13
Antisocial Personality Disorder

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Abstract The present chapter discusses the medical understanding of antisocial personality disorder (APSD), including research concerning its etiology, prevalence, pathology, differential diagnosis, and treatment. ASPD, and the closely related diagnosis of psychopathy, appear to be products of a strong genetic disposition interacting with a variety of environmental contributions. Epidemiological studies indicate that ASPD and psychopathy are much more prevalent in men than in women, a finding that is supported by general personality research. Theories of pathology are numerous, but generally point to several distinct deficits; psychopathy has been associated empirically with abnormal affective processing, neuroanatomical abnormalities, psychophysiological arousal system impairments, deficits in cognitive functioning, and maladaptive personality constellations. While considered diagnostically reliable, ASPD and psychopathy are highly comorbid with substance dependence and narcissistic personality disorder due to similar criteria, making differential diagnosis difficult. Finally, treatment for psychopathy and ASPD remains a very controversial subject; while meta-analytic findings demonstrate positive results, considerable evidence also indicates that these disorders are resistant to typical interventions.

Keywords Antisocial • Psychopathy • Personality disorders • Pathology • Dimensional models

13.1. Definition

The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (1) defined antisocial personality disorder (ASPD) as a pervasive pattern of disregard for and violation of the rights of others. DSM-IV has been supplanted by the fifth edition of this diagnostic manual [DSM-5; (2)]. However, no changes were made to the personality disorders section. Therefore, all references to DSM-IV apply as well to DSM-5. DSM-5 does include within Section 3, for emerging models and measures, a reference to a dimensional trait model conceptualization of the personality disorders. This conceptualization is also included herein.

The primary diagnostic criteria for ASPD include criminal activity, deceitfulness, impulsivity, aggression, recklessness, irresponsibility, and indifference to the mistreatment of others. The DSM-IV conceptualization of ASPD was based substantially on the features of psychopathy originally outlined by Cleckley (3, 4). In fact, the text of the DSM-IV indicated that psychopathy is another term for the disorder (1). However, some have argued that the constructs of ASPD and psychopathy are not interchangeable due to the failure of DSM ASPD to include the breadth of Cleckley's psychopathy traits (5, 6). In support, the most widely recognized psychopathy measure, the Psychopathy Checklist-Revised (PCL-R) (7, 8), includes a few traits not found in the DSM-IV definition of ASPD: Glib charm, lack of empathy, shallow affect, and arrogance. Additionally, Cleckley identified other psychopathy traits not present in either the DSM-IV or the PCL-R criterion sets, notably the "absence of 'nervousness'" (3) (p. 206), which some suggest is a fundamental trait of psychopathy (9).

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TABLE 13.1 Domains and facets of the Five Factor Model (FFM).

Domains	Facets
Neuroticism (N)	N1: Anxiety
	N2: Angry hostility
	N3: Depression
	N4:Self-consciousness
	N5: Impulsiveness
	N6: Vulnerability
Extroversion (E)	E1: Warmth
	E2: Gregariousness
	E3: Assertiveness
	E4: Activity
	E5: Excitement seeking
	E6: Positive emotions
Openness to Experience (O)	O1: Fantasy
	O2: Aesthetics
	O3: Feelings
	O4: Actions
	O5: Ideas
	O6: Values
Agreeableness (A)	A1: Trust
	A2: Straightforwardness
	A3: Altruism
	A4: Compliance
	A5: Modesty
	A6: Tender-mindedness
Conscientiousness (C)	C1: Competence
	C2: Order
	C3: Dutifulness
	C4: Achievement striving
	C5: Self-discipline
	C6: Deliberation

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It is also helpful to understand ASPD from the perspective of general personality structure; more specifically, as a maladaptive variant of personality traits evident within the general population. Our preference is to use the five factor model of personality (FFM), the predominant dimensional model of general personality (10–12). The FFM includes five broad domains, each with six specific facets. The domains include neuroticism (N: anxiousness, angry hostility, trait depression, self-consciousness, impulsiveness, vulnerability), extroversion (E; warmth, gregariousness, assertiveness, activity, excitement seeking, positive emotions), openness to experience (O; fantasy, aesthetics, feelings, actions, ideas, values), agreeableness (A; trust, straightforwardness, altruism, compliance, modesty, tender mindedness), and conscientiousness (C; competence, order, dutifulness, achievement striving, self-discipline, deliberation). A complete list of the domains and facets of the FFM can be found in Table 13.1. The five domains of the FFM align with the five domains of the DSM-5 dimensional trait model (DSM-5 negative affectivity aligns with FFM neuroticism, DSM-5 detachment with introversion, DSM-5 antagonism with low agreeableness, DSM-5 disinhibition with low conscientiousness, and DSM-5 psychoticism with openness) (13).

Considerable research has been conducted using the five-factor model to understand psychopathy (14–17) and ASPD (17–20). Importantly, the five-factor model conceptualization articulates the similarities and differences between the psychopathy and ASPD constructs within a common framework. For instance, while both ASPD and psychopathy are represented by the A facets of low straightforwardness (deception), low altruism (exploitation), and low compliance (antagonistic aggression), psychopathy also includes the other A facets of low modesty (arrogance), low tender-mindedness (callousness), and low trust (suspiciousness). ASPD and psychopathy share several facets of C, including low dutifulness (irresponsible), low self-discipline (negligent), and low deliberation (rash). With regard to N, both ASPD and psychopathy are represented by high angry hostility and high impulsiveness. However, psychopathy is also characterized by the N facets of low self-

consciousness (glib), low anxiety (absence of nervousness), low depressiveness (self-contentment) and low vulnerability (fearless). In terms of E, both psychopathy and ASPD are represented by high excitement seeking (foolhardy), and high assertiveness (dominant), but the psychopath is also low in warmth (cold and distant). An advantage of conceptualizing psychopathy from the perspective of the FFM is that it allows for a clear distinction between the successful psychopath (who manages to avoid exposure or arrest) and the unsuccessful psychopath (21). The successful psychopath is characterized by the traits of high conscientiousness (self-discipline, achievement-striving, and competence), whereas, conversely, the unsuccessful psychopath by low traits of conscientiousness [rash, irresponsible, and negligent; (22)]. There is also now a published measure to assess psychopathy from the perspective of the FFM (23), which aligns well with the DSM-5 traits for ASPD (24), albeit the latter does not include traits involving low neuroticism (i.e., glib charm and fearlessness) or extroversion (e.g., dominance).

