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PERSONALITY DISORDERS (C SCHMAHL, SECTION EDITOR)

Antisocial Personality Disorder: A Current Review

Andrea L. Glenn · Alexandria K. Johnson · Adrian Raine

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Abstract The Diagnostic and Statistical Manual of Mental Disorders (DSM 5) classification of antisocial personality disorder (ASPD) describes individuals who engage in repetitive irresponsible, delinquent, and criminal behavior. The diagnosis is highly controversial, with many researchers and clinicians arguing that the category is too heterogeneous, overinclusive, and demonstrates considerable overlap with other disorders. This review focuses on recent studies that have improved our understanding of the characteristics of individuals who fit the ASPD definition by exploring how subtypes differ and how comorbid conditions influence the presentation of ASPD. In addition, we discuss research on the etiology of ASPD that has identified genetic and environmental factors that may contribute to the development and persistence of antisocial behavior, and brain imaging research that has improved our understanding of the relationships between ASPD and other psychopathology. Finally, we discuss promising preliminary research on treatment for this disorder.

Keywords Crime · subtypes · DSM 5 · Psychopathy · Substance use · Brain imaging · Genetics · Child abuse · Treatment · Personality disorder

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A. L. Glenn (✉)
Center for the Prevention of Youth Behavior Problems, Department of Psychology, University of Alabama, Box 870348, Tuscaloosa, AL 35487, USA
e-mail: Andrea.L.Glenn@ua.edu

A. K. Johnson
Department of Psychology, University of Alabama, Box 870348, Tuscaloosa, AL, USA
e-mail: akjohnson6@crimson.ua.edu

A. Raine
Departments of Criminology, Psychiatry, and Psychology, University of Pennsylvania, 3809 Walnut Street, Philadelphia, PA 19104, USA
e-mail: araine@sas.upenn.edu

Introduction

Antisocial personality disorder (ASPD) describes individuals with a pervasive pattern of disregard for, and violation of, the rights of others that begins in childhood or early adolescence and continues into adulthood. Based on the DSM-IV criteria for ASPD, recent epidemiological studies report a prevalence of 2-3 % in the general population [1], with estimates of approximately 3 % in men and 1 % in women. In prison samples, studies have found rates of ASPD to be 47 % in men and 21 % in women [2]. In addition to the costs placed on the criminal justice system, APSD inflicts considerable costs on health and social service agencies.

How to best define the construct of ASPD is a topic of debate that has gained traction in recent years. Many researchers have expressed concerns that the current ASPD criteria place too much emphasis on observable behaviors, rather than on the underlying personality structure [3]. However, the newly released update of the DSM, the DSM-5, retains the same diagnostic criteria for ASPD as was included in the previous edition. Criteria include behaviors such as repeatedly performing acts that are grounds for arrest, repeated lying, repeated fights or assaults, disregard for the safety of oneself and others, repeated failure to sustain consistent work behavior, and mistreating other individuals. The personality disorders work group, charged with ensuring that the diagnostic criteria for personality disorders reflects current understanding based on up-to-date research, suggested an alternate model for personality disorders that differed in significant ways from the retained criteria. This proposed model is included in the DSM-5 under Section III, which is reserved for emerging measures and models. The alternate model places a much greater emphasis on personality characteristics than the current model, evaluating traits such as egocentrism, empathy, and self-direction, and seems to be more closely aligned with the construct of psychopathy by including traits such as manipulativeness, callousness, deceitfulness, and impulsivity.

This potential future direction of ASPD is deserving of careful consideration and further research.

ASPD has high rates of comorbidity with several psychiatric disorders, including psychopathy, substance abuse, anxiety, depression, bipolar disorder, and borderline personality disorder. One of the primary questions that has been the focus of research in the last few years is how the category of APSD relates to other constructs. Understanding the overlap between these constructs, as well as how ASPD may contain subtypes, will likely provide information about etiology and may be beneficial in developing treatment programs.

Subtypes of ASPD and Associations with Other Disorders

Several recent studies suggest that people who meet criteria for ASPD can be subtyped into more precise and homogeneous groups based on characteristics that offer greater acuity than solely the tendency to act in ways that are irresponsible and illegal. In particular, recent research has examined associations with psychopathy, substance use, and mood disorders.

