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PERSONALITY AND IMPULSE CONTROL DISORDERS (R LEE, SECTION EDITOR)

Antisocial Personality Disorder: Neurophysiological Mechanisms and Distinct Subtypes

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Abstract

Purpose of Review The trait-oriented triarchic model of psychopathy emphasizes heterogeneity in mechanisms that give rise to antisocial behavior. We review findings from neurophysiological studies that provide evidence for distinct variants of antisocial personality involving different configurations of triarchic model traits—boldness, meanness, and disinhibition.

Recent Findings High boldness is evident in some manifestations of ASPD, where it operates as a "mask" to conceal callous-disinhibitory proclivities. Meanness involves features of low empathy, weak affiliation, and an antagonistic social style that appear related to deficits in sensitivity to pain and distress in others. Disinhibition is associated with impairments in frontal-executive functioning manifested in deficient behavioral restraint and poor affect regulation.

Summary We propose alternative subtypes of ASPD, including "primarily detached" and "primarily disinhibited" variants, with differing propensities for aggression and distinct neurophysiological profiles. Further research is needed to clarify mechanisms for these ASPD subtypes and how best to address them clinically.

Keywords Antisocial · Personality · Psychopathy · Neurophysiology · Subtypes

Introduction

Antisocial personality disorder (ASPD) is a form of personality pathology involving rule- and law-breaking, irresponsibility, impulsivity, and aggressiveness that begins in childhood and persists into adulthood. Psychopathy shares features with ASPD, and both are predictive of violent offenses and other criminal activity [3, 33], but psychopathy is distinguished by additional proclivities toward fearlessness, social dominance, callousness, and emotional insensitivity [75, 79]. Traditional conceptualizations of these conditions [1, 2, 13, 33] have emphasized prototypic features and a categorical approach to diagnosis. In contrast, more recent accounts conceive of antisocial-psychopathic behavior and concomitant cognitive-affective deficits as arising from different pathogenic traits, each continuous/dimensional in nature [22, 83]. Consistent with this view, several studies have documented subtypes of antisocial and psychopathic offenders, showing distinct trait profiles, bolstering the case that psychopathic and antisocial profiles can vary widely [37, 66, 72].

In this paper, we review evidence for different neurophysiological mechanisms contributing to antisocial-psychopathic behavior within the context of the triarchic model of psychopathy [62], a conceptual framework that emphasizes the multidimensionality of ASPD and psychopathy and helps to account for distinct variants (subtypes) of these conditions. Careful delineation of the neurophysiological correlates of the constituent traits associated with ASPD and psychopathy can provide a clearer understanding of etiological processes that contribute to antisocial behavior, and the characteristic diagnostic configurations in which this behavior occurs. We conclude with the proposal of several subtypes of ASPD, depending on the configuration of triarchic traits present within each profile, and the putative neurophysiological mechanisms driving these manifestations.

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Background Information

ASPD is most closely aligned with the construct of disinhibition or dysconstraint [55, 73], which involves impairments in the cognitive control of behavior and affect, resulting in weak regulation of emotions and a lack of appreciation for the feelings and welfare of others [17, 68]. While psychopathy also manifests some of these impairments, it is less consistently associated with cognitive deficits and is instead characterized by reduced reactivity to fear cues and depictions of others' distress [10, 40, 42, 47, 57]. Although the criteria for ASPD focus on criminal and impulsive behaviors (making ASPD highly prevalent among offenders; [5, 26]), some have suggested that ASPD and psychopathy may be better viewed as occupying a common continuum than as separate diagnoses (for more on this topic, see [14]). Accordingly, this review surveys existing literature on neurophysiological correlates of both psychopathy and ASPD, with the ultimate goal of discerning antisocial subtypes that manifest distinct profiles involving alterations in cognitive and affective control, threat and pain processing, and affiliative capacity and reward sensitivity.

