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Antisocial Personality Disorder

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Antisocial personality disorder (ASPD) is a complex mental health condition with major implications for criminal justice and public policy due to its prevalence among incarcerated populations and the cost of its associated criminal behavior on society. This entry discusses the history of ASPD as a diagnostic condition and how it is represented in the official psychiatric nosology, its comorbidity and personality correlates, its relation with the construct of psychopathy, causal theories and neurobiological indicators, and perspectives on treatment.

ASPD is a well-known but commonly misunderstood clinical condition whose modern conceptualization reflects the culmination of decades of observation and empirical investigation. Although relatively uncommon in the general population—with about 3% of men and 1% of women meeting the criteria for diagnosis—ASPD is commonplace in correctional and forensic settings, with estimated prevalence among incarcerated offenders falling within the 50% to 80% range. Research on the etiology and treatment of ASPD is largely motivated by governmental and public interest in reducing the costly toll of criminal behavior on society, including damage and loss to victims and expenses for incarceration and intervention.

History

Early editions of the *Diagnostic and Statistical Manual of Mental Disorders (DSM)* recognized chronic antisocial deviance as a mental health condition. The first edition, *DSM-I*, defined “sociopathic personality disturbance—antisocial reaction” as entailing the persistent presence of aggressive, criminal behavior; and *DSM-II* introduced the term *antisocial personality* for this condition.

Notably, these early *DSM* conceptions of ASPD included features of psychopathic personality as described by Hervey Cleckley. Based on his many years of experience with diverse inpatients in a psychiatric hospital setting, Cleckley characterized psychopathy as entailing emotional insensitivity and detached social relations along with persistent behavioral deviance that is “masked” by a veneer of apparent psychological health (i.e., lucidity, lack of distress, and social efficacy). The *DSM-I* and *DSM-II* definitions of antisocial personality in particular emphasized features of callousness, disloyalty, and rationalization of harmful behavior. However, the *DSM-I* and *DSM-II*'s reliance on prototype descriptions as referents for diagnoses, rather than specific observable behavioral criteria, resulted in the weak reliability of classifications. To address this problem, the *DSM-III* provided specific diagnostic criteria for personality disorders.

The criteria for ASPD in *DSM-III* consisted of behavioral indicants of deviancy in childhood (e.g., evidence of conduct disorder, in the form of lying, stealing, fighting or bullying, and other rule breaking) with continuance into adulthood (e.g., repeated impulsive, reckless or irresponsible behavior, aggressiveness, and illicit actions). In the revision to this third edition (*DSM-III-R*), remorselessness was added to the adult criteria for ASPD in response to criticisms that the behaviorally based definition in the *DSM-III* strayed too far from Cleckley's conception of psychopathy.

The criteria for ASPD were revised further in the *DSM-IV*, with a somewhat expanded list of child behavioral indicators (reflecting revisions to the diagnosis of conduct disorder) and a reduced set of adult criteria (including deleting one indicator and combining three others). This conception of ASPD, which was carried over from the *DSM-IV* to the main “Diagnostic Criteria and Codes” portion (Section II) of *DSM-5*, can be characterized as polythetic—

meaning that diagnostic criteria can be met in many different ways, such that individuals designated as ASPD exhibit diverse symptomatic expressions rather than comprising a homogeneous clinical group; this symptomatic heterogeneity presents problems for formulating and implementing treatment programs, as discussed later in this entry.

Notably, the *DSM-5* also includes an alternative trait-based conception of ASPD, as part of the dimensional system for personality pathology included in Section III of the manual —“Emerging Measures and Models.” The Section III dimensional system characterizes ASPD as entailing high levels of traits within two broad domains: (1) disinhibition (i.e., traits of impulsivity, irresponsibility, and risk taking) and (2) antagonism (i.e., traits of hostility, callousness, deceitfulness, and manipulativeness). The latter trait domain parallels a new “limited prosocial emotions” specifier for the diagnosis of child conduct disorder in *DSM-5* Section II, denoting the presence versus absence of callous-unemotional (CU) traits as described in the youth psychopathy literature; children with conduct disorder accompanied by CU traits exhibit more persistent and severe behavioral problems, more aggressive tendencies, and a stronger biological-genetic basis for their behavior. Notably, the Section III conception of adult ASPD also includes a trait-based psychopathy specifier, encompassing high attention-seeking along with low anxiousness and social withdrawal—tendencies characteristic of boldness as described in the triarchic model of psychopathy but absent in ASPD itself (see later section “Antisocial Personality Disorder and Psychopathy”).

Diagnostic Comorbidity

ASPD co-occurs routinely with substance use disorders (SUDs) but in an asymmetric manner: SUDs are far more prevalent than ASPD, and the vast majority of people with SUDs do not have comorbid ASPD, but most individuals diagnosed with ASPD (~80%) exhibit SUDs. In addition, ASPD, which has an earlier age of onset, confers an enhanced risk for the development of SUDs.

