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Antisocial Personality Disorder

Conceptualization and Treatment

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Among the various disorders of personality, antisocial personality disorder (ASPD) and its diagnostic cousin, psychopathy (psychopathic personality), stand as unique in terms of the costly toll they exact on society. Because of this, these disorders have long held the fascination of the public at large as well as that of investigators in the scientific community. Our goal in this chapter is to review what is currently known about the nature and causal bases of ASPD, highlighting ways in which it resembles and differs from psychopathy, and discuss currently available and potentially new approaches to the treatment of ASPD in light of this existing knowledge base. In reviewing what is known about ASPD, we consider relationships that ASPD has with phenomena in the domains of personality and psychopathology more broadly and consider what these relationships might tell us about psychological mechanisms and maintaining factors that can serve as targets for intervention.

The chapter begins with an historical overview of the construct of ASPD, highlighting its links to the concept of psychopathy. The next section summarizes contemporary conceptual perspectives and empirical findings on ASPD. Here, our emphasis is on placing ASPD into a broader conceptual framework that encompasses other impulse control disorders (e.g., conduct disorder, alcohol dependence, and drug dependence) with which ASPD is closely associated. Specifically, drawing on recent research findings, we argue that ASPD comprises one phenotypic manifestation of a broader genotypic disposition toward disinhibitory (externalizing) problems. The next section discusses physiological (including brain response) correlates of ASPD in relation to what is known about corresponding correlates of externalizing proneness and of psychopathy. This is followed by a section on currently available treatments for ASPD, focusing in particular on behavioral and cognitive behavioral methods, and a further section on possible alternative approaches to intervention suggested by recent developments in the empirical literature.



Conceptualizing and Assessing Antisocial Personality Disorder

Historical Overview

Early conceptions. The earliest accounts of the condition that came to be known as ASPD emphasized extreme behavioral deviance in the context of intact reasoning and communicative abilities. French physician Philippe Pinel (1801) described cases of individuals who engaged repeatedly in impulsive acts injurious to themselves and others despite recognizing at a verbal/conceptual level the irrationality of such acts. The label Pinel used for this condition was *manie sans delire* (“insanity without delirium”). Around the same time in the United States, Benjamin Rush (1812) documented similar cases, but postulated moral weakness (i.e., incapacity for guilt or shame in relation to actions and potential consequences) as the root cause. In his account, Rush highlighted the manipulative, deceitful nature of such individuals.

Reflecting a perspective similar to Rush’s, British physician J. C. Pritchard (1835) applied the term “moral insanity” to cases of this type. However, Pritchard applied this term much more broadly than Rush, for conditions ranging from drug or alcohol addiction to sexual deviations to mood disorders, along with conditions that would be classified today as mental retardation or schizophrenia. The alternative term “psychopathic” was introduced by German psychiatrist J. L. Koch (1891) to denote conditions of a chronic nature presumed to have an underlying organic (physical, brain-based) cause. Like Pritchard, Koch applied this term to a much broader array of clinical conditions than would be encompassed by current conceptions of ASPD or psychopathy. Operating from a similar etiologic perspective, Emil Kraepelin (1915) used the term “psychopathic personalities” for a somewhat narrower range of conditions including impulse-related problems, sexual deviations, obsessional disorders, and other “degenerative” personalities. The latter category included antisocial (callous-destructive) and quarrelsome (hostile-alienated) subgroups that would be classifiable today as APSD.

Reversing the trend toward broad application of the term “psychopathic,” Hervey Cleckley ([1941], 1976) proposed that the label be reserved for a specific condition with a distinct set of diagnostic features. Cleckley’s diagnostic criteria focused on three sets of features: (a) indications of psychological stability (i.e., good intelligence and social charm, absence of delusions/irrationality, absence of nervousness, and suicide rarely carried out); (b) tendencies toward emotional underresponsiveness and superficial/insincere relationships with others (i.e., deceitfulness, poverty in affective reactions, self-centeredness and incapacity for love, lack of reciprocity in social relations, lack of insight); and (c) persistent behavioral deviance in the form of repeated antisocial acts (often without obvious motives), irresponsibility, promiscuity, and absence of any clear life plan. According to Cleckley, the overt presentation of psychological stability in such individuals functioned as a convincing “mask of sanity,” concealing their affective-interpersonal deficits and behavioral deviancy.



Emergence of the concept of antisocial personality disorder. The first edition of the *Diagnostic and statistical manual of mental disorders (DSM-I*; American Psychiatric Association [APA], 1952) included a category of mental disorders termed “sociopathic personality disturbance.” Following early conceptions of psychopathy, this designation encompassed a broad array of clinical phenomena including sexual deviations of various types, addictions, and a condition referred to as “sociopathic personality disturbance: antisocial reaction,” entailing persistent aggressive and criminally deviant behavior. In the next edition (*DSM-II*; APA, 1968), the term “reaction” was eliminated as a descriptor for disorders, and sexual deviations, addictions, and delinquent personality types were grouped together in the category of “personality disorders and other non-psychotic mental disorders,” which included an “antisocial personality” designation resembling the syndrome of psychopathy described by Cleckley (i.e., with features including defective socialization, selfishness, callousness, untrustworthiness, and absence of guilt).

However, a serious limitation of the *DSM-II* (as with the *DSM-I*) was that diagnoses were assigned on the basis of prototypic descriptions of disorders rather than through use of specific behavioral criteria. As a result, the reliability of diagnoses using the *DSM-II* was poor (APA, 1980). This problem was addressed in the *DSM-III* (APA, 1980) by specifying more explicit, behaviorally oriented criteria for diagnoses. The criteria for ASPD in the *DSM-III* were strongly influenced by the work of Lee Robins (1966), who undertook longitudinal research to investigate the developmental course of “sociopathy.” Following Cleckley, Robins’s initial criteria for sociopathy included items relating to lack of guilt, remorse, and shame, but (due in part to weak reliability) these criteria showed weak discrimination in her work and thus were discarded as indicators. In line with this, the criteria for ASPD adopted in the *DSM-III* focused exclusively on behavioral indicants of deviance in childhood and adulthood, including such things as truancy, delinquency, stealing, vandalism, irresponsibility, aggressiveness, impulsivity, recklessness, and lying.

As a function of this change, the *DSM-III* diagnosis of ASPD proved highly reliable. However, prominent researchers (e.g., Frances, 1980; Hare, 1983) challenged the validity of the diagnosis on the grounds that it excluded many of the features identified by Cleckley as essential to psychopathy, including superficial charm, absence of anxiety, lack of remorse or empathy, and general poverty of affect. Some effort was made to address these criticisms in the revised third edition (*DSM-III-R*; APA, 1987) by adding lack of remorse (i.e., “feels justified in having hurt, mistreated, or stolen from another”; p. 346) as an adult criterion for ASPD. Despite an extensive field trial (Widiger et al., 1996) that provided for evaluation of alternative criterion sets including a 10-item version of Hare’s (1991) Psychopathy Checklist-Revised (PCL-R), intended to provide greater coverage of affective-interpersonal features of psychopathy, the diagnostic criteria for ASPD in the *DSM-IV* (APA, 1994, 2000) remain much the same as those in the *DSM-III*.

