# Physiological Correlates of Psychopathy, Antisocial Personality Disorder, Habitual Aggression, and Violence

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**Abstract** This chapter reviews the existing literature on physiological correlates of psychopathy, antisocial personality disorder, and persistent violence/aggression. Coverage is provided of findings from studies utilizing peripheral, electrocortical, and neuroimaging measures. The review begins with a discussion of how psychopathy and antisocial personality are defined, and how these conditions relate to one another and to violent behavior. A case is made that the relationships psychopathy and ASPD show with violent and aggressive behavior, and similarities and differences in associations of each with physiological measures of various types can be understood in terms of symptomatic features these conditions have in common versus features that distinguish them. Following this, an overview is provided of major lines of evidence emerging from psychophysiological and neuroimaging studies conducted to date on these conditions. The final section of the chapter summarizes what has been learned from these existing studies and discusses implications and directions for future research.

**Keywords** Psychopathy · Antisocial personality disorder · Aggression · Violence · Autonomic response · EEG/ERP · Neuroimaging

## Contents



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## 1 Introduction

This chapter reviews what is known about physiological correlates of psychopathy, antisocial personality disorder (ASPD), and aggression/violence based on findings from studies employing peripheral, electrocortical, and neuroimaging measures. A key theme of the review is that the relationships psychopathy and ASPD show with violence and aggression can be understood in terms of diagnostic features these conditions share and those that distinguish them. In turn, divergences in observed physiological correlates of psychopathy as compared to antisocial personality and aggression can be understood in terms of common and distinctive features.

The chapter is organized into three sections. The first discusses conceptions of psychopathy and antisocial personality and their relations with one another and with violent behavior. The second section provides an overview of major lines of evidence emerging from psychophysiological and neuroimaging investigations of these conditions that have been published to date. The third section summarizes existing findings and discusses implications and directions for future research.

## 2 Phenotype Descriptions and Interrelations

### *2.1 Psychopathy and ASPD: Conceptions, Measures, and Distinguishable Facets*

Historic conceptions of psychopathy have emphasized reckless, unrestrained behavior in conjunction with distinct affective–interpersonal symptoms including shallow affect, lack of close relationships, and an appearance of psychological stability ("a convincing mask of sanity"; Cleckley 1941/1976; see also Hare 1980, 2003; Lykken 1957). By contrast, the diagnosis of antisocial personality disorder (ASPD) in the third and fourth editions of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III and IV; American Psychiatric Association (APA) 1980, 2000) focused predominantly on impulsive–antisocial tendencies—beginning in childhood, and continuing on into adulthood—with limited representation of affective–interpersonal features aside from deceptiveness and lack of remorse. The DSM-IV conception of ASPD was maintained without revision in the main diagnostic codes part (Section II) of the latest, fifth edition of the DSM (APA 2013). However, DSM-5 also contains a new dimensional system for characterizing personality pathology (in Section III, "Emerging Measures and Models") that includes

alternative trait-based definitions of certain personality disorders, including ASPD. This trait-based definition provides more balanced coverage of affective–interpersonal and impulsive–antisocial features (Strickland et al. 2013; Anderson et al. in press), and includes a trait-based specifier for designating a classically low-anxious, socially efficacious (i.e., "primary psychopathic"; Karpman 1941; Skeem et al. 2007) variant of ASPD.

Alternative conceptions of psychopathy are embodied in differing contemporary assessment instruments. The dominant inventory used with adults in clinical and forensic settings is the interview-based Psychopathy Checklist-Revised (PCL-R; Hare 2003). Adaptations have been developed for children and adolescents, including an interview-based youth version (PCL-YV; Forth et al. 2003) and the child-oriented Antisocial Process Screening Device (APSD; Frick and Hare 2001) and Child Psychopathy Scale (CPS; Lynam 1997), which rely on informant ratings. Various self-report instruments also exist for assessing psychopathy. Some are patterned after the PCL-R, including Hare's Self-Report Psychopathy Scale (SRP; Williams et al. 2007), the Levenson Self-Report Psychopathy Scale (LSRP; Levenson et al. 1995), and the Youth Psychopathic Traits Inventory (YPI; Andershed et al. 2002). Others have been developed separately from the PCL-R. The most widely used of these in recent years has been the Psychopathic Personality Inventory (PPInv; Lilienfeld and Andrews 1996; Lilienfeld and Widows 2005).[1]

*Subdimensions of psychopathy*: A shift has occurred over the past several years from the idea of psychopathy as a unitary entity to a view of psychopathy as multifaceted—that is, as composed of distinguishable symptomatic subdimensions, or factors. The PCL-R, for example, contains distinct affective–interpersonal and impulsive–antisocial factors (labeled "1" and "2", respectively) even though its items were selected to index psychopathy as a unitary construct (Hare 1980). While intercorrelated, these factors show differing relationships with various criterion variables (Hare 2003; Patrick and Bernat 2009). PCL-R Factor 1 shows selective associations with narcissism, instrumental aggression, and adaptive qualities such as lack of anxiety or depression, whereