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Psychopathy, Etiology of

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Psychopathy is a clinical condition marked by interpersonal-affective characteristics (charm, manipulativeness, callous-exploitativeness) along with impulsive-irresponsible tendencies and persistent antisocial behavior. According to the triarchic model of psychopathy, the interpersonal-affective features are expressions of more basic dispositions termed *boldness* and *meanness*, whereas the impulsive-irresponsible tendencies reflect general proneness toward externalizing problems (*disinhibition*). Persistent engagement in antisocial activities is seen to arise from these basic dispositional tendencies operating jointly.

This entry covers what is known about causal influences contributing to psychopathy, using the triarchic model as a reference point. A synopsis of historical perspectives on etiology and evolutionary models is first provided, followed by a discussion of the genetic and environmental bases of psychopathy, the biological mechanisms contributing to it, and the developmental precursors of adult psychopathy.

Historical Ideas About Etiology

In 1976, American psychiatrist Hervey Cleckley theorized that the root cause of psychopathy is a general deficit in the capacity for emotional experience. Being unaware of this deficit on their part, psychopathic individuals learn to mimic the normal responses of others (including emotional reactions) in order to *blend in* and achieve desired goals, resulting in an outward *mask of sanity*. Benjamin Karpman, a contemporary of Cleckley, highlighted the importance of diagnostic subtypes in seeking to understand the causal bases of psychopathy. He distinguished between a *primary* subtype, presumed to be largely constitutional (heritable) in origin and entailing a core deficit in emotional sensitivity as hypothesized by Cleckley, and a *secondary* subtype, believed to arise more from adverse environmental influences and marked by excessive negative affect (anxiousness and hostility).

A 1957 study by David Lykken, considered the first empirical-laboratory investigation of psychopathy, tested the hypothesis that primary psychopathic delinquents meeting Cleckley's criteria differed in emotional (specifically, anxiety or fear) reactivity from secondary psychopathic delinquents exhibiting impulsive-antisocial behavior but lacking in Cleckley's core features. More recent cluster analytic studies have confirmed the existence of subgroups of psychopathic offenders that differ markedly in negative affectivity.

Evolutionary Models

Three main evolutionary models have been proposed to account for the persistence of genes contributing to psychopathy. The *balancing-selection model* proposes that within the totality of genes contributing to psychopathy, there is a subset that promotes certain adaptive qualities that offset the negative selection pressure conferred by the maladaptive aspects of the condition—that is, Cleckley's *mask* features (superficial charm, low anxiousness and immunity to internalizing problems, disinclination toward suicide). A second evolutionary model is the *antagonistic-pleiotropy model*, which posits that certain genes contributing to the expression of psychopathy have both evolutionarily beneficial and detrimental effects. However, the genes that are beneficial—in particular, those that promote precocious and unbridled sexual activity—exert their effects earlier in life than those that are detrimental, resulting in a net gain in reproductive effectiveness. The third model, termed the *frequency-dependent model*, hypothesizes that genes associated with psychopathy will contribute maximally to adaptive fitness in societies where they are uncommon. Specifically, in societies where most people are trusting and accommodating, manipulative-exploitative individuals will be especially successful at surviving and reproducing. However, as the proportion of such individuals increases and awareness of their adverse impact on society grows, the survival advantage will diminish due to increased caution and societal protections against such behavior.

Although helpful for thinking about the origins and maintenance of psychopathic behavior in society, evolutionary theories are limited in important respects. For one thing, these models assume that variations in psychopathic tendencies are driven by natural selection, but direct evidence for this is lacking and potentially unattainable. In addition, evolutionary models generally conceive of psychopathy as a unitary condition rather than a configuration of dispositional tendencies (i.e., boldness, disinhibition, and meanness, in triarchic model terms) that may have differing selective advantages and disadvantages.

Genetic and Environmental Influences

A growing literature has emerged on the role of genetic and environmental factors in psychopathy based on studies of monozygotic and dizygotic twins, who are presumed to share 100% and 50% of their genes, respectively—allowing for statistical estimation of sources of genetic and nongenetic influence in psychological characteristics. Twin research up through the 1990s focused on delinquent/antisocial behavior without consideration of core interpersonal-affective symptoms, yielding a limited picture of the etiology of psychopathy. Studies since the early 2000s focusing on psychopathic tendencies as a whole, assessed through self-report, have indicated heritability to be around 50%, with the remainder of variance attributable to nonshared (nonfamilial) environmental influences.

