

## Psychobiological Aspects of Antisocial Personality Disorder, Psychopathy, and Violence

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The key focus is on understanding violent offending (eg, reactive, proactive, firearm violence) tied to antisocial personality disorder and psychopathy using a psychobiological lens.

Antisocial personality disorder (ASPD) and the related condition of psychopathy (psychopathic personality) hold strong interest for researchers and practitioners because of their chronic nature and costly impact on society. Individuals who exhibit symptoms of these clinical conditions account for a disproportionate number of crimes, including violent offenses, and are more likely to reoffend than other adjudicated individuals.<sup>1</sup> A clear understanding of psychological and biological processes underlying these conditions and their predictive relations with violence is essential for developing effective treatment and prevention programs for curtailing violent victimization.

The key focus of this article is on understanding violent offending (eg, reactive, proactive, firearm violence) tied to ASPD and psychopathy using a psychobiological lens to aid in characterizing pathological processes that give rise to acts of violence.

In reviewing the literature on ASPD and psychopathy and the biological bases of their overlap and relations with violent offending, we argue that most forms of ASPD-related violence reflect disturbances in the functioning of negative valence and cognitive control systems, and in the normal adaptive interplay between the two. This is in contrast to affective-interpersonal features specific to psychopathy that entail disturbances in emotional processing (deficient fear and empathy) and social relations (domination, exploitativeness) that give rise to more proactive forms of violence. After reviewing relevant existing work, we extend from the general case to discuss the rare but highly impactful phenomenon of mass shootings. In these cases, hostile-alienated tendencies commonly seen in ASPD take an unusually dark turn, toward fantasy-fueled acts of violent vengeance.

### Definitions and distinctions

Contemporary diagnostic nosology defines ASPD primarily in terms of observable norm-violating behaviors, rather than more internal affective states or personality traits (conceived in DSM-III). DSM-5 contains 2 definitions of ASPD—one in “Diagnostic Criteria and Codes,” the other in “Emerging Measures and Models.” The first is a traditional criterion-based, categorical diagnosis carried over without revision from DSM-IV. The diagnosis is assigned to individuals aged 18 or older if 3 or more of 7 adult symptoms are exhibited together with symptoms of conduct disorder earlier in life (ie, before age 15). With the exception of items pertaining to remorse and deceptiveness, the diagnostic criteria for ASPD largely reflect behavioral tendencies associated with a highly reckless, impulsive (ie, disinhibited) disposition.

The second definition contains a new dimensional system for characterizing personality pathology that includes trait-based definitions of 6 distinct personality disorders, including ASPD. ASPD is characterized in this system by elevations on personality disorder-related traits from 2 categories: antagonism and disinhibition—a combination supported by structural and biometric analysis of adult symptoms of ASPD.<sup>2</sup>

While ASPD is defined primarily in terms of impulsive, aggressive, and illicit behaviors, psychopathy is defined by impulsive-antisocial behavior occurring together with distinct affective-interpersonal features that entail a dominant and forceful social style, manipulativeness, callousness/cruelty, and emotional insensitivity. The assessment instrument that has dominated contemporary research on psychopathy is the Psychopathy Checklist-Revised (PCL-R), developed to identify the condition in incarcerated offenders.<sup>1</sup> While the PCL-R is devised to assess psychopathy as a unitary condition, factor analyses of its 20 items have revealed correlated ( $r \approx 0.5$ ) subdimensions or factors. Most published work has focused on a 2-factor model that specifies an interpersonal-affective factor (Factor 1) marked by superficial charm, grandiosity, conning/deceptiveness, absence of remorse or empathy, shallow affect, and externalization of blame, and an impulsive-antisocial factor (Factor 2)

that encompasses early behavior problems, impulsivity, irresponsibility, prone to boredom, lack of long-term goals, and hot-tempered aggressiveness.

These PCL-R factors show contrasting relations with a variety of criterion measures, particularly when their shared variance is accounted for.<sup>3</sup> Variance unique to Factor 1 correlates negatively with measures of anxiousness, internalizing problems, and empathy, and positively with measures of social dominance, grandiose narcissism, Machiavellianism, and proactive aggression.<sup>3,4</sup> By contrast, variance unique to Factor 2 correlates positively with trait anxiety, internalizing problems, and suicidality, as well as with measures of impulsivity, aggressiveness, general sensation seeking, and substance dependence.<sup>4,5</sup> These differential associations for Factors 1 and 2 indicate that the items of the PCL-R, although selected to index psychopathy as a unitary diagnostic entity, are in fact tapping separate constructs—one that entails emotional insensitivity and social dominance, and the other impulsiveness and hostile negative affectivity—common to both is antagonism or callous aggressiveness.<sup>6,7</sup>

A framework that is useful for understanding how ASPD relates to the two factors of PCL-R-defined psychopathy and, in turn, to violent behavior, is the externalizing spectrum model.<sup>8</sup> ASPD co-occurs reliably with other DSM disorders—particularly disruptive behavioral conditions and substance use disorders—and like ASPD, these disorders show associations with impulsiveness and hostile negative affectivity.<sup>6</sup> Krueger and colleagues<sup>8</sup> proposed that a broad dispositional liability contributes to various disorders in the externalizing spectrum and personality traits that relate to them. Using a biometric analysis of twin data, they demonstrated that the common factor is substantially (approximately 80%) heritable.

Statistical modeling research indicates that variance specific to Factor 2 of the PCL-R, which accounts largely for the relationship with ASPD, reflects this broad externalizing liability.<sup>3</sup> Impulsive tendencies and hostile negative affectivity associated with externalizing proneness, prominent in ASPD, account substantially for the reactive violence associated with this diagnostic condition. By contrast, callous-unemotional tendencies characteristic of PCL-R Factor 1 and its overlap with Factor 2 are associated with more predatory, proactive expressions of aggression.<sup>7,9</sup>

### **ASPD, psychopathy, and aggression/violence**

It is not surprising that ASPD includes proclivities toward aggression and violence. ASPD-related violence can be characterized as predominantly reactive (ie, involving immediate, angry responses to provocations).<sup>9</sup> Impulsive fighting and assaults toward acquaintances or family members are in fact common.<sup>4</sup> Items associated with Factor 2 of the PCL-R also index externalizing proneness in a way that intersects with callous-aggressive tendencies, which may substantially account for the relationship between PCL-R psychopathy and violent behavior. Consistent with this, a meta-analysis by Kennealy and colleagues<sup>10</sup> of data from several studies revealed that PCL-R psychopathy was predictive of violent behavior largely as a function of the impulsive-antisocial features encompassed by Factor 2.

One interpretation of these results is that disinhibitory and callous-aggressive dispositional tendencies associated with Factor 2 account for the predictive relationship between PCL-R scores and violent behavior. However, an alternative possibility raised by Kennealy and colleagues<sup>10</sup> is that scores on PCL-R Factor 2 may appreciably reflect the stability across time of aggressive antisocial behavior (most directly, through inclusion of “early behavior problem” and “poor behavioral control” items that specifically refer to aggressive acts).

Although violence appears to be strongly influenced by genes associated with externalizing proneness (disinhibition), the specific manner in which this propensity is expressed (eg, aggression, alcohol or drug dependency, reckless risk taking) appears to be determined substantially by environmental influences.<sup>8</sup> From this perspective, persistent severe aggressive behavior associated with ASPD may partly reflect high levels of externalizing proneness shaped toward violent criminal expression by adverse physical and social experiences. Indeed, ASPD and Factor 2 of psychopathy, in contrast to Factor 1, have been associated with elevated rates of adversity and abuse, and lower educational attainment.<sup>5</sup>

Given such evidence, it is important to consider that a high proportion of persons with ASPD live in communities with poor economic resources and limited opportunities that may promote norm-violating behaviors. Adverse experiences and threats to safety associated with violent communities may, in concert with aberrant dispositional tendencies (ie, impulsiveness and hostile negative affectivity), account substantially for the elevated sensitivity to provocation and threat seen in individuals with ASPD.

