippocrates and Aretaeus, the last describing the vacillating presence of impulsive anger, melancholia and mania within a single person. The concept was revived by Swiss physician Théophile Bonet in 1684 who, using the term *folie maniaco-mélancolique*, described the phenomenon of unstable moods that followed an unpredictable course. Other writers noted the same pattern, including the American psychiatrist C. Hughes in 1884 and J.C. Rosse in 1890, who called the disorder "borderline insanity". In 1921, Kraepelin identified an "excitable personality" that closely parallels the borderline features outlined in the current concept of BPD.

The first significant psychoanalytic work to use the term "borderline" was written by Adolf Stern in 1938. It described a group of patients suffering from what he thought to be a mild form of schizophrenia, on the borderline between neurosis and psychosis.

The 1960s and 1970s saw a shift from thinking of the condition as borderline schizophrenia to thinking of it as a borderline affective disorder (mood disorder), on the fringes of bipolar disorder, cyclothymia and dysthymia. In the DSM-II, stressing the intensity and variability of moods, it was called cyclothymic personality (affective personality). While the term "borderline" was evolving to refer to a distinct category of disorder, psychoanalysts such as Otto Kernberg were using it to refer to a broad spectrum of issues, describing an intermediate level of personality organization between neurosis and psychosis.

After standardized criteria were developed to distinguish it from mood disorders and other Axis I disorders, BPD became a personality disorder diagnosis in 1980 with the publication of the DSM-III. The diagnosis was distinguished from sub-syndromal schizophrenia, which was termed "Schizotypal personality disorder". The DSM-IV Axis II Work Group of the American Psychiatric Association finally decided on the name "borderline personality disorder," which is still in use by the DSM-IV today. However, the term "borderline" has been described as uniquely inadequate for describing the symptoms characteristic of this disorder.

Controversies

Credibility and validity of testimony

The credibility of individuals with personality disorders has been questioned at least since the 1960s. Two concerns are the incidence of dissociative episodes among people with BPD, and the belief that lying is a key component of this condition.

Dissociation

Researchers disagree about whether dissociation, or a sense of detachment from emotions and physical experiences, impacts the ability of people with BPD to recall the specifics of past events. A 1999 study reported that the specificity of autobiographical memory was decreased in BPD patients. The researchers found that decreased ability to recall specifics was correlated with patients' levels of dissociation. However, a larger study in 2010 found that people with BPD and without depression had more specific autobiographical memory than did people without BPD and with depression. The presence of depression (though not its severity) was the main factor related to a decreased ability to recall the specifics of past events. This decreased ability was found to be unrelated to dissociation and other symptoms of BPD, thus supporting the reliability of the testimony of people with BPD.

Lying as a feature of BPD

Some theorists argue that patients with BPD often lie. However, others write that they have rarely seen lying among patients with BPD in clinical practice. Regardless, lying is not one of the diagnostic criteria for BPD.

The belief that lying is a distinguishing characteristic of BPD can impact the quality of care that people with this diagnosis receive in the legal and healthcare systems. For instance, Jean Goodwin relates an anecdote of a patient with multiple personality disorder, now called dissociative identity disorder, who suffered from pelvic pain due to traumatic events in her childhood. Due to their disbelief in her accounts of these events, physicians diagnosed her with borderline personality disorder, reflecting a belief that lying is a key feature of BPD. Based upon her BPD diagnosis, the physicians then disregarded the patient's assertion that she was allergic to adhesive tape. The patient was in fact allergic to adhesive tape, which later caused complications in the surgery to relieve her pelvic pain.

Gender

Feminists question why women are three times more likely to be diagnosed with BPD than men, while other stigmatizing diagnoses, such as antisocial personality disorder, are diagnosed three times as often in men.

One explanation is that some of the diagnostic criteria of BPD uphold stereotypes about women. For example, the criteria of "a pattern of unstable personal relationships, unstable self-image, and instability of mood," can all be linked to the stereotype that women are "neither decisive nor constant". Women may be more likely to receive a personality disorder diagnosis if they reject the traditional female role by being assertive, successful, or sexually active. If a woman presents with psychiatric symptoms but does not conform to a traditional, passive sick role, she may be labelled as a "difficult" patient and given a BPD diagnosis.

Since BPD is a stigmatizing diagnosis even within the mental health community (see Stigma), some survivors of childhood sexual abuse who are diagnosed with BPD are thus re-traumatized by the negative responses they receive from healthcare providers. One camp argues that it would be better to diagnose these women with post-traumatic stress disorder, as this would acknowledge the impact of abuse on their behavior. Critics of the PTSD diagnosis argue that it medicalizes abuse rather than addressing the root causes in society. Regardless, a diagnosis of PTSD does not encompass all aspects of the disorder (see Brain abnormalities and Terminology).

Manipulative behavior

Manipulative behavior to obtain nurturance is considered by the DSM-IV-TR and many mental health professionals to be a defining characteristic of borderline personality disorder. However, Marsha Linehan notes that doing so relies upon the assumption that people with BPD who communicate intense pain, or who engage in self-harm and suicidal behavior, do so with the intention of influencing the behavior of others. The impact of such behavior on others – often an intense emotional reaction in concerned friends, family members, and therapists – is thus assumed to have been the person's intention.

However, since people with BPD lack the ability to successfully manage painful emotions and interpersonal challenges, their frequent expressions of intense pain, self-harming, or suicidal behavior may instead represent a method of mood regulation or an escape mechanism from situations that feel unbearable. Linehan notes that if, for example, one were to withhold pain medication from burn victims and cancer patients, leaving them unable to regulate their severe pain, they would also exhibit "attention-seeking" and self-destructive behavior in order to cope.

Stigma

The features of BPD include emotional instability, intense unstable interpersonal relationships, a need for intimacy, and a fear of rejection. As a result, people with BPD often evoke intense emotions in those around them. Pejorative terms to describe people with BPD, such as "difficult," "treatment resistant," "manipulative," "demanding" and "attention seeking," are often used, and may become a self-fulfilling prophecy as the negative treatment of these individuals triggers further self-destructive behavior.

Physical violence

The stigma surrounding borderline personality disorder includes the belief that people with BPD are prone to violence toward others. While movies and visual media often sensationalize people with BPD by portraying them as violent, the majority of researchers agree that people with BPD are unlikely to physically harm others. Although people with BPD often struggle with experiences of intense anger, a defining characteristic of BPD is that they direct it inward toward themselves. One of the key differences between BPD and antisocial personality disorder (ASPD) is that people with BPD tend to internalize anger by hurting themselves, while people with ASPD tend to externalize it by hurting others. In addition, adults with BPD have often experienced abuse in childhood, so many people with BPD adopt a "no-tolerance" policy toward expressions of anger of any kind. Their extreme aversion to violence can cause many people with BPD to overcompensate and experience difficulties being assertive and expressing their needs. This is one way in which people with BPD choose to harm themselves over potentially causing harm to others. Another way in which people with BPD avoid expressing their anger through violence is by causing physical damage to themselves, such as engaging in non-suicidal self injury.

Mental healthcare providers

People with BPD are considered to be among the most challenging groups of patients to work with in therapy, requiring a high level of skill and training in the psychiatrists, therapists and nurses involved in their treatment. A majority of psychiatric staff report finding individuals with BPD moderately to extremely difficult to work with, and more difficult than other client groups. Efforts are ongoing to improve public and staff attitudes toward people with BPD.

In psychoanalytic theory, the stigmatization among mental healthcare providers may be thought to reflect countertransference (when a therapist projects their own feelings on to a client). Thus a diagnosis of BPD "often says more about the clinician's negative reaction to the patient than it does about the patient" and "explains away the breakdown in empathy between the therapist and the patient and becomes an institutional epithet in the guise of pseudoscientific jargon". This inadvertent countertransference can give rise to inappropriate clinical responses, including excessive use of medication, inappropriate mothering, and punitive use of limit setting and interpretation.

Some clients feel the diagnosis is helpful, allowing them to understand that they are not alone and to connect with others with BPD who have developed helpful coping mechanisms. However, others experience the term "Borderline Personality Disorder" as a pejorative label rather than an informative diagnosis. They report concerns that their self-destructive behavior is incorrectly perceived as manipulative, and that the stigma surrounding this disorder limits their access to healthcare. Indeed, mental health professionals frequently refuse to provide services to those who have received a BPD diagnosis.

Terminology

Because of the above concerns, and because of a move away from the original theoretical basis for the term (see history), there is ongoing debate about renaming Borderline Personality Disorder. While some clinicians agree with the current name, others argue that it should be changed, since many who are labelled with "Borderline Personality Disorder" find the name unhelpful, stigmatizing, or inaccurate. Valerie Porr, president of Treatment and Research Advancement Association for Personality Disorders states that "the name BPD is confusing, imparts no relevant or descriptive information, and reinforces existing stigma."

Alternative suggestions for names include *emotional regulation disorder* or *emotional dysregulation disorder*. *Impulse disorder* and *interpersonal regulatory disorder* are other valid alternatives, according to John Gunderson of McLean Hospital in the United States. Another term suggested by psychiatrist Carolyn Quadrio is *post traumatic personality disorganization* (PTPD), reflecting the condition's status as (often) both a form of chronic post traumatic stress disorder (PTSD) as well as a personality disorder. However, although many with BPD do have traumatic histories, some do not report any kind of traumatic event, which suggests that BPD is not necessarily a trauma spectrum disorder.

The Treatment and Research Advancements National Association for Personality Disorders (TARA-APD) campaigned unsuccessfully to change the name and designation of BPD in DSM-5, published in May 2013, in which the name Borderline personality disorder remains unchanged and it is not considered a Trauma- and stressor-related disorder.

Society and culture

Film and television

There are several films portraying characters either explicitly diagnosed or with traits suggestive of BPD. Some of these films may be misleading if they are thought to depict this disorder. The films *Play Misty for Me* and *Girl, Interrupted,* based on the memoir by Susanna Kaysen, with Winona Ryder playing Kaysen both suggest the emotional instability of the disorder; however, the first case shows a person more aggressive to others than to herself, which is not characteristic of the disorder. The 1992 film *Single White Female,* like the first example, also suggests characteristics, some of which are actually atypical of the disorder: the character Hedy suffers from a markedly disturbed sense of identity and abandonment leads to drastic measures.[11] The main character in the film American Psycho (2000), Patrick Bateman, was psychologically evaluated and diagnosed with borderline personality disorder with schizotypal features. In the HBO series *The Sopranos* Dr. Melfi, Tony Soprano's therapist, suggests that his mother may suffer from BPD and quotes from the DSM definition of the disorder. The characterization definitely exhibits all the traits. In the NBC sitcom "Will & Grace" Grace Adler asks best friend and roommate Will Truman to forge her doctor's signature to evade jury duty because of borderline personality disorder and a high risk for a psychotic break.[12] Another film directed by Lasse Hallström, *What's Eating Gilbert Grape,* shows a clear example of the disorder in the seductive neighbor Betty (Mary Steenburgen).

Psychiatrists Eric Bui and Rachel Rodgers argue that the character of Anakin Skywalker/Darth Vader in the *Star Wars* films meets six of the nine diagnostic criteria; Bui also found Anakin a useful example to explain BPD to medical students. In particular, Bui points to the character's abandonment issues, uncertainty over his identity, and

dissociative episodes. Other films attempting to depict characters with the disorder include *Fatal Attraction*, *The Crush*, *Mad Love*, *Malicious*, *Interiors*, *Notes On a Scandal*, *The Cable Guy*, *Mr. Nobody* and *Cracks*.

Unfortunately, dramatic portrayals of people with BPD in movies and other forms of visual media contribute to the stigma surrounding borderline personality disorder, especially the myth that people with BPD are violent toward others. The majority of researchers agree that in reality, people with BPD are very unlikely to harm others.

Literature

The Buddha and the Borderline: My Recovery from Borderline Personality Disorder through Dialectical Behavior Therapy, Buddhism, and Online Dating is a memoir by Kiera Van Gelder.

Girl, Interrupted is a memoir by American author Susanna Kaysen, relating her experiences as a young woman in a psychiatric hospital in the 1960s after being diagnosed with borderline personality disorder.

Get Me Out of Here: My Recovery from Borderline Personality Disorder is a memoir by author Rachel Reiland, relating her treatment and recovery from borderline personality disorder.

Songs of Three Islands, by Millicent Monks, is a memoir speculating about the impact of BPD upon the Carnegie family. Readers have criticized it for presenting a biased and stigmatizing view of BPD.

In Lois McMaster Bujold's science fiction novel *Komarr*, Tien Vorsoisson has BPD, per the author; his disorder drives a large part of the story.

Kreisman, Jerold J MD. *I Hate You—Don't Leave Me: Understanding the Borderline Personality*{{inconsistent citations}}

Awareness

In early 2008, the United States House of Representatives declared the month of May as Borderline Personality Disorder Awareness Month.[13]

Notes

[1] http://apps.who.int/classifications/icd10/browse/2010/en#/F60.3
[2] http://www.icd9data.com/getICD9Code.ashx?icd9=301.83
[3] http://www.nlm.nih.gov/medlineplus/ency/article/000935.htm
[4] http://emedicine.medscape.com/article/913575-overview
[5] http://www.nlm.nih.gov/cgi/mesh/2014/MB_cgi?field=uid&term=D001883
[6] http://en.wikipedia.org/w/index.php?title=Template:Personality_disorders_sidebar&action=edit
[7] Emotionally unstable personality disorder – [[International Statistical Classification of Diseases and Related Health Problems (http://www.mentalhealth.com/icd/p22-pe05.html)] 10th Revision (ICD-10) – World Health Organization]
[8] Gunderson 2011, p. 2040
[9] name="Harris-1998">
[10] Anthony C. Ruocco, Sathya Amirthavasagam, Lois W. Choi-Kain, Shelley F. McMain. Neural Correlates of Negative Emotionality in Borderline Personality Disorder: An Activation-Likelihood-Estimation Meta-Analysis" *Biological Psychiatry* 2013; 73 (2) 153
[11] Robinson (*Reel Psychiatry: Movie Portrayals of Psychiatric Conditions*), p. 235
[12] http://www.durfee.net/will/scripts/s0312.htm
[13] HR 1005, 4/1/08

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Further reading

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External links

- Borderline personality disorder (http://www.dmoz.org/Health/Mental_Health/Disorders/Personality/Borderline/) on the Open Directory Project
- "Borderline Personality Disorder" (http://www.nimh.nih.gov/topics/topic-page-borderline-personality-disorder.shtml). National Institute of Mental Health.

Conduct disorder

Conduct disorder	
Classification and external resources	
ICD-10	F91 [1]
ICD-9	312 [2]
MedlinePlus	000919 [3]
MeSH	D019955 [4]

Conduct disorder is a psychological disorder diagnosed in childhood or adolescence that presents itself through a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate norms are violated. These behaviors are often referred to as "antisocial behaviors."[5] Indeed, the disorder is often seen as the precursor to antisocial personality disorder, which is not diagnosed until the individual is 18 years old.

Diagnosis

Conduct disorder is classified in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM).[6] It is diagnosed based on a prolonged pattern of antisocial behaviour such as serious violation of laws and social norms and rules. There are no proposed revisions for the main criteria of conduct disorder in the *DSM-5*; there is a recommendation by the work group to add an additional specifier for callous and unemotional traits. Almost all adolescents who have a substance use disorder have conduct disorder-like traits, but after successful treatment of the substance use disorder, about half of these adolescents no longer display conduct disorder-like symptoms. Therefore it is important to exclude a substance-induced cause and instead address the substance use disorder prior to making a psychiatric diagnosis of conduct disorder.

Etiology

While the etiology (origin or cause) of conduct disorder is complicated by an intricate interplay of biological and environmental factors, identifying etiological mechanisms is crucial for obtaining accurate assessment and implementing effective treatment.[7] These mechanisms serve as the fundamental building blocks on which evidence-based treatments are developed. Despite the complexities, several domains have been implicated in the development of conduct disorder including cognitive variables, neurological factors, intraindividual factors, familial and peer influences, and wider contextual factors. These factors may also vary based on the age of onset, with different variables related to early (e.g., neurodevelopmental basis) and adolescent (e.g., social/peer relationships) onset.[8]

Cognitive factors

In terms of cognitive function, intelligence and cognitive deficits are common amongst youths with conduct disorder, particularly those with early-onset and have intelligence quotients (IQ) one standard deviation below the mean[9] and severe deficits in verbal reasoning and executive function.[10] Executive function difficulties may manifest in terms of one's ability to shift between tasks, plan as well as organize, and also inhibit a prepotent response. These findings hold true even after taking into account other variables such as socioeconomic status (SES), and education. However, IQ and executive function deficits are only one piece of the puzzle, and the magnitude of their influence is increased during transactional processes with environmental factors.[11]

Structural and functional brain differences

Beyond difficulties in executive function, youth with conduct disorder may also demonstrate differences in brain anatomy and function. Compared to normal controls, youths with early and adolescent onset of conduct disorder displayed reduced responses in brain regions associated with antisocial behavior (i.e., amygdala, ventromedial prefrontal cortex, insula, and orbitofrontal cortex). In addition, youths with conduct disorder also demonstrated less responsiveness in the orbitofrontal regions of the brain during a stimulus-reinforcement and reward task.[12] This provides a neural explanation for why youths with conduct disorder may be more likely to repeat poor decision making patterns. Lastly, youths with conduct disorder display a reduction in grey matter volume in the amygdala, which may account for the fear conditioning deficits.[13] This reduction has been linked to difficulty processing social emotional stimuli, regardless of the age of onset.[14] Aside from the differences in neuroanatomy and activation patterns between youth with conduct disorder and controls, neurochemical profiles also vary between groups. Individuals with conduct disorder are characterized as having reduced serotonin and cortisol levels (e.g., reduced hypothalamic-pituitary-adrenal (HPA) axis), as well as reduced autonomic nervous system (ANS) functioning. These reductions are associated with the inability to regulate mood and impulsive behaviors, weakened signals of anxiety and fear, and decreased self-esteem. Taken together, these findings may account for some of the variance in the psychological and behavioral patterns of youth with conduct disorder.

Intra-individual factors

Aside from findings related to neurological and neurochemical profiles of youth with conduct disorder, intraindividual factors such as genetics may also be relevant. Having a sibling or parent with conduct disorder increases the likelihood of having the disorder, with a heritability rate of .53.[15] There also tends to be a stronger genetic link for individuals with childhood-onset compared to adolescent onset.[16] In addition, youth with conduct disorder also exhibit polymorphism in the monoamine oxidase A gene,[17] low resting heart rates,[18] and increased testosterone.[19]

Family and peer influences

Elements of the family and social environment may also play a role in the development and maintenance of conduct disorder. For instance, antisocial behavior suggestive of conduct disorder is associated with single parent status, parental divorce, large family size, and young age of mothers. However, these factors are difficult to tease apart from other demographic variables that are known to be linked with conduct disorder, including poverty and low SES. Family functioning and parent-child interactions also play a substantial role in childhood aggression and conduct disorder, with low levels of parental involvement, inadequate supervision, and unpredictable discipline practices reinforcing youth's defiant behaviors. Peer influences have also been related to the development of antisocial behavior in youth, particularly peer rejection in childhood and association with deviant peers. Peer rejection is not only a marker of a number of externalizing disorders, but also a contributing factor for the continuity of the disorders over time. Hinshaw and Lee (2003) also explain that association with deviant peers has been thought to influence the development of conduct disorder in two ways: 1) a "selection" process whereby youth with aggressive characteristics choose deviant friends, and 2) a "facilitation" process whereby deviant peer networks bolster patterns of antisocial behavior. In a separate study by Bonin and colleagues, parenting programs were shown to positively affect child behavior and reduce costs to the public sector.[20]

Wider contextual factors

In addition to the individual and social factors associated with conduct disorder, research has highlighted the importance of environment and context in youth with antisocial behavior. However, it is important to note that these are not static factors, but rather transactional in nature (e.g., individuals are influenced by and also influence their environment). For instance, neighborhood safety and exposure to violence has been studied in conjunction with conduct disorder, but it is not simply the case that youth with aggressive tendencies reside in violent neighborhoods. Transactional models propose that youth may resort to violence more often as a result of exposure to community violence, but their predisposition towards violence also contributes to neighborhood climate.

Developmental course

Currently, there are thought to be two possible developmental courses to conduct disorder. The first is known as the "childhood-onset type" and occurs when conduct disorder symptoms are present before the age of 10 years. This course is often linked to a more persistent life course and more pervasive behaviors. Specifically, children in this group have greater levels of ADHD symptoms, neuropsychological deficits, more academic problems, increased family dysfunction, and higher likelihood of aggression and violence.[21]

There is debate among professionals regarding the validity of diagnosing young children with conduct disorder. The characteristics of the diagnosis are commonly seen in young children that are referred to mental health professionals.[22] It is concerning that a premature diagnosis may be made in young children, and thus labeling an individual may be inappropriate. It is also argued that some children may not in fact have conduct disorder, but are engaging in developmentally appropriate disruptive behavior.

The second developmental course is known as the "adolescent-onset type" and occurs when conduct disorder symptoms are present after the age of 10 years. Individuals with adolescent-onset conduct disorder exhibit less impairment than those with the childhood-onset type and are not characterized by similar psychopathology.[23] At times, these individuals will remit in their deviant patterns before adulthood. Research has shown that there is a greater number of children with adolescent-onset conduct disorder than those with childhood-onset, suggesting that adolescent-onset conduct disorder is an exaggeration of developmental behaviors that are typically seen in adolescence, such as rebellion against authority figures and rejection of conventional values. However, this argument is not established[24] and empirical research suggests that these subgroups are not as valid as once thought.

In addition to these two courses that are recognized by the DSM-IV-TR, there appears to be a relationship among oppositional defiant disorder, conduct disorder and antisocial personality disorder. Specifically, research has demonstrated continuity in the disorders such that conduct disorder is often diagnosed in children who have been previously diagnosed with oppositional defiant disorder, and most adults with antisocial personality disorder were previously diagnosed with conduct disorder. For example, some research has shown that 90% of children diagnosed with conduct disorder had a previous diagnosis of oppositional defiant disorder.[25] Moreover, both disorders share relevant risk factors and disruptive behaviors, suggesting that oppositional defiant disorder is a developmental precursor and milder variant of conduct disorder. However, this is not to say that this trajectory occurs in all individuals. In fact, only about 25% of children with oppositional defiant disorder will receive a later diagnosis of conduct disorder. Correspondingly, there is an established link between conduct disorder and the diagnosis of antisocial personality disorder as an adult. In fact, the current diagnostic criteria for antisocial personality disorder require a conduct disorder diagnosis before the age of 15. However, again, only 25-40% of youths with conduct disorder will develop antisocial personality disorder.[26] Nonetheless, many of the individuals who do not meet full criteria for antisocial personality disorder still exhibit a pattern of social and personal impairments or antisocial behaviors.[27] These developmental trajectories suggest the existence of antisocial pathways in certain individuals, which have important implications for both research and treatment.

Epidemiology

Prevalence and incidence

Prevalence estimates for conduct disorder range from 1-10%. However, among incarcerated youth or youth in juvenile detention facilities, rates of conduct disorder are between 23% and 87%.[28]

Gender differences

The majority of research on conduct disorder suggests that there are a significantly greater number of males than females with the diagnosis, with some reports demonstrating a threefold to fourfold difference in prevalence.[29] However, this difference may be somewhat biased by the diagnostic criteria which focus on more overt behaviors, such as aggression and fighting, which are more often exhibited by males. Females are more likely to be characterized by covert behaviors, such as stealing or running away. Moreover, conduct disorder in females is linked to several negative outcomes, such as antisocial personality disorder and early pregnancy,[30] suggesting that sex differences in disruptive behaviors need to be more fully understood.

Females are more responsive to peer pressure[31] including feelings of guilt[32] than males.

Racial and ethnic differences in the US

Research on racial or cultural differences on the prevalence or presentation of conduct disorder is limited. However, it appears that African-American youth are more often diagnosed with conduct disorder,[33] while Asian-American youth are about one-third as likely[34] to develop conduct disorder when compared to White American youth.

Risk and protective factors

It is important to note that the development of conduct disorder is not immutable or predetermined. There is a number of interactive risk and protective factors that can influence and change outcomes, and in most cases conduct disorder develops due to an interaction and gradual accumulation of risk factors.[35] In addition to the risk factors identified under etiology, several other variables place youth at increased risk for developing the disorder, including child physical abuse and prenatal alcohol abuse and maternal smoking during pregnancy.[36] Protective factors have also been identified, and most notably include high IQ, being female, positive social orientations, good coping skills, and supportive family and community relationships.[37]

Lack of empathy

Empathy is recognizing feelings that other people are experiencing; lack of empathy is inability to recognize feelings of others. The child diagnosed with CD often presents with a lack of empathy. Because the child with CD is unable to place themselves in the other person's shoes, they are unable to understand their consequences.

One of the factors of conduct disorder is a lower level of fear. Research performed on the impact of toddlers who are exposed to fear and distress show negative emotionality (fear) predict toddlers' empathy-related responding to distress. The findings support that if a caregiver is able to respond to infant cues, the toddler has a better ability to respond to fear and distress. If a child does not learn how to handle fear or distress the child will be more likely to lash out at other children. If the caregiver is able to provide therapeutic intervention teaching children at risk better empathy skills, the child will have a lower incident level of conduct disorder.[38]

Comorbidity

Children with conduct disorder have a high risk of developing other adjustment problems. Specifically, risk factors associated with conduct disorder and the effects of conduct disorder symptomatolology on a child's psychosocial context have been linked to overlap with other psychological disorders.[39] In this way, there seems to be reciprocal effects of comorbidity with certain disorders, leading to increased overall risk for these youth.

Attention deficit hyperactivity disorder

ADHD is the condition most commonly associated with conduct disorders, with approximately 25-30% of boys and 50-55% of girls with conduct disorder having a comorbid ADHD diagnosis.[40] While it is unlikely that ADHD alone is a risk factor for developing conduct disorder, children who exhibit hyperactivity and impulsivity along with aggression is associated with the early onset of conduct problems. Moreover, children with comorbid conduct disorder and ADHD show more severe aggression.

Substance use disorders

Conduct disorder is also highly associated with both substance use and abuse. Children with conduct disorder have an earlier onset of substance use, as compared to their peers, and also tend to use multiple substances.[41] However, substance use disorders themselves can directly or indirectly cause conduct disorder like traits in about half of adolescents who have a substance use disorder. As mentioned above, it seems that there is a transactional relationship between substance use and conduct problems, such that aggressive behaviors increase substance use, which leads to increased aggressive behavior.[42]

Learning disabilities

While language impairments are most common, approximately 20-25% of youth with conduct disorder have some type of learning disability.[43] Although the relationship between the disorders is complex, it seems as if learning disabilities result from a combination of ADHD, a history of academic difficulty and failure, and long-standing socialization difficulties with family and peers.[44] However, confounding variables, such as language deficits, SES disadvantage, or neurodevelopmental delay also need to be considered in this relationship, as they could help explain some of the association between conduct disorder and learning problems.

Treatment

The most effective treatment for an individual with conduct disorder is one that seeks to integrate individual, school, and family settings. Additionally, treatment should also seek to address familial conflict such as marital discord or maternal depression. In this manner, a treatment would serve to address many of the possible triggers of conduct problems. Several treatments currently exist, the most effective of which is multi-systemic treatment (MST).

Multisystemic treatment (MST)

MST is an intensive, integrative treatment that emphasizes how an individual's conduct problems fit within a broader context. The individual is viewed functioning within a series of interconnected systems (home, school, neighborhood etc.), that reinforces their antisocial behavior. MST seeks to break this connection through empowering the individual and family members.

The success rate of MST among severely antisocial youths has been found to be superior to other office-based therapy approaches. Adolescents that have undergone this treatment show decreased levels of aggression and improved familial relations. MST has also been found to decrease long-term rates of crime.

Treatment considerations

MST has not yet been shown to differentiate between rates of improvement for those presenting a child-onset path and those with an adolescent-onset path. Perceived gains from this treatment may stem from the fact that adolescent onset of the disorder is typically associated with troubled teens befriending other troubled teens. MST may serve to deter these bonds and thus improve their prognosis. The child-onset type has proved to be more impairing, and resilient, and thus may not respond as well.[45]

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External links

- Conduct Disorder Symptoms and Treatment (http://www.abct.org/sccap/?m=sPublic&fa=pub_Conduct#ConductTable) Society of Clinical Child and Adolescent Psychology
- Bullying tendency wired in brain (http://news.bbc.co.uk/2/hi/health/7714072.stm) from the BBC News.
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- Support for Parents (http://www.conductdisorders.com/)
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Depression (mood)

Depression is a state of low mood and aversion to activity that can affect a person's thoughts, behavior, feelings and sense of well-being. Depressed people can feel sad, anxious, empty, hopeless, worried, helpless, worthless, guilty, irritable, hurt, or restless. They may lose interest in activities that once were pleasurable, experience loss of appetite or overeating, have problems concentrating, remembering details, or making decisions, and may contemplate, attempt, or commit suicide. Insomnia, excessive sleeping, fatigue, loss of energy, or aches, pains, or digestive problems may also be present.

Depressed mood is not always a psychiatric disorder. It may also be a normal reaction to certain life events, a symptom of some medical conditions, or a side effect of some drugs or medical treatments. Depressed mood is also a primary or associated feature of certain psychiatric syndromes such as clinical depression.

Melencolia I (ca. 1514), by Albrecht Dürer

Causes

Lifestyle

Lifestyle factors that may play a role in depressed moods include irregular sleep, poor diet, and lack of exercise.

Life events

Life events and changes that may precipitate depressed mood include childbirth, menopause, financial difficulties, job problems, a medical diagnosis (cancer, HIV, etc.), bullying, loss of a loved one, natural disasters, social isolation, relationship troubles, separation, and catastrophic injury.

Poor diet may contribute to a depressed mood

Medical treatments

Certain medications are known to cause depressed mood in a significant number of patients. These include hepatitis C drug therapy and some drugs used to treat high blood pressure, such as beta-blockers or reserpine.

Serious injuries may predispose a person to have a depressed mood

Non-psychiatric illnesses

Depressed mood can be the result of a number of infectious diseases, neurological conditions [1] and physiological problems including hypoandrogenism (in men), Addison's disease, Lyme disease, multiple sclerosis, chronic pain, stroke, diabetes, cancer, sleep apnea, and disturbed circadian rhythm. It is often one of the early symptoms of hypothyroidism (reduced activity of the thyroid gland). For a discussion of non-psychiatric conditions that can cause depressed mood, see Depression (differential diagnoses).

Psychiatric syndromes

A number of psychiatric syndromes feature depressed mood as a main symptom. The mood disorders are a group of disorders considered to be primary disturbances of mood. These include major depressive disorder (MDD; commonly called major depression or clinical depression) where a person has at least two weeks of depressed mood or a loss of interest or pleasure in nearly all activities; and dysthymia, a state of chronic depressed mood, the symptoms of which do not meet the severity of a major depressive episode. Another mood disorder, bipolar disorder, features one or more episodes of abnormally elevated mood, cognition and energy levels, but may also involve one or more depressive episodes. When the course of depressive episodes follows a seasonal pattern, the disorder (major depressive disorder, bipolar disorder, etc.) may be described as a seasonal affective disorder.

Outside the mood disorders: borderline personality disorder commonly features depressed mood; adjustment disorder with depressed mood is a mood disturbance appearing as a psychological response to an identifiable event or stressor, in which the resulting emotional or behavioral symptoms are significant but do not meet the criteria for a major depressive episode;[:355] and posttraumatic stress disorder, an anxiety disorder that sometimes follows trauma, is commonly accompanied by depressed mood.

Assessment

Many psychological assessments related to testing for depression and/or for the severity of depressive symptoms exist, assessments such as the Beck Depression Inventory and Children's Depression Inventory test for depression and/or depressive symptoms.[2]

Treatment

Depressed mood may not require any professional treatment, and may be a normal reaction to certain life events, a symptom of some medical conditions, or a side effect of some drugs or medical treatments. A prolonged depressed mood, especially in combination with other symptoms, may lead to a diagnosis of a psychiatric or medical condition by a counselor or doctor, which may benefit from treatment.[3] Different sub-divisions of depression have different treatment approaches.[4]

Meditation may help people with a depressed mood.

Over-diagnosis of depression may be common, because while the prevalence of major depressive disorder has remained unchanged,[*citation needed*] its diagnosis has doubled in 20 years. Depression is more likely over-diagnosed than under-diagnosed in primary care, resulting in patients without evidence of major depressive disorder being prescribed medications. The elderly may be at more risk of over-diagnosis.

Given an accurate diagnosis of major depressive disorder, in general the type of treatment (psychotherapy and/or antidepressants, alternative therapies, or active intervention) is "less important than getting depressed patients involved in an active therapeutic program."

Lifestyle strategies that may improve depressed mood include wake therapy, light therapy, eating a healthy diet, meditation, exercise, and smoking cessation.

Social

Gender differences in depression (for which women are much more likely) could be explained by gender roles and norms attached to gender roles. Mental problems of women are closely correlated with todays social roles of women, they are expected to care and look after family and friends, but without strong, stable relationships that foster support they are more susceptible to depressive symptoms. And because relationships are often dependent on social norms this can effect a women's mental health.[5] Situations in life including divorce, widowhood and aspects of child rearing all have effects on women's mental health as there is a certain norm attached to roles a woman plays.[6] Women who are married tend to be more psychological stable than their unmarried counterparts. Life experiences can have serious effects on mental health.

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Diagnostic and Statistical Manual of Mental Disorders

The ***Diagnostic and Statistical Manual of Mental Disorders*** (**DSM**), published by the American Psychiatric Association, offers a common language and standard criteria for the classification of mental disorders. It is used, or relied upon, by clinicians, researchers, psychiatric drug regulation agencies, health insurance companies, pharmaceutical companies, the legal system, and policy makers together with alternatives such as the International Statistical Classification of Diseases and Related Health Problems (ICD), produced by the World Health Organization (WHO). As of February 2014[1], the current version of the DSM is the fifth edition, DSM-5, published on May 18, 2013.

The DSM evolved from systems for collecting census and psychiatric hospital statistics, and from a United States Army manual. Revisions since its first publication in 1952 have incrementally added to the total number of mental disorders, although also removing those no longer considered to be mental disorders.

The International Statistical Classification of Diseases and Related Health Problems (ICD), produced by the World Health Organization (WHO), is the other commonly used manual for mental disorders. It is distinguished from the DSM in that it covers health as a whole. It is in fact the official diagnostic system for mental disorders in the US, but is used more widely in Europe and other parts of the world. The coding system used in the DSM is designed to correspond with the codes used in the ICD, although not all codes may match at all times because the two publications are not revised synchronously.

While the DSM has been praised for standardizing psychiatric diagnostic categories and criteria, it has also generated controversy and criticism. Critics, including the National Institute of Mental Health, argue that the DSM represents an unscientific and subjective system. There are ongoing issues concerning the validity and reliability of the diagnostic categories; the reliance on superficial symptoms; the use of artificial dividing lines between categories and from 'normality'; possible cultural bias; medicalization of human distress.[2] The publication of the DSM, with tightly guarded copyrights, now makes APA over $5 million a year, historically totaling over $100 million.

Uses and definition

Many mental health professionals use the manual to determine and help communicate a patient's diagnosis after an evaluation; hospitals, clinics, and insurance companies in the US also generally require a DSM diagnosis for all patients treated. The DSM can be used clinically in this way, and also to categorize patients using diagnostic criteria for research purposes. Studies done on specific disorders often recruit patients whose symptoms match the criteria listed in the DSM for that disorder. An international survey of psychiatrists in 66 countries comparing use of the ICD-10 and DSM-IV found the former was more often used for clinical diagnosis while the latter was more valued

for research.

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The current version of the DSM characterizes a mental disorder as "a clinically significant behavioral or psychological syndrome or pattern that occurs in an individual [which] is associated with present distress...or disability...or with a significant increased risk of suffering." It also notes that "...no definition adequately specifies precise boundaries for the concept of 'mental disorder'...different situations call for different definitions". It states that "there is no assumption that each category of mental disorder is a completely discrete entity with absolute boundaries dividing it from other mental disorders or from no mental disorder" (APA, 1994 and 2000). There are attempts to adjust the wording for the upcoming DSM-V.Wikipedia:Manual of Style/Dates and numbers#Precise language[3]

History

The initial impetus for developing a classification of mental disorders in the United States was the need to collect statistical information. The first official attempt was the 1840 census, which used a single category, "idiocy/insanity". Three years later, the American Statistical Association made an official protest to the U.S. House of Representatives stating that "the most glaring and remarkable errors are found in the statements respecting nosology, prevalence of insanity, blindness, deafness, and dumbness, among the people of this nation" and pointing out that in many towns African-Americans were all marked as insane, and the statistics were essentially useless.

The Association of Medical Superintendents of American Institutions for the Insane was formed in 1844, changing its name in 1892 to the American Medico-Psychological Association, and in 1921 to the present American Psychiatric Association (APA).

Edward Jarvis and later Francis Amasa Walker helped expand the census, from 2 volumes in 1870 to 25 volumes in 1880. Frederick H. Wines was appointed to write a 582-page volume called "Report on the Defective, Dependent, and Delinquent Classes of the Population of the United States, As Returned at the Tenth Census (June 1, 1880)" (published 1888). Wines used seven categories of mental illness: dementia, dipsomania (uncontrollable craving for alcohol), epilepsy, mania, melancholia, monomania and paresis. These categories were also adopted by the Association.[4]

In 1917, together with the National Commission on Mental Hygiene (now Mental Health America), the APA developed a new guide for mental hospitals called the "Statistical Manual for the Use of Institutions for the Insane". This included 22 diagnoses and would be revised several times by the APA over the years.[5] Along with the New York Academy of Medicine, the APA also provided the psychiatric nomenclature subsection of the US general medical guide, the *Standard Classified Nomenclature of Disease*, referred to as the "Standard".

DSM-I (1952)

World War II saw the large-scale involvement of US psychiatrists in the selection, processing, assessment, and treatment of soldiers. This moved the focus away from mental institutions and traditional clinical perspectives. A committee headed by psychiatrist Brigadier General William C. Menninger developed a new classification scheme called Medical 203, that was issued in 1943 as a War Department Technical Bulletin under the auspices of the Office of the Surgeon General. The foreword to the DSM-I states the US Navy had itself made some minor revisions but "the Army established a much more sweeping revision, abandoning the basic outline of the Standard and attempting to express present day concepts of mental disturbance. This nomenclature eventually was adopted by all Armed Forces", and "assorted modifications of the Armed Forces nomenclature [were] introduced into many clinics and hospitals by psychiatrists returning from military duty." The Veterans Administration also adopted a slightly modified version of Medical 203.[*citation needed*] In 1949, the World Health Organization published the sixth revision of the International Statistical Classification of Diseases (ICD), which included a section on mental disorders for the

first time. The foreword to DSM-1 states this "categorized mental disorders in rubrics similar to those of the Armed Forces nomenclature." An APA Committee on Nomenclature and Statistics was empowered to develop a version specifically for use in the United States, to standardize the diverse and confused usage of different documents. In 1950, the APA committee undertook a review and consultation. It circulated an adaptation of Medical 203, the VA system, and the Standard's Nomenclature to approximately 10% of APA members. 46% replied, of which 93% approved, and after some further revisions (resulting in its being called DSM-I), the *Diagnostic and Statistical Manual of Mental Disorders* was approved in 1951 and published in 1952. The structure and conceptual framework were the same as in Medical 203, and many passages of text were identical. The manual was 130 pages long and listed 106 mental disorders.[6] These included several categories of "personality disturbance", generally distinguished from "neurosis" (nervousness, egodystonic). In 1952, the APA listed homosexuality in the DSM as a sociopathic personality disturbance. *Homosexuality: A Psychoanalytic Study of Male Homosexuals*, a large-scale 1962 study of homosexuality, was used to justify inclusion of the disorder as a supposed pathological hidden fear of the opposite sex caused by traumatic parent–child relationships. This view was widely influential in the medical profession.[7] In 1956, however, the psychologist Evelyn Hooker performed a study that compared the happiness and well-adjusted nature of self-identified homosexual men with heterosexual men and found no difference.[8] Her study stunned the medical community and made her a hero to many gay men and lesbians,[9] but homosexuality remained in the DSM until May 1974.

DSM-II (1968)

In the 1960s, there were many challenges to the concept of mental illness itself. These challenges came from psychiatrists like Thomas Szasz, who argued that mental illness was a myth used to disguise moral conflicts; from sociologists such as Erving Goffman, who said mental illness was merely another example of how society labels and controls non-conformists; from behavioural psychologists who challenged psychiatry's fundamental reliance on unobservable phenomena; and from gay rights activists who criticised the APA's listing of homosexuality as a mental disorder. A study published in *Science* by Rosenhan received much publicity and was viewed as an attack on the efficacy of psychiatric diagnosis.

Although the APA was closely involved in the next significant revision of the mental disorder section of the ICD (version 8 in 1968), it decided to go ahead with a revision of the DSM. It was published in 1968, listed 182 disorders, and was 134 pages long. It was quite similar to the DSM-I. The term "reaction" was dropped, but the term "neurosis" was retained. Both the DSM-I and the DSM-II reflected the predominant psychodynamic psychiatry, although they also included biological perspectives and concepts from Kraepelin's system of classification. Symptoms were not specified in detail for specific disorders. Many were seen as reflections of broad underlying conflicts or maladaptive reactions to life problems, rooted in a distinction between neurosis and psychosis (roughly, anxiety/depression broadly in touch with reality, or hallucinations/delusions appearing disconnected from reality). Sociological and biological knowledge was incorporated, in a model that did not emphasize a clear boundary between normality and abnormality. The idea that personality disorders did not involve emotional distress was discarded.

An influential 1974 paper by Robert Spitzer and Joseph L. Fleiss demonstrated that the second edition of the DSM (DSM-II) was an unreliable diagnostic tool. They found that different practitioners using the DSM-II were rarely in agreement when diagnosing patients with similar problems. In reviewing previous studies of 18 major diagnostic categories, Fleiss and Spitzer concluded that "there are no diagnostic categories for which reliability is uniformly high. Reliability appears to be only satisfactory for three categories: mental deficiency, organic brain syndrome (but not its subtypes), and alcoholism. The level of reliability is no better than fair for psychosis and schizophrenia and is poor for the remaining categories".

Seventh printing of the DSM-II, 1974

As described by Ronald Bayer, a psychiatrist and gay rights activist, specific protests by gay rights activists against the APA began in 1970, when the organization held its convention in San Francisco. The activists disrupted the conference by interrupting speakers and shouting down and ridiculing psychiatrists who viewed homosexuality as a mental disorder. In 1971, gay rights activist Frank Kameny worked with the Gay Liberation Front collective to demonstrate against the APA's convention. At the 1971 conference, Kameny grabbed the microphone and yelled, "Psychiatry is the enemy incarnate. Psychiatry has waged a relentless war of extermination against us. You may take this as a declaration of war against you."[10]

This activism occurred in the context of a broader anti-psychiatry movement that had come to the fore in the 1960s and was challenging the legitimacy of psychiatric diagnosis. Anti-psychiatry activists protested at the same APA conventions, with some shared slogans and intellectual foundations.[11][12]

Presented with data from researchers such as Alfred Kinsey and Evelyn Hooker, the seventh printing of the DSM-II, in 1974, no longer listed homosexuality as a category of disorder. After a vote by the APA trustees in 1973, and confirmed by the wider APA membership in 1974, the diagnosis was replaced with the category of "sexual orientation disturbance".

DSM-III (1980)

In 1974, the decision to create a new revision of the DSM was made, and Robert Spitzer was selected as chairman of the task force. The initial impetus was to make the DSM nomenclature consistent with the International Statistical Classification of Diseases and Related Health Problems (ICD), published by the World Health Organization. The revision took on a far wider mandate under the influence and control of Spitzer and his chosen committee members.[13] One goal was to improve the uniformity and validity of psychiatric diagnosis in the wake of a number of critiques, including the famous Rosenhan experiment. There was also a need to standardize diagnostic practices within the US and with other countries after research showed that psychiatric diagnoses differed markedly between Europe and the USA. The establishment of these criteria was an attempt to facilitate the pharmaceutical regulatory process.

The criteria adopted for many of the mental disorders were taken from the Research Diagnostic Criteria (RDC) and Feighner Criteria, which had just been developed by a group of research-orientated psychiatrists based primarily at Washington University in St. Louis and the New York State Psychiatric Institute. Other criteria, and potential new categories of disorder, were established by consensus during meetings of the committee, as chaired by Spitzer. A key aim was to base categorization on colloquial English descriptive language (which would be easier to use by federal administrative offices), rather than assumptions of etiology, although its categorical approach assumed each particular pattern of symptoms in a category reflected a particular underlying pathology (an approach described as "neo-Kraepelinian"). The psychodynamic or physiologic view was abandoned, in favor of a regulatory or legislative model. A new "multiaxial" system attempted to yield a picture more amenable to a statistical population census, rather than just a simple diagnosis. Spitzer argued that "mental disorders are a subset of medical disorders" but the task force decided on the DSM statement: "Each of the mental disorders is conceptualized as a clinically significant behavioral or psychological syndrome." The personality disorders were placed on axis II along with mental retardation.

The first draft of the DSM-III was prepared within a year. Many new categories of disorder were introduced, while some were deleted or changed. A number of the unpublished documents discussing and justifying the changes have recently come to light. Field trials sponsored by the U.S. National Institute of Mental Health (NIMH) were conducted between 1977 and 1979 to test the reliability of the new diagnoses. A controversy emerged regarding deletion of the concept of neurosis, a mainstream of psychoanalytic theory and therapy but seen as vague and unscientific by the DSM task force. Faced with enormous political opposition, the DSM-III was in serious danger of not being approved by the APA Board of Trustees unless "neurosis" was included in some capacity; a political

compromise reinserted the term in parentheses after the word “disorder” in some cases. Additionally, the diagnosis of ego-dystonic homosexuality replaced the DSM-II category of “sexual orientation disturbance”.

Finally published in 1980, the DSM-III was 494 pages and listed 265 diagnostic categories. It rapidly came into widespread international use and has been termed a revolution or transformation in psychiatry. However, Robert Spitzer later criticized his own work on it in an interview with Adam Curtis, saying it led to the medicalization of 20-30 percent of the population who may not have had any serious mental problems.

When DSM-III was published, the developers made extensive claims about the reliability of the radically new diagnostic system they had devised, which relied on data from special field trials. However, according to a 1994 article by Stuart A. Kirk:

> Twenty years after the reliability problem became the central focus of DSM-III, there is still not a single multi-site study showing that DSM (any version) is routinely used with high reliably by regular mental health clinicians. Nor is there any credible evidence that any version of the manual has greatly increased its reliability beyond the previous version. There are important methodological problems that limit the generalisability of most reliability studies. Each reliability study is constrained by the training and supervision of the interviewers, their motivation and commitment to diagnostic accuracy, their prior skill, the homogeneity of the clinical setting in regard to patient mix and base rates, and the methodological rigor achieved by the investigator...

DSM-III-R (1987)

In 1987, the DSM-III-R was published as a revision of the DSM-III, under the direction of Spitzer. Categories were renamed and reorganized, and significant changes in criteria were made. Six categories were deleted while others were added. Controversial diagnoses, such as pre-menstrual dysphoric disorder and masochistic personality disorder, were considered and discarded. “Sexual orientation disturbance” was also removed and was largely subsumed under “sexual disorder not otherwise specified”, which can include “persistent and marked distress about one’s sexual orientation.”[14] Altogether, the DSM-III-R contained 292 diagnoses and was 567 pages long. Further efforts were made for the diagnoses to be purely descriptive, although the introductory text stated that for at least some disorders, “particularly the Personality Disorders, the criteria require much more inference on the part of the observer” (p. xxiii).

DSM-IV (1994)

In 1994, DSM-IV was published, listing 297 disorders in 886 pages. The task force was chaired by Allen Frances. A steering committee of 27 people was introduced, including four psychologists. The steering committee created 13 work groups of 5–16 members. Each work group had approximately 20 advisers.Wikipedia:Please clarify The work groups conducted a three-step process: first, each group conducted an extensive literature review of their diagnoses; then, they requested data from researchers, conducting analyses to determine which criteria required change, with instructions to be conservative; finally, they conducted multicenter field trials relating diagnoses to clinical practice.[15][16] A major change from previous versions was the inclusion of a clinical significance criterion to almost half of all the categories, which required that symptoms cause “clinically significant distress or impairment in social, occupational, or other important areas of functioning”. Some personality disorder diagnoses were deleted or moved to the appendix.

DSM-IV-TR (2000)

A "text revision" of the DSM-IV, known as the DSM-IV-TR, was published in 2000. The diagnostic categories and the vast majority of the specific criteria for diagnosis were unchanged.[17] The text sections giving extra information on each diagnosis were updated, as were some of the diagnostic codes to maintain consistency with the ICD. The DSM-IV-TR was organized into a five-part axial system. The first axis incorporated clinical disorders. The second axis covered personality disorders and intellectual disabilities. The remaining axes covered medical, psychosocial, environmental, and childhood factors functionally necessary to provide diagnostic criteria for health care assessments.

DSM-5 (2013)

The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM), the DSM-V, was approved by the Board of Trustees of the American Psychiatric Association (APA) on December 1, 2012. Published on May 18, 2013, the DSM-5 contains extensively revised diagnoses and, in some cases, broadens diagnostic definitions while narrowing definitions in other cases. The DSM-5 is the first major edition of the manual in twenty years, and the Roman numerals numbering system has been discontinued to allow for greater clarity in regard to revision numbers. A significant change in the fifth edition is the proposed deletion of the subtypes of schizophrenia. During the revision process, the APA website periodically listed several sections of the DSM-5 for review and discussion.

DSM-IV-TR

Categorization

The DSM-IV is a categorical classification system. The categories are prototypes, and a patient with a close approximation to the prototype is said to have that disorder. DSM-IV states, "there is no assumption each category of mental disorder is a completely discrete entity with absolute boundaries" but isolated, low-grade and noncriterion (unlisted for a given disorder) symptoms are not given importance.[18] Qualifiers are sometimes used, for example mild, moderate or severe forms of a disorder. For nearly half the disorders, symptoms must be sufficient to cause "clinically significant distress or impairment in social, occupational, or other important areas of functioning," although DSM-IV-TR removed the distress criterion from tic disorders and several of the paraphilias due to their egosyntonic nature. Each category of disorder has a numeric code taken from the ICD coding system, used for health service (including insurance) administrative purposes.

Multi-axial system

The DSM-IV organizes each psychiatric diagnosis into five dimensions (axes) relating to different aspects of disorder or disability:

- ***Axis I****:* All psychological diagnostic categories except mental retardation and personality disorder
- ***Axis II****:* Personality disorders and mental retardation
- ***Axis III****:* General medical condition; acute medical conditions and physical disorders
- ***Axis IV****:* Psychosocial and environmental factors contributing to the disorder
- ***Axis V****:* Global Assessment of Functioning or Children's Global Assessment Scale for children and teens under the age of 18

Common Axis I disorders include depression, anxiety disorders, bipolar disorder, ADHD, autism spectrum disorders, anorexia nervosa, bulimia nervosa, and schizophrenia.

Common Axis II disorders include personality disorders: paranoid personality disorder, schizoid personality disorder, schizotypal personality disorder, borderline personality disorder, antisocial personality disorder, narcissistic personality disorder, histrionic personality disorder, avoidant personality disorder, dependent personality disorder,

obsessive-compulsive personality disorder; and intellectual disabilities.

Common Axis III disorders include brain injuries and other medical/physical disorders which may aggravate existing diseases or present symptoms similar to other disorders.