Current *DSM* Conception of Antisocial Personality Disorder

Clinical features. Table 53.1 summarizes the diagnostic criteria for ASPD in the current fourth edition of the *DSM (DSM-IV-TR*; APA, 2000). As with other disorders,

Table 53.1 Summary of Diagnostic Criteria for DSM-IV Antisocial Personality Disorder

<i>Criterion category</i>	<i>Summary description of criterion</i>
A. Adult antisocial behavior (3 or more of the following since age 15):	<ol style="list-style-type: none"> 1. repeated participation in illegal acts 2. deceitfulness 3. impulsiveness or failure to make plans in advance 4. hostile-aggressive behavior 5. engagement in actions that endanger self or others 6. frequent irresponsible behavior 7. absence of remorse
B. Age criterion	Current age at least 18
C. Evidence of child conduct disorder (3 or more of the following before age 15, resulting in impaired social, academic, or occupational function): ^a	<p><i>Aggression toward people or animals:</i></p> <ol style="list-style-type: none"> 1. frequent bullying, threatening, or intimidation of others 2. frequent initiation of physical fights 3. use of dangerous weapons 4. physical cruelty toward people 5. physical cruelty toward animals 6. theft involving victim confrontation 7. forced sexual contact <p><i>Destroying property:</i></p> <ol style="list-style-type: none"> 8. deliberate fire setting with intent to cause damage 9. deliberate destruction of property <p><i>Deceptiveness or stealing:</i></p> <ol style="list-style-type: none"> 10. breaking/entering (house, building, or vehicle) 11. frequent lying to acquire things or to avoid duties 12. non-trivial theft without victim confrontation <p><i>Serious rule violations:</i></p> <ol style="list-style-type: none"> 13. frequent violations of parental curfew, starting before age 13 14. running away from home 15. frequent truancy, starting before age 13
D. Comorbidity criterion	Antisocial behavior does not occur exclusively during episodes of schizophrenic or mania

Notes. ^aThe *DSM-IV* criteria for conduct disorder require the occurrence of three or more of these behavioral symptoms before age 15. Criterion C for antisocial personality disorder (ASPD) is vague as to the number of child symptoms needing to be met, specifying only “evidence of Conduct Disorder with onset before age 15 years.” Some approaches to assessing ASPD, for example the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II; First, Gibbon, Spitzer, Williams, & Benjamin, 1997), interpret “evidence of” as denoting a lower threshold (i.e., occurrence of two child symptoms, as opposed to three). Adapted from American Psychiatric Association (2000).



the criteria for ASPD are polythetic. That is, because only a portion of designated child and adult criteria need to be met, individuals can achieve the diagnosis in many different ways, subject to fulfilling inclusionary requirements (i.e., age 18 or older, antisocial behavior not attributable to mania or psychosis). As shown in Table 53.1, the child criteria for ASPD include aggressive and destructive behaviors on one hand, and deceitfulness/theft and nonaggressive rule breaking on the other. Formal factor-analytic investigations of the child criteria (e.g., Frick et al., 1991; Tackett, Krueger, Sawyer, & Graetz, 2003) have established that the aggressive and rule-breaking symptoms define separate, albeit correlated, factors. Tackett et al. (2003) reported that these two conduct disorder factors showed discriminative associations with aggressive behavior syndrome and delinquent behavior syndrome, respectively, as defined by scores on the Child Behavior Checklist (Achenbach, 1991).

An implication of this work is that there may be distinct variants of child antisocial deviance with different etiologic underpinnings. Along these lines, Moffitt (1993) proposed a distinction between adolescence-limited and life-course-persistent subgroups of delinquent individuals. The former was distinguished by a later onset and predominantly nonaggressive forms of deviancy and rule breaking, the latter by early age of onset, aggressive-destructive as well as nonaggressive delinquent behaviors, and continuation of child and adolescent deviancy into adulthood. Moffitt postulated that the early-onset, aggressive subtype of delinquency may have a stronger underlying neurobiological basis (see also Lynam, 1997).

Tackett, Krueger, Iacono, and McGue (2005) further examined the structure of conduct disorder symptoms in a male twin sample, permitting an analysis of etiologic contributions to aggressive versus nonaggressive subfactors. Their results indicated that these two components of conduct disorder have common as well as distinctive etiologic underpinnings. Additive genetic influences and nonshared environment (i.e., experiences unique to the individual) contributed significantly to both components, with the proportion of symptom variance attributable to genes somewhat higher for the aggressive than the nonaggressive component (35% vs. 28%). In addition, a significant contribution of shared environment (i.e., influences common to two siblings growing up in the same household) was found for the nonaggressive component only. Recently, Kendler, Aggen, and Patrick (2013) extended this work by presenting behavioral genetic evidence that: (a) aggressive and rule-breaking components of conduct disorder reflect differing sources of genetic influence, and (b) the shared environmental contribution to the rule-breaking component is concentrated in a distinct subset of symptoms reflecting covert delinquent acts (e.g., stealing, telling lies).

The adult criteria for ASPD include deceitfulness, impulsivity, irresponsibility, irritability and aggressiveness, reckless disregard for safety of self or others, lack of remorse, and failure to conform to norms with respect to lawful behaviors. As with child conduct disorder symptoms, evidence exists for differing etiological influences underlying aggressive and nonaggressive antisocial behavior patterns in adulthood. In a study involving adult twins, Kendler, Aggen, and Patrick (2012) reported two distinct factors emerging from a structural analysis of the adult criteria for ASPD, one (labeled *disinhibition*) reflecting tendencies toward impulsivity, irresponsibility,



and deceitfulness, and the other (labeled *aggressive-disregard*) reflecting irritability/aggressiveness and behaviors indicative of recklessness and lack of concern for oneself and others. Paralleling what has been reported for distinct factors of conduct disorder (Kendler et al., 2013; Tackett et al., 2005), these authors found that nonaggressive and aggressive facets of antisociality in adulthood were associated with differing sources of genetic influence.

Other evidence in the literature also supports the idea that aggressive forms of adult antisocial behavior have unique neurobiological underpinnings. For example, published studies have reported consistent evidence for reduced levels of the neurotransmitter serotonin (indexed by concentrations of the serotonin metabolite 5-Hydroxyindoleacetic acid [5-HIAA] in cerebrospinal fluid) in antisocial individuals exhibiting severe episodes of impulsive aggressive behavior (for a review, see Minzenberg & Siever, 2006). Evidence of reduced brain serotonin has also been reported in antisocial individuals who engage in impulsive suicidal acts (Linnoila & Virkkunen, 1992), which have been conceptualized as an alternative, self-directed expression of impulsive aggressive tendencies (Verona & Patrick, 2000).

Notably, findings demonstrating distinct aggressive and nonaggressive (disinhibitory or rule-breaking) components of antisocial behavior in childhood and adulthood dovetail with findings of recent research on the structure of impulse-related (externalizing) problems more broadly (Krueger, Markon, Patrick, Benning, & Kramer, 2007). As discussed in more detail later in this chapter, this work has yielded evidence of separate disinhibitory and callous-aggression factors (along with a third substance-addiction factor) underlying this domain of problems. Given these various converging lines of evidence, it will be valuable in future research to evaluate (through longitudinal investigations) the temporal stability of these distinct symptomatic facets of antisocial behavior from childhood to adulthood. For example, it might be hypothesized that the aggressive facet would exhibit greater stability across time than the disinhibitory/rule-breaking facet (cf. Moffitt, 1993; Tackett et al., 2005).

Antisocial personality disorder in the DSM-5. The fifth edition of the *DSM* (*DSM-5*; APA, 2012), scheduled for publication in 2013, contains major proposed changes to the diagnoses of child conduct disorder and adult ASPD that incorporate the developments noted earlier, along with new perspectives on the distinction between externalizing proneness and psychopathy (see the section on “Antisocial Personality Disorder and Psychopathy”). At the child level, a specifier has been proposed for the diagnosis of child conduct disorder to distinguish between variants with and without “callous-unemotional traits.” The callous-unemotional variant is characterized in particular by proactive aggressive tendencies and disregard for the feelings and welfare of others. At the adult level, a dimensional approach to the diagnosis of ASPD has been proposed that defines the disorder in terms of high levels of traits from two separate domains: antagonism, reflecting callousness, hostility, manipulativeness, and deceitfulness; and disinhibition, reflecting impulsivity, irresponsibility, and risk-taking. Although the criteria for ASPD in the *DSM-5* no longer include direct reference to conduct disorder symptoms, the inclusion of antagonism-related traits in the definition of ASPD clearly parallels the demarcation of a distinct variant of conduct disorder entailing callous-unemotional traits.



Comorbidity with other DSM disorders. ASPD shows well-documented patterns of comorbidity with other disorders in the *DSM*, most notably substance use disorders. In the Epidemiologic Catchment Area study (Robins & Regier, 1991), the base rate of substance use disorders of any type among individuals diagnosed with ASPD exceeded 80%. Earlier studies employing *DSM-III* criteria reported similar high rates of alcohol and drug problems among individuals diagnosed with ASPD (e.g., Koenigsberg, Kaplan, Gilmore, & Cooper, 1985; Lewis, Rice, & Helzer, 1983). ASPD has also been shown to be associated with greatly enhanced risk for alcohol and drug use disorders in more recent comorbidity studies (e.g., Grant et al., 2004; Kessler & Walters, 2002).