Thus, while ASPD and psychopathy appear to have substantial overlap, the pronounced differences with regard to aspects of personality indicate potentially meaningful divergence. This divergence is reflected in epidemiological and pathological differences across the two alternative conceptualizations. For this reason, these diagnoses will be discussed separately in the relevant sections. In addition, among the two conceptualizations, considerably more research has been conducted for psychopathy, particularly within the pathology domain. Again, while we do not want to use the disorders interchangeably, the weight of the psychopathy literature deserves consideration.

13.2. Etiology

There is considerable evidence of the heritability of antisocial behavior. In animal studies of temperament, selection studies (where brother-sister matings are carried out over many generations) have been successful in breeding rats for specific traits, including aggression, indicating that part of what is genetically transmitted is temperament (25, 26). In research with humans, the results of twin and adoption studies indicate a strong genetic component for antisocial behavior. Generally speaking, genetic factors are believed to account for approximately 50% of variation in antisocial behavior, although this estimate may be influenced by the interaction among genes, or between genes and environment (27, 28). However, when additive (interactive) and nonadditive (singular) genetic contributions are assessed, the genetic contribution remains resilient. Waldman and Rhee (29) provided results of a meta-analysis of 51 twin and adoption studies of antisocial behavior that indicated a substantial contribution of both additive genetic factors (*effect size* = .32) and nonadditive genetic factors (*effect size* = .09). These results indicate that specific, heritable genes may be important contributors to generalized antisocial behavior. Of interest, twin studies that have explored the development of antisocial behavior in children suggest that there may be differences in heritability of antisocial behavior dependent on the presence of psychopathic features. Viding and colleagues (30) found that the additive genetic heritability of antisocial behavior without traits of psychopathy is similar to prior estimates (30%) and the expression more heavily influenced by shared environmental factors, whereas antisocial behavior with traits of psychopathy is considerably more genetically-based (81%), with little or no influence of shared environment [see (31) for a review]. Other research has indicated that the stability of psychopathic traits is also primarily genetically-based (32), suggesting a chronic and refractory course. This may suggest that assessing for psychopathic traits may be of value when exploring the development of antisocial behavior, and this resilience to environment may be a factor in treatment development efforts for psychopathy.

Although no genes have been clearly identified as etiological precursors to ASPD or psychopathy, several candidates remain a focus of this research, including those that are thought to underlie the related predisposing disorder of attention-deficit/hyperactivity (ADHD), and those that are related to neurotransmitter systems relevant to aggressive and criminal behavior, such as the dopaminergic and serotonergic systems (29, 33, 34). In a 2006 review of this area, Minzenberg and Siever provided several genetic polymorphisms that are the focus of recent research in antisocial and aggressive behavior (33). Within the serotonergic system, alleles that are involved in the synthesis (*U* and *LL*), transportation (*s*), reception (*5HTR1B, 5HTR1A*), and metabolism (*MAO-A*) of neuronal serotonin have all been associated with anger, aggressive behavior, impulsivity, and antisociality, as have several receptor polymorphisms (*DRD2, DRD3, DRD4*) and genes related to metabolism (DBH) of the dopaminergic system, and catechol-O-methyltransferase (*COMT*), a polymorphism associated with the breakdown of dopamine and norepinephrine [see (33, 35) for a review]. A 2012 review of genetic contributions specific to psychopathy has implicated the valine allele of the COMT gene, the low activity allele of the MAOA gene (MAOA-L), and the short allele of the serotonin transporter linked polymorphic region gene (5-HTTLPRs) (36). However, as others have noted, this area of research is still very new and almost no replication of these findings has been achieved, suggesting that these preliminary findings are not yet considered conclusive evidence of any specific genetic contribution (31).

Numerous environmental factors have also been implicated in the etiology of antisocial behavior. Shared, or common, environmental influences account for 15% to 20% of variation in criminality or delinquency (28, 37). This finding is remarkably robust even when compared to other psychiatric disorders with known environmental components such as affective and substance use disorders (38), and indicates something distinct about the shared environmental influence on antisocial behavior.

The modeling or learning of aggressive behaviors is more likely to occur in environments that have higher incidents of this type of behavior, or that condone antisociality and violence (39). Not surprisingly, shared environmental factors such as low family income, inner city residence, poor parental supervision, single-parent households, rearing by antisocial parents, delinquent siblings, parental conflict, harsh discipline, neglect, large family size, young mother, and depressed mother have all been implicated as risk factors for antisocial behavior (40). The effects of these factors are not limited to learning, however. For instance, neglect and physical abuse can generate several possible courses to antisocial and aggressive behavior, such as desensitization to pain, impulsive coping styles, changes in self-esteem, and early contact with the justice system (41). Nonshared environmental influences are also substantial contributors. Factors specific to the individual appear to account for fully 30% of antisocial behavior variance (27). In short, this is the remaining variance not accounted for by genetic (50%) or shared environmental (20%) influences. Nonshared environmental factors may include delinquent peers, individual social and academic experiences, sexual abuse, or sustaining an injury not shared by siblings, such as a head injury.

Unfortunately, the interactive effects of genetic and environmental influences are difficult to tease apart, and likely create confusion about what these estimates mean in terms of causation. For example, the individual who is genetically predisposed to antisocial behavior will subsequently elicit environmental factors associated with criminal outcomes, such as peer problems, academic difficulty, and harsh discipline from parents. In addition, antisocial individuals receive their genes from antisocial parents who also exhibit delinquent and irresponsible behavior, thus creating an immediate home environment that is likely to model instability and criminality. Concerns surrounding the interaction of environmental and genetic factors have led to research designs that have focused more directly at making these distinctions. Studies that explicitly address this issue have found that environmental factors continue to play a large part in etiology of antisocial behavior beyond genetic factors alone. For instance, after controlling for the genetic component of physical maltreatment, Jaffee, Caspi, Moffitt, and Taylor (42) found that the environmental etiological effect of physical maltreatment remained.

In addition to genetic and environmental influences, other work has focused on the relative contribution of the interaction of genes and environment to the development of antisocial behavior. For example, Caspi and colleagues (34) found that MAOA interacted with adverse environment to form a vulnerability to antisocial spectrum behaviors in children, suggesting that the phenotype of antisocial behavior is much more than a sum of the genetic and environmental parts. However, these findings are difficult to replicate (43, 44), indicating that considerably more work is needed in this area to understand the relative contributions of genes, environmental influences, and their interactions.