Cautions

The **DSM-IV-TR** states, because it is produced for the completion of federal legislative mandates, its use by people without clinical training can lead to inappropriate application of its contents. Appropriate use of the diagnostic criteria is said to require extensive clinical training, and its contents "cannot simply be applied in a cookbook fashion". The APA notes diagnostic labels are primarily for use as a "convenient shorthand" among professionals. The DSM advises laypersons should consult the DSM only to obtain information, not to make diagnoses, and people who may have a mental disorder should be referred to psychological counseling or treatment. Further, a shared diagnosis or label may have different causes or require different treatments; for this reason the DSM contains no information regarding **treatment** or **cause**. The range of the DSM represents an extensive scope of psychiatric and psychological issues or conditions, and it is not exclusive to what may be considered "illnesses".

Sourcebooks

The DSM-IV does not specifically cite its sources, but there are four volumes of "sourcebooks" intended to be APA's documentation of the guideline development process and supporting evidence, including literature reviews, data analyses and field trials. The Sourcebooks have been said to provide important insights into the character and quality of the decisions that led to the production of DSM-IV, and hence the scientific credibility of contemporary psychiatric classification.[19][20]

Criticism

Reliability and validity concerns

The revisions of the DSM from the 3rd Edition forward have been mainly concerned with diagnostic reliability—the degree to which different diagnosticians agree on a diagnosis. It was argued that a science of psychiatry can only advance if diagnosis is reliable. If clinicians and researchers frequently disagree about a diagnosis with a patient, then research into the causes and effective treatments of those disorders cannot advance. Hence, diagnostic reliability was a major concern of DSM-III. When the diagnostic reliability problem was thought to be solved, subsequent editions of the DSM were concerned mainly with "tweaking" the diagnostic criteria. Unfortunately, neither the issue of reliability (accurate measurement) or validity (do these disorders really exist) was settled. However, most psychiatric education post DSM-III focused on issues of treatment—especially drug treatment—and less on diagnostic concerns. In fact, Thomas R. Insel, M.D., Director of the NIMH, has recently stated the agency would no longer fund research projects that rely exclusively on DSM criteria due to its lack of validity.

Superficial symptoms

By design, the DSM is primarily concerned with the signs and symptoms of mental disorders, rather than the underlying causes. It claims to collect them together based on statistical or clinical patterns. As such, it has been compared to a naturalist's field guide to birds, with similar advantages and disadvantages.[21] The lack of a causative or explanatory basis, however, is not specific to the DSM, but rather reflects a general lack of pathophysiological understanding of psychiatric disorders. As DSM-III chief architect Robert Spitzer and DSM-IV editor Michael First outlined in 2005, "little progress has been made toward understanding the pathophysiological processes and etiology of mental disorders. If anything, the research has shown the situation is even more complex than initially imagined, and we believe not enough is known to structure the classification of psychiatric disorders according to etiology."[22]

The DSM's focus on superficial symptoms is claimed to be largely a result of necessity (assuming such a manual is nevertheless produced), since there is no agreement on a more explanatory classification system.[*citation needed*] Reviewers note, however, that this approach is undermining research, including in genetics, because it results in the grouping of individuals who have very little in common except superficial criteria as per DSM or ICD diagnosis.

Despite the lack of consensus on underlying causation, advocates for specific psychopathological paradigms have nonetheless faulted the current diagnostic scheme for not incorporating evidence-based models or findings from other areas of science. A recent example is evolutionary psychologists' criticism that the DSM does not differentiate between genuine cognitive malfunctions and those induced by psychological adaptations, a key distinction within evolutionary psychology, but one widely challenged within general psychology.[23] Another example is a strong operationalist viewpoint, which contends that reliance on operational definitions, as purported by the DSM, necessitates that intuitive concepts such as depression be replaced by specific measurable concepts before they are scientifically meaningful. One critic states of psychologists that "Instead of replacing 'metaphysical' terms such as 'desire' and 'purpose', they used it to legitimize them by giving them operational definitions...the initial, quite radical operationalist ideas eventually came to serve as little more than a 'reassurance fetish' (Koch 1992) for mainstream methodological practice."[24]

A 2013 review published in the *European archives of psychiatry and clinical neuroscience* states "that psychiatry targets the phenomena of consciousness, which, unlike somatic symptoms and signs, cannot be grasped on the analogy with material thing-like objects." As an example of the problem of the superficial characterization of psychiatric signs and symptoms, the authors gave the example of a patient saying they "feel depressed, sad, or down," showing that such a statement could indicate various underlying experiences: "not only depressed mood but also, for instance, irritation, anger, loss of meaning, varieties of fatigue, ambivalence, ruminations of different kinds, hyper-reflectivity, thought pressure, psychic anxiety, varieties of depersonalization, and even voices with negative content, and so forth." The structured interview comes with "danger of over confidence in the face value of the answers, as if a simple 'yes' or 'no' truly confirmed or denied the diagnostic criterion at issue." The authors gave an example: A patient who was being administered the Structured Clinical Interview for the DSM-IV Axis I Disorders denied thought insertion, but during a "conversational, phenomenological interview," a semi-structured interview tailored to the patient, the same patient admitted to experiencing thought insertion, along with a delusional elaboration. The authors suggested 2 reasons for this discrepancy: That the patient didn't "recognize his own experience in the rather blunt, implicitly either/or formulation of the structured-interview question," or the experience didn't "fully articulate itself" until the patient started talking about his experiences.

Dividing lines

Despite caveats in the introduction to the DSM, it has long been argued that its system of classification makes unjustified categorical distinctions between disorders, and uses arbitrary cut-offs between normal and abnormal. A 2009 psychiatric review noted that attempts to demonstrate natural boundaries between related DSM syndromes, or between a common DSM syndrome and normality, have failed. Some argue that rather than a categorical approach, a fully dimensional, spectrum or complaint-oriented approach would better reflect the evidence.[25][26]

In addition, it is argued that the current approach based on exceeding a threshold of symptoms does not adequately take into account the context in which a person is living, and to what extent there is internal disorder of an individual versus a psychological response to adverse situations.[27] The DSM does include a step ("Axis IV") for outlining "Psychosocial and environmental factors contributing to the disorder" once someone is diagnosed with that particular disorder.

Because an individual's degree of impairment is often not correlated with symptom counts, and can stem from various individual and social factors, the DSM's standard of distress or disability can often produce false positives. On the other hand, individuals who do not meet symptom counts may nevertheless experience comparable distress or disability in their life.

Cultural bias

Some psychiatrists also argue that current diagnostic standards rely on an exaggerated interpretation of neurophysiological findings and so understate the scientific importance of social-psychological variables. Advocating a more culturally sensitive approach to psychology, critics such as Carl Bell and Marcello Maviglia contend that the cultural and ethnic diversity of individuals is often discounted by researchers and service providers.[28] In addition, current diagnostic guidelines have been criticized as having a fundamentally Euro-American outlook. Although these guidelines have been widely implemented, opponents argue that even when a diagnostic criteria set is accepted across different cultures, it does not necessarily indicate that the underlying constructs have any validity within those cultures; even reliable application can only demonstrate consistency, not legitimacy. Cross-cultural psychiatrist Arthur Kleinman contends that the Western bias is ironically illustrated in the introduction of cultural factors to the DSM-IV: the fact that disorders or concepts from non-Western or non-mainstream cultures are described as "culture-bound", whereas standard psychiatric diagnoses are given no cultural qualification whatsoever, is to Kleinman revelatory of an underlying assumption that Western cultural phenomena are universal. Kleinman's negative view toward the culture-bound syndrome is largely shared by other cross-cultural critics, common responses included both disappointment over the large number of documented non-Western mental disorders still left out, and frustration that even those included were often misinterpreted or misrepresented.[29] Many mainstream psychiatrists have also been dissatisfied with these new culture-bound diagnoses, although not for the same reasons. Robert Spitzer, a lead architect of the DSM-III, has held the opinion that the addition of cultural formulations was an attempt to placate cultural critics, and that they lack any scientific motivation or support. Spitzer also posits that the new culture-bound diagnoses are rarely used in practice, maintaining that the standard diagnoses apply regardless of the culture involved. In general, the mainstream psychiatric opinion remains that if a diagnostic category is valid, cross-cultural factors are either irrelevant or are only significant to specific symptom presentations. One of the results was the development of the Azibo Nosology by Daudi Ajani Ya Azibo as an alternative to the DSM to treat African and African American patients.[30]

Medicalization and financial conflicts of interest

It has also been alleged that the way the categories of the DSM are structured, as well as the substantial expansion of the number of categories, are representative of an increasing medicalization of human nature, which may be attributed to disease mongering by psychiatrists and pharmaceutical companies, the power and influence of the latter having grown dramatically in recent decades.[31] Of the authors who selected and defined the DSM-IV psychiatric disorders, roughly half have had financial relationships with the pharmaceutical industry at one time, raising the prospect of a direct conflict of interest.[32] The same article concludes that the connections between panel members and the drug companies were particularly strong in those diagnoses where drugs are the first line of treatment, such as schizophrenia and mood disorders, where 100% of the panel members had financial ties with the pharmaceutical industry. In 2005, then American Psychiatric Association President Steven Sharfstein released a statement in which he conceded that psychiatrists had "allowed the biopsychosocial model to become the bio-bio-bio model".[33]

However, although the number of identified diagnoses has increased by more than 200% (from 106 in DSM-I to 365 in DSM-IV-TR), psychiatrists such as Zimmerman and Spitzer argue it almost entirely represents greater specification of the forms of pathology, thereby allowing better grouping of more similar patients. William Glasser, however, refers to the DSM as "phony diagnostic categories", arguing that "it was developed to help psychiatrists – to help them make money". In addition, the publishing of the DSM, with tightly guarded copyrights, has in itself earned over $100 million for the American Psychiatric Association.

Consumers and survivors

A "consumer" is a person who accesses psychiatric services and may have been given a diagnosis from the *Diagnostic and Statistical Manual of Mental Disorders*, while a "survivor" self-identifies as a person who has endured a psychiatric intervention and the mental health system (which may have involved involuntary commitment and involuntary treatment).[*citation needed*] Some individuals are relieved to find that they have a recognized condition that they can apply a name to and this has led to many people self-diagnosing.[*citation needed*] Others, however, question the accuracy of the diagnosis, or feel they have been given a "label" that invites social stigma and discrimination (the terms "mentalism" and "sanism" have been used to describe such discriminatory treatment).[34]

Diagnoses can become internalized and affect an individual's self-identity, and some psychotherapists have found that the healing process can be inhibited and symptoms can worsen as a result.[35] Some members of the psychiatric survivors movement (more broadly the consumer/survivor/ex-patient movement) actively campaign against their diagnoses, or the assumed implications, and/or against the DSM system in general. The Mad Pride movement has been particularly vocal in its criticism of the DSM.[*citation needed*] Additionally, it has been noted that the DSM often uses definitions and terminology that are inconsistent with a recovery model, and such content can erroneously imply excess psychopathology (e.g. multiple "comorbid" diagnoses) or chronicity.[36]

DSM-5 critiques

Psychiatrist Allen Frances has been critical of proposed revisions to the DSM-5. In a 2012 *New York Times* editorial, Frances warned that if this DSM version is issued unamended by the APA, it will "medicalize normality and result in a glut of unnecessary and harmful drug prescription."[37] In a December 2, 2012 blog post in *Psychology Today*, Frances lists the ten "most potentially harmful changes" to DSM-5:

- Disruptive Mood Dysregulation Disorder, for temper tantrums
- Major Depressive Disorder, includes normal grief
- Minor Neurocognitive Disorder, for normal forgetting in old age
- Adult Attention Deficit Disorder, encouraging psychiatric prescriptions of stimulants
- Binge Eating Disorder, for excessive eating
- Autism, defining the disorder more specifically, possibly leading to decreased rates of diagnosis and the disruption of school services
- First time drug users will be lumped in with addicts
- Behavioral Addictions, making a "mental disorder of everything we like to do a lot."
- Generalized Anxiety Disorder, includes everyday worries
- Post-traumatic stress disorder, changes opening "the gate even further to the already existing problem of misdiagnosis of PTSD in forensic settings."

Frances and others have published debates on what they see as the six most essential questions in psychiatric diagnosis:

- are they more like theoretical constructs or more like diseases
- how to reach an agreed definition
- whether the DSM-5 should take a cautious or conservative approach
- the role of practical rather than scientific considerations
- the issue of use by clinicians or researchers
- whether an entirely different diagnostic system is required.

In 2011, psychologist Brent Robbins co-authored a national letter for the Society for Humanistic Psychology that has brought thousands into the public debate about the DSM. Approximately 14,000 individuals and mental health professionals have signed a petition in support of the letter. Thirteen other American Psychological Association divisions have endorsed the petition. Robbins has noted that under the new guidelines, certain responses to grief could be labeled as pathological disorders, instead of being recognized as being normal human experiences.

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External links

- Official DSM-5 development website (http://www.dsm5.org/pages/default.aspx)
- Topic Center from the Psychiatric Times: DSM-5 (http://www.psychiatrictimes.com/DSM-5)
- DSM-IV-TR Official Site (http://www.psychiatry.org/practice/dsm/dsm-iv-tr) - American Psychiatric Association
- Diagnostic Criteria from DSM-IV-TR (http://www.behavenet.com/capsules/disorders/dsm4TRclassification.htm)
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- DSM-IV-TR In Action (http://www.spiritualmentoring.com/page2/page46/page46.html) Powerpoint slide handouts by G. Scott Sparrow

DSM-5

Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)	
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The ***Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition**, abbreviated as **DSM-5**, is the 2013 update to the American Psychiatric Association's (APA) classification and diagnostic tool. In the United States the DSM serves as a universal authority for psychiatric diagnosis. Treatment recommendations, as well as payment by health care providers, are often determined by DSM classifications, so the appearance of a new version has significant practical importance.

The DSM-5 was published on May 18, 2013, superseding the DSM-IV-TR, which was published in 2000. The development of the new edition began with a conference in 1999, and proceeded with the formation of a Task Force in 2007, which developed and field-tested a variety of new classifications. In most respects DSM-5 is not greatly changed from DSM-IV-TR. Notable changes include dropping Asperger syndrome as a distinct classification; loss of subtype classifications for variant forms of schizophrenia; dropping the "bereavement exclusion" for depressive disorders; a revised treatment and naming of *gender identity disorder* to *gender dysphoria*, and removing the A2 criterion for posttraumatic stress disorder (PTSD) because its requirement for specific emotional reactions to trauma did not apply to combat veterans and first responders with PTSD.

The fifth edition was criticized by various authorities both before and after it was formally published. Critics assert, for example, that many DSM-5 revisions or additions lack empirical support; inter-rater reliability is low for many disorders; several sections contain poorly written, confusing, or contradictory information; and the psychiatric drug industry unduly influenced the manual's content. Various scientists have argued that the DSM-5 forces clinicians to make distinctions that are not supported by solid evidence, distinctions that have major treatment implications, including drug prescriptions and the availability of health insurance coverage. General criticism of the DSM-5 ultimately resulted in a petition signed by 13,000, and sponsored by many mental health organizations, which called for outside review of the document.

Changes

Section I

Section I describes DSM-5 chapter organization, its change from the multiaxial system, and Section III's dimensional assessments. The DSM-5 deleted the chapter that includes "disorders usually first diagnosed in infancy, childhood, or adolescence" opting to list them in other chapters. A note under Anxiety Disorders says that the "sequential order" of at least some DSM-5 chapters has significance that reflects the relationships between diagnoses.

This introductory section describes the process of DSM revision, including field trials, public and professional review, and expert review. It states its goal is to harmonize with the ICD systems and share organizational structures as much as is feasible. Concern about the categorical system of diagnosis is expressed, but the conclusion is the reality that alternative definitions for most disorders is scientifically premature.

The new version replaces the NOS categories with two options: *other specified disorder* and *unspecified disorder* to increase the utility to the clinician. The first allows the clinician to specify the reason that the criteria for a specific disorder are not met; the second allows the clinician the option to forgo specification.

DSM-5 has discarded the multiaxial system of diagnosis (formerly Axis I, Axis II, Axis III), listing all disorders in Section II. It has replaced Axis IV with significant psychosocial and contextual features and dropped Axis V (Global Assessment of Functioning, known as GAF). The World Health Organization's (WHO) Disability Assessment Schedule is added to Section III (Emerging measures and models) under Assessment Measures, as a suggested, but not required, method to assess functioning.

Section II: diagnostic criteria and codes

Neurodevelopmental disorders

- "Mental retardation" has a new name: "intellectual disability (intellectual developmental disorder)."
- Phonological disorder and stuttering are now called communication disorders—which include language disorder, speech sound disorder, childhood-onset fluency disorder, and a new condition characterized by impaired social verbal and nonverbal communication called social (pragmatic) communication disorder.
- Autism spectrum disorder incorporates Asperger disorder, childhood disintegrative disorder, and pervasive developmental disorder not otherwise specified (PDD-NOS) - see Diagnosis of Asperger syndrome#Proposed changes to DSM-5.
- A new sub-category, motor disorders, encompasses developmental coordination disorder, stereotypic movement disorder, and the tic disorders including Tourette syndrome.

Schizophrenia spectrum and other psychotic disorders

- All subtypes of schizophrenia were deleted (paranoid, disorganized, catatonic, undifferentiated, and residual).
- A major mood episode is required for schizoaffective disorder (for a majority of the disorder's duration after criterion A [related to delusions, hallucinations, disorganized speech or behavior, and negative symptoms such as avolition] is met).
- Criteria for delusional disorder changed, and it is no longer separate from shared delusional disorder.
- Catatonia in all contexts requires 3 of a total of 12 symptoms. Catatonia may be a specifier for depressive, bipolar, and psychotic disorders; part of another medical condition; or of another specified diagnosis.

Bipolar and related disorders

- New specifier "with mixed features" can be applied to bipolar I disorder, bipolar II disorder, bipolar disorder NED (not elsewhere defined, previously called "NOS", not otherwise specified) and MDD.
- Allows other specified bipolar and related disorder for particular conditions.
- Anxiety symptoms are a specifier (called "anxious distress") added to bipolar disorder and to depressive disorders (but are not part of the bipolar diagnostic criteria).

Depressive disorders

- The bereavement exclusion in DSM-IV was removed from depressive disorders in DSM-5.
- New disruptive mood dysregulation disorder (DMDD) for children up to age 18 years.
- Premenstrual dysphoric disorder moved from an appendix for further study, and became a disorder.
- Specifiers were added for mixed symptoms and for anxiety, along with guidance to physicians for suicidality.
- The term dysthymia now also would be called persistent depressive disorder.

Anxiety disorders

- For the various forms of phobias and anxiety disorders, DSM-5 removes the requirement that the subject (formerly, over 18 years old) "must recognize that their fear and anxiety are excessive or unreasonable". Also, the duration of at least 6 months now applies to everyone (not only to children).
- Panic attack became a specifier for all DSM-5 disorders.
- Panic disorder and agoraphobia became two separate disorders.
- Specific types of phobias became specifiers but are otherwise unchanged.
- The generalized specifier for social anxiety disorder (formerly, social phobia) changed in favor of a performance only (i.e., public speaking or performance) specifier.
- Separation anxiety disorder and selective mutism are now classified as anxiety disorders (rather than disorders of early onset).

Obsessive-compulsive and related disorders

- A new chapter on obsessive-compulsive and related disorders includes four new disorders: excoriation (skin-picking) disorder, hoarding disorder, substance-/medication-induced obsessive-compulsive and related disorder, and obsessive-compulsive and related disorder due to another medical condition.
- Trichotillomania (hair-pulling disorder) moved from "impulse-control disorders not elsewhere classified" in DSM-IV, to an obsessive-compulsive disorder in DSM-5.
- A specifier was expanded (and added to body dysmorphic disorder and hoarding disorder) to allow for good or fair insight, poor insight, and "absent insight/delusional" (i.e., complete conviction that obsessive-compulsive disorder beliefs are true).
- Criteria were added to body dysmorphic disorder to describe repetitive behaviors or mental acts that may arise with perceived defects or flaws in physical appearance.
- The DSM-IV specifier “with obsessive-compulsive symptoms” moved from anxiety disorders to this new category for obsessive-compulsive and related disorders.
- There are two new diagnoses: other specified obsessive-compulsive and related disorder, which can include body-focused repetitive behavior disorder (behaviors like nail biting, lip biting, and cheek chewing, other than hair pulling and skin picking) or obsessional jealousy; and unspecified obsessive-compulsive and related disorder.

Trauma- and stressor-related disorders

- Posttraumatic stress disorder (PTSD) is now included in a new section titled "Trauma- and Stressor-Related Disorders."
- The PTSD diagnostic clusters were reorganized and expanded from a total of three clusters to four based on the results of confirmatory factor analytic research conducted since the publication of DSM-IV.
- Separate criteria were added for children six years old or younger.
- For the diagnosis of acute stress disorder and PTSD, the stressor criteria (Criterion A1 in DSM-IV) was modified to some extent. The requirement for specific subjective emotional reactions (Criterion A2 in DSM-IV) was eliminated because it lacked empirical support for its utility and predictive validity. Previously certain groups, such as military personnel involved in combat, law enforcement officers and other first responders, did not meet criterion A2 in DSM-IV because their training prepared them to not react emotionally to traumatic events.
- Two new disorders that were formerly subtypes were named: reactive attachment disorder and disinhibited social engagement disorder.
- Adjustment disorders were moved to this new section and reconceptualized as stress-response syndromes. DSM-IV subtypes for depressed mood, anxious symptoms, and disturbed conduct are unchanged.

Dissociative disorders

- Depersonalization disorder is now called depersonalization/derealization disorder.
- Dissociative fugue became a specifier for dissociative amnesia.
- The criteria for dissociative identity disorder were expanded to include "possession-form phenomena and functional neurological symptoms". It is made clear that "transitions in identity may be observable by others or self-reported". Criterion B was also modified for people who experience gaps in recall of everyday events (not only trauma).

Somatic symptom and related disorders

- Somatoform disorders are now called somatic symptom and related disorders.
- Diagnoses of somatization disorder, hypochondriasis, pain disorder, and undifferentiated somatoform disorder were deleted.
- People with chronic pain can now be diagnosed with *somatic symptom disorder with predominant pain*; or *psychological factors that affect other medical conditions*; or with an *adjustment disorder*.
- Somatization disorder and undifferentiated somatoform disorder were combined to become somatic symptom disorder, a diagnosis which no longer requires a specific number of somatic symptoms.
- Somatic symptom and related disorders are defined by positive symptoms, and the use of medically unexplained symptoms is minimized, except in the cases of conversion disorder and pseudocyesis (false pregnancy).
- A new diagnosis is psychological factors affecting other medical conditions. This was formerly found in the DSM-IV chapter "Other Conditions That May Be a Focus of Clinical Attention".
- Criteria for conversion disorder (functional neurological symptom disorder) were changed.

Feeding and eating disorders

- Criteria for pica and rumination disorder were changed and can now refer to people of any age.
- Binge eating disorder graduated from DSM-IV's "Appendix B -- Criteria Sets and Axes Provided for Further Study" into a proper diagnosis.
- Requirements for bulimia nervosa and binge eating disorder were changed from "at least twice weekly for 6 months to at least once weekly over the last 3 months".
- The criteria for anorexia nervosa were changed; there is no longer a requirement of amenorrhea.
- "Feeding disorder of infancy or early childhood", a rarely used diagnosis in DSM-IV, was renamed to avoidant/restrictive food intake disorder, and criteria were expanded.

Sleep–wake disorders

- "Sleep disorders related to another mental disorder, and sleep disorders related to a general medical condition" were deleted.
- Primary insomnia became insomnia disorder, and narcolepsy is separate from other hypersomnolence.
- There are now three breathing-related sleep disorders: obstructive sleep apnea hypopnea, central sleep apnea, and sleep-related hypoventilation.
- Circadian rhythm sleep–wake disorders were expanded to include advanced sleep phase syndrome, irregular sleep–wake type, and non-24-hour sleep–wake type. Jet lag was removed.
- Rapid eye movement sleep behavior disorder and restless legs syndrome are each a disorder, instead of both being listed under "dyssomnia not otherwise specified" in DSM-IV.

Sexual dysfunctions

- DSM-5 has sex-specific sexual dysfunctions.
- For females, sexual desire and arousal disorders are combined into female sexual interest/arousal disorder.
- Sexual dysfunctions (except substance-/medication-induced sexual dysfunction) now require a duration of approximately 6 months and more exact severity criteria.
- A new diagnosis is genito-pelvic pain/penetration disorder which combines vaginismus and dyspareunia from DSM-IV.
- Sexual aversion disorder was deleted.
- Subtypes for all disorders include only "lifelong versus acquired" and "generalized versus situational" (one subtype was deleted from DSM-IV).
- Two subtypes were deleted: "sexual dysfunction due to a general medical condition" and "due to psychological versus combined factors".

Gender dysphoria

- DSM-IV gender identity disorder is similar to, but not the same as, gender dysphoria in DSM-5. Separate criteria for children, adolescents and adults that are appropriate for varying developmental states are added.
- Subtypes of gender identity disorder based on sexual orientation were deleted.
- Among other wording changes, criterion A and criterion B (cross-gender identification, and aversion toward one's gender) were combined. Along with these changes comes the creation of a separate gender dysphoria in children as well as one for adults and adolescents. The grouping has been moved out of the sexual disorders category and into its own. The name change was made in part due to stigmatization of the term "disorder" and the relatively common use of "gender dysphoria" in the GID literature and among specialists in the area. The creation of a specific diagnosis for children reflects the lesser ability of children to have insight into what they are experiencing and ability to express it in the event that they have insight.

Disruptive, impulse-control, and conduct disorders

Some of these disorders were formerly part of the chapter on early diagnosis, oppositional defiant disorder; conduct disorder; and disruptive behavior disorder not otherwise specified became other specified and unspecified disruptive disorder, impulse-control disorder, and conduct disorders. Intermittent explosive disorder, pyromania, and kleptomania moved to this chapter from the DSM-IV chapter "Impulse-Control Disorders Not Otherwise Specified".

- Antisocial personality disorder is listed here *and* in the chapter on personality disorders (but ADHD is listed under neurodevelopmental disorders).
- Symptoms for oppositional defiant disorder are of three types: angry/irritable mood, argumentative/defiant behavior, and vindictiveness. The conduct disorder exclusion is deleted. The criteria were also changed with a note on frequency requirements and a measure of severity.

- Criteria for conduct disorder are unchanged for the most part from DSM-IV. A specifier was added for people with limited "prosocial emotion", showing callous and unemotional traits.
- People over the disorder's minimum age of 6 may be diagnosed with intermittent explosive disorder without outbursts of physical aggression. Criteria were added for frequency and to specify "impulsive and/or anger based in nature, and must cause marked distress, cause impairment in occupational or interpersonal functioning, or be associated with negative financial or legal consequences".

Substance-related and addictive disorders

- Gambling disorder and tobacco use disorder are new.
- Substance abuse and substance dependence have been combined into single substance use disorders specific to each substance of abuse within a new "addictions and related disorders" category. "Recurrent legal problems" was deleted and "craving or a strong desire or urge to use a substance" was added to the criteria. The threshold of the number of criteria that must be met was changed. Severity from mild to severe is based on the number of criteria endorsed. Criteria for cannabis and caffeine withdrawal were added. New specifiers were added for early and sustained remission along with new specifiers for "in a controlled environment" and "on maintenance therapy".

Categories of abuse and dependence have been eliminated.Now it is Substance use disorder with different levels of severity - mild moderate and severe. Gambling has been included as an addictive disorder.

Neurocognitive disorders

- Dementia and amnestic disorder became major or mild neurocognitive disorder (major NCD, or mild NCD). DSM-5 has a new list of neurocognitive domains. "New separate criteria are now presented" for major or mild NCD due to various conditions. Substance/medication-induced NCD and unspecified NCD are new diagnoses.

Paraphilic disorders

- New specifiers "in a controlled environment" and "in remission" were added to criteria for all paraphilic disorders.
- A distinction is made between paraphilic behaviors, or paraphilias, and paraphilic disorders. All criteria sets were changed to add the word disorder to all of the paraphilias, for example, pedophilia is now pedophilic disorder. There is no change in the basic diagnostic structure since DSM-III-R; however, people now must meet both qualitative (criterion A) and negative consequences (criterion B) criteria to be diagnosed with a paraphilic disorder. Otherwise they have a paraphilia (and no diagnosis).

Personality disorders

- Personality disorder previously belonged to a different axis than almost all other disorders, but is now in one axis with all mental and other medical diagnoses. However, the same ten types of personality disorder are retained.

Section III: emerging measures and models

Alternative DSM-5 model for personality disorders

An alternative hybrid dimensional-categorical model for personality disorders is included to stimulate further research on this modified classification system.

Conditions for further study

These conditions and criteria are set forth to encourage future research and are not meant for clinical use.

- Attenuated psychosis syndrome
- Depressive episodes with short-duration hypomania

- Persistent complex bereavement disorder
- Caffeine use disorder
- Internet gaming disorder
- Neurobehavioral disorder associated with prenatal alcohol exposure
- Suicidal behavior disorder
- Non-suicidal self-injury

Development

In 1999, a DSM–5 Research Planning Conference; sponsored jointly by APA and the National Institute of Mental Health (NIMH), was held to set the research priorities. Research Planning Work Groups produced "white papers" on the research needed to inform and shape the DSM-5 and the resulting work and recommendations were reported in an APA monograph and peer-reviewed literature. There were six workgroups, each focusing on a broad topic: Nomenclature, Neuroscience and Genetics, Developmental Issues and Diagnosis, Personality and Relational Disorders, Mental Disorders and Disability, and Cross-Cultural Issues. Three additional white papers were also due by 2004 concerning gender issues, diagnostic issues in the geriatric population, and mental disorders in infants and young children. The white papers have been followed by a series of conferences to produce recommendations relating to specific disorders and issues, with attendance limited to 25 invited researchers.

On July 23, 2007, the APA announced the task force that would oversee the development of DSM-5. The DSM-5 Task Force consisted of 27 members, including a chair and vice chair, who collectively represent research scientists from psychiatry and other disciplines, clinical care providers, and consumer and family advocates. Scientists working on the revision of the DSM had a broad range of experience and interests. The APA Board of Trustees required that all task force nominees disclose any competing interests or potentially conflicting relationships with entities that have an interest in psychiatric diagnoses and treatments as a precondition to appointment to the task force. The APA made all task force members' disclosures available during the announcement of the task force. Several individuals were ruled ineligible for task force appointments due to their competing interests.

The DSM-5 field trials included test-retest reliability which involved different clinicians doing independent evaluations of the same patient—a common approach to the study of diagnostic reliability.[2]

Criticism

General

Robert Spitzer, the head of the DSM-III task force, has publicly criticized the APA for mandating that DSM-5 task force members sign a nondisclosure agreement, effectively conducting the whole process in secret: "When I first heard about this agreement, I just went bonkers. Transparency is necessary if the document is to have credibility, and, in time, you're going to have people complaining all over the place that they didn't have the opportunity to challenge anything." Allen Frances, chair of the DSM-IV task force, expressed a similar concern.[3]

Although the APA has since instituted a disclosure policy for DSM-5 task force members, many still believe the Association has not gone far enough in its efforts to be transparent and to protect against industry influence. In a 2009 Point/Counterpoint article, Lisa Cosgrove, PhD and Harold J. Bursztajn, MD noted that "the fact that 70% of the task force members have reported direct industry ties---an increase of almost 14% over the percentage of DSM-IV task force members who had industry ties---shows that disclosure policies alone, especially those that rely on an honor system, are not enough and that more specific safeguards are needed."[4]

David Kupfer, chair of the DSM-5 task force, and Darrel A. Regier, MD, MPH, vice chair of the task force, whose industry ties are disclosed with those of the task force,[5] countered that "collaborative relationships among government, academia, and industry are vital to the current and future development of pharmacological treatments for mental disorders." They asserted that the development of DSM-5 is the "most inclusive and transparent

developmental process in the 60-year history of DSM." The developments to this new version can be viewed on the APA website.[6] Public input was requested for the first time in the history of the manual.[*citation needed*] During periods of public comment, members of the general public could sign up at the DSM-5 website[7] and provide feedback on the various proposed changes.[8]

In June 2009, Allen Frances issued strongly worded criticisms of the processes leading to DSM-5 and the risk of "serious, subtle, (…) ubiquitous" and "dangerous" unintended consequences such as new "false 'epidemics'". He writes that "the work on DSM-V has displayed the most unhappy combination of soaring ambition and weak methodology" and is concerned about the task force's "inexplicably closed and secretive process". His and Spitzer's concerns about the contract that the APA drew up for consultants to sign, agreeing not to discuss drafts of the fifth edition beyond the task force and committees, have also been aired and debated.

The appointment, in May 2008, of two of the taskforce members, Kenneth Zucker and Ray Blanchard, led to an internet petition to remove them. According to MSNBC, "The petition accuses Zucker of having engaged in 'junk science' and promoting 'hurtful theories' during his career, especially advocating the idea that children who are unambiguously male or female anatomically, but seem confused about their gender identity, can be treated by encouraging gender expression in line with their anatomy." According to *The Gay City News*, "Dr. Ray Blanchard, a psychiatry professor at the University of Toronto, is deemed offensive for his theories that some types of transsexuality are paraphilias, or sexual urges. In this model, transsexuality is not an essential aspect of the individual, but a misdirected sexual impulse." Blanchard responded, "Naturally, it's very disappointing to me there seems to be so much misinformation about me on the Internet. [They didn't distort] my views, they completely reversed my views." Zucker "rejects the junk-science charge, saying there 'has to be an empirical basis to modify anything' in the DSM. As for hurting people, 'in my own career, my primary motivation in working with children, adolescents and families is to help them with the distress and suffering they are experiencing, whatever the reasons they are having these struggles. I want to help people feel better about themselves, not hurt them.'"

In 2011, psychologist Brent Robbins co-authored a national letter for the Society for Humanistic Psychology that brought thousands into the public debate about the DSM. Approximately 13,000 individuals and mental health professionals signed a petition in support of the letter. Thirteen other American Psychological Association divisions endorsed the petition. In a November 2011 article about the debate in the *San Francisco Chronicle*, Robbins notes that under the new guidelines, certain responses to grief could be labeled as pathological disorders, instead of being recognized as being normal human experiences. In 2012, a footnote was added to the draft text which explains the distinction between grief and depression.

DSM-5 has been criticized for purportedly saying nothing about the biological underpinnings of mental disorders.[9]

Borderline personality disorder controversy

In 2003, the Treatment and Research Advancements National Association for Personality Disorders (TARA-APD) campaigned to change the name and designation of borderline personality disorder in DSM-5.[10] The paper *How Advocacy is Bringing BPD into the Light*[11] reported that "the name BPD is confusing, imparts no relevant or descriptive information, and reinforces existing stigma." Instead, it proposed the name "emotional regulation disorder" or "emotional dysregulation disorder." There was also discussion about changing borderline personality disorder, an Axis II diagnosis (personality disorders and mental retardation), to an Axis I diagnosis (clinical disorders).

The TARA-APD recommendations do not appear to have had an impact on the American Psychiatric Association (publisher of the DSM). As noted above, the DSM-5 does not employ a multi-axial diagnostic scheme, therefore distinction between Axis I and II disorders no longer exist in the DSM nosology. The diagnostic criteria for, and description of, Borderline Personality Disorder remain largely unchanged from DSM-IV-TR.

More radical criticisms

Some authors believe that the problem is not simply of a few criteria to be deleted or modified. For example, a Kuhnian reformulation of the diagnostic debate suggested that apparently trivial problems of the DSM, like the extremely high rates of comorbidity, might fruitfully be analysed as Kuhnian anomalies leading the DSM system to a scientific crisis.[12] As a consequence, a radical rethinking of the concept of mental disorder was proposed, addressing its constructive nature.[13] Based on similar views, several revolutionary approaches were proposed, ranging from dimensional diagnosis to various forms of etiopathogenetic diagnosis.[14]

The financial association of DSM-5 panel members with industry continues to be a concern for financial conflict of interest. Of the DSM-5 task force members, 69% report having ties to the pharmaceutical industry, an increase from the 57% of DSM-IV task force members.

British Psychological Society response

The British Psychological Society in the United Kingdom stated in its June 2011 response that it had "more concerns than plaudits".[15] It criticized proposed diagnoses as "clearly based largely on social norms, with 'symptoms' that all rely on subjective judgements... not value-free, but rather reflect[ing] current normative social expectations", noting doubts over the reliability, validity, and value of existing criteria, that personality disorders were not normed on the general population, and that "not otherwise specified" categories covered a "huge" 30% of all personality disorders.

It also expressed a major concern that "clients and the general public are negatively affected by the continued and continuous medicalisation of their natural and normal responses to their experiences... which demand helping responses, but which do not reflect illnesses so much as normal individual variation".

The Society suggested as its primary specific recommendation, a change from using "diagnostic frameworks" to a description based on an individual's specific experienced problems, and that mental disorders are better explored as part of a spectrum shared with normality:

> *[We recommend] a revision of the way mental distress is thought about, starting with recognition of the overwhelming evidence that it is on a spectrum with 'normal' experience, and that psychosocial factors such as poverty, unemployment and trauma are the most strongly-evidenced causal factors. Rather than applying preordained diagnostic categories to clinical populations, we believe that any classification system should begin from the bottom up – starting with specific experiences, problems or 'symptoms' or 'complaints'... We would like to see the base unit of measurement as specific problems (e.g. hearing voices, feelings of anxiety etc.)? These would be more helpful too in terms of epidemiology.*
>
> *While some people find a name or a diagnostic label helpful, our contention is that this helpfulness results from a knowledge that their problems are recognised (in both senses of the word) understood, validated, explained (and explicable) and have some relief. Clients often, unfortunately, find that diagnosis offers only a spurious promise of such benefits. Since – for example – two people with a diagnosis of 'schizophrenia' or 'personality disorder' may possess no two symptoms in common, it is difficult to see what communicative benefit is served by using these diagnoses. We believe that a description of a person's real problems would suffice. Moncrieff and others have shown that diagnostic labels are less useful than a description of a person's problems for predicting treatment response, so again diagnoses seem positively unhelpful compared to the alternatives.* - British Psychological Society June 2011 response

National Institute of Mental Health

National Institute of Mental Health director Thomas R. Insel, MD, wrote in an April 29, 2013 blog post:

> *The goal of this new manual, as with all previous editions, is to provide a common language for describing psychopathology. While DSM has been described as a "Bible" for the field, it is, at best, a dictionary, creating a set of labels and defining each. The strength of each of the editions of DSM has been "reliability" – each edition has ensured that clinicians use the same terms in the same ways. The weakness is its lack of validity ... Patients with mental disorders deserve better.*

Insel also discussed an NIMH effort to develop a new classification system, Research Domain Criteria (RDoC), currently for research purposes only. Insel's post sparked a flurry of reaction, some of which might be termed sensationalistic, with headlines such as "Goodbye to the DSM-V", "Federal institute for mental health abandons

controversial 'bible' of psychiatry", "National Institute of Mental Health abandoning the DSM", and "Psychiatry divided as mental health 'bible' denounced." Other responses provided a more nuanced analysis of the NIMH Director's post.

In May 2013, Insel, on behalf of NIMH, issued a joint statement with Jeffrey A. Lieberman, MD, president of the American Psychiatric Association, that emphasized that DSM-5 "... represents the best information currently available for clinical diagnosis of mental disorders. Patients, families, and insurers can be confident that effective treatments are available and that the DSM is the key resource for delivering the best available care. The National Institute of Mental Health (NIMH) has not changed its position on DSM-5." Insel and Lieberman say that DSM-5 and RDoC "represent complementary, not competing, frameworks" for characterizing diseases and disorders.

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External links

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Empathy

Empathy is the capacity to recognize emotions that are being experienced by another sentient or fictional being. One may need to have a certain amount of empathy before being able to experience accurate sympathy or compassion. The English word was coined in 1909 by the psychologist Edward B. Titchener in an attempt to translate the German word "*Einfühlungsvermögen*", a new phenomenon explored at the end of 19th century mainly by philosopher Theodor Lipps. It was later re-translated into the German language as "*Empathie*", and is still in use there.

Etymology

The English word is derived from the Ancient Greek word ἐμπάθεια (*empatheia*), "physical affection, passion, partiality" which comes from ἐν (*en*), "in, at" + πάθος (*pathos*), "passion" or "suffering".[1] The term was adapted by Hermann Lotze and Robert Vischer to create the German word *Einfühlung* ("feeling into"), which was translated by Edward B. Titchener into the English term empathy.

Alexithymia (the word comes from the Ancient Greek words λέξις (*lexis*, "diction", "word") and θυμός (*thumos*, "soul, as the seat of emotion, feeling, and thought") modified by an alpha-privative, literally meaning "without words for emotions"), is a term to describe a state of deficiency in understanding, processing, or describing emotions in oneself.[2][3]

Note that in Modern Greek the word empathy (εμπάθεια) translates as "hatred", "loathing","malevolence" and "spitefulness" (a situation of causing passion, rather than mutual relation to one's passion); ενσυναίσθηση is the correct modern equivalent of empathy.[*citation needed*]

Definition

Empathy has many different definitions that encompass a broad range of emotional states, such as caring for other people and having a desire to help them; experiencing emotions that match another person's emotions; discerning what another person is thinking or feeling; and making less distinct the differences between the self and the other.[4]

Since empathy involves understanding the emotional states of other people, the way it is characterized is derivative of the way emotions themselves are characterized. If, for example, emotions are taken to be centrally characterized by bodily feelings, then grasping the bodily feelings of another will be central to empathy. On the other hand, if emotions are more centrally characterized by a combination of beliefs and desires, then grasping these beliefs and desires will be more essential to empathy. The ability to imagine oneself as another person is a sophisticated imaginative process. However, the basic capacity to recognize emotions is probably innate[5] and may be achieved unconsciously. Yet it can be trained and achieved with various degrees of intensity or accuracy.

Empathy necessarily has a "more or less" quality. The paradigm case of an empathic interaction, however, involves a person communicating an accurate recognition of the significance of another person's ongoing intentional actions, associated emotional states, and personal characteristics in a manner that the recognized person can tolerate. Recognitions that are both accurate and tolerable are central features of empathy.

The human capacity to recognize the bodily feelings of another is related to one's imitative capacities and seems to be grounded in an innate capacity to associate the bodily movements and facial expressions one sees in another with the proprioceptive feelings of producing those corresponding movements or expressions oneself. Humans seem to make the same immediate connection between the tone of voice and other vocal expressions and inner feeling.

Empathy is distinct from sympathy, pity, and emotional contagion.[6] Sympathy or empathic concern is the feeling of compassion or concern for another, the wish to see them better off or happier. Pity is feeling that another is in trouble and in need of help as they cannot fix their problems themselves, often described as "feeling sorry" for someone. Emotional contagion is when a person (especially an infant or a member of a mob) imitatively "catches" the

emotions that others are showing without necessarily recognizing this is happening.

Types

Affective and cognitive empathy

Empathy can be divided into two major components:

- **Affective empathy**, also called **emotional empathy**: the capacity to respond with an appropriate emotion to another's mental states. Our ability to empathize emotionally is supposed to be based on emotional contagion: being affected by another's emotional or arousal state.
- **Cognitive empathy**: the capacity to understand another's perspective or mental state.[7] The terms *cognitive empathy* and *theory of mind* are often used synonymously, but due to a lack of studies comparing theory of mind with types of empathy, it is unclear whether these are equivalent.

Although science has not yet agreed upon a precise definition of these constructs, there is consensus about this distinction.

Affective empathy can be subdivided into the following scales:

- **Empathic concern**: sympathy and compassion for others in response to their suffering.
- **Personal distress**: self-centered feelings of discomfort and anxiety in response to another's suffering. There is no consensus regarding whether personal distress is a basic form of empathy or instead does not constitute empathy. There may be a developmental aspect to this subdivision. Infants respond to the distress of others by getting distressed themselves; only when they are 2 years old do they start to respond in other-oriented ways, trying to help, comfort and share.

Cognitive empathy can be subdivided into the following scales:

- **Perspective taking**: the tendency to spontaneously adopt others' psychological perspectives.
- **Fantasy**: the tendency to identify with fictional characters.

In research

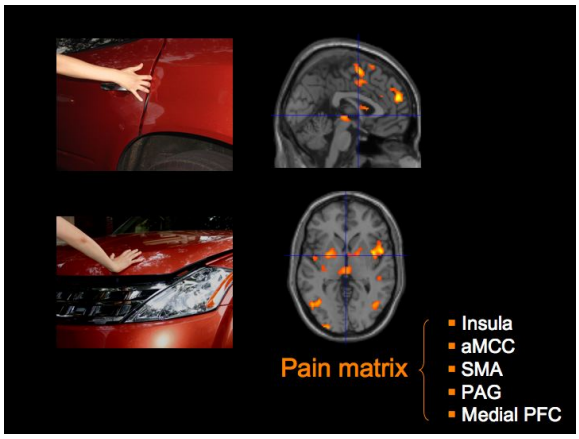
A difference in distribution between affective and cognitive empathy has been observed in various conditions. Psychopathy, schizophrenia, and narcissism have been associated with impairments in affective but not cognitive empathy, whereas bipolar disorder and borderline traits have been associated with deficits in cognitive but not affective empathy. Autism spectrum disorders have been associated with various combinations, including deficits in cognitive empathy as well as deficits in both cognitive and affective empathy. Even in people without conditions such as these, the balance between affective and cognitive empathy varies.

A meta-analysis of recent fMRI studies of empathy confirmed that different brain areas are activated during affective–perceptual empathy and cognitive–evaluative empathy. Also, a study with patients with different types of brain damage confirmed the distinction between emotional and cognitive empathy. Specifically, the inferior frontal gyrus appears to be responsible for emotional empathy, and the ventromedial prefrontal gyrus seems to mediate cognitive empathy.

The Interpersonal Reactivity Index (IRI) is the only published measurement tool to date that accounts for a multi-dimensional assessment of empathy. It comprises a self-report questionnaire of 28 items, divided into four 7-item scales covering the above subdivisions of affective and cognitive empathy.[]

Development

By the age of two years, children normally begin to display the fundamental behaviors of empathy by having an emotional response that corresponds with another person's emotional state.[8] Even earlier, at one year of age, infants have some rudiments of empathy, in the sense that they understand that, just like their own actions, other people's actions have goals.[9][10] Sometimes, toddlers will comfort others or show concern for them at as early an age as two. Also during the second year, toddlers will play games of falsehood or "pretend" in an effort to fool others, and this requires that the child know what others believe before he or she can manipulate those beliefs.



When children are shown videoclips with situations where they see people suffering pain by coincidence, neural circuits related to pain are being activated in their brain.

According to researchers at the University of Chicago who used functional magnetic resonance imaging (fMRI), children between the ages of 7 and 12 years appear to be naturally inclined to feel empathy for others in pain. Their findings are consistent with previous fMRI studies of pain empathy with adults. The research also found additional aspects of the brain were activated when youngsters saw another person intentionally hurt by another individual, including regions involved in moral reasoning.[11]

Despite being able to show some signs of empathy, such as attempting to comfort a crying baby, from as early as 18 months to two years, most children do not show a fully fledged theory of mind until around the age of four. Theory of mind involves the ability to understand that other people may have beliefs that are different from one's own, and is thought to involve the cognitive component of empathy. Children usually become capable of passing "false belief" tasks, considered to be a test for a theory of mind, around the age of four. Individuals with autism often find using a theory of mind very difficult (e.g. Baron-Cohen, Leslie & Frith, 1988; the Sally-Anne test).

Empathetic maturity is a cognitive structural theory developed at the Yale University School of Nursing and addresses how adults conceive or understand the personhood of patients. The theory, first applied to nurses and since applied to other professions, postulates three levels that have the properties of cognitive structures. The third and highest level is held to be a meta-ethical theory of the moral structure of care. Those adults operating with level-III understanding synthesize systems of justice and care-based ethics.

Neurological basis

Research in recent years has focused on possible brain processes underlying the experience of empathy. For instance, functional magnetic resonance imaging (fMRI) has been employed to investigate the functional anatomy of empathy.[12] These studies have shown that observing another person's emotional state activates parts of the neuronal network involved in processing that same state in oneself, whether it is disgust, touch,[13] or pain.[14] The study of the neural underpinnings of empathy has received increased interest following the target paper published by Preston and Frans de Waal, following the discovery of mirror neurons in monkeys that fire both when the creature watches another perform an action as well as when they themselves perform it.

In their paper, they argued that attended perception of the object's state automatically activates neural representations, and that this activation automatically primes or generates the associated autonomic and somatic responses (idea of perception-action-coupling[15]), unless inhibited. This mechanism is similar to the common coding theory between perception and action. Another recent study provides evidence of separate neural pathways activating reciprocal suppression in different regions of the brain associated with the performance of "social" and "mechanical" tasks. These findings suggest that the cognition associated with reasoning about the "state of another

person's mind" and "causal/mechanical properties of inanimate objects" are neurally suppressed from occurring at the same time.[16]

In animals

Empathy is easily noted and recognized in humans. However, new research has shown that the ability of empathy in other species is indeed attainable. Many instances of empathy have been recorded throughout many species, including but not limited to canines, felines, dolphins, primates, rats and mice. In animals, empathy-related responding could in fact have an ulterior motive such as survival, the sharing of food, companionship and pack-oriented mentality. It is certainly difficult to understand an animals intention behind an empathic response. Many researchers maintain that applying the term empathy in general to animal behavior is an act of anthropomorphism.

Researchers Clay and Zana studied the socio-emotional development of the bonobo chimpanzee. They focused on the interplay of nuemerous skills such as empathy-related responding, and how different rearing backgrounds of the juvenile bonobo affected their response to stressful events, related to themselves (loss of a fight) and of stressful events of others. It was found that the bonobos sought out body contact as a coping mechanism with one another. A finding of this study was that the bonobos sought out more body contact after watching a distressing event upon the other bonobos rather than their individually experienced stressful event. Mother-reared bonobos as opposed to orphaned bonobos sought out more physical contact after a stressful event happened to another. This finding shows the importance of mother-child attachment and bonding, and how it may be crucial to successful socio-emotional development, such as empathic-like behaviors.

Empathic-like responding has been observed in chimpanzees in various different aspects of their natural behaviors. For example, chimpanzees are known to spontaneously contribute comforting behaviors to victims of aggressive behavior in natural and unnatural settings, a behavior recognized as consolation. Researchers Romero and Teresa observed these empathic and sympathetic-like behaviors in chimpanzees at two separate outdoor housed groups. The act of consolation was observed in both of the groups of chimpanzees. This behavior is found in humans, and particularly in human infants. Another similarity found between chimpanzees and humans is that empathic-like responding was disproportionately provided to individuals of kin. Although comforting towards non-family chimpanzees was also observed, as with humans, chimpanzees showed the majority of comfort and concern to close/loved ones. Another similarity between chimpanzee and human expression of empathy is that females provided more comfort than males on average. The only exception to this discovery was that high-ranking males showed as much empathy-like behavior as their female counterparts. This is believed to be because of policing-like behavior and the authoritative status of high-ranking male chimpanzees.

It is thought that species that possess a more intricate and developed prefrontal cortex have more of an ability of experiencing empathy. It has however been found that empathic and altruistic responses may also be found in sand dwelling Mediterranean ants. Researcher Hollis studied the *Cataglyphis cursor* sand dwelling Mediterranean ant and their rescue behaviors by ensnaring ants from a nest in nylon threads and partially buried beneath the sand. The ants not ensnared in the nylon thread proceeded to attempt to rescue their nest mates by sand digging, limb pulling, transporting sand away from the trapped ant, and when efforts remained unfruitful, began to attack the nylon thread itself; biting and pulling apart the threads. Similar rescue behavior was found in other sand-dwelling Mediterranean ants, but only *Cataglyphis floricola* and *Lasius Grandis* species of ants showed the same rescue behaviors of transporting sand away from the trapped victim and directing attention towards the nylon thread. It was observed in all ant species that rescue behavior was only directed towards nest mates. Ants of the same species, even conspecifics were treated with aggression and were continually attacked and pursued, which speaks to the depths of ants discriminative abilities. This study brings up the possibility that if ants have the capacity for empathy and/or altruism, these complex processes may be derived from primitive and simpler mechanisms.