The *configural-trait* model of psychopathy emphasizes that ASPD and other personality disorders can result from different combinations of distinct trait dispositions, and depending on the configuration of these dispositions, manifest in quite different ways [45]. As an example, Hicks et al. [37] identified *emotionally stable* and *aggressive* psychopathic subtypes, who scored similarly on psychopathy measures but displayed distinct personality profiles and criminal behavior patterns, with the latter subtype overlapping most with diagnostic features of ASPD. The *configural-trait* model is distinct from the *discrete-syndrome* model, which suggests that conditions such as psychopathy or ASPD are taxonic in nature [36]—representing discrete, nonarbitrary classes, analogous to entities like gender or species. The dimensional model of personality has garnered interest from many researchers in the field, and it was included in a supplemental section within the most recent edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; [3]). In general, there is ample evidence to support the idea that antisocial personality traits occur on a continuum and that individuals with ASPD can present in dramatically different forms [22, 83].

One version of the *configural-trait* model, the triarchic conceptualization of psychopathy [62], focuses on three core dispositions in terms of their combined and differential roles in variants of psychopathy and antisocial-aggressive behavior: boldness, meanness, and disinhibition. Within the context of the triarchic model, ASPD/psychopathy reflects the disinhibition (impulsivity, recklessness) and meanness (aggression, lack of empathy) emphasized in diagnostic criteria for ASPD [3]. Boldness (interpersonal charm, social dominance) is believed by some to be specific to psychopathy [75]; however, there is research to suggest that aspects of boldness (fearlessness, low anxiety) may be exhibited in variants of ASPD (perhaps those more similar to psychopathy; [79]). Theoretically, individuals with ASPD could show distinct configurations of the triarchic traits, with disinhibition a primary mover in some (i.e., impulsive, reactive), meanness in others (i.e., violent victimization of others; [57]), or some combination of these in conjunction with boldness. Research examining subtypes based on the triarchic model dispositions is limited, with only one published study of this type in the literature so far [21]. Of note, this study revealed variants of psychopathy consistent with primary and secondary variants [akin to Hick et al.'s [37] emotionally stable and aggressive subtypes] identified in studies that have used other psychopathy assessments (e.g., [24, 72]).

A key advantage of the triarchic model is that the trait dispositions it specifies are explicitly *neurobehavioral*—that is, they reflect attributes that relate directly to neurobiological systems and behaviors mediated by those systems [61]. With this in mind, our major aim in this paper is to consider different neural processes that have been implicated in ASPD and psychopathy, and discuss how they relate to relevant trait constructs specified by the triarchic model. In particular, we consider research that has examined associations between trait constructs related to ASPD/psychopathy and mechanisms governing cognitive and affective control, threat and pain processing, and affiliation-related reward responding.

Neurophysiological Processes

Low Threat Sensitivity/Emotional Processing: Boldness

The dispositional construct of boldness helps to highlight differences between psychopathy and ASPD, particularly in regard to associations with threat sensitivity. Namely, psychopathic individuals exhibit lower threat sensitivity, whereas many ASPD individuals do not—highlighting the fact that boldness/fearlessness is especially relevant to psychopathy [38, 39]. In fact, Wall et al. [79] have argued that boldness is a key component differentiating psychopathy from ASPD. The research evidence supports this. Hyde et al. [39] found that high psychopathy scores were associated with lower negative emotionality and lower amygdala activity, whereas high ASPD scores were associated with the converse (higher negative emotionality and higher amygdala activity), after controlling for overlapping variance between the two constructs.

Nonetheless, boldness traits have implications for manifestations of ASPD that involve reduced threat reactivity. Research has indicated that boldness is related to fear deficits involving a high tolerance for stress, general unconcern about consequences, and blunted emotional reactivity, resulting in

social poise and calmness under pressure [62]. For example, Ellis, Schroder, Patrick, & Moser [23] found that higher boldness, and *not* meanness or disinhibition, was associated with reduced reactivity to emotionally relevant stimuli (also highlighting the difference between mechanisms driving boldness and meanness). Interestingly, self-report results yielded inconsistencies with the neurophysiological data, in which persons high in boldness reported no blunted emotionality in response to the visual stimuli; this seems to suggest either a lack of insight into the blunted emotionality or a level of deception/positive impression management among individuals high in boldness. Other studies that have examined the interpersonal features of psychopathy in relation to emotional or fear processing attribute these to deficiencies in amygdala functioning [30, 70].

Dispositional boldness can be expressed in either socially problematic or adaptive ways. In terms of problematic expressions, decreased threat sensitivity could contribute to a variant of ASPD that is characterized by an outward appearance of charm and emotional stability coupled with reckless, untrustworthy behavior. Examples of this bold expression of ASPD include manipulative individuals who display a superficially charming persona while casually exploiting others for personal gain—e.g., unscrupulous salesmen, con artists, and racketeers. More adaptive expressions of boldness include prosocial leadership and heroism in military or civilian-emergency service [43]. While meanness and disinhibition appear more closely related to the symptomatic features of ASPD, individuals who exhibit high levels of fearlessness/boldness in conjunction with these other traits are likely to present as most prototypically psychopathic.

Affective Deficits, Pain Processing, and Affiliation/Reward: Meanness

Affective deficits resulting in lack of empathy and disregard for the welfare of others are considered central in traditional conceptualizations of ASPD [3, 34]. However, it is worth noting that only two of the DSM-5 criteria for ASPD are indicative of callousness/meanness (e.g., lacks remorse, irritability/aggression; [3]). Given this, callous-unemotionality or meanness appears specific to more predatory manifestations of ASPD, as evidenced, for example, by findings pointing to different developmental pathways for antisociality with callousness and without callousness [28, 78].

One perspective on the empathic deficits implicated in meanness is that they arise from a fundamental deficit in fear response that interrupts the development of conditioned affective responses to others' distress cues, essential for the development of "conscience" [31, 41, 48]. In contrast to the affective deficits in boldness, those related to meanness are compounded by low affiliative tendencies and reduced sensitivity to the pain of others, manifested in a lack of social concern and a propensity to aggress in proactive ways [18, 60, 62]. Consistent with this perspective, Blair and colleagues (2015) suggested that individuals with ASPD or psychopathic personality traits possess an evolutionary weakness in a neurocognitive process deemed the "violence inhibition mechanism," which curbs aggressive responses to distress cues in normally functioning individuals. In studies seeking to confirm this neurocognitive impairment in antisocial individuals, Blair and his colleagues presented evidence for reduced reactivity to both direct threat and threat toward others in individuals high in psychopathy; further examination of these results uncovered reduced physiological reactivity primarily in relation to the callous-unemotional traits of psychopathy, which bear strong resemblance to the triarchic model meanness construct [6, 62]. It appears that meanness traits, in particular, are related to reduced distress in stressful or potentially harmful situations; thus, it may be more likely that an individual would act aggressively ("fight") instead of instinctually withdrawing ("fleeing") when confronted with threat.

Drawing on work showing that high-ASPD individuals exhibit reduced responses to negative emotional faces and vocalizations (fearful, sad, angry, etc.; [8, 9]), Blair [7] also proposed an *integrated emotion systems* model in which deficits in affective processing were linked to impairment in amygdala functioning—the area of the brain considered most central to fear processing. This hypothesis has been supported by fMRI studies linking reduced amygdala reactivity to deficits in affective face recognition and other forms of emotional processing [81, 82]. Moul et al. [53] postulated that this deficient amygdala activity and resultant inability to detect fear in facial displays is part of a broader inability to develop fear learning and a general insensitivity to distress—leading to a fundamental lack of empathy. This literature strongly suggests that meanness traits, implicated particularly in predatory expressions of ASPD, are linked to functional impairments in the amygdala and affiliated structures comprising the brain's defensive (fear) system. Of note, weak responsiveness of this neural system has also been posited as a mechanism for boldness in the triarchic model [62]. This common element of weak defensive reactivity may account for the partial phenotypic overlap between boldness and meanness [20]. However, these two dispositions clearly differ in important ways, and thus, it is important to consider neural mechanisms that differentiate the two.

One factor that may distinguish callous-unemotionality or meanness from boldness is deficits in the processing of pain, either in oneself or on the part of others. Neural networks for pain processing have been implicated in empathic responding, providing a basis for predicting pain-processing deficits in high-callous individuals. The findings in this domain are mixed, however. Whereas most studies have found evidence

of reduced pain-network activation in individuals identified as high in antisociality or psychopathy [50, 51, 71], a smaller number of studies have reported the opposite—i.e., increased pain-network activation in such individuals [16, 18]. Importantly, there appears to be a difference between "imagine-other" vs. "imagine-self" conditions in tasks requiring individuals to rate pain, such that the imagine-self condition results in higher amygdala activity for antisocial/psychopathic individuals than the imagine-other condition [18, 50]. In the absence of specific instructions one way or the other, findings appear more mixed—with some studies reporting decreased amygdala activation (e.g., [46]) and others enhanced activation [15]. Speculatively, these contrasting findings could be due to differing manifestations of ASPD across studies. Studies finding greater amygdala activation in ASPD may have relied on participants who were primarily deficient in their ability to regulate distressing emotions (high in disinhibition, described below), rather than participants representing antagonistic or fearless manifestations of ASPD. Conversely, studies reporting reduced activation in ASPD may have recruited a more detached or "mean" group of participants, embodying the typical "coldheartedness" of the prototypical psychopath. It is for these reasons that more specific variants/profiles may be specified in order to reconcile seemingly contradictory findings from studies in the realm of ASPD/psychopathy.

Certain brain regions implicated in pain [74], combined with systems for reward processing [57], may influence capacities for affiliation and attachment, integral to developing empathy—capacities likely to be impaired in individuals scoring high on meanness [62]. From this perspective, persons high on meanness would not reap rewards from social connections and consequently would not experience normal levels of pain from loss of relationships. As such, antagonistic/mean individuals are prone to seek out alternative asocial avenues for attainment of pleasure (e.g., selfish exploitation or destructiveness; [60]). This lack of affiliative capacity operates, in tandem with impaired fear and pain processing, to create the profile of an individual who simply does not care about other people and will victimize them in the pursuit of tangible rewards. This variant of ASPD, more so than the disinhibited manifestation, is comparable to typologies of "organized" murderers and power-oriented mass shooters [19, 32, 44, 45]; furthermore, this ASPD variant is reflective of the emotionally stable psychopathic variant discussed by Hicks et al. [37], which is characterized by high agency (assertiveness and control), low stress reactivity, and low social connectedness.

In summary, existing neuroscientific evidence links antagonistic traits in ASPD with deficient sensitivity to distress in others (and, in some cases, lowered nocioceptive reactivity), implicating reduced amygdala activity, impairment in the neural circuitry for pain processing, and affiliative reward system dysfunction that allows for pursuit of goals without regard for others.

Cognitive and Affective Control: Disinhibition

Cognitive control, involving regions of the prefrontal cortex (PFC), is crucial for inhibition of behavioral responses (e.g., [11, 49]). Impairments in PFC functioning are associated with elevated levels of impulsivity; as an example, individuals high in impulsivity tend to perform poorly on inhibitory control tasks such as the Stroop, antisaccade, and stop-signal tasks [85]. Highly impulsive individuals also show reduced amplitude of P3 brain response in a variety of lab-based cognitive processing tasks (e.g., visual oddball, flanker, choice feedback, picture/startle; [54, 64, 65]). Importantly, the observed correlation between trait impulsivity and performance or brain reactivity in tasks of these types appears to reflect common genetic influences to a considerable degree [84, 85]; this makes a compelling argument for a contribution of biological influences to the etiology of disinhibitory conditions including ASPD, along with environmental influences that have been documented (e.g., low SES, deviant peers; [25]).

Recent research on the symptom facets of psychopathy represented in Hare's PCL-R (2003) lend credence to the idea that PFC impairments are more strongly linked to trait disinhibition than to callous-unemotionality (meanness). Specifically, a number of studies have reported that the impulsive-antisocial features of psychopathy (factor 2) are significantly more associated with weak executive functioning than the interpersonal-affective (factor 1) features [56, 69, 80]. Impairments are particularly evident in laboratory tasks that require sustained attention and repeated effortful control [58]. This may be because the processing style of the impulsive individual is reactive and opportunistic: These individuals are *not* playing the long game, which may in fact be adaptive in some contexts of our evolutionary history (i.e., making split-second survival decisions), but maladaptive for behavior patterns in our modern world that require restraint and deliberation.

Abnormalities in P3 brain response have been attributed specifically to disinhibition as defined in the triarchic model, rather than meanness or boldness, based on the overlap between disinhibition and ASPD, as well as the impulsive-antisocial features of psychopathy (versus affective-interpersonal features; [59]). In a meta-analysis of 38 relevant studies, Gao and Raine [29] found significantly reduced P3 amplitudes ($d = 0.252$, $p < .001$) and longer P3 latencies ($d = 0.130$, $p = .019$) for antisocial individuals relative to both normal and clinical comparison groups. Venables & Patrick [76] also reported evidence of reduced P3 amplitude in a prison sample, specifically in relation to the impulsive-antisocial

(factor 2) component of the Psychopathy Checklist-Revised (PCL-R; [33])—the factor known to be most associated with ASPD [35, 63]. Further highlighting the link between low P3 amplitude and trait impulsivity, Carlson et al. [12] reported a negative correlation between P3 amplitude and scores on the Self-Centered Impulsivity dimension of the PPI-R in an undergraduate sample. These lines of evidence indicate that P3 amplitude is a reliable neurophysiological indicator of disinhibition and can be used, in conjunction with other indicators, to identify variants of ASPD that arise from high trait disinhibition—the dispositional factor that relates most to impulsive-antisocial features of psychopathy and that is presumed to underlie "secondary" manifestations of psychopathy. Disinhibitory/impulsive proclivities also manifest in reduced interplay between cognitive and affective processing systems, which is critical to regulation of impulses, especially in emotionally evocative situations. For example, studies have shown that impairment in the PFC, resulting in reduced cognitive and behavioral control, can lower affect regulation capacity and actually heighten levels of negative emotions (e.g., leading to the development of mood and anxiety disorders; [52, 67]). In particular, recent research suggests that poor regulation of emotions and affectively charged cognitions may contribute especially to reactive forms of antisocial or aggressive behavior and may help to explain the progressive dysfunction that leads to acts of mass murder [77]. Another example of the impact of regulatory dysfunction comes from the literature on secondary psychopathy, which characterizes this variant as impulsively aggressive and lacking in emotion regulation capacity (as evidence by prominent anxiousness, depression, social alienation, and compulsive use of substances; [72]). We argue that high trait disinhibition factor is especially prominent in antisocial individuals who are less socially adept and unable to function effectively in society (i.e., "unsuccessful"), given the pervasiveness of their impulsive-irresponsible and reactive aggressive behaviors.

Taken together, PFC impairment and affiliated reductions in executive functioning have wide-range effects on cognitive, behavioral, and affective control; a weakened control capacity creates the propensity for uncontrollable emotions that can then manifest in impulsive and reactive behaviors (i.e., opportunistic antisocial behavior, reactive violent acts).