13.3. Epidemiology

The prevalence of ASPD in the general population indicates strong gender differences, with higher incidence in men than in women. Using the Diagnostic Interview Schedule (2), the Epidemiologic Catchment Area study estimated ASPD prevalence to be 4.5% in men and 0.8% in women (45). Similarly, the National Comorbidity Survey (NCS) indicated substantial gender differences, with 5.8% of men and only 1.2% of women meeting ASPD criteria (46). In addition, ASPD prevalence rates tend to be similar across all races. For example, ECA estimates demonstrated little difference between African American and Caucasian races (2.3% vs. 2.6%, respectively), suggesting that ASPD tends to present with equal incidence across race and ethnicity (45).

In contrast to the substantial epidemiological research conducted for ASPD, studies of the prevalence of psychopathy are lacking in number and scope. Importantly, psychopathy prevalence estimates have previously been based primarily on incarcerated samples, thereby making comparison with general population ASPD epidemiology difficult. Many individuals in corrections settings meet the criteria for ASPD, thus raising the prevalence rates to 50% to 60% for incarcerated offenders (5). Psychopathy prevalence rates in prisons tend to be significantly lower than those for ASPD [estimated from 15.0% to 7.7% for men and 7.0% to 1.9% in women in corrections settings (7, 47)], leading researchers to believe that psychopathy must be quite rare in the broader general population. Low prevalence rates appear to be supported in empirical findings; the few epidemiological studies using psychopathy-specific criteria have estimated prevalence rates between 0.6% (48) and 3.6% (49), although these two studies were based upon the same small sample using different cutoff criteria.

It should be noted that these prevalence differences between ASPD and psychopathy, and the relative "rarity" of psychopathy, may be indicative of a confound between the criteria and the correctional setting. It has been suggested that the heavy weighting of the DSM-IV (and DSM-5) ASPD criteria toward criminal and delinquent behavior inflates ASPD prevalence in prison settings due to the nature of a correctional population (50). In addition to the behavioral elements of ASPD, the diagnosis of psychopathy is contingent on the presence of several personality traits (e.g., glib charm, arrogance) that would not necessarily be intrinsic to correctional populations. Because of this asymmetric criterion overlap, it is little wonder that 90% of incarcerated offenders who meet the PCL-R criteria for psychopathy also meet the behavioral criteria for ASPD, but as few as 30% of those with ASPD also meet the trait criteria for psychopathy (51). It may be that the widely accepted incidence differences between ASPD and psychopathy would cease to exist (or even be reversed) in other populations where the psychopathy traits of manipulation and glib charm are emphasized, such as the professions of law or politics (50).

Very few studies have exclusively focused on racial or gender differences in psychopathy prevalence. At this point, there is little evidence that psychopathy exists differentially across race in terms of how the construct validity is preserved (52–54) although a handful of studies have reported a higher incidence in African Americans than Caucasians or European Americans (55, 56). Gender differences in psychopathy prevalence are generally consistent with the ASPD findings (57), indicating that women are less psychopathic than men overall (58). Known gender differences in the facets of the FFM (59) may explain why. For example, Costa et al. (59) report that women score much higher on all facets of agreeableness and neuroticism than men, as well as on the warmth and positive emotions facets of the extroversion domain, and the dutifulness facet of the conscientiousness domain. Additionally, women score lower than men on the excitement seeking and assertiveness facets of extroversion. In sum, the facets in which the psychopath is low (see Definition section) are precisely those facets in which men tend to score lower than women (e.g., all facets of agreeableness, the anxiety, depression, self-consciousness and vulnerability facets of neuroticism, the warmth facet of the extroversion domain, and the dutifulness facet of the conscientiousness domain). Likewise, the facets in which the psychopath is high are facets in which men score higher than women (e.g., the excitement seeking and assertiveness facets of extroversion). That is, the facets of general personality structures involved in psychopathy are ones that are more characteristic of men than women. Thus, from a personality standpoint large gender differences in psychopathy are to be expected. Despite gender differences, evidence to date suggests that when clinical levels of disorder are present, psychopathy and antisocial traits look remarkably similar in presentation, and lead to similar outcomes (60, 61).

13.4. Clinical Picture

According to the DSM-IV (and DSM-5), a diagnosis of ASPD is contingent upon the early manifestation of conduct problems with onset before age 15 years, thereby documenting a stable and pervasive pathology. In adulthood, the antisocial individual has little regard for societal norms, and is often engaged in unlawful behaviors such as gambling, stealing, drug use, and destruction of property. Irresponsibility, recklessness, and impulsivity are hallmark features of ASPD. The antisocial individual is often unable to plan ahead, and generally fails to consider the consequences of his hedonistic actions to himself or others. This failure to construct organized plans and deliberate about the consequences of behavior creates pervasive instability in many areas of the antisocial individual's life, both in personal and professional domains. The employment histories of those with ASPD are often marred by unexplained absences and early terminations from jobs, and personal relations tend to be short-lived, and filled with strife and conflict. Further, antisocial individuals are often irritable and aggressive, leading to numerous physical and verbal altercations with others. Contact with the legal system is not uncommon for those with ASPD. Interpersonally, ASPD individuals are known to be remorseless, exhibiting little or no consideration for those whom they harm with their delinquent acts. In addition, those with ASPD are notoriously deceitful and manipulative, and are known for their ability to lie, con, and cheat others without detection.

As stated previously, the psychopathy criteria of the PCL-R have considerable overlap with the DSM-IV (and DSM-5) ASPD criteria. Both conceptualizations call for early diagnosis of conduct problems (although childhood conduct disorder is not in fact required for the PCL-R), and indicate several similar traits and behaviors, such as failure to plan ahead, impulsivity, delinquent and criminal behaviors, irresponsibility, remorselessness, and deceitfulness. However, the psychopathy criteria of the PCL-R also include a few personality characteristics absent from the DSM-IV (and DSM-5) ASPD criterion set, specifically glibness, arrogant self-appraisal, lack of empathy, and shallow affect (62). These indicators might suggest that the psychopath is more charming, self-assured, and cold-hearted than his ASPD counterpart, thereby making the psychopath seem both capable of, and successful at completing the most heinous of crimes.