### **Neurophysiology of ASPD and violence**

*ASPD and reactive violence.* Given the evidence that the shared variance across externalizing

spectrum behaviors is highly heritable, consideration of neural systems implicated in antisociality and violence may help identify the mechanisms for the higher incidence of violence among those with ASPD. Neuropsychological studies of antisocial individuals have yielded evidence of deficits in cognitive and executive function, as assessed by frontal lobe tasks, and abnormalities in brain potential measures, such as the P300 response.<sup>3,11</sup> Given theoretic models that interpret the P300 as reflecting post-perceptual processing of salient stimuli within a task, reduced amplitude of this brain response in ASPD (and externalizing conditions more broadly) implies some impairment in higher cognitive processing of events.

Consistent with this, brain imaging studies of antisocial individuals—including structural-anatomic studies, and investigations of regional brain activation at rest and during performance of differing task procedures—indicate impairment in neural systems undergirding cognitive functions and inhibitory control, specifically the prefrontal cortex and temporal lobes.<sup>11</sup> Yang and Raine<sup>12</sup> undertook a quantitative review of individuals with externalizing disorders (ASPD, conduct disorder, psychopathy, criminality, aggression) and found consistent evidence of deficits in the right orbitofrontal cortex, left dorsolateral prefrontal cortex, and right anterior cingulate cortex. These regions govern cognitive control and inhibition through top-down mechanisms, including engagement of frontoparietal circuitry to increase attentional control in the face of reward/motivational signals.

In addition to deficits in cognitive/inhibitory control systems, there is evidence that ASPD, particularly when involving violence, is associated with heightened reactivity to environmental irritants and increased sensitivity to motivationally relevant cues. ASPD, its childhood precursors (conduct disorder, delinquency), and variance specific to psychopathy Factor 2 show positive relations with scale measures of dispositional negative affect.<sup>3</sup> Moreover, the presence of emotional events can interfere with appropriate behavioral inhibition in ASPD.<sup>13</sup>

A typical functional neuroimaging profile of violent individuals entails the combination of reduced activity in the prefrontal regions and increased activity in subcortical (limbic) regions.<sup>11,12</sup> Thus, while disinhibition and prefrontal functioning deficits may represent the core vulnerability to ASPD, the affective disruption of cognitive control is a highly relevant predisposing process for violence, particularly reactive violence, in individuals with ASPD.

The **Figure** illustrates potential neurophysiological processes in reactive violence among persons with ASPD that involve functional connections between negative valence and cognitive system circuits in the brain. The model posits reciprocal interplay between the acute threat system and cognitive control circuitry, including subdivisions of prefrontal cortex and the anterior cingulate.<sup>14</sup>

The activation of threat-relevant circuits introduces bottom-up signaling of the visual cortex and the vigilance network to bias attention toward perceived threats. Dysregulation in threat responding can occur in a sustained way, manifested in the chronic activation of the brain's vigilance network and the hypothalamic-pituitary-adrenal axis (hormonal stress response), reduced threshold for defensive activation, impaired attentional disengagement, and rumination.<sup>15</sup>

These sustained negative affective processes would, in turn, be expected to exert ever more disruptive effects on cognitive control circuitry. Activity in the orbitofrontal regions, the inferior frontal gyrus, and anterior cingulate networks is particularly disrupted.<sup>16</sup> Engagement of other disrupting cognitive processes, such as rumination or hypervigilance, may place additional load on cognitive control systems and further limit resources required for affective and behavioral regulation. *Psychopathy and proactive aggression.* The previous model, which emphasizes interplay between negative valence and cognitive control systems, is most relevant for understanding reactive violence. Other processes are likely to be of greater importance in proactive (instrumental) violence, including deficits in perception and understanding of others (eg, empathy), dispositional fearlessness, and a socially dominant orientation. Processes of these types, central to callous-aggressive tendencies typical of criminal psychopathy, are expected to foster more extreme and proactive forms of violence. Data from child and adult psychopathy literature indicate that features of callous unconcern, guiltlessness, and emotional unresponsiveness are linked to higher engagement in proactive forms of aggression.<sup>9,17</sup>

### Mass shootings

The model of reactive violence depicted in the **Figure** can be applied to an understanding of extreme acts of gun violence that have occurred in the US in recent years. Given the dearth of empirical research on this class of severely violent individuals and the low prevalence of such acts, any descriptive-explanatory account is partly speculative. However, concepts and findings from the neurophysiological literature on violent individuals can provide unique perspective on factors contributing to this type of violence, which may have important implications for early identification

and prevention.

Case studies of individuals who have engaged in mass shootings indicate a history of social isolation and awkwardness or rejection.<sup>18,19</sup> Symptoms of distress and dysphoria, low self-esteem, and suicidal thoughts or urges are common.<sup>19,20</sup> These symptoms are linked to higher threat sensitivity, which may, in turn, interfere with (possibly already weak) executive functions and cognitive control—in particular, the ability to reflect, gain perspective, and engage in useful problem solving. In opposition to constructive goal setting and action, the intersection of negative emotionality and decreased cognitive-reflective functions is expressed as a hopeless perspective for the future and an antagonistic orientation toward others (ie, externalizing blame for problems, social alienation, misanthropy) and angry rumination.<sup>21</sup>

We posit one additional process that differentiates mass murder from more common instances of violence: a transition from chronic, passive, angry rumination to more proactive fantasies or obsessive thoughts of revenge.<sup>20</sup> Recurrent thoughts that place the blame of one's misfortunes on others can evolve into fantasies of retribution that engage attention in a focused, energizing manner, and provide a gratifying escape from pervasive negativity and hopelessness/powerlessness. Indeed, obsession with firearms or violent media, which help promote feelings of agency and control, is common in such cases.<sup>19</sup> Although this sense of empowerment appears more naturally characteristic of individuals exhibiting the core affective-interpersonal features of psychopathy, it becomes attainable mainly through fantasy in hostile/alienated individuals.

The potential for hostile negative affect to shift toward more empowering drive states is supported by neuroscientific research findings. In particular, there is evidence that in contrast to other negative emotional states, such as fear or disgust, anger connects to distinct neural circuitry and is associated with an approach-oriented pattern of frontal brain activation.<sup>22</sup> Other research has shown that stress exposure involving socially aversive contexts can activate brain changes indicative of approach motivation that, in turn, predict level of aggressive behavior.<sup>14</sup> Specific mechanisms that contribute to activation of approach tendencies under conditions of provocation include release of dopamine within the prefrontal cortex and connected areas.

As applied to the phenomenon of retribution-motivated mass murder, the involvement of the dopamine system provides a mechanism for a recursive process that can dampen, and over time supersede, feelings of distress and low self-worth. Specifically, thoughts or images of active retaliation instigate a natural appetitive process associated with dopamine release, which opposes negative withdrawal-oriented processes. In certain susceptible individuals, the release of dopamine and attendant reduction in distress energizes and strengthens such thoughts and increases the likelihood of re-occurrence.

Incentive sensitization via dopamine operates in a self-amplifying manner (ie, desire increases with repetition), particularly in susceptible individuals when distress-reduction is involved.<sup>23</sup> Thoughts or images of retaliation that are at first fleeting can evolve into persistent, elaborative fantasies of retribution. First, they support the fantasized revenge act, so that it becomes more satisfying and possible with every iteration. Second, fantasies enhance psychological capability by desensitizing the individual to fears as well as to the suffering of victims, which, in turn, increases the ability to enact the imagined attack.

The case of the Atlanta day trader, Mark Barton (**Sidebar**) illustrates the pathological processes associated with acts of mass murder.