Associations with Psychopathy

Antisocial personality disorder has perhaps the most overlap with the construct of psychopathy. Psychopathy, while not recognized in the diagnostic criteria of DSM 5, describes individuals with many of the features of ASPD, but who, in addition, demonstrate a characteristic set of interpersonal and affective features, including superficial charm, manipulativeness, callousness, and shallow affect [4, 5]. Although some individuals diagnosed with ASPD may have psychopathic traits, others may not. Some suggest that although psychopathy and ASPD share many features, the underlying psychobiological processes may be distinct.

The current diagnostic criteria for ASPD are primarily behavior based, in contrast to the construct of psychopathy, which contains interpersonal and affective features in addition to behavioral criteria. There has been some debate as to whether the personality features captured by the psychopathy construct are simply associated features of the maladaptive behavior essential to ASPD, or whether psychopathy and ASPD differ more substantially. Examining this question within a sample of 691 offenders who met criteria for ASPD, Poythress et al. [6] conducted a cluster analysis which revealed that these offenders could be separated into meaningful subgroups based on significant differences in personality traits commonly identified within the psychopathy literature (e.g., low anxiety/fear, impulsivity, dominance). Poythress et al. [6] identified four ASPD subtypes (primary, secondary, and "fearful" psychopathy, as well as non-psychopathic ASPD) within their sample. The four subgroups identified within ASPD offer some evidence that ASPD may in fact be a heterogeneous diagnosis.

A follow-up study focused on participants within the sample who were court-ordered to attend substance abuse treatment found correlations suggestive of differences in the etiology of substance abuse among the four previously identified subgroups and a fifth non-ASPD subgroup [7]. Specifically, this study reported that for the two non-psychopathic subgroups (non-psychopathic ASPD and non-ASPD) negative emotionality and impulsivity were both significantly correlated with drug use, whereas in the three psychopathic ASPD subgroups only impulsivity was significantly correlated with drug use (and only in two of the three psychopathic subgroups). Similarly, within the two non-psychopathic subgroups, impulsivity was significantly correlated with alcohol abuse, whereas this relationship was not significant among the three psychopathic subgroups.

Another study [8] attempted to replicate the same four clusters found by Poythress et al., this time using the Psychopathic Personality Inventory (PPI) [9]. Despite the poor classification accuracy reported, the four subtypes still showed significant differences, with those in the non-psychopathic ASPD subgroup being the least likely to commit any infraction and the least likely to commit a violent infraction while in prison. Results from these studies offer support for the idea that individuals with ASPD who do not demonstrate psychopathic traits differ in meaningful ways from those diagnosed with ASPD who also meet criteria for psychopathy.

In addition to studies that have examined subtypes based on psychological characteristics, a number of recent studies have also examined the underlying biological factors that may either unify or distinguish individuals with ASPD. In a sample of female offenders, Anton et al. [10] examined the process of fear-potentiated startle, finding distinct patterns of cognitive processing and fear reactivity between psychopathy and ASPD. Specifically, psychopathy was associated with selective attention that favored goal-relevant information and filtered out peripheral information, including information related to threats. Those with ASPD, on the other hand, displayed a more distinct fear deficit as well as possible deficits in executive functioning, as evidenced by greater fear-related distraction when conditions placed participants under greater cognitive demand. Drislane et al. [11] also examined differing responses to threat cues between those with psychopathy and ASPD. Psychopathic participants (nearly all of whom also met criteria for comorbid ASPD) had a diminished defensive response to threat compared to non-psychopathic ASPD participants, whose threat-response did not significantly differ from those who did not meet criteria for either ASPD or psychopathy. Moreover, results showed that the deficit in defensively responding to threat which psychopaths in this study displayed was associated with the affective-interpersonal features of the disorder. This finding highlights the importance of affective-interpersonal traits in distinguishing psychopathy from ASPD.

A recent study examining the brain through structural MRI also revealed, as in the prior study, that, at least in some respects, participants with ASPD but not psychopathy appear more similar to the control group than they do to participants with co-morbid psychopathy and ASPD. Gregory et al. [12•] found that, compared to violent offenders who met criteria for ASPD but not psychopathy, violent offenders who met criteria for both disorders had significantly reduced gray matter volume in areas of the brain related to empathy, morality, and processing prosocial emotions such as guilt. The gray matter of the former group appeared similar to that of non-offenders without either disorder. These studies provide further evidence that although there are some shared deficits, ASPD and psychopathy are characterized by distinct neurobiological processes, suggesting that there are differences in the etiology of these disorders. These studies support the idea that there may be a distinct subgroup of individuals with ASPD for whom antisocial behavior results from different underlying pathology.

Associations with Mood Disorders

Many characteristics common to mood disorders (e.g., emotional reactivity, impulsivity) overlap with characteristics commonly associated with ASPD. The examination of ASPD as it presents co-morbidly with specific mood disorders can aid in better understanding the variety of possible mechanisms that may underlie the problematic behaviors characteristic of ASPD.

For example, in a recent study [13•] examining the presence of anxiety disorders among offenders diagnosed with ASPD, two-thirds of the participants were reported to have symptoms of an anxiety disorder at some point in their lifetime. Offenders whose ASPD was accompanied by co-morbid anxiety had significantly greater ASPD symptoms, as well as significantly increased suicidal ideation and suicide attempts, and greater alcohol and drug abuse compared to offenders with ASPD alone. Those with co-morbid ASPD had more convictions for homicide, attempted homicide, and physical and sexual aggression. One possible explanation for these associations between ASPD behaviors and anxiety is that the low-activity variant of the MAOA gene is associated with both enhanced reactivity to threat as well as an increased likelihood to experience anger, making a carrier of this gene more prone to anxiety and more likely to engage in reactive aggression. This possibility offers some evidence that among offenders with ASPD, the mechanisms underlying violent behavior may differ for those who also have co-morbid anxiety.

Impulsivity is another feature that is present in ASPD as well as several other disorders. Recent studies have examined how impulsivity may differ in individuals with ASPD depending on whether another diagnosis is present. For example, borderline personality disorder and ASPD share impulsivity as a common characteristic, which can make differentiating two disorders difficult. DeShong and Kurtz [14] found evidence in support of using a four factor model of impulsivity [15] to more accurately distinguish the two disorders. Results showed that each disorder was uniquely associated with two of the four impulsivity factors. Borderline features were associated with urgency (acting rashly in reaction to intense negative affect) and a lack of perseverance, whereas ASPD was associated with sensation seeking and a lack of premeditation.

Impulsivity is also a common feature in bipolar disorder. In a study comparing the role of impulsivity in individuals with bipolar disorder only, ASPD only, ASPD with bipolar disorder, and controls without either disorder, Swann et al. [16] found that although both disorders are associated with impulsivity, when ASPD was co-morbid with bipolar disorder significant deficits in the ability to delay reward were present compared to participants with either disorder alone. This suggests that perhaps the mechanisms underlying impulsivity in the two disorders may be slightly different or perhaps when the disorders are combined the ability to compensate for impulsivity is diminished. In another study of bipolar disorder concurrent with ASPD, Mueser et al. [17] examined schizophrenia, schizoaffective disorder, or bipolar disorder and found that among these patients, those who also had co-morbid ASPD had: greater functional impairment, greater strain on relationships with relatives, higher rates of drug abuse, more severe depression, and less education. Overall, the results from these studies suggest that when ASPD is co-morbid with other disorders, associated negative effects are exacerbated.

Associations with Substance Use Disorders

Studies have reported that 80–85 % of individuals with ASPD also meet criteria for a substance use disorder (SUD) [18, 19]. This is compared to the estimated US population lifetime prevalence rates of 13.5 % for alcohol use disorders and 6.1 % for other drug use disorders [18]. A recent study found that approximately 71 % of patients in a rural inpatient psychiatric facility diagnosed with ASPD abused alcohol, and approximately 62 % abused multiple substances. Over half abused cannabis and nearly one third abused amphetamines [19]. A study examining heavy episodic drinking among college undergraduates found that ASPD was significantly correlated with heavy episodic drinking. In fact, ASPD severity accounted for 9–26 % percent of the variance in heavy episodic drinking behavior within this particular sample [20]. In the previously mentioned study by Mueser and colleagues [17], it was found that among participants with a serious mental illness, those who had a co-occurring ASPD diagnosis were significantly more likely to abuse drugs and to use drugs more frequently, especially amphetamines and opiates. A recent study by Schiffer et al. [21] attempted to identify structural differences within the brains of violent offenders

as compared to non-offenders while accounting for long-term substance abuse, which often co-occurs alongside violence and can have substantial effects on brain structures. Results showed that greater gray matter volume in the mesolimbic reward system of the brain may be associated with violence, whereas decreased gray matter in the prefrontal cortex, orbitofrontal cortex, and premotor area is associated with substance abuse.