An additional psychopathy criterion that has remained absent from both the ASPD and PCL-R conceptualizations is the absence of anxiety. According to Cleckley, the psychopath "appears almost as incapable of anxiety as of profound remorse," (3) (p. 340) and demonstrates "a relative immunity from such anxiety or worry as might be judged normal or appropriate" (p. 206). Many experts in the psychopathy field continue to support Cleckley's assertion that the psychopath is low in anxiousness (14) although this criterion ultimately failed to appear in the PCL-R due to poor item-total correlations (63). In sharp contrast to psychopathy, ASPD is said to be associated with high levels of anxiety and other affective disorders (1). The DSM-IV stated that individuals with ASPD "may also experience dysphoria, including complaints of tension, inability to tolerate boredom, and depressed mood" (p. 702) and may be prone to both anxiety and depressive disorders (1). While the presence of anxiety disorders may be an artifact of the psychiatric samples traditionally used to study ASPD, epidemiological studies also support the diagnostic comorbidity of ASPD and anxiety in community samples, suggesting that the relation is resilient beyond the clinical domain (64, 65). Thus, in the anxiety domain, the clinical pictures of psychopathy and ASPD are strikingly different in how they present. Further research is needed to better understand why these conceptualizations diverge in their respective relations to anxiety and to provide insight into whether this divergence is clinically meaningful to outcomes.

The inclusion of additional personality criteria in the psychopathy conceptualization also indicates that psychopathy has a heavier weighting toward the interpersonal and affective traits associated with crime than ASPD. The strong behavioral

focus of the ASPD criteria has received extensive criticism, as it makes the assumption that criminal behavior, rather than personality features, is a primary symptom of the disorder (5, 52). Hare makes explicit use of both behavioral and personality characteristics in the PCL-R, and has designated these domains as separate but equal through a two-factor structure. Hare's original PCL-R two factor solution characterized Factor 1 as consisting of the affective and interpersonal set of items termed the "selfish, callous, remorseless use of others", and Factor 2 as the behavioral criteria which he termed the "chronically unstable, antisocial, and socially deviant lifestyle" (52) (p. 79). Many studies have indicated that the ASPD criterion set correlates more highly with Factor 2 than with Factor 1 [e.g., (8, 62, 66, 67)], thereby supporting the heavy concentration of behaviors and the relative lack of personality characteristics in ASPD. However, while smaller than the relations with Factor 2, correlations between PCL-R Factor 1 and ASPD are significant, and indicate that at least some personality features are represented in both conceptualizations. In addition, studies of the ASPD criterion set have also indicated a two-factor structure, with facets that distinguish between the callous exploitation of others and impulsive disinhibition (68, 69), indicating that interpersonal characteristics play at least some part in the diagnosis of ASPD, albeit a more minor role. It should also be acknowledged that despite a concerted effort by the authors of the PCL-R to include distinct interpersonal and affective characteristics, much of the assessment of the PCL-R personality traits relies heavily on the existence and consideration of criminal behaviors. Due to this saturation of antisocial behavior, the PCL-R has received criticism comparable to the ASPD criterion set (12). To date, it remains unclear whether the PCL-R can be effectively applied within non-criminal settings, as the reliable assessment of antisocial activity becomes much more difficult in such populations.

While criminal and irresponsible behaviors appear to be important to the construct of psychopathy, some maintain that antisocial behavior deserves no role in the diagnosis of psychopathy whatsoever due to its role as a consequence, rather than a symptom, of the disorder (70). These authors argue that while trait descriptions of psychopathy characterize an individual who is prone to delinquency and antisociality, criminal behavior itself may arise from many alternative sources, with psychopathic personality being only one potential cause (71). By designating behavioral criteria as primary, rather than secondary symptoms, a diagnosis of ASPD may be given regardless of the actual genesis of the antisocial acts. Research using Structural Equation Modeling (SEM) supports a secondary hierarchical position for behavioral symptoms in psychopathy (72). Model fit estimates indicated that the simultaneous inclusion of behavioral items from the PCL-R (e.g., criminal behavior, criminal versatility, promiscuous sexual behavior) with impulsive, interpersonal and affective PCL-R items resulted in worse fit estimates than using impulsive, interpersonal and affective PCL-R items alone (72), and "actually degraded the measurement of psychopathy" with their inclusion (70) (p. 98). SEM fit estimates improved dramatically when behavioral items were placed as products (consequences) of the impulsive, affective, and interpersonal factors, leading Cooke and colleagues to argue that "it may be time to 'reconstruct' psychopathy by reducing or eliminating reliance on criteria that are overly saturated with antisocial and deviant behavior, thus putting personality back at the heart of this personality disorder" (70) (p. 99).

Work has begun in placing psychopathy back into the realm of personality. Trait-based alternatives to PCL-R assessment are beginning to gain credence, and demonstrate adequate reliability and validity as indicators of psychopathy (73). Among these are the Psychopathic Personality Inventory (9) and the FFM conceptualization of psychopathy (14), both of which have demonstrated positive associations with criminal and delinquent behaviors (9, 14, 74), convergence with other psychopathy measures (75, 76), and predicted relations to other known correlates of psychopathy including performance on laboratory tasks of aggression and deliberation (15). Thus, the assessment of psychopathy does not appear to be reliant on antisocial behavior, and can be achieved through a personality-based measure.

13.5. Pathology

Considerable research effort has been focused on the pathology of antisocial behavior. Within this domain, various proximal pathways to ASPD have been advanced, including psychoanalytic defenses, neuroanatomical abnormalities, psychophysiological arousal system impairments, deficits in cognitive functioning, and personality factors. Interestingly, rather than supporting one causal factor, this extensive research base indicates that many deficits are involved in antisocial behavior, leading to a very complex picture of pathology.

13.5.1. Psychoanalytic Defenses

The historical conceptualization of antisocial pathology comes from psychoanalytic thought. The antisocial individual was believed to suffer from "superego lacunae" or holes in the conscience (77). This superego pathology is associated with an "incapacity to experience self-reflective sadness" that ultimately results in callous, tough-minded behavior (78). This classical picture of the psychopath was modified in later conceptualizations, and is reflected in Cleckley's and Hare's descriptions of "semantic

dementia," where abnormal affective processing is the prime feature of the psychopath's pathology (3–5). Hare has described the psychopath as being "without conscience," a deficit that ultimately results in ruthless, manipulative, cold-hearted, and violent behavior (79). This prevailing and longstanding conceptualization of psychopathic pathology has pervaded the research, and has recently been extended into laboratory task designs (80). Studies assessing the psychopath's autonomic reaction to emotional words and fearful images appear to be supportive of abnormally deficient affective processing, although the psychopath's cognitive reports of emotional responses have been found to be similar to those of nonpsychopaths (5, 80).