Conclusions/Implications for Expressions of ASPD

Our dual aims in this review are to encourage a focus on the heterogeneity of psychopathy/ASPD and to discuss neural-systems constructs that represent key targets for research aimed at advancing our understanding of the etiology of antisocial behavior and developing effective interventions for it. Mapping extant neuroscientific findings in the area of ASPD onto the triarchic neurobehavioral trait model lends support to the idea that antisocial and psychopathic proclivities are dimensional and configural [45] in nature—reflecting the occurrence of different combinations of dispositions [62]. From a triarchic model perspective and considering the neurophysiological evidence, we propose the existence of (at least) three distinct subtypes of ASPD, with the three triarchic model traits being differentially influential in these variants. We delineate these ASPD subtypes in Table 1, each paired with the triarchic traits and neurobiological mechanisms with which they are associated, as well as a sample description of how this subtype may manifest.

First, the primarily detached subtype is characterized by affective detachment, as demonstrated by reduced reactivity to the distress of others as a function of deficits in amygdala and pain-network function. The resulting lack of affiliative capacity associated with meanness contributes to the victimization of others, a process distinguishable from disinhibition-related violence that is partly attributable to PFC dysfunction. The primarily detached individual is expected to be strongly prone to premeditated, instrumental violence, unlike the reactive violence more likely to be exhibited by the primarily disinhibited subtype (see below). Violence would be especially likely among individuals of this type who attain dopamine-related reinforcement from exploiting and victimizing others, in lieu of rewards gained from social affiliation per se [60]. Because the mechanisms for this subtype appear different from those for disinhibitory ASPD [27], and given the centrality of impulsiveness in defining ASPD [3], we hypothesize that individuals of this type make up a smaller proportion of ASPD cases.

The primarily detached subtype may also exhibit aspects of boldness (akin to a *DSM* modifier: "with boldness features"), as this profile is most consistent with low threat sensitivity. Boldness would add a distinct appearance of healthy adjustment to the primarily detached subtype, in terms of emotional stability and charm that facilitate gaining the trust of potential victims, in ways consistent with Cleckley's [13] description of psychopathy as "masked" pathology. Individuals high on both meanness and boldness may alternate between their charming/socially slick personas and their callous/predatory presentations, depending on immediate circumstances. For example, the famous serial killer, Ted Bundy, lured his victims through charm and deception, but exhibited extreme callousness and savage violence when alone with the victims.

A second subtype, primarily disinhibited, characterizes ASPD individuals high in impulsiveness and risk-taking behaviors, consistent with representations of ASPD familiar to forensic professionals and researchers. These individuals show PFC-related impairments, resulting in combined dysregulation of affect and behaviors, which manifest in reactively aggressive, impulsive behavior. Supporting this conceptualization, ASPD individuals are typically characterized in the

Table 1 Subtypes of antisocial personality disorder (ASPD)

Type	Triarchic traits	Neurological constructs and mechanisms	Example
Primarily detached	High meanness Low disinhibition and boldness	Impaired fear and pain processing, low affiliative capacity ➔ deficits in limbic and dopamine-striatal systems, altered pain pathways	Find reward through victimization of others; without disinhibition, their violence against others is often premeditated (i.e., rape, assault, murder).
Detached with boldness	High meanness and boldness Low disinhibition	Same as above, with: Blunted emotional processing ➔ impaired amygdala functioning	Similar to the primarily detached subtype, but with the addition of charm and affability to manipulate/seduce victims (i.e., Ted Bundy).
Primarily disinhibited	High disinhibition Low meanness and boldness	Low cognitive/behavioral control and affect dysregulation ➔ prefrontal cortex, prefrontal regulation of affective centers	Typical of offenders who engage in common types of antisocial behavior (i.e., theft, substance use, selling drugs, etc.) due to impulsive and dysregulated tendencies.
Combined liability	High meanness and disinhibition Low boldness	Same as above, with: Impaired fear and pain processing, low affiliative capacity ➔ deficits in the limbic and dopamine-striatal systems, altered pain pathways	These individuals have impulsive tendencies combined with detachment from others and gratification from victimization of others; ensures high likelihood for recidivism, especially violence.
Combined liability with boldness	High boldness, meanness, and disinhibition	Same as above, with: Blunted emotional processing ➔ impaired amygdala functioning	This modification of the combined liability subtype includes the presence of social dominance and interpersonal skills, potentially decreasing the likelihood of apprehension by law enforcement