The fact that substance use disorders co-occur with ASPD to a much greater extent than would be expected if each occurred by chance, given their respective population prevalence rates, implies that something systematic underlies the association between the two. This hypothesis has been supported by factor-analytic investigations of the diagnostic overlap among common disorders within the *DSM*. For example, employing diagnostic data from the National Comorbidity Survey (Kessler & Walters, 2002), Krueger (1999a) reported evidence for two broad factors underlying the most common Axis I disorders: an “externalizing” factor encompassing ASPD, alcohol dependence, and drug dependence, and an “internalizing” dimension encapsulating the mood and anxiety disorders (see also Krueger, Caspi, Moffitt, & Silva, 1998; Vollebergh et al., 2001). One possible explanation for the systematic association between ASPD and substance use disorders is that it reflects overlap between the criteria for the two types of disorders. For example, it could be argued that some of the behaviors that define ASPD (e.g., irresponsibility, recklessness, aggressiveness) are common sequelae of alcohol or drug abuse, so that increased rates of such behaviors would be expected among individuals with substance use problems. However, this seems unlikely to explain the systematic association between ASPD and substance use disorders. For one thing, the onset of ASPD typically precedes that of substance use problems in cases where the two are comorbid. In addition, the relationship between ASPD and substance abuse problems is asymmetric, due to the higher population prevalence of the latter—that is, whereas most individuals diagnosed with ASPD also show evidence of substance use disorders, the majority of individuals from the general community diagnosed with substance abuse or dependence do not meet criteria for ASPD.

Another possibility is that ASPD and substance use problems arise from a common diathesis—that is, a common underlying trait factor that predisposes individuals toward the development of both types of disorders. Consistent with this possibility, behavior genetic (twin) studies have revealed evidence of shared genetic factors underlying ASPD and substance use disorders (e.g., Pickens, Svikis, McGue, & LaBuda, 1995; Slutske et al., 1998). More recent quantitative analyses of etiologic factors contributing to the broad externalizing factor representing the systematic covariance among these disorders have revealed that this factor is substantially heritable (Kendler, Prescott, Myers, & Neale, 2003; Krueger et al., 2002; Young, Stallings, Corley, Krauter, & Hewitt, 2000). This work is described in more detail later in this chapter.



Personality correlates. Two personality trait variables in particular, represented in various models of personality, have been shown to be related to ASPD. One is impulsiveness, represented in the five-factor model (FFM; cf. Digman, 1990) by the conscientiousness factor (reversed) and in Tellegen's (2003) Multidimensional Personality Questionnaire (MPQ) model by the higher-order factor of constraint (reversed). The other is aggressiveness, represented in the FFM by the agreeableness factor (reversed) and in the MPQ by the lower-order trait of aggression. Notably, these same personality variables (impulsivity and aggression) show reliable associations with substance use disorders (Casillas & Clark, 2002; Krueger, 1999b; Lynam, Leukefeld, & Clayton, 2003; Trull & Sher, 1994).

These observed relations between personality trait variables and clinical disorders have been interpreted in various ways. One perspective is that traits indexed by personality scales reflect basic individual difference processes from which mental disorder symptoms arise; another is that deviations in personality emerge as a consequence of psychopathology (Widiger, Verheul, & van den Brink, 1999). A third perspective is that psychopathology symptoms and personality trait variables correlate with one another because they are indicators of a shared underlying (latent) individual differences factor. With regard to ASPD and substance dependence, this perspective would suggest that these disorders are related to one another and in turn to personality traits of impulsivity and aggression because all of these variables are manifest indicators of a shared underlying externalizing factor. Krueger et al. (2002) evaluated this hypothesis for the broad MPQ factor of constraint by including this personality variable along with child and adult symptoms of ASPD and alcohol and drug dependence symptoms in a joint-factor analysis. Consistent with the aforementioned hypothesis, the analysis revealed the presence of a single latent factor on which constraint loaded significantly together with all four symptom variables.

Krueger et al. (2007) extended this work by undertaking a fine-grained analysis of traits and problem behaviors within the domain of externalizing psychopathology to elucidate the scope and structure of this spectrum more fully. They began by identifying various constructs embodied in the *DSM* definitions of the disorders included in the Krueger et al. (2002) analysis, and then developed self-report items to tap these constructs. They also surveyed the literature to identify other behavioral and trait constructs related empirically or conceptually to externalizing psychopathology and developed additional items to index these constructs. Across multiple rounds of data collection and analysis, item response modeling and factor analysis were used to refine the overall item set and thereby clarify the nature of constructs associated with the broad externalizing factor.

Employing this strategy, Krueger et al. (2007) arrived at a final set of 23 constructs, each operationalized by a unique subscale. These constructs included alcohol, marijuana, and other drug use and problems; aggression of various sorts; impulsiveness; irresponsibility; rebelliousness; excitement seeking; and blame externalization. Structural analyses of these 23 subscales yielded evidence of one broad superordinate factor (*externalizing*) on which all subscales loaded (the strongest indicators being "irresponsibility" and "problematic impulsivity") and two subordinate factors accounting for residual variance in specific subscales—a *callous-aggression* factor marked by subscales indexing aggression (all forms), callousness, and excitement seeking, and a *substance*



abuse factor marked by subscales indexing excessive use and problems with alcohol, marijuana, and other drugs. These findings provide support for the idea that problem behaviors and affiliated personality traits within this domain are indicators of a shared underlying factor (externalizing). In addition, consistent with results from structural analyses of the child and adult symptom criteria for ASPD, this more comprehensive analysis of constructs within the externalizing domain revealed evidence of distinctive aggressive and nonaggressive expressions of this general factor.

Neurobiological correlates. A variety of neurobiological correlates of ASPD have been identified. For example, as noted earlier, antisocial individuals—in particular, those displaying impulsive aggressive behavior—show evidence of reduced levels of the neurotransmitter serotonin in the brain. Other research has consistently demonstrated that low resting heart rate is a correlate of antisocial deviance, with prospective studies showing that low heart rate in childhood predicts later antisocial behavior (Ortiz & Raine, 2004). This finding has been interpreted as indicating that physiological hypoarousal confers liability to antisocial behavior by promoting sensation-seeking behavior (Raine, 2002).

Neuropsychological studies provide evidence of deficits in frontal brain function in individuals diagnosed as antisocial. Morgan and Lilienfeld (2000) reported meta-analytic evidence for deficits on frontal lobe tasks in individuals exhibiting conduct disorder and adult antisocial behavior. Notably, individuals at risk for alcoholism by virtue of a positive parental history also show evidence of impairment on neuropsychological tests of frontal lobe function (Peterson & Pihl, 1990; Tarter, Alterman, & Edwards, 1985). Iacono, Carlson, and Malone (2000) suggested that impairments in frontal brain function may be generally characteristic of individuals with externalizing problems.

There is also evidence that reduced amplitude of the P3 brain potential response, long known to be an indicator of risk for alcohol problems (Polich, Pollock, & Bloom, 1994), may be a marker of externalizing problems more generally, including ASPD. A number of studies have reported evidence of reduced P3 brain response amplitude in individuals with ASPD (Bauer, Hesselbrock, O'Connor, & Roberts, 1994; Bauer, O'Connor, & Hesselbrock, 1994; Costa et al., 2000; Iacono, Carlson, Malone, & McGue, 2002). Reduced P3 response amplitude has also been found in individuals with other impulse control problems, including nicotine dependence (Anokhin et al., 2000; Iacono et al., 2002), child conduct disorder (Bauer & Hesselbrock, 1999a, 1999b, 2002; M. S. Kim, Kim, & Kwon, 2001), and attention-deficit/hyperactivity disorder (ADHD; Johnstone & Barry, 1996; Klorman, 1991). The implication is that reduced P3 amplitude could be an indicator of the general externalizing factor that these disorders share.

Patrick et al. (2006) evaluated this possibility in a sample of 969 males recruited from the community by examining the association between reduced P3 amplitude and scores on the externalizing factor, defined as the primary component derived from a principal components analysis of symptoms of various *DSM-III-R* impulse control disorders (i.e., conduct disorder, adult antisocial behavior, and alcohol, drug, and nicotine dependence). These investigators found a highly significant negative association between scores on the externalizing factor and P3 brain response amplitude (i.e.,



higher externalizing scores, reflecting a greater number of impulse problems, were associated with smaller P3 amplitude). Moreover, significant associations between each individual diagnostic variable and P3 amplitude were accounted for entirely by the externalizing factor—that is, after controlling for scores on this common factor, all associations for individual disorders dropped to nonsignificance. These results suggest that reduced P3 amplitude represents a neural indicator of general proneness to externalizing problems, rather than an indicator of ASPD per se (or of other specific disorders, such as alcohol dependence).