13.5.2. Neuroanatomical Abnormalities

Structural and functional brain impairments have also been advanced as possible underlying pathologies of antisocial behavior (81, 82). Reviews of brain imaging studies of antisocial populations implicate abnormal functioning in the temporal cortex (83, 84), amygdala and hippocampus (85, 86), angular gyrus (87), and prefrontal cortex (87–89). Research in the psychopathy domain suggests much more widespread structural and functional issues, ranging from reduced volumes of the amygdala to abnormal shape of the hippocampus, and aberrant activity in all four lobes of the cortex (frontal, temporal, parietal and occipital), as well as several subcortical structures [see (90) for a review].

The neural dysfunctions implicated appear to be generally consistent with the existing research on both antisocial behavior and psychopathy. However, emerging research in this area suggests that there may also be subtle functional differences between psychopathy and antisocial behavior; work with children suggests that conduct disorder is associated with *increased* amygdalar activity in affective scenarios, but psychopathy is associated with *decreased* amygdalar activity in these same scenarios (91). Further, it has been demonstrated that for typical children and children with ADHD, unexpected punishment results in a reduction in ventromedial prefrontal cortex activity, whereas there is no such reduction for children with psychopathic traits (92). It may be the case that broad abnormalities are similar across disorders, but distinct areas of dysfunction exist, particularly for brain activity surrounding emotion and reward pathways.

While functional and anatomical deficits appear to be fairly replicable, causal conclusions have yet to be determined. Environmental factors may also play a part in creating neural abnormalities in antisocial individuals. For example, closed head injuries, drug and alcohol abuse, and early health factors may serve to exacerbate a genetic propensity, rather than act independently.

13.5.3. Psychophysiological Arousal System Impairments

Another influential theory of ASPD pathology comes from Gray's three arousal model of the nervous system (93). Briefly, this model entails the interaction of three neurophysiological arousal systems that are hypothesized to control behavior. The behavioral inhibition system (BIS) is said to inhibit behavior in response to punishment, in opposition to a behavioral activation system (BAS) that activates behavior in response to reward. The overarching nonspecific arousal system (NAS) can be activated by either the BIS or BAS system. Activation of the NAS generally results in an increase in arousal, with the valence of this arousal (inhibit or interrupt vs. approach) directed by the BIS or BAS. Within this context, normal, adaptive functioning is reliant on the balance of activation between the arousal systems. The observed symptoms of ASPD could be evidence of a malfunctioning BIS acting in concert with a normal or strong BAS (94, 95). In this manner, normal sensitivity and anxiety in response to threatening and stressful situations may be reduced or altogether absent in the antisocial individual. Low arousal may also be a factor in the observed deficits in feelings of guilt or remorse and may serve to increase resistance to aversive conditioning.

In support of Gray's model as applied to ASPD, many psychophysiological deficits have been associated with psychopathy. Lykken's (96) classical conditioning paradigm demonstrated that psychopathic inmates had abnormally low physiological responses (reduced skin conductance) to a conditioned stimulus paired with electric shock, indicating that the psychopath does not develop the expected anticipatory arousal from threat of physical punishment. Additionally, this conditioning showed a more rapid extinction in the primary psychopathic group when compared to secondary or "neurotic" psychopaths. Although low skin conductance is widely discussed in the literature, Raine's (97) review of this research indicates that this finding has not been altogether consistent. In contrast to Lykken's findings, contemporary research does not support group differences in skin conductance levels for psychopathic versus nonpsychopathic offenders (97). Interestingly, while low skin conductance has been associated with crimes of evasion [e.g., white collar crimes and customs offenses; (98)], it has not been found to be associated with other criminal activity, such as violent offenses (98). Additionally, although low skin conductance is associated with later institutionalization in behaviorally disordered children, it does not appear to be predictive of arrest (99).

Other autonomic arousal assessments have also been used to investigate psychophysiological functioning in the psychopath, including heart rate and startle response (100, 101). Low resting heart rate levels have been associated consistently with antisocial behavior in noninstitutionalized individuals, providing support for Gray's theory (97). However, studies of incar-

cerated populations generally fail to find group differences between psychopaths and nonpsychopaths, indicating that this finding may be a predisposing factor to antisocial behavior in general rather than psychopathy (97).

Some have argued that it is not generalized arousal deficits, but arousal deficits associated with the experience of emotion that best characterizes the psychopathic dysfunction (102). Interestingly, Casey, Rogers, Burns, and Yiend (2013) found that individuals with higher psychopathy scores were more autonomically responsive (as assessed by cardiovascular activity) when processing negative information, possibly indicating that psychopaths find unpleasant material somewhat rewarding (103). Emotionally valenced startle response tasks have also demonstrated reliable psychopathic psychophysiological deficits. Patrick, Bradley, and Lang (101) found that psychopaths do not show normal startle potentiation when viewing negatively valenced photos, although normal attenuation of startle was documented with positively valenced photos. Startle response deficits have been replicated numerous times, and may be considered supportive of a generalized deficit in behavioral inhibition dysregulation (104–107).

13.5.4. Deficits in Cognitive Functioning

Cognitive functioning deficits have also been implicated in the pathology of antisocial behavior. Historically, psychopathy has not been associated with "classic" cognitive dysfunction (e.g., intelligence, memory, executive ability), as the psychopath typically appears to be intact in most of these areas (3, 108). In fact, recent evidence indicates that violence is positively correlated with intelligence scores in psychopathic adults (109), and psychopathy scores are positively related to verbal, analytic, creative, and practical abilities in children (110, 111). However, the psychopath's notorious disconnect between successful planning and understanding of contingencies and subsequent violent, impulsive behavior indicates that a psychopathic cognitive deficit may exist, albeit in a more subtle form (112, 113).