## Conclusions

From a neuroscientific standpoint, proneness toward angry-reactive aggression reflects dysfunction in brain systems that mediate negative emotional reactivity and cognitive control. In aggression-prone individuals, fantasies of active retaliation can provide relief from feelings of dysphoria and alienation by engaging appetitive drive systems; if nurtured, the fantasies can progress toward enactment.

The key questions that need to be addressed through further research are:

- What dispositional tendencies, identifiable at an early age, place certain individuals at risk for violent behavior?
- What steps can be taken to prevent dispositional risk for violence from being actualized over time?
- Can processes that contribute to episodes of extreme retaliatory violence, such as mass shootings, be detected as they emerge—and be interrupted through intervention?

We can have effective answers to these questions sooner rather than later if violent behavior is viewed as the major public health problem it in fact is.

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Figure. Interplay between cognitive and negative valence systems as re...

### Case Study | Mark Barton

"I have been dying since October. Wake up at night so afraid, so terrified that I couldn't be that afraid while awake. It has taken its toll. I have come to hate this life in this system of things."

Having driven himself to the point of financial ruin through risky stock trading over a period of months, Mark Barton murdered his wife and children in July 1996. He then walked into 2 brokerage offices in Atlanta and killed 8 people and wounded 13 others before taking his own life. The above excerpt from his suicide note characterizes Barton's frame of mind at the time of the murders.

As a child, Barton was emotionally distant and ostracized by other children. During his teen years, he exhibited impulsive, thrill-seeking behavior in the form of burglaries and regular use of illegal drugs. Indications are that he used drugs in part to cope with persistent feelings of loneliness and dysphoria. Despite his emotional problems, Barton was able to complete college and hold a series of jobs, including one as president of a manufacturing company. The first documented instance of retaliatory behavior occurred when he was fired from this executive position. He sabotaged company data files, was caught, and served a brief jail term.

Barton's first marriage began to dissolve and he became involved in an affair. Around this time, his wife and mother-in-law were found bludgeoned to death by an unknown assailant; Barton was considered a prime suspect, but he denied involvement in the killings (a denial reiterated in the suicide note he composed before his 1996 shootings) and the available evidence was insufficient to charge him.

Barton married his mistress following his wife's death, and reportedly he lapsed into episodes of depression and paranoia within a short time. He and his second wife drifted apart and were separated at the time of the fatal shootings. Using the insurance money from his first wife's death, Barton began engaging in high-risk stock transactions (day trading). However, he lost over \$180K by the summer of 1999, at which time his access to accounts required for trading was removed.

In his suicide note, Barton referred to securities personnel he'd dealt with as "people who greedily sought my destruction." He likewise described his wife as "one of the main reasons for my demise." According to his note, he shot his children after killing his wife to protect them from the pain associated with the loss of their parents. However, Barton's angry accusations were mingled with expressions of regret and loss: "I wish I didn't..." and "I had to take him [his son] with me..." Barton killed himself when cornered by police. In his possession at the time of his death were multiple firearms and over 200 rounds of ammunition.

### Case Study | Mark Barton

#### Disclosures:

Dr Verona is Associate Professor and Director of Clinical Training at the University of South Florida, Tampa. Dr Patrick is Professor of Psychology at Florida State University, Tallahassee. The authors report no conflicts of interest concerning the subject matter of this article.