Etiologic perspectives on antisocial personality disorder. A variety of etiologic models of ASPD have been proposed, some of them based on the aforementioned neurobiological findings (for reviews, see Raine, 1997; Zuckerman, 1999). Most of these models focus on the underpinnings of ASPD as a distinctive syndrome, without considering its relations to other forms of psychopathology (e.g., substance use disorders). However, recent efforts have been made to develop integrative etiologic models that accommodate ASPD's associations with other disorders and distinctive personality traits by conceptualizing ASPD as one facet of a broader spectrum of traits and problem behaviors. An example of this is the hierarchical spectrum model proposed by Krueger et al. (2002).

The essence of the hierarchical spectrum model is that there is a broad dispositional factor that disorders within a spectrum share, along with unique etiologic influences that determine the unique symptomatic expression of each disorder. The database on which the model was based consisted of symptom scores on four diagnostic variables (child conduct disorder, adult antisocial behavior, alcohol dependence, and drug dependence) along with a trait measure of impulsiveness (the broad Constraint scale of the MPQ) for a sample of male and female twins recruited from the community ($N = 1,048$). A biometric structural analysis revealed a large common factor (“externalizing”) on which all of these diagnostic variables loaded substantially (.58–.78); more than 80% of the variance in this common factor was attributable to additive genetic influence (see also Kendler et al., 2003; Young et al., 2000). The remaining variance in each disorder not accounted for by the broad externalizing factor was attributable primarily to nonshared environmental influence—although for conduct disorder there was also a significant contribution of shared environment.

Based on these findings, Krueger et al. (2002) proposed that a general constitutional factor contributes to the development of various disorders in this spectrum, but that the precise expression of this underlying vulnerability (i.e., as antisocial deviance or substance problems of different kinds) is determined by disorder-specific etiologic influences. Although the analysis pointed to unique environmental experience as the main determinant of diagnostic specificity (with some contribution of family environment for conduct disorder), owing to the somewhat modest sample size and large confidence intervals around parameters in the model, the authors acknowledged that specific genetic factors might also contribute to the uniqueness of certain disorders. Indeed, Kendler et al. (2003) presented evidence for this possibility in a subsequent study. As noted earlier, Krueger et al. (2007) extended the work of Krueger et al. (2002) by providing a more comprehensive analysis of traits and problem behaviors within the externalizing spectrum. However, an etiologic analysis





of this newer, more comprehensive model remains to be undertaken. In particular, it will be important to evaluate differences in etiologic contributions to the two subordinate factors (callous-aggression, substance abuse) identified by Krueger et al. (2007) in comparison with those for the broad externalizing factor (for a discussion of possible neurobiological mechanisms, see Patrick & Bernat, 2006; Patrick, Drislane, & Strickland, 2012).

Antisocial Personality Disorder and Psychopathy: Overlap and Distinctiveness

Psychopathy has been conceptualized in diverse ways historically, and contemporary assessment instruments such as the interview-based Psychopathy Checklist-Revised (PCL-R; Hare, 1991, 2003) and the self-report-based Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996; Lilienfeld & Widows, 2005) differ in the types of features they use to identify psychopathic individuals. A model that was recently advanced to reconcile differing conceptions of psychopathy, and clarify its relationship with ASPD, is the Triarchic Model (Patrick, Fowles, & Krueger, 2009). This model conceives of psychopathy as encompassing three distinguishable symptomatic components—disinhibition, meanness, and boldness—that represent thematic building blocks for alternative conceptions of psychopathy.

Disinhibition entails tendencies toward impulsiveness, deficient behavioral restraint, and difficulty in regulating emotion. Impulse-related problems of differing types, including child and adult antisocial behavior and substance-related disorders, feature disinhibition as a strong common element. *Meanness* as defined in the Triarchic Model is marked by deficient empathy, low social connectedness, exploitativeness, excitement-seeking, and empowerment through cruelty. In contrast with disinhibition, which reflects general proneness to problems of impulse control, meanness entails more specific tendencies toward callous insensitivity, instrumental use of aggression, destructive fun-seeking, and predatory manipulation of others. The third component of the Triarchic Model, *boldness*, entails social efficacy, calmness under pressure and rapid recovery from stressors, and high tolerance for unfamiliarity and danger. Correlates of boldness include confidence, interpersonal assertiveness, narcissism and thrill-seeking behavior, and low anxiety and depression (Benning, Patrick, Blonigen, Hicks, & Iacono, 2005).

Alternative instruments for assessing psychopathy provide differing coverage of disinhibition, meanness, and boldness in their item content. The PCL-R, designed for use with adult offenders, assesses psychopathy in terms of two distinct factors, an affective-interpersonal factor reflecting a blend of meanness and boldness (Hall, Benning, & Patrick, 2004; Venables & Patrick, 2012) and an antisocial-deviance factor reflecting a blend of disinhibition and meanness (Patrick, Hicks, Nichol, & Krueger, 2007; Venables & Patrick, 2012). The PPI, developed for use with adults from the general community, assesses psychopathy in terms of a fearless-dominance factor that predominantly reflects boldness (Patrick et al., 2009; Sellbom & Phillips, 2013) and an impulsive-antisociality factor that reflects disinhibition and to a lesser extent meanness (Sellbom & Phillips, 2013; Venables & Patrick, 2012); the PPI also contains one subscale, Coldheartedness, that is distinct from its two factors and



that appears to tap meanness specifically (Sellbom & Phillips, 2013). The Antisocial Process Screening Device (Frick & Hare, 2001), designed for use with children and younger adolescents exhibiting conduct problems, assesses psychopathy in terms of impulsive-externalizing and callous-unemotional factors that show contrasting relations with external criterion variables (Frick & Marsee, 2006; Frick, O'Brien, Wooten, & McBurnett, 1994; Frick & White, 2008); inspired by this work, revisions have been proposed for the *DSM-5* that would recognize distinct variants of conduct disorder with and without callous-unemotional traits. ASPD as currently defined in the *DSM-5* emphasizes disinhibition and to a lesser extent meanness (Venables & Patrick, 2012), with negligible coverage of boldness (Patrick, Venables, & Drislane, 2012). As noted earlier, proposed changes to the diagnosis of ASPD in the *DSM-5* would place more emphasis on traits related to meanness (i.e., antagonism) in the diagnosis.

Available research findings point to differing causal factors underlying the disinhibition and meanness components of psychopathy that comprise its points of overlap with ASPD, and the boldness component that distinguishes psychopathy most from ASPD (Patrick et al., 2012). As noted in earlier sections, biometric analyses of child and adult criteria for ASPD point to differing sources of genetic influence for aggressive and nonaggressive symptom components. Behavior genetic research on the etiologic bases of the fearless-dominance (akin to boldness) and impulsive-antisociality factors of the PPI (Blonigen et al., 2005) likewise demonstrates distinct sources of genetic influence for the two. With regard to brain mechanisms, disinhibition is hypothesized to reflect dysfunction in anterior brain systems—including the prefrontal cortex and anterior cingulate cortex—that operate to guide decision-making and action and regulate emotional reactivity. As a result, highly disinhibited individuals operate in the present moment, failing to moderate their actions and reactions as a function of past experiences or anticipated future outcomes.

Different neurobiological processes have been hypothesized to contribute to the meanness (callous-aggression) component of psychopathy, which is represented to some extent also in ASPD. One of these is a weakness in regions of the brain that govern fear reactivity (Fowles & Dindo, 2009; Frick & Marsee, 2006), or perhaps emotional responsiveness more generally (Blair, 2006). As evidence of this, high scores on the callousness-unemotional factor of the Antisocial Process Screening Device are associated with low levels of reported anxiety, lack of responsiveness to distressing stimuli, impaired ability to learn from punishment, and affinity for activities entailing novelty and risk. Beyond this, it seems likely that impairments in biological systems underlying caring (nurturance) and affiliative capacity also contribute to the expression of meanness. For example, disturbances in the function of neuromodulatory hormones such as oxytocin and vasopressin, which are known to influence a range of social phenomena including social bonding, altruism, cooperation versus competition, and recognition of others' emotional displays (e.g., Domes, Heinrichs, Michel, Berger, & Herpertz, 2007; Gobrogge, Liu, Jia, & Wang, 2007; Kosfeld, Heinrichs, Zak, Fischbacher, & Fehr, 2005; Lim et al., 2004), may contribute to the emergence and maintenance of callous-aggressive tendencies.