Existing literature on the cognitive attributes associated with psychopathy indicates that the psychopath experiences stable deficits in the cognitive domains of attention (113, 114) and response modulation (115, 116). Laboratory task paradigms designed to assess the allocation of attention indicate that despite intact perceptual and autonomic processes, the psychopath is unable to switch attention from an ongoing task to secondary (or peripheral) information when appropriate (114, 117–119). The deficits in attention associated with psychopathy have been incorporated into the limbic dysfunction literature, and contribute to what Newman has coined the "response modulation hypothesis" (116). Many researchers believe that this may underlie the behavior control problems that characterize psychopathy (116). According to Newman, psychopaths continue approach behaviors even while maladaptive, and are unlikely to consider contextual information that may be helpful in choosing alternate responses (115). Newman, Patterson and Kosson (116) explored the inability of the psychopath to inhibit a dominant response to a card playing task of worsening odds and found that psychopaths continued for more trials of unlikely success with a dominant response set in comparison to nonpsychopaths. This effect has been replicated several times over, with different forms of stimuli and in conjunction with event-related brain potentials, and continues to a productive area of research in the pathology of psychopathy (116, 119, 120).

13.5.5. Personality Factors

Finally, personality differences are also considered an important aspect of the pathology of ASPD and psychopathy. Antisocial behavior has been associated with various personality traits and trait-like behaviors which are believed to underlie the construct, such as aggressiveness, impulsivity, sensation-seeking, lack of empathy, and impairments in cognitive functioning (14, 121). Eysenck's theoretical framework placed personality between the physiological processes of arousal and antisocial behavior, implying that personality moderates the relation (122). In other words, physiological functioning deficits may or may not develop into antisocial behavior depending on the personality characteristics present in the individual.

Indeed, Cleckley's description of the psychopathic personality is a testament to the importance of this aspect to the construct of ASPD. Since Cleckley's time, many other researchers have proposed personality-based models to understand psychopathy. Lykken's fearlessness hypothesis (96) proposed that the absence of anxious behaviors typically demonstrated by psychopaths is due to the psychopath's deficient emotional response to punishment or danger. In a description of this deficit, Lykken states that for the psychopath, "the fear of punishment and the coercive voice of conscience both are, for some reason, weak or ineffectual" [(123); p. 134]. According to Lykken, this absence of fear allows the psychopath to remain collected in high-stress situations, and inoculates against anxiety disorders. Rather than considering fearlessness as a correlate of psychopathy, Lykken considered it a precursor to the disorder.

Other models of psychopathic trait pathology abound, and have gravitated toward integrating dimensional models of personality with the psychopathy literature. Rather than focusing on individual characteristics, dimensional models of personality disorders incorporate the broad spectrum of personality to improve predictive capacity (124). By viewing psychopathy as a constellation of personality traits, the model can be used to subsume existent literature on the notable deficits associated

with ASPD and psychopathic pathology (16). The multifaceted nature of psychopathy is reflected in the varied pathology; simply put, different investigators are exploring different aspects of the psychopathy profile. For instance, the disinhibition and poor deliberation associated with response modulation deficits are likely representative of low conscientiousness, whereas the lack of empathy and ruthlessness of semantic dementia appear to represent low agreeableness or antagonism. Likewise, Lykken's fearlessness hypothesis seems to relate to hasty decision-making and recklessness, traits also associated with low conscientiousness. While an elegant conceptualization, the dimensional modeling of psychopathy remains in its early stages, and proposed mappings of traits to deficits have yet to be tested empirically.

13.6. Clinical Course

Although ASPD and psychopathy are considered pervasive disorders, the specific antisocial behaviors associated with these diagnoses tend to remit with age (3, 4, 125). Robins' influential longitudinal study of delinquent children indicated that approximately 40% of antisocial youths show a reduction in antisocial activity in adulthood, and that the median age of clinical improvement was 35 years (4). Similar findings have been reported in the psychopathy research, albeit with slightly higher age estimates for remission of symptoms (125, 126). In addition, cross-sectional prevalence estimates in prisoners reflect this trend with a linear decline in PCL-R and ASPD scores beginning at age 20 (127). Simply put, there appears to be a higher prevalence of ASPD and psychopathy in prisoners between the ages of 20 to 40 than after age 40. However, the clinical improvement documented is relative to the group; before the drop in criminal behaviors, psychopathic individuals participate in more criminal activity, have higher conviction rates, and serve longer sentences than nonpsychopathic offenders, and after age 40, conviction rates drop but remain comparable for psychopathic and nonpsychopathic criminals (125, 127). Thus, while the reduction of criminal behaviors over time is significant for the psychopath, this "improvement" merely renders them comparable in criminality to their nonpsychopathic counterparts.

Interestingly, while the psychopath appears to "age out" of his criminal activity over time, there is evidence that the personality characteristics that accompany psychopathy remain remarkably stable. In their cross-sectional study, Harpur and Hare (127) demonstrated that the psychopathy factors were differentially related to age; while Factor 2, which assesses the "traits and behaviors associated with an unstable and antisocial lifestyle" (p. 605) was found to have the predicted negative relation with age, Factor 1, which describes the "affective and interpersonal traits central to the classical clinical descriptions of the psychopath [including] egocentricity, manipulativeness, callousness, and lack of empathy" (pp. 604–605) was unrelated to age. In fact, Factor 1 scores of the 15–20 year-old age group were strikingly similar to Factor 1 scores of the 46–70 year-old age group, indicating that the personality characteristics present in Factor 1 show no significant age reduction. Thus, although criminal behaviors become less prevalent over the life course, the traits associated with psychopathy appear to continue to cause problems for the psychopath long after his criminal career ends.

The personality literature also supports these findings. Longitudinal studies of the NEO PI-R indicate that the factors of agreeableness and conscientiousness tend to increase across age (128). Importantly, these domains are those believed to be most important to psychopathy, ASPD, and antisocial behavior in general (15, 16, 124). Thus, independently of the psychopathy and ASPD research, predictions about the course of these disorders are supported from the broad personality literature.

13.7. Differential Diagnosis

Differentiation between ASPD and psychopathy and other DSM-5 diagnoses can be problematic as many other disorders may present with overlapping symptoms. For instance, the ASPD criteria of irresponsibility, aggressiveness, and impulsivity may also be associated with DSM-5 diagnoses such as schizophrenia, bipolar disorder, or major depression. In fact, longitudinal studies of delinquent children indicate that early conduct problems can sometimes be predictive of adult manifestations of schizophrenia, rather than ASPD (4). Conversely, the substance abuse and psychiatric malingering associated with ASPD and psychopathy may initially present as schizophrenia, also leading to difficulties in diagnosis. However, the antagonistic personality criteria (e.g., deceitfulness and lack of remorse), lack of psychotic symptoms, and pervasiveness of ASPD and psychopathy typically allow for sufficient differentiation between these disorders and most other DSM-5 diagnoses. Despite this, two disorders continue to cause concerns in categorical diagnosis of ASPD. Substance use disorder and narcissistic personality disorder remain difficult to distinguish from ASPD, and in fact substance use and antisocial behaviors have been shown to form a coherent "externalizing factor" according to epidemiological studies (129), suggesting that some variety of true overlap in pathology exists. For the purposes of this chapter, substance use disorders and narcissistic personality disorder will be discussed to identify potential divergence in diagnostic features and to inform treatment efforts.