Other research has generated estimates for more specific subdimensions (facets) of psychopathy as described in the triarchic model. Studies focusing on the disinhibitory facet have yielded heritability estimates of around 80%. Initial work on the heritability of callous-unemotional traits (i.e., meanness) in young children suggested comparable heritability (~80%) for this facet of psychopathy. However, subsequent work indicates that this high heritability may characterize callous-unemotionality when accompanied by salient conduct problems (i.e., meanness plus disinhibition), with heritability lower for callous-unemotional traits per se (50–60%). Other twin work focusing on the boldness facet of psychopathy as assessed by self-report indicates heritability of around 50%.

In sum, available data point to moderate heritability for psychopathy scores as a whole, with distinctive facets of psychopathy exhibiting moderate to high heritability when examined separately. Of note, evidence for the high heritability of the disinhibitory facet challenges the notion that impulsive-antisocial behavior unaccompanied by salient affective-interpersonal features (i.e., so-called secondary psychopathy) is predominantly environmental in origin. Rather, it appears that there is a substantial heritability component to differing variants (subtypes) of psychopathy.

Biological Mechanisms

A number of theories have been advanced regarding biological mechanisms accounting for the observable symptoms of psychopathy. These can be grouped into two broad categories. One consists of theories that posit some underlying deficit in emotional reactivity, as suggested originally by Cleckley. Lykken presented the first experimental evidence that individuals meeting Cleckley's criteria were deficient in anxiety responses and later postulated that the various symptoms of psychopathy arise from a core deficit in fear reactivity. Other researchers have proposed that psychopathy entails an abnormally steep gradient of fear arousal or a weak behavioral inhibition (anxiety) system. Some have hypothesized that psychopathy results specifically from dysfunction in the amygdala, a subcortical brain structure involved in aversive learning and fear responding.

The other category consists of theories that postulate some sort of higher cognitive processing deviation. For

example, Joseph Newman and his colleagues proposed during the 1990s that psychopathy involves a basic impairment in *response modulation*, defined as the ability to switch from an ongoing (dominant) action set to an alternative mode of responding when environmental cues signal the need for a shift. Similarly, other cognitive theorists have postulated a deficit in the ability to detect and respond to peripheral stimuli when attention is directed toward a high-priority ongoing task. Yet another view is that psychopathy involves a dissociation between semantic-denotative and affective-connotative aspects of language—such that high-psychopathic individuals are adept at saying the right things but without normal conviction or follow-through. This linguistic dissociation model of psychopathy intersects with affective-deficit theories.

An alternative to theories positing a single core deficit accounting for all symptoms of psychopathy is the possibility that different mechanisms contribute to its distinguishable subdimensions. Evidence for this comes from research demonstrating contrasting biological correlates for the interpersonal-affective and impulsive-antisocial components of psychopathy and newer work documenting differential correlates for its boldness, meanness, and disinhibition facets. For example, a well-established correlate of the interpersonal-affective component is a lack of normal enhancement of the startle blink reflex to sudden noises presented during viewing of aversive visual images (e.g., frightening pictures) relative to neutral or pleasant images. This result indicates a lack of normal fear reactivity. Research with adults has shown this deficit to be associated specifically with the boldness facet of psychopathy.

Other work has shown that reduced brain potential response to target stimuli within cognitive performance tasks relates to the impulsive-disinhibitory component of psychopathy. This finding converges with a larger literature indicating impaired cortical-attentional responding in individuals with impulse-control problems more generally. Yet other research using functional neuroimaging has demonstrated deficits in amygdala reactivity to social distress cues (i.e., fearful human faces) in individuals scoring high on the callous-unemotionality (meanness) facet of psychopathy.

Taken together, these lines of evidence point to a role for more than just one type of biologically based processing deviation in psychopathy. Deficits in fear reactivity and impaired sensitivity to others' distress appear to be associated with the affective-interpersonal features of psychopathy (i.e., boldness and meanness, as described in the triarchic model), whereas reduced cortical-attentional processing appears to be associated more with its impulsive-disinhibitory features.