The third component of the Triarchic Model, boldness, has been conceptualized as reflecting the behavioral (phenotypic) expression of an underlying fearless disposition





(genotype; Fowles & Dindo, 2009; Lykken, 1995; Patrick et al., 2009). Boldness entails a more straightforward, adaptive expression of low fear than meanness; as just noted, the expression of fearlessness as meanness may entail co-occurring disturbances in systems underlying social connectedness and caring. Individual differences in the functioning of the brain's defensive motivational system, including the amygdala and affiliated structures, have been posited to play a role in boldness (Kramer, Patrick, Krueger, & Gaspari, 2012; Patrick et al., 2009). Consistent with this perspective, individuals high on the fearless-dominance factor of the PPI show reduced responsiveness to affective visual (including aversive) stimuli (Benning, Patrick, & Iacono, 2005; Gordon, Baird, & End, 2004). Additionally, experiential factors that promote a sense of personal efficacy and effective top-down regulatory control of emotion may also contribute to individual differences in boldness.

Treatment of Antisocial Personality Disorder

Contemporary Perspectives and Approaches

The symptoms of ASPD and its effects on others present unique challenges for treatment, as a number of elements inherent to the disorder, and its typical pathways to treatment, stand in stark contrast to many theoretically based and empirically supported tenets of effective psychotherapy. For example, a client's internal motivation for change is considered by many to be a prerequisite for successful intervention (Olver, Stockdale, & Wormith, 2011; Ryan, Plant, & O'Malley, 1995), yet the impetus for initiating treatment with ASPD clients often comes from external sources (i.e., the courts). Relatedly, whereas addressing patient-relevant goals is known to enhance motivation and thereby improve treatment outcomes (Michalek, Klappheck, & Kosfelder, 2004; Ryan & Deci, 2008), the primary goal of treatment with antisocial individuals has traditionally been to protect society rather than help these individuals achieve personal goals (Andrews, Bonta, & Hoge, 1990; Skeem & Manchak, 2008; Skeem, Polascheck, & Manchak, 2009). Moreover, while a strong therapeutic alliance can enhance treatment adherence and outcome irrespective of other factors (Martin, Garske, & Davis, 2000), characteristics common to antisocial individuals (e.g., dishonesty, low motivation for treatment; APA, 2000; Reid & Gacono, 2000) and their providers (e.g., pessimism regarding the potential for change; e.g., Salekin, 2002) can hinder development of effective therapist–client relationships. Alliances are especially complex in mandated treatment, where the therapist's dual role (both to care for and control the client) further complicates this tenuous but crucial relationship (Skeem, Eno Loudon, Manchak, Vidal, & Haddad, 2009; Skeem, Eno Loudon, Polaschek, & Camp, 2007).

These challenges are compounded by the fact that researchers and practitioners have historically been cynical about the potential for successful treatment of individuals with psychopathy in particular (e.g., Salekin, 2002), and of criminal offenders more generally (Bailey, 1966; Reid & Gacono, 2000; Robison & Smith, 1971). Up to the 1970s, the field of criminology viewed the potential to rehabilitate offenders in a positive light, but then underwent a paradigm shift in part due to reviews of



intervention studies indicating that existing methods of treatment had “no appreciable effect on recidivism” (Martinson, 1974, p. 25). Two competing perspectives on criminal justice policy emerged at this time, each of which contributed to the devaluation of rehabilitation efforts: On one hand, “left wing” criminologists rejected the possibility that psychological characteristics of individuals could motivate the occurrence of criminal behaviors, advocating for changes in social structures as the best means for reducing recidivism; on the other hand, “right wing” criminologists advocated for a “just deserts” approach (i.e., punishment rather than treatment) for addressing the behavior of offenders (Cooke & Philip, 2000).

Fortunately, the pessimism expressed by these and other groups sparked a new wave of research that has generated a more hopeful outlook regarding prospects for successful treatment of antisocial individuals. Even opponents of rehabilitation, such as Martinson (1974), have acknowledged the pervasive methodological weaknesses in traditional research on treatment of offenders: “It is just possible that some of our treatment programs are working to some extent, but that our research is so bad that it is incapable of telling” (p. 49). Since the 1980s, increases in well-controlled studies and meta-analytic reviews of such studies have challenged longstanding beliefs about the stability of crime-relevant personality traits and the potential for antisocial individuals to undergo change (e.g., Rogers, Jackson, Sewell, & Johansen, 2004). In light of these developments, criminal justice agencies have shifted from the viewpoint that “nothing works” to an emphasis on determining “what works.” Beyond adapting traditional psychotherapeutic techniques for use with antisocial populations, advances in understanding of the neurobiological underpinnings and developmental pathways of antisocial behavior have inspired new perspectives on treatment that focus on dispositional and environmental factors underlying the development and maintenance of antisocial tendencies, and on developing alternative interventions for distinct subsets of antisocial individuals with unique treatment needs (Frick, 2001; Patrick, Drislane, & Strickland, 2012; Seto & Quinsey, 2006).

Cognitive behavioral interventions. At present, cognitive behavioral therapy (CBT) methods comprise the most widely accepted approaches to treatment of antisocial offenders. In general, treatments of this type for offenders emphasize the teaching of cognitive skills theorized to be deficient in these individuals and considered causally relevant to criminal behavior (e.g., Friendship, Blud, Erikson, Travers, & Thornton, 2003; Lipsey, Chapman, & Landenberger, 2001; Samenow, 1991; Yochelson & Samenow, 1976, 1977).

Reasoning and rehabilitation therapy. Although many variants of CBT have been employed with antisocial (particularly offender) populations, arguably the best-known and most widely used method is the reasoning and rehabilitation (R&R) program, developed in the 1980s and now used across a range of settings in various countries (Polaschek, 2011; Robinson & Porporino, 2000). Also referred to as cognitive skills training, R&R is a structured, multifaceted intervention that focuses on criminogenic beliefs and thinking patterns and the role they play in maintaining offense behavior. Because of its focus on behavior patterns as well as affiliated thought processes, R&R



is considered a cognitive behavioral treatment as opposed to a more purely cognitive intervention (Antonowitz, 2005; Fernandez, Shingler, & Marshall, 2006).

R&R was developed in response to evidence that offenders exhibit cognitive deficits relevant to interpersonal problem solving and social interactions, including tendencies to think concretely, fail to consider the consequences of behavior, and disregard or misapprehend others' behavior, thoughts, and feelings (Ross, Fabiano, & Ewles, 1988). The R&R approach contains a number of components designed to address such problems, including modules directed at social perspective taking, interpersonal problem solving, and assertiveness (i.e., nonaggressive ways to communicate), along with training in self-control, critical reasoning, and consideration of values (Robinson & Porporino, 2000; Ross et al., 1988). Typically administered over multiple 2-hour sessions involving groups of six to 12 individuals, and requiring extensive training on the part of therapists (referred to as "trainers" or "coaches"), R&R emphasizes development of skills in a step-wise manner, through repetition, and from diverse learning modalities entailing active participation (e.g., role playing, games) to engage offenders' attention and accommodate a range of client learning styles. A further feature of the R&R approach is that it seeks to enhance motivation on the part of participants by framing the training as an opportunity to develop new skills or ways of thinking that individuals can choose to use outside the training context (Robinson & Porporino, 2000).

Moral reconnection therapy. Another well-known treatment for offenders is moral reconnection therapy (MRT). Developed in the 1980s for the purpose of improving offenders' behavior through improvements in moral and social capacities (Little & Robinson, 1988; Wilson, Bouffard, & MacKenzie, 2005), MRT is also considered cognitive behavioral in orientation. However, in contrast with R&R, it draws specifically on ideas from Kohlberg's (1976) theory of moral development and places distinct emphasis on development of individual identity and self-concept and on drawing connections between moral reasoning and behavior. MRT views offenders as entering therapy with "low levels of moral development, strong narcissism, low ego/identity strength, poor self-concept, low self-esteem, inability to delay gratification, relatively strong defense mechanisms, and relative strong resistance to change and treatment" (Little & Robinson, 1988, p. 135).