Despite the relative reliability of the diagnostic criteria for ASPD, controversy remains about the adequate differentiation between ASPD/psychopathy and substance use disorders. Comorbidity estimates indicate that ASPD is strongly associated

with substance use disorders (1), and in the ECA study, 84% of those diagnosed with ASPD also reported some form of substance use or abuse (64). Additionally, PCL-R scores are strongly associated with substance use, particularly with Factor 2 of the PCL-R (130). However, the comorbidity estimates reported may be indicative of overlapping criterion sets (131); the history of those involved with dyscontrolled drug use generally include some of the same traits and behaviors associated with ASPD and psychopathy, including theft, deception, poor work history, and irresponsibility. Thus, differentiation between the disorders remains difficult. While suggestions have been made to incorporate exclusion criteria for DSM-IV ASPD in lieu of substance use disorder presence (132), the early onset of behavioral problems specific to ASPD (versus the lack of early onset for substance use disorders) has been presumed to be an adequate differentiation criterion, ultimately preventing a substance use exclusion criterion from being included in revisions of the DSM (50). On the other hand, both disorders appear to share a common underlying pathology and course (133, 134), and each may contribute to the development of the other, making the use of the early onset criterion a troublesome differentiation factor.

The other psychiatric diagnosis that is often reported to be comorbid with psychopathy and ASPD is narcissistic personality disorder [NPD; (135, 136)]. In contrast to the criterion overlap with the irresponsibility and antisocial behaviors associated with substance use, NPD appears to share the manipulative, exploitative, and callous traits associated with ASPD and psychopathy. In support, PCL-R total scores and Factor 1 scores correlate significantly with NPD, but Factor 2 scores do not (66), a pattern of correlations that is the mirror image for ASPD. While the clinical and theoretical literatures of NPD and ASPD/psychopathy have grown independently, psychodynamic views generally incorporate narcissism into the psychopathy conceptualization (137, 138). In fact, many have explicitly suggested that NPD is a lower-order facet of the psychopathy construct, and have argued that NPD is a closer conceptualization of psychopathy than ASPD (139). However, due to concerns about diagnostic overlap and differentiation, authors of the DSM-IV ultimately decided to incorporate components of the PCL-R (i.e., lack of remorse) into the ASPD criterion set to increase the validity of the assessment of ASPD within prisons and other forensic settings (140).

From a personality standpoint, the diagnostic comorbidity and poor differentiation between personality disorders is understandable, and even predicted. Dimensional models of ASPD and psychopathy indicate that these disorders obtain diagnostic comorbidity with other DSM-IV (and DSM-5) personality disorders to the degree that they share overlapping constellations of personality traits (141). Lynam and Derefinko (142) conducted a comparison of predicted comorbidity (based on expert-generated personality prototypes) and empirical comorbidity between the psychopathy and the DSM personality disorders. Expert prototype predictions indicated that psychopathy would share the highest comorbidity with ASPD (with shared low agreeableness and low conscientiousness), followed by NPD (with shared low agreeableness). These predictions were supported by meta-analytic results of empirical findings (142).

13.8. Treatment

There is considerable debate surrounding the efficacy of treatment for ASPD and psychopathy (143). Although some treatment-outcome research has indicated positive results for therapeutic interventions (144–147), other empirical evidence appears to suggest that the antisocial behaviors associated with ASPD and psychopathy are resistant to intervention, particularly for the psychopath (148–150). Authors on both sides of the argument cite significant shortcomings in the existing treatment-outcome research, such as the lack of control groups, the use of clinically insignificant outcome measures, the use of inappropriate treatment strategies, high variability in results, and the clinical (vs. statistical) meaningfulness of effect sizes (143, 147, 151, 152). All of these factors contribute to the confusion about whether treatment of those with ASPD and psychopathy is a viable pursuit, or should be abandoned in favor of traditional management through incarceration.

Several studies indicate that psychopaths benefit less from treatment than nonpsychopaths, demonstrating higher attrition rates, lower clinical improvement, higher violent recidivism, and more immediate recidivism upon release than their nonpsychopathic counterparts (153–155). In addition, high PCL-R scores (particularly Factor 1 scores) have been associated with significantly higher recidivism rates in treated psychopaths than untreated psychopaths, suggesting that psychopaths actually get worse with therapy (148, 149, 156). Thus, it is generally accepted that "nothing works" with respect to treating psychopathy (157); the psychopath seems to benefit less from treatment than nonpsychopaths, and in some cases, therapeutic interventions appear to increase future criminal activity in the psychopath, indicating that management, rather than treatment, may be the prescribed course for this type of offender (158).

This pessimism regarding treatment is enhanced by the over-reliance on findings of a handful of landmark studies that report notably disappointing outcomes (159). This is unfortunate, given that some of the studies cited are of questionable scientific value. For instance, in the most famous treatment outcome study of psychopathy, the Penetanguishene study, Harris and colleagues (148) reported that therapeutic community (TC) treatment significantly increased recidivism in psychopaths compared to the untreated psychopathic group (77% vs. 55%, respectively). However, the TC treatment employed in this study was an unlikely candidate for success; the "total encounter capsule" involved nude encounter groups, feeding through tubes in the

walls, and LSD and alcohol administration for many days at a time [(148)pp. 285–288]. Remarkably, the Penetanguishene study continues to be used as evidence that treatment of the psychopath is contra-indicated. Perhaps even more surprisingly, although it seems apparent that therapeutic communities are not effective at reducing future criminal behavior (160, 161), they remain popular in prisons and psychiatric hospitals in Europe (143). But perhaps it is not surprising that unconventional therapies are often used. It is recognized that the characteristics of antisocial individuals create very difficult obstacles for treatment, thereby limiting the number of available options for intervention. While pharmacological research suggests that specific symptoms of ASPD can be effectively reduced through medication (151, 162), concerns about compliance outside of controlled settings and the high potential for abuse associated with some substances inhibits the degree to which this type of treatment can be used. For instance, while dopaminergic stimulants such as methylphenidate have been found to improve symptoms of inattention, irritability, conduct problems, and impulsivity in adults and adolescents with antisocial behaviors (163–165), the comorbidity between ASPD and substance abuse limits the use of this intervention beyond controlled settings due to its high potential for abuse when appropriate use of this medication cannot be monitored (151).