The comorbidity between ASPD and SUD can be accounted for by the externalizing spectrum model, which posits a general dispositional liability contributing to both disorders. This model is based on analyses demonstrating systematic covariance between antisocial and SUD symptoms, reflected in a common externalizing factor accounting for portions of each and a twin-based research showing this externalizing factor to be highly heritable. The model characterizes ASPD and SUDs as alternative symptomatic expressions of the general liability to externalizing problems, with environmental influences such as peers who are antisocial or early access to alcohol or drugs contributing importantly to one or the other direction of expression.

Personality Correlates

The idea of a dispositional liability contributing in common to ASPD and SUDs is also supported by data indicating shared personality correlates for the two. In particular, these clinical conditions show relations in common with traits reflecting impulsiveness and negative affectivity, which are represented in one way or another in most models of personality. For example, impulsiveness is represented in the Multidimensional Personality Questionnaire by the broad dimension of constraint (reversed; encompassing traits of control, traditionalism, and harm avoidance), and negative affectivity is represented by the broad dimension of negative emotionality (encompassing traits of aggression, alienation, and stress reaction). Within the well-known five-factor model of personality, impulsivity and negative affectivity are

reflected in facet traits from the broad domains of conscientiousness (reversed), antagonism (reversed), and neuroticism.

ASPD and Psychopathy

The distinction between ASPD and psychopathy has often been a source of confusion, particularly when it comes to the assessment of these conditions. Psychopathy is considered to encompass affective-interpersonal symptoms not included in the criterion-based conception of ASPD (e.g., superficial charm, grandiosity, callousness, shallow affectivity), and assessment instruments for the two conditions capture distinct dispositional tendencies to differing degrees.

The triarchic model of psychopathy was put forth in part to clarify what differing conceptions and measures of psychopathy reflect, and how they differ in turn from ASPD, in terms of three symptomatic-dispositional dimensions (facets) that make up psychopathy: (1) disinhibition, (2) meanness, and (3) boldness. The disinhibition facet entails weak inhibitory control, expressed as a lack of behavioral restraint and emotional volatility. This psychopathy facet relates closely to the externalizing liability factor that connects SUDs with ASPD and in turn with the impulsive-antisocial symptoms of psychopathy. Meanness entails deficient empathy, callous exploitativeness, and low affiliative tendencies, paralleling the concept of CU traits in the child psychopathy literature. Although considered central to psychopathy, meanness is represented secondarily to disinhibition in ASPD. Finally, boldness, considered unique to psychopathy and absent from ASPD, is a partially adaptive trait that entails social effectiveness, calmness in the face of threat or pressure, and enjoyment of novel situations and risky adventure. Meanness and boldness are both considered manifestations of a fearless temperament, but boldness lacks the social detachment associated with meanness.

Differing established measures of psychopathy can be interpreted through the lens of the triarchic model in terms of their content coverage and relations with ASPD. The interview-based Psychopathy Checklist-Revised, which is the most widely used measure in adult clinical-forensic samples, contains 20 items that index two distinct symptom dimensions (factors): (1) affective-interpersonal symptoms (Factor 1) and (2) impulsive-antisocial symptoms (Factor 2). Factor 1 encompasses symptoms of glibness, grandiosity, lying/manipulativeness, callous insensitivity, remorselessness, and blame externalization and reflects the triarchic dimensions of meanness and boldness. Factor 2 encompasses symptoms of boredom proneness, impulsiveness, reckless irresponsibility, aggressiveness, and proneness to illicit rule-breaking behavior from an early age, and as such, it reflects the triarchic dimensions of disinhibition and meanness and accounts for Psychopathy Checklist-Revised psychopathy's intersection with ASPD.

Another widely used measure, the self-report-based Psychopathic Personality Inventory-Revised, is designed for work with community adults and contains eight content scales that demarcate two distinct symptom dimensions: (1) fearless dominance (reflecting primarily boldness) and (2) impulsive antisociality (reflecting disinhibition and, to a lesser degree, meanness). The PPI also includes a coldheartedness subscale that relates distinctively to the meanness facet of the triarchic model. As such, the PPI's impulsive-antisociality dimension appears to capture tendencies in common with ASPD as represented in Section II of the *DSM-5*. In addition, the PPI's coldheartedness scale appears to capture distinct aspects of Section III ASPD reflected in traits from the antagonism domain (callousness, in particular), with PPI fearless dominance providing coverage of bold tendencies reflected in the Section III psychopathy specifier.

Causal Theories and Neurobiological Correlates

A number of differing etiological theories have been proposed for ASPD. One of these is the aforementioned externalizing spectrum model, which postulates a general heritable liability contributing to impulse control problems including child conduct disorder, adult ASPD, and SUDs—with the specific symptomatic expression of this broad liability determined substantially by environmental influences. Other work on this psychopathology domain has suggested that specific genetic factors may also play some role in the distinct symptomatic expression of the general liability.