Summary

The fact that there is significant overlap between ASPD and several other forms of psychopathology makes understanding the developmental of it even more complicated. When examining the biological and environmental factors associated with antisocial personality disorder it is important that researchers consider whether these factors are specific to antisocial personality disorder, or are factors that may more broadly contribute to the co-occurrence of antisocial personality disorder and other disorders.

Etiology of ASPD

Results from a growing body of research, including from prospective longitudinal studies, suggest a complex interplay between biological (genetic/physiological/neurobiological) and environmental factors contribute to the development and maintenance of ASPD. In the last few years, several studies have furthered our knowledge in this area.

Genetics

Extensive research has shown that genetic factors contribute to approximately half of the variance in antisocial behavior [22, 23]. Recently, Barnes, Beaver, and Boutwell [24•] assessed how genetic contributions to antisocial behavior may differ depending on the developmental trajectories that have been defined by Moffitt. This model defines two groups of offenders—life-course persistent offenders who manifest antisocial behavior beginning in childhood and whose problems remain relatively stable throughout adulthood, and adolescence-limited offenders, who exhibit behavioral problems primarily in adolescence. In a study of sibling pairs, Barnes, Beaver and Boutwell found that genetic factors explained a larger percent of the variance in being classified as a life-course persistent offender than being an adolescent-limited offender. These findings support Moffit's theory [25] which suggested that environmental influences contribute more to the development of adolescent-limited offending.

Although it is clear that there is a significant genetic contribution to antisocial behavior, an important next step is to understand which specific genes confer risk. Several genes have been examined in relation to antisocial personality disorder [26–28]. Recently Basoglu et al. [29] examined two variants of the synaptosomal-associated protein 25 (SNAP25) gene, a gene that has been associated with attention-deficit hyperactivity disorder and cognitive performance, and may be associated with the functioning of several neurotransmitters. Specific variants of these two polymorphisms were more frequently present in male participants with ASPD than in sex-matched healthy controls. Notably, these polymorphisms were not associated with psychopathic traits, suggesting that they may be associated with traits that are seen across the spectrum of externalizing behavior such as novelty seeking and reward dependence, rather than traits that are unique to psychopathy.

Although single genes only contribute to a small proportion of the overall variance in antisocial behavior [30], identifying genes that confer risk may aid in the development of treatment methods that could potentially be tailored to specific risk factors of the individual. In addition, it may improve our understanding of the biological pathways that lead to antisocial behavior.

Environmental Factors

Although genes may contribute to half of the variance in antisocial behavior, this still leaves a large proportion of variance that results from environmental influences. In many cases, it is difficult to tease apart the influences of genes versus the environment because the two are confounded. If one or both parents are antisocial, it is likely that genetic risk factors for antisocial behavior will be transmitted to the child. Because of this, it is difficult to determine the extent to which environmental influences associated with having an antisocial parent have an effect. A child with an antisocial parent may be more likely to experience maltreatment, or may witness violence in the home. Data from an epidemiologic survey suggests that childhood witnessing of intimate partner violence increases the risk for adult perpetration of intimate partner violence [31]. Antisocial parents may also have poorer parental management strategies (harsh and inconsistent discipline, less supervision of the child, lack of warmth toward the child), and may be less able to provide adequate resources for the child.

Berg-Nielsen and Wichström [32] recently examined the influence of parents' personality disorder status on child problems at the preschool age. Similar to prior work showing that parent antisociality places older children and adolescents at risk for developing a range of externalizing and internalizing problems [33], they found that parents' personality disorder symptoms (antisocial, borderline, or narcissistic) explained 13.2 % of the variance in children's behavioral symptoms. Although this study does not disentangle the genetic versus environmental contributions to the generational transmission of behavioral and emotional problems, it demonstrates that these problems can be observed as early as the preschool years.

Another factor that has been examined is television viewing. In a 26-year longitudinal study assessing a birth cohort of 1037 individuals, young adults who had spent more time watching television during childhood and adolescence were significantly more likely to have a diagnosis of ASPD and more likely to have a criminal conviction [34]. These associations remained significant when controlling for sex, IQ, socioeconomic status, previous antisocial behavior, and parental control. The authors speculate about the mechanisms that could explain the effect of television viewing on antisocial behavior. One possibility is based on observational learning theory, whereby the behaviors that are viewed on television are imitated or internalized. Youth may also become emotionally desensitized to violence or the suffering of others, or may develop normative beliefs about the use of aggression and violence in response to particular situations. Additional mechanisms include reduced social interactions with peers and/or parents, poorer educational achievement, and increased risk of unemployment [34].