#### References:

1. Hare RD. *The Hare Psychopathy Checklist— Revised*. Toronto: Multi-Health Systems; 1991.
2. Kendler KS, Aggen SH, Patrick CJ. A multivariate twin study of the DSM-IV criteria for antisocial personality disorder. *Biol Psychiatry*. 2012;71:247-253.
3. Patrick CJ, Bernat EM. Neurobiology of psychopathy: a two-process theory. In: Berntson GG, Cacioppo JT, eds. *Handbook of Neuroscience for the Behavioral Sciences*. New York: John Wiley & Sons; 2009: 1110-1131.
4. Schoenleber M, Sadeh N, Verona E. Parallel syndromes: two dimensions of narcissism and the facets of psychopathic personality in criminally involved individuals. *Personal Disord*. 2011;2:113-127.
5. Verona E, Hicks BM, Patrick CJ. Psychopathy and suicidality in female offenders: mediating influences of personality and abuse. *J Consult Clin Psychol*. 2005;73:1065-1073.
6. Lynam DR, Leukefeld C, Clayton RR. The contribution of personality to the overlap between antisocial behavior and substance use/misuse. *Aggress Behav*. 2003;29:316-331.
7. Venables NC, Patrick CJ. Validity of the Externalizing Spectrum Inventory in a criminal offender sample: relations with disinhibitory psychopathology, personality, and psychopathic features.

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*Psychol Assess.* 2012;24:88-100.

- 8.** Krueger RF, Hicks BM, Patrick CJ, et al. Etiologic connections among substance dependence, antisocial behavior, and personality: modeling the externalizing spectrum. *J Abnorm Psychol.* 2002;111: 411-424.
- 9.** Porter S, Woodworth M. Psychopathy and aggression. In: Patrick CJ, ed. *Handbook of Psychopathy.* New York: Guilford Press; 2006:481-494.
- 10.** Kennealy PJ, Skeem JL, Walters GD, Camp J. Do core interpersonal and affective traits of PCL-R psychopathy interact with antisocial behavior and disinhibition to predict violence? *Psychol Assess.* 2010;22:569-580.
- 11.** Patrick CJ. Physiological correlates of psychopathy, antisocial personality disorder, habitual aggression, and violence. In: Kumari V, Boutros N, Bob P, eds. *Current Topics in Behavioral Neuroscience, Vol. 14: Psychophysiology in Psychiatry and Psychopharmacology.* New York: Springer; 2014:197-226.
- 12.** Yang Y, Raine A. Prefrontal structural and functional brain imaging findings in antisocial, violent, and psychopathic individuals: a meta-analysis. *Psychiatry Res.* 2009;174:81-88.
- 13.** Verona E, Sprague J, Sadeh N. Inhibitory control and negative emotional processing in psychopathy and antisocial personality disorder. *J Abnorm Psychol.* 2012;121:498-510.
- 14.** Verona E, Sadeh N, Curtin JJ. Stress-induced asymmetric frontal brain activity and aggression risk. *J Abnorm Psychol.* 2009;118:131-145.
- 15.** Brosschot JF. Markers of chronic stress: prolonged physiological activation and (un)conscious perseverative cognition. *Neurosci Biobehav Rev.* 2010;35:46-50.
- 16.** Arnsten AF. Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Rev Neurosci.* 2009;10:410-422.
- 17.** Frick PJ, Ray JV, Thornton LC, Kahn RE. Can callous-unemotional traits enhance the understanding, diagnosis, and treatment of serious conduct problems in children and adolescents? a comprehensive review. *Psychol Bull.* 2014;140:1-57.
- 18.** O'Toole ME. *The School Shooter: A Threat Assessment Perspective.* Darby, PA: DIANE Publishing; 2000.
- 19.** Verlinden S, Hersen M, Thomas J. Risk factors in school shootings. *Clin Psychol Rev.* 2000;20:3-56.
- 20.** Scheff TJ. Social-emotional origins of violence: a theory of multiple killing. *Aggress Violent Behav.* 2011;16:453-460.
- 21.** Liem M. Homicide followed by suicide: a review. *Aggress Violent Behav.* 2010;15:153-161.
- 22.** Carver CS, Harmon-Jones E. Anger is an approach-related affect: evidence and implications. *Psychol Bull.* 2009;135:183-204.
- 23.** Karoly HC, Harlaar N, Hutchison KE. Substance use disorders: a theory-driven approach to the integration of genetics and neuroimaging. *Ann N Y Acad Sci.* 2013;1282:71-91.

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