Canines have been hypothesized to share empathic-like responding towards human species. Researchers Custance and Mayer put individual dogs in an enclosure with their owner and a stranger. When the participants were talking or humming, the dog showed no behavioral changes, however when the participants were pretending to cry, the dogs oriented their behavior toward the person in distress whether it be the owner or stranger. The dogs approached the participants when crying in a submissive fashion, by sniffing, licking and nuzzling the distressed person. The dogs did not approach the participants in the usual form of excitement, tail wagging or painting. Since the dogs did not direct their empathic-like responses only towards their owner, it is hypothesized that dogs generally seek out humans showing distressing body behavior. Although this could insinuate that dogs have the cognitive capacity for empathy, this could also mean that domesticated dogs have learned to comfort distressed humans through generations of being rewarded for that specific behavior.

When witnessing chicks in distress, domesticated hens, *gallus gallus domesticus* show emotional and physiological responding. Researchers Edgar, Paul and Nicol found that in conditions where the chick was susceptible to danger, the mother hens heart rate increased, vocal alarms were sounded, personal preening decreased and body temperature increased. This responding happened whether or not the chick felt as if they were in danger. Mother hens experienced stress-induced hyperthermia only when the chick's behavior correlated with the perceived threat. Animal maternal behavior may be perceived as empathy, however, it could be guided by the evolutionary principles of survival and not emotionality.

Anger and distress

Anger

Empathic anger is an emotion, a form of empathic distress. Empathic anger is felt in a situation where someone else is being hurt by another person or thing. It is possible to see this form of anger as a pro-social emotion.[*citation needed*]

Empathic anger has direct effects on both helping and punishing desires. Empathic anger can be divided into two sub-categories: trait empathic anger and state empathic anger.[17]

The relationship between empathy and anger response towards another person has also been investigated, with two studies basically finding that the higher a person's perspective taking ability, the less angry they were in response to a provocation. Empathic concern did not, however, significantly predict anger response, and higher personal distress was associated with increased anger.

Distress

Empathic distress is feeling the perceived pain of another person. This feeling can be transformed into empathic anger, feelings of injustice, or guilt. These emotions can be perceived as pro-social, and some say they can be seen as motives for moral behavior.

Atypical empathic response

Atypical empathic responses have been associated with autism spectrum disorders; particular personality disorders such as psychopathy, borderline, narcissistic, and schizoid personality disorders; conduct disorder; schizophrenia; bipolar disorder; and depersonalization.

Autism spectrum

The interaction between empathy and the autism spectrum is a complex and ongoing field of research. Several different factors are proposed to be at play here.

Alexithymia

A study of high-functioning adults with autism spectrum disorders found an increased prevalence of alexithymia, a personality construct characterized by the inability to recognize and articulate emotional arousal in oneself or others.[18][19] Based on fMRI studies, alexithymia is responsible for a lack of empathy.[20] The lack of empathic attunement inherent to alexithymic states may reduce quality and satisfaction[21] of relationships. Recently, a study has shown that high-functioning adults with autism appear to have a range of responses to music similar to that of neurotypical individuals, including the deliberate use of music for mood management. Clinical treatment of alexithymia could involve using a simple associative learning process between musically induced emotions and their cognitive correlates.[22] A study has suggested that the empathy deficits associated with the autism spectrum may be due to significant comorbidity between alexithymia and autism spectrum conditions rather than a result of social impairment.

Mirror neuron activity

One study found that, relative to typically developing children, high-functioning children with autism showed reduced mirror neuron activity in the brain's inferior frontal gyrus (pars opercularis) while imitating and observing emotional expressions. EEG evidence revealed that there was significantly greater mu suppression in the sensorimotor cortex of autistic individuals. Activity in this area was inversely related to symptom severity in the social domain, suggesting that a dysfunctional mirror neuron system may underlie social and communication deficits observed in autism, including impaired theory of mind and empathy.[23] The mirror neuron system is essential for emotional empathy.

Theory of mind

Previous studies have suggested that autistic individuals have impaired theory of mind. Theory of mind is the ability to understand the perspectives of others. The terms cognitive empathy and theory of mind are often used synonymously, but due to a lack of studies comparing theory of mind with types of empathy, it is unclear whether these are equivalent. Theory of mind relies on structures of the temporal lobe and the pre-frontal cortex, and empathy, i.e. the ability to share the feelings of others, relies on the sensorimotor cortices as well as limbic and para-limbic structures.[*citation needed*] Francesca Happe showed that autistic children who demonstrate a lack of theory of mind lack it for their self as well as for others.[24]Wikipedia:Link rot[*citation needed*] The lack of clear distinctions between theory of mind and empathy may have resulted in an incomplete understanding of the empathic abilities of those with Asperger syndrome; many reports on the empathic deficits of individuals with Asperger syndrome are actually based on impairments in theory of mind.[25][26]

Cognitive and affective empathy

Studies have found that individuals on the autism spectrum self-report lower levels of empathic concern, show less or absent comforting responses toward someone who is suffering, and report equal or higher levels of personal distress compared to controls. The combination of reduced empathic concern and increased personal distress may lead to the overall reduction of empathy in those on the autism spectrum. Professor Simon Baron-Cohen suggests that those with classic autism often lack both cognitive and affective empathy. Research also suggests that people with Asperger syndrome may have problems understanding others' perspectives in terms of theory of mind, but on average demonstrate equal empathic concern as and higher personal distress than controls. The generally heightened personal distress in those with autism spectrum conditions has been offered as an explanation to the claim that at least some people with autism would appear to have heightened emotional empathy,[] although emotional empathy

depends on mirror neuron activity, which (as described previously) has been found to be reduced in those with autism, and empathy in people on the autism spectrum is generally reduced.

Empathizing–systemizing theory

The empathizing–systemizing (E-S) theory suggests that people may be classified on the basis of their capabilities along two independent dimensions, empathizing (E) and systemizing (S). These capabilities may be inferred through tests that measure someone's Empathy Quotient (EQ) and Systemizing Quotient (SQ). Five different "brain types" can be observed among the population based on the scores, which should correlate with differences at the neural level. In the E-S theory, autism and Asperger syndrome are associated with below-average empathy and average or above-average systemizing. The E-S theory has been extended into the Extreme Male Brain theory, which suggests that people with an autism spectrum condition are more likely to have an "Extreme Type S" brain type, corresponding with above-average systemizing but challenged empathy (see the next section).

Sex differences and autism

It has been shown that males are generally less empathetic than females.[27] The Extreme Male Brain (EMB) theory proposes that individuals on the autistic spectrum are characterized by impairments in empathy due to sex differences in the brain: specifically, people with autism spectrum conditions show an exaggerated male profile. A study showed that some aspects of autistic neuroanatomy seem to be extremes of typical male neuroanatomy, which may be influenced by elevated levels of fetal testosterone rather than gender itself.[28] Another study involving brain scans of 120 men and women suggested that autism affects male and female brains differently; females with autism had brains that appeared to be closer to those of non-autistic males than females, yet the same kind of difference was not observed in males with autism.

Personality disorders

Atypical empathy is associated with some personality disorders, including psychopathy, borderline, narcissistic, and schizoid personality disorders.

Psychopathy

Psychopathy is a personality disorder partly characterized by antisocial and aggressive behaviors, as well as emotional and interpersonal deficits including shallow emotions and a lack of remorse and empathy. The *Diagnostic and Statistical Manual of Mental Disorders* (DSM) and *International Classification of Diseases* (ICD) list antisocial personality disorder (ASPD) and dissocial personality disorder, stating that these have been referred to or include what is referred to as psychopathy.[29][30]

Studies by R.J.R. Blair and others suggest psychopathy is associated with atypical responses to distress cues (e.g. facial and vocal expressions of fear and sadness), including decreased activation of the fusiform and extrastriate cortical regions, which may partly account for impaired recognition of and reduced autonomic responsiveness to expressions of fear, and impairments of empathy.[31] Studies by Blair on children with psychopathic tendencies have also shown such associations. The underlying biological surfaces for processing expressions of happiness are functionally intact in psychopaths, although less responsive than those of controls.

A recent study in which psychopathic criminals were brain-scanned while they watched videos of a person harming another individual found that the psychopaths' empathic reaction (theorized to occur through the mirror system) initiated the same way it did for controls when they were instructed to empathise with the harmed individual, and that the area of the brain relating to pain was activated when the psychopaths were asked to imagine how the harmed individual felt. The research demonstrated how psychopaths could switch empathy on at will and would enable them to be both callous and charming. Professor Simon Baron-Cohen suggests that, unlike the combination of both reduced cognitive and affective empathy often seen in those with classic autism, psychopaths are associated with intact cognitive empathy, implying non-diminished awareness of another's feelings when they hurt someone.

Borderline personality disorder

Borderline personality disorder is characterized by extensive behavioral and interpersonal difficulties that arise from emotional and cognitive dysfunction. Dysfunctional social and interpersonal behavior has been shown to play a crucial role in the emotionally intense way people with borderline personality disorder react. While individuals with borderline personality disorder may show their emotions too much, several authors have suggested that they might have a compromised ability to reflect upon mental states (impaired cognitive empathy), as well as an impaired theory of mind.

People with borderline personality disorder are very good at recognizing emotions in people's faces, suggesting increased empathic capacities. It is, therefore, possible that impaired cognitive empathy (the capacity for understanding another person's experience and perspective) may account for borderline personality disorder individuals' tendency for interpersonal dysfunction, while "hyper-emotional empathy"Wikipedia:Verifiability may account for the emotional over-reactivity observed in these individuals. One primary study confirmed that patients with borderline personality disorder were significantly impaired in cognitive empathy, yet there was no sign of impairment in affective empathy.

Narcissistic personality disorder

One diagnostic criterion of narcissistic personality disorder is a lack of empathy and an unwillingness or inability to recognize or identify with the feelings and needs of others.[32]

Schizoid personality disorder

Characteristics of schizoid personality disorder include emotional coldness, detachment, and impaired affect corresponding with an inability to be empathetic and sensitive towards others.[33][34][35][36]

Conduct disorder

A study conducted by Jean Decety and colleagues at the University of Chicago demonstrated that subjects with aggressive conduct disorder elicit atypical empathic responses to viewing others in pain. Subjects with conduct disorder were at least as responsive as controls to the pain of others, but unlike controls, subjects with conduct disorder showed strong and specific activation of the amygdala and ventral striatum (areas that enable a general arousing effect of reward), yet impaired activation of the neural regions involved in self-regulation and metacognition (including moral reasoning), in addition to diminished processing between the amygdala and the prefrontal cortex.

Schizophrenia

Schizophrenics are characterized by impaired affective empathy, and have been observed to have severe cognitive and empathy impairments as measured by the Empathy Quotient (EQ). These empathy impairments are also associated with impairments in social cognitive tasks.

Bipolar disorder

Bipolar individuals have been observed to have impaired cognitive empathy and theory of mind, but increased affective empathy. Despite cognitive flexibility being impaired, planning behavior is intact. It has been suggested that dysfunctions in the prefrontal cortex could result in the impaired cognitive empathy, since impaired cognitive empathy has been related with neurocognitive task performance involving cognitive flexibility.

Depersonalization

Lieutenant Colonel Dave Grossman, in his book *On Killing*, suggests that military training artificially creates depersonalization in soldiers, suppressing empathy and making it easier for them to kill other human beings.

Practical issues

The capacity to empathize is a revered trait in society. Empathy is considered a motivating factor for unselfish, prosocial behavior,[37] whereas a lack of empathy is related to antisocial behavior.[38][39]

Proper empathic engagement helps an individual understand and anticipate the behavior of another. Apart from the automatic tendency to recognize the emotions of others, one may also deliberately engage in empathic reasoning. Two general methods have been identified here. An individual may simulate fictitious versions of the beliefs, desires, character traits and context of another individual to see what emotional feelings it provokes. Or, an individual may simulate an emotional feeling and then access the environment for a suitable reason for the emotional feeling to be appropriate for that specific environment.[*citation needed*]

Some research suggests that people are more able and willing to empathize with those most similar to themselves. In particular, empathy increases with similarities in culture and living conditions. Empathy is more likely to occur between individuals whose interaction is more frequent. (See Levenson and Reuf 1997 and Hoffman 2000: 62). A measure of how well a person can infer the specific content of another person's thoughts and feelings has been developed by William Ickes (1997, 2003). Ickes and his colleagues have developed a video-based method to measure empathic accuracy and have used this method to study the empathic inaccuracy of maritally aggressive and abusive spouses, among other topics.[*citation needed*]

There are concerns that the empathiser's own emotional background may affect or distort what emotions they perceive in others (e.g. Goleman 1996: p. 104). Empathy is not a process that is likely to deliver certain judgments about the emotional states of others. It is a skill that is gradually developed throughout life, and which improves the more contact we have with the person with whom one empathizes. Accordingly, any knowledge gained of the emotions of the other must be revisable in light of further information.[*citation needed*]

Ethical issues

The extent to which a person's emotions are publicly observable, or mutually recognized as such has significant social consequences. Empathic recognition may or may not be welcomed or socially desirable. This is particularly the case where we recognize the emotions that someone has towards us during real time interactions. Based on a metaphorical affinity with touch, philosopher Edith Wyschogrod claims that the proximity entailed by empathy increases the potential vulnerability of either party. The appropriate role of empathy in our dealings with others is highly dependent on the circumstances. For instance, Tania Singer claims that clinicians or caregivers must take care not to be too sensitive to the emotions of others, to over-invest their own emotions, at the risk of draining away their own resourcefulness.[40] Furthermore an awareness of the limitations of empathic accuracy is prudent in a caregiving situation.

Disciplinary approaches

Philosophy

Ethics

In his 2008 book, *Solving the Riddle of Right and Wrong*, philosopher Iain King presents two reasons why empathy is the "essence" or "DNA" of right and wrong. First, he argues that empathy uniquely has all the characteristics we can know about an ethical viewpoint[41] – including that it is "partly self-standing", and so provides a source of

motivation that is partly within us and partly outside, as moral motivations seem to be.[42] This allows empathy-based judgements to have sufficiently distance from a personal opinion to count as "moral". His second argument is more practical: he argues, "Empathy for others really is the route to value in life", and so the means by which a selfish attitude can become a moral one.[42] By using empathy as the basis for a system of ethics, King is able to reconcile ethics based on consequences with virtue-ethics and act-based accounts of right and wrong.[43] His empathy-based system has been taken up by some Buddhists,[44] and is used to address some practical problems, such as when to tell lies,[45] and how to develop culturally-neutral rules for romance.

In the 2007 book *The Ethics of Care and Empathy*, philosopher Michael Slote introduces a theory of care-based ethics that is grounded in empathy. His claim is that moral motivation does, and should, stem from a basis of empathic response. He claims that our natural reaction to situations of moral significance are explained by empathy. He explains that the limits and obligations of empathy and in turn morality are natural. These natural obligations include a greater empathic, and moral obligation to family and friends, along with an account of temporal and physical distance. In situations of close temporal and physical distance, and with family or friends, our moral obligation seems stronger to us than with strangers at a distance naturally. Slote explains that this is due to empathy and our natural empathic ties. He further adds that actions are wrong if and only if they reflect or exhibit a deficiency of fully developed empathic concern for others on the part of the agent.[46]

Phenomenology

In phenomenology, empathy describes the experience of something from the other's viewpoint, without confusion between self and other. This draws on the sense of agency. In the most basic sense, this is the experience of the other's body and, in this sense, it is an experience of "my body over there". In most other respects, however, the experience is modified so that what is experienced is experienced as being the other's experience; in experiencing empathy, what is experienced is not "my" experience, even though *I* experience it. Empathy is also considered to be the condition of intersubjectivity and, as such, the source of the constitution of objectivity.[47]

History

Some postmodern historians such as Keith Jenkins in recent years have debated whether or not it is possible to empathise with people from the past. Jenkins argues that empathy only enjoys such a privileged position in the present because it corresponds harmoniously with the dominant liberal discourse of modern society and can be connected to John Stuart Mill's concept of reciprocal freedom. Jenkins argues the past is a foreign country and as we do not have access to the epistemological conditions of by gone ages we are unable to empathise.[48]

It is impossible to forecast the effect of empathy on the future.[*citation needed*] A past subject may take part in the present by the so-called historic present. If we watch from a fictitious past, can tell the present with the future tense, as it happens with the trick of the false prophecy. There is no way of telling the present with the means of the past.[49]

Evolution

An increasing number of studies in animal behavior and neuroscience claim that empathy is not restricted to humans, and is in fact as old as the mammals, or perhaps older. Examples include dolphins saving humans from drowning or from shark attacks. Professor Tom White suggests that reports of cetaceans having three times as many spindle cells — the nerve cells that convey empathy — in their brains as we do might mean these highly social animals have a great awareness of one another's feelings.[50]

A multitude of behaviors observed in primates, both in captivity and in the wild, and in particular in bonobos, which are reported as the most empathetic of all the primates.[51] A recent study has demonstrated prosocial behavior elicited by empathy in rodents.

Rodents have been shown to demonstrate empathy for cagemates (but not strangers) in pain. One of the most widely read studies on the evolution of empathy, which discusses a neural perception-action mechanism (PAM), is the one by Stephanie Preston and de Waal (). This review postulates a bottom-up model of empathy that ties together all levels, from state matching to perspective-taking. For University of Chicago neurobiologist Jean Decety, [empathy] is not specific to humans. He argues that there is strong evidence that empathy has deep evolutionary, biochemical, and neurological underpinnings, and that even the most advanced forms of empathy in humans are built on more basic forms and remain connected to core mechanisms associated with affective communication, social attachment, and parental care. Core neural circuits that are involved in empathy and caring include the brainstem, the amygdala, hypothalamus, basal ganglia, insula and orbitofrontal cortex.

Psychotherapy

Heinz Kohut is the main introducer of the principle of empathy in psychoanalysis. His principle applies to the method of gathering unconscious material. The possibility of not applying the principle is granted in the cure, for instance when you must reckon with another principle, that of reality. Developing skills of empathy is often a central theme in the recovery process for drug addicts.[*citation needed*]

In evolutionary psychology, attempts at explaining pro-social behavior often mention the presence of empathy in the individual as a possible variable. Although exact motives behind complex social behaviors are difficult to distinguish, the "ability to put oneself in the shoes of another person and experience events and emotions the way that person experienced them" is the definitive factor for truly altruistic behavior according to Batson's empathy-altruism hypothesis. If empathy is not felt, social exchange (what's in it for me?) supersedes pure altruism, but if empathy is felt, an individual will help by actions or by word, regardless of whether it is in their self-interest to do so and even if the costs outweigh potential rewards.

Education

An important target of the method Learning by teaching (LbT) is to train systematically and, in each lesson, teach empathy. Students have to transmit new content to their classmates, so they have to reflect continuously on the mental processes of the other students in the classroom. This way it is possible to develop step-by-step the students' feeling for group reactions and networking. Carl R. Rogers pioneered research in effective psychotherapy and teaching which espoused that empathy coupled with unconditional positive regard or caring for students and authenticity or congruence were the most important traits for a therapist or teacher to have. Other research and publications by Tausch, Aspy, Roebuck. Lyon, and meta-analyses by Cornelius-White, corroborated the importance of these person-centered traits.[52][53]

Business and management

In the 2009 book *Wired to Care*, strategy consultant Dev Patnaik argues that a major flaw in contemporary business practice is a lack of empathy inside large corporations. He states that lacking any sense of empathy, people inside companies struggle to make intuitive decisions and often get fooled into believing they understand their business if they have quantitative research to rely upon. Patnaik claims that the real opportunity for companies doing business in the 21st Century is to create a widely held sense of empathy for customers, pointing to Nike, Harley-Davidson, and IBM as examples of "Open Empathy Organizations". Such institutions, he claims, see new opportunities more quickly than competitors, adapt to change more easily, and create workplaces that offer employees a greater sense of mission in their jobs.[54] In the 2011 book *The Empathy Factor*, organizational consultant Marie Miyashiro similarly argues the value of bringing empathy to the workplace, and offers Nonviolent Communication as an effective mechanism for achieving this. In studies by the Management Research Group, empathy was found to be the strongest predictor of ethical leadership behavior out of 22 competencies in its management model, and empathy was one of the three strongest predictors of senior executive effectiveness.

Intercultural

Intercultural empathy is the ability to perceive the world as it is perceived by a culture different from the subject's own. Empathy interculturally regards a variety of issues, such as the approach to time perception (deadlines, temporal precision, perspective time), how to negotiate with people from different cultures and organizations, and be able to integrate all possible difference of communication styles due to differences in culture. The literature distinguishes four levels of empathy, identified by the Italian researcher Daniele Trevisani (2005) that examines the dimensions useful for applying empathic component on the intercultural setting:

1. **Behavioral empathy**: understanding the behavior of a different culture and their causes, the ability to understand why the behavior is adopted and the chains of related behaviors.
2. **Emotional empathy**: being able to feel the emotions experienced by others, even in cultures different from their own, understand what emotions feels the culturally different person (which emotion is flowing), of which intensity, which are the emotional lives, how emotions are associated to people, objects, events, situations, in private or public aspects of different cultures.
3. **Relational empathy**: understanding the map of the relations of the subject and its affective value in the culture of belonging, to understand with whom the subject relates whether voluntarily or compulsorily, who has to deal with that subject in order to decide, in work or life, what is his map of "significant others ", the referents, the interlocutors, "other relevant "and influencers affecting their decisions, who are enemies and friends, who can affects his/her professional and life decisions.
4. **Cognitive empathy** (understanding of different cognitive or prototypes): understanding the cognitive prototypes active in a given moment of time in a certain culture in a single person, the beliefs that generate the visible values, ideologies underlying behaviors, identifying the mental structures that the individuals own and which parts are culturally-depending" (Trevisani, 2005).

Fiction

Some philosophers (such as Martha Nussbaum) suggest that novel reading cultivates readers' empathy and leads them to exercise better world citizenship. For a critique of this application of the empathy-altruism hypothesis to experiences of narrative empathy, see Keen's *Empathy and the Novel* (Oxford, 2007). In some works of science fiction and fantasy, empathy is understood to be a paranormal or psychic ability to sense the emotions of others, as opposed to telepathy, which allows one to perceive thoughts as well. A person who has that ability is also called an "empath" or "telepath" in this context. Occasionally these empaths are also able to project their own emotions, or to affect the emotions of others.[*citation needed*]

Measurement

Research into the measurement of empathy has sought to answer a number of questions: who should be carrying out the measurement? What should pass for empathy and what should be discounted? What unit of measure (UOM) should be adopted and to what degree should each occurrence precisely match that UOM are also key questions that researchers have sought to investigate.

Researchers have approached the measurement of empathy from a number of perspectives.

Behavioural measures normally involve raters assessing the presence or absence of certain either predetermined or ad-hoc behaviours in the subjects they are monitoring. Both verbal and non-verbal behaviours have been captured on video by experimenters such as Truax (1967b).[55] Other experimenters, including Mehrabian and Epstein (1972), have required subjects to comment upon their own feelings and behaviours, or those of other people involved in the experiment, as indirect ways of signalling their level of empathic functioning to the raters.

Physiological responses tend to be captured by elaborate electronic equipment that has been physically connected to the subject's body. Researchers then draw inferences about that person's empathic reactions from the electronic

readings produced (e.g. Levenson and Ruef, 1992; Leslie et al., 2004).

Bodily or "somatic" measures can be looked upon as behavioural measures at a micro level. Their focus is upon measuring empathy through facial and other non-verbally expressed reactions in the empathiser. These changes are presumably underpinned by physiological changes brought about by some form of "emotional contagion" or mirroring (e.g. Levenson and Ruef, 1992*; Leslie et al., 2004*). It should be pointed out that these reactions, whilst appearing to reflect the internal emotional state of the empathiser, could also, if the stimulus incident lasted more than the briefest period, be reflecting the results of emotional reactions that are based upon more pieces of thinking through (cognitions) associated with role-taking ("if I were him I would feel ...").

Paper-based indices involve one or more of a variety of methods of responding. In some experiments, subjects are required to watch video scenarios (either staged or authentic) and to make written responses which are then assessed for their levels of empathy (e.g. Geher, Warner and Brown, 2001); scenarios are sometimes also depicted in printed form (e.g. Mehrabian and Epstein, 1972). Measures also frequently require subjects to self-report upon their own ability or capacity for empathy, using Likert-style numerical responses to a printed questionnaire that may have been designed to tap into the affective, cognitive-affective or largely cognitive substrates of empathic functioning. Some questionnaires claim to have been able to tap into both cognitive and affective substrates (e.g. Davis, 1980). More recent paper-based tools include The Empathy Quotient (EQ) created by Baron-Cohen and Wheelwright which comprises a self-report questionnaire consisting of 60 items.

For the very young, picture or puppet-story indices for empathy have been adopted to enable even very young, pre-school subjects to respond without needing to read questions and write answers (e.g. Denham and Couchoud, 1990). Dependent variables (variables that are monitored for any change by the experimenter) for younger subjects have included self reporting on a 7-point smiley face scale and filmed facial reactions (Barnett, 1984).

A certain amount of confusion exists about how to measure empathy. These may be rooted in another problem: deciding what is empathy and what is not. In general, researchers have until now been keen to pin down a singular definition of empathy which would allow them to design a measure to assess its presence in an exchange, in someone's repertoire of behaviours or within them as a latent trait. As a result they have been frequently forced to ignore the richness of the empathic process in favour of capturing surface, explicit self-report or third-party data about whether empathy between two people was present or not. In most cases, instruments have unfortunately only yielded information on whether someone had the potential to demonstrate empathy (Geher et al., 2001)*. Gladstein (1987)[56] summarises the position noting that empathy has been measured from the point of view of the empathiser, the recipient for empathy and the third-party observer. He suggests that since the multiple measures used have produced results that bear little relation to one another, researchers should refrain from making comparisons between scales that are in fact measuring different things. He suggests that researchers should instead stipulate what kind of empathy they are setting out to measure rather than simplistically stating that they are setting out to measure the unitary phenomenon "empathy"; a view more recently endorsed by Duan and Hill (1996).

In the field of medicine, a measurement tool for carers is the *Jefferson Scale of Physician Empathy, Health Professional Version (JSPE-HP).* At least one study using this tool with health sciences' students has found that levels of empathy are greater amongst females than males, and also are greater amongst older students than younger students.

The Interpersonal Reactivity Index (IRI) is the only published measurement tool accounting for a multi-dimensional assessment of empathy, consisting of a self-report questionnaire of 28 items, divided into four 7-item scales covering the subdivisions of affective and cognitive empathy.

Gender differences

The issue of gender differences in empathy is quite controversial. It is often believed that females are more empathic than males. Evidence for gender differences in empathy are important for self-report questionnaires of empathy in which it is obvious what was being indexed (e.g., impact of social desirability and gender stereotypes) but are smaller or nonexistent for other types of indexes that are less self-evident with regard to their purpose. On average female subjects score higher than males on the Empathy Quotient (EQ), while males tend to score higher on the Systemizing Quotient (SQ).

Both males and females with Autistic Spectrum Disorders usually score higher on the SQ (Baron-Cohen, 2003). However, a series of recent studies, using a variety of neurophysiological measures, including MEG, spinal reflex excitability, and electroencephalography have documented the presence of a gender difference in the human mirror neuron system, with female participants exhibiting stronger motor resonance than male participants. In addition, these aforementioned studies found that female participants scored higher on empathy self-report dispositional measures and that these measures positively correlated with the physiological response. However, other studies show that women do not possess greater empathic abilities than men, and perceived gender differences are the result of motivational differences.[57] Using fMRI, neuroscientist Tania Singer showed that empathy-related neural responses are significantly lower in males when observing an "unfair" person experiencing pain.

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External links

- The dictionary definition of empathy at Wiktionary
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Fictional portrayals of psychopaths

Fictional portrayals of psychopaths, or **sociopaths**, are some of the most notorious in film and literature but may only vaguely or partly relate to the concept of psychopathy, which is itself used with varying definitions by mental health professionals, criminologists and others. The character may be identified as a diagnosed/assessed psychopath or sociopath within the fictional work itself, or by its creator when discussing their intentions with the work, which might be distinguished from opinions of audiences or critics based only on a character appearing to show traits or behaviors associated with an undefined popular stereotype of psychopathy.

Such characters are often portrayed in an exaggerated fashion and typically in the role of a villain or antihero, where the general characteristics of a psychopath are useful to facilitate conflict and danger. Because the definitions and criteria in the history of psychopathy have varied over the years and continue to change even now, many characters in notable films may have been designed to fall under the category of a psychopath at the time of the film's production or release, but not necessarily in subsequent years. There are several stereotypical images of psychopathy in both lay and professional accounts which only partly overlap and can involve contradictory traits: the charming con artist, the deranged serial killer, the successful corporate psychopath, or the chronic low-level offender with Juvenile delinquency. The public concept reflects some combination of fear of the mythical bogeyman, fascination with human evil, and sometimes perhaps envy of people who might appear to go through life unencumbered by the same levels of guilt, anguish or insecurity.

Early depictions

In the 19th century the diagnostic categories of monomania or moral insanity (the word 'moral' then meant either emotional or ethical) made their way into works of literature, covering numerous eccentricities, obsessions or breakdowns - and sometimes acts of apparently senseless criminality, occasionally violent! This period also saw the rise of crime fiction such as sensation novels, where often someone in a local community who appeared normal would turn out to be criminally insane, and detective novels, playing on increasing anxieties about the characters of people in the newly expanding and diversifying industrial cities. The term 'psychopath' came into use in the late 19th century (as did the term it would often be confused with, psychotic), and also spanned a very wide range of conditions (etymologically and originally equivalent to 'mentally ill'). Nevertheless an early rise to prominence followed its use in a Russian trial between 1883 and 1885 concerning a child murder, contributing to the release of a probable false confessor while the original suspect was found guilty. 'Psychopaths' began to appear in vaudevilles, ditties (songs) and press articles. The psychopathy defense was reported internationally as having enabled a remorseless female child killer to go free, a usage still quoted in dictionaries today.[1][2]

'Degenerates' were also depicted in popular fiction of the 19th through to mid 20th century, sometimes in similar ways to the modern usages of the concept of psychopaths, sometimes being cited as a cause of psychopathy. The concept fell into disrepute due partly to its use by the Nazis to justify eradicating their opposition.[3]

Early 20th century

The meaning gradually narrowed, initially as 'psychopathic inferiors' covering all of what today might be called personality disorders and various other conditions, then intertwining with the terminology of the 'sociopath' (and eventually antisocial personality disorder), though psychopathy remained variously defined in both broad and narrow ways.

Early representations of psychopaths in film were often caricatured as sadistic, unpredictable, sexually depraved, and emotionally unstable (manic) characters with a compulsion to engage in random violence and destruction, usually with a series of bizarre mannerisms such as giggling, laughing, or facial tics. Up until the late 1950s, American cinematic conventions usually relegated the psychopath to roles of genre villains such as gangsters, mad scientists, supervillains, and many types of generic criminals. Examples of this type are Tommy Udo (Richard Widmark) in *Kiss of Death*, Cody Jarrett (James Cagney) in *White Heat*, and Antonio 'Tony' Camonte (Paul Muni) in the 1932 version of *Scarface*.[*citation needed*] Homosexuals were also referred to as psychopaths under the broad definition then in use; the American Psychiatric Association in the first Diagnostic Statistics Manual in 1952 would list it under 'sociopathic personality disturbance'.

One exceptional depiction in this period was the character of child murderer Hans Beckert (Peter Lorre) in the 1931 Fritz Lang film *M*. Lorre portrays Beckert as an outwardly unremarkable man tormented by a compulsion to ritualistically murder children. A German film (allegedly based on the real life Peter Kürten), it was released in America in 1933 and has been seen as indicative of a turning point in American media depictions of psychopaths. Until the 1930s psychiatrists typically applied the diagnosis to unemployed males or 'hypersexual' women, but several psychiatric, cultural and economic trends, together with sex crime panics, converged to transform the popular psychopath into a violent, male, sexual deviant or criminal - a threat to innocence, gender roles and the social order.[4]

Mid 20th century

One of the earliest real life cases which had a pervasive influence on American movies was that of Ed Gein, arrested in 1957. A farmer who had resided with his mother until her death, he had then killed two women and dug up female bodies from the local cemetery, making various items out of their skin. Rumours spread that he was also a necrophiliac, cannibal or transvestite, though these appear to have been unsupported other than by brief affirmations from Gein to leading questions by interrogators.[5][6] Gein was found mentally ill and legally insane before trial, deemed to have had schizophrenia (psychosis including delusions and hallucinations) for at least 12 years, though at least one media psychiatrist dubbed him instead a 'sexual psychopath'.[7][8] Robert Bloch, a prolific pulp horror author, says his 1959 novel *Psycho* was based on the Gein murders and the idea of an apparently sane person in a local community committing heinous crimes, but not necessarily on Gein himself, despite numerous similarities.[9] The villain, Norman Bates, is portrayed as an outwardly unremarkable man who murders a woman while under the control of an alternate personality that takes the form of his domineering mother, who he himself murdered. Both the novel and Alfred Hitchcock's 1960 film adaptation were influences on the popular media portrayal of psychopaths.[10][11] Neither the book or film elaborates on the term 'psycho', though it is commonly taken to refer to either psychotic or psychopath. The psychiatrist describes Bates as having a split personality. Multiple Personality Disorder was at that time very popular (cf 1957 movie *The Three Faces of Eve*) and is commonly confused with schizophrenia. Bloch later wrote a script for the 1966 film *The Psychopath*, the original working title for which was "Schizo".[12]

A different thread within fictional portrayals of psychopathy continued to focus on low-level rebelliously antisocial characters. The title of the 1955 film *Rebel Without a Cause*, starring James Dean, came from a 1944 book of the same name detailing The Hypnoanalysis of a diagnosed psychopath, in which psychiatrist Robert M. Lindner also discussed psychopaths in general as pointlessly selfish individuals who appear unable to accept society's rules. In Ken Kesey's 1962 novel *One Flew Over the Cuckoo's Nest*, the protagonist Randle McMurphy is repeatedly referred

to by the authorities, other patients and himself as a possible or definite psychopath. He reads from his record: 'repeated outbreaks of passion that suggest the possible diagnosis of psychopath', and adds that a doctor told him it means 'I fight and fuh-pardon me, ladies-means I am he put it overzealous in my sexual relations.' The current doctor then reads out the note: "Don't overlook the possibility that this man might be feigning psychosis".[13] In the script for the popular film adaptation in 1975, only the latter is retained and the term psychopath is never used. Ironically the coldly clinical and controlling Nurse Ratched would be described as a psychopath by observers under later understandings of the term.[14]

Late 20th century

The 1973 film Badlands involved two lead characters based loosely on a young serial killing couple from the late 1950s, Charles Starkweather (who was influenced by watching Rebel Without a Cause) and Caril Ann Fugate. While the male lead Kit (Martin Sheen) is sometimes described as a psychopath or sociopath, psychologist Robert D. Hare, a leading proponent of the assessment of psychopathy, has identified Holly (Sissy Spacek) as exemplifying his concept of a psychopath due to her poor emotional sense of the meaning of events and her attempted mask of normality. However, writer and director Terrence Malick has said he considered Kit's shallow bitter insensitivity to be a result of suffering and neglect growing up in the Midwest, and 15-year old Holly, though immature and humorously mis-estimating her audience, to be a quite typical Southern (born in Texas) girl wanting to help narrate and come off well but still give the hard facts, and not dwell on herself or on personal tragedies as that would not be proper.[15][16]

An increasing panic about civilian serial killers from the late 1960s, fuelled by cases described as psychopaths such as John Wayne Gacy (1978), Ted Bundy (1978) and Jeffrey Dahmer (1991), lent an additional momentum in the way psychopathy was both perceived and portrayed in film and literature, sometimes incorporating a hybrid of traditional psychopaths from early film and late-19th Century literature with the high-functioning behaviors detected in some serial killers.[17][18]

The cannibalistic psychiatrist Dr. Hannibal Lecter, most notably portrayed by Anthony Hopkins in the Academy Award-winning 1991 film *The Silence of the Lambs*, is perhaps the most infamous fictional 'psychopath'. Lecter is intelligent and sophisticated (whereas psychopathy is generally associated with lower than average verbal intelligence), and his disarming charisma and wit disguise his true nature as a serial killer. He spends most of the film in a cell, taunting protagonist Clarice Starling with clues to the identity of another serial killer, Buffalo Bill, in exchange for intimate details of Starling's troubled childhood. The Lecter movies are based on books by Thomas Harris first published in 1981, in which Lecter's patient evaluations are described as all being different but they call him a sociopath as they can't understand him. In the Silence of the Lambs he is simply referred to as a pure psychopath.[19] In 2013 Harris revealed that he originally based the Lecter character on Alfredo Balli Trevino, a Mexican physician who had killed and chopped up his homosexual lover who was also a doctor, in what was classed as a crime of passion over a financial dispute.[20][21] While Lecter is not described as homosexual, Buffalo Bill is bisexual and (pseudo) transgender and trying to use the skin of victims to look like a woman.

American Psycho was also published in 1991, turned into a movie in 2000. Bret Easton Ellis has told interviewers that the book is a satire on shallow consumerist lifestyles, but also that the writing of the violent scenes was based on fictional horror and FBI material on serial killers, along with how he imagined "a psychotic who worked on Wall Street" (Patrick Bateman) would describe such incidents. Some commentary, including in scientific journals, has suggested the Bateman character appears to be a psychopath, but Bateman appears to be an unreliable narrator with psychosis so that it is unclear which of his reported acts really occurred.[22][23]

In the 1993 book Girl, Interrupted and its 1999 film adaptation, which have been described as female versions of One Flew Over The Cuckoo's Nest, the character of Lisa, played by Angelina Jolie, is a mischievously rebellious young women who is proudly diagnosed as a sociopath.[24]

1996's Primal Fear (film) played on fears about the insanity defense and malingering, depicting a killer and possible victim of sex abuse appearing to suffer from multiple personality disorder, who at the end lets slip his deliberate creation of his alter ego. In the film one character asks "But don't we all invent psychopaths to do our dirty work for us, doctor?", while in the book of the same name by William Diehl the psychiatrist 'Molly' and others repeatedly explain psychopathy and psychosis as if the same, inherently antisocial, condition.

The author J. K. Rowling has described her Dark Lord character of Voldemort in the Harry Potter books and films as "a raging psychopath, devoid of the normal human responses to other people's suffering, and there ARE people like that in the world".

21st century

The book and film The Corporation discussed the legal fiction, especially in America, that a business entity is a legal personality, and concluded that it inherently meets the criteria for being a psychopath, which some consider to be a metaphorical usage.[25][26]

Numerous characters in television shows are informally described as psychopaths by the actors who play the parts, including in the UK the characters Natalie Buxton in Bad Girls,[27] Sean Slater and Michael Moon in EastEnders,[28][29] Dexter Morgan in the American show Dexter[30] and the Master in Doctor Who.[*citation needed*]

The Dan Wells novel *I Am Not a Serial Killer* and its sequels, *Mr. Monster* and *I Don't Want to Kill You*, are narrated by a teenage diagnosed sociopath who sets himself rigid rules of behavior in order to lead a moral life.[*citation needed*]

One Chicago academic has argued in a review of TV trends that the contemporary fantasy of sociopathy is of someone whose emotional disconnection from others in society, rather than being the hindrance that it can represent in real clinical cases, enables them to be an amazingly successful manipulator due to a breakdown in the social contract.[31]

Contemporary advice on writing psychopathic/sociopathic characters suggests that lack of a conscience and lack of empathy are always the chief characteristics, along with an ability to fool others, while the type of selfish antisocial behavior, and any quirky secondary characteristics, can vary.[32]

In 2013, the same year in which the DSM-5 was published, again with a category of antisocial personality disorder noted to also have been called psychopathy or sociopathy, the video console game GTA 5 was released with numerous references to psychopaths and sociopaths, including in tailored reports from a fictional psychiatrist. One of the lead characters 'Trevor' is described as a psychopath but also as psychotic; the voice actor who played the part says his acting was influenced by Tom Hardy's portrayal of Charles Bronson (prisoner) (assumed name after the actor Charles Bronson) in Bronson (film).[33]

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History of psychopathy

Psychopathy, from psych (soul or mind) and pathy (suffering or disease), was coined by German psychiatrists in the 19th century and originally just meant what would today be called mental disorder, the study of which is still known as psychopathology. By the turn of the century 'psychopathic inferiority' referred to the type of mental disorder that might now be termed personality disorder, along with a wide variety of other conditions now otherwise classified. Through the early 20th century this and other terms such as 'constitutional (inborn) psychopaths' or 'psychopathic personalities', were used very broadly to cover anyone who violated legal or moral expectations or was considered inherently socially undesirable in some way.

The term **sociopathy** was popularized from 1929/30 by an American psychologist, originally intended as an alternative term to indicate that the defining feature was a pervasive failure to adhere to societal norms in a way that could harm others. The term psychopathy also gradually narrowed to the latter sense, based on interpretations of the work of a Scottish psychiatrist and especially checklists popularized by an American psychiatrist and later a Canadian psychologist. Psychopathy became defined in these quarters as a constellation of personality traits allegedly associated with immorality, criminality, or in some cases socioeconomic success.

Official psychiatric diagnostic manuals adopted a mixture of approaches, eventually going by the term Antisocial or Dissocial Personality Disorder. In the meantime concepts of psychopaths/sociopaths had became notorious among the general public and as characters in fiction.

Early literature

Historical descriptions of people or characters are sometimes noted in discussions of psychopathy, with claims of superficial resemblance or retrospective diagnosis. For example a brief vignette by Theophrastus in Ancient Greece concerning *The Unscrupulous Man*.[1] On the other hand, the ancient Greek military statesman Alcibiades has been described as the best example of a probable psychopath - due to inconsistent failures despite his potential and confident speaking.[2] Figures of insanity (e.g. vagabonds, libertines, the "mad") have, at least since the 18th century, often represented an image of darkness and threat to society, as later would "the psychopath" - a mixture of concepts

of dangerousness, evil and illness.[3]

Early clinical concepts

Psychiatric concepts began to develop in the early 19th century which to some extent fed into the use of the term psychopathy from the late 19th century, when that term still had a different and far broader meaning than today. In 1801, French psychiatrist Philippe Pinel described without moral judgment patients who appeared mentally unimpaired but who nonetheless engaged in impulsive and self-defeating acts. He described this as insanity without confusion/delusion (manie sans délire), or rational insanity (la folie raisonnante), and his anecdotes generally described people carried away by instinctive fury (instincte fureur). American Benjamin Rush wrote in 1812 about individuals with an apparent "perversion of the moral faculties", which he saw as a sign of innate defective organization. He also saw such people as objects of compassion whose mental alienation could be helped, even if that was in prison or what he referred to as the "christian system of criminal jurisprudence". In 1835 English psychiatrist James Cowles Prichard, based partly on Pinel's publications, developed a broad category of mental disorder he called moral insanity - a "madness" of emotional or social dispositions without significant delusions or hallucinations. Generally Prichard referred more to eccentric behaviour than, as had Pinel, out of control passions. Prichard's diagnosis came into widespread use in Europe for several decades. None of these concepts are comparable to current specific constructs of psychopathy, or even to the broader category of personality disorders. Moreover, "moral" did not necessarily refer at that time to morality but to the psychological or emotional faculties.

In the latter half of the 19th century the (pseudo) scientific study of individuals thought to lack a conscience flourished. Notably the Italian physician Cesare Lombroso rejected the view that criminality could occur in anyone and sought to identify particular "born criminals" whom he thought showed certain physical signs, such as proportionately long arms or a low and narrow forehead.[4] By the beginning of the 20th century the English psychiatrist Henry Maudsley was writing about not just "moral insanity" but the "moral imbecile" and "criminal psychosis", conditions he believed were genetic in origin and impervious to punishment or correction, and which he applied to the lower class of chronic offenders by comparison to "the higher industrial classes".[][5]

First uses of term

Initially physicians who specialised in mental disorders might be referred to as psychopaths (e.g. the *American Journal of Medical Science* in 1864) and their hospitals as psychopathic institutions (compare to the etymologically similar use of the term homeopathic). Treatments of physical conditions by psychological or spiritualist methods might be referred to as psychopathic.[6]

Up to the 1840s, the term psychopathy was also used in a way consistent with its etymology to refer to any illness of the mind. German psychiatrist von Feuchtersleben's (1845) *The Principles of Medical Psychology*, which was translated into English, used it in this sense, as well as the roughly equivalent new term psychosis, now traced back to Karl Friedrich Canstatt's *Handbuch der Medicinischen Klinik* (1841).[7][8][9] William Griesinger (1868) and Krafft-Ebing (1886) also notably employed the term in distinct ways.

The use of the term in a criminological context was popularised by a high profile legal case in Russia between 1883 and 1885, concerning the murder of a girl who had previously lived in Britain for some time, Sarah Becker (Sarra Bekker). The owner of the pawnbroker shop in which she worked and where her body was found, a retired military man Mr Mironovich, was eventually convicted on circumstantial evidence and imprisoned. In the meantime, however, a Ms Semenova had handed herself in saying she had killed Becker while trying to steal jewellery with her lover Bezak, a married policeman, though she soon recanted and changed her confession. Semenova was found not guilty following testimony from eminent Russian psychiatrist Prof Ivan M. Balinsky, who described her as a psychopath, still then a very general term. Dictionaries to this day note this as the first use of the noun, via British or American articles which had suggested a known murderer had been released and in some cases that psychopaths should be immediately hanged.[10][11]

In 1888 Julius Ludwig August Koch first published on his concept of "psychopathic inferiority" (psychopathische Minderwertigkeiten), which would become influential domestically and internationally. He used it to refer to various kinds of dysfunction or strange conduct noted in patients in the absence of obvious mental illness or retardation. Koch was a Christian and also influenced by the degeneration theory popular in Europe at the time, though he referred to both congenital and acquired types. Habitual criminality was only a small part of his concept but the German public soon used the shortened version "inferiors" to refer to anyone supposedly suffering from an inherent ('constitutional') disposition toward crime.[12][13][14]

Early 20th century

Some writers would still use psychopathy in the general sense of mental illness, such as Austrian psychiatrist Sigmund Freud in *Psychopathic Characters on Stage*.[15] By contrast influential German psychiatrist Emil Kraepelin, who had previously included a section on moral insanity in his psychiatric classification scheme, was by 1904 referring to specific psychopathic subtypes all involving antisocial, criminal or dissocial behaviour, including: born criminals (inborn delinquents), liars and swindlers, querulous persons, and driven persons (including vagabonds, spendthrifts, and dipsomaniacs). The influential Adolf Meyer (psychiatrist) spread the concept of constitutional psychopathy when he emigrated to the US, though unlike Koch he separated out cases of what was termed psychoneurosis.

After World War I German psychiatrists dropped the term inferiors/defectives (Minderwertigkeiten) and used psychopathic (psychopathisch) and its derivatives instead, at that time a more neutral term covering a wide range of conditions. Emil Kraepelin, Kurt Schneider and Karl Birnbaum developed categorisation schemes under the heading 'psychopathic personality', only some subtypes of which were thought to have particular links to antisocial behaviour. Schneider in particular advanced the term and tried to formulate it in less judgemental terms than Kraepelin, though infamously defining it as ‘those abnormal personalities who suffer from their abnormality or from whose abnormality society suffers.’[16] In a similar vein, Birnbaum, a biological psychiatrist, suggested from 1909 a concept similar to sociopathy, implying the social environment could determine whether dispositions became criminal or not.

From 1917 a forerunner to later diagnostic manuals, called the Statistical Manual for the Use of Institutions for the Insane, included a category of 'psychoses with constitutional psychopathic inferiority'. This covered abnormalities in the emotional and volitional spheres associated with episodic disturbances which did not fit into the established categories of psychosis: "The type of behavior disorder, the social reactions, the trends of interests, etc., which the psychopathic inferior may show give special features to many cases, e. g., criminal traits, moral deficiency, tramp life, sexual perversions and various temperamental peculiarities." Constitutional psychopathic inferiority without psychosis was listed separately as one term to apply to patients considered 'Not insane'.[17] Meanwhile the American Prison Association had its own definition, in which psychopathic personalities were considered non-psychotic and characterized by failure to adjust to environment, lacking purpose, ambition and proper feelings, while often showing tendencies towards delinquency, lying and various eccentricities, perversions or manias (including dromomania (compulsion to travel or experience new lifestyles), kleptomania (stealing), pyromania (fire-setting) etc.). In the UK the Mental Deficiency Act 1913 included a category or moral imbeciles, who were not intellectually idiots but displayed from an early age an alleged mental defect coupled with alleged vicious or criminal propensities, and on whom punishment has little or no deterrent effect. Cyril Burt and others pointed out that 'psychopathic personality' was used in a broader and somewhat different way in America than in the UK.

In the first decades of the 20th century, "constitutional psychopathic inferiority" had become a commonly used term in the US, implying the issue was inherent to the genetics or makeup of the person, an organic disease.[18] As a category it was used to target any and all dysfunctional or antisocial behavior, and in psychiatric categorization it labeled a broad range of alleged mental deviances, including homosexuality.[19] Some courts began to develop "psychopathic laboratories" for the classification and treatment of offenders; the term psychopathic was chosen to

avoid the social stigma of "lunacy" or "insanity", while emphasizing variance from normality rather than simply a mental hygiene issue.[20] Nevertheless, at least one such laboratory issued a report on eugenic sterilization initiatives.[21] From the 1930s, "sexual psychopath" laws (a term going back to Krafft-Ebing) started to be implemented in many US states, allowing for the indeterminate psychiatric commitment of sex offenders.[22]

From the late 1920s American psychologist George E. Partridge influentially narrowed the definition of psychopathy to antisocial personality, and from 1930 suggested that a more apt name for it would be sociopathy. He suggested that anyone, and indeed groups of people acting together, could be considered sociopathic at times, but that sociopaths - or technically 'essential sociopaths' - were chronically and pervasively so in their motivation and behavior.[] Scottish psychiatrist David Henderson published in 1939 a theory of "psychopathic states" which, although he described different types and unusually suggested that psychopaths might not all be criminals, included a violently antisocial type which ended up contributing to that being the popular meaning of the term.[23] In the 1940s a diagnosis of autistic psychopathy was introduced, later coming to wider notice and renamed Asperger syndrome to avoid the stigma of the term psychopathy.

Mid-20th century

The Mask of Sanity by American psychiatrist Hervey M. Cleckley, first published in 1941 and with revised editions for several decades, is considered a seminal work which provided a vivid series of case studies of individuals described as psychopaths. Cleckley proposed 16 characteristics of psychopathy, derived mainly from his work with male psychiatric patients in a locked institution. The title refers to the "mask" of normal functioning that Cleckley thought concealed the disorganization, amorality and disorder of the psychopathic personality. This marked the start in America of the current clinical and popularist conception of psychopathy as a particular type of antisocial, emotionless and criminal character. Cleckley would produce five editions of the book over subsequent decades, including a substantial revision in 1950, expanding his case studies and theories to more non-prisoners and non-criminals.[24]

In Nazi Germany, especially during World War II, psychiatrists and others in programmes such as Action T4 and Action 14f13 systematically deported, sterilised, interned and euthanised (killed) patients and prisoners who could be classed as mentally ill, feebleminded, psychopathic, criminally insane or just asocial. In the aftermath of the war, therefore, concepts of antisocial psychopathic personalities fell out of favour in Europe to some extent.[25] At the same time, however, in America and other countries the concept became increasingly prominent, used to categorise allied soldiers as fit or unfit for duty or on return to society, or, conversely, in the more specific sinister sense of the term, as a way to explain the actions of Nazi's.