literature by high negative affectivity (elevated anxiousness or stress reactivity and hostility/aggression) and high impulsiveness, are prone to engage in heavy substance use [37], and exhibit high overall levels of psychopathology [72]. The deficits in cognitive and behavioral control inherent in disinhibited manifestations of ASPD are further supported by a recent neurological model proposed by Verona & Patrick [77], which suggests that chronic activation of the acute threat system leads to impairment of cognitive control functioning over time, thus limiting the resources required for affective and behavioral regulation (and making individuals more likely to act out aggressively). While no study has yet directly examined the prevalence rates of psychopathic/antisocial subtypes, we hypothesize, based on previous subtyping studies [37, 72], that the primarily disinhibited subtype is the most common representation of the proposed subtypes.

Our third proposed ASPD subtype is a combined liability variant, reflecting individuals who possess high trait disinhibition together with core affective detachment—that is, deficits in both executive control and affective/pain-processing networks. In this variant, the presence of affective deficits exacerbates the problems associated with disinhibitory tendencies. Antisocial and violent behavior in these individuals reflects the combined neurophysiological deficits present in the primarily disinhibited and primarily detached subtypes; this combined deficit profile would result in an individual who impulsively victimizes others based on perceived need without regard for harmful consequences. Rather than the controlled, premeditated violence exhibited by the primarily detached subtype, this type is impulsively driven to violence and receives gratification from victimization of others (presumably, via the dopaminergic-striatal system; [60]). This subtype represents a dangerous variant, as the reckless impulse to engage in violent behavior all but ensures that victimization will occur repeatedly.

An additional combined subtype could contain aspects of boldness, in addition to disinhibition and meanness, which would further "fuel the fire" by introducing social efficacy and dominance into the individual's arsenal, making them more manipulative and predatory with their victims. In a way, this subtype could elude detection for a longer period of time, as the boldness features serve to "mask" maladaptive traits. When allocating resources for intervention efforts, individuals showing combined subtypes should be assigned high priority, as they have the highest potential for recidivism.

Throughout this review, we have discussed the critical biobehavioral mechanisms that operate to create varying manifestations of ASPD, focusing on deficits in pain and threat processing, affiliative capacity and cognitive control as major mechanisms underlying dispositional constructs of boldness, meanness/callousness, and disinhibition. We propose three main variants of ASPD—the primarily disinhibited, primarily detached (with or without boldness), and combined liability subtypes (with or without boldness)—reflecting different configurations of these dispositions (and affiliated neural deficits)

and associated with differing propensities for violence/victimization. Future research should seek to validate the existence of these (or similar) subtypes, especially now that the *DSM-5* contains (a) a specifier for conduct disorder (in Diagnostic Criteria and Codes) that differentiates callous and noncallous subtypes and (b) a dimensional-trait system (in Emerging Measures and Models) for personality disorders. In particular, more work is needed to conceptualize and refine the potential subtype manifestations of ASPD/psychopathy, and more work in general is needed to move from the traditional view of ASPD as taxonic toward a continuous, disposition-based conceptualization.

In the context of intervention efforts, Baskin-Sommers, Curtin, & Newman [4] have preliminarily identified distinct cognitive interventions that target the different cognitive-affective deficits associated with disinhibited and bold-mean manifestations of antisociality. This work should continue. We believe that mapping configural trait models to their neurobehavioral correlates can facilitate treatment development, scientifically informed approaches, and tailored interventions. Our review shows that trait-based approaches can allow for more nuanced understanding of ASPD and psychopathy, potentially leading to success in reducing and/or preventing violence.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

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

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