Like R&R, MRT is a structured, manualized program administered in treatment groups, typically including 10–15 offenders, with sessions lasting 1–2 hours and offered twice per week. The program revolves around use of a workbook that calls for engagement in a variety of tasks, including identifying goals, exploring good and bad times in one's life, recognizing behaviors that lead to negative outcomes, and recognizing and discussing sources of unhappiness (Little & Robinson, 1988; Wilson et al., 2005).

Other cognitive behavioral approaches. Other CBT-oriented approaches to the treatment of antisocial individuals include Thinking for a Change, developed by the National Institute of Corrections for use with adult offenders on probation (Golden, Gatchel, & Cahill, 2006), and aggression replacement therapy, designed for use with persistently aggressive youth (Glick & Goldstein, 1987). Despite procedural



differences, these and other variants of CBT all emphasize training-based acquisition of thinking skills directed at enhancing self-control and interpersonal relations. This commonality of emphasis might be expected to translate into similar levels of effectiveness for differing treatments of this type. Consistent with this, a recent review of alternative approaches to the treatment of offenders (Landenberger & Lipsey, 2005) found no evidence of differential effectiveness for “brand name” as compared to “generic” forms of CBT with clientele of this type.

Effectiveness of cognitive behavioral therapy for antisocial populations. Various qualitative and quantitative reviews of the empirical literature on treatments for antisocial behavior have converged on the conclusion that cognitive behavioral interventions produce modest but stable reductions in recidivism for offending populations (e.g., Andrews et al., 1990; Antonowitz & Ross, 1994; Friendship et al., 2003; Garrett, 1985; Izzo & Ross, 1990; Landenberger & Lipsey, 2005; Lipsey et al., 2001; Whitehead & Lab, 1989; Wilson et al., 2005). Meta-analyses, in which data from multiple studies are aggregated quantitatively (Rosenthal & DiMatteo, 2001), have been particularly valuable due to their ability to provide composite indices of treatment impact (effect sizes) and identify moderating variables (variables that interact with antisocial status to determine the effectiveness of treatment).

Overall, meta-analytic reviews have established that, on average, offender rehabilitation programs reduce reoffending by approximately 10% (Lösel, 1996). Although small in absolute terms, this level of effect is considered valuable from financial and social standpoints given the heavy costs associated with reoffending—including court, legal, and incarceration-related fees as well as negative effects on victims. Consistent with this, numerous cost–benefit analyses of cognitive behavioral and other intensive treatment programs have reported that programs of these types reduce costs over the long term relative to traditional punishment or inappropriate treatments (e.g., Robertson, Grimes, & Rogers, 2001; Romani, Morgan, Gross, & McDonald, 2012). Beyond improvements in the standard outcome criterion of recidivism, interventions with offenders also appear to produce reliable effects on more clinically relevant indices of improvement such as psychological, community, and vocational adjustment as well as academic performance (Garrett, 1985; Lipsey, 1995).

Factors that moderate effectiveness of treatment. Several factors have been shown to moderate the effectiveness of treatments for antisocial behavior across studies and sites. Some of these are highlighted in the risk–need–responsivity (RNR) model, the dominant conceptual framework guiding treatment of adolescent and adult offenders (Andrews et al., 1990). The RNR model articulates a set of broad principles distilled from reviews of the empirical literature for designing rehabilitation services based on relevant characteristics of individual clients, namely: *risk* (i.e., likelihood of reoffending in more vs. less severe ways), criminogenic *needs* (i.e., underlying motives for antisocial behavior that represent targets for treatment), and abilities and learning styles that affect *responsivity* to treatment (e.g., general intellect). Although these principles have been subject to debate (e.g., risk level and intensiveness of service can be defined in varying ways; Polaschek, 2011) and may be difficult to translate into practice in some cases, they provide useful guidelines that



appear to enhance outcomes (e.g., reduce recidivism) when followed (Andrews et al., 1990; Lowenkamp, Latessa, & Smith, 2004).

Beyond the RNR model components, researchers have identified other specific factors that contribute to treatment effectiveness through additional studies of offender populations. One of these is quality of program implementation (Landenberger & Lipsey, 2005; Lowenkamp et al., 2004)—which poses distinct challenges given that most programs for treatment of offenders reviewed by Lowenkamp et al. (2004) were classified as having “unsatisfactory” adherence to RNR principles. Another factor that has been increasingly recognized as a moderator of treatment success consists of provider characteristics that affect therapist–client (or staff–offender) relationships. Specifically, providers with “firm but fair” or authoritative (as opposed to authoritarian) styles appear most effective in fostering positive alliances that enhance treatment outcomes (Skeem et al., 2007; Skeem, Eno Louden, et al., 2009).

Other therapeutic approaches. Other change techniques that have been used with offenders include psychodynamic therapy and traditional behavior therapies (e.g., token economies; Pearson, Lipton, Cleland, & Yee, 2002). In the case of psychodynamic therapy, evidence for effectiveness is decidedly mixed (Cooke & Philip, 2000), with some studies reporting worse outcomes for treated versus untreated offenders (Andrews et al., 1990; Antonowicz & Ross, 1994). Interestingly, strict behavior modification techniques appear to be less effective than cognitive behavioral interventions for juvenile and adult offenders, according to findings of a meta-analysis of 58 experimental and quasi-experimental studies reported by Landenberger and Lipsey (2005).

Another common approach in the literature is the therapeutic community, a term that has been applied both to psychoanalytically oriented treatments for offenders with serious mental illness used in the United Kingdom and to nonpsychoanalytic treatments used in the United States for offenders with comorbid substance use problems. Empirical findings for approaches of these types are less encouraging, with some prominent studies reporting evidence of adverse effects with certain offender populations (e.g., studies by Ogloff, Wong, & Greenwood, 1990, and Rice, Harris, & Cormier, 1992, have demonstrated worse outcomes for psychopathic offenders undergoing therapeutic community intervention; but see Skeem, Polaschek, Patrick, & Lilienfeld, 2011, for a critique of these studies).

Summary of Existing Treatments of Antisocial Personality Disorder

As a whole, available evidence supports the use of R&R and other cognitive behavioral treatment approaches with offender populations (e.g., Friendship et al., 2003; Landenberger & Lipsey, 2005; Lipsey et al., 2001; Wilson et al., 2005). However, a number of questions remain regarding the essential components of these treatments and how to maximize their effectiveness with more severely antisocial individuals and those exhibiting prominent psychopathic features. Although compelling arguments have been made for the use of multimodal interventions with antisocial clients (Frick, 2001), findings indicating that some treatment approaches may produce adverse effects with certain subgroups of offenders (e.g., psychopaths; Harris & Rice, 2006)



suggest caution in implementing treatments of this kind on a widespread basis. Findings of this kind also highlight the need for further research directed at evaluating the effectiveness of customized interventions for distinct subtypes of individuals in the broader antisocial population (i.e., those exhibiting particular symptomatic features; see the next section). Another question requiring further research is whether positive findings reported for treatments of antisocial individuals (including cognitive behavioral interventions) simply reflect preexisting characteristics of those individuals who appear willing to complete treatment as opposed to effects of the treatments per se. This question is not resolvable through existing literature, because the majority of published studies have used no-treatment control groups and not accounted adequately for dropout. In light of evidence that individuals who stay in treatment tend to be those with lower severity/risk raises the possibility of inflation of estimates of treatment effectiveness in many studies, and suggests that currently available treatments may not be reaching those individuals who need it most (Olver et al., 2011; Olver & Wong, 2010).