The first version of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders in 1952 did not use the term psychopathy as a diagnosis, but "sociopathic personality disturbance".[26] Individuals to be placed in this category were said to be "...ill primarily in terms of society and of conformity with the prevailing milieu, and not only in terms of personal discomfort and relations with other individuals". There were four subtypes (called 'reactions' after Adolf Meyer): antisocial, dyssocial, sexual and addiction. The antisocial reaction was said to include "individuals who are chronically in trouble and do not seem to change as a result of experience or punishment, with no loyalties to anyone", as well as being frequently callous and lacking responsibility, with an ability to 'rationalize' their behaviour. The dyssocial reaction was for "individuals who disregard societal rules, although they are capable of strong loyalties to others or groups." Although the sociopathy category was very broad by today's definitions, the DSM-I itself pointed out that it was more specific and limited than the then current notions of 'constitutional psychopathic state' or 'psychopathic personality'.

Meanwhile other subtypes of psychopathy were sometimes proposed, notably by psychoanalyst Benjamin Karpman from the 1940s. He described psychopathy due to psychological problems (e.g. psychotic, hysterical or neurotic conditions) and idiopathic psychopathy where there was no obvious psychological cause, concluding that the former could not be attributed to a psychopathic personality and that the latter appeared so absent of any redeeming features

that it couldn't be seen as a personality issue either but must be a constitutional "anethopathy" (amorality or antipathy).[27] Various theories of distinctions between primary and secondary psychopathy remain to this day.

Cleckley's concept of psychopathy as expanded on in new editions of his book, particularly the sense of a conscience-less man beneath a mask of normality, caught the public imagination around this time. It also became increasingly influential in psychiatric circles. It later fell out of favor for some time, however, such that when he died in 1984 he was better remembered for a vivid case study of a female patient published in 1956, turned into a movie The Three Faces of Eve in 1957, which had (re)popularized in America another controversial diagnosis, Multiple Personality Disorder.[28]

A sociologist reviewing the field in 1958 wrote that "Without exception, on every point regarding psychopathic personality, psychiatrists present varying or contradictory views."[29]

Nevertheless criminologist sociologists William and Joan McCord[30] were influential in narrowing the definition of psychopathy in some quarters to mean an antisocial lack of guilt accompanied by reactive aggression.[31][32] From another direction, sociologist Lee Robins was also an influential figure in sociopathy research, stemming largely from her research-based 1966 book 'Deviant Children Grown Up: a sociological and psychiatric study of sociopathic personality', based on operational criteria provided by Eli Robins, which would shape the later diagnosis of Antisocial Personality Disorder.[33]

In the Mental Health Act in England, a new category of 'Psychopathic Personality' was added in 1959, renamed Psychopathic Disorder in 1983 (then in 2007 removed entirely). This was a legal subcategory in addition to 'mental illness' which did not equate to any one psychiatric diagnosis but covered anyone with "a persistent disorder or disability of mind which results in abnormally aggressive or seriously irresponsible conduct."[34]

On the other hand, various analysts began to identify "successful" psychopaths in society, some even suggesting it was but an adaption to the social or economic mores of the age, others noting they could be hard to spot either because they were so good at hiding their lack of conscience, or because many people showed the traits to some degree.[35]

Late 20th century

In 1968 the second edition of the DSM, in place of the antisocial subtype of socipathic personality disturbance, listed "antisocial personality" as one of ten personality disorders. This was still described in similar terms as the DSM-I's category, for individuals who are "basically unsocialized", in repeated conflicts with society, incapable of significant loyalty, selfish, irresponsible, unable to feel guilt or learn from prior experiences, and tend to blame others and rationalise. It warned that a history of legal or social offenses was not by itself enough to justify the diagnosis and that a 'group delinquent reaction' of childhood or adolescence or 'social maladjustment without manifest psychiatric disorder' should be ruled out first. The dyssocial type from the DSM-I was relegated, though would resurface as the main diagnosis in the ICD manual of the World Health Organisation.

In 1974 (and republished in 1984) clinical psychologist Bobby E. Wright wrote about 'The Psychopathic Racial Personality', in which he suggested that negative aspects of the overall behavior of white peoples towards non-white peoples could be understood by seeing the former as displaying psychopathic traits - involving predatory behavior and senseless destruction combined with ability to persuade.[36][37][38]

There remained no international clinical agreement on the diagnosis of psychopathy. A 1977 study found little relationship with the characteristics commonly attributed to psychopaths and concluded that the concept was being used too widely and loosely.[39] Robert D. Hare had published a book in 1970 summarizing research on psychopathy, and was subsequently at the forefront of psychopathy research. Frustrated by a lack of agreed definitions or rating systems for psychopathy, including at a ten-day international North Atlantic Treaty Organization (NATO) conference in 1975, Hare began developing a Psychopathy Checklist. Produced for initial circulation in 1980, it was based largely on the list of traits advanced by Cleckley and partly on the theories of other authors and on

his own experiences with clients in prisons. Meanwhile a DSM-III task force instead developed the diagnosis of antisocial personality disorder, based on 1972 Feighner Criteria for research and published in the DSM in 1980.[40] This was based on some of the criteria put forward by Cleckley but operationalized in behavioral rather than personality terms, more specifically related to conduct. APA was most concerned to demonstrate inter-rater reliability rather than necessarily validity.

Nevertheless, one author referred to the concept of psychopathy in 1987 as an "infinitely elastic, catch-all category". In 1988, psychologist Blackburn wrote in the *British Journal of Psychiatry* that as commonly used in psychiatry it is little more than a moral judgment masquerading as a clinical diagnosis, and should be scrapped. Ellard argued similarly in the same year in the *Australian and New Zealand Journal of Psychiatry*, describing the concept as 'a reflection of the customs and prejudices of a particular social group. Most psychiatrists are from that group and therefore fail to see the incongruity.' By the 1970s and 80s the sexual psychopath laws were falling out of favor in many states; the Group for the Advancement of Psychiatry called them a failure based on a confusing label mixing law and psychiatry.[41]

Hare redrafted his checklist in 1985 (Cleckley had died in 1984), renaming it the Hare Psychopathy Checklist Revised and finalising it as a first edition in 1991, updated with extra data in a 2nd edition in 2003. Hare's list differed from Cleckley's not just in rewordings and introducing quantitative scores for each point. Cleckley had required an absence of delusions and an absence of nervousness, which was central to how he defined psychopathy, whereas neither were mentioned in Hare's list. Hare also left out mention of suicidality being rarely completed and behavior with alcohol. Moreover, while Cleckley only listed "inadequately motivated antisocial behavior", Hare turned this into an array of specific antisocial behaviors covering a person's whole life, including juvenile delinquency, parasitic lifestyle, poor behavioural controls, and criminal versatility.[42] Blackburn has noted that overall Hare's checklist is closer to the criminological concept of the McCords than that of Cleckley.[43] Hare himself, while noting his promotion of Cleckley's work for four decades, would subsequently distance himself from it to some extent.

Meanwhile, following some criticism over the lack of psychological criteria in the DSM, further studies were conducted leading up the DSM-IV in 1994 and some personality criteria were included as "associated features" which were outlined in the text.[44] The World Health Organization's ICD incorporated a similar diagnosis of Dissocial Personality Disorder. Both state that psychopathy (or sociopathy) may be considered synonyms of their diagnosis.

Hare wrote two bestsellers on psychopathy, "Without Conscience" in 1993 and "Snakes in Suits: When Psychopaths Go to Work" in 2006. Cleckley had described psychopathic patients as "carr[ying] disaster lightly in each hand" and "not deeply vicious", but Hare presented a more malevolent picture; the "mask of sanity" had acquired a more sinister and Machiavellian meaning.[45]

21st century

In 2002 an academic dispute arose around claims and counterclaims of racism in the use of the concept of psychopathy. British psychologist Richard Lynn claimed that some races were inherently more psychopathic than others, while other psychologists criticized his data and interepretations.[46][47]

The Federal Bureau of Investigation's monthly outreach and communication bulletin focused on psychopathy in June 2012, featuring articles introduced and co-authored by the main contemporary proponent of the construct, Robert D. Hare.[48]

The DSM-5 published in 2013 had criteria for an overall diagnosis of Antisocial (Dissocial) Personality Disorder similar to DSM-IV, still noting that it has also been known as psychpathy or sociopathy. In an 'alternative model' suggested at the end of the manual, there is an optional specifier for "psychopathic features" - where there is a lack of anxiety/fear accompanied by a bold and efficacious interpersonal style.[49]

Overall trends

One exhaustive analysis by a Canadian psychologist describes the various lines of work as 'a psychopathy project' attempting to establish psychopathy as an object of science. Overall this was found to have suffered from 'a number of serious logical confusions and deliberate mischaracterizations of its scientific merits' - including its early basis in degeneration theory, tautological definitions and associated neuroscience findings, routinely unclarified assumptions and shifting levels of explanation about the core concept, and exaggerated statistical claims such as based on Hare's use of factor analysis. It was noted, however, that some of the limited research findings may prove useful in a better explanatory framework (i.e. not necessarily under the umbrella of 'psychopathy').[50]

A Swedish sociologist has further placed the more recent resurgence in popular coverage of psychopathy in the context of "the Enlightenment project" to use rationality and technology to deal with problems in human life and society.[51] A Scottish sociologist of biomedical ethics has suggested that the DSM's attempt to develop different standards for Antisocial Personality Disorder have been limited and modified by path dependence on the concept of psychopathy/sociopathy, due to the latter being embedded in diverse sociotechnological networks and thereby demanded by various users.

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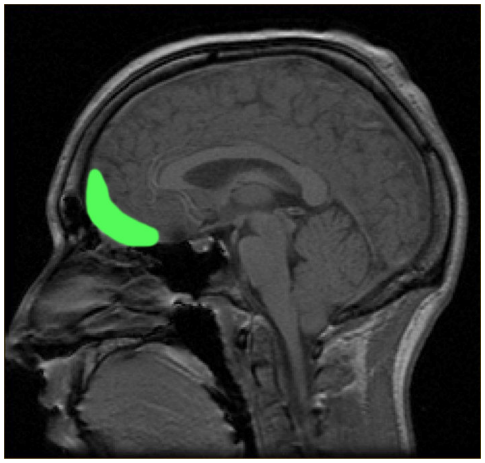
Impulsivity


Orbitofrontal cortex, part of the prefrontal cortex

Impulsivity (or **impulsiveness**) is a multifactorial construct that involves a tendency to act on a whim, displaying behavior characterized by little or no forethought, reflection, or consideration of the consequences. Impulsive actions typically are "poorly conceived, prematurely expressed, unduly risky, or inappropriate to the situation that often result in undesirable consequences," which imperil long term goals and strategies for success. A functional variety of impulsivity has also been suggested, which involves action without much forethought in appropriate situations that can and does result in desirable consequences. "When such actions have positive outcomes, they tend not to be seen as signs of impulsivity, but as indicators of boldness, quickness, spontaneity, courageousness, or unconventionality" Thus, the construct of impulsivity includes at least two independent components: (1) acting without an appropriate amount of deliberation, which may or may not be functional, and (2) choosing short-term over long-term gains.

Impulsivity is both a facet of personality, as well as a major component of various disorders including: ADHD, substance use disorders, bipolar disorder, antisocial personality disorder, and borderline personality disorder. Impulsiveness is also shown by some research to be a factor in procrastination. Abnormal patterns of impulsivity have also been noted instances of acquired brain injury and neurodegenerative diseases. Neurobiological findings suggest that there are specific brain regions involved in impulsive behavior although different brain networks may contribute to different manifestations of impulsivity, and that genetics may play a role.

Many actions contain both impulsive and compulsive features, but impulsivity and compulsivity are functionally distinct. Impulsivity and compulsivity are interrelated in that each exhibits a tendency to act prematurely or without considered thought and often include negative outcomes. However, compulsivity may be on a continuum with compulsivity on one end and impulsivity on the other, but research has been contradictory on this point. Compulsivity occurs in response to a perceived risk or threat, impulsivity occurs in response to a perceived immediate gain or benefit, and whereas compulsivity involves repetitive actions, impulsivity involves unplanned reactions.

Associated behavioral and societal problems

Attention deficit hyperactivity disorder

Attention deficit-hyperactivity disorder (ADHD) is a multiple component disorder involving inattention, impulsivity, and hyperactivity. The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) breaks ADHD into three subtypes according to the behavioral symptoms:

- Attention-Deficit/Hyperactivity Disorder Predominantly Inattentive Type
- Attention-Deficit/Hyperactivity Disorder Predominantly Hyperactive-Impulsive Type
- Attention-Deficit/Hyperactivity Disorder Combined Type

Predominantly hyperactive-impulsive type symptoms may include:

- Fidgeting and squirming in seats
- Talking nonstop
- Dashing around, touching or playing with anything and everything in sight
- Having trouble sitting still during dinner, school, and story time
- Being constantly in motion
- Having difficulty doing quiet tasks or activities

and also these manifestations primarily of impulsivity:

- Be very impatient
- Blurt out inappropriate comments, show their emotions without restraint, and act without regard for consequences
- Have difficulty waiting for things they want or waiting their turns in games
- Often interrupts conversations or others' activities

Prevalence of the disorder worldwide is estimated to be between 4% and 10%, with reports as low as 2.2% and as high as 17.8%. Variation in rate of diagnoses may be attributed to differences between populations (i.e., culture), and differences in diagnostic methodologies. Prevalence of ADHD among females is less than half that of males, and females more commonly fall into the inattentive subtype.

Despite an upward trend in diagnoses of the inattentive subtype of ADHD, impulsivity is commonly considered to be the central feature of ADHD, and the impulsive and combined subtypes are the major contributors to the societal costs associated with ADHD. The estimated cost of illness (COI) for a child with ADHD is $14,576 (in 2005 dollars) annually. Prevalence of ADHD among prison populations is significantly higher than that of the normal population. In both adults and children, ADHD has a high rate of comorbidity with other mental health disorders such as learning disability, conduct disorder, anxiety disorder, major depressive disorder, bipolar disorder, and substance use disorders.

The precise genetic and environmental factors contributing to ADHD are relatively unknown, but endophenotypes offer a potential middle ground between genes and symptoms. ADHD is commonly linked to "core" deficits involving "executive function," "delay aversion," or "activation/arousal" theories that attempt to explain ADHD through its symptomology. Endophenotypes, on the other hand, purport to identify potential behavioral markers that correlate with specific genetic etiology. There is some evidence to support deficits in response inhibition as one such marker. Problems inhibiting prepotent responses are linked with deficits in pre-frontal cortex (PFC) functioning, which is a common dysfunction associated with ADHD and other impulse-control disorders.

Evidence based psychopharmacological and behavioral interventions exist for ADHD.[*citation needed*]

Substance abuse

Impulsivity appears to be linked to all stages of substance abuse.

The acquisition phase of substance abuse involves the escalation from single use to regular use. Impulsivity may be related to the acquisition of substance abuse because of the potential role that instant gratification provided by the substance may offset the larger future benefits of abstaining from the substance, and because people with impaired inhibitory control may not able to overcome motivating environmental cues, such as peer pressure. "Similarly, individuals that discount the value of delayed reinforcers begin to abuse alcohol, marijuana, and cigarettes early in life, while also abusing a wider array of illicit drugs compared to those who discounted delayed reinforcers less."

Escalation or dysregulation is the next and more severe phase of substance abuse. In this phase individuals "lose control" of their addiction with large levels of drug consumption and binge drug use. Animal studies suggest that individuals with higher levels of impulsivity may be more prone to the escalation stage of substance abuse.

Impulsivity is also related to the abstinence, relapse, and treatment stages of substance abuse. People who scored high on the Barratt Impulsivity Scale (BIS) were more likely to stop treatment for cocaine abuse. Additionally, they adhered to treatment for a shorter duration than people that scored low on impulsivity. Also, impulsive people had greater cravings for drugs during withdrawal periods and were more likely to relapse. This effect was shown in a study where smokers that test high on the BIS had increased craving in response to smoking cues, and gave into the cravings more quickly than less impulsive smokers. Taken as a whole the current research suggests that impulsive individuals are less likely to abstain from drugs and more likely to relapse earlier than less impulsive individuals.

While it is important to note the effect of impulsivity on substance abuse, the reciprocating effect whereby substance abuse can increase impulsivity has also been researched and documented. The promoting effect of impulsivity on substance abuse and the effect of substance abuse on increased impulsivity creates a positive feedback loop that maintains substance seeking behaviors. It also makes conclusions about the direction of causality difficult. This phenomenon has been shown to be related to several substances, but not all. For example, alcohol has been shown to increase impulsivity while amphetamines have had mixed results.

Substance use disorder treatments include prescription of medications such as acamprosate, buprenorphine, disulfiram, LAAM, methadone, and naltrexone., as well as effective psychotherapeutic treatment like behavioral couples therapy, CBT, contingency management, motivational enhancement therapy, and relapse prevention.

Eating

Impulsive overeating spans from an episode of indulgence by an otherwise healthy person to chronic binges by a person with an eating disorder.[*citation needed*]

Consumption of a tempting food by non-clinical individuals increases when self-regulatory resources are previously depleted by another task, suggesting that it is caused by a breakdown in self control. Impulsive eating of unhealthy snack foods appears to be regulated by individual differences in impulsivity when self-control is weak and by attitudes towards the snack and towards healthy eating when self-control is strong. There is also evidence that greater food consumption occurs when people are in a sad mood, although it is possible that this is due more to emotional regulation than to a lack of self-control. In these cases, overeating will only take place if the food is palatable to the person, and if so individual differences in impulsivity can predict the amount of consumption.

Chronic overeating is a behavioral component of binge eating disorder, compulsive overeating, and bulimia nervosa. These diseases are more common for women and may involve eating thousands of calories at a time. Depending on which of these disorders is the underlying cause, an episode of overeating can have a variety of different motivations. Characteristics common among these three disorders include low self-esteem, depression, eating when not physically hungry, preoccupation with food, eating alone due to embarrassment, and feelings of regret or disgust after an episode. In these cases, overeating is not limited to palatable foods.

Impulsivity differentially affects disorders involving the over control of food intake (such as anorexia nervosa) and disorders involving the lack of control of food intake (such as bulimia nervosa). Cognitive impulsivity, such as risk-taking, is a component of many eating disorders, including those that are restrictive. However, only people with disorders involving episodes of overeating have elevated levels of motoric impulsivity, such as reduced response inhibition capacity.

One theory suggests that binging provides a short-term escape from feelings of sadness, anger, or boredom, although it may contribute to these negative emotions in the long-term. Another theory suggests that binge eating involves reward seeking, as evidenced by decreased serotonin binding receptors of binge-eating women compared to matched-weight controls and predictive value of heightened reward sensitivity/drive in dysfunctional eating.[1]

Treatments for clinical-grade overeating include cognitive behavioral therapy to teach people how to track and change their eating habits and actions, interpersonal psychotherapy to help people analyze the contribution of their friends and family in their disorder, and pharmacological therapies including antidepressants and SSRIs.[*citation needed*]

Impulse buying

Impulse buying consists of purchasing a product or service without any previous intent to make that purchase and has been speculated to account for as much as eighty percent of all purchases[2] in the United States.WP:TOPIC

There are several theories pertaining to impulsive buying. One theory suggests that it is exposure combining with the speed that a reward can be obtained that influences an individual to choose lesser immediate rewards over greater rewards that can be obtained later. For example a person might choose to buy a candy bar because they are in the candy isle even though they had decided earlier that they would not buy candy while in the store.

Another theory is one of self-regulation which suggests that the capacity to refrain from impulsive buying is a finite resource. As this capacity is depleted with repeated acts of restraint susceptibility to purchasing other items on impulse increases.[*citation needed*]

Finally, a third theory suggests an emotional and behavioral tie between the purchaser and the product which drives both the likelihood of an impulsive purchase as well as the degree that a person will retroactively be satisfied with that purchase result. Some studies have shown a large number of individuals are happy with purchases made on impulse (41% in one study) which is explained as a preexisting emotional attachment which has a positive relationship both with the likelihood of initiating the purchase as well as mitigating post purchase satisfaction. As an example, when purchasing team-related college paraphernalia a large percentage of those purchases are made on impulse and are tied to the degree with which a person has positive ties to that team.

Impulsive buying is seen both as an individual trait in which each person has a preconditioned or hereditary allotment, as well as a situational construct which is mitigated by such things as emotion in the moment of the purchase and the preconditioned ties an individual has with the product.

Psychotherapy and pharmacological treatments have been shown to be helpful interventions for patients with impulsive-compulsive buying disorder.

- Psychotherapy interventions include the use of desensitization techniques, self-help books or attending a support group.
- Pharmacological interventions include the use of SSRIs, such as fluvoxamine, citalopram, escitalopram, and naltrexone

Impulse control disorders not elsewhere classified

Impulse control disorder (ICDs) are a class of DSM diagnoses that do not fall into the other diagnostic categories of the manual (e.g. substance use disorders), and that are characterized by extreme difficulty controlling impulses or urges despite negative consequences. Individuals suffering from an impulse control disorder frequently experience five stages of symptoms: compelling urge or desire, failure to resist the urge, a heightened sense of arousal, succumbing to the urge (which usually yields relief from tension), and potential remorse or feelings of guilt after the behavior is completed. Specific disorders included within this category include intermittent explosive disorder, kleptomania, pathological gambling, pyromania, trichotillomania (hair pulling disorder), and impulse control disorders not otherwise specified (ICD NOS). ICD NOS includes other significant difficulties that seem to be related to impulsivity but do not meet the criteria for a specific DSM diagnosis.

There has been much debate over whether or not the ICDs deserve a diagnostic category of their own, or whether they are in fact phenomenologically and epidemiologically related to other major psychiatric conditions like obsessive-compulsive disorder (OCD), affective disorders, and addictive disorders. In fact, the ICD classification is likely to change with the release of the DSM-V in May 2013.[3] In this new revision the ICD NOS will likely be reduced or removed; proposed revisions include reclassifying trichotillomania (to be renamed hair-pulling disorder) and skin picking disorder as obsessive-compulsive and related disorders, moving Intermittent Explosive Disorder under the diagnostic heading of disruptive, impulse control, and conduct disorders, and gambling disorder may be included in addiction and related disorders.

The role of impulsivity in the ICDs varies. Research on kleptomania and pyromania are lacking, though there is some evidence that greater kleptomania severity is tied to poor executive functioning.

Trichotillomania and skin picking disorder seem to be disorders that primarily involve motor impulsivity, and will likely be classified in the DSM-V within the obsessive-compulsive and related disorders category.

Pathological gambling, in contrast, seems to involve many diverse aspects of impulsivity and abnormal reward circuitry (similar to substance use disorders) that has led to it being increasingly conceptualized as a non-substance or behavioral addiction. Evidence elucidating the role of impulsivity in pathological gambling is accumulating, with pathological gambling samples demonstrating greater response impulsivity, choice impulsivity, and reflection impulsivity than comparison control samples. Additionally, pathological gamblers tend to demonstrate greater response perseveration (compulsivity) and risky decision making in laboratory gambling tasks compared to controls, though there is no strong evidence suggesting that attention and working memory are impaired in pathological gamblers. These relations between impulsivity and pathological gambling are confirmed by brain function research: pathological gamblers demonstrate less activation in the frontal cortical regions (implicated in impulsivity) compared to controls during behavioral tasks tapping response impulsivity, compulsivity, and risk/reward. Preliminary, though variable, findings also suggest that striatal activation is different between gamblers and controls, and that neurotransmitter differences (e.g. dopamine, serotonin, opioids, glutamate, norepinephrine) may exist as well.

Individuals with Intermittent Explosive Disorder, also known as impulsive aggression, have exhibited serotonergic abnormalities and show differential activation in response to emotional stimuli and situations. Notably, Intermittent Explosive Disorder is not associated with a higher likelihood of diagnosis with any of the other ICDs but is highly comorbid with disruptive behavior disorders in childhood. Intermittent Explosive Disorder is likely be re-classified in the DSM-V as under the heading of disruptive, impulse control, and conduct disorders.

These sorts of impulse control disorders are most often treated using certain types of psychopharamcological interventions (e.g. antidepressants) and behavioral treatments like cognitive behavior therapy.[*citation needed*]

Theories of impulsivity

Ego (cognitive) depletion

According to the ego (or cognitive) depletion theory of impulsivity, self-control refers to the capacity for altering one's own responses, especially to bring them into line with standards such as ideals, values, morals, and social expectations, and to support the pursuit of long-term goals. Self-control enables a person to restrain or override one response, thereby making a different response possible. A major tenet of the theory is that engaging in acts of self-control draws from a limited "reservoir" of self-control that, when depleted, results in reduced capacity for further self-regulation. Self-control is viewed as analogous to a muscle: Just as a muscle requires strength and energy to exert force over a period of time, acts that have high self-control demands also require strength and energy to perform. Similarly, as muscles become fatigued after a period of sustained exertion and have reduced capacity to exert further force, self-control can also become depleted when demands are made of self-control resources over a period of time. Baumeister and colleagues termed the state of diminished self-control strength ego depletion (or cognitive depletion).

The strength model of self-control asserts that:

- Just as exercise can make muscles stronger, there are signs that regular exertions of self-control can improve willpower strength. These improvements typically take the form of resistance to depletion, in the sense that performance at self-control tasks deteriorates at a slower rate. Targeted efforts to control behavior in one area, such as spending money or exercise, lead to improvements in unrelated areas, such as studying or household chores. And daily exercises in self-control, such as improving posture, altering verbal behavior, and using one's nondominant hand for simple tasks, gradually produce improvements in self-control as measured by laboratory tasks. The finding that these improvements carry over into tasks vastly different from the daily exercises shows that the improvements are not due to simply increasing skill or acquiring self-efficacy from practice.
- Just as athletes begin to conserve their remaining strength when their muscles begin to tire, so do self-controllers when some of their self-regulatory resources have been expended. The severity of behavioral impairment during depletion depends in part on whether the person expects further challenges and demands. When people expect to have to exert self-control later, they will curtail current performance more severely than if no such demands are anticipated.
- Consistent with the conservation hypothesis, people can exert self-control despite ego depletion if the stakes are high enough. Offering cash incentives or other motives for good performance counteracts the effects of ego depletion. This may seem surprising but in fact it may be highly adaptive. Given the value and importance of the capacity for self-control, it would be dangerous for a person to lose that capacity completely, and so ego depletion effects may occur because people start conserving their remaining strength. When people do exert themselves on the second task, they deplete the resource even more, as reflected in severe impairments on a third task that they have not anticipated.

Empirical tests of the ego-depletion effect typically adopt dual-task paradigm. Participants assigned to an experimental ego-depletion group are required to engage in two consecutive tasks requiring self-control. Control participants are also required to engage in two consecutive tasks, but only the second task requires self-control. The strength model predicts that the performance of the experimental-group on the second self-control task will be impaired relative to that of the control group. This is because the finite self-control resources of the experimental participants will be diminished after the initial self-control task, leaving little to draw on for the second task.

The effects of ego depletion do not appear to be a product of mood or arousal. In most studies, mood and arousal has not been found to differ between participants who exerted self-control and those who did not. Likewise, mood and arousal was not related to final self-control performance. The same is true for more specific mood items, such as frustration, irritation, annoyance, boredom, or interest as well. Feedback about success and failure of the self-control efforts does not appear to affect performance. In short, the decline in self-control performance after exerting

self-control appears to be directly related to the amount of self-control exerted and cannot be easily explained by other, well-established psychological processes.

Automatic vs. controlled processes/cognitive control

Dual process theory states that mental processes operate in two separate classes: automatic and controlled. In general, automatic processes are those that are experiential in nature, occur without involving higher levels of cognition, and are based on prior experiences or informal heuristics. Controlled decisions are effortful and largely conscious processes in which an individual weighs alternatives and makes a more deliberate decision.[*citation needed*]

- *Automatic Process*: Automatic processes have four main features. They occur unintentionally or without a conscious decision, the cost of the decision is very low in mental resources, they cannot be easily stopped, and they occur without conscious thought on the part of the individual making them.
- *Controlled Process*: Controlled processes also have four main features that are very close to the opposite in spectrum from their automatic counterparts. Controlled processes occur intentionally, they require the expenditure of cognitive resources, the individual making the decision can stop the process voluntarily, and the mental process is a conscious one.

Dual process theories at one time considered any single action/thought as either being automatic or controlled. However, currently they are seen as operating more along a continuum as most impulsive actions will have both controlled and automatic attributes. Automatic processes are classified according to whether they are meant to inhibit or to facilitate a thought process. For example in one study researchers offered individuals a choice between a 1:10 chance of winning a prize and a 10 in 100 chance. Many participants chose one of the choices over the other without identifying that the chances inherent in each were the same as they saw either only 10 chances total as more beneficial, or of having 10 chances to win as more beneficial. In effect impulsive decisions can be made as prior information and experiences dictate one of the courses of action is more beneficial when in actuality careful consideration would better enable the individual to make a more informed and improved decision.[*citation needed*]

Intertemporal choice

Intertemporal choice is defined as "decisions with consequences that play out over time". This is often assessed using the relative value people assign to rewards at different points in time, either by asking experimental subjects to choose between alternatives or examining behavioral choices in a naturalistic setting.[*citation needed*]

Intertemporal choice is commonly measured in the laboratory using a "delayed discounting" paradigm, which measures the process of devaluing rewards and punishments that happen in the future. In this paradigm, subjects must choose between a smaller reward delivered soon and a larger reward delivered at a delay in the future. Choosing the smaller-sooner reward is considered impulsive. By repeatedly making these choices, indifference points can be estimated. For example, if someone chose $70 now over $100 in a week, but chose the $100 in a week over $60 now, it can be inferred that they are indifferent between $100 in a week and an intermediate value between $60 and $70. A delay discounting curve can be obtained for each participant by plotting their indifference points with different reward amounts and time delays. Individual differences in discounting curves are affected by personality characteristics such as self-reports of impulsivity and locus of control; personal characteristics such as age, gender, IQ, race, and culture; socioeconomic characteristics such as income and education; and many other variables. Lesions of the nucleus accumbens core subregion or basolateral amygdala produce shifts towards choosing the smaller-sooner reward, suggesting the involvement of these brain regions in the preference for delayed reinforcers. There is also evidence that the orbitofrontal cortex is involved in delay discounting, although there is currently debate on whether lesions in this region result in more or less impulsivity.

Economic theory suggests that optimal discounting involves the exponential discounting of value over time. This model assumes that people and institutions should discount the value of rewards and punishments at a constant rate according to how delayed they are in time. While economically rational, recent evidence suggests that this people

and animals do not discount exponentially. Many studies suggest that humans and animals discount future values according to a hyperbolic discounting curve where the discount factor decreases with the length of the delay (for example, waiting from today to tomorrow involves more loss of value than waiting from twenty days to twenty-one days). Further evidence for non-constant delay discounting is suggested by the differential involvement of various brain regions in evaluating immediate versus delayed consequences. Specifically, the prefrontal cortex is activated when choosing between rewards at a short delay or a long delay, but regions associated with the dopamine system are additionally activated when the option of an immediate reinforcer is added. Additionally, intertemporal choices differ from economic models because they involve anticipation (which may involve a neurological "reward" even if the reinforcer is delayed), self-control (and the breakdown of it when faced with temptations), and representation (how the choice is framed may influence desirability of the reinforcer), none of which are accounted for by a model that assumes economic rationality.[*citation needed*]

One facet of intertemporal choice is the possibility for preference reversal, when a tempting reward becomes more highly valued than abstaining only when immediately available. For example, when sitting home alone, a person may report that they value the health benefit of not smoking a cigarette over the effect of smoking one. However, later at night when the cigarette is immediately available, their subjective value of the cigarette may rise and they may choose to smoke it.[*citation needed*]

A theory called the "primrose path" is intended to explain how preference reversal can lead to addiction in the long run. As an example, a lifetime of sobriety may be more highly valued than a lifetime of alcoholism, but, at the same time, one drink now may be more highly valued than not drinking now. Because it is always "now," the drink is always chosen, and a paradoxical effect occurs whereby the more-valued long-term alternative is not achieved because the more-valued short-term alternative is always chosen. This is an example of complex ambivalence, when a choice is made not between two concrete alternatives but between one immediate and tangible alternative (i.e., having a drink) and one delayed and abstract alternative (i.e., sobriety).[*citation needed*]

Similarities between humans and non-human animals in intertemporal choice have been studied. Pigeons and rats also discount hyperbolically; tamarin monkeys do not wait more than eight seconds to triple the amount of a food reward. The question arises as to whether this is a difference of homology or analogy—that is, whether the same underlying process underlies human-animal similarities or whether different processes are manifesting in similar patterns of results.[*citation needed*]

Inhibitory control

Inhibitory control, often conceptualized as an executive function, is the ability to inhibit or hold back a prepotent response. It is theorized that impulsive behavior reflects a deficit in this ability to inhibit a response; impulsive people may find it more difficult to inhibit action whereas non-impulsive people may find it easier to do so. There is evidence that, in normal adults, commonly used behavioral measures of inhibitory control correlate with standard self-report measures of impulsivity.

Inhibitory control may itself be multifaceted, evidenced by numerous distinct inhibition constructs that can be measured in different ways, and relate to specific types of psychopathology. Nigg developed a useful working taxonomy of these different types of inhibition, drawing heavily from the fields of cognitive and personality psychology Nigg's eight proposed types of inhibition include the following:

Executive Inhibition

Interference control

Suppression of a stimulus that elicits an interfering response, enabling a person to complete the primary response. Interference control can also refer to suppressing distractors.

Interference control has been measured using cognitive tasks like the stroop test, flanker tasks, dual task interference, and priming tasks. Personality researchers have used the Rothbart effortful control measures and the conscientiousness scale of the Big Five as inventory measures of interference control. Based on imaging and neural research is theorized that the anterior cingulate, the dorsolateral prefrontal/premotor cortex, and the basal ganglia are related to interference control.

Cognitive inhibition

Cognitive inhibition is the suppression of unwanted or irrelevant thoughts to protect working memory and attention resources.

Cognitive inhibition is most often measured through tests of directed ignoring, self-report on one's intrusive thoughts, and negative priming tasks. As with interference control, personality psychologists have measured cognitive inhibition using the Rothbart Effortful Control scale and the Big Five Conscientiousness scale. The anterior cingulate, the prefrontal regions, and the association cortex seem to be involved in cognitive inhibition.

Behavioral inhibition

Behavioral Inhibition is the suppression of prepotent response.

Behavioral inhibition is usually measures using the Go/No Go task, Stop signal task, and reports of suppression of attentional orienting. Surveys that are theoretically relevant to behavioral inhibition include the Rothbart effortful control scale, and the Big Five Conscientiousness dimension. The rationale behind the use of behavioral measures like the Stop signal task is that "go" processes and "stop processes" are independent, and that, upon "go" and "stop" cues, they "race" against each other; if the go process wins the race, the prepotent response is executed, whereas if the stop processes wins the race, the response is withheld. In this context, impulsivity is conceptualized as a relatively slow stop process. The brain regions involved in behavioral inhibition appear to be the lateral and orbital prefrontal regions along with premotor processes.

Oculomotor Inhibition

Oculomotor Inhibition is the effortful suppression of reflexive saccade.

Oculomotor inhibition is tested using antisaccade and oculomotor tasks. Also, the Rothbart effortful control measure and the Big Five Conscientiousness dimension are thought to tap some of the effortful processes underlying the ability to suppress saccade. The Frontal eye fields/orbitofrontal cortex are involved in oculomotor inhibition.

Motivational inhibition

In response to punishment

Motivational inhibition and response in the face of punishment can be measured using tasks tapping inhibition of primary response, modified go/no go tasks, inhibition of competing response, and emotional Stroop tasks. Personality psychologists also use the Gray behavioral inhibition system measure, the Eysenck scale for neurotic introversion, and the Zuckerman Neuroticism-Anxiety scale. The Septal-hippocampal formation, cingulate, and motor systems seem to be the brain areas most involved in response to punishment.

In response to novelty

Response to novelty has been measured using the Kagan behavioral inhibition system measure and scales of neurotic introversion. The amygdaloid system is implicated in novelty response.

Automatic inhibition of attention

Recently inspected stimuli

Suppression of recently inspected stimuli for both attention and oculomotor saccade is usually measured using attentional and oculomotor inhibition of return tests. The superior colliculus and the midbrain, oculomotor pathway are involved in suppression of stimuli.

Neglected stimuli

Information at locations that are not presently being attended to is suppressed, while attending elsewhere.

This involves measures of covert attentional orienting and neglect, along with personality scales on neuroticism. The posterior association cortex and subcortical pathways are implicated in this sort of inhibition.

Assessment of impulsivity

Personality tests and reports

Barratt Impulsiveness Scale

The *Barratt Impulsiveness Scale* (BIS) is one of the oldest and most widely used measures of impulsive personality traits. The first BIS was developed in 1959 by Dr. Ernest Barratt. It has been revised extensively to achieve two major goals: (1) to identify a set of "impulsiveness" items that was orthogonal to a set of "anxiety" items as measured by the Taylor Manifest Anxiety Scale (MAS) or the Cattelll Anxiety Scale, and (2) to define impulsiveness within the structure of related personality traits like Eysenck's Extraversion dimension or Zuckerman's Sensation-Seeking dimension, especially the disinhibition subfactor. The BIS-11 with 30 items was developed in 1995. According to Patton and colleagues, there are 3 subscales (Attentional Impulsiveness, Motor Impulsiveness, and Non-Planning Impulsiveness) with six factors:

1. Attention: "focusing on a task at hand".
2. Motor impulsiveness: "acting on the spur of the moment".
3. Self-control: "planning and thinking carefully".
4. Cognitive complexity: "enjoying challenging mental tasks".
5. Perseverance: "a consistent life style".
6. Cognitive instability: "thought insertion and racing thoughts".

Eysenck Impulsiveness Scale

The Eysenck Impulsiveness Scale (EIS) is a 54-item yes/no questionnaire designed to measure impulsiveness. Three subscales are computed from this measure: Impulsiveness, Venturesomeness, and Empathy. Impulsiveness is defined as "behaving without thinking and without realizing the risk involved in the behavior". Venturesomeness is conceptualized as "being conscious of the risk of the behavior but acting anyway" The questionnaire was constructed through factor analysis to contain items that most highly loaded on impulsiveness and venturesomeness. The EIS is a widely used and well-validated measure.

Dickman Impulsivity Inventory

The *Dickman Impulsivity Inventory* was first developed in 1990 by Scott J. Dickman. This scale is based on Dickman's proposal that there are two types of impulsivity that are significantly different from one another. This includes functional impulsivity which is characterized by quick decision making when it is optimal, a trait that is often considered to be a source of pride. The scale also includes dysfunctional impulsivity which is characterized by making quick decisions when it is not optimal. This type of impulsivity is most often associated with life difficulties including substance abuse problems and other negative outcomes.[4]

This scale includes 63 items of which 23 are related to dysfunctional impulsivity, 17 are related to functional impulsivity, and 23 are filler questions that relate to neither construct. This scale has been developed into a version for use with children[5] as well as into several languages. Dickman showed there is no correlation between these two tendencies across individuals, and they also have different cognitive correlates.

UPPS Impulsive Behavior Scale

The *UPPS Impulsive Behavior Scale* is a 45-item self-report questionnaire that was designed to measure impulsivity across dimensions of the Five Factor Model of personality. The UPPS includes 4 sub-scales: lack of premediation, urgency, lack of perseverance, and sensation-seeking.

UPPS-P Impulsive Behavior Scale (UPPS-P) is a revised version of the UPPS, including 59 items. It assesses an additional personality pathway to impulsive behavior, Positive Urgency, in addition to the four pathways assessed in the original version of the scale: Urgency (now Negative Urgency), (lack of) Premeditation, (lack of) Perseverance, and Sensation Seeking

UPPS-P short version (UPPS-Ps) is 20-item scale that evaluates five different impulsivity facets (4 items per dimension).

UPPS-R Interview is a semi-structured interview that measures the degree to which individuals exhibit the various components of impulsivity assessed by the UPPS-P.

Lifetime History of Impulsive Behaviors

Lifetime History of Impulsive Behaviors (LHIB). is a 53-item questionnaire designed to assess lifetime history of impulsive behavior (as opposed to impulsive tendencies) as well as the level of distress and impairment associated with these behaviors. The assessment battery was designed to measure the following six dimensions: (a) impulsivity, (b) sensation seeking, (c) trait anxiety, (d) state depression, (e) empathy, and (f) social desirability. The LHIB consists of scales for clinically significant impulsivity, non-clinically significant impulsivity, and impulsivity related distress/impairment.

Behavioral Inhibition System/Behavioral Activation System

Behavioral Inhibition System/Behavioral Activation System (BIS/BAS) was developed based on the Gray's biopsychological theory of personality which suggests that there are two general motivational systems that underlie behavior and affect: BIS and BAS. This 20-item self-report questionnaire is designed to assess dispositional BIS and BAS sensitivities.

Impulsive/Premeditated Aggression Scale

Impulsive/Premeditated Aggression Scale (IPAS) is a 30-item self-report questionnaire. Half of the items describe impulsive aggression and half the items describe premeditated aggression. Aggressive behavior has traditionally been classified into two distinct subtypes, impulsive or premeditated. Impulsive aggression is defined as a hair-trigger aggressive response to provocation with loss of behavioral control. Premeditated aggression is defined as a planned or conscious aggressive act, not spontaneous or related to an agitated state. The IPAS is designed to characterize aggressive behavior as predominately impulsive or predominately premeditated in nature. Those

subjects who clustered on the impulsive factor showed a broad range of emotional and cognitive impairments; those who clustered on the premeditated factor showed a greater inclination for aggression and antisocial behavior.

Padua Inventory

The *Padua Inventory (PI)* consists of 60 items describing common obsessional and compulsive behavior and allows investigation of such problems in normal and clinical subjects.

Behavioral paradigms

A wide variety of behavioral tests have been devised for the assessment of impulsivity in both clinical and experimental settings. While no single test is a perfect predictor or a sufficient replacement for an actual clinical diagnosis, when used in conjunction with parent/teacher reports, behavioral surveys, and other diagnostic criteria, the utility of behavioral paradigms lies in their ability to narrow in on specific, discrete aspects of the impulsivity umbrella. Quantifying specific deficits is of use to the clinician and the experimenter, both of whom are generally concerned with obtaining objectively measurable treatment effects.[*citation needed*]

Marshmallow test

One widely recognizable test for impulsivity is the delay of gratification paradigm commonly known as the 'marshmallow test'. Developed in the 1960s to assess 'willpower' and self-control in preschoolers, the marshmallow test consists of placing a single marshmallow in front of a child and informing them that they will be left alone in the room for some duration. The child is told that if the marshmallow remains uneaten when the experimenter returns, they will be awarded a second marshmallow, both of which can then be eaten.[*citation needed*]

Despite its simplicity and ease of administration, evidence from longitudinal studies suggests that the number of seconds preschoolers wait to obtain the second marshmallow is predictive of higher SAT scores, better social and emotional coping in adolescence, higher educational achievement, and less cocaine/crack use.

Delay discounting

Like the marshmallow test, delay discounting is also a delay of gratification paradigm. It is designed around the principle that the subjective value of a reinforcer decreases, or is 'discounted,' as the delay to reinforcement increases. Subjects are given varying choices between smaller, immediate rewards and larger, delayed rewards. By manipulating reward magnitude and/or reward delay over multiple trials, 'indifference' points can be estimated whereby choosing the small, immediate reward, or the large, delayed reward are about equally likely. Subjects are labeled impulsive when their indifference points decline more steeply as a function of delay compared to the normal population (i.e., greater preference for immediate reward). Unlike the marshmallow test, delay discounting does not require verbal instruction and can be implemented on non-human animals.[6]

Go/no-go and Stop-signal reaction time tasks

Two common tests of response inhibition used in humans are the go/no-go task, and a slight variant known as the stop signal reaction time test (SSRT). During a go/no-task, the participant is trained over multiple trials to make a particular response (e.g., a key-press) when presented with a 'go' signal. On some trials, a 'stop' signal is presented just prior to, or simultaneously with the 'go' signal, and the subject must inhibit the impending response. The SSRT is similar, except that the 'stop' signal is presented after the 'go' signal. This small modification increases the difficulty of inhibiting the 'go' response, because the participant has typically already initiated the 'go' response by the time the 'stop' signal is presented.

Balloon Analogue Risk Task

The balloon analogue risk task (BART) was designed to assess risk-taking behavior. Subjects are presented with a computer depiction of a balloon that can be incrementally inflated by pressing a response key. As the balloon inflates, the subject accumulates rewards with each new key-press. The balloon is programmed with a constant probability of popping. If the balloon pops, all rewards for that balloon are lost, or the subject may choose to stop inflating and 'bank' the reward for that balloon at any time. Therefore, more key-presses equate to greater reward, but also greater probability of popping and cancelling rewards for that trial. The BART assumes that those with an affinity for 'risk-taking' are more likely pop the balloon, earning less reward overall than the typical population.[*citation needed*]

Iowa Gambling Test

The Iowa gambling task (IGT) is a test originally meant to measure decision making specifically within individuals who have ventromedial prefrontal cortex damage. The concept of impulsivity as relates to the IGT is one in which impulsive decisions are a function of an individual's lack of ability to make rational decisions over time due to an over amplification of emotional/somatic reward. In the IGT individuals are provided four decks of cards to choose from. Two of these decks provide much higher rewards but the deductions are also much higher while the second two decks have lower rewards per card but also much lower deductions. Over time anyone who chooses predominantly from the high rewards decks will lose money while those who choose from the smaller rewards decks will gain money.

The IGT uses hot and cold processes in its concept of decision making. Hot decision making involves emotional responses to the material presented based on motivation related to reward and punishment. Cold processes occur when an individual uses rational cognitive determinations when making decisions. Combined an individual should gain a positive emotional reaction when choices have beneficial consequences and will have negative emotional responses tied to choices that have greater negative consequences. In general, healthy responders to the IGT will begin to drift to the lower gain decks as they realize that they are gaining more money than they lose both through an ability to recognize that one is more consistently providing rewards as well as through the emotions related to winning consistently. However, those who have emotional deficits will fail to recognize that they are losing money over time and will continue to be more influenced by the exhilaration of higher value rewards without being influenced by the negative emotions of the loses associated with them.[*citation needed*]

For more information concerning these process refer to the **Somatic marker hypothesis**

Differential Reinforcement of Low Response Rate Task

Differential reinforcement of low response rate (DRL) described by Ferster and Skinner is used to encourage low rates of responding. It is derived from research in operant conditioning that provides an excellent opportunity to measure the hyperactive child's ability to inhibit behavioral responding. Hyperactive children were relatively unable to perform efficiently on the task, and that this deficit endured regardless of age, IQ, or experimental condition. Therefore, it can be used to discriminate accurately between teacher rated and parent rated hyperactive and nonhyperactive children. In this procedure, responses that occur before a set time interval has passed are not reinforced and reset the time required between behaviors.[*citation needed*]

In a study, a child was taken to the experimental room and told that they were going to play a game in which they had a chance to win a lot of M&M's. Every time they made the light of the reward indicator by pressing a red button, they would earn an M&M's. However, they had to wait a while (6 seconds) before they could press it to get another point. If they had pressed the button too soon, then they would have not gotten a point, and the light would not go on, and they had to wait a while before they could press it to get another point.[*citation needed*]

Researchers have also observed that subjects in a time-based situation will often engage in a sequence or chain of behaviors between reinforceable responses. This is because this collateral behavior sequence helps the subject "wait

out" the required temporal delay between responses.[*citation needed*]

Other

Other common impulsivity tasks include the Continuous performance task (CPT), 5-choice serial reaction time task (5-CSRTT), Stroop task, and Matching Familiar Figures Task.

Pharmacology and neurobiology

Neurobiological findings

Although the precise neural mechanisms underlying disorders of impulse control are not fully known, the prefrontal cortex (PFC) is the brain region most ubiquitously implicated in impulsivity. Damage to the prefrontal cortex has been associated with difficulties preparing to act, switching between response alternatives, and inhibiting inappropriate responses. Recent research has uncovered additional regions of interest, as well as highlighted particular subregions of the PFC, that can be tied to performance in specific behavioral tasks.[*citation needed*]

Delay discounting

Excitotoxic lesions in the nucleus accumbens core have been shown to increase preference for the smaller, immediate reward, whereas lesions to the nucleus accumbens shell have had no observable effect. Additionally, lesions of the basolateral amygdala, a region tied closely to the PFC, negatively affect impulsive choice similarly to what is observed in the nucleus accumbens core lesions.

Go/No-go and Stop-signal reaction time test

The orbital frontal cortex has been thought to play a role in behavioral disinhibition, and damage to the right inferior frontal gyrus, a specific subregion of the PFC, has been associated with deficits in stop-signal inhibition.

5-Choice Serial Reaction Time Task (5-CSRTT) and Differential Reinforcement of Low rates (DRL)

As with delay discounting, lesion studies have implicated the core region of the nucleus accumbens in response inhibition for both DRL and 5-CSRTT. Premature responses in the 5-CSRTT may also be modulated by other systems within the ventral striatum.[*citation needed*] In the 5-CSRTT, lesions of the anterior cingulate cortex have been shown to increase impulsive responding, and lesions to the prelimbic cortex impair attentional performance.

Iowa Gambling Task

Patients with damage to the ventromedial frontal cortex exhibit poor decision-making and persist in making risky choices in the Iowa Gambling Task.

Neurochemical and pharmacological findings

The primary pharmacological treatments for ADHD are methylphenidate (Ritalin) and amphetamine. Both methylphenidate and amphetamines block re-uptake of dopamine and norepinephrine into the pre-synaptic neuron, acting to increase post-synaptic levels of dopamine and norepinephrine. Of these two monoamines, increased availability of dopamine is considered the primary cause for the ameliorative effects of ADHD medications, whereas increased levels of norepinephrine may be efficacious only to the extent that it has downstream, indirect effects on dopamine. The effectiveness of dopamine re-uptake inhibitors in treating the symptoms of ADHD has led to the hypothesis that ADHD may arise from low tonic levels of dopamine (particularly in the fronto-limbic circuitry), but evidence in support of this theory is mixed.

Genetics

There are several difficulties when it comes to trying to identify a gene for complex traits such as impulsivity, such as genetic heterogeneity. Another difficulty is that the genes in question might sometimes show incomplete penetrance, "where a given gene variant does not always cause the phenotype". Much of the research on the genetics of impulsivity-related disorders, such as ADHD, is based on family or linkage studies. There are several genes of interest that have been studied in an attempt to find the major genetic contributors to impulsivity. Some of these genes are:

- DAT1 is the dopamine transporter gene which is responsible for the active reuptake of dopamine from the neural synapse. DAT1 polymorphisms have been shown to be linked to hyperactivity and ADHD.
- DRD4 is the dopamine D4 receptor gene and is associated with ADHD and novelty seeking behaviors. It has been proposed that novelty seeking is associated with impulsivity. Mice deficient for DRD4 have shown less behavioral responses to novelty.
- 5HT2A is the serotonin receptor gene. The serotonin 2A receptor gene has been associated with both hyper locomotion, ADHD, as well as impulsivity. Subjects with a particular polymorphism of the 5HT2A gene made more commission errors during a punishment-reward condition in a go/no-go task.
- HTR2B a serotonin receptor gene.
- CTNNA2 encodes for a brain-expressed α-catenin that has been associated with Excitement-Seeking in a genome-wide association study GWAS of 7860 individuals.

Intervention

Interventions to impact impulsivity generally

While impulsivity can take on pathological forms (e.g. substance use disorder, ADHD), there are less severe, non-clinical forms of problematic impulsivity in many people's daily lives. Research on the different facets of impulsivity can inform small interventions to change decision making and reduce impulsive behavior For example, changing cognitive representations of rewards (e.g. making long term rewards seem more concrete) and/or creating situations of "pre-commitment" (eliminating the option of changing one's mind later) can reduce the preference for immediate reward seen in delay discounting.