In another prospective longitudinal study, Shi et al. [35] examined different components of the early childhood environment that were related to ASPD features nearly 20 years later. They found that quality of early care, as indexed by clinician referral for problems in the parent-infant relationship during the first 18 months of life, was a significant predictor of ASPD in adulthood. Signs of maternal withdrawal, such as interacting silently, failing to greet the infant, and using toys instead of the self to soothe the infant were also found to be predictive of later ASPD outcomes. At age 8, disorganized attachment was predictive of later ASPD. Similarly, Liu et al. [36] found that childhood abuse was associated with ASPD features in adulthood. Psychosocial deprivation, including abuse and neglect, has been found to be very common in individuals with ASPD [37]. In both of these studies, genetic factors may also be involved. Parents who demonstrate lower quality caregiving may also pass on genes to their offspring that confer risk for antisocial behavior. Twin and adoption studies will be necessary to determine whether these environmental factors themselves confer risk for antisocial behavior, or whether there may be confounding genetic factors.

One of the ways in which environmental factors such as childhood abuse may result in antisocial behavior is through direct effects on biological systems. The environment can influence how genes are expressed (e.g., whether genes are "turned on or off") and can alter hormone and neurotransmitter levels, which in turn affect brain functioning. Environmental factors early in life may have a particularly pronounced effect on biological systems. A recent brain imaging study highlights this idea. Kumari et al. [37] examined how psychosocial deprivation, including childhood physical and sexual abuse, related to brain structure in violent individuals with ASPD. They found that the volume in the thalamus was reduced in psychosocially deprived violent individuals compared to non-deprived violent individuals and healthy controls. The thalamus is a region which filters incoming sensory information. Although speculative, the authors suggest that a thalamic deficit may make it more difficult for individuals to suppress intrusive memories and thoughts related to prior abuse and maltreatment. They also found a negative relationship between psychosocial deprivation and volume in the inferior frontal region of the prefrontal cortex across all individuals. This region is involved in inhibition and behavioral control; deficits in this region may contribute to an inability to plan and regulate one's behavior.

Brain Imaging

Prospective longitudinal studies implementing neuropsychological tests in children as young as 3 years suggest that abnormalities in brain functioning may contribute to the development of ASPD [38]. A number of neuroimaging studies have identified brain regions in which the structure or function differs in antisocial groups [39]. One of the most replicated findings is that individuals with antisocial personality disorder have reduced volume and functioning in the prefrontal cortex [40]. A recent study suggests that differences in the structure of the prefrontal cortex may partially explain the gender differences in antisocial behavior. Raine et al. [41•] found that there are significant differences between men and women in gray matter volume specifically in the orbitofrontal and middle frontal regions of the prefrontal cortex. Controlling for these brain differences reduced the gender difference in antisocial personality by 77.3 %. These findings suggest that part of the gender difference in antisocial behavior is attributable to gender differences in the volume of prefrontal brain regions.

Other recent studies have questioned whether reduced volumes in prefrontal regions are associated with ASPD generally, or whether they are a result of comorbid conditions. As mentioned above, two studies have examined differences in the brain in different subgroups of individuals with ASPD. Gregory et al. [12•] found reduced prefrontal gray matter only in individuals with comorbid psychopathic traits. No prefrontal volume differences were observed between controls and individuals with ASPD and low levels of psychopathic traits. Schiffer et al. [21] examined the potential effects of substance use on findings in antisocial groups. Using a 2 (violent offenders / non-offenders) × 2 (substance use disorders / no substance use disorders) design, they were able to examine the effects of antisocial behavior and substance use separately. They found that although men with SUDs exhibited smaller gray matter volume in the prefrontal cortex, there were no differences in this region between violent offenders and non-offenders. These findings differ from those of Raine et al. [41•], who found that individuals with ASPD *did* demonstrate reductions in the prefrontal cortex compared to non-antisocial substance abuse control participants. In the study by Gregory

et al., there were no differences between the ASPD groups with and without psychopathic traits in the proportion of lifetime substance use disorders, but there were significant differences between ASPD and control participants. However, they found no regions in which brain volumes differed between offenders with ASPD without psychopathic traits and non-offenders.