Toward the Future: Interventions for Separable Facets of Antisocial Personality Disorder that Reflect Distinct Underlying Processes

Lessons from the Treatment of Child Conduct Problems

The literature on childhood precursors to ASPD offers some useful insights that can help to guide efforts to improve adult treatment. Within the domain of disruptive behavior disorders in the *DSM-IV-TR*, constituent diagnoses include oppositional defiant disorder (ODD), characterized by unusual disobedience and defiance, and conduct disorder (CD), involving more severe behavioral symptoms than ODD and considered the child precursor to adult ASPD. In addition to these established diagnoses, research over the past 15 years has highlighted the diagnostic importance of callous-unemotional (CU) traits for evaluating behavioral risk and treatment amenability in children with conduct problems. This influential body of research has led to a proposal for inclusion of a “CU specifier” in the *DSM-5* to distinguish variants of CD entailing the presence versus absence of traits of this kind, which are considered to be indicative of psychopathy (Frick, 2006; Frick & Ellis, 1999). Because of significant overlap between conduct problems and attentional deficits (up to 90% of children with CD or ODD also have a diagnosis of ADHD; Abikoff & Klein, 1992), research on children with ADHD and multiple disruptive behavior diagnoses is also relevant. Furthermore, the fact that the course of ADHD appears more chronic in children with as opposed to without co-occurring conduct problems (Barkley, Fischer, Smallish, & Fletcher, 2002; Broidy et al., 2003) calls for separate consideration of individuals with differing levels of comorbidity in deciding among alternative methods of intervention.

Currently, four approaches to the treatment of child conduct disorder are considered well-supported: behavioral (contingency) management strategies, parent training, cognitive behavioral programs, and stimulant medications. Behavioral management



strategies entail setting concrete behavioral goals relevant to a child's problem behavior, establishing reinforcement and punishment contingencies in order to gradually shape behavior in more appropriate directions, and providing for effective ongoing monitoring of children's behavior and treatment progress (Abramowitz & O'Leary, 1991). Parent training programs, the best-supported intervention for children with severe CD, teach parents to run contingency management programs within the home and emphasize improving the quality of parent-child relationships (Kazdin & Whitley, 2003). Similar to adult CBT interventions, cognitive behavioral programs for conduct problems address children's social-cognitive and social problem-solving deficits (e.g., through procedures directed at overriding tendencies to attribute hostile intent in others and strengthening ability to inhibit impulsive responding). Stimulant medications are well supported for children with comorbid ADHD and are widely used (Hinshaw, 1991; Hinshaw, Heller, & McHale, 1992).

Considering Individual Differences within the Antisocial Population

Two lessons from this literature are perhaps most relevant to future work on the treatment of ASPD. The first is that there appear to be individual differences among children diagnosed with CD (i.e., subgroups within this diagnostic category) that warrant special consideration in determining treatment. In particular, CU traits and comorbidity with ADHD appear particularly relevant to understanding individual prognoses and treatment needs. A number of studies have reported that CD children with CU traits have more persistent conduct problems (Frick & Ellis, 1999), more difficulties in academic, social, and other areas of functioning (e.g., Pettit, Bates, & Dodge, 1993; Sanders, Dadds, Johnston, & Cash, 1992), and poorer response to standard treatments than CD children without CU traits (Hawes & Dadds, 2005). For example, Hawes and Dadds (2005) found that boys high in CU traits showed less affective reactivity to discipline involving time-outs and as such responded less well to this form of behavioral intervention. Similarly, Haas et al. (2011) found time-outs to be less effective for CD children with CU traits, and in addition, reported that children high in CU traits responded more disruptively to time-outs than those low in CU traits.

Another study that focused on children with comorbid CD and ADHD found that those high in CU traits (CU+) fared worse than those low in CU traits (CU-) in a behavioral-only intervention (Waschbush, Carrey, Willoughby, King, & Andrade, 2007). By contrast, outcomes for CU- and CU+ groups were largely comparable for a combined intervention approach including stimulant medication along with behavioral treatment. The implication of this finding is that behavioral therapy in itself is ineffective with ADHD/CD children exhibiting high levels of CU traits, but that these higher-risk children can be effectively treated with more intensive combined treatments. Despite evidence for a prominent genetic contribution to CU traits (Viding, Blair, Moffitt, & Plomin, 2005) and relative stability of these traits over time (Waller et al., 2012), other work has shown that comprehensive and structured treatments can produce improvements in high-CU/psychopathic children (Hawes & Dadds, 2007; Kolko et al., 2009; McDonald, Dodson, Rosenfield, & Jouriles, 2011).



Notably, just as high-risk CD children appear responsive to more intensive treatment, adolescents and adults with severe antisocial and psychopathic features appear capable of responding to more intensive forms of appropriate treatment (Skeem, Polaschek, & Manchak, 2009). But despite these promising findings, the fact remains that a relatively high proportion of antisocial children and adults who enter treatment (in particular, those with prominent psychopathic features) fail to complete it, or do not respond appreciably even though they do (e.g., Brestan & Eyberg, 1998). Moreover, generalizability of treatment gains across settings and maintenance of treatment gains over time tend to be poor for such individuals (e.g., Kimonis & Frick, 2012). Taken together, these findings suggest that, rather than viewing “offenders” as a broad population with common deficits, subsets of antisocial individuals with particular symptomatic (phenotypic) patterns may require distinct treatment approaches.

In thinking about how CBT-oriented methods might be applied to the treatment of particular subgroups of antisocial individuals, it is useful to consider components of existing treatments in relation to models of disinhibitory (externalizing) psychopathology. For example, the Externalizing Spectrum Model (ESM; Krueger et al., 2007) delineates a range of interrelated problems and traits, some involving more antisocial-aggressive expressions and others more addiction-proneness. Certain components of existing treatments like R&R may be more effective for addressing some elements of externalizing relative to others. In teaching offenders to be more reflective (vs. reactive) and more systematic in pursuing actions and goals, modules focusing on aspects of externalizing relevant to inhibitory control (e.g., planful control, problematic impulsivity, impatient urgency) are likely to be especially valuable. Components aimed at improving perspective taking and flexibility of thought may be particularly useful in modifying interpersonal biases or deficits (e.g., alienation, externalization of blame, deficient empathy, relational aggression, and other tendencies involving hostility toward or lack of consideration for others). By contrast, the R&R intervention approach does not directly target alcohol or drug problems, which are considered part of the externalizing spectrum and co-occur frequently with ASPD, nor does it include modules directed at boredom proneness or tendencies toward excitement seeking. Targeting and evaluating change in specific facets of disinhibition/externalizing such as these would contribute to understanding of the mechanisms by which existing treatments effect change, and also to new approaches for targeting elements of antisociality not addressed by current interventions.

Designing Structured yet Individualized Interventions

The second major lesson to be learned from the child literature is that treatments can be structured and empirically sound while also being tailored to meet the unique needs of individuals. CD (and, by extension, ASPD) appears to develop via multiple pathways (genetic, environmental), affects children across numerous domains/contexts (school, home, community), and is maintained by diverse factors within affected children (e.g., temperament characteristics, social-cognitive deficits) and their environments (Frick, 2006). As such, researchers are increasingly recognizing the need for interventions to be comprehensive and individualized in order to be maximally effective with this



heterogeneous population. One intervention that appears to fulfill these criteria is multisystemic therapy (MST). Based on a systems-oriented perspective on family therapy, MST is inherently comprehensive in that it views child adjustment as embedded within a broader familial, social, and cultural context (Kimonis & Frick, 2010). Importantly, while explicitly tailoring interventions to meet differing needs of individual children, MST in general has garnered strong empirical support (e.g., Henggeler, Melton, & Smith, 1992; Sawyer & Borduin, 2011), setting an example for how treatments can achieve a balance between programmatic implementation and flexibility.

Although adherence to general principles of effective treatment is important to the success of specific intervention approaches (Landenbeger & Lipsey, 2005; Lowenkamp et al., 2004), the degree to which CBT and other treatments are manualized and highly structured limits their ability to accommodate the treatment needs of individuals. The literature reviewed in this chapter suggests that considering client-specific variables is important and that components of treatment may need to be tailored to patterns of strengths and weaknesses on the part of individuals (e.g., intellectual level, personality and temperament characteristics, environmental risk and protective factors) in order to maximize the efficacy of adult therapies in the broader antisocial population.