Brain training

Brain training interventions include laboratory-based interventions (e.g. training using tasks like go/no go) as well as community, family, and school based interventions that are ecologically valid (e.g. teaching techniques for regulating emotions or behaviors) and can be used with individuals with non-clinical levels of impulsivity. Both sorts of interventions are aimed at improving executive functioning and self-control capacities, with different interventions specifically targeting different aspects of executive functioning like inhibitory control, working memory, or attention. Emerging evidence suggests that brain training interventions may succeed in impacting executive function, including inhibitory control. Inhibitory control training specifically is accumulating evidence that it can help individuals resist temptation to consume high calorie food and drinking behavior. Some have voiced concerns that the favorable results of studies testing working memory training should be interpreted with caution, claiming that conclusions regarding changes to abilities are measured using single tasks, inconsistent use of working memory tasks, no-contact control groups, and subjective measurements of change.

Treatment of specific disorders of impulsivity

Behavioral, psychosocial, and psychopharmacological treatments for disorders involving impulsivity are common.

Psychopharmacological intervention

Psychopharmacological intervention in disorders of impulsivity has shown evidence of positive effects; common pharmacological interventions include the use of stimulant medication, selective serotonin reuptake inhibitors (SSRIs) and other antidepressants. ADHD has a well-established evidence base supporting the use of stimulant medication for the reduction of ADHD symptoms. Pathological gambling has also been studied in drug trials, and there is evidence that gambling is responsive to SSRIs and other antidepressants. Evidence based pharmacological treatment for trichotillomania is not yet available, with mixed results of studies investigating the use of SSRIs, though Cognitive Behavioral Therapy (CBT) has shown positive effects. Intermittent Explosive Disorder is most often treated with mood stabilizers, SSRIs, beta blockers, alpha agonists, and anti-psychotics (all of which have shown positive effects). There is evidence that some pharmacological interventions are efficacious in treating substance use disorders, though their use can depend on the type substance that is abused. Pharmacological treatments for SUD include the use of acamprosate, buprenorphine, disulfiram, LAAM, methadone, and naltrexone.

Behavioral interventions

Behavioral interventions also have a fairly strong evidence base in impulse control disorders. In ADHD, the behavioral interventions of behavioral parent training, behavioral classroom management, and intensive peer-focused behavioral interventions in recreational settings meet stringent guidelines qualifying them for evidence based treatment status. Empirically validated behavioral treatments for substance use disorder are fairly similar across substance use disorders, and include behavioral couples therapy, CBT, contingency management, motivational enhancement therapy, and relapse prevention. Pyromania and kleptomania are understudied (due in large part to the illegality of the behaviors), though there is some evidence that psychotherapeutic interventions (CBT, short term counseling, day treatment programs) are efficacious in treating pyromania, while kleptomania seems to be best impacted using SSRIs. Additionally, therapies including CBT, family therapy, and social skill training have shown positive effects on explosive aggressive behaviors.

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International Statistical Classification of Diseases and Related Health Problems

The ***International Statistical Classification of Diseases*** (**ICD**) is the international "standard diagnostic tool for epidemiology, health management and clinical purposes". The ICD is maintained by the World Health Organization, the directing and coordinating authority for health within the United Nations System. The ICD is designed as a health care classification system, providing a system of diagnostic codes for classifying diseases, including nuanced classifications of a wide variety of signs, symptoms, abnormal findings, complaints, social circumstances, and external causes of injury or disease. This system is designed to map health conditions to corresponding generic categories together with specific variations, assigning for these a designated code, up to six characters long. Thus, major categories are designed to include a set of similar diseases.

The International Classification of Diseases is published by the World Health Organization (WHO) and used worldwide for morbidity and mortality statistics, reimbursement systems, and automated decision support in health care. This system is designed to promote international comparability in the collection, processing, classification, and presentation of these statistics. As in the case of the analogous (but limited to mental and behavioral disorders) Diagnostic and Statistical Manual of Mental Disorders (DSM, currently in version 5), the ICD is a major project to statistically classify health disorders, and provide diagnostic assistance. The ICD is a core statistically-based classificatory diagnostic system for health care related issues of the WHO Family of International Classifications (WHO-FIC).

The ICD is revised periodically and is currently in its tenth revision. The ICD-10, as it is therefore known, was developed in 1992 to track health statistics. ICD-11 is planned for 2017.[1] As of 2007[2], development plans included using Web 2.0 principles to support detailed revision. Annual minor updates and triennial major updates are published by the WHO.[3] The ICD is part of a "family" of guides that can be used to complement each other, including also the International Classification of Functioning, Disability and Health which focuses on the domains of functioning (disability) associated with health conditions, from both medical and social perspectives.

Historical synopsis

In 1893, a French physician, Jacques Bertillon, introduced the *Bertillon Classification of Causes of Death* at a congress of the International Statistical Institute in Chicago.[4] A number of countries and cities adopted Dr. Bertillon's system, which was based on the principle of distinguishing between general diseases and those localized to a particular organ or anatomical site, as used by the City of Paris for classifying deaths. Subsequent revisions represented a synthesis of English, German and Swiss classifications, expanding from the original 44 titles to 161 titles. In 1898, the American Public Health Association (APHA) recommended that the registrars of Canada, Mexico, and the United States also adopt it. The APHA also recommended revising the system every ten-years to ensure the system remained current with medical practice advances. As a result, the first international conference to revise the International Classification of Causes of Death took place in 1900; with revisions occurring every ten-years thereafter. At that time the classification system was contained in one book, which included an Alphabetic Index as well as a Tabular List. The book was small compared with current coding texts.

The revisions that followed contained minor changes, until the sixth revision of the classification system. With the sixth revision, the classification system expanded to two volumes. The sixth revision included morbidity and mortality conditions, and its title was modified to reflect the changes: *International Statistical Classification of Diseases, Injuries and Causes of Death (ICD)*. Prior to the sixth revision, responsibility for ICD revisions fell to the Mixed Commission, a group composed of representatives from the International Statistical Institute and the Health Organization of the League of Nations. In 1948, the World Health Organization (WHO) assumed responsibility for preparing and publishing the revisions to the ICD every ten-years. WHO sponsored the seventh and eighth revisions

in 1957 and 1968, respectively. It later become clear that the established ten-year interval between revisions was too short.

The ICD is currently the most widely used statistical classification system for diseases in the world. International health statistics using this system are available at the Global Health Observatory (GHO) [5] ([6])

In addition, some countries—including Australia, Canada and the United States—have developed their own adaptations of ICD, with more procedure codes for classification of operative or diagnostic procedures.

Versions of ICD

ICD-6

The ICD-6, published in 1949, was the first to be shaped to become suitable for morbidity reporting. Accordingly the name changed from International List of Causes of Death to International Statistical Classification of Diseases. The combined code section for injuries and their associated accidents was split into two, a chapter for injuries, and a chapter for their external causes. With use for morbidity there was a need for coding mental conditions, and for the first time a section on mental disorders was added. [7]

ICD-7

The International Conference for the Seventh Revision of the International Classification of Diseases was held in Paris under the auspices of WHO in February 1955. In accordance with a recommendation of the WHO Expert Committee on Health Statistics, this revision was limited to essential changes and amendments of errors and inconsistencies.

ICD-8

The Eighth Revision Conference convened by WHO met in Geneva, from 6 to 12 July 1965. This revision was more radical than the Seventh but left unchanged the basic structure of the Classification and the general philosophy of classifying diseases, whenever possible, according to their etiology rather than a particular manifestation. During the years that the Seventh and Eighth Revisions of the ICD were in force, the use of the ICD for indexing hospital medical records increased rapidly and some countries prepared national adaptations which provided the additional detail needed for this application of the ICD. In the USA, a group of consultants was asked to study the 8th revision of ICD (ICD-8) for its applicability to various users in the United States. This group recommended that further detail be provided for coding hospital and morbidity data. The American Hospital Association's "Advisory Committee to the Central Office on ICDA" developed the needed adaptation proposals, resulting in the publication of the International Classification of Diseases, Adapted (ICDA). In 1968, the United States Public Health Service published the International Classification of Diseases, Adapted, 8th Revision for use in the United States (ICDA-8). Beginning in 1968, ICDA-8 served as the basis for coding diagnostic data for both official morbidity [and mortality] statistics in the United States.[8]

ICD-9

The International Conference for the Ninth Revision of the International Classification of Diseases, convened by WHO, met in Geneva from 30 September to 6 October 1975. In the discussions leading up to the conference, it had originally been intended that there should be little change other than updating of the classification. This was mainly because of the expense of adapting data processing systems each time the classification was revised.

There had been an enormous growth of interest in the ICD and ways had to be found of responding to this, partly by modifying the classification itself and partly by introducing special coding provisions. A number of representations was made by specialist bodies which had become interested in using the ICD for their own statistics. Some subject areas in the classification were regarded as inappropriately arranged and there was considerable pressure for more detail and for adaptation of the classification to make it more relevant for the evaluation of medical care, by classifying conditions to the chapters concerned with the part of the body affected rather than to those dealing with the underlying generalized disease.

At the other end of the scale, there were representations from countries and areas where a detailed and sophisticated classification was irrelevant, but which nevertheless needed a classification based on the ICD in order to assess their progress in health care and in the control of disease. A field test with a bi-axial classification approach - one axis for anatomy, another for etiology - showed the impracticability of such approach for routine use.

The final proposals presented to and accepted by the Conference retained the basic structure of the ICD, although with much additional detail at the level of the four digit subcategories, and some optional five digit subdivisions. For the benefit of users not requiring such detail, care was taken to ensure that the categories at the three digit level were appropriate.

For the benefit of users wishing to produce statistics and indexes oriented towards medical care, the Ninth Revision included an optional alternative method of classifying diagnostic statements, including information about both an underlying general disease and a manifestation in a particular organ or site. This system became known as the dagger and asterisk system and is retained in the Tenth Revision. A number of other technical innovations were included in the Ninth Revision, aimed at increasing its flexibility for use in a variety of situations. It was eventually replaced by ICD-10, the version currently in use by the WHO and most countries. Given the widespread expansion in the tenth revision, it is not possible to convert ICD-9 data sets directly into ICD-10 data sets, although some tools are available to help guide users.[9] Publication of ICD-9 without IP restrictions in a world with evolving electronic data systems led to a range of products based on ICD-9, such as MeDRA or the Read directory.

ICPM

When ICD-9 was published by the World Health Organization (WHO), the International Classification of Procedures in Medicine (ICPM) was also developed (1975) and published (1978). The ICPM surgical procedures fascicle was originally created by the United States, based on its adaptations of ICD (called ICDA), which had contained a procedure classification since 1962. ICPM is published separately from the ICD disease classification as a series of supplementary documents called fascicles (bundles or groups of items). Each fascicle contains a classification of modes of laboratory, radiology, surgery, therapy, and other diagnostic procedures. Many countries have adapted and translated the ICPM in parts or as a whole and are using it with amendments since then.

ICD-9-CM

International Classification of Diseases, Clinical Modification (ICD-9-CM) is an adaption created by the U.S. National Center for Health Statistics (NCHS) and used in assigning diagnostic and procedure codes associated with inpatient, outpatient, and physician office utilization in the United States. The ICD-9-CM is based on the ICD-9 but provides for additional morbidity detail. It is updated annually on October 1.[10][11]

It consists of two or three volumes:

- Volumes 1 and 2 contain diagnosis codes. (Volume 1 is a tabular listing, and volume 2 is an index.) Extended for ICD-9-CM
- Volume 3 contains procedure codes. ICD-9-CM only

The NCHS and the Centers for Medicare and Medicaid Services are the U.S. governmental agencies responsible for overseeing all changes and modifications to the ICD-9-CM.

ICD-10

Work on ICD-10 began in 1983, and the new revision was endorsed by the Forty-third World Health Assembly in May 1990. The latest version came into use in WHO Member States starting in 1994.[12] The classification system allows more than 155,000 different codes and permits tracking of many new diagnoses and procedures, a significant expansion on the 17,000 codes available in ICD-9. Adoption was relatively swift in most of the world. Several materials are made available online by WHO to facilitate its use, including a manual, training guidelines, a browser, and files for download. Some countries have adapted the international standard, such as the "ICD-10-AM" published in Australia in 1998 (also used in New Zealand),[13] and the "ICD-10-CA" introduced in Canada in 2000.[14]

ICD-10-CM

Adoption of ICD-10-CM has been slow in the United States. Since 1979, the USA had required ICD-9-CM codes[15] for Medicare and Medicaid claims, and most of the rest of the American medical industry followed suit. On 1 January 1999 the ICD-10 (without clinical extensions) was adopted for reporting mortality, but ICD-9-CM was still used for morbidity. Meanwhile, NCHS received permission from the WHO to create a clinical modification of the ICD-10, and has production of all these systems:

- ICD-10-CM, for diagnosis codes, is intended to replace volumes 1 and 2. Annual updates are provided.
- ICD-10-PCS, for procedure codes, is intended to replace volume 3. Annual updates are provided.

On August 21, 2008, the US Department of Health and Human Services (HHS) proposed new code sets to be used for reporting diagnoses and procedures on health care transactions. Under the proposal, the ICD-9-CM code sets would be replaced with the ICD-10-CM code sets, effective October 1, 2013. On April 17, 2012 the Department of Health and Human Services (HHS) published a proposed rule that would delay, from October 1, 2013 to October 1, 2014,the compliance date for the ICD-10-CM and PCS.

ICD-10-CA

ICD-10-CA is a clinical modification of ICD-10 developed by the Canadian Institute for Health Information for morbidity classification in Canada. ICD-10-CA applies beyond acute hospital care, and includes conditions and situations that are not diseases but represent risk factors to health, such as occupational and environmental factors, lifestyle and psycho-social circumstances.

ICD-11

The World Health Organization is currently revising the International Classification of Diseases (ICD) towards the ICD-11. The development is taking place on an internet-based workspace, called iCAT (Collaborative Authoring Tool) Platform, somewhat similar to a wiki – yet it requires more structure and peer review process. The WHO collaborates through this platform with all interested parties.

The final draft of the ICD-11 system is expected to be submitted to WHO's World Health Assembly (WHA) for official endorsement by 2017.[16] The beta draft[17] was made available online in May 2012 for initial consultation and commenting.[18]

In ICD-11 each disease entity will have definitions that give key descriptions and guidance on what the meaning of the entity/category is in human readable terms - to guide users. This is an advancement over ICD-10, which had only title headings. The Definitions have a standard structure according to a template with standard definition templates and further features exemplified in a “Content Model”. The Content Model is a structured framework that captures the knowledge that underpins the definition of an ICD entity. The Content Model therefore allows computerization (with links to ontologies and SNOMED CT). Each ICD entity can be seen from different dimensions or “parameters”. For example, there are currently 13 defined main parameters in the Content Model (see below) to describe a category in ICD.

1. ICD Entity Title - *Fully Specified Name*
2. Classification Properties - *disease, disorder, injury, etc*.
3. Textual Definitions - *short standard description*
4. Terms - *synonyms, other inclusion and exclusions*
5. Body System/Structure Description - *anatomy and physiology*
6. Temporal Properties - *acute, chronic or other*
7. Severity of Subtypes Properties - *mild, moderate, severe, or other scales*
8. Manifestation Properties - *signs, symptoms*
9. Causal Properties - *etiology: infectious, external cause, etc.*
10. Functioning Properties - *impact on daily life: activities and participation*
11. Specific Condition Properties - *relates to pregnancy etc.*
12. Treatment Properties - *specific treatment considerations: e.g. resistance*
13. Diagnostic Criteria - *operational definitions for assessment*

ICD exists in 41 Languages in electronic versions and its expression in multiple languages will be systematically pursued in ICD11.

Usage and current topics

History and usage in the United States

In the United States, the U.S. Public Health Service published *The International Classification of Diseases, Adapted for Indexing of Hospital Records and Operation Classification (ICDA),* completed in 1962 and expanding the ICD-7 in a number of areas to more completely meet the indexing needs of hospitals. The U.S. Public Health Service later published the *Eighth Revision, International Classification of Diseases, Adapted for Use in the United States,* commonly referred to as ICDA-8, for official national morbidity and mortality statistics. This was followed by the *ICD, 9th Revision, Clinical Modification*, known as ICD-9-CM, published by the U.S. Department of Health and Human Services and used by hospitals and other healthcare facilities to better describe the clinical picture of the patient. The diagnosis component of ICD-9-CM is completely consistent with ICD-9 codes, and remains the data standard for reporting morbidity. National adaptations of the ICD-10 progressed to incorporate both clinical code (ICD-10-CM) and procedure code (ICD-10-PCS) with the revisions completed in 2003. In 2009, the U.S. Centers for Medicare and Medicaid Services announced that it would begin using ICD-10 on April 1, 2010, with full compliance by all involved parties by 2013.

The years for which causes of death in the United States have been classified by each revision as follows:

- ICD-1 – 1900
- ICD-2 – 1910
- ICD-3 – 1921
- ICD-4 – 1930
- ICD-5 – 1939
- ICD-6 – 1949
- ICD-7 – 1958
- ICD-8A – 1968
- ICD-9 – 1979
- ICD-10 – 1999

Mental and behavioral disorders

The ICD includes a section classifying mental and behavioral disorders (Chapter V). This has developed alongside the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM) and the two manuals seek to use the same codes. There are significant differences, however, such as the ICD including personality disorders in the same way as other mental disorders, while the DSM-IV-TR lists them on a separate 'axis'. The WHO is revising their classifications in these sections as part the development of the ICD-11 (scheduled for 2015), and an "International Advisory Group" has been established to guide this.[19] An international survey of psychiatrists in 66 countries comparing use of the ICD-10 and DSM-IV found that the former was more often used for clinical diagnosis while the latter was more valued for research. The ICD is actually the official system for the US, although many mental health professionals do not realize this due to the dominance of the DSM. The US is due to adopt a modified version of the ICD-10 in 2013. Psychologists state, "Serious problems with the clinical utility of both the ICD and the DSM are widely acknowledged."

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[16] http://www.who.int/classifications/icd/revision/timeline/en/index.html

[17] http://apps.who.int/classifications/icd11/browse/f/en#

[18] World Health Organization. "The International Classification of Diseases 11th Revision is due by 2015." (http://www.who.int/classifications/icd/revision/en/) Accessed 21 June 2012.

[19] http://www.who.int/mental_health/evidence/en/

External links

- Official website (http://www.who.int/classifications/icd/en/) at World Health Organization (WHO)
- ICD-10 online browser (http://apps.who.int/classifications/icd10) (WHO)
- ICD-10 online training direct access (http://apps.who.int/classifications/apps/icd/ICD10Training) (WHO)
- ICD-10 online training support (http://sites.google.com/site/icd10onlinetraining) (WHO)
- ICD-10-CM (http://www.cdc.gov/nchs/icd/icd10cm.htm) (USA – modification) at Centers for Disease Control and Prevention
- ICD-11 Revision (http://sites.google.com/site/icd11revision/home) (WHO)
- Code Browser for ICD-9-CM, ICD-10-CM, ICD-10-PCS, HCPCS, DRGs (http://www.findacode.com/tools/browse-a-code.php)
- ICD-9-CM to ICD-10-CM code conversions (http://www.ecodingnow.com/OnlineCodes/OnlineCodes/i9toi10.html)
- ICD-9-CM and DRG on-line coding engine (http://www.icd9coding.com)
- Free ICD-9-CM Code search (https://drchrono.com/public_billing_code_search/)
- ICD-10 and ICD-10 PCS (http://www.med-code.info/?country=us&locale=us)
- ICD-9 and ICD-10 code lookup (http://icdx.org)
- Free ICD-9 and ICD-10 online browser in english and spanish (http://www.itserver.es)

Narcissism

Narcissism is a term that originated with Narcissus in Greek mythology who fell in love with his own image reflected in a pool of water. Currently it is used to describe the pursuit of gratification from vanity, or egotistic admiration of one's own physical or mental attributes, that derive from arrogant pride. Narcissism has included particular meanings in specific fields:

- A concept in psychoanalytic theory, introduced in Sigmund Freud's *On Narcissism*
- An Axis II disorder, Narcissistic personality disorder, in DSM-IV
- A social or cultural problem
- A factor in trait theory used in some self-report inventories of personality such as the Millon Clinical Multiaxial Inventory

Except in the sense of primary narcissism or healthy self-love, narcissism is usually considered a problem in a person or group's relationships with self and others. Narcissism is not the same as egocentrism.

Narcissus (1590s) by Caravaggio (Galleria Nazionale d'Arte Antica, Rome)

History

The term *narcissism* comes from the Greek myth of Narcissus, a handsome Greek youth who rejected the desperate advances of the nymph Echo. These advances eventually led Narcissus to fall in love with his own reflection in a pool of water. Unable to consummate his love, Narcissus "lay gazing enraptured into the pool, hour after hour," and finally changed into a flower that bears his name, the narcissus.

The concept of excessive selfishness has been recognized throughout history. In ancient Greece the concept was understood as hubris. It is only in recent times that it has been defined in psychological terms.

- In 1752 Jean-Jacques Rousseau's play *Narcissus: or the Self-Admirer* was performed in Paris.
- In 1898 Havelock Ellis, an English sexologist, used the term "narcissus-like" in reference to excessive masturbation, whereby the person becomes his or her own sex object.[1]
- In 1899, Paul Näcke was the first person to use the term "narcissism" in a study of sexual perversions.
- Otto Rank in 1911 published the first psychoanalytical paper specifically concerned with narcissism, linking it to vanity and self-admiration.
- Sigmund Freud published a paper exclusively devoted to narcissism in 1914 called On Narcissism: An Introduction.[2]
- In 1923, Martin Buber published an essay "Ich und Du" (I and You), in which he pointed out that our narcissism often leads us to relate to others as objects instead of as equals.
- Since 2000, on psychological tests designed to detect narcissism, the scores of residents of the United States have continually increased. Psychologists have suggested a link to social networking.[3]

Traits and signs

> Life is a stage, and when the curtain falls upon an act, it is finished and forgotten. The emptiness of such a life is beyond imagination.
>
> —Alexander Lowen, describing the existence of a narcissist

Four dimensions of narcissism as a personality variable have been delineated: leadership/authority, superiority/arrogance, self-absorption/self-admiration, and exploitativeness/entitlement.[4]

A 2012 popular book on power-hungry narcissists suggests that narcissists typically display most, and sometimes all, of the following traits:[5]

- An obvious self-focus in interpersonal exchanges
- Problems in sustaining satisfying relationships
- A lack of psychological awareness (see insight in psychology and psychiatry, egosyntonic)
- Difficulty with empathy
- Problems distinguishing the self from others (see narcissism and boundaries)
- Hypersensitivity to any insults or imagined insults (see criticism and narcissists, narcissistic rage and narcissistic injury)
- Vulnerability to shame rather than guilt
- Haughty body language
- Flattery towards people who admire and affirm them (narcissistic supply)
- Detesting those who do not admire them (narcissistic abuse)
- Using other people without considering the cost of doing so
- Pretending to be more important than they really are
- Bragging (subtly but persistently) and exaggerating their achievements
- Claiming to be an "expert" at many things
- Inability to view the world from the perspective of other people
- Denial of remorse and gratitude

Hotchkiss' seven deadly sins of narcissism

Hotchkiss identified what she called the seven deadly sins of narcissism:[6]

1. **Shamelessness**: Shame is the feeling that lurks beneath all unhealthy narcissism, and the inability to process shame in healthy ways.
2. **Magical thinking**: Narcissists see themselves as perfect, using distortion and illusion known as magical thinking. They also use projection to dump shame onto others.
3. **Arrogance**: A narcissist who is feeling deflated may reinflate by diminishing, debasing, or degrading somebody else.
4. **Envy**: A narcissist may secure a sense of superiority in the face of another person's ability by using contempt to minimize the other person.
5. **Entitlement**: Narcissists hold unreasonable expectations of particularly favorable treatment and automatic compliance because they consider themselves special. Failure to comply is considered an attack on their superiority, and the perpetrator is considered an "awkward" or "difficult" person. Defiance of their will is a narcissistic injury that can trigger narcissistic rage.
6. **Exploitation**: Can take many forms but always involves the exploitation of others without regard for their feelings or interests. Often the other is in a subservient position where resistance would be difficult or even impossible. Sometimes the subservience is not so much real as assumed.
7. **Bad boundaries**: Narcissists do not recognize that they have boundaries and that others are separate and are not extensions of themselves. Others either exist to meet their needs or may as well not exist at all. Those who provide narcissistic supply to the narcissist are treated as if they are part of the narcissist and are expected to live up to those expectations. In the mind of a narcissist there is no boundary between self and other.

Clinical and research aspects

Narcissistic personality disorder

Narcissistic personality disorder affects 1% of the population.

Although most individuals have some narcissistic traits, high levels of narcissism can manifest themselves in a pathological form as narcissistic personality disorder (NPD), whereby the patient overestimates his or her abilities and has an excessive need for admiration and affirmation. A revision of NPD took place in the DSM V. In this revision, NPD saw dramatic changes to its definition. The general move towards a dimensional (personality trait-based) view of the Personality Disorders has been maintained.

-Some may have a limited or minimal capability of experiencing emotions.[7]

Healthy narcissism

Healthy narcissism is a structural truthfulness of the self, achievement of self and object constancy, synchronization between the self and the superego and a balance between libidinal and aggressive drives (the ability to receive gratification from others and the drive for impulse expression). Healthy narcissism forms a constant, realistic self-interest and mature goals and principles and an ability to form deep object relations.[8] A feature related to healthy narcissism is the feeling of greatness. This is the antithesis of insecurity or inadequacy.

A required element within normal development

Healthy narcissism might exist in all individuals. Freud says that this is an original state from which the individual develops the love object. He argues that healthy narcissism is an essential part of normal development. According to Freud the love of the parents for their child and their attitude toward their child could be seen as a revival and reproduction of their own narcissism. The child has an omnipotence of thought; the parents stimulate that feeling because in their child they see the things that they have never reached themselves. Compared to neutral observations, the parents tend to overvalue the qualities of their child. When parents act in an extreme opposite style and the child is rejected or inconsistently reinforced depending on the mood of the parent, the self-needs of the child are not met.[*citation needed*]

Karen Horney saw the narcissistic personality as the product of a certain kind of early environment molding a certain kind of temperament. She did not see narcissistic needs and tendencies as inherent in human nature.[9]

In relation to the pathological condition

Healthy narcissism has to do with a strong feeling of "own love" protecting the human being against illness. Eventually, however, the individual must love the other, "the object love to not become ill." The individual becomes ill as a result of the frustration created when he is unable to love the object.[10] In pathological narcissism such as the narcissistic personality disorder, the person's libido has been withdrawn from objects in the world and produces megalomania. The clinical theorists Kernberg, Kohut and Millon all see pathological narcissism as a possible outcome in response to unempathic and inconsistent early childhood interactions. They suggested that narcissists try to compensate in adult relationships. The pathological condition of narcissism is, as Freud suggested, a magnified, extreme manifestation of healthy narcissism.

With regard to the condition of healthy narcissism, it is suggested that this is correlated with good psychological health. Self-esteem works as a mediator between narcissism and psychological health. Therefore, because of their elevated self-esteem, deriving from self-perceptions of competence and likability, high narcissists are relatively free of worry and gloom. Other researchers suggested that healthy narcissism cannot be seen as 'good' or 'bad'; however, it depends on the contexts and outcomes being measured. In certain social contexts such as initiating social relationships, and with certain outcome variables, such as feeling good about oneself, healthy narcissism can be helpful. In other contexts, such as maintaining long-term relationships and with other outcome variables, such as accurate self-knowledge, healthy narcissism can be unhelpful.[11]

Commonly used measures

Narcissistic Personality Inventory

The Narcissistic Personality Inventory (NPI) is the most widely used measure of narcissism in social psychological research. Although several versions of the NPI have been proposed in the literature, a forty-item forced-choice version (Raskin & Terry, 1988) is the one most commonly employed in current research. The NPI is based on the DSM-III clinical criteria for narcissistic personality disorder (NPD), although it was designed to measure these features in the general population. Thus, the NPI is often said to measure "normal" or "subclinical" (borderline) narcissism (i.e., in people who score very high on the NPI do not necessarily meet criteria for diagnosis with NPD).

The Millon Clinical Multiaxial Inventory

The Millon Clinical Multiaxial Inventory (MCMI) is a widely used diagnostic test developed by Theodore Millon. The MCMI includes a scale for Narcissism. Auerbach compared the NPI and MCMI and found them well correlated, $r(146) = .55$, $p<.001$. It should be noted that whereas the MCMI measures narcissistic personality disorder (NPD), the NPI measures narcissism as it occurs in the general population. In other words, the NPI measures "normal" narcissism; i.e., most people who score very high on the NPI do not have NPD. Indeed, the NPI does not capture any sort of narcissism taxon as would be expected if it measured NPD.[12]

Empirical studies

Within psychology, there are two main branches of research into narcissism, clinical and social psychology. These approaches differ in their view of narcissism with the former treating it as a disorder, thus as discrete, and the latter treating it as a personality trait, thus as a continuum. These two strands of research tend loosely to stand in a divergent relation to one another, although they converge in places.

Campbell and Foster (2007)[13] review the literature on narcissism. They argue that narcissists possess the following "basic ingredients":

- **Positive**: Narcissists think they are better than others.
- **Inflated**: Narcissists' views tend to be contrary to reality. In measures that compare self-report to objective measures, narcissists' self-views tend to be greatly exaggerated.
- **Agentic**: Narcissists' views tend to be most exaggerated in the agentic domain, relative to the communion domain.Wikipedia:Please clarify
- **Special**: Narcissists perceive themselves to be unique and special people.
- **Selfish**: Research upon narcissists' behaviour in resource dilemmas supports the case for narcissists as being selfish.[14]
- **Oriented toward success**: Narcissists are oriented towards success by being, for example, approach oriented.Wikipedia:Please clarify[15]

Narcissists tend to demonstrate a lack of interest in warm and caring interpersonal relationships. [Campbell and Forster (2007)]. There are several ongoing controversies within narcissism literature, namely whether narcissism is healthy or unhealthy, a personality disorder, a discrete or continuous variable, defensive or offensive, the same across genders, the same across cultures, and changeable or unchangeable.

Campbell and Foster (2007) argue that self-regulatory strategies are of paramount importance to understanding narcissism. Self-regulation in narcissists involves such things as striving to make one's self look and feel positive, special, successful and important. It comes in both intra-psychic, such as blaming a situation rather than self for failure, and interpersonal forms, such as using a relationship to serve one's own self. Some differences in self-regulation between narcissists and non-narcissists can be seen with Campbell, Reeder, Sedikides & Elliot (2000) who conducted a study with two experiments. In each experiment, participants took part in an achievement task, following which they were provided with false feedback; it was either bogus success or failure. The study found that both narcissists and non-narcissists self-enhanced, but non-narcissists showed more flexibility in doing so. Participants were measured on both a comparative and a non-comparative self-enhancement strategy. Both narcissists and non-narcissists employed the non-comparative strategy similarly; however, narcissists were found to be more self-serving with the comparative strategy, employing it far more than non-narcissists, suggesting a greater rigidity in their self-enhancement. When narcissists receive negative feedback that threatens the self, they self-enhance at all costs, but non-narcissists tend to have limits.

Heritability of narcissism utilizing twin studies

Livesley et al. concluded, in agreement with other studies, that narcissism as measured by a standardized test was a common inherited trait. Additionally, in similar agreement with those other studies, it was found that there exists a continuum between normal and disordered personality.

The study subjects were 175 volunteer twin pairs (ninety identical, eighty-five fraternal) drawn from the general population. Each twin completed a questionnaire that assessed eighteen dimensions of personality disorder. The authors estimated the heritability of each dimension of personality by standard methods, thus providing estimates of the relative contributions of genetic and environmental causation.

Of the eighteen personality dimensions, narcissism was found to have the highest heritability (0.64), indicating that the concordance of this trait in the identical twins was significantly influenced by genetics. Of the other dimensions

of personality, only four were found to have heritability coefficients of greater than 0.5: callousness, identity problems, oppositionality and social avoidance.

Stigmatising attitude of narcissists to psychiatric illness

Arikan found that a stigmatising attitude to psychiatric patients is associated with narcissistic personality traits.

Narcissism in evolutionary psychology

The concept of narcissism is used in evolutionary psychology in relation to the mechanisms of assortative mating, or the non-random choice of a partner for purposes of procreation.

Evidence for assortative mating among humans is well established; humans mate assortatively regarding age, IQ, height, weight, nationality, educational and occupational level, physical and personality characteristics, and family relatedness.[16] In the "self seeking like" hypothesis, individuals unconsciously look for a mirror image of themselves in others, seeking criteria of beauty or reproductive fitness in the context of self-reference.

Alvarez et al. found that facial resemblance between couples was a strong driving force among the mechanisms of assortative mating: human couples resemble each other significantly more than would be expected from random pair formation. Since facial characteristics are known to be inherited, the "self seeking like" mechanism may enhance reproduction between genetically similar mates, favoring the stabilization of genes supporting social behavior, with no kin relationship among them.

Types

Masterson's subtypes (exhibitionist and closet)

In 1993, James F. Masterson proposed two categories for pathological narcissism, **exhibitionist** and **closet**.[17] Both fail to adequately develop an age- and phase- appropriate self because of defects in the quality of psychological nurturing provided, usually by the mother. The exhibitionist narcissist is the one described in DSM-IV and differs from the closet narcissist in several important ways.

The closet narcissist is more likely to be described as having a deflated, inadequate self-perception and greater awareness of emptiness within. The exhibitionist narcissist would be described as having an inflated, grandiose self-perception with little or no conscious awareness of the emptiness within. Such a person would assume that this condition was normal and that others were just like them.

The closet narcissist seeks constant approval from others and appears similar to the borderline in the need to please others. The exhibitionist narcissist seeks perfect admiration all the time from others.

Millon's variations

Theodore Millon identified five variations of narcissist. Any individual narcissist may exhibit none or one of the following:

- **unprincipled narcissist**: including antisocial features. A charlatan—is a fraudulent, exploitative, deceptive and unscrupulous individual.
- **amorous narcissist**: including histrionic features. The Don Juan or Casanova of our times—is erotic, exhibitionist.
- **compensatory narcissist**: including negativistic (passive-aggressive), avoidant features.
- **elitist narcissist**: variant of pure pattern. Corresponds to Wilhelm Reich's "phallic narcissistic" personality type.
- **fanatic type**: including paranoid features. An individual whose self-esteem was severely arrested during childhood, usually with major paranoid tendencies, who holds onto an illusion of omnipotence. These people are fighting delusions of insignificance and lost value and are trying to re-establish their self-esteem through

grandiose fantasies and self-reinforcement. If unable to gain recognition or support from others, they take on the role of a heroic or worshipped person with a grandiose mission.

Other forms of narcissism

Acquired situational narcissism

Acquired situational narcissism (ASN) is a form of narcissism that develops in late adolescence or adulthood, brought on by wealth, fame and the other trappings of celebrity. It was coined by Robert B. Millman, professor of psychiatry at the Weill Cornell Medical College of Cornell University.

ASN differs from conventional narcissism in that it develops after childhood and is triggered and supported by the celebrity-obsessed society. Fans, assistants and tabloid media all play into the idea that the person really is vastly more important than other people, triggering a narcissistic problem that might have been only a tendency, or latent, and helping it to become a full-blown personality disorder. "Millman says that what happens to celebrities is that they get so used to people looking at them that they stop looking back at other people."[18]

In its presentation and symptoms, it is indistinguishable from narcissistic personality disorder, differing only in its late onset and its support by large numbers of others. "The lack of social norms, controls, and of people telling them how life really is, also makes these people believe they're invulnerable,"[19] so that the person with ASN may suffer from unstable relationships, substance abuse and erratic behaviour.

A famous fictional character with ASN is Norma Desmond, the main character of *Sunset Boulevard*.

Aggressive narcissism

This is Factor 1 in the Hare Psychopathy Checklist,[*citation needed*] which includes the following traits:

- Glibness/superficial charm
- Grandiose sense of self-worth
- Pathological lying
- Cunning/manipulative
- Lack of remorse or guilt
- Callous/lack of empathy
- Failure to accept responsibility for own actions

Codependency (inverted narcissism or co-narcissism)

Codependency is a tendency to behave in overly passive or excessively caretaking ways that negatively impact one's relationships and quality of life. Narcissists are considered to be natural magnets for the codependent. Rappoport identifies codependents of narcissists as "co-narcissists".[20]

Collective or group narcissism

Collective narcissism (or group narcissism) is a type of narcissism where an individual has an inflated self-love of his or her own ingroup, where an “ingroup” is a group in which an individual is personally involved.[21] While the classic definition of narcissism focuses on the individual, collective narcissism asserts that one can have a similar excessively high opinion of a group, and that a group can function as a narcissistic entity. Collective narcissism is related to ethnocentrism; however, ethnocentrism primarily focuses on self-centeredness at an ethnic or cultural level, while collective narcissism is extended to any type of ingroup beyond just cultures and ethnicities.[22]

Conversational narcissism

Conversational narcissism is a term used by sociologist Charles Derber in his book, *The Pursuit of Attention: Power and Ego in Everyday Life*.

Derber observed that the social support system in America is relatively weak, and this leads people to compete mightily for attention. In social situations, they tend to steer the conversation away from others and toward themselves. "Conversational narcissism is the key manifestation of the dominant attention-getting psychology in America," he wrote. "It occurs in informal conversations among friends, family and coworkers. The profusion of popular literature about listening and the etiquette of managing those who talk constantly about themselves suggests its pervasiveness in everyday life."

What Derber describes as "conversational narcissism" often occurs subtly rather than overtly because it is prudent to avoid being judged an egotist.

Derber distinguishes the "shift-response" from the "support-response".Wikipedia:Please clarify

Corporate narcissism

Organizational psychologist Alan Downs wrote a book in 1997 describing corporate narcissism.[23] He explores high-profile corporate leaders (such as Al Dunlap and Robert Allen) who, he suggests, literally have only one thing on their minds: profits. According to Downs, such narrow focus actually may yield positive short-term benefits, but ultimately it drags down individual employees as well as entire companies. Alternative thinking is proposed, and some firms now utilizing these options are examined. Downs' theories are relevant to those suggested by Victor Hill in his book, *Corporate Narcissism in Accounting Firms Australia*.[24]

Cross-cultural narcissism

Joan Lachkar describes the phenomenon of cross-cultural narcissism thus:[25]

> The cross-cultural narcissist brings to his new country a certain amount of nationalistic pride, which he holds onto relentlessly. He refuses to adapt and will go to great lengths to maintain his sense of special identity. Cross-cultural narcissists often hook up with borderline women, who tend to idealize and be mesmerized by men from another culture.

Cultural narcissism

In *The Culture of Narcissism*, Christopher Lasch defines a narcissistic culture as one where every activity and relationship is defined by the hedonistic need to acquire the symbols of wealth,[26] this becoming the only expression of rigid, yet covert, social hierarchies. It is a culture where liberalism only exists insofar as it serves a consumer society, and even art, sex and religion lose their liberating power.

In such a society of constant competition, there can be no allies, and little transparency. The threats to acquisitions of social symbols are so numerous, varied and frequently incomprehensible, that defensiveness, as well as competitiveness, becomes a way of life. Any real sense of community is undermined—or even destroyed—to be replaced by virtual equivalents that strive, unsuccessfully, to synthesize a sense of community.

Destructive narcissism

Destructive narcissism is the constant exhibition of numerous and intense characteristics usually associated with the pathological narcissist but having fewer characteristics than pathological narcissism.[27]

Malignant narcissism

Malignant narcissism, a term first coined in a book by Erich Fromm in 1964,[28] is a syndrome consisting of a cross breed of the narcissistic personality disorder, the antisocial personality disorder, as well as paranoid traits. The malignant narcissist differs from one suffering from narcissistic personality disorder in that the malignant narcissist derives higher levels of psychological gratification from accomplishments over time (thus worsening the disorder). Because the malignant narcissist becomes more involved in this psychological gratification, in the context of the right conditions, the narcissist is apt to develop the antisocial, the paranoid, and the schizoid personality disorders. The term malignant is added to the term *narcissist* to indicate that individuals with this disorder have a powerful form of narcissism that has made them ill in the forms of paranoid and anti-social traits.

Medical narcissism

Medical narcissism is a term coined by John Banja in his book, *Medical Errors and Medical Narcissism*.[29][30]

Banja defines "medical narcissism" as the need of health professionals to preserve their self-esteem leading to the compromise of error disclosure to patients.

In the book he explores the psychological, ethical and legal effects of medical errors and the extent to which a need to constantly assert their competence can cause otherwise capable, and even exceptional, professionals to fall into narcissistic traps.

He claims that:

> ...most health professionals (in fact, most professionals of any ilk) work on cultivating a self that exudes authority, control, knowledge, competence and respectability. It's the narcissist in us all—we dread appearing stupid or incompetent.

Primordial narcissism

Psychiatrist Ernst Simmel first defined primordial narcissism in 1944. Simmel's fundamental thesis is that the most primitive stage of libidinal development is not the oral, but the gastro-intestinal one. Mouth and anus are merely to be considered as the terminal parts of this organic zone. Simmel terms the psychological condition of prenatal existence "primordial narcissism." It is the vegetative stage of the pre-ego, identical with the id. At this stage there is complete instinctual repose, manifested in unconsciousness. Satiation of the gastro-intestinal zone, the representative of the instinct of self-preservation, can bring back this complete instinctual repose, which, under pathological conditions, can become the aim of the instinct.

Contrary to Lasch, Bernard Stiegler argues in his book, *Acting Out*, that consumer capitalism is in fact destructive of what he calls primordial narcissism, without which it is not possible to extend love to others.[31]

In other words he is referring to the natural state of an infant as a fetus and in the first few days of its life, before it has learned that other people exist besides itself, and therefore cannot possibly be aware that they are human beings with feelings, rather than having anything to do with actual narcissism.

Sexual narcissism

Sexual narcissism has been described as an egocentric pattern of sexual behavior that involves an inflated sense of sexual ability and sexual entitlement. In addition, sexual narcissism is the erotic preoccupation with oneself as a superb lover through a desire to merge sexually with a mirror image of oneself. Sexual narcissism is an intimacy dysfunction in which sexual exploits are pursued, generally in the form of extramarital affairs, to overcompensate for low self-esteem and an inability to experience true intimacy. This behavioral pattern is believed to be more common

in men than in women and has been tied to domestic violence in men and sexual coercion in couples. Hurlbert argues that sex is a natural biological given and therefore cannot be deemed as an addiction. He and his colleagues assert that any sexual addiction is nothing more than a misnomer for what is actually sexual narcissism or sexual compulsivity.

Spiritual narcissism

Spiritual narcissism is turning the pursuit of spirituality into an ego-building and confusion-creating endeavor.[32] This is based on the idea that ego development is counter to spiritual progress.

Narcissistic parents

Narcissistic parents demand certain behavior from their children because they see the children as extensions of themselves, and need the children to represent them in the world in ways that meet the parents' emotional needs. This parenting 'style' most often results in estranged relationships with the children, coupled with feelings of resentment and self-destructive tendencies.[20]

Narcissistic leadership

Narcissistic leadership is a common form of leadership. The narcissism may be healthy or destructive although there is a continuum between the two. A study published in the journal Personality and Social Psychology Bulletin suggests that when a group is without a leader, you can often count on a narcissist to take charge. Researchers found that people who score high in narcissism tend to emerge as group leader.[33]

Narcissism and popular culture

> According to recent cultural criticism, Narcissus has replaced Oedipus as the myth of our time. Narcissism is now seen to be at the root of everything from the ill-fated romance with violent revolution to the enthralled mass consumption of state-of-the-art products and the 'lifestyles of the rich and famous'
>
> Jessica Benjamin (2000), "The Oedipal Riddle," p. 233[34]

It is a habitual observation that an integral facet of the consumerist and media-suffused culture of the present is the ubiquity of self-regard and self-absorption. This claim finds apparent confirmation in the pervasive fascination with celebrity and fame,[35] the frequency with which "reality" programs populate the television schedules, and the growth of an online culture in which digital media and the "will-to-fame" are generating a "new era of public narcissism [that] is mutating with new media forms." In this analysis, narcissism, rather than being the pathologized property of a discrete personality type, has been asserted as a constituent cultural feature of an entire generation since the end of World War II.[36]

Supporting the contention that American culture has become more narcissistic and that this is increasingly reflected in its cultural products is an analysis of US popular song lyrics between 1987 and 2007. This found a growth in the use of first-person singular pronouns, reflecting a greater focus on the self, and also of references to antisocial behavior; during the same period, there was a diminution of words reflecting a focus on others, positive emotions, and social interactions. Similar patterns of change in cultural production are observable in other Western states. A linguistic analysis of the largest circulation Norwegian newspaper found that the use of self-focused and individualistic terms increased in frequency by 69 per cent between 1984 and 2005 while collectivist terms declined by 32 per cent. References to narcissism and self-esteem in American popular print media have experienced vast inflation since the late 1980s. Between 1987 and 2007 direct mentions of self-esteem in leading US newspapers and magazines increased by 4,540 per cent while narcissism, which had been almost non-existent in the press during the 1970s, was referred to over 5,000 times between 2002 and 2007.

Cross-cultural studies of differences in narcissism are rare. Instead, as there is a positive association between narcissism and individualism and a negative one between it and collectivism, these traits have been used as proxies for narcissism in some studies. This approach, however, risks the misapplication of the concepts of individualism and collectivism to create overly-fixed, "caricature-like",[37] oppositional categories. Nonetheless, one study looked at differences in advertising products between an individualistic culture, America, and a collectivist one, South Korea. In American magazine advertisements, it found, there was a greater tendency to stress the distinctiveness and uniqueness of the person; conversely the South Korean ones stressed the importance of social conformity and harmony. This observation holds true for a cross-cultural analysis across a wide range of cultural outputs where individualistic national cultures produce more individualistic cultural products and collectivist national cultures produce more collectivist national products; these cultural effects were greater than the effects of individual differences within national cultures.

Fictional Narcissists

- The gang from It's Always Sunny in Philadelphia is narcissistic, but Glenn Howerton's character Dennis Reynolds displays the most extreme narcissistic traits with more sociopathic tendencies.
- Jay Gatsby, the titular character of F. Scott Fitzgerald's 1925 novel *The Great Gatsby*, "an archetype of self-made American men seeking to join high society," has been described as a "pathological narcissist" for whom the "ego-ideal" has become "inflated and destructive" and whose "grandiose lies, poor sense of reality, sense of entitlement, and exploitive treatment of others" conspire toward his own demise.[38]
- In the film *To Die For*, Nicole Kidman's character wants to appear on television at all costs, even if this involves murdering her husband. A psychiatric assessment of her character noted that she "was seen as a prototypical narcissistic person by the raters: on average, she satisfied 8 of 9 criteria for narcissistic personality disorder... had she been evaluated for personality disorders, she would receive a diagnosis of narcissistic personality disorder."
- Abigail "Misty" Briarton from Call of Duty: Black Ops II she sees herself as better than the rest of the group.
- Kuja Tribal from Final Fantasy IX had the chance to kill the main protagonist Zidane Tribal but instead decided to let him live and show him that he is superior. When he found out that he is a mortal, he decided that no one deserved to live while he wasn't alive and thus not only destroying their home world Terra, along with the inhabitants, but also attempted to wipe out all of existence.
- Johnny Bravo, from the show of the same name, has narcissistic behaviors toward himself and of women he sees around him.
- The Riddler from Batman has narcissistic belief that he is the smartest person in the world.
- Niklaus Mikaelson from The Vampire Diaries, having be a bastard to his family, has developed into a narcissistic personality after having his emotions heightened as a Vampire.

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External links

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- Distinctions between Self-Esteem and Narcissism: Implications for Practice, [[Lilian Katz|Lilian G. Katz (http://ceep.crc.uiuc.edu/eecearchive/books/selfe.html)]]
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Narcissistic personality disorder

Narcissistic personality disorder	
Classification and external resources	
 Narcissus by Caravaggio. Gazing at his own reflection.	
ICD-10	F60.8 [1]
ICD-9	301.81 [2]
MedlinePlus	000934 [3]
MeSH	D010554 [4]

Narcissistic personality disorder (**NPD**) is a personality disorder[5] in which a person is excessively preoccupied with personal adequacy, power, prestige and vanity. This condition affects one percent of the population.WP:NOTRS First formulated in 1968, it was historically called megalomania, and is severe egocentrism.[*citation needed*]

Symptoms

Some people diagnosed with a narcissistic personality disorder are characterized by exaggerated feelings of self-importance. They have a sense of entitlement and demonstrate grandiosity in their beliefs and behavior. They have a strong need for admiration, but lack feelings of empathy.

Symptoms of this disorder, as defined by the DSM-IV-TR, include:[5]

- Expects to be recognized as superior and special, without superior accomplishments
- Expects constant attention, admiration and positive reinforcement from others
- Envies others and believes others envy him/her
- Is preoccupied with thoughts and fantasies of great success, enormous attractiveness, power, intelligence
- Lacks the ability to empathize with the feelings or desires of others
- Is arrogant in attitudes and behavior
- Has expectations of special treatment that are unrealistic

Other symptoms in addition to the ones defined by DSM-IV-TR include: Is interpersonally exploitative, i.e., takes advantage of others to achieve his or her own ends, has trouble keeping healthy relationships with others, easily hurt

or rejected, appears unemotional, and exaggerating special achievements and talents, setting unrealistic goals for himself/herself.

Narcissistic personality disorder is characterized by dramatic, emotional behavior, and an over-inflated sense of self-importance that is in the same category as antisocial and borderline personality disorders.[6]

In addition to these symptoms, the person may display arrogance, show superiority, and seek power.[7] The symptoms of narcissistic personality disorder can be similar to the traits of individuals with strong self-esteem and confidence; differentiation occurs when the underlying psychological structures of these traits are considered pathological. Narcissists have such an elevated sense of self-worth that they value themselves as inherently better than others, when in reality they have a fragile self-esteem, cannot handle criticism, and often try to compensate for this inner fragility by belittling or disparaging others in an attempt to validate their own self-worth. Comments and criticisms about others are vicious from sufferers of NPD, in an attempt to boost their own poor self-esteem.[8]

Another narcissist symptom is a lack of empathy. They are unable to relate, understand, and rationalize the feelings of others. Instead of behaving in a way that shows how they are feeling in the moment, they behave in the way that they feel they are expected to behave or what gives them the most attention.

In children, inflated self-views and grandiose feelings, which are characteristics of narcissism, are part of the normal self-development. Children typically cannot understand the difference between their actual and their ideal self, which causes an unrealistic perception of the self. After about age 8, views of the self, both positive and negative, begin to develop based on comparisons of peers, and become more realistic. Two factors that cause self-view to remain unrealistic are dysfunctional interactions with parents that can be either excessive attention or a lack thereof. For example but not limited to, the excessive attention and lack of attention go hand in hand when a child's parents are divorced. Usually, one is overindulgent (typically the one seeing the child less) and the other shows less affection. The child either compensates for lack of attention or acts in terms of unrealistic self-perception.[9]

An extensive US survey found a high association with other disabilities, especially amongst men: mental disability, substance use, mood, anxiety disorders and other personality disorders, bipolar I disorder, post-traumatic stress disorder, and schizotypal and borderline personality disorders were among the associated disabilities.

Eating disorders

The study of Narcissism and the Narcissistic Defenses in the Eating Disorders was concerned with the correlation between eating pathology and narcissism. Two types of narcissism were observed: core narcissism, having extremely positive (high) self-esteem combined with delusions about the level and ability of achievement; and narcissistic defenses, defenses that are triggered when self-esteem is threatened. Such narcissists maintain self-esteem by seeing themselves as misunderstood and a subject to intolerable demands.[10]

Two types of narcissistic defenses that were measured with eating pathology were "poisonous pedagogy" and "narcissistically abused". Poisonous pedagogy is one who places blame on others and is overly critical of others' inadequacies. The narcissistically abused are those who put others' needs before theirs yet see themselves as being poorly treated. Two groups were measured: Clinical (83 women and one male with the mean age of 28.4) and Non Clinical (70 women mean age of 23.2). BMI of groups did not significantly vary. They filled out a questionnaire that was measured by eating characteristic and narcissism levels by the OMNI (O'Brien Multiphasic Narcissism Inventory) and the EDE-Q (Eating Disorder Examination Questionnaire). OMNI measures pathological narcissism of narcissistic personality, poisonous pedagogy, and narcissistically abused personality. EDE-Q measures the common eating disorders: restraint, eating concern, body shape concern, and body weight concern.