In sum, these studies suggest that the finding of reduced prefrontal gray matter volumes may be influenced by comorbid conditions such as psychopathy and substance abuse, but relationships remain unclear. Given the heterogeneity of ASPD, it is difficult to integrate results from neuroimaging studies. In many samples it is unclear how many participants also exhibit psychopathic traits, substance use disorders, or other forms of psychopathology. The issue of substance use in particular is a complicated factor to disentangle. Research suggests that common genetic factors contribute to risk for antisocial personality and substance use problems [42]. Additionally, the use of substances from an early age may directly affect brain structure and functioning. Longitudinal studies will be necessary to understand the concurrent development of antisocial behavior and substance use disorders.

Additional studies have used novel tasks and methods to further our understanding of the neurobiological differences in individuals with ASPD. Tang et al. [43] examined brain activity during a resting period in a sample of young adult offenders with ASPD. Using machine learning, they were able to develop a classifier that could discriminate ASPD individuals from normal controls with 86.57 % accuracy. The authors suggest that this classifier may be able to improve the diagnosis of ASPD and aid in the understanding of the etiology. ASPD participants were found to have decoupling between the regions that become active while at rest (referred to as the default mode network) and regions that are involved in attention. The default mode network is thought to be involved in processes such as emotion regulation, planning for the future, reflecting on past experiences, and self-reflection. Decreased functioning in this network may result in impairment in these processes. Tang et al. also used voxel-based morphometry to examine brain volumes. They found altered gray matter volumes in the parietal lobe and altered white matter volumes in the precuneus, but no volumetric differences in the prefrontal cortex.

Jiang et al. [44] examined the neural correlates of deception in a sample of offenders with ASPD. Similar to findings from healthy populations, they found that areas of the dorsolateral prefrontal cortex, inferior parietal lobe, and anterior cingulate were associated with lying versus truth-telling. These regions have previously been associated with cognitive control and inhibition, and thus may be necessary for inhibiting the normal propensity toward truth-telling. Offenders who scored higher on ASPD criteria associated with deception had less activity in these regions. Although this study cannot provide information about causal relationships (i.e., whether individuals with specific brain functioning find it easier to lie or whether frequent lying alters brain functioning), it demonstrates that there is variation within individuals with ASPD.

The source of differences in brain structure and functioning in individuals with ASPD may result from either genetic or environmental influences. As noted above, some brain differences may result from psychosocial deprivation, such as childhood physical and sexual abuse [37]. Such environmental factors, particularly early in life, can significantly affect the development of the brain, and thus increase the risk for antisocial behavior.

Advances in Treatment of ASPD

ASPD has long been recognized as one of the more difficult forms of psychopathology to treat. Individuals rarely seek treatment and many service providers are reluctant to attempt to treat these individuals. When in treatment, therapists report difficulty establishing a therapeutic alliance and find poor compliance with treatment. The presence of comorbid psychopathology often further hinders treatment progress. High quality trials on the treatment of ASPD are lacking. One study compared cognitive behavioral therapy with treatment as usual in a community sample with a diagnosis of ASPD [45]. They found that individuals receiving CBT showed a small but nonsignificant improvement in social functioning and physical aggression, but no improvements in anger or verbal aggression compared to individuals receiving treatment as usual. One form of treatment that has emerged in the last decade is mentalization-based treatment [46]. Mentalization is the capacity to think about one's own mental state and the mental states of others. This form of treatment was originally developed for individuals with borderline personality disorder. Preliminary evidence suggests that this treatment may be effective in reducing self-reported aggressive behavior individuals with ASPD with moderate levels of psychopathic traits [47].

Conclusions

Research in the last few years has highlighted the fact that subtypes of ASPD exist. These subtypes differ in meaningful ways, including exhibiting different biological risk factors. Furthermore, research has begun to clarify how comorbid conditions such as anxiety, substance use disorders, and bipolar disorders influence the presentation of ASPD. Research on the etiology of ASPD has established that genetic factors have a significant role in antisocial behavior that begins early and persists into adulthood. Specific genes and specific environmental factors have been identified as contributors to the disorder. Brain imaging research suggests that the

consideration of comorbid conditions is particularly important in understanding the deficits that accompany ASPD, and also highlight the potential for brain imaging to aid in the diagnosis of the disorder. Finally, although much work remains to be done in the realm of treatment, preliminary evidence suggests that newer methods hold promise. Continued research on the subtypes of individuals with ASPD and its comorbidity with other psychopathology may prove useful in informing treatment in these individuals.

Compliance with Ethics Guidelines

Conflict of Interest Andrea L. Glenn, Alexandria K. Johnson, and Adrian Raine declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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