Future Directions for Treatment of Antisocial Personality Disorder: Incorporating Neuroscientific Concepts and Findings

Another potential avenue for development of improved treatments entails application of findings from research on the neural substrates of ASPD and affiliated conditions, with the aim of more directly targeting cognitive and affective processing deficits characteristic of antisocial and psychopathic individuals (Patrick, Drislane, & Stickland, 2012; Seto & Quinsey, 2006). Child and adult externalizing problems, which show a close relationship with disinhibitory personality traits (Iacono, Carlson, Taylor, Elkins, & McGue, 1999; Krueger et al., 2002), appear to be associated with dysfunction in anterior brain systems critical for monitoring and regulating behavior. For example, integrative reviews have described evidence for impairments in the functioning of the prefrontal and temporal lobes in violent offenders (Davidson, Putnam, & Larson, 2000; Patrick, 2008; Raine & Yang, 2006). Other work has reported impairments in brain event-related potential (ERP) responses, including the P3 (Iacono et al., 2002; Patrick et al., 2006) and the error-related negativity (ERN; Dikman & Allen, 2000; Hall, Bernat, & Patrick, 2007), in individuals with antisocial-externalizing tendencies. Of particular note, the ERN, which occurs following behavioral errors in performance tasks, has direct conceptual relevance to impulse control problems because it directly reflects the extent to which individuals monitor their own actions as they occur.

Pharmacologic interventions may prove useful as an adjunct to forms of CBT in the treatment of antisocial behavior problems. As suggested by the abovementioned work of Waschbush et al. (2007), induction of changes in the functioning of underlying neural systems through pharmacological means may facilitate the effectiveness of



cognitive and behavioral interventions in modifying maladaptive behaviors associated with ASPD and psychopathy. Along these lines, evidence exists for the effectiveness of drug treatments for various impulse-related problems, including impulsive aggression (e.g., lithium, selective serotonin reuptake inhibitors; Minzenberg & Siever, 2006), alcoholism (e.g., acamprosate, naltrexone; O'Malley, Croop, Wroblewski, Labriola, & Volpicelli, 1995; Paille et al., 1995), sexual deviancy (e.g., antiandrogens; Briken, Hill, & Berner, 2003), and pathological gambling (e.g., naltrexone; S. W. Kim & Grant, 2001). That certain drug treatments (e.g., naltrexone) show effectiveness with differing impulse control disorders accords with the idea of a common neurobiological liability underlying problems of this type. From the standpoint of the hierarchical model of externalizing problems (Krueger et al., 2002, 2007), particular pharmacological treatments may prove effective in ameliorating neurocognitive impairments associated with general externalizing proneness, whereas others may be needed to address distinct cognitive and affective processing deviations associated with aggressive and addictive expressions of externalizing.

Other treatment approaches could also be developed to address brain-based processing deviations. For example, behavioral or brain feedback interventions could be used to train individuals to augment internal self-monitoring processes critical to regulation of behavior. Recent work showing that the ERN deficiency characteristic of high-externalizing individuals can be normalized through a feedback-learning procedure (Nelson, Bernat, & Patrick, 2012) provides a demonstration of this approach. Another demonstration is provided by recent research showing that activation in the anterior cingulate cortex (the neural source of the ERN) can be modulated through functional magnetic resonance imaging-based brain biofeedback (Weiskopf et al., 2003).

Beyond cognitive-attentional deficits characteristic of impulse-related problems, antisocial individuals diagnosable as psychopathic show distinct deficits in interpersonal relations and emotional sensitivity. Specialized intervention strategies may be needed to address the lack of empathic concern, exploitativeness, and social disconnectedness that appear central to callous-unemotionality (or "meanness"; Patrick et al., 2009). Features of this sort present obvious, direct obstacles to forming therapeutic alliances. In addition, some portion of individuals meeting criteria for ASPD will exhibit features of dominance, stress immunity, and fearlessness associated with the boldness component of psychopathy (Patrick et al., 2009). Characteristics of these types, entailing strong perceptions of self-efficacy and a lack of concern about the effects of one's behavior on others, are likely to interfere with treatment success by compromising motivation for change.

Neurobiologically, individuals high in affective-interpersonal features of psychopathy (callousness/meanness, boldness) appear to have a heightened threshold for activation of the brain's defensive (fear) system, such that threatening cues must be particularly salient to evoke fear (Levenston, Patrick, Bradley, & Lang, 2000; Patrick, 2007). This is hypothesized to reflect in part dysfunction of the amygdala, a subcortical brain structure crucial to fear and other emotional reactions (Blair, 2006; LeDoux, 2000). Consistent with this perspective, a well-documented finding in the research literature is that antisocial offenders high in affective-interpersonal features of psychopathy show reduced potentiation of the noise-elicited startle reflex during





viewing of threatening stimuli (e.g., Patrick, 1994; Vaidyanathan, Hall, Patrick, & Bernat, 2011)—an effect believed to be mediated by activation of the amygdala (Davis, Walker, & Lee, 1997; Lang, Bradley, & Cuthbert, 1990). Relatedly, children high in callous-unemotional traits showed diminished amygdala reactivity to depictions of fear faces (Jones et al., 2009; Marsh et al., 2008).

Pharmacologic or feedback-based procedures could potentially be used to augment responsiveness in brain regions such as the amygdala that appear underreactive in psychopathic individuals. Alternatively, variants of attentional retraining, a newer class of treatment that has proven effective with clinical conditions of differing types including anxiety disorders (Hakamata et al., 2010), might be developed to enhance emotional sensitivity in psychopathic individuals. In contrast to individuals with anxiety, however, psychopathic individuals would be trained to *enhance* attentional processing of stimuli indicative of threat to oneself or distress on the part of others (cf. Patrick, Drislane, & Strickland, 2012).

Conclusion

ASPD as defined in the *DSM* can be seen as one behavioral expression (facet) of a broader underlying propensity to problems of impulse control. Among disorders within the externalizing spectrum, ASPD is characterized in particular by irritability and aggressiveness along with impulsiveness and irresponsibility. Psychopathy as defined by Hare's (1991) PCL-R intersects with ASPD through its social deviance (Factor 2) component, which taps the broad externalizing factor of which ASPD is an indicator. However, in addition to impulsive-externalizing tendencies, the diagnostic criteria for psychopathy include affective-interpersonal features that reflect emotional insensitivity and interpersonal detachment. Available evidence suggests that this component of psychopathy may reflect different neurobiological mechanisms (i.e., dysfunction in brain circuits underlying defensive/fear reactivity and affiliation/attachment) from the externalizing component (i.e., impairments in anterior regulatory circuitry).

Ultimately, to achieve satisfactory levels of effectiveness, therapeutic interventions for ASPD will need to recognize and contend with the heterogeneity of the disorder. Multifaceted treatment programs that employ cognitive behavioral techniques and brain-oriented training procedures (potentially in conjunction with pharmacologic manipulations) to target specific processing impairments associated with distinct symptomatic features—including general impulsiveness, callous aggression, addictive urges, and insouciant narcissism—may in time offer the best hope for dealing with these challenging and costly disorders (cf. Seto & Quinsey, 2006).

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Abstract: Antisocial personality disorder exacts a costly toll on society and poses many unique challenges for treatment. This chapter reviews historic conceptions of antisocial personality disorder and the related but distinct condition of psychopathy. The systematic co-occurrence of antisocial personality disorder with other diagnostic conditions (e.g., alcoholism, other forms of drug dependence) is discussed, and research is reviewed indicating that disorders of these types (termed "externalizing" disorders) share a common underlying dispositional vulnerability. This is followed by a review of current diagnostic criteria for antisocial personality disorder and changes to the diagnosis that have been proposed for the *DSM-5*, highlighting parallels to the literature (including changes proposed for the *DSM-5*) on conduct disorder, the childhood precursor to adult antisocial personality disorder. The chapter concludes with a discussion of currently available methods of treatment for antisocial populations, emphasizing the best-supported cognitive behavioral approaches, and future directions for treatment based on recent developments in the literature reviewed. Particular emphasis is placed on ways in which interventions could be tailored to meet the unique treatment needs of phenotypically distinct subgroups of antisocial individuals, and how emerging knowledge of the neurobiological underpinnings of antisocial personality disorder and psychopathy might be applied to developing alternative methods of treatment such as pharmacologically based or neuro-reprogramming (e.g., brain-process oriented training, or direct brain biofeedback) approaches that directly target cognitive and affective processing deficits common in these populations.

Keywords: antisocial personality disorder, psychopathy, externalizing, offenders, aggression, treatment, cognitive behavioral, neuroscience