The basic summaries of the questionnaire's findings were the poisonous pedagogy defenses was related to restrictive mind-set; narcissistically abused defense related to restraint, eating concern, body shape concern, and body weight concern. The only main difference between the groups was the role of core narcissism in the clinical women's levels of eating concerns. Further research is needed to better understand the relationship approaches in both groups.

Professional attainment

In 2005, Board and Fritzon published the results of a study in which they interviewed senior business managers, assessing them for the presence of personality disorder.[11] Comparing their findings to three samples of psychiatric patients, they found that their senior business managers were as likely to demonstrate narcissistic traits as the patient population, although were less physically aggressive.

Causes

The cause of this disorder is unknown; however, Groopman and Cooper (2006) listed the following factors identified by various researchers as possibilities:

- An oversensitive temperament (personality traits) at birth.
- Excessive admiration that is never balanced with realistic feedback.
- Excessive praise for good behaviors or excessive criticism for bad behaviors in childhood.
- Overindulgence and overvaluation by parents, other family members, or peers.
- Being praised for perceived exceptional looks or abilities by adults.
- Severe emotional abuse in childhood.
- Unpredictable or unreliable caregiving from parents.
- Learning manipulative behaviors from parents.
- Valued by parents as a means to regulate their own self-esteem.

Some narcissistic traits are common with a normal developmental phase. When these traits are compounded by a failure of the interpersonal environment and continue into adulthood, they may intensify to the point where NPD is diagnosed.[12]

In addition, biological differences may cause narcissism as structural abnormalities in the brain have been recently documented. Specifically, researchers have noted that narcissists have less volume in gray matter in the left anterior insula, which is related to empathy (Schulze et al., 2013).[13]

Theories

Pathological narcissism occurs in a spectrum of severity. In its more extreme forms, it is narcissistic personality disorder (NPD). NPD is considered to result from a person's belief that they are flawed in a way that makes them fundamentally unacceptable to others.[14] This belief is held below the person's conscious awareness; such a person would, if questioned, typically deny thinking such a thing. To protect themselves against the intolerably painful rejection and isolation that (they imagine) would follow if others recognized their (perceived) defective nature, such people make strong attempts to control others' views of them and behavior towards them.

Pathological narcissism can develop from an impairment in the quality of the person's relationship with their primary caregivers, usually their parents, in that the parents could not form a healthy and empathic attachment to them. This results in the child's perception of himself/herself as unimportant and unconnected to others. The child typically comes to believe they have some personality defect that makes them unvalued and unwanted.

To the extent that people are pathologically narcissistic, they can be controlling, blaming, self-absorbed, intolerant of others' views, unaware of others' needs and of the effects of their behavior on others, and insistent that others see them as they wish to be seen.[15]

Narcissistic individuals use various strategies to protect the self at the expense of others. They tend to devalue, derogate and blame others, and they respond to threatening feedback with anger and hostility.[16]

People who are narcissistic commonly feel rejected, humiliated and threatened when criticised. To protect themselves from these dangers, they often react with disdain, rage, and/or defiance to any slight criticism, real or imagined.[17] To avoid such situations, some narcissistic people withdraw socially and may feign modesty or humility. In cases where the narcissistic personality-disordered individual feels a lack of admiration, adulation,

attention and affirmation, they may also manifest a desire to be feared and be notorious (narcissistic supply).

Although individuals with NPD are often ambitious and capable, the inability to tolerate setbacks, disagreements or criticism, along with lack of empathy, make it difficult for such individuals to work cooperatively with others or to maintain long-term professional achievements.[18] With narcissistic personality disorder, the individual's self-perceived fantastic grandiosity, often coupled with a hypomanic mood, is typically not commensurate with his or her real accomplishments.

Splitting

People who are diagnosed with narcissistic personality disorder use splitting as a central defense mechanism. According to psychoanalyst Kernberg, "The normal tension between actual self on the one hand, and ideal object on the other, is eliminated by the building up of an inflated self-concept within which the actual self and the ideal self and ideal object are confused. At the same time, the remnants of the unacceptable images are repressed and projected onto external objects, which are devalued."[19]

The merging of the "inflated self-concept" and the "actual self" is seen in the inherent grandiosity of narcissistic personality disorder. Also inherent in this process are the defense mechanisms of devaluation, idealization and denial.[20] Other people are either manipulated as an extension of one's own self, who serve the sole role of giving "admiration and approval"[21] or they are seen as worthless (because they cannot collude with the narcissist's grandiosity).[22]

Relationship to shame

It has been suggested that narcissistic personality disorder may be related to defenses against shame.[23] Psychiatrist Glen Gabbard suggested NPD could be broken down into two subtypes.[24] He saw the "oblivious" subtype as being grandiose, arrogant, and thick-skinned, and the "hypervigilant" subtype as being easily hurt, oversensitive, and ashamed. In his view, the oblivious subtype presents for admiration, envy, and appreciation of a powerful, grandiose self that is the antithesis of a weak internalized self, which hides in shame, while the hypervigilant subtype neutralizes devaluation by seeing others as unjust abusers. Dr. Jeffrey Young, who coined the term "Schema Therapy", a technique originally developed by psychiatrist Aaron T. Beck (1979), also links NPD and shame. He sees the so-called Defectiveness Schema as a core schema of NPD, along with the Emotional Deprivation and Entitlement Schemas.[25]

Diagnosis

DSM-5

The formulation of Narcissistic personality disorder in the diagnostic manual DSM-IV has been criticised for failing to describe the range and complexity of the disorder. Critics say it focuses overly on "...the narcissistic individual's external, symptomatic, or social interpersonal patterns—at the expense of ... internal complexity and individual suffering," which reduces its clinical utility.

The *Personality and Personality Disorders Work Group* originally proposed the elimination of NPD as a distinct disorder in DSM-5 as part of a major revamping of the diagnostic criteria for personality disorders, replacing a categorical with a dimensional approach based on the severity of dysfunctional personality trait domains.

Some clinicians objected to this, characterizing the new diagnostic system as an "unwieldy conglomeration of disparate models that cannot happily coexist" and may have limited usefulness in clinical practice.

In July 2011, the Work Group came back with a major revision to their original proposal. In this revision, NPD was reinstated with dramatic changes to its definition.[26] The general move towards a dimensional (personality trait-based) view of the Personality Disorders has been maintained despite the reintroduction of NPD.

ICD-10

The World Health Organization's ICD-10 lists narcissistic personality disorder under *(F 60.8 [1]) Other specific personality disorders*.[27]

It is a requirement of ICD-10 that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

Subtypes

Theodore Millon identified five narcissist subtypes, however, there are few pure variants of any subtype, and the subtypes are not recognized in the DSM or ICD.

Subtype	Description	Personality Traits
Unprincipled narcissist	Including antisocial features. A charlatan who is a fraudulent, exploitative, deceptive, and unscrupulous individual	Deficient conscience; unscrupulous, amoral, disloyal, fraudulent, deceptive, arrogant, exploitive; a con man and charlatan; dominating, contemptuous, vindictive.
Amorous narcissist	Including histrionic features. The Don Juan or Casanova of our times who is erotic, exhibitionist	Sexually seductive, enticing, beguiling, tantalizing; glib and clever; disinclines real intimacy; indulges hedonistic desires; bewitches and inveigles others; pathological lying and swindling.
Compensatory narcissist	Including negativistic features	Seeks to counteract or cancel out deep feelings of inferiority and lack of self-esteem; offsets deficits by creating illusions of being superior, exceptional, admirable, noteworthy; self-worth results from self-enhancement.
Elitist narcissist	Variant of “pure” pattern. Corresponds to Wilhelm Reich's "phallic narcissistic" personality type	Feels privileged and empowered by virtue of special childhood status and pseudo achievements; entitled façade bears little relation to reality; seeks favored and good life; is upwardly mobile; cultivates special status and advantages by association.
Fanatic narcissist	Including paranoid features	An individual whose self-esteem was severely arrested during childhood, who usually displays major paranoid tendencies, and who holds on to an illusion of omnipotence. These people are fighting delusions of insignificance and lost value, and trying to re-establish their self-esteem through grandiose fantasies and self-reinforcement. When unable to gain recognition or support from others, they take on the role of a heroic or worshipped person with a grandiose mission.

Other theorists have identified two types of narcissism. Those narcissists who have been diagnosed with narcissistic grandiosity express behavior "through interpersonally exploitative acts, lack of empathy, intense envy, aggression, and exhibitionism."[28] Another type of narcissism is narcissistic vulnerability. It entails (on a conscious level) "helplessness, emptiness, low self-esteem, and shame, which can be expressed in the behavior as being socially avoidant in situations where their self-presentation is not possible so they withdraw, or the approval they need/expect is not being met."

Treatment

Clinical strategies are outlined by Heinz Kohut, Stephen M. Johnson and James F. Masterson, while Johns discusses a continuum of severity and the kinds of therapy most effective in different cases. Schema Therapy, a form of therapy developed by Jeffrey Young that integrates several therapeutic approaches (psychodynamic, cognitive, behavioral etc.), also offers an approach for the treatment of NPD.[29] It is unusual for people to seek therapy for NPD. This is partly due to the NPD sufferers' not believing they have a problem. Most, if not all, are unable to see the destructive damage they are causing to themselves and to others and usually only seek treatment at the insistence of relatives and friends. Unconscious fears of exposure or inadequacy often cause defensive disdain of therapeutic processes.[30][31] Pattern change strategies, over a long period of time, are for narcissists to work on increasing their

ability to become more empathetic in everyday relationships. To help modify their sense of entitlement and self-centeredness schema, the strategy is to help them identify how to utilize their unique talents and to help others for reasons other than their own personal gain. This is not so much to change their self-perception of their "entitlement" feeling but more to help them empathize with others. Another type of treatment would be temperament change.[32]

Anger, rage, impulsivity and impatience can be worked on with skill training. Therapy is not one hundred percent effective because patients receive feedback poorly and defensively. Anxiety disorders and somatoma dysfunctions are prevalent but the most common would be depression. Medication has proven ineffective for treating narcissistic personality disorder, but psychoanalytic psychotherapy has a higher success rate. Therapists must recognize the patient's traits and use caution in tearing down narcissistic defenses too quickly.

Group treatment has its benefits as the effectiveness of receiving peer feedback rather than the clinician's may be more accepted, but group therapy can also contradict itself as the patient may show "demandingness, egocentrism, social isolation and withdrawal, and socially deviant behavior." Researchers originally thought group therapy among Narcissists would fail because it was believed that group therapy required empathy that NPD patients lack. However, studies show group therapy does hold value for patients because it lets them explore boundaries, develop trust, increase self-awareness, and accept feedback. Relationship therapy stresses the importance of learning and applying four basic interpersonal skills: "...effective expression, empathy, discussion and problem solving/conflict resolution."

Marital/relationship therapy is most beneficial when both partners participate.

Epidemiology

Lifetime prevalence is estimated at 1% in the general population and 2% to 16% in clinical populations.[33]

In 2009, Twenge and Campbell conducted studies suggesting that the incidence of NPD had more than doubled in the US in the prior 10 years, and that 1 in 16 of the population have experienced NPD.[34]

History

The use of the term "narcissism" to describe excessive vanity and self-centeredness predates by many years the modern medical classification of narcissistic personality disorder. The condition was named after Narcissus, a mythological Greek youth who became infatuated with his own reflection in a lake. He did not realize at first that it was his own reflection, but when he did, he died out of grief for having fallen in love with someone that did not exist outside of himself.

The term "narcissistic personality structure" was introduced by Kernberg in 1967[35] and "narcissistic personality disorder" first proposed by Heinz Kohut in 1968.[36]

Society and culture

In the film *To Die For*, Nicole Kidman's character wants to appear on television at all costs, even if this involves murdering her husband. A psychiatric assessment of her character noted that she "was seen as a prototypical narcissistic person by the raters: on average, she satisfied 8 of 9 criteria for narcissistic personality disorder... had she been evaluated for personality disorders, she would receive a diagnosis of narcissistic personality disorder."

Asuka Langley Soryu from *Neon Genesis Evangelion* was written to correspond with the DSM-criteria for Narcissistic personality disorder.

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- How to Survive Working for a Narcissistic Leader (http://www.bnet.com/blog/business-strategy/how-to-survive-working-for-a-narcissistic-leader/1718) (2011-05-19) BNet
- The Narcissism Epidemic, Jean M. Twenge, Ph.D. and W. Keith Campbell, Ph.D., New York, Free Press 2009 ISBN 978-1-4165-7625-9

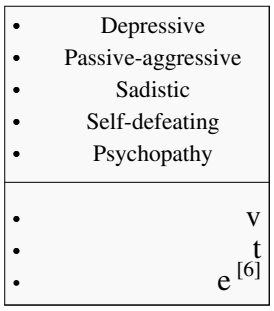
External links

- DSM-IV-TR Narcissistic Personality Disorder Diagnostic Criteria (http://www.behavenet.com/capsules/disorders/narcissisticpd.htm)
- Narcissistic personality disorder (http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001930/) PubMed
- Narcissistic personality disorder (http://www.mayoclinic.com/health/narcissistic-personality-disorder/DS00652/DSECTION=symptoms) Mayo Clinic
- Narcissistic Personality Disorder (http://my.clevelandclinic.org/disorders/personality_disorders/hic_narcissistic_personality_disorder.aspx) Cleveland Clinic

Personality disorder

Personality disorders	
Classification and external resources	
ICD-10	F60 [1]
ICD-9	301.9 [2]
DiseasesDB	9889 [3]
MedlinePlus	000939 [4]
MeSH	D010554 [4]

Personality disorders
Cluster A (odd)
• Paranoid • Schizoid • Schizotypal
Cluster B (dramatic)
• Antisocial • Borderline • Histrionic • Narcissistic
Cluster C (anxious)
• Avoidant • Dependent • Obsessive–compulsive
Not specified



Personality disorders are a class of mental disorders characterised by enduring maladaptive patterns of behavior, cognition and inner experience, exhibited across many contexts and deviating markedly from those accepted by the individual's culture. These patterns develop early, are inflexible and are associated with significant distress or disability. The definitions may vary some according to other sources.

Official criteria for diagnosing personality disorders are listed in the *Diagnostic and Statistical Manual of Mental Disorders*, published by the American Psychiatric Association, and in the mental and behavioral disorders section of the *International Statistical Classification of Diseases and Related Health Problems*, published by the World Health Organization. The DSM-5 published in 2013 now lists personality disorders in exactly the same way as other mental disorders, rather than on a separate 'axis' as previously.[5]

Personality, defined psychologically, is the set of enduring behavioral and mental traits that distinguish human beings. Hence, personality disorders are defined by experiences and behaviors that differ from societal norms and expectations. Those diagnosed with a personality disorder may experience difficulties in cognition, emotiveness, interpersonal functioning or control of impulses. In general, personality disorders are diagnosed in 40–60 percent of psychiatric patients, making them the most frequent of all psychiatric diagnoses.[6]

These behavioral patterns in personality disorders are typically associated with substantial disturbances in some behavioral tendencies of an individual, usually involving several areas of the personality, and are nearly always associated with considerable personal and social disruption. A person is classified as having a personality disorder if their abnormalities of behavior impair their social or occupational functioning. Additionally, personality disorders are inflexible and pervasive across many situations, due in large part to the fact that such behavior may be ego-syntonic (i.e. the patterns are consistent with the ego integrity of the individual) and are, therefore, perceived to be appropriate by that individual. This behavior can result in maladaptive coping skills, which may lead to personal problems that induce extreme anxiety, distress or depression.[7] These patterns of behavior typically are recognized in adolescence and the beginning of adulthood and, in some unusual instances, childhood.

There are many issues with classifying a personality disorder.[8] There are many categories of definition,Wikipedia:Please clarify some mild and some extreme. Because the theory and diagnosis of personality disorders stem from prevailing cultural expectations, their validity is contested by some experts on the basis of invariable subjectivity. They argue that the theory and diagnosis of personality disorders are based strictly on social, or even sociopolitical and economic considerations.[9][10][11]

Classification

The two major systems of classification, the ICD and DSM, have deliberately merged their diagnoses to some extent, but some differences remain. For example, ICD-10 does not include narcissistic personality disorder as a distinct category, while DSM-5 does not include enduring personality change after catastrophic experience or after psychiatric illness. ICD-10 classifies the DSM-5 schizotypal personality disorder as a form of schizophrenia rather than as a personality disorder. There are accepted diagnostic issues and controversies with regard to distinguishing particular personality disorder categories from each other. ICD classifies Transsexualism as a personality disorder;[12] while DSM-5 addresses Gender dysphoria.

World Health Organization

The ICD-10 section on mental and behavioral disorders includes categories of personality disorder and enduring personality changes. They are defined as ingrained patterns indicated by inflexible and disabling responses that significantly differ from how the average person in the culture perceives, thinks and feels, particularly in relating to others.[13]

The specific personality disorders are: paranoid, schizoid, dissocial, emotionally unstable (borderline type and impulsive type), histrionic, anankastic, anxious (avoidant) and dependent.[14]

There is also an 'Others' category involving conditions characterized as eccentric, *haltlose* (derived from "haltlos" (*German*) = drifting, aimless and irresponsible), immature, narcissistic, passive-aggressive or psychoneurotic. An additional category is for unspecified personality disorder, including character neurosis and pathological personality.

There is also a category for Mixed and other personality disorders, defined as conditions that are often troublesome but do not demonstrate the specific pattern of symptoms in the named disorders. Finally there is a category of Enduring personality changes, not attributable to brain damage and disease. This is for conditions that seem to arise in adults without a diagnosis of personality disorder, following catastrophic or prolonged stress or other psychiatric illness.

American Psychiatric Association

The Diagnostic and Statistical Manual of Mental Disorders (currently the DSM-5) provides a definition of a General personality disorder that stress such disorders are an enduring and inflexible pattern of long duration that lead to significant distress or impairment and are not due to use of substances or another medical condition. DSM-5 lists ten personality disorders, grouped into three clusters. The DSM-5 also contains three diagnoses for personality patterns that do not match these ten disorders, but nevertheless exhibit characteristics of a personality disorder.

Cluster A (odd disorders)

- **Paranoid personality disorder:** characterized by a pattern of irrational suspicion and mistrust of others, interpreting motivations as malevolent
- **Schizoid personality disorder:** lack of interest and detachment from social relationships, and restricted emotional expression
- **Schizotypal personality disorder:** a pattern of extreme discomfort interacting socially, distorted cognitions and perceptions

Cluster B (dramatic, emotional or erratic disorders)

- **Antisocial personality disorder:** a pervasive pattern of disregard for and violation of the rights of others, lack of empathy
- **Borderline personality disorder:** pervasive pattern of instability in relationships, self-image, identity, behavior and affects often leading to self-harm and impulsivity
- **Histrionic personality disorder:** pervasive pattern of attention-seeking behavior and excessive emotions
- **Narcissistic personality disorder:** a pervasive pattern of grandiosity, need for admiration, and a lack of empathy

Cluster C (anxious or fearful disorders)

- **Avoidant personality disorder:** pervasive feelings of social inhibition and inadequacy, extreme sensitivity to negative evaluation
- **Dependent personality disorder:** pervasive psychological need to be cared for by other people.
- **Obsessive-compulsive personality disorder (not the same as obsessive-compulsive disorder):** characterized by rigid conformity to rules, perfectionism and control

Other personality disorders

- **Personality change due to another medical condition –** is a personality disturbance due to the direct effects of a medical condition
- **Other specified personality disorder –** symptoms characteristic of a personality disorder but fails to meet the criteria for a specific disorder, with the reason given
- **Personality disorder not otherwise specified**

Other

Some types of personality disorder were in previous versions of the diagnostic manuals but have been deleted. This includes two types that were in the DSM-III-R appendix as "Proposed diagnostic categories needing further study" without specific criteria, namely Sadistic personality disorder (a pervasive pattern of cruel, demeaning and aggressive behavior) and Self-defeating personality disorder (masochistic personality disorder) (characterised by behaviour consequently undermining the person's pleasure and goals). The psychologist Theodore Millon and others consider some relegated diagnoses to be equally valid disorders, and may also propose other personality disorders or subtypes, including mixtures of aspects of different categories of the officially accepted diagnoses.[15]

Personality disorder diagnoses in each edition of American Psychiatric Association's Diagnostic Manual[16]					
DSM-I	**DSM-II**	**DSM-III**	**DSM-III-R**	**DSM-IV(-TR)**	**DSM-V Proposals**
Personality					
Pattern disturbance:					
Inadequate	Inadequate				
Schizoid	Schizoid	Schizoid	Schizoid	Schizoid	
Cyclothymic	Cyclothymic				
Paranoid	Paranoid	Paranoid	Paranoid	Paranoid	
		Schizotypal	Schizotypal	Schizotypal	Schizotypal*
Personality					
Trait disturbance:					
Emotionally unstable	Hysterical	Histrionic	Histrionic	Histrionic	
		Borderline	Borderline	Borderline	Borderline
Compulsive	Obsessive-compulsive	Compulsive	Obsessive-compulsive	Obsessive-compulsive	Obsessive-compulsive
Passive-aggressive:					
Passive-depressive subtype		Dependent	Dependent	Dependent	
Passive-aggressive subtype	Passive-aggressive	Passive-aggressive	Passive-aggressive		
Aggressive subtype					
	Explosive				
	Asthenic				

		Avoidant	Avoidant	Avoidant	Avoidant
		Narcissistic	Narcissistic	Narcissistic	Narcissistic**
Sociopathic personality					
Disturbance:					
Antisocial reaction	Antisocial	Antisocial	Antisocial	Antisocial	Antisocial-psychopathic
Dyssocial reaction					
Sexual deviation					
Addiction					
			Appendix:	*Appendix:*	*Appendix:*
			Self-defeating	Negativistic	Dependent
			Sadistic	Depressive	Histrionic
					Paranoid
					Schizoid
					Negativistic
					Depressive

** – Not actually to be classified as a personality disorder; classified instead as a form of schizophrenia-spectrum disorder.*

*** – Originally proposed for deletion; status remains unclear for DSM-5.*

Millon's description

Psychologist Theodore Millon, who has written numerous popular works on personality, proposed the following description of personality disorders:

Millon's brief description of personality disorders[17]	
Type of personality disorder	**Description**
Paranoid	Guarded, defensive, distrustful and suspiciousness. Hypervigilant to the motives of others to undermine or do harm. Always seeking confirmatory evidence of hidden schemes. Feels righteous, but persecuted.
Schizoid	Apathetic, indifferent, remote, solitary, distant, humorless. Neither desires nor needs human attachments. Withdrawal from relationships and prefer to be alone. Little interest in others, often seen as a loner. Minimal awareness of feelings of self or others. Few drives or ambitions, if any.
Schizotypal	Eccentric, self-estranged, bizarre, absent. Exhibits peculiar mannerisms and behaviors. Thinks can read thoughts of others. Preoccupied with odd daydreams and beliefs. Blurs line between reality and fantasy. Magical thinking and strange beliefs.
Antisocial	Impulsive, irresponsible, deviant, unruly. Acts without due consideration. Meets social obligations only when self-serving. Disrespects societal customs, rules, and standards. Sees self as free and independent.
Borderline	Unpredictable, manipulative, unstable. Frantically fears abandonment and isolation. Experiences rapidly fluctuating moods. Shifts rapidly between loving and hating. Sees self and others alternatively as all-good and all-bad. Unstable and frequently changing moods.
Histrionic	Dramatic, seductive, shallow, stimulus-seeking, vain. Overreacts to minor events. Exhibitionistic as a means of securing attention and favors. Sees self as attractive and charming. Constant seeking for others' attention.
Narcissistic	Egotistical, arrogant, grandiose, insouciant. Preoccupied with fantasies of success, beauty, or achievement. Sees self as admirable and superior, and therefore entitled to special treatment.

Avoidant	Hesitant, self-conscious, embarrassed, anxious. Tense in social situations due to fear of rejection. Plagued by constant performance anxiety. Sees self as inept, inferior, or unappealing. Feels alone and empty.
Dependent	Helpless, incompetent, submissive, immature. Withdraws from adult responsibilities. Sees self as weak or fragile. Seeks constant reassurance from stronger figures.
Obsessive–compulsive	Restrained, conscientious, respectful, rigid. Maintains a rule-bound lifestyle. Adheres closely to social conventions. Sees the world in terms of regulations and hierarchies. Sees self as devoted, reliable, efficient, and productive.
Depressive	Somber, discouraged, pessimistic, brooding, fatalistic. Presents self as vulnerable and abandoned. Feels valueless, guilty, and impotent. Judges self as worthy only of criticism and contempt.
Passive–aggressive (Negativistic)	Resentful, contrary, skeptical, discontented. Resists fulfilling others' expectations. Deliberately inefficient. Vents anger indirectly by undermining others' goals. Alternately moody and irritable, then sullen and withdrawn.
Sadistic	Explosively hostile, abrasive, cruel, dogmatic. Liable to sudden outbursts of rage. Feels selfsatisfied through dominating, intimidating and humiliating others. Is opinionated and close-minded.
Self-defeating (Masochistic)	Deferential, pleasure-phobic, servile, blameful, self-effacing. Encourages others to take advantage. Deliberately defeats own achievements. Seeks condemning or mistreatful partners.

Additional classification factors

Except for classifying by category and cluster, it is possible to classify personality disorders using such additional factors as severity, impact on social functioning, and attribution.[18]

Severity

This involves both the notion of personality difficulty as a measure of subthreshold scores for personality disorder using standard interviews and the evidence that those with the most severe personality disorders demonstrate a "ripple effect" of personality disturbance across the whole range of mental disorders. In addition to subthreshold (personality difficulty) and single cluster (simple personality disorder), this also derives complex or diffuse personality disorder (two or more clusters of personality disorder present) and can also derive severe personality disorder for those of greatest risk.

Dimensional System of Classifying Personality Disorders[19]		
Level of Severity	**Description**	**Definition by Categorical System**
0	No Personality Disorder	Does not meet actual or subthreshold criteria for any personality disorder
1	Personality Difficulty	Meets sub-threshold criteria for one or several personality disorders
2	Simple Personality Disorder	Meets actual criteria for one or more personality disorders within the same cluster
3	Complex (Diffuse) Personality Disorder	Meets actual criteria for one or more personality disorders within more than one cluster
4	Severe Personality Disorder	Meets criteria for creation of severe disruption to both individual and to many in society

There are several advantages to classifying personality disorder by severity:

- It not only allows for but also takes advantage of the tendency for personality disorders to be comorbid with each other.
- It represents the influence of personality disorder on clinical outcome more satisfactorily than the simple dichotomous system of no personality versus personality disorder.
- This system accommodates the new diagnosis of severe personality disorder, particularly "dangerous and severe personality disorder" (DSPD). Politicians and the public both want to know who comprise the most dangerous group.

Effect on social functioning

Social function is affected by many other aspects of mental functioning apart from that of personality. However, whenever there is persistently impaired social functioning in conditions in which it would normally not be expected, the evidence suggests that this is more likely to be created by personality abnormality than by other clinical variables. The Personality Assessment Schedule gives social function priority in creating a hierarchy in which the personality disorder creating the greater social dysfunction is given primacy over others in a subsequent description of personality disorder.

Attribution

Many who have a personality disorder do not recognize any abnormality and defend valiantly their continued occupancy of their personality role. This group have been termed the Type R, or treatment-resisting personality disorders, as opposed to the Type S or treatment-seeking ones, who are keen on altering their personality disorders and sometimes clamor for treatment. The classification of 68 personality disordered patients on the caseload of an assertive community team using a simple scale showed a 3 to 1 ratio between Type R and Type S personality disorders with Cluster C personality disorders being significantly more likely to be Type S, and paranoid and schizoid (Cluster A) personality disorders significantly more likely to be Type R than others.

Signs and symptoms

In the workplace

Depending on the diagnosis, severity and individual, and the job itself, personality disorders can be associated with difficulty coping with work or the workplace - potentially leading to problems with others by interfering with interpersonal relationships. Indirect effects also play a role; for example, impaired educational progress or complications outside of work, such as substance abuse and co-morbid mental diseases, can plague sufferers. However, personality disorders can also bring about above-average work abilities by increasing competitive drive or causing the sufferer to exploit his or her co-workers.[20]

In 2005, psychologists Belinda Board and Katarina Fritzon at the University of Surrey, UK, interviewed and gave personality tests to high-level British executives and compared their profiles with those of criminal psychiatric patients at Broadmoor Hospital in the UK. They found that three out of eleven personality disorders were actually more common in executives than in the disturbed criminals:

- Histrionic personality disorder: including superficial charm, insincerity, egocentricity and manipulation
- Narcissistic personality disorder: including grandiosity, self-focused lack of empathy for others, exploitativeness and independence.
- Obsessive-compulsive personality disorder: including perfectionism, excessive devotion to work, rigidity, stubbornness and dictatorial tendencies.

According to leading leadership academic Manfred F.R. Kets de Vries, it seems almost inevitable these days that there will be some personality disorders in a senior management team.

Relationship with other mental disorders

The disorders in each of the three clusters may share some underlying common vulnerability factors involving cognition, affect and impulse control, and behavioral maintenance or inhibition, respectively, and may have a spectrum relationship to certain syndromal mental disorders:

- Paranoid or schizotypal personality disorders may be observed to be premorbid antecedents of delusional disorders or schizophrenia.
- Borderline personality disorder is seen in association with mood and anxiety disorders and with impulse control disorders, eating disorders, ADHD, or a substance use disorder.
- Avoidant personality disorder is seen with social anxiety disorder.

Diagnosis

The DSM-IV lists General diagnostic criteria for a personality disorder, which must be met in addition to the specific criteria for a particular named personality disorder. This requires that there be (to paraphrase):[21]

- An enduring pattern of psychological experience and behavior that differs prominently from cultural expectations, as shown in two or more of: cognition (i.e. perceiving and interpreting the self, other people or events); affect (i.e. the range, intensity, lability, and appropriateness of emotional response); interpersonal functioning; or impulse control.
- The pattern must appear inflexible and pervasive across a wide range of situations, and lead to clinically significant distress or impairment in important areas of functioning.
- The pattern must be stable and long-lasting, have started as early as at least adolescence or early adulthood.
- The pattern must not be better accounted for as a manifestation of another mental disorder, or to the direct physiological effects of a substance (e.g. drug or medication) or a general medical condition (e.g. head trauma).

The ICD-10 'clinical descriptions and diagnostic guidelines' introduces its specific personality disorder diagnoses with some general guideline criteria that are similar. To quote:[22]

- Markedly disharmonious attitudes and behavior, generally involving several areas of functioning; e.g. affectivity, arousal, impulse control, ways of perceiving and thinking, and style of relating to others;
- The abnormal behavior pattern is enduring, of long standing, and not limited to episodes of mental illness;
- The abnormal behavior pattern is pervasive and clearly maladaptive to a broad range of personal and social situations;
- The above manifestations always appear during childhood or adolescence and continue into adulthood;
- The disorder leads to considerable personal distress but this may only become apparent late in its course;
- The disorder is usually, but not invariably, associated with significant problems in occupational and social performance.

The ICD adds: "For different cultures it may be necessary to develop specific sets of criteria with regard to social norms, rules and obligations."

In clinical practice, individuals are generally diagnosed by an interview with a psychiatrist based on a mental status examination, which may take into account observations by relatives and others. One tool of diagnosing personality disorders is a process involving interviews with scoring systems. The patient is asked to answer questions, and depending on their answers, the trained interviewer tries to code what their responses were. This process is fairly time consuming.

Normal personality

The issue of the relationship between normal personality and personality disorders is one of the important issues in personality and clinical psychology. The personality disorders classification (DSM IV TR and ICD-10) follows a categorical approach that views personality disorders as discrete entities that are distinct from each other and from normal personality. In contrast, the dimensional approach is an alternative approach that personality disorders represent maladaptive extensions of the same traits that describe normal personality. Thomas Widiger and his collaborators have contributed to this debate significantly. He discussed the constraints of the categorical approach and argued for the dimensional approach to the personality disorders. Specifically, he proposed that Five Factor Model of personality is alternative to the classification of personality disorders. For example, this view specifies that Borderline Personality Disorder can be understood as a combination of emotional lability (i.e., high neuroticism), impulsivity (i.e., low conscientiousness), and hostility (i.e., low agreeableness). Many studies across cultures have explored the relationship between personality disorders and the Five Factor Model.[23] This research has demonstrated that personality disorders largely correlate in expected ways with measures of the Five Factor Model and has set the stage for including the Five Factor Model within the upcoming DSM-5.[24]

DSM-IV-TR Personality Disorders from the Perspective of the Five-Factor Model of General Personality Functioning[]												
Factors	**PPD**	**SzPD**	**StPD**	**ASPD**	**BPD**	**HPD**	**NPD**	**AvPD**	**DPD**	**OCPD**	**PAPD**	**DpPD**
Neuroticism (vs. emotional stability)												
Anxiousness (vs. unconcerned)	n/a	n/a	High	Low	High	n/a	n/a	High	High	High	n/a	n/a
Angry hostility (vs. dispassionate)	High	n/a	n/a	High	High	n/a	High	n/a	n/a	n/a	High	n/a
Depressiveness (vs. optimistic)	n/a	n/a	n/a	n/a	High	n/a	n/a	n/a	n/a	n/a	n/a	High
Self-consciousness (vs. shameless)	n/a	n/a	High	Low	n/a	Low	Low	High	High	n/a	n/a	High
Impulsivity (vs. restrained)	n/a	n/a	n/a	High	High	High	n/a	Low	n/a	Low	n/a	n/a
Vulnerability (vs. fearless)	n/a	n/a	n/a	Low	High	n/a	n/a	High	High	n/a	n/a	n/a
Extraversion (vs. introversion)												
Warmth (vs. coldness)	Low	Low	Low	n/a	n/a	n/a	Low	n/a	High	n/a	Low	Low
Gregariousness (vs. withdrawal)	Low	Low	Low	n/a	n/a	High	n/a	Low	n/a	n/a	n/a	Low
Assertiveness (vs. submissiveness)	n/a	n/a	n/a	High	n/a	n/a	High	Low	Low	n/a	Low	n/a
Activity (vs. passivity)	n/a	Low	n/a	High	n/a	High	n/a	n/a	n/a	n/a	Low	n/a
Excitement seeking (vs. lifeless)	n/a	Low	n/a	High	n/a	High	High	Low	n/a	Low	n/a	Low
Positive emotionality (vs. anhedonia)	n/a	Low	Low	n/a	n/a	High	n/a	Low	n/a	n/a	n/a	n/a
Openness (vs. closedness)												
Fantasy (vs. concrete)	n/a	n/a	High	n/a	n/a	High	n/a	n/a	n/a	n/a	n/a	n/a
Aesthetics (vs. disinterest)	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Feelings (vs. alexithymia)	n/a	Low	n/a	n/a	High	High	Low	n/a	n/a	Low	n/a	n/a
Actions (vs. predictable)	Low	Low	n/a	High	High	High	High	Low	n/a	Low	Low	n/a
Ideas (vs. closed-minded)	Low	n/a	High	n/a	n/a	n/a	n/a	n/a	n/a	Low	Low	Low
Values (vs. dogmatic)	Low	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	Low	n/a	n/a
Agreeableness (vs. antagonism)												
Trust (vs. mistrust)	Low	n/a	n/a	Low	n/a	High	Low	n/a	High	n/a	n/a	Low
Straightforwardness (vs. deception)	Low	n/a	n/a	Low	n/a	n/a	Low	n/a	n/a	n/a	Low	n/a
Altruism (vs. exploitative)	Low	n/a	n/a	Low	n/a	n/a	Low	n/a	High	n/a	n/a	n/a

Compliance (vs. aggression)	Low	n/a	n/a	Low	n/a	n/a	Low	n/a	High	n/a	Low	n/a
Modesty (vs. arrogance)	n/a	n/a	n/a	Low	n/a	n/a	Low	High	High	n/a	n/a	High
Tender-mindedness (vs. tough-minded)	Low	n/a	n/a	Low	n/a	n/a	Low	n/a	High	n/a	n/a	n/a
Conscientiousness (vs. disinhibition)												
Competence (vs. laxness)	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	High	Low	n/a
Order (vs. disorderly)	n/a	n/a	Low	n/a	n/a	n/a	n/a	n/a	n/a	n/a	High	Low
Dutifulness (vs. irresponsibility)	n/a	n/a	n/a	Low	n/a	n/a	n/a	n/a	n/a	High	Low	High
Achievement striving (vs. lackadaisical)	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	High	n/a	n/a
Self-discipline (vs. negligence)	n/a	n/a	n/a	Low	n/a	Low	n/a	n/a	n/a	High	Low	n/a
Deliberation (vs. rashness)	n/a	n/a	n/a	Low	Low	Low	n/a	n/a	n/a	High	n/a	High

Abbreviations used: *PPD – Paranoid Personality Disorder, SzPD – Schizoid Personality Disorder, StPD – Schizotypal Personality Disorder, ASPD – Antisocial Personality Disorder, BPD – Borderline Personality Disorder, HPD – Histrionic Personality Disorder, NPD – Narcissistic Personality Disorder, AvPD – Avoidant Personality Disorder, DPD – Dependent Personality Disorder, OCPD – Obsessive-Compulsive Personality Disorder, PAPD – Passive-Aggressive Personality Disorder, DpPD – Depressive Personality Disorder, n/a – not available.*

Causes

There are numerous possible causes of mental disorders, and they may vary depending on the disorder, the individual, and the circumstances. There may be genetic dispositions as well as particular life experiences, which may or may not include particular incidents of trauma or abuse.

A study of almost 600 male college students, averaging almost 30 years of age and who were not drawn from a clinical sample, examined the relationship between childhood experiences of sexual and physical abuse and currently reported personality disorder symptoms. Childhood abuse histories were found to be definitively associated with greater levels of symptomatology. Severity of abuse was found to be statistically significant, but clinically negligible, in symptomatology variance spread over Cluster A, B and C scales.

Child abuse and neglect consistently evidence themselves as antecedent risks to the development of personality disorders in adulthood. In the following study, efforts were taken to match retrospective reports of abuse with a clinical population that had demonstrated psychopathology from childhood to adulthood who were later found to have experienced abuse and neglect. In a study of 793 mothers and children, researchers asked mothers if they had screamed at their children, and told them that they did not love them or threatened to send them away. Children who had experienced such verbal abuse were three times as likely as other children (who did not experience such verbal abuse) to have borderline, narcissistic, obsessive-compulsive or paranoid personality disorders in adulthood. The sexually abused group demonstrated the most consistently elevated patterns of psychopathology. Officially verified physical abuse showed an extremely strong correlation with the development of antisocial and impulsive behavior. On the other hand, cases of abuse of the neglectful type that created childhood pathology were found to be subject to partial remission in adulthood.

Epidemiology

The prevalence of personality disorder in the general community was largely unknown until surveys starting from the 1990s. In 2008 the median rate of diagnosable PD was estimated at 10.6%, based on six major studies across three nations. This rate of around one in ten, especially as associated with high use of services, is described as a major public health concern requiring attention by researchers and clinicians.

The prevalence of individual personality disorders ranges from about 2% to 3% for the more common varieties, such as schizotypal, antisocial, borderline, and histrionic, to 0.5–1% for the least common, such as narcissistic and avoidant.

A screening survey across 13 countries by the World Health Organization using DSM-IV criteria, reported in 2009 a prevalence estimate of around 6% for personality disorders. The rate sometimes varied with demographic and socioeconomic factors, and functional impairment was partly explained by co-occurring mental disorders. In the US, screening data from the National Comorbidity Survey Replication between 2001 and 2003, combined with interviews of a subset of respondents, indicated a population prevalence of around 9% for personality disorders in total. Functional disability associated with the diagnoses appeared to be largely due to co-occurring mental disorders (Axis I in the DSM).

A UK national epidemiological study (based on DSM-IV screening criteria), reclassified into levels of severity rather than just diagnosis, reported in 2010 that the majority of people show some personality difficulties in one way or another (short of threshold for diagnosis), while the prevalence of the most complex and severe cases (including meeting criteria for multiple diagnoses in different clusters) was estimated at 1.3%. Even low levels of personality symptoms were associated with functional problems, but the most severely in need of services was a much smaller group.

There are also some sex differences in the frequency of personality disorders. They are shown in the table below.

Sex differences in the frequency of personality disorders	
Type of personality disorder	**Sex**
Paranoid personality disorder	**Male**
Schizoid personality disorder	**Male**
Schizotypal personality disorder	**Male**
Antisocial personality disorder	**Male**
Borderline personality disorder	**Female**
Histrionic personality disorder	**Female**
Narcissistic personality disorder	**Male**
Avoidant personality disorder	**Equal**
Dependent personality disorder	**Female**
Obsessive–compulsive personality disorder	**Male**

Comorbidity

There is a considerable personality disorder diagnostic co-occurrence. Patients who meet the DSM-IV-TR diagnostic criteria for one personality disorder are likely to meet the diagnostic criteria for another. Diagnostic categories provide clear, vivid descriptions of discrete personality types but the personality structure of actual patients might be more accurately described by a constellation of maladaptive personality traits.

DSM-III-R personality disorder diagnostic co-occurrence aggregated across six research sites											
Type of Personality Disorder	**PPD**	**SzPD**	**StPD**	**ASPD**	**BPD**	**HPD**	**NPD**	**AvPD**	**DPD**	**OCPD**	**PAPD**
Paranoid (PPD)		8	19	15	41	28	26	44	23	21	30
Schizoid (SzPD)	38		39	8	22	8	22	55	11	20	9
Schizotypal (StPD)	43	32		19	4	17	26	68	34	19	18
Antisocial (ASPD)	30	8	15		59	39	40	25	19	9	29
Borderline (BPD)	31	6	16	23		30	19	39	36	12	21
Histrionic (HPD)	29	2	7	17	41		40	21	28	13	25
Narcissistic (NPD)	41	12	18	25	38	60		32	24	21	38
Avoidant (AvPD)	33	15	22	11	39	16	15		43	16	19
Dependent (DPD)	26	3	16	16	48	24	14	57		15	22
Obsessive-Compulsive (OCPD)	31	10	11	4	25	21	19	37	27		23
Passive-Aggressive (PAPD)	39	6	12	25	44	36	39	41	34	23	

Sites used DSM-III-R criterion sets. Data obtained for purposes of informing the development of the DSM-IV-TR personality disorder diagnostic criteria.

Abbreviations used: *PPD – Paranoid Personality Disorder, SzPD – Schizoid Personality Disorder, StPD – Schizotypal Personality Disorder, ASPD – Antisocial Personality Disorder, BPD – Borderline Personality Disorder, HPD – Histrionic Personality Disorder, NPD – Narcissistic Personality Disorder, AvPD – Avoidant Personality Disorder, DPD – Dependent Personality Disorder, OCPD – Obsessive-Compulsive Personality Disorder, PAPD – Passive-Aggressive Personality Disorder.*

Management

Specific approaches

There are many different forms (modalities) of treatment used for personality disorders:[25]

- Individual psychotherapy has been a mainstay of treatment. There are long-term and short-term (brief) forms.
- Family therapy, including couples therapy.
- Group therapy for personality dysfunction is probably the second most used.
- Psychological-education may be used as an addition.
- Self-help groups may provide resources for personality disorders.
- Psychiatric medications for treating symptoms of personality dysfunction or co-occurring conditions.
- Milieu therapy, a kind of group-based residential approach, has a history of use in treating personality disorders, including therapeutic communities.

There are different specific theories or schools of therapy within many of these modalities. They may, for example, emphasize psychodynamic techniques, or cognitive or behavioral techniques. In clinical practice, many therapists use an 'eclectic' approach, taking elements of different schools as and when they seem to fit to an individual client. There is also often a focus on common themes that seem to be beneficial regardless of techniques, including

attributes of the therapist (e.g. trustworthiness, competence, caring), processes afforded to the client (e.g. ability to express and confide difficulties and emotions), and the match between the two (e.g. aiming for mutual respect, trust and boundaries).

Response of Patients with Personality Disorders to Biological and Psychosocial Treatments			
Cluster	**Evidence for Brain Dysfunction**	**Response to Biological Treatments**	**Response to Psychosocial Treatments**
A	Evidence for relationship of schizotypal personality to schizophrenia; otherwise none known	Schizotypal patients may improve on antipsychotic medication; otherwise not indicated	Poor. Supportive psychotherapy may help
B	Evidence suggestive for antisocial and borderline personalities; otherwise none known	Antidepressants, antipsychotics, or mood stabilizers may help for borderline personality; otherwise not indicated	Poor in antisocial personality. Variable in borderline, narcissistic, and histrionic personalities
C	None known	No direct response. Medications may help with comorbid anxiety and depression	Most common treatment for these disorders. Response variable

Challenges

The management and treatment of personality disorders can be a challenging and controversial area, for by definition the difficulties have been enduring and affect multiple areas of functioning. This often involves interpersonal issues, and there can be difficulties in seeking and obtaining help from organizations in the first place, as well as with establishing and maintaining a specific therapeutic relationship. On the one hand, an individual may not consider themselves to have a mental health problem, while on the other, community mental health services may view individuals with personality disorders as too complex or difficult, and may directly or indirectly exclude individuals with such diagnoses or associated behaviors. The disruptiveness people with personality disorders can create in an organisation makes these, arguably, the most challenging conditions to manage.

Apart from all these issues, an individual may not consider their personality to be disordered or the cause of problems. This perspective may be caused by the patient's ignorance or lack of insight into their own condition, an ego-syntonic perception of the problems with their personality that prevents them from experiencing it as being in conflict with their goals and self-image, or by the simple fact that there is no distinct or objective boundary between 'normal' and 'abnormal' personalities. Unfortunately, there is substantial social stigma and discrimination related to the diagnosis.

The term 'personality disorder' encompasses a wide range of issues, each with different a level of severity or disability; thus, personality disorders can require fundamentally different approaches and understandings. To illustrate the scope of the matter, consider that while some disorders or individuals are characterized by continual social withdrawal and the shunning of relationships, others may cause *fluctuations* in forwardness. The extremes are worse still: at one extreme lie self-harm and self-neglect, while at another extreme some individuals may commit violence and crime. There can be other factors such as problematic substance use or dependency or behavioral addictions. A person may meet criteria for multiple personality disorder diagnoses and/or other mental disorders, either at particular times or continually, thus making coordinated input from multiple services a potential requirement.

Therapists in this area can become disheartened by lack of initial progress, or by apparent progress that then leads to setbacks. Clients may be experienced as negative, rejecting, demanding, aggressive or manipulative. This has been looked at in terms of both therapist and client; in terms of social skills, coping efforts, defence mechanisms, or deliberate strategies; and in terms of moral judgements or the need to consider underlying motivations for specific behaviors or conflicts. The vulnerabilities of a client, and indeed therapist, may become lost behind actual or apparent strength and resilience. It is commonly stated that there is always a need to maintain appropriate professional personal boundaries, while allowing for emotional expression and therapeutic relationships. However,

there can be difficulty acknowledging the different worlds and understandings that client and therapist may live with. A therapist may assume that the kinds of relationships and ways of interacting that make them feel safe and comfortable, have the same effect on clients. As an example at one extreme, people who may in their lives have been used to hostility, deceptiveness, rejection, aggression or abuse, may in some cases be made confused, intimidated or suspicious by presentations of warmth, intimacy or positivity. On the other hand, reassurance, openness and clear communication are usually helpful and needed. It can take several months of sessions, and perhaps several stops and starts, to begin to develop a trusting relationship that can meaningfully address issues.[26]

History

Personality disorder is a term with a distinctly modern meaning, owing in part to its clinical usage and the institutional character of modern psychiatry. The currently accepted meaning must be understood in the context of historical changing classification systems such as DSM-IV and its predecessors. Although highly anachronistic, and ignoring radical differences in the character of subjectivity and social relations, some have suggested similarities to other concepts going back to at least the ancient Greeks. For example, the Greek philosopher Theophrastus described 29 'character' types that he saw as deviations from the norm, and similar views have been found in Asian, Arabic and Celtic cultures. A long-standing influence in the Western world was Galen's concept of personality types which he linked to the four humours proposed by Hippocrates.

Such views lasted into the 18th century, when experiments began to question the supposed biologically based humours and 'temperaments'. Psychological concepts of character and 'self' became widespread. In the 19th century, 'personality' referred to a person's conscious awareness of their behavior, a disorder of which could be linked to altered states such as dissociation. This sense of the term has been compared to the use of the term 'multiple personality disorder' in the first versions of the DSM.[27]

Physicians in the early 19th century started to diagnose forms of insanity that involved disturbed emotions and behaviors but seemingly without significant intellectual impairment or delusions or hallucinations. Philippe Pinel referred to this as 'manie sans délire' – insanity without delusion – and described a number of cases mainly involving excessive or inexplicable anger or rage. James Cowles Prichard advanced a similar concept he called moral insanity, which would be used to diagnose patients for some decades. 'Moral' in this sense referred to affect (emotion or mood) rather than necessarily ethics, but it was arguably based in part on religious, social and moral beliefs, with a pessimism about medical intervention so that social control should take precedence. These categories were much different and broader than later definitions of personality disorder, while also being developed by some into a more specific meaning of moral degeneracy akin to later ideas about 'psychopaths'. Separately, Richard von Krafft-Ebing popularized the terms sadism and masochism, as well as homosexuality, as psychiatric issues.

The German psychiatrist Koch sought to make the moral insanity concept more scientific, suggesting in 1891 the phrase 'psychopathic inferiority', theorized to be a congenital disorder. This referred to continual and rigid patterns of misconduct or dysfunction in the absence of apparent mental retardation or illness, supposedly without a moral judgement. Described as deeply rooted in his Christian faith, his work has been described as a fundamental text on personality disorders that is still of use today.

20th century

In the early 20th century, another German psychiatrist, Emil Kraepelin, included a chapter on psychopathic inferiority in his influential work on clinical psychiatry for students and physicians. He suggested six types – excitable, unstable, eccentric, liar, swindler and quarrelsome. The categories were essentially defined by the most disordered criminal offenders observed, distinguished between criminals by impulse, professional criminals, and morbid vagabonds who wandered through life. Kraepelin also described three paranoid (meaning then delusional) disorders, resembling later concepts of schizophrenia, delusional disorder and paranoid personality disorder. A diagnostic term for the latter concept would be included in the DSM from 1952, and from 1980 the DSM would also

include schizoid and schizotypal personality disorders; interpretations of earlier (1921) theories of Ernst Kretschmer led to a distinction between these and another type later included in the DSM, avoidant personality disorder.

In 1933 Russian psychiatrist Pyotr Borisovich Gannushkin published his book *Manifestations of psychopathies: statics, dynamics, systematic aspects*, which was one of the first attempts to develop a detailed typology of psychopathies. Regarding maladaptation, ubiquity, and stability as the three main symptoms of behavioral pathology, he distinguished 9 clusters of psychopaths: cycloids (including constitutionally depressive, constitutionally excitable, cyclothymics, and emotionally labile), asthenics (including psychasthenics), schizoids (including dreamers), paranoiacs (including fanatics), epileptoids, hysterical personalities (including pathological liars), unstable psychopaths, antisocial psychopaths, and constitutionally stupid.[28] Some elements of Gannushkin's typology were later incorporated into the theory developed by a Russian adolescent psychiatrist, Andrey Yevgenyevich Lichko, who was also interested in psychopathies along with their milder forms, the so-called accentuations of character.[29]

Psychiatrist David Henderson published in 1939 a theory of 'psychopathic states' which ended up contributing to the term becoming popularly linked to anti-social behavior. Hervey M. Cleckley's 1941 text, The Mask of Sanity, based on his personal categorization of similarities he noted in some prisoners, marked the start of the modern clinical conception of psychopathy and its popularist usage.