Research on the neurobiological correlates of ASPD and its affiliated conditions supports the idea of a general liability contributing to each. Consistent evidence for performance deficits in frontal lobe (executive) tasks among individuals who are antisocial dovetails with earlier work pointing to frontal brain dysfunction in individuals at risk for alcohol problems. These functional deficits in frontal systems have been interpreted as characteristic of externalizing problems in general, with subsequent work on common brain correlates corroborating this perspective. One particularly notable example is the finding that reduced amplitude of the P300 brain potential to rare target stimuli in a visual “oddball” task, at one time considered a distinct marker for alcoholism risk, indexes general proneness to externalizing problems—with the association between reduced P300 and externalizing proneness mediated by common genetic influences.

Other neurobiological variables have also been found to correlate with diagnoses or symptoms of ASPD. For example, multiple studies have reported reduced brain levels of the neurotransmitter serotonin in individuals who are antisocial and who exhibit impulsive-aggressive or violent behavior. Other prospective work has shown that low resting heart rate in childhood significantly predicts later antisocial behavior, suggesting that physiological hypoarousal may promote sensation-seeking behaviors typical of individuals who are antisocial. Given the findings for frontal-task performance and P300, further systematic research is needed to establish whether neurobiological variables such as reduced brain serotonin and low resting heart rate represent distinct correlates of ASPD or are indicators of the general liability to externalizing problems that can be expressed in this or other forms.

The neurobiology of psychopathy has also been extensively studied and can lend insight into findings for ASPD, particularly when considered in relation to the triarchic model. Each facet of the triarchic model is presumed to reflect a specific neurobehavioral mechanism, associated with distinct biological correlates. Disinhibition’s theorized root in anterior brain dysfunction, based on studies of frontal-executive and cognitive task performance, may give rise to executive control and emotion regulation deficiencies that prohibit the use of past experience, including adverse consequences, in guiding action for individuals with externalizing disorders in general. Meanness or callous unemotionality is likely related to abnormalities in fear reactivity or emotional responsiveness more generally, in combination with deficits in the hormones oxytocin and vasopressin, which drive evolutionarily adaptive, affiliative, and nurturing tendencies. The antisocial and often instrumentally cruel or aggressive behavior displayed by high-callous individuals is more pronounced but in common with that seen in ASPD. Individual differences in boldness may be related to variations in the sensitivity of the brain’s defensive motivational system (i.e., the amygdala and related structures). Consistent with its phenotypic distinctiveness from ASPD, neurobiological variables related to boldness tend to show negligible associations with ASPD.

Treatment Approaches

The enormous financial and personal toll exacted by ASPD on society necessitates the development of effective interventions. Central to the treatment process is the therapeutic alliance (i.e., mutual collaborative relationship between client and therapist), which depends importantly on the client's internal motivation to improve; however, as treatment of ASPD is often court ordered, development of an effective therapist-client relationship is difficult, and a "firm but fair" approach is empirically supported.

Theoretically speaking, cognitive behavioral methods that explicitly teach cognitive skills have been most successful with clients who are antisocial, in terms of enhancing self-regulation and interpersonal problem solving beyond the therapeutic context. Relative to traditional punitive measures or non-skill-oriented treatments, cognitive behavioral therapy (CBT) methods are associated with modestly greater clinical improvements across domains and modest but stable reductions in recidivism and intervention-related costs.

However, treatments gains associated with CBT may be overstated due to selection bias in the subset of individuals with ASPD who actually complete treatment studies; clients who are more severely antisocial, requiring more intensive treatment, may not persist in the program, participate inconsistently, or fail to maintain improvements across contexts. Some researchers have called for individually customized interventions to maximize efficacy with the heterogeneous antisocial population; multisystemic therapy, which treats the client as embedded in multiple wider social-behavioral contexts, is particularly promising. In contrast to CBT approaches, other treatments such as psychodynamic interventions and therapeutic community interventions may actually have adverse effects on individuals who are antisocial and their criminal proclivities.

As with interventions for psychological conditions more generally, the treatment of ASPD needs to incorporate neurobiological principles and findings to an increasing degree in order to effect significant, lasting changes in behavior. Feedback-based interventions, focusing either on performance or on brain responding in problem-relevant tasks, have great potential to improve self-awareness of dysfunctional behavior patterns and to enhance affective and social sensitivity. Against historic views of ASPD as intractable and untreatable, continuing advances in neuroscientific methods and insights provide hope for the future.

See also [Alternative DSM-5 Model for Personality Disorders](#); [Amygdala](#); [Cognitive Behavioral Therapy](#); [Conduct Disorder](#); [Conduct Disorder: Treatment](#); [Externalizing Disorders](#); [Serotonin](#); [Substance Use Disorders and Co-Occurring Mental Health Disorders](#)

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Further Readings

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