Developmental Processes

Child psychopathology research has identified distinct risk factors that are predictive of psychopathic tendencies across the life span. One important category of risk factors is subsumed by the notion of child temperament, referring to early emerging affective-behavioral tendencies presumed to have a strong basis in biology. The constructs of difficult temperament and fearless temperament are particularly relevant to psychopathy. Children exhibiting difficult temperaments tend to be irritable and easily distressed, high in activity, have problems adapting to novelty and change, and perform poorly in contexts requiring sustained attention. This temperament profile is associated with increased risk of early conduct problems and conflictual interactions with caretakers and peers—and as such, can be seen as inherently related to the impulsive-disinhibitory facet of psychopathy.

Fearless temperament, marked by venturesomeness and low reactivity to unexpected events or stressors, has been described both as a dispositional substrate for boldness and an early risk factor for callous-unemotional traits. On one hand, a lack of normal fearfulness could operate to smooth social interactions and facilitate adaptive coping and thus contribute to successful outcomes. On the other hand, weak fear conditioning and affiliated deficits in behavioral inhibition could result in the impairments in conscience and punishment

sensitivity observed in callous-unemotional children and psychopathic adults. As noted earlier, evidence for reduced sensitivity to aversive stimuli has been reported in relation to the affective-interpersonal features of psychopathy—both in terms of a lack of aversive startle potentiation (associated with bold tendencies in particular) and in terms of diminished reactivity to fearful face expressions (related to callous-unemotional [mean] tendencies especially).

However, deficits in early social attachment appear relevant to the interpersonal-affective features of psychopathy as well. For example, secure parent–child attachment, characterized by a developmentally appropriate balance of protectiveness and independence, has been shown to be predictive of positive outcomes across the life span. By contrast, insecure attachment can give rise to a defective internal working model of relationships, as evidenced by work showing insecure attachment to be more common in psychopathic offenders than other offenders. In particular, disrupted bonding—resulting from prolonged early separation from parents or deficits in parental care—is associated with higher psychopathy scores in adulthood. Other work indicates that child perceptions of parental relationship quality predict variations in callous-unemotionality, reflecting the importance of children's schematic understanding of interpersonal relationships. Importantly, child temperament may contribute in part to attachment strength. For example, children with difficult or fearless temperaments may pose greater rearing challenges that contribute to impaired parental relationships.

These lines of evidence highlight the importance of developmental processes and the complexity of forces that likely contribute to the emergence of psychopathic behavior across time. As opposed to one cause producing all symptoms, or separate specific causes accounting for distinct symptom facets, it seems likely that different causal factors contribute in interweaving ways to the observable symptoms of psychopathy—with alternative variants (subtypes) of psychopathy reflecting varying contributions of particular causal influences relative to others.

See also Juvenile Delinquents, Callous Unemotional Traits of; Psychopathic Offenders, Treatment of; Psychopathic Traits, Structure of; Psychopathy

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- psychopathy
- etiology
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Further Readings

Cleckley, H. (1976). The mask of sanity (5th ed.). St. Louis, MO: Mosby. (Original work published 1941)
Karpman, B. (1941). On the need for separating psychopathy into two distinct clinical types: Symptomatic and idiopathic. Journal of Criminology and Psychopathology, 3, 112–137.
Lykken, D. T. (1957). A study of anxiety in the sociopathic personality. Journal of Abnormal and Clinical Psychology, 55, 6–10.
Patrick, C. J., Fowles, D. C., & Krueger, R. F. (2009). Triarchic conceptualization of psychopathy: Developmental origins of disinhibition, boldness, and meanness. Development and Psychopathology, 21, 913–938. doi:http://dx.doi.org/10.1017/S0954579409000492
Skeem, J., Polaschek, D., Patrick, C., & Lilienfeld, S. (2011). Psychopathic personality: Bridging the gap be-

tween scientific evidence and public policy. Psychological Science in the Public Interest, 12, 95–162. Retrieved from https://doi.org/10.1177/1529100611426706