Towards the mid 20th century, psychoanalytic theories were coming to the fore based on work from the turn of the century being popularized by Sigmund Freud and others. This included the concept of 'character disorders', which were seen as enduring problems linked not to specific symptoms but to pervasive internal conflicts or derailments of normal childhood development. These were typically understood as weaknesses of character or willful deviance, and were distinguished from neurosis or psychosis. The term 'borderline' stems from a belief that some individuals were functioning on the edge of those two categories, and a number of the other personality disorder categories were also heavily influenced by this approach, including dependent, obsessive-compulsive and histrionic,[30] the latter starting off as a conversion symptom of hysteria particularly associated with women, then a hysterical personality, then renamed histrionic personality disorder in later versions of the DSM. A passive aggressive style was defined clinically by Colonel William Menninger during World War II in the context of men's reactions to military compliance, which would later be referenced as a personality disorder in the DSM. Otto Kernberg was influential with regard to the concepts of the borderline and narcissistic personalities which were later incorporated as disorders into the DSM in 1980.

Meanwhile, a more general personality psychology had been developing in academia and to some extent clinically. Gordon Allport was publishing theories of personality traits from the 1920s, and Henry Murray advanced a theory called 'personology' which influenced a later key advocate of personality disorders, Theodore Millon. Tests were developing or being applied for personality evaluation, including projective tests such as the Rorshach, as well as questionnaires such as the Minnesota Multiphasic Personality Inventory. Around mid-century, Hans Eysenck was analysing traits and personality types, and psychiatrist Kurt Schneider was popularising a clinical use in place of the previously more usual terms 'character', 'temperament' or 'constitution'.

American psychiatrists officially recognised concepts of enduring personality disturbances in the first Diagnostic and Statistical Manual of Mental Disorders in the 1950s, which relied heavily on psychoanalytic concepts. Somewhat more neutral language was employed in the DSM-II in 1968, though the terms and descriptions had only a slight resemblance to current definitions. The DSM-III published in 1980 made some major changes, notably putting all personality disorders onto a second separate 'axis' along with mental retardation, intended to signify more enduring patterns, distinct from what were considered axis one mental disorders. 'Inadequate' and 'asthenic' personality disorder' categories were deleted, and others were unpacked into more types, or changed from being personality disorders to regular disorders. Sociopathic personality disorder, which had been the term for psychopathy, was renamed Antisocial Personality Disorder. Most categories were given more specific 'operationalized' definitions, with standard criteria that psychiatrists could agree on in order to conduct research and diagnose patients.[31] In the DSM-III revision, self-defeating personality disorder and sadistic personality disorder were included as provisional

diagnoses requiring further study. They were dropped in the DSM-IV, though a proposed 'depressive personality disorder' was added; in addition, the official diagnosis of passive-aggressive personality disorder was dropped, tentatively renamed 'negativistic personality disorder.'

International differences have been noted in how attitudes have developed towards the diagnosis of personality disorder. Kurt Schneider had argued that they were simply 'abnormal varieties of psychic life' and therefore not necessarily the domain of psychiatry, a view said to still have influence in Germany today. British psychiatrists have also been reluctant to address such disorders or consider them on a par with other mental disorders, which has been attributed partly to resource pressures within the National Health Service, as well as to negative medical attitudes towards behaviors associated with personality disorders. In the US, the prevailing healthcare system and psychanalytic tradition has been said to provide a rationale for private therapists to diagnose some personality disorders more broadly and provide ongoing treatment for them.

Children

Early stages and preliminary forms of personality disorders need a multi-dimensional and early treatment approach. Personality development disorder is considered to be a childhood risk factor or early stage of a later personality disorder in adulthood. In addition, in Robert F. Krueger's review of their research indicates that some children and adolescents do suffer from clinically significant syndromes that resemble adult personal disorders, and that these syndromes have meaningful correlates and are consequential. Much of this research has been framed by the adult personality disorder constructs from Axis II of the Diagnostic and Statistical Manual. Hence, they are less likely to encounter the first risk they described at the outset of their review: clinicians and researchers are not simply avoiding use of the PD construct in youth. However, they may encounter the second risk they described: under-appreciation of the developmental context in which these syndromes occur. That is, although PD constructs show continuity over time, they are probabilistic predictors; not all youths who exhibit PD symptomatology become adult PD cases.

Five Factor Model

As of 2002, there were over fifty published studies relating the Five Factor Model (FFM) to personality disorders.[32] Since that time, quite a number of additional studies have expanded on this research base and provided further empirical support for understanding the DSM personality disorders in terms of the FFM domains.[33]

In her seminal review of the personality disorder literature published in 2007, Dr. Lee Anna Clark asserted that "the five-factor model of personality is widely accepted as representing the higher-order structure of both normal and abnormal personality traits".[34]

The Five Factor Model has been shown to significantly predict all ten personality disorder symptoms and outperform the Minnesota Multiphasic Personality Inventory (MMPI) in the prediction of borderline, avoidant, and dependent personality disorder symptoms.[35]

Research results examining the relationships between the FFM and each of the ten DSM personality disorder diagnostic categories are widely available. For example, in a study published in 2003 titled "The five-factor model and personality disorder empirical literature: A meta-analytic review",[36] the authors analyzed data from 15 other studies to determine how personality disorders are different and similar, respectively, with regard to underlying personality traits. In terms of how personality disorders differ, the results showed that each disorder displays a FFM profile that is meaningful and predictable given its unique diagnostic criteria. With regard to their similarities, the findings revealed that the most prominent and consistent personality dimensions underlying a large number of the personality disorders are positive associations with neuroticism and negative associations with agreeableness.

Openness to experience

At least three aspects of openness to experience are relevant to understanding personality disorders: cognitive distortions, lack of insight and impulsivity. Problems related to high openness that can cause problems with social or professional functioning are excessive fantasising, peculiar thinking, diffuse identity, unstable goals and nonconformity with the demands of the society.

High openness is characteristic to schizotypal personality disorder (odd and fragmented thinking), narcissistic personality disorder (excessive self-valuation) and paranoid personality disorder (sensitivity to external hostility). Lack of insight (shows low openness) is characteristic to all personality disorders and could explain the persistence of maladaptive behavioral patterns.

The problems associated with low openness are difficulties adapting to change, low tolerance for different worldview or lifestyles, emotional flattening, alexithymia and a narrow range of interests. Rigidity is the most obvious aspect of (low) openness among personality disorders and that shows lack of knowledge of one's emotional experiences. It is most characteristic of obsessive-compulsive personality disorder, the opposite of it known as impulsivity (here: an aspect of openness that shows a tendency to behave unusually or autistically) is characteristic of schizotypal and borderline personality disorders.

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External links

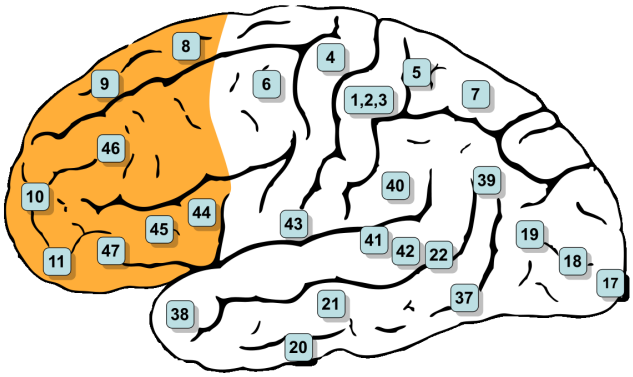
Library resources about **Personality disorders**
• Resources in your library (http://tools.wmflabs.org/ftl/cgi-bin/ftl?st=wp&su=Personality+disorder)

- The Institute for Advanced Studies in Personology and Psychopathology (http://www.millon.net/) the official website for Theodore Millon, Ph.D., D.Sc.
- Personality Disorders Foundation (http://counsellingresource.com/distress/personality-disorders/foundation/index.html)
- National Personality Disorder website for England (http://personalitydisorder.org.uk/)
- Dangerous severe personality disorder (http://apt.rcpsych.org/cgi/content/full/9/5/349?ck=nck)
- National Mental Health Association Personality Disorder Fact Sheet (http://www.nmha.org/go/information/get-info/personality-disorders)

- Personality Disorders information leaflet from The Royal College of Psychiatrists (http://www.rcpsych.ac.uk/expertadvice/problemsdisorders/personalitydisorder.aspx)

Prefrontal cortex

Brain: Prefrontal cortex



Brodmann areas #9, #10, #11, #12, #46, and #47 are all in the prefrontal cortex.

Latin	*Cortex praefrontalis*
Part of	Frontal lobe
Components	Superior frontal gyrus Middle frontal gyrus Inferior frontal gyrus
Artery	Anterior cerebral Middle cerebral
Vein	Superior sagittal sinus
NeuroNames	*ancil-101* [1]
MeSH	*Prefrontal+Cortex* [2]

The **prefrontal cortex**, also called **PFC**, refers to the anterior part of the frontal lobes of the brain, lying in front of the motor and premotor areas. PFC contains Brodmann's areas 9, 10, 11, 12, 46, and 47.

This brain region has been implicated in planning complex cognitive behavior, personality expression, decision making, and moderating social behavior. The basic activity of this brain region is considered to be orchestration of thoughts and actions in accordance with internal goals. Destruction of the anterior two-thirds results in deficits in concentration, orientation, abstracting ability, judgment, and problem solving ability; destruction of the orbital (frontal) lobe results in inappropriate social behavior.

The most typical psychological term for functions carried out by the prefrontal cortex area is executive function. Executive function relates to abilities to differentiate among conflicting thoughts, determine good and bad, better and best, same and different, future consequences of current activities, working toward a defined goal, prediction of outcomes, expectation based on actions, and social "control" (the ability to suppress urges that, if not suppressed, could lead to socially unacceptable outcomes).

Many authors have indicated an integral link between a person's personality and the functions of the prefrontal cortex.

Structure

Definition

There are three possible ways to define the prefrontal cortex:

- as the granular frontal cortex
- as the projection zone of the [mediodorsal nucleus] of the thalamus
- as that part of the frontal cortex whose electrical stimulation does not evoke movements

The prefrontal cortex has been defined based on cytoarchitectonics by the presence of a cortical granular layer IV. It is not entirely clear who first used this criterion. Many of the early cytoarchitectonic researchers restricted the use of the term prefrontal to a much smaller region of cortex including the gyrus rectus and the gyrus rostralis (Campbell, 1905; G. E. Smith, 1907; Brodmann, 1909; von Economo and Koskinas, 1925). In 1935, however, Jacobsen used the term prefrontal to distinguish granular prefrontal areas from agranular motor and premotor areas. In terms of Brodmann areas, the prefrontal cortex traditionally includes areas 8, 9, 10, 11, 44, 45, 46, and 47 (to complicate matters, not all of these areas are strictly granular—44 is dysgranular, caudal 11 and orbital 47 are agranular). The main problem with this definition is that it works well only in primates but not in nonprimates, as the latter lack a granular layer IV.

To define the prefrontal cortex as the projection zone of the mediodorsal nucleus of the thalamus builds on the work of Rose and Woolsey who showed that this nucleus projects to anterior and ventral parts of the brain in nonprimates. Rose and Woolsey however termed this projection zone "orbitofrontal." It seems to have been Akert, who in 1964 for the first time explicitly suggested that this criterion could be used to define homologues of the prefrontal cortex in primates and nonprimates. This allowed the establishment of homologies despite the lack of a granular frontal cortex in nonprimates. The projection zone definition is still widely accepted today (e.g. Fuster), although its usefulness has been questioned. Modern tract tracing studies have shown that projections of the mediodorsal nucleus of the thalamus are not restricted to the granular frontal cortex in primates. As a result, it was suggested to define the prefrontal cortex as the region of cortex that has stronger reciprocal connections with the mediodorsal nucleus than with any other thalamic nucleus. Uylings et al. acknowledge, however, that even with the application of this criterion it might be rather difficult to unequivocally define the prefrontal cortex.

A third definition of the prefrontal cortex is the area of frontal cortex whose electrical stimulation does not lead to observable movements. For example, in 1890 David Ferrier used the term in this sense. One complication with this definition is that the electrically "silent" frontal cortex includes both granular and non-granular areas.

Subdivisions

The table below shows different ways to subdivide the prefrontal cortex starting from Brodmann areas. Note that the term "dorsolateral" has been used to denote areas 8, 9, and 46 as well as areas 8, 9, 44, 45, 46, and lateral 47[*citation needed*] and several terms are given to areas 47, 11 and 10.

<table>
<tr><th>8</th><th>9</th><th>46</th><th>44</th><th>45</th><th>lateral 47</th><th>orbital 47</th><th>11</th><th>10</th></tr>
<tr><td colspan="6">dorsolateral</td><td colspan="2" rowspan="3">orbitofrontal, ventromedial, basal, orbital</td><td rowspan="3">frontopolar, anterior, rostral</td></tr>
<tr><td colspan="4">dorsolateral</td><td colspan="2" rowspan="2">ventrolateral</td></tr>
<tr><td colspan="2">posterior dorsolateral</td><td colspan="2">mid-dorsolateral</td></tr>
</table>

Interconnections

The prefrontal cortex is highly interconnected with much of the brain, including extensive connections with other cortical, subcortical and brain stem sites. The dorsal prefrontal cortex is especially interconnected with brain regions involved with attention, cognition and action, while the ventral prefrontal cortex interconnects with brain regions involved with emotion. The prefrontal cortex also receives inputs from the brainstem arousal systems, and its function is particularly dependent on its neurochemical environment. Thus, there is coordination between our state of arousal and our mental state.

The medial prefrontal cortex has been implicated in the generation of slow-wave sleep (SWS), and prefrontal atrophy has been linked to decreases in SWS. Prefrontal atrophy occurs naturally as individuals age, and it has been demonstrated that older adults experience impairments in memory consolidation as their medial prefrontal cortices degrade. Significant atrophy can also occur as a result of neuroleptic or antipsychotic psychiatric medication. In older adults, instead of being transferred and stored in the neocortex during SWS, memories start to remain in the hippocampus where they were encoded, as evidenced by increased hippocampal activation compared to younger adults during recall tasks when subjects learned word associations, slept, and then were asked to recall the learned words.

Function

Executive function

The original studies of Fuster and of Goldman-Rakic emphasized the fundamental ability of the prefrontal cortex to represent information not currently in the environment, and the central role of this function in creating the "mental sketch pad". Goldman-Rakic spoke of how this representational knowledge was used to intelligently guide thought, action, and emotion, including the inhibition of inappropriate thoughts, distractions, actions, and feelings. In this way, working memory can be seen as fundamental to attention and behavioral inhibition. Fuster speaks of how this prefrontal ability allows the wedding of past to future, allowing both cross-temporal and cross-modal associations in the creation of goal-directed, perception-action cycles. This ability to represent underlies all other higher executive functions.

Shimamura proposed Dynamic Filtering Theory to describe the role of the prefrontal cortex in executive functions. The prefrontal cortex is presumed to act as a high-level gating or filtering mechanism that enhances goal-directed activations and inhibits irrelevant activations. This filtering mechanism enables executive control at various levels of processing, including selecting, maintaining, updating, and rerouting activations. It has also been used to explain emotional regulation.

Miller and Cohen proposed an Integrative Theory of Prefrontal Cortex Function, that arises from the original work of Goldman-Rakic and Fuster. The two theorize that “cognitive control stems from the active maintenance of patterns of activity in the prefrontal cortex that represents goals and means to achieve them. They provide bias signals to other brain structures whose net effect is to guide the flow of activity along neural pathways that establish the proper mappings between inputs, internal states, and outputs needed to perform a given task”. In essence, the two theorize that the prefrontal cortex guides the inputs and connections, which allows for cognitive control of our actions.

The prefrontal cortex is of significant importance when top-down processing is needed. Top-down processing by definition is when behavior is guided by internal states or intentions. According to the two, “The PFC is critical in situations when the mappings between sensory inputs, thoughts, and actions either are weakly established relative to other existing ones or are rapidly changing”. An example of this can be portrayed in the Wisconsin Card Sorting Test (WCST). Subjects engaging in this task are instructed to sort cards according to the shape, color, or number of symbols appearing on them. The thought is that any given card can be associated with a number of actions and no single stimulus-response mapping will work. Human subjects with PFC damage are able to sort the card in the initial simple tasks, but unable to do so as the rules of classification change.

Miller and Cohen conclude that the implications of their theory can explain how much of a role the PFC has in guiding control of cognitive actions. In the researchers' own words, they claim that, “depending on their target of influence, representations in the PFC can function variously as attentional templates, rules, or goals by providing top-down bias signals to other parts of the brain that guide the flow of activity along the pathways needed to perform a task”.

Experimental data indicate a role for the prefrontal cortex in mediating normal sleep physiology, dreaming and sleep-deprivation phenomena.[3]

When analyzing and thinking about attributes of other individuals, the medial prefrontal cortex is activated. However, it is not activated when contemplating about the characteristics of inanimate objects.[4]

As of recent, researchers have used neuroimaging techniques to find that along with the basal ganglia, the prefrontal cortex is involved with learning exemplars, which is part of the *exemplar theory*, one of the three main ways our mind categorizes things. The exemplar theory states that we categorize judgements by comparing it to a similar past experience within our stored memories.[5]

Clinical significance

In the last few decades, brain imaging systems have been used to determine brain region volumes and nerve linkages. Several studies have indicated that reduced volume and interconnections of the frontal lobes with other brain regions is observed in patients diagnosed with mental disorders and prescribed potent antipsychotics; those subjected to repeated stressors; suicide victims; those incarcerated; criminals; sociopaths; those affected by lead poisoning; It is believed that at least some of the human abilities to feel guilt or remorse, and to interpret reality, are dependent on a well-functioning prefrontal cortex. It is also widely believed that the size and number of connections in the prefrontal cortex relates directly to sentience, as the prefrontal cortex in humans occupies a far larger percentage of the brain than any other animal. And it is theorized that, as the brain has tripled in size over 5 million years of human evolution, the prefrontal cortex has increased in size sixfold.

History

Perhaps the seminal case in prefrontal cortex function is that of Phineas Gage, whose left frontal lobe was destroyed when a large iron rod was driven through his head in an 1848 accident. The standard presentation (e.g.[6]) is that, although Gage retained normal memory, speech and motor skills, his personality changed radically: He became irritable, quick-tempered, and impatient—characteristics he did not previously display — so that friends described him as "no longer Gage"; and, whereas he had previously been a capable and efficient worker, afterward he was unable to complete tasks. However, careful analysis of primary evidence shows that descriptions of Gage's psychological changes are usually exaggerated when held against the description given by Gage's doctor, the most striking feature being that changes described years after Gage's death are far more dramatic than anything reported while he was alive.[7]

Subsequent studies on patients with prefrontal injuries have shown that the patients verbalized what the most appropriate social responses would be under certain circumstances. Yet, when actually performing, they instead

pursued behavior aimed at immediate gratification, despite knowing the longer-term results would be self-defeating.

The interpretation of this data indicates that not only are skills of comparison and understanding of eventual outcomes harbored in the prefrontal cortex but the prefrontal cortex (when functioning correctly) controls the mental option to delay immediate gratification for a better or more rewarding longer-term gratification result. This ability to wait for a reward is one of the key pieces that define optimal executive function of the human brain.

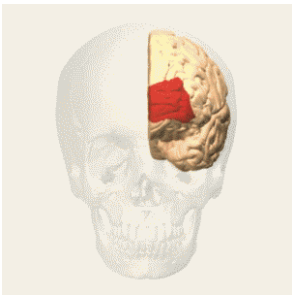
There is much current research devoted to understanding the role of the prefrontal cortex in neurological disorders. Many disorders, such as schizophrenia, bipolar disorder, and ADHD, have been related to dysfunction of the prefrontal cortex, and thus this area of the brain offers the potential for new treatments of these conditions.[*citation needed*] Clinical trials have begun on certain drugs that have been shown to improve prefrontal cortex function, including guanfacine, which acts through the alpha-2A adrenergic receptor. A downstream target of this drug, the HCN channel, is one of the most recent areas of exploration in prefrontal cortex pharmacology.

Etymology

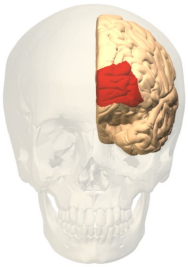
The term "prefrontal" as describing a part of the brain appears to have been introduced by Richard Owen in 1868. For him, the prefrontal area was restricted to the anterior-most part of the frontal lobe (approximately corresponding to the frontal pole). It has been hypothesized that his choice of the term was based on the prefrontal bone present in most amphibians and reptiles.

Additional images

These images of Brodmann area 10 give a clearer idea of the location of one particular sub-region of the prefrontal cortex.

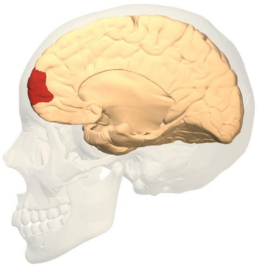


Animation.



front view.

Lateral view.



Medial view.

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[5] Schacter, Daniel L., Daniel Todd Gilbert, and Daniel M. Wegner. Psychology. 2nd ed, pages 364-366 New York, NY: Worth Publishers, 2011. Print.

[6] Antonio Damasio, *Descartes' Error*. Penguin Putman Pub., 1994

[7] Malcolm Macmillan, *An Odd Kind of Fame: Stories of Phineas Gage* (MIT Press, 2000), pp.116-119, 307-333, esp. pp.11,333.

External links

- Diagram (ua.edu) (http://bama.ua.edu/~sprentic/672 aggression-brain.jpg)
- Diagram (universe-review.ca) (http://universe-review.ca/I10-80-prefrontal.jpg)
- Stained brain slice images which include the "Prefrontal cortex" (http://brainmaps.org/index.php?q=Prefrontal cortex) at the BrainMaps project

Psychological manipulation

Psychological manipulation is a type of social influence that aims to change the perception or behavior of others through underhanded, deceptive, or even abusive tactics. By advancing the interests of the manipulator, often at another's expense, such methods could be considered exploitative, abusive, devious, and deceptive. Social influence is not necessarily negative. For example, doctors can try to persuade patients to change unhealthy habits. Social influence is generally perceived to be harmless when it respects the right of the influenced to accept or reject and is not unduly coercive. Depending on the context and motivations, social influence may constitute underhanded manipulation.

Requirements for successful manipulation

According to psychology author George K. Simon, successful psychological manipulation primarily involves the manipulator:

1. concealing aggressive intentions and behaviors.
2. knowing the psychological vulnerabilities of the victim to determine what tactics are likely to be the most effective.
3. having a sufficient level of ruthlessness to have no qualms about causing harm to the victim if necessary.

Consequently, the manipulation is likely to be accomplished through covert aggressive (relational aggressive or passive aggressive) means.[1]

How manipulators control their victims

According to Braiker

Clinical psychologist Harriet Braiker wrote a self help book which identified the following basic ways that manipulators control their victims:

- Positive reinforcement: includes praise, superficial charm, superficial sympathy (crocodile tears), excessive apologizing, money, approval, gifts, attention, facial expressions such as a forced laugh or smile, and public recognition.
- Negative reinforcement: involves removing one from a negative situation as a reward, e.g. "You won't have to do your homework if you allow me to do this to you."
- Intermittent or partial reinforcement: Partial or intermittent negative reinforcement can create an effective climate of fear and doubt. Partial or intermittent positive reinforcement can encourage the victim to persist - for example in most forms of gambling, the gambler is likely to win now and again but still lose money overall.
- Punishment: includes nagging, yelling, the silent treatment, intimidation, threats, swearing, emotional blackmail, the guilt trip, sulking, crying, and playing the victim.
- Traumatic one-trial learning: using verbal abuse, explosive anger, or other intimidating behavior to establish dominance or superiority; even one incident of such behavior can condition or train victims to avoid upsetting, confronting or contradicting the manipulator.

According to Simon

Simon identified the following manipulative techniques:

- Lying: It is hard to tell if somebody is lying at the time they do it, although often the truth may be apparent later when it is too late. One way to minimize the chances of being lied to is to understand that some personality types (particularly psychopaths) are experts at the art of lying and cheating, doing it frequently, and often in subtle ways.
- Lying by omission: This is a very subtle form of lying by withholding a significant amount of the truth. This technique is also used in propaganda.
- Denial: Manipulator refuses to admit that he or she has done something wrong.
- Rationalization: An excuse made by the manipulator for inappropriate behavior. Rationalization is closely related to spin.
- Minimization: This is a type of denial coupled with rationalization. The manipulator asserts that his or her behavior is not as harmful or irresponsible as someone else was suggesting, for example, saying that a taunt or insult was only a joke.
- Selective inattention or selective attention: Manipulator refuses to pay attention to anything that may distract from his or her agenda, saying things like "I don't want to hear it".
- Diversion: Manipulator not giving a straight answer to a straight question and instead being diversionary, steering the conversation onto another topic.
- Evasion: Similar to diversion but giving irrelevant, rambling, vague responses, weasel words.
- Covert intimidation: Manipulator throwing the victim onto the defensive by using veiled (subtle, indirect or implied) threats.
- Guilt trip: A special kind of intimidation tactic. A manipulator suggests to the conscientious victim that he or she does not care enough, is too selfish or has it easy. This usually results in the victim feeling bad, keeping them in a self-doubting, anxious and submissive position.
- Shaming: Manipulator uses sarcasm and put-downs to increase fear and self-doubt in the victim. Manipulators use this tactic to make others feel unworthy and therefore defer to them. Shaming tactics can be very subtle such as a fierce look or glance, unpleasant tone of voice, rhetorical comments, subtle sarcasm. Manipulators can make one feel ashamed for even daring to challenge them. It is an effective way to foster a sense of inadequacy in the victim.
- Playing the victim role: Manipulator portrays him- or herself as a victim of circumstance or of someone else's behavior in order to gain pity, sympathy or evoke compassion and thereby get something from another. Caring and conscientious people cannot stand to see anyone suffering and the manipulator often finds it easy to play on sympathy to get cooperation.
- Vilifying the victim: More than any other, this tactic is a powerful means of putting the victim on the defensive while simultaneously masking the aggressive intent of the manipulator.
- Playing the servant role: Cloaking a self-serving agenda in guise of a service to a more noble cause, for example saying he is acting in a certain way to be "obedient" to or in "service" to an authority figure or "just doing their job".
- Seduction: Manipulator uses charm, praise, flattery or overtly supporting others in order to get them to lower their defenses and give their trust and loyalty to him or her.
- Projecting the blame (blaming others): Manipulator scapegoats in often subtle, hard-to-detect ways.
- Feigning innocence: Manipulator tries to suggest that any harm done was unintentional or that they did not do something that they were accused of. Manipulator may put on a look of surprise or indignation. This tactic makes the victim question his or her own judgment and possibly his own sanity.

- Feigning confusion: Manipulator tries to play dumb by pretending he or she does not know what the victim is talking about or is confused about an important issue brought to his or her attention.
- Brandishing anger: Manipulator uses anger to brandish sufficient emotional intensity and rage to shock the victim into submission. The manipulator is not actually angry, he or she just puts on an act. He just wants what he wants and gets "angry" when denied.

Vulnerabilities exploited by manipulators

According to Braiker's self-help book, manipulators exploit the following vulnerabilities (buttons) that may exist in victims:

- the "disease to please"
- addiction to earning the approval and acceptance of others
- Emotophobia (fear of negative emotion; *i.e.* a fear of expressing anger, frustration or disapproval)
- lack of assertiveness and ability to say no
- blurry sense of identity (with soft personal boundaries)
- low self-reliance
- external locus of control

According to Simon, manipulators exploit the following vulnerabilities that may exist in victims:

- naïveté - victim finds it too hard to accept the idea that some people are cunning, devious and ruthless or is "in denial" if he or she is being victimized.
- over-conscientiousness - victim is too willing to give manipulator the benefit of the doubt and see their side of things in which they blame the victim.
- low self-confidence - victim is self-doubting, lacking in confidence and assertiveness, likely to go on the defensive too easily.
- over-intellectualization - victim tries too hard to understand and believes the manipulator has some understandable reason to be hurtful.
- emotional dependency - victim has a submissive or dependent personality. The more emotionally dependent the victim is, the more vulnerable he or she is to being exploited and manipulated.

Manipulators generally take the time to scope out the characteristics and vulnerabilities of their victim.

Kantor advises in his book, the following are vulnerable to psychopathic manipulators:

- too dependent - dependent people need to be loved and are therefore gullible and liable to say yes to something to which they should say no.
- too immature - has impaired judgment and believes the exaggerated advertising claims.
- too naïve - cannot believe there are dishonest people in the world, taking for granted that if there were they would not be allowed to operate.
- too impressionable - overly seduced by charmers. For example, they might vote for the seemingly charming politician who kisses babies.
- too trusting - people who are honest often assume that everyone else is honest. They are more likely to commit themselves to people they hardly know without checking credentials, etc., and less likely to question so-called experts.
- too lonely - lonely people may accept any offer of human contact. A psychopathic stranger may offer human companionship for a price.
- too narcissistic - narcissists are prone to falling for unmerited flattery.
- too impulsive - make snap decisions about, for example, what to buy or whom to marry without consulting others.
- too altruistic - the opposite of psychopathic: too honest, too fair, too empathetic.
- too frugal - cannot say no to a bargain even if they know the reason it is so cheap.

- too materialistic - easy prey for loan sharks or get-rich-quick schemes.
- too greedy - the greedy and dishonest may fall prey to a psychopath who can easily entice them to act in an immoral way.
- too masochistic - lack self-respect and so unconsciously let psychopaths take advantage of them. They think they deserve it out of a sense of guilt.
- the elderly - the elderly can become fatigued and less capable of multi-tasking. When hearing a sales pitch they are less likely to consider that it could be a con. They are prone to giving money to someone with a hard-luck story. See elder abuse.

Motivations of manipulators

Manipulators can have various possible motivations, including but not limited to:

- the need to advance their own purposes and personal gain at virtually any cost to others
- a strong need to attain feelings of power and superiority in relationships with others
- a want and need to feel in control (aka. control freak)
- a desire to gain a feeling of power over others in order to raise their perception of self-esteem
- boredom, or growing tired of his/her surroundings, seeing it as a game more than hurting others

Psychological conditions of manipulators

Manipulators may have any of the following psychological conditions:

- machiavellian personality
- narcissistic personality disorder
- paranoid personality disorder
- borderline personality disorder
- dependent personality disorder
- histrionic personality disorder
- passive–aggressive behavior
- type A angry personalities
- antisocial personality disorder
- behavioral addiction

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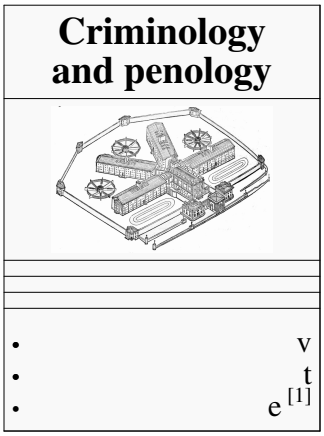
Psychopathy



Psychopathy (/saɪˈkɒpəθi/) (or **sociopathy** /ˈsoʊsiəˌpæθi/) is traditionally defined as a personality disorder characterized by enduring antisocial behavior, diminished empathy and remorse, and disinhibited or bold behavior. It may also be defined as a continuous aspect of personality, representing scores on different personality dimensions found throughout the population in varying combinations. The definition of psychopathy has varied significantly throughout the history of the concept; different definitions continue to be used that are only partly overlapping and sometimes appear contradictory.

American psychiatrist Hervey M. Cleckley's work on psychopathy probably influenced the initial diagnostic criteria for antisocial personality reaction/disturbance in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM), as did American psychologist George E. Partridge's work on sociopathy.[*citation needed*] The DSM and *International Classification of Diseases* (ICD) subsequently introduced the diagnoses of antisocial personality disorder (ASPD) and dissocial personality disorder, stating that these have been referred to (or include what is referred to) as psychopathy or sociopathy. Canadian psychologist Robert D. Hare later repopularised the construct of psychopathy in criminology with his Psychopathy Checklist.

Although no psychiatric or psychological organization has sanctioned a diagnosis titled "psychopathy", assessments of psychopathy characteristics are widely used in criminal justice settings in some nations, and may have important consequences for individuals. The term is also used by the general public, in popular press, and in fictional portrayals.

Definition

Conceptions

Researchers have noted that there appear to be at least two different conceptions of psychopathy, each with differing policy implications. Jennifer L. Skeem et al. distinguished *Cleckleyan psychopathy* (named after Hervey Cleckley's early conception of psychopathy, entailing bold, disinhibited behavior, low anxiety and "feckless disregard") and *criminal psychopathy* (a "meaner, more aggressively disinhibited conception of psychopathy that explicitly entails persistent and sometimes serious criminal behavior", typically operationalized with the Hare Psychopathy Checklist). Due to the profound implications that a label of "psychopath" can have—including in terms of decisions about punishment severity, treatment, etc.—efforts have been made to clarify the meaning of the term, e.g. by reconciling seemingly disparate conceptions such as those mentioned.

Triarchic model

The triarchic model, formulated by Christopher J. Patrick et al., suggests that different conceptions of psychopathy emphasize three observable characteristics to varying degrees. Analyses have been made with respect to the applicability of measurement tools such as the Hare Psychopathy Checklist (PCL, PCL-R) and Psychopathic Personality Inventory (PPI) to this model.

- **Boldness**. Low fear including stress-tolerance, toleration of unfamiliarity and danger, and high self-confidence and social assertiveness. The PCL-R measures this relatively poorly and mainly through Facet 1 of Factor 1. Similar to PPI Fearless dominance. May correspond to differences in the amygdala and other neurological systems associated with fear.
- **Disinhibition**. Poor impulse control including problems with planning and foresight, lacking affect and urge control, demand for immediate gratification, and poor behavioral restraints. Similar to PCL-R Factor 2 and PPI Impulsive antisociality. May correspond to impairments in frontal lobe systems that are involved in such control.
- **Meanness**. Lacking empathy and close attachments with others, disdain of close attachments, use of cruelty to gain empowerment, exploitative tendencies, defiance of authority, and destructive excitement seeking. The PCL-R in general is related to this but in particular some elements in Factor 1. Similar to PPI Coldheartedness but also includes elements of subscales in Impulsive antisociality. Meanness may possibly be caused by either high boldness or high disinhibition combined with an adverse environment. Thus, a child with high boldness may respond poorly to punishment but may respond better to rewards and secure attachments which may not be available under adverse conditions. A child with high disinhibition may have increased problems under adverse conditions with meanness developing in response.

Measurement

A crucial issue regarding the concept of psychopathy is whether it identifies a distinct condition that can be separated from other conditions and "normal" personality types, or whether it is simply a combination of scores on various dimensions of personality found throughout the population in varying combinations.[*citation needed*]

An early and influential analysis from Harris and colleagues indicated a discrete category may underlie PCL-R psychopathy, but this was only found for the behavioral Factor 2 items. Marcus, John, and Edens more recently performed a series of statistical analysis on PPI scores and concluded psychopathy may best be conceptualized as having a "dimensional latent structure" like depression.

Marcus et al. repeated the study on a larger sample of prisoners, using the PCL-R and seeking to rule out other experimental or statistical issues that may have produced the previously different findings. They again found that the psychopathy measurements do not appear to be identifying a discrete type (a taxon). They suggest that while for legal or other practical purposes an arbitrary cut-off point on trait scores might be used, there is actually no clear scientific evidence for an objective point of difference by which to call some people "psychopaths"; in other words, a "psychopath" may be more accurately described as someone who is "relatively psychopathic".[2] The PCL-R was developed for research, not clinical forensic diagnosis, and even for research purposes to improve understanding of the underlying issues, it is necessary to examine dimensions of personality in general rather than only this constellation of traits.

Personality dimensions

Psychopathy represents a cluster of different dimensions of personality found throughout the general population to varying degrees. There are different views as to which dimensions are more central in regard to psychopathy.

Besides dimensions described elsewhere in this article, some studies have linked psychopathy to alternative dimensions, such as antagonism (high), conscientiousness (low) and anxiousness (low, or sometimes high). Psychopathy has also been linked to high psychoticism—a theorized dimension referring to tough, aggressive or hostile tendencies. Aspects of this that appear associated with psychopathy are lack of socialization and responsibility, impulsivity, sensation-seeking in some cases, and aggression.

Otto Kernberg, from a particular psychoanalytic perspective, believes psychopathy should be considered as part of a spectrum of pathological narcissism, that would range from narcissistic personality on the low end, malignant narcissism in the middle, and psychopathy at the high end. However, narcissism is generally seen as only one possible aspect of psychopathy as generally defined.[*citation needed*]

Signs and symptoms

Offending

Criminality

In terms of simple correlations, the PCL-R manual states an average score of 22.1 has been found in North American prisoner samples, and that 20.5% scored 30 or higher. An analysis of prisoner samples from outside North America found a somewhat lower average value of 17.5. Studies have found that psychopathy scores correlated with repeated imprisonment, detention in higher security, disciplinary infractions, and substance misuse.

Psychopathy, as measured with the PCL-R in institutional settings, show in meta-analyses small to moderate effect sizes with institutional misbehavior, postrelease crime, or postrelease violent crime with similar effects for the three outcomes. Individual studies give similar results for adult offenders, forensic psychiatric samples, community samples, and youth. The PCL-R is poorer at predicting sexual re-offending. However, this small to moderate effect appears to be due largely to the scale items that assess impulsive behaviors and past criminal history, which are well-established but very general risk factors. The aspects of core personality often held to be distinctively psychopathic, generally show little or no predictive link to crime by themselves. Thus Factor 1 of the PCL-R and Fearless dominance of the PPI-R have smaller or no relationship to crime, including violent crime. In contrast Factor 2 and Impulsive antisociality of the PPI-R are associated more strongly with criminality. Factor 2 has a relationship of similar strength to that of the PCL-R as a whole. The antisocial facet of the PCL-R is still predictive of future violence after controlling for past criminal behavior which, together with results regarding the PPI-R which by design does not include past criminal behavior, suggests that impulsive behaviors is an independent risk factor. The concept of psychopathy may perform poorly when attempted to be used as a general theory of crime.[3]

Violence

Studies have suggested a strong correlation between psychopathy scores and violence, and the PCL-R emphasizes features that are somewhat predictive of violent behavior. Additionally, the Federal Bureau of Investigation reports that psychopathic behavior is consistent with traits common to some serial killers, including sensation seeking, a lack of remorse or guilt, impulsivity, the need for control, and predatory behavior. Researchers, however, have noted that psychopathy is dissociable from and not synonymous with violence.

It has been suggested that psychopaths tend to commit more "instrumental" violence than "reactive" violence. One conclusion in this regard was made by a 2002 study of homicide offenders, which reported that the homicides committed by psychopaths were almost always (93.3%) primarily instrumental, while about half (48.4%) of those committed by non-psychopaths were. However, contrary to the equating of this to mean "in cold blood", more than a

third of the homicides by psychopaths involved emotional reactivity as well. In addition, the non-psychopaths still accounted for most of the instrumental homicides, because most of these murderers were not psychopaths. In any case, FBI profilers indicate that serious victim injury is generally an emotional offense, and some research supports this, at least with regard to sexual offending. One study has found more serious offending by non-psychopaths on average than by psychopaths (e.g. more homicides versus more armed robbery and property offenses) and another that the Affective facet of PCL-R predicted reduced offense seriousness.

Although psychopathy is associated with an increased risk of violence, it is difficult to know how to manage the risk. Some clinicians suggest that assessment of the construct of psychopathy does not necessarily add value to violence risk assessment. A large systematic review and meta-regression found that the PCL performed the poorest out of nine tools for predicting violence.[4][5] In addition, studies conducted by the authors or translators of violence prediction measures, including the PCL, show on average more positive results than those conducted by more independent investigators.[6] There are several other risk assessment instruments which can predict further crime with an accuracy similar to the PCL-R and some of these are considerably easier, quicker, and less expensive to administrate. This may even be done automatically by a computer simply based on data such as age, gender, number of previous convictions, and age of first conviction. Some of these assessments may also identify treatment change and goals, identify quick changes that may help short-term management, identify more specific kinds of violence that may be at risk, and may have established specific probabilities of offending for specific scores. Nonetheless, the PCL-R may continue to be popular for risk assessment because of its pioneering role and the large amount of research done using it.

Sexual offending

A 2011 study of conditional releases for Canadian male federal offenders found that psychopathy was related to more violent and non-violent offences but not more sexual offences. For child molesters, psychopathy was associated with more offences. It is purported that high-psychopathy offenders (both sexual and non-sexual offenders) are about 2.5 times more likely to be granted conditional release compared to non-psychopathic offenders.

Some researchers have argued that psychopaths have a preference for violent sexual behavior. A study examining the relationship between psychopathy scores and types of aggression in a sample of sexual murderers, in which 84.2% of the sample had PCL-R scores above 20 and 47.4% above 30, found that 82.4% of those with scores above 30 had engaged in sadistic violence (defined as enjoyment indicated by self-report or evidence) compared to 52.6% of those with scores below 30, and total PCL-R and Factor 1 scores correlated significantly with sadistic violence.

In considering the issue of possible reunification of some sex offenders into homes with a non-offending parent and children, it has been advised that any sex offender with a significant criminal history should be assessed on the PCL-R, and if they score 18 or higher than they should be excluded from any consideration of being placed in a home with children under any circumstances. There is, however, increasing concern that PCL scores are too inconsistent between different examiners, including in its use to evaluate sex offenders.[7]

Other offending

Researchers have discussed the possibility of psychopathy being associated with organised crime, economic crime and war crimes. Terrorists are sometimes called psychopaths, and comparisons may be drawn with traits such as antisocial violence, a selfish world view that precludes the welfare of others, a lack of remorse or guilt, and blame externalization. However, John Horgan, author of *The Psychology of Terrorism*, argues that such comparisons could also then be drawn more widely: for example, to soldiers in wars. It has also been noted that coordinated terrorist activity requires organization, loyalty and ideology, and that traits such as a self-centered disposition, unreliability, poor behavioral controls, and unusual behaviors may be disadvantages.

It has been speculated that some psychopaths may be socially successful, due to factors such as low disinhibition as defined in the triarchic model, in combination with other advantages such as a favorable upbringing and good

intelligence. However, there is little research on this, in part because the PCL-R does not include positive adjustment characteristics, and most researchers have used the PCL-R on confirmed criminals. Some research using the PPI report that some interpersonal and affective traits associated with psychopathy, and boldness and/or meanness as defined in the triarchic model, can exist in non-criminals and correlate with stress immunity and stability. Psychologists Fritzon and Board, in their study comparing the incidence of personality disorders in business executives against criminals detained in a mental hospital, found that the profiles of senior business managers contained some significant elements of personality disorders, particularly those referred to as the "emotional components" of psychopathy.

Childhood and adolescent precursors

The PCL:YV is an adaptation of the PCL-R for individuals aged 13–18 years. It is, like the PCL-R, done by a trained rater based on an interview and an examination of criminal and other records. The "Antisocial Process Screening Device" (APSD) is also an adaptation of the PCL-R. It can be administered by parents or teachers for 6–13 year olds or it can be self-administered by 13–18 years olds. High psychopathy scores for both juveniles, as measured with these instruments, and adults, as measured with the PCL-R and other measurement tools, have similar associations with other variables, including similar ability in predicting violence and criminality.

There are moderate to high correlations between psychopathy rankings from late childhood to early adolescence. The correlations are considerably lower from early- or mid-adolescence to adulthood. In one study most of the similarities were on the Impulsive- and Antisocial-Behavior scales. Of those adolescents who scored in the top 5% highest psychopathy scores at age 13, less than one third (29%) were classified as psychopathic at age 24. Some recent studies have also found poorer ability at predicting long-term, adult offending. In one study, predictive ability was found to be not better than unaided clinical judgment.

Juvenile psychopathy appears to be associated with more negative emotionality such as anger, hostility, anxiety, and depression.

Conduct disorder

Conduct disorder is diagnosed based on a prolonged pattern of antisocial behavior in childhood and/or adolescence, and may be seen as a precursor to ASPD.[8] Some researchers have speculated that there are two subtypes of conduct disorder which mark dual developmental pathways to adult psychopathy.

The DSM allows differentiating between childhood onset before age 10 and adolescent onset at age 10 and later. Childhood onset is argued to be more due to a personality disorder caused by neurological deficits interacting with an adverse environment. For many, but not all, childhood onset is associated with what is in Terrie Moffitt's developmental theory of crime referred to as "life-course- persistent" antisocial behavior as well as poorer health and economic status. Adolescent onset is argued to more typically be associated with short-term antisocial behavior.

It has been suggested that the combination of early-onset conduct disorder and ADHD may be associated with life-course-persistent antisocial behaviors as well as psychopathy. There is evidence that this combination is more aggressive and antisocial than those with conduct disorder alone. However, it is not a particularly distinct group since the vast majority of young children with conduct disorder also have ADHD. Some evidence indicates that this group have deficits in behavioral inhibition similar to adult psychopaths. They may not be more likely than those with conduct disorder alone to have the interpersonal/affective features and the deficits in emotional processing characteristic of adult psychopaths. Proponents of different types/dimensions of psychopathy have seen this type as possibly corresponding to adult secondary psychopathy and increased disinhibition in the triarchic model.

The DSM-5 includes a specifier for those with conduct disorder who also display a callous, unemotional interpersonal style across multiple settings and relationships. The specifier is based on research which suggests that those with conduct disorder who also meet criteria for the specifier tend to have a more severe form of the disorder as well as a different response to treatment. Proponents of different types/dimensions of psychopathy have seen this

as possibly corresponding to adult primary psychopathy and increased boldness and/or meanness in the triarchic model.

Macdonald triad

Three behaviors—bedwetting, cruelty to animals and firestarting, known as the Macdonald triad—were first described by John Macdonald as possible indicators, if occurring together over time during childhood, of future episodic aggressive behavior. However, subsequent research has found that bedwetting is not a significant factor and the triad as a particular profile has been called an urban legend.[9] Questions remain about a connection between animal cruelty and later violence, though it has been included in the DSM as a possible factor in conduct disorder and later antisocial behavior.

Mental deficits

Learning impairment

Dysfunctions in the prefrontal cortex and amygdala regions of the brain are associated with specific learning impairments in psychopathy. Since the 1980s, scientists have linked traumatic brain injury, including damage to these regions, with violent and psychopathic behavior. Patients with damage in such areas resembled "psychopathic individuals" whose brains were incapable of acquiring social and moral knowledge; those who acquired damage as children may have trouble conceptualizing social or moral reasoning, while those with adult-acquired damage may be aware of proper social and moral conduct but be unable to behave appropriately. Dysfunctions in the amygdala and ventromedial prefrontal cortex may also impair stimulus-reinforced learning in psychopaths, whether punishment-based or reward-based. People scoring 25 or higher in the PCL-R, with an associated history of violent behavior, appear to have significantly reduced mean microstructural integrity in their uncinate fasciculus—white matter connecting the amygdala and orbitofrontal cortex. There is DT-MRI evidence of breakdowns in the white matter connections between these two important areas.

Studies also suggest inverse relationships between psychopathy and intelligence, including with regards to verbal IQ (although in fiction psychopaths are often portrayed as having superior intelligence). Studies also indicate that different aspects of the definition of psychopathy (e.g. interpersonal, affective (emotion), behavioral and lifestyle components) can show different links to intelligence, and the result can also depend on the type of intelligence assessment (e.g. verbal, creative, practical, analytical). However, Hare and Neumann state that a large literature demonstrates at most only a weak association between psychopathy and IQ. They consider that the early pioneer Cleckley included good intelligence in his checklist due to selection bias (since many of his patients were "well educated and from middle-class or upper-class backgrounds") and state that "there is no obvious theoretical reason why the disorder described by Cleckley or other clinicians should be related to intelligence; some psychopaths are bright, others less so."

Emotion recognition and empathy

Studies by R.J.R. Blair and others suggest psychopathy is associated with atypical responses to distress cues (e.g. facial and vocal expressions of fear and sadness), including decreased activation of the fusiform and extrastriate cortical regions, which may partly account for impaired recognition of and reduced autonomic responsiveness to expressions of fear, and impairments of empathy.[10] Studies by Blair on children with psychopathic tendencies have also shown such associations. The underlying biological surfaces for processing expressions of happiness are functionally intact in psychopaths, although less responsive than those of controls.

A recent study in which psychopathic criminals were brain-scanned while they watched videos of a person harming another individual found that the psychopaths' empathic reaction (theorized to occur through the mirror system) initiated the same way it did for controls when they were instructed to empathise with the harmed individual, and that the area of the brain relating to pain was activated when the psychopaths were asked to imagine how the harmed

individual felt. The research demonstrated how psychopaths could switch empathy on at will and would enable them to be both callous and charming. Professor Simon Baron-Cohen suggests that, unlike the combination of both reduced cognitive and affective empathy often seen in those with classic autism, psychopaths are associated with intact cognitive empathy, implying non-diminished awareness of another's feelings when they hurt someone.

Moral judgment

Psychopaths have been considered notoriously amoral – an absence of, indifference towards, or disregard for moral beliefs. There are few firm data on patterns of moral judgment, however. Studies of developmental level (sophistication) of moral reasoning found all possible results – lower, higher or the same as non-psychopaths. Studies that compared judgments of personal moral transgressions versus judgments of breaking conventional rules or laws, found that psychopaths rated them as equally severe, whereas non-psychopaths rated the rule-breaking as less severe.

A study comparing judgments of whether personal or impersonal harm would be endorsed in order to achieve the rationally maximum (utilitarian) amount of welfare, found no significant differences between psychopaths and non-psychopaths. However, a further study using the same tests found that prisoners scoring high on the PCL were more likely to endorse impersonal harm or rule violations than non-psychopaths were. Psychopaths who scored low in anxiety were also more willing to endorse personal harm on average.

Assessing accidents, where one person harmed another unintentionally, psychopaths judged such actions to be more morally permissible. This result is perhaps a reflection of psychopaths' failure to appreciate the emotional aspect of the victim's harmful experience, and furnishes direct evidence of abnormal moral judgment in psychopathy.

Other characteristics

Cleckley's clinical profile

In his book *The Mask of Sanity*, Hervey Cleckley described 16 (originally 21 in the first edition) "common qualities" he thought were characteristic of the individuals he termed psychopaths:

1. Superficial charm and good "intelligence"
2. Absence of delusions and other signs of irrational thinking
3. Absence of "nervousness" or psychoneurotic manifestations
4. Unreliability
5. Untruthfulness and insincerity
6. Lack of remorse and shame
7. Inadequately motivated antisocial behavior
8. Poor judgment and failure to learn by experience
9. Pathologic egocentricity and incapacity for love
10. General poverty in major affective reactions
11. Specific loss of insight
12. Unresponsiveness in general interpersonal relations
13. Fantastic and uninviting behavior with drink and sometimes without
14. Suicide threats rarely carried out
15. Sex life impersonal, trivial, and poorly integrated
16. Failure to follow any life plan.

Cleckley stated in the first edition of *The Mask of Sanity* (p. 257) that those he was calling psychopaths were "frankly and unquestionably psychotic", but in later editions suggested that they are not psychotic according to prevailing definitions. He did not on the whole describe them as particularly hostile or aggressive, contrary to more sinister depictions that others later developed. In addition, he proposed the existence of a milder and extremely common form of the condition: "If we consider, in addition to these patients (nearly all of whom have records of the utmost

folly and misery and idleness over many years and who have had to enter a psychiatric hospital), the vast number of similar people in every community who show the same behavior pattern in milder form but who are sufficiently protected and supported by relatives to remain at large, the prevalence of this disorder is seen to be appalling."

Despite possible discrepancies, Cleckley's work on psychopathy may have influenced the PCL-R and the initial diagnostic criteria for antisocial personality reaction/disturbance in the DSM,[*citation needed*] and some researchers continue to consider Cleckley's clinical profile to be a prominent model of psychopathy.