Other pharmacological treatments have also shown promise in the reduction of the aggressive behaviors associated with ASPD and psychopathy, but may also be limited in their use due to concerns about long-term treatment compliance (166, 167). Lithium has received considerable attention for use in those with ASPD due to its efficacy at reducing impulsive violent behaviors in nonbipolar adults and adolescents (168, 169). Additionally, selective serotonin reuptake inhibitors (SSRIs) such as sertraline and fluoxetine have been associated with significant reductions in overt hostility, aggression, and antisocial behavior (170–172), as have anticonvulsants, including valproic acid (173), or its salt form, divalproex sodium (174, 175), and phenytoin (176). Like methylphenidate, the use of lithium and antipsychotics to control aggression is suitable in forensic settings, but the poor treatment compliance demonstrated by those with ASPD may reduce the effectiveness of this treatment in the long-term (166, 167).

In addition, the manipulative and remorseless traits associated with psychopathy do not bolster optimism for therapeutic interventions. Some researchers believe that despite good compliance with therapy and reported therapeutic improvement in correctional settings, the psychopath is simply using what he uses in therapy to enrich his criminal versatility and skill, thus accounting for negative outcomes (143). For instance, structured cognitive-behavioral techniques designed to target the behaviors associated with ASPD and psychopathy have demonstrated insignificant, or even inverse relations with recidivism, despite reported therapeutic gains such as conduct during sessions and therapists' ratings of motivation ((149); see (143) for a review). Radical therapeutic change techniques aimed at modifying the character of the psychopath have been proposed, but to date, have limited empirical support (177). One example of this type of treatment is Cloninger's (151) coherence therapy which is designed to address the unseemly character of those with ASPD in the hopes of increasing "trust, hope, and compassion" in the remorseless individual. Cloninger (151) posits that deficiencies in self-transcendence and emotional awareness underlie antisocial traits, and can be improved through meditative exercises, exposure to classical music, and therapeutic exercises in self-efficacy. While many agree that therapy targeting dysfunctional characteristics may be a very important aspect of the treatment of those with ASPD, it is perhaps unrealistic to expect self-transcendence exercises to be the most effective way of eliciting this type of change (178).

In contrast to those who hold little hope for changing the outcomes of those with ASPD and psychopathy, some authors contend that the treatment of ASPD and psychopathy can be beneficial, and that the reported failures in treatment response are simply not the norm (147, 151, 159). In fact, large meta-analytic studies indicate that many forms of treatment (e.g., electroconvulsive therapy, psychodrama, cognitive-behavioral therapy, psychoanalysis, therapeutic communities, and pharmacotherapy) have a positive overall effect on the reduction of recidivism for adult offenders (147, 179, 180), and juvenile offenders alike (181, 182). In addition to overall effect sizes, Lipsey (181) conducted moderator analyses on over 400 treatment-outcome studies to identify important factors that contribute to therapeutic success; reductions in criminal recidivism are associated with greater therapeutic intensity (longer duration and more frequent contacts), structured treatments (e.g., cognitive-behavioral therapy and skills training), and multimodal treatments (e.g., individual and group therapy coupled with vocational training, pharmacological treatment and work assignments). Thus, the quantitative treatment results appear to indicate something quite different than the dismal conventional standard. When specific guidelines are followed, it appears as though significant gains can be attained (159, 182).

Many of these same techniques have been recommended in a recent review of treatment outcomes for psychopathy (152). Salekin and colleagues suggest several possible "practical problem areas" (p. 256) when conducting therapy with psychopathic individuals, including addressing motivation to change, deceptiveness/manipulativeness, and lack of emotion and empathy leading to poor attachment, as these appear to be areas that prevent optimal outcomes. However, this review also noted that of the recent treatment studies, only 3 of 8 adult studies indicated any success. This same review identified 8 recent studies of treatment in psychopathic youth, and unlike the adult outcomes, youth treatment seemed much more effective; 6 of 8 studies indicated some therapeutic gain in terms of recidivism and latency to recidivism suggesting that early intervention is likely to be the best practice in terms of instituting relevant changes (152).

However, even though individual studies and some meta-analytic work indicates that treatment has a positive influence on recidivism, it is still questionable whether these are truly meaningful gains. Importantly, reported effect sizes in this area tend

to gravitate toward a value of .20, which is comparable to that of a placebo effect (179–183). The clinical meaningfulness of this improvement may simply be insignificant. For instance, the treated offender may have committed rape fewer times in the five years following release than the untreated offender, but the fact that he continues to rape (albeit with less frequency) speaks to the inadequacy of contemporary interventions. Although modest reductions in antisocial behavior are important, these reductions do not indicate that treatment is eliciting substantive change. In addition, many of the studies used in meta-analytic research of the treatment of ASPD and psychopathy rely on one group, pre-post treatment designs (143). This type of study design has been found to overestimate treatment effects, thereby making these positive effect sizes even less convincing (182). Finally, very few treatment studies have examined long-term efficacy of treatments, reporting primarily on short-term outcomes. This can result in the inflation of positive results, as well as mask discouraging survival statistics. As can be seen in a recent study by Olver, Lewis, & Wong (184), even large therapeutic treatment gains in the short-term may not have lasting effects; of 38 incarcerated offenders with high psychopathy scores who responded well to treatment, only 10% had violently recidivated during the first year post-treatment. However, this number increased to over 60% within 10 years (184). Thus, while meta-analytic results provide compelling suggestions that treatment interventions addressing the criminal behaviors of ASPD and psychopathy should continue to be pursued, they do not actually document that contemporary programs are having a substantial, meaningful effect.

In sum, clear conclusions about the efficacy of treatment for ASPD and psychopathy are difficult to draw. While meta-analytic findings appear to support further investigation into treatment interventions, they also fail to fully contradict the argument that these disorders are largely unresponsive to treatment in general. Although even mild to moderate changes in antisocial personality traits can be associated with benefits to the person and to the wider society, the relative modesty of therapeutic gains indicates that treatment needs to improve before we can decisively conclude that ASPD and psychopathy are treatable disorders.

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