Causes

Genetic

One approach to studying the role of genetics for crime is to calculate the heritability coefficient. It describes the proportion of the variance that is due to genetic factors for some characteristic that differs between individuals. The non-heritability proportion can be further divided into the "shared environment" which is the non-genetic factors which make siblings similar while the "non-shared environment" is the non-genetic factors which makes siblings different from another. Studies on the personality characteristics typical of psychopathy have found moderate genetic and moderate "non-shared environmental" influences while none from the "shared environment." A study using the PPI found the two factors fearless dominance and impulsive antisociality to be similarly moderately influenced by genetics and uncorrelated with one another which indicated separate genetic influences. Genetic factors may generally influence the development of psychopathy while environmental factors affect the specific traits that predominate. A study on a large group of children found more than 60% heritability for "callous-unemotional traits" and that conduct problems among children with these traits had a higher heritability than among children without these traits.

Studies have also suggested a connection between a variant of the monoamine oxidase A (MAO-A) gene (dubbed the "warrior gene") and psychopathy. In the variant, the allele associated with behavioral traits consists of 30 bases, and produces comparatively less MAO-A enzyme. Low MAO-A activity was found to result in a significantly increased risk of aggression and antisocial behavior. The variant was found to vary widely in demographic prevalence among different ethnic groups. 59% of African-American men, 56% of Maori men and 54% of Chinese men carry the MOA-A 3R genetic variant, compared to 34% of Caucasians.[11]

Environment

A study by Farrington of a sample of London males followed between age 8 and 48 included studying which factors scored 10 or more on the PCL:SV at age 48. The strongest factors included having a convicted parent, being physically neglected, low involvement of the father with the boy, low family income, and coming from a disrupted family. Other significant factors included poor supervision, harsh discipline, large family size, delinquent sibling, young mother, depressed mother, low social class, and poor housing. There has also been association between psychopaths and detrimental treatment by peers.

Researchers have linked head injuries with psychopathy and violence.[23][24] Since the 1980s, scientists have associated traumatic brain injury, such as damage to the prefrontal cortex, including the orbitofrontal cortex, with psychopathic behavior and an inability to make morally and socially acceptable decisions. Children with early damage in the prefrontal cortex may never fully develop social or moral reasoning and become "psychopathic individuals ... characterized by high levels of aggression and antisocial behavior performed without guilt or empathy for their victims." Additionally, damage to the amygdala may impair the ability of the prefrontal cortex to interpret feedback from the limbic system, which could result in uninhibited signals that manifest in violent and aggressive behavior.

Proponents of the triarchic model see psychopathy as due to the interaction of an adverse environment and genetic predispositions. What is adverse may differ depending on the underlying predisposition. Thus, persons having high

boldness may respond poorly to punishment but may respond better to rewards and secure attachments.

Biochemical

High levels of testosterone combined with low levels of cortisol and/or serotonin have been theorized as contributing factors. Testosterone is "associated with approach-related behavior, reward sensitivity, and fear reduction". Injecting testosterone "shift[s] the balance from punishment to reward sensitivity", decreases fearfulness, and increases "responding to angry faces". Some studies have found that high testosterone levels are associated with antisocial and aggressive behaviors, yet other research suggests that testosterone alone does not cause aggression but increases dominance-seeking. It is unclear from studies if psychopathy correlates with high testosterone levels, but a few studies have found psychopathy to be linked to low cortisol levels. Cortisol increases "the state of fear, sensitivity to punishment, and withdrawal behavior". Furthermore, high testosterone levels combined with low serotonin levels, which are associated with "impulsive and highly negative reactions", may increase violent aggression when an individual becomes frustrated. Several animal studies note the role of serotonergic functioning in impulsive aggression and antisocial behavior.

Studies have suggested other correlations. Psychopathy was associated in two studies with an increased ratio of HVA (a dopamine metabolite) to 5-HIAA (a serotonin metabolite). Studies have indicated that individuals with the traits meeting criteria for psychopathy show a greater dopamine response to potential "rewards" such as monetary promises or taking drugs such as amphetamines. This has been theoretically linked to increased impulsivity. A 2010 British study found that a large 2D:4D digit ratio, an indication of high prenatal estrogen exposure, was a "positive correlate of psychopathy in females, and a positive correlate of callous affect (psychopathy sub-scale) in males". Monoamine oxidase A also affected the predictive ability of PCL-R in one study.

Other

Evolutionary explanations

Psychopathy is associated with several adverse life outcomes as well as increased risk of early death due to factors such as homicides, accidents, and suicides. This, in combination with the evidence for genetic influences, is evolutionarily puzzling and may suggest that there are compensating evolutionary advantages. Researchers within evolutionary psychology have proposed several evolutionary explanations. Some psychopaths may possibly be very socially successful. Another is that some associated traits such as early, promiscuous, adulterous, and coercive sexuality may increase reproductive success. A third is that psychopathy represents a frequency-dependent, socially parasitic strategy.[*clarify*] This may work as long as there are few other psychopaths in the community since more psychopaths means increasing the risk of encountering another psychopath as well as non-psychopaths likely adapting more countermeasures against cheaters.

Criticisms include that it may be better to look at the contributing personality factors rather than treat psychopathy as a unitary concept due to poor testability and a lack of empirical evidence regarding reproductive success of psychopaths. Furthermore, if psychopathy is caused by the combined effects of a very large number of adverse mutations then each mutation may have so small an effect that it escapes natural selection.

Mechanisms

Psychosocial

Some laboratory research demonstrate correlations between psychopathy and atypical responses to aversive stimuli, including weak conditioning to painful stimuli and poor learning of avoiding responses that cause punishment, as well as low reactivity in the autonomic nervous system as measured with skin conductance while waiting for a painful stimulus but not when the stimulus occurs. While it has been argued that the reward system functions

normally, some studies have also found reduced reactivity to pleasurable stimuli. Psychopaths have also had difficulty switching from an ongoing action despite environmental cues signaling a need to do so. This may explain the difficulty responding to punishment, although it is unclear if it can explain findings such as deficient conditioning. There may be methodological issues regarding the research. While establishing a range of idiosyncrasies on average in linguistic and affective processing under certain conditions, this research program has not confirmed a common pathology of psychopathy.[12]

Neurological

A 2008 review by Weber et al. suggested that psychopathy is sometimes associated with brain abnormalities in prefrontal-temporo-limbic regions that are involved in emotional and learning processes, among others. Neuroimaging studies have found structural and functional differences between those scoring high and low on the PCL-R with a 2011 review by Skeem et al. stating that they are "most notably in the amygdala, hippocampus and parahippocampal gyri, anterior and posterior cingulate cortex, striatum, insula, and frontal and temporal cortex".

The amygdala and frontal areas have been suggested as particularly important. People scoring 25 or higher in the PCL-R, with an associated history of violent behavior, appear on average to have significantly reduced microstructural integrity between the white matter connecting the amygdala and orbitofrontal cortex (such as the uncinate fasciculus). The evidence suggested that the degree of abnormality was significantly related to the degree of psychopathy and may explain the offending behaviors. Furthermore, changes in the amygdala have been associated with "callous-unemotional" traits in children. However, the amygdala has also been associated with positive emotions, and there have been inconsistent results in the studies in particular areas, which may be due to methodological issues.

Some of the findings above are consistent with other research and theories. For example, in a study of how psychopaths respond to emotional words, widespread differences in activation patterns have been shown across the temporal lobe when criminal psychopaths were compared to "normal" volunteers, which is consistent with views in clinical psychology. Additionally, the notion of psychopathy being characterized by low fear is consistent with abnormalities in the amygdala, since detriments in aversive conditioning and instrumental learning are thought to result from amygdala dysfunction, potentially compounded by orbital frontal cortex dysfunction, although the specific reasons are unknown.

Proponents of the primary-secondary psychopathy distinction and triarchic model argue that there are neuroscientific differences between subgroups of psychopaths which support their views. For instance, the boldness factor in the triarchic model is argued to be associated with reduced activity in the amygdala during fearful or aversive stimuli and reduced startle response, while the disinhibition factor is argued to be associated with impairment of frontal lobe tasks. There is evidence that boldness and disinhibition are genetically distinguishable.

Diagnosis

Tools

Hare Psychopathy Checklist

Psychopathy is most commonly assessed with the Hare Psychopathy Checklist, Revised (PCL-R) created by Robert Hare, based on Cleckley's criteria from the 1940s, criminological concepts such as those of William and Joan McCord, and his own research on criminals and incarcerated offenders in Canada.[13][14] The PCL-R is widely used and is referred to by someWikipedia:Avoid weasel words as the "gold standard" for assessing psychopathy. There are also numerous criticisms of the PCL as as a theoretical tool and in real-world usage.[15]

Each of the 20 items in the PCL-R is scored on a three-point scale, with a rating of 0 if it does not apply at all, 1 if there is a partial match or mixed information, and 2 if there is a reasonably good match to the offender. This is said

to be ideally done through a face-to-face interview together with supporting information on lifetime behavior (e.g. from case files), but is also done based only on file information. It can take up to three hours to collect and review the information. High PCL-R scores are positively associated with measures of impulsivity and aggression, Machiavellianism, persistent criminal behavior, and negatively associated with measures of empathy and affiliation. Out of a maximum score of 40, the cut-off for the label of psychopathy is 30 in the United States and 25 in the United Kingdom, although there is little scientific support for these as particular break points.[*citation needed*] A cut-off score of 25 is also sometimes used for research purposes.

The PCL-R items were designed to be split in two. Factor 1 involves interpersonal or affective (emotion) personality traits and higher values are associated with narcissism and low empathy as well as social dominance and less fear or depression. Factor 2 involves either impulsive-irresponsible behaviors or antisocial behaviors and is associated with a maladaptive lifestyle including criminality. The two factors correlate with each other to some extent. Each factor is sometimes further subdivided in two: interpersonal versus affect items for Factor 1, and impulsive-irresponsible lifestyle versus antisocial behavior items for Factor 2. "Promiscuous sexual behavior" and "many short-term marital relationships" have sometimes been left out in such divisions.

Some have argued that a three-factor structure may provide a better model than the two-factor structure. Cooke and Michie suggested that those items from Factor 2 strictly relating to antisocial behavior (criminal versatility, juvenile delinquency, revocation of conditional release, early behavioral problems, and poor behavioral controls) should be removed, and that the remaining items be divided into three factors: Arrogant and Deceitful Interpersonal Style, Deficient Affective Experience, and Impulsive and Irresponsible Behavioral Style. Hare and colleagues have published detailed critiques of the model and argue that there are statistical and conceptual problems.

Because scores may have important consequences for an individual's future, the potential for harm if the test is used or administered incorrectly is considerable. The test can only be considered valid if administered by a suitably qualified and experienced clinician under controlled conditions.

There are additional inventories derived directly from the PCL-R, including the Psychopathy Checklist: Screening Version (PCL:SV) and Psychopathy Checklist: Youth Version (PCL:YV). The PCL:SV was developed as a labor-saving assessment for the same forensic settings as the PCL-R and to meet the needs of settings where clients do not necessarily have criminal records (e.g. civil psychiatric patients). The PCL:YV assesses juvenile psychopathy in children and adolescents.

Psychopathic Personality Inventory

Unlike the PCL, the Psychopathic Personality Inventory (PPI) was developed to comprehensively index personality traits without explicitly referring to antisocial or criminal behaviors themselves. It is a self-report scale that was developed originally for non-clinical samples (e.g. university students) rather than prisoners, though may be used with the latter. It was revised in 2005 to become the PPI-R and now comprises 154 items organized into eight subscales.[16] The item scores have been found to group into two overarching and largely separate factors (unlike the PCL-R factors), plus a third factor which is largely independent on scores on the other two: Factor I is associated with social efficacy while Factor 2 is associated with maladaptive tendencies. A person may score at different levels on the different factors, but the overall score indicates the extent of psychopathic personality.

DSM and ICD

There are currently two widely established systems for classifying mental disorders—the *International Classification of Diseases* (ICD) produced by the World Health Organization (WHO) and the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) produced by the American Psychiatric Association (APA). Both list categories of disorders thought to be distinct types, and have deliberately converged their codes in recent revisions so that the manuals are often broadly comparable, although significant differences remain.[*citation needed*]

The first edition of the DSM in 1952 had a section on sociopathic personality disturbances, then a general term that included such things as homosexuality and alcoholism as well as an "antisocial reaction" and "dyssocial reaction". The latter two eventually became antisocial personality disorder (ASPD) in the DSM and dissocial personality disorder in the ICD.[*citation needed*] Both manuals have stated that their diagnoses have been referred to, or include what is referred to, as psychopathy or sociopathy, although neither diagnostic manual has ever included a disorder officially titled as such.

Researchers such as Robert Hare and Stephen Hart regard the mainstream psychiatric view as deeply flawed, calling for a return to a traditional model of psychopathy as a distinct disorder.Wikipedia:Citing sources

DSM

Antisocial personality disorder

Antisocial personality disorder (ASPD), the criteria of which were based on American psychiatrist Hervey Cleckley's work[*citation needed*] on psychopathy, is described in the DSM-IV-TR as "... a pervasive pattern of disregard for, and violation of, the rights of others that begins in childhood or early adolescence and continues into adulthood. This pattern has also been referred to as psychopathy, sociopathy, or dissocial personality disorder."[17]

A diagnosis of ASPD is based largely on explicit behavioral patterns, whereas measurement tools such as the PCL or PPI also largely, or solely, rely on judgment or self-reports of personality traits. The diagnostic criteria for ASPD focus relatively less on personality traits partly due to the belief that such traits are difficult to measure reliably and it is "easier to agree on the behaviors that typify a disorder than on the reasons why they occur". As a result, critics have argued that psychopathy and ASPD are not synonymous, despite the DSM's statement that ASPD has been referred to as psychopathy.

Nonetheless, psychopathy has been proposed as a specifier under an alternative model for ASPD. In the DSM-5, under "Alternative DSM-5 Model for Personality Disorders", ASPD with psychopathic features is described as characterized by "a lack of anxiety or fear and by a bold interpersonal style that may mask maladaptive behaviors (e.g., fraudulence)." Low levels of withdrawal and high levels of attention-seeking combined with low anxiety demonstrate "social potency" and "stress immunity" in psychopathy.:765 Under the specifier, affective and interpersonal characteristics are comparatively emphasized over behavioral components.

ICD

Dissocial personality disorder

The ICD defines a conceptually similar or equivalent disorder to ASPD called dissocial personality disorder, "usually coming to attention because of a gross disparity between behaviour and the prevailing social norms, and characterized by" at least 3 of 6 specific issues. The manual states that its diagnosis includes "amoral, antisocial, asocial, psychopathic, and sociopathic personality (disorder)".[29][18]

Other tools

There are some traditional personality tests that contain subscales relating to psychopathy, though they assess relatively non-specific tendencies towards antisocial or criminal behavior. These include the Minnesota Multiphasic Personality Inventory (Psychopathic Deviate scale), California Psychological Inventory (Socialization scale), and Millon Clinical Multiaxial Inventory Antisocial Personality Disorder scale. There is also the Levenson Self-Report Psychopathy Scale (LSRP) and the Hare Self-Report Psychopathy Scale (HSRP), but in terms of self-report tests, the PPI/PPI-R has become more used than either of these in modern psychopathy research on adults.

Comorbidity

As with other mental disorders, psychopathy as a personality disorder may be present with a variety of other diagnosable conditions. Studies especially suggest strong comorbidity with antisocial personality disorder. Among numerous studies, positive correlations have also been reported between psychopathy and histrionic, narcissistic, borderline, paranoid, and schizoid personality disorders, panic and obsessive–compulsive disorders, but not neurotic disorders, schizophrenia, or depression.

Attention deficit hyperactivity disorder (ADHD) is known to be highly comorbid with conduct disorder (a theorized precursor to ASPD), and may also co-occur with psychopathic tendencies. This may be explained in part by deficits in executive function. Anxiety disorders often co-occur with ASPD, and contrary to assumptions, psychopathy can sometimes be marked by anxiety; this appears to be related to items from Factor 2 but not Factor 1 of the PCL-R.[*citation needed*] Psychopathy is also associated with substance use disorders.

It has been suggested that psychopathy may be comorbid with several other conditions than these, but limited work on comorbidity has been carried out. This may be partly due to difficulties in using inpatient groups from certain institutions to assess comorbidity, owing to the likelihood of some bias in sample selection.

Further considerations

Subtypes

Primary and secondary

Several researchers have argued that there exist two variants of psychopathy. There is also empirical support for separating persons scoring high on the PCL-R into two groups that do not simply reflect Factor 1 and Factor 2. There is at least preliminary evidence of differences regarding cognition and affect as measured in laboratory tests. Different theories characterize these two variants somewhat differently.

Compared to "primary" psychopaths, "secondary" psychopaths have been characterized as having more fear, anxiety, and negative emotions. They are often seen as more impulsive and with more reactive anger and aggression. David T. Lykken, using Gray's biopsychological theory of personality, argued that primary psychopaths innately have little fear while secondary psychopaths innately have increased sensitivity to rewards. Studies also suggest that secondary psychopaths manifest more borderline personality features than primary psychopaths, and comparable levels of antisocial behavior.

There are also different theories as to the predominant causes of either variant. Some researchers, such as Benjamin Karpman, believe that primary psychopaths are born with an emotional deficit and that secondary psychopaths acquire it through adverse environmental experiences, although others, such as Lykken, link both variants to biological predispositions. Some preliminary research suggests that secondary psychopaths may have had a more abusive childhood, a higher risk of future violence, and potentially a better response to treatment.

Other proposed subtypes

Results of a study on male patients at a maximum-security forensic hospital suggested four potential subtypes of psychopathy: narcissistic, borderline, sadistic, and antisocial. The researchers have stated that additional data are needed to understand the observed variations.

Sex differences

Research on psychopathy has largely been done on men and the PCL-R was developed using mainly male criminal samples, raising the question of how well the results apply to women. There have also been research investigating the sex differences. Men score higher than women on both the PCL-R and the PPI and on both of their main scales. The differences tend to be somewhat larger on the interpersonal-affective scale than on the antisocial scale. Most but not all studies have found broadly similar factor structure for men and women.

Many associations with other personality traits are similar, although in one study the antisocial factor was more strongly related with impulsivity in men and more strongly related with openness to experience in women. It has been suggested that psychopathy in men manifest more as an antisocial pattern while in women it manifests more as a histrionic pattern. Studies on this have shown mixed results. PCL-R scores may be somewhat less predictive of violence and recidivism in women. On the other hand, psychopathy may have a stronger relationship with suicide and possibly internalizing symptoms in women. A suggestion is that psychopathy manifests more as externalizing behaviors in men and more as internalizing behaviors in women.

Management

Medical

Psychopathy has often been considered untreatable. Harris and Rice's *Handbook of Psychopathy* says that there is little evidence of a cure or effective treatment for psychopathy; no medications can instill empathy, and psychopaths who undergo traditional talk therapy might become more adept at manipulating others and more likely to commit crime. The only study finding increased criminal recidivism after treatment was in a 2011 retrospective study of a treatment program in the 1960s that had several methodological problems likely not approved today. Some relatively rigorous quasi-experimental studies using more modern treatment methods have found improvements regarding reducing future violent and other criminal behavior, regardless of PCL-R scores, although none were randomized controlled trials. Various other studies have found improvements in risk factors for crime such as substance abuse. No study had in a 2011 review examined if the personality traits could be changed by such treatments. It has been shown in some studies that punishment and behavior modification techniques may not improve the behavior of psychopaths.

A recent study on psychopathic criminals found that under certain circumstances, they could willfully empathize with others, and that their empathic reaction initiated the same way it does for controls. The team who conducted the study say it is still unknown how to transform this willful empathy into the spontaneous empathy most people have, though they propose it could be possible to bring psychopaths closer to rehabilitation by helping them to activate their "empathy switch". Others suggested that despite the results of the study, it remained unclear whether psychopaths' experience of empathy was the same as that of controls, and also questioned the possibility of devising therapeutic interventions that would make the empathic reactions more automatic.

Certain psychiatric medications may alleviate conditions sometimes associated with ASPD (which has been referred to as, may present with features of, or is often comorbid with psychopathy), or with symptoms such as aggression, including antipsychotic, antidepressant or mood-stabilizing medications.Wikipedia:Vagueness

Legal

The PCL-R, the PCL:SV, and the PCL:YV are highly regarded and widely used in criminal justice settings, particularly in North America. They may be used for risk assessment and for assessing treatment potential and be used as part of the decisions regarding bail, sentence, which prison to use, parole, and regarding whether to a youth should be tried as a juvenile or as an adult. There have been several criticisms against this. They include the general criticisms against the PCL-R, the availability of other risk assessment tools which may have advantages, and excessive pessimism regarding prognosis and treatment possibilities.

The interrater reliability of the PCL-R can be high when used carefully in research but tend to be poor in applied settings. In particular Factor 1 items are somewhat subjective. In sexually violent predator cases the PCL-R scores given by prosecution experts were consistently higher than those given by defense experts in one study. The scoring may also be influenced by other differences between raters. In one study it was estimated that of the PCL-R variance, about 45% was due to true offender differences, 20% was due to which side the rater testified for, and 30% was due to other rater differences.

United Kingdom

The PCL-R cut-off for a label of psychopathy is 25 in the United Kingdom, instead of 30 as it is in the United States.

In the United Kingdom, "psychopathic disorder" was legally defined in the Mental Health Act (UK), under MHA1983,[19] as "a persistent disorder or disability of mind (whether or not including significant impairment of intelligence) which results in abnormally aggressive or seriously irresponsible conduct on the part of the person concerned." This term was intended to reflect the presence of a personality disorder in terms of conditions for detention under the Mental Health Act 1983. With the subsequent amendments to the Mental Health Act 1983 within the Mental Health Act 2007, the term "psychopathic disorder" has been abolished, with all conditions for detention (e.g. mental illness, personality disorder, etc.) now being contained within the generic term of "mental disorder".[*citation needed*]

In England and Wales, the diagnosis of dissocial personality disorder is grounds for detention in secure psychiatric hospitals under the Mental Health Act if they have committed serious crimes, but since such individuals are disruptive for other patients and not responsive to treatment this alternative to prison is not often used.

United States

"Sexual psychopath" laws

Starting in the 1930s, before some modern concepts of psychopathy were developed, "sexual psychopath" laws were introduced by some states, and by the mid-1960s more than half of the states had such laws. "Sexual psychopaths" were seen as a distinct group of sex offenders who were not seriously mentally ill but had a "psychopathic personality" that could be treated. This was in agreement with the general rehabilitative trends at this time. Courts sent such sex offenders to a mental health facility for community protection and treatment.[20]

Starting in 1970, many of these laws were modified or abolished in favor of more traditional responses such as imprisonment due to criticism of the "sexual psychopath" concept as lacking scientific evidence, the treatment being ineffective, and predictions of future offending being dubious. There were also a series of cases where persons treated and released committed new sex crimes. Starting in the 1990s several states have passed sexually dangerous person laws, not synonymous with the modern concept of psychopathy, which permit confinement after a sentence has been completed. Psychopathy measurements may be used in the confinement decision process.

Epidemiology

A 2008 study using the PCL:SV found that 1.2% of a US sample scored 13 or more out of 24, indicating "potential psychopathy". The scores correlated significantly with violence, alcohol use, and lower intelligence. A 2009 British study by Coid et al., also using the PCL:SV, reported a community prevalence of 0.6% scoring 13 or more. The scores correlated with younger age, male gender, suicide attempts, violence, imprisonment, homelessness, drug dependence, personality disorders (histrionic, borderline and antisocial), and panic and obsessive–compulsive disorders.

Robert Hare has stated that many male psychopaths have a pattern of mating with and quickly abandoning women, and thereby have a high fertility rate, resulting in children that may inherit a predisposition to psychopathy. However, empirical evidence regarding the reproductive success of psychopaths is lacking.

History

Etymology

The word *psychopathy* is a joining of the Greek words psyche (ψυχή)—meaning soul—and pathos (πάθος)—meaning suffering or feeling.[21] The first documented use is from 1847 in Germany as *psychopatisch*,[22] and the noun *psychopath* has been traced to 1885.[23] In medicine, *patho-* has a more specific meaning of disease (thus *pathology* has meant the study of disease since 1610, and *psychopathology* has meant the study of mental disorder in general since 1847. A sense of "a subject of pathology, morbid, excessive" is attested from 1845,[24] including the phrase *pathological liar* from 1891 in the medical literature).

The term *psychopathy* initially had a very general meaning referring to all sorts of mental disorders and social aberrations, popularised from 1891 in Germany by Koch's concept of "psychopathic inferiority" (psychopathische Minderwertigkeiten). Some medical dictionaries still define psychopathy in both a narrow and broad sense, such as MedlinePlus from the U.S. National Library of Medicine.[25] On the other hand, Stedman's Medical Dictionary defines psychopathy only as an outdated term for an antisocial type of personality disorder.[26]

The term *psychosis* was also used in Germany from 1841, originally in a very general sense. The suffix -ωσις (-osis) meant in this case "abnormal condition". This term or its adjective *psychotic* would come to refer to the more severe mental disturbances and then specifically to mental states or disorders characterized by hallucinations, delusions or in some other sense markedly out of touch with reality.

The slang *psycho* has been traced to a shortening of the adjective *psychopathic* from 1936, and from 1942 as a shortening of the noun *psychopath*,[27] but it is also used as shorthand for psychotic or just mentally crazed in some way.

The label *psychopath* has been described as nonspecific but probably persisting because it indicates that the source of behavior lies in the psyche rather than in the situation. The media usually uses the term to designate any criminal whose offenses are particularly abhorrent and unnatural, but that is not its original or general psychiatric meaning.

Sociopathy

The word element *socio* has been used in compound words since around 1880.[28] The term *sociopathy* may have been first introduced in 1909 in Germany by biological psychiatrist Karl Birnbaum and in 1930 in the US by educational psychologist George E. Partridge, as an alternative to, or a subtype of, the concept of *psychopathy*. It was used to indicate that the defining feature is violation of social norms, or antisocial behavior, and has often also been associated with postulating social as well as biological causation.[29][30][31][32]

There are various contemporary usages of the term. Robert Hare, who may believe that biological factors are predominant in causing psychopathy,[33] claimed in a 1999 popular science book that *sociopathy* and *psychopathy* are often used interchangeably, but in some cases the term *sociopathy* is preferred because it is less likely than is *psychopathy* to be confused with psychosis, whereas in other cases which term is used may "reflect the user's views on the origins and determinates of the disorder". Hare contended that the term *sociopathy* is preferred by those that see the causes as due to social factors and early environment, and the term *psychopathy* preferred by those who believe that there are psychological, biological, and genetic factors involved in addition to environmental factors. Hare also suggests another possible distinction: he defines psychopathy as not having a sense of empathy or morality, but sociopathy as only differing in sense of right and wrong from the average person.Wikipedia:Verifiability

Precursors

The concept of psychopathy has been indirectly connected to the early 1800s with the work of Pinel (1801; "mania without delirium") and Pritchard (1835; "moral insanity"), although historians have largely discredited the idea of a direct equivalence. The term *psychopathic* was coined toward the end of the 19th century, by the German psychiatrist Julius Koch (1891). In contrast with current usage, Koch applied the term *psychopathic inferiority* (psychopathischen Minderwertigkeiten) to various chronic conditions and character disorders.

The term *psychopathic* came to be used to describe a diverse range of dysfunctional or antisocial behavior and mental and sexual deviances, including at the time homosexuality. It was often used to imply an underlying "constitutional" or genetic origin. Disparate early descriptions likely set the stage for modern controversies about the definition of psychopathy.

20th century

An influential figure in shaping modern American conceptualizations of psychopathy was American psychiatrist Hervey Cleckley. In his classic monograph, *The Mask of Sanity* (1941), Cleckley drew on a small series of vivid case studies of psychiatric patients at a Veterans Administration hospital in Georgia to describe the disorder. Cleckley used the metaphor of the "mask" to refer to the tendency of psychopaths to appear confident, personable, and well-adjusted compared to most psychiatric patients, while revealing underlying pathology through their actions over time. Cleckley formulated sixteen criteria to describe the disorder. The Scottish psychiatrist David Henderson had also been influential in Europe from 1939 in narrowing the diagnosis.

The diagnostic category of *sociopathic personality* in early editions of the *Diagnostic and Statistical Manual* (DSM) had some key similarities to Cleckley's ideas, though in 1980 when renamed Antisocial Personality Disorder some of the underlying personality assumptions were removed. In 1980, Canadian psychologist Robert D. Hare introduced an alternative measure, the "Psychopathy Checklist" (PCL) based largely on Cleckley's criteria, which was revised in 1991 (PCL-R), and is the most widely used measure of psychopathy. There are also several self-report tests, with the Psychopathic Personality Inventory (PPI) used more often among these in contemporary adult research.

Famous individuals have sometimes been diagnosed, albeit at a distance, as psychopaths. As one example out of many possible from history, in a 1972 version of a secret report originally prepared for the Office of Strategic Services in 1943, and which may have been intended to be used as propaganda,[34][35] non-medical psychoanalyst Walter C. Langer suggested Adolf Hitler was probably a psychopath. However, others have not drawn this conclusion; clinical forensic psychologist Glenn Walters argues that Hitler's actions do not warrant a diagnosis of psychopathy as, although he showed several characteristics of criminality, he was not always egocentric, callously disregarding of feelings or lacking impulse control and there is no proof he couldn't learn from mistakes.

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Relational aggression

Relational aggression, also known as **covert aggression**,[1] or **covert bullying**[2] is a type of aggression in which harm is caused through damage to one's relationships or social status.[3] Although it can be used in many contexts and among different age groups, relational aggression among adolescents, in particular, has received a lot of attention with the help of popular media including movies like *Mean Girls* and books like *Odd Girl Out* by R. Simmons (2003) and *Queen Bees and Wannabes* by R. Wiseman (2003). Relational aggression can have various lifelong consequences. Relational aggression has been primarily observed and studied among girls.

Overview

Peers become increasingly significant in adolescence. Peer relationships are especially important for adolescents' healthy psychological development: peers provide many new behavioral models and feedback essential for successful identity formation and development of one's sense of self.[4][5] Interactions with peers encourage positive practice of autonomy and independent decision-making skills,[6] as well as are essential for healthy sexual development including the development of the capacity for intimate friendships and learning appropriate sexual behavior.[7] Peer relationships are also very important for determining how much adolescents value school, how much effort they put into it, and how well they perform in class.[8][9] However, quite frequently adolescents take part in peer relationships that are harmful for their psychological development. Adolescents tend to form various cliques and belong to different crowds based on their activity interests, music and clothing preferences, as well as their cultural or ethnic background.[10] Such groups differ in their sociometric or popularity status, which often create unhealthy, aggression-victimization based dynamics between groups. Different forms of aggression can also be used to control dynamics and sociometric status within a group. Sometimes aggression is directed to an individual rather than to any apparent social group. Primary reasons for victimization include looks and speech; adolescents are also frequently bullied because of a disability, particular ethnicity, or religion.[11]

Definition

Relational aggression is defined as a type of aggression that is "intended to harm other adolescents through deliberate manipulation of their social standing and relationships." [12] Relational aggression, according to Dan Olweus [13] is a type of bullying. Bullying in general, is defined as physically or psychologically violent re-occurring and not provoked acts, where the bully and victim have unequal physical strength or/and psychological power.[14] These key conditions apply to all types of bullying: verbal, physical, relational.

Forms

The main three forms of relational aggression include:

- Excluding others from social activities;
- Damaging victim's reputations with others by spreading rumors and gossiping about the victim, or humiliating him/her in front of others;
- Withdrawing attention and friendship.

Psychological manipulation and coercion can also be considered as a type of relational aggression.

Most recent research has been focusing on cyber-bullying, which is a relatively new yet increasingly popular way of engaging in both verbal and relational aggression due to growing importance of various communication and technology devices in modern societies.[15] Some studies show that internet meanness is more common among girls than boys.

Prevalence

Many studies in the U.S. and Europe show that at least 30% of students report having been bullied in one or another way.[*citation needed*] Some studies indicate even higher percentages of victimization. Bullying in schools happens in all forms and at various ages, although peer bullying has the highest prevalence in 6th-8th grades.[16] The most common forms of bullying are verbal with relational, or various forms of ostracism, coming in second.[17]

Gender differences

Although it can be used by both genders, relational aggression is more commonly associated with girls.[] Findings of a study by Rivers and Smith [18] have shown that while verbal aggression occurs with similar frequency in both sexes, direct physical aggression is more common among boys and indirect aggression is more common among girls. In another study by Baldry [19] it was found that boys are more likely to engage in bullying behaviors such as threats, physical harm, rejection, and name-calling, while girls are most likely to use name-calling, teasing, rumors, rejection, and taking personal belongings. Based on these findings, girls do seem to use relational aggression somewhat more than boys. In addition, recent international research shows that both genders tend to use relational aggression, but girls are more aware and distressed by it.[20][21][22] For example, a study by Horn [23] found that girls are more likely to say that it is morally wrong to exclude someone based on their crowd membership. However, this tendency for girls to engage in relational aggression more so than for boys might be influenced by different socialization of male and female children and different social expectations associated with gender roles.[*citation needed*] Boys are allowed to be more physically and verbally aggressive than girls.[*citation needed*] Some research shows that there are certain implications when boys and girls engage in gender- atypical aggression as girls who are more physically aggressive or boys who are highly relationally aggressive are more maladjusted than their peers, who engage in gender- typical aggression.[24]

Sociometric status

Sociometric status, commonly referred as popularity, is one of the most significant predictors of victimization or bullying as differences in popularity can be associated with differences in social power. It is commonly believed that aggressive adolescents belong to rejected social groups. However, some research shows that they can be popular among their peers.[25][26] Rodkin et al. (2000),[27] for example, describes two types of popular boys: "model" boys, who are "physically and academically competent, friendly and neither shy nor aggressive." Second type is described as "tough" and such adolescents are "aggressive, physically competent, and average or below average in friendliness, academic competence, and shyness.". Usually the more popular aggressive adolescents use instrumental aggression and not reactive aggression.[28][29] Instrumental aggression is defined as behavior that is deliberate and planned while reactive aggression is unplanned and impulsive. Relational aggression can be greatly instrumental for maintaining the popularity status of a group among other groups, as well specific relationship and status dynamics inside a group. Ojala and Nesdale (2004) [30] found that both victims and bullies normally come from rejected groups. Bullies chose to bully students, who are members of their social out-groups that are similar to their own in-group as a result of threatened distinctiveness. Hence, the need to maintain a unique social identity and status can be one of the causes to engage in bullying. Using relational aggression to maintain a particular social order inside the group has been mostly observed in girl groups: if some member of the group becomes too popular and this causes imbalance in the group, other members might start rumors about the overly popular girl to diminish her status.[31] Amanda Rose (2004) [32] claims that the main purpose of using relational aggression in first place is to enhance or maintain one's social status. Many skills that are needed to be popular are also essential for being "successful" at employing relational aggression, e.g. ability to "read" people and adjust one's behavior accordingly, etc. The researcher suggests that some aggressive boys are popular because they are also good at using relational aggression, and, therefore, their primary reason for popularity is not their physical but relational aggression.

Different participation roles

Research shows that there are three types of rejected or unpopular adolescents who are very likely to be involved in bullying behavior.[33][34][35][36][37][38] First type includes adolescents who are overly aggressive: they tend to get into fights, get involved in antisocial activities, and are often involved in bullying; second type includes adolescents who are withdrawn or timid and exceedingly shy and inhibited and who are more likely to be victims; third- aggressive- withdrawn- type adolescents tend to have trouble controlling their hostility, but they are also very shy and nervous about initiating friendships. The latter are likely to be bully-victims. Other students- bystanders can also choose between several roles: victim-defender, bully-reinforcer and/or assistant, and outsiders.

Victims

Victims or the unpopular withdrawn children are excessively anxious, lack social skills needed to initiate new contacts or break into a group activity.[39] Their lack of confidence combined with submissiveness make them perfect targets for bullying.[40][41] Some of the most common underlying reasons for bullying include low socioeconomic status, disability, and obesity.[42][43][44] Research shows that in comparison with other adolescents victims often use worse problem solving strategies.[45] They often score less than their bullies and defenders in the tests of moral competence and theory of mind.[46] Moral competence refers to the ability to carefully consider both the consequences and prior beliefs in determining how morally right or wrong one's actions are. Victims seemed to focus primarily on the outcomes and not being as good in integrating the moral beliefs. They have difficulties in social skills, and social problem solving, as well as emotional regulation.[47] And because of their lack of social competence, victims score low on peer acceptance and popularity.[48][49] Victims are often overly sensitive to being rejected, which might originate in their relationships with parents.[50]

Bullies

Bullies, despite being quite morally competent, tend to engage in morally wrong behaviors because of several reasons, including a lack of moral compassion. In general, bullies seem to engage in a kind of cold cognition and have a good theory of mind. They also have an average to good social intelligence. These skills seem to be especially important in order to use relational aggression in an instrumental manner—for achieving specific social goals. As mentioned previously, male and female bullies usually score differently on sociometric measures. Male bullies often fall in the socially rejected category while female bullies tend to fall in the controversial category. They can be popular yet not liked.

Hostile Attributional Bias

Many unpopular aggressive kids seem to engage in hostile attributional bias when analyzing the actions of others: they are more likely to interpret other children's behavior as hostile while it is not,[51][52] which can cause the perpetuation of their aggressive behaviors.

Bully-victims

Bully- victims are adolescents who have both experienced aggression directed towards them and have themselves engaged in bullying. They often choose to be bully assistants or reinforcers.[53] Seeing others victimized can serve as a buffer against some psychological problems, for which these adolescents are at risk (see Consequences of victimization below). In comparison to all other groups, bully-victims are the worst off regarding their psychological adjustment and problems. They are least liked among the peers.

Bystanders

Although early research has mostly focused on victims and bullies, currently more and more attention has been given to the roles of other students, or bystanders: bully-reinforcer's and assistants, victim-defenders, and outsiders.

Bully- reinforcers and assistants

Bully-reinforcers and assistants do not normally initiate aggressive actions themselves, but they support, reinforce, and/or assist the bully. They often have rather large friendship networks when compared to outsiders, victims, and their defenders. These individuals are similar to bullies in regards of their personal characteristics. Female bully-reinforcers and assistants usually score low on social acceptance and high on rejection by their peers while male bully assistants have average scores on both and bully- reinforcers are often quite popular among their peers. The characteristic that is common among all these individuals across both genders is low level of empathy.

Victim-defenders

Victim-defenders are individuals who stand up for the victim. They are usually popular among their peers, although occasionally rejected and victimized adolescents take on the defender's role. Defenders like to befriend other defenders and usually belong to the smallest social network of all other previously mentioned groups. Defenders have both advanced moral competence and high level of compassion. They also score high on the theory of mind tests. They are usually very morally engaged, have a high sense of responsibility, and self-efficacy. They are also good at emotion regulation.[54]

Outsiders

Outsiders are adolescents who like to stay away from the conflict situations, participate in spreading rumors, or actively support either side. They usually befriend other outsiders. Both male and female outsiders usually score below average on both social acceptance and rejection by their peers. In general, the best predictor for whether an adolescent will choose to be a defender or an outsider in a particular situation is their relationship to the victim and/or bully. Occasionally, adolescents will feel more comfortable to intervene if they are friends of the offender.[55] However, in general they will take the side of the bully or victim based on who they know better.[56] Bullies are more likely to be friends of other bullies, as well as their reinforcers, and assistants, while victims befriend other victims.

Consequences of victimization

There are serious negative consequences associated with being involved in any aggressive behaviors. And while problems with peers might be a result of one's poor social skills and maladjustment, difficulty making friends, and regular experience of aggression can also be a cause of many short and long term negative consequences on one's mental health and academic and professional achievements.[57][58][59] Experience of relational aggression, peer rejection, and unpopularity are shown to be linked to various problems in adolescence, which are listed below:[60][61][62][63]

- depression
- behavior problems;
- poor social skills;
- lack of close peer relationships;
- difficulties in academic performance;
- low school engagement;
- undermined feelings of competence;
- low self-esteem

- occasionally distress due to victimization can also result in physical symptoms such as wetting, abdominal pain, or/and headaches.[64]

Some negative effects persist into adulthood. In a longitudinal study, Dan Olweus (2003) found that young adults, who were victims of bullying in adolescence, had more symptoms of depression and lower self-esteem than did their non-victimized peers. Victims are also much more likely to engage in heavy smoking later in life.[65] Decreased academic engagement due to victimization can have some long term consequences as victim's lower educational attainment in adulthood leads to lower earnings.[66]

Differences in consequences of victimization for victims and bully-victims

There are differences in consequences among the children who are rejected and aggressive, also known as bully-victims, and children who are rejected and withdrawn, also referred to as simply victims. Aggressive individuals often have conduct problems and are involved in antisocial activity.;[67][68][69][70] withdrawn children feel exceedingly lonely, at risk of low self-esteem, depression, and diminished social competence.[71] Adolescents, who are both aggressive and withdrawn, are at greatest risk for various mental and behavioral problems [72][73]

Suicide ideation and attempts

Although victims respond to bullying in various ways, some of the most common ways include avoidance or escape behaviors, such as not going to school and running away from home. However, in some extreme cases, suicide attempts might occur.[74] Compared to non-victims, victims exhibit increased levels of suicidal ideation.[75][76] and are more likely to have attempted suicide [77][78][79] Researcher Y.S. Kim (2005) found that there are some gender differences as victimized female but not male students were at significantly greater risk for suicidal ideation. Further research has shown that increased risk for suicidal ideation and attempts depend on a specific interaction between gender, frequency, and type of aggression. Relational or indirect aggression was found to be associated with depression and suicidal ideation among both genders.[80] According to Brustein and Klomek (2007), victimization at any frequency increased the risk of depression, ideation, and attempts among girls, while only frequent victimization increased the risk of depression and ideation among males, yet Katliala-Heino et al.(1999) [81] found that severe ideation was associated with frequent victimization only among girls.

Environmental buffers and prevention programs

Some adolescents are more resilient to victimization due to their personal characteristics, but there are some environmental factors such as having a best friend or great family support can decrease the risk for many negative consequences associated with victimization.[82] In addition, research shows that support from teachers can be a significant environmental factor for higher academic achievement and school engagement. It can also increase general well- being in the classroom.[83] Teacher attitudes towards bullying were found to moderate the extend of which victims internalize and feel distressed and express it by avoiding school and similar behavior.[84] Close teacher- student relationship moderates perceived safety in the classroom, and higher perceived safety is directly linked to better classroom concentration and improved coping strategies [85] Therefore, supportive friends, family, and teachers can be great buffers for victimized students against all negative effects of victimization. Interestingly, witnessing the harassment of others can also reduce some harmful effects of being victimized:[86] victims-only feel more humiliated and angry than victims-witnesses on the same day. Being singled out and picked on feels worse than being one of many victimized students. This explains why in ethnically diverse schools victimized students experience worse psychological outcomes when their ethnic group is in majority, because then they are more likely to attribute it to their personal shortcomings and not to their group membership.[87]

Prevention programs

There are many prevention programs, which have been designed to improve social skills of the unpopular and victimized adolescents. Prevention programs usually focus on one of the three strategies:

1. teaching social skills like self-expression, leadership, and questioning of others about themselves;[88][89]
2. have unpopular adolescents participate in group activities together with the popular adolescents under supervision of psychologists;
3. some programs focus on training on how to combine and use one's cognitive and behavioral abilities, including social problem solving.[90][91]

Different types of programs have shown to have somewhat different effects: the first type seems to best improve adolescent's ability to get along with others while the second type has shown to improve adolescents' self-conceptions and their acceptance by others.[92] One of the examples of the programs using the third approach is PATHS (Promoting Alternative Thinking Strategies) [93] teaches skills needed for successfully analyzing social situations, controlling one's negative emotions, and making more rational social decisions. It has been shown to successfully reduce behavioral problems among elementary school children.[94] However, it is difficult to prevent relational aggression from happening as often adolescents who use it are seen to be more popular among their peers.

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Further reading

Books

- Kupkovits, Jamie, *Relational Aggression in Girls* (2008)
- Randall, Kaye & Bowen, Allyson A., *Mean Girls: 101$^{1}/_{2}$ Creative Strategies for Working With Relational Aggression* (2007)

Academic articles

- Carpenter, E.M. & Nangle, D.W. (2006). "Caught between stages: Relational aggression emerging as a developmental advance in at-risk preschoolers." *Journal of Research in Childhood Education*, 21, 177–188.
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External links

- Website on Relational Aggression (http://www.relationalaggression.com)
- The Ophelia Project (http://www.opheliaproject.org/main/index.htm)
- Youth & Family Resource Center, Inc. (http://www.hopehouseonline.org/pages/girls.shtml)

Simon Baron-Cohen

Simon Baron-Cohen	
 Baron-Cohen in 2011	
Born	15 August 1958
Residence	England
Nationality	British
Fields	Psychology and Cognitive Neuroscience
Institutions	University of Cambridge
Alma mater	• New College, Oxford • King's College London • University College London
Doctoral advisor	Uta Frith
Known for	Autism research
Notable awards	Kanner-Asperger Medal 2013 (WGAS)

Simon Baron-Cohen FBA is Professor of Developmental Psychopathology at the University of Cambridge in the United Kingdom. He is the Director of the University's Autism Research Centre, and a Fellow of Trinity College. He is best known for his work on autism, including the theory that autism involves degrees of mind-blindness (or delays in the development of theory of mind) and his later theory that autism is an extreme form of what he calls the "male brain", which involved a re-conceptualisation of typical psychological sex differences in terms of empathizing–systemizing theory.

Personal life and education

Baron-Cohen completed a BA in Human Sciences at New College, Oxford, and an MPhil in Clinical Psychology at the Institute of Psychiatry, King's College London. He completed a PhD in Psychology at University College London; his doctoral research was in collaboration with his supervisor Uta Frith.

Baron-Cohen's cousin is Sacha Baron Cohen.

Autism research

Baron-Cohen was lead author in 1985 of the first study of children with autism and delays in the development of a theory of mind, known as ToM. The theory of mind is the ability to detect other people's emotions and thoughts, and it is a skill that according to Baron-Cohen's research is typically delayed developmentally in children with autism.

Baron-Cohen and his colleagues discovered in 1987 the first evidence that experiences in synaesthesia remain consistent over time; they also found synaesthesia to be measurable via neuroimaging techniques. His team has

investigated whether synaesthesia is connected to autism.

In 1997, Baron-Cohen developed the empathizing–systemizing theory, which theorises that a cognitive profile he terms "empathising-systemising", associated with math, science and technology skills, exists in families with autism spectrum disorders. He suspects that if individuals with a "systemising" focus are selecting each other as mates, they are more likely to have children with autism. He postulates that more individuals with autistic traits are marrying each other and having children. He said that "In essence, some geeks may be carriers of genes for autism: in their own life, they do not demonstrate any signs of severe autism, but when they pair up and have kids, their children may get a double dose of autism genes and traits. In this way, assortative mating between technical-minded people might spread autism genes." *Time* magazine said that his views on systemising traits had "earned him the ire of some parents of autistic children, who complain that he underestimates their families' suffering". *Time* said that while research from Washington University in St. Louis did not support the assortive mating theory, a survey finding that autism was twice as high in Eindhoven (the Silicon Valley of the Netherlands) had "breathed new life" into Baron-Cohen's theory.

Baron-Cohen's work in systemising-empathising led him to investigate whether higher levels of foetal testosterone explain the increased prevalence of autism spectrum disorders among males; his theory is known as the "extreme male brain" theory of autism. A review of his book *The Essential Difference* published in *Nature* in 2003 summarises his proposal as: "the male brain is programmed to systemize and the female brain to empathize ... Asperger's syndrome represents the extreme male brain". Critics say that because his work has focused on higher-functioning individuals with autism spectrum disorders, it requires independent replication with broader samples. A *Nature* article published in 2011 says, "Some critics are also rankled by Baron-Cohen's history of headline-grabbing theories—particularly one that autism is an 'extreme male' brain state. They worry that his theory about technically minded parents may be giving the public wrong ideas, including the impression that autism is linked to being a 'geek'."

In 2001 he developed the Autism Spectrum Quotient, a set of fifty questions that can be used to help determine whether or not an adult exhibits symptoms of autism. Neuroscientist Francesca Happé questions whether the questionnaires produce objective enough results to be useful, as they rely on the subject's self-evaluation rather than independent observations. Psychologist Uta Frith agrees, and notes that "rigorous studies are still missing".

Baron-Cohen developed the Mindreading software for special education, which was nominated for an award from the British Academy of Film and Television Arts (BAFTA) interactive award in 2002. His lab developed *The Transporters*, an animation series designed to teach children with autism to recognise and understand emotions. The series was also nominated for a BAFTA award.

Organizations

Baron-Cohen is a Fellow of the British Psychological Society (BPS), the British Academy, and the Association for Psychological Science. He is a BPS Chartered Psychologist.

He serves as Vice-President of the National Autistic Society (UK), and was the 2012 Chair of the National Institute for Health and Care Excellence (NICE) Guideline Development Group for adults with autism. He has served as Vice-President of the International Society for Autism Research (INSAR). He is co-editor in chief of the journal *Molecular Autism*.

Recognition

Baron-Cohen was awarded the 1990 Spearman Medal from the BPS, the McAndless Award from the American Psychological Association, the 1993 May Davidson Award for Clinical Psychology from the BPS, and the 2006 Presidents' Award from the BPS. He was awarded the Kanner-Asperger Medal in 2013 by the *Wissenschaftliche Gesellschaft Autismus-Spektrum* as a Lifetime Achievement Award for his contributions to autism research.

Media appearances

In 2005, Baron-Cohen appeared in the 2005 Science Channel documentary *Brainman* about Daniel Tammet. In 2008, he confirmed the Asperger syndrome diagnosis of Gary McKinnon, the British computer hacker accused of breaking into United States military and NASA computer networks, and appeared in *Private Passions*, the biographical music discussion programme hosted by Michael Berkeley on BBC Radio 3. He presented a 2012 TEDx talk on the causes of cruelty at Parliament UK entitled "The erosion of empathy". His theory that high-functioning autism or Asperger syndrome can lead to a career choice in the sciences was explored in a talk at the Wired 2012 Conference.

Selected publications

Single-authored books

- *Mindblindness: An Essay on Autism and Theory of Mind*. MIT Press/Bradford Books. 1995. ISBN 978-0-262-02384-9.
- *The Essential Difference: Men, Women and the Extreme Male Brain*. Penguin/Basic Books. 2003. ISBN 978-0-7139-9671-5.
- *Autism and Asperger Syndrome*. Facts. Oxford University Press. 2008. ISBN 978-0-19-850490-0.
- *Zero Degrees of Empathy: A New Theory of Human Cruelty*. Penguin/Allen Lane. 2011. ISBN 978-0-7139-9791-0. (published in the US as *The Science of Evil: On Empathy and the Origins of Human Cruelty*, ISBN 978-0-465-02353-0)

Other books

- Baron-Cohen S, Tager-Flusberg H, Lombardo MV, ed. (2013). *Understanding Other Minds: Perspectives From Social Cognitive Neuroscience* (3rd ed.). Oxford University Press. ISBN 978-0-19-852446-5.
- Hadwin J, Howlin P, Baron-Cohen S (2008). *Teaching Children with Autism to Mindread: A Practical Guide for Teachers and Parents*. Wiley. ISBN 978-0-471-97623-3.
- Baron-Cohen S, Lutchmaya S, Knickmeyer R (2005). *Prenatal Testosterone in Mind: Amniotic Fluid Studies*. MIT Press/Bradford Books. ISBN 978-0-262-26774-8.
- Baron-Cohen S, Wheelwright S (2004). *An Exact Mind: An Artist with Asperger Syndrome*. Jessica Kingsley. ISBN 978-1-84310-032-4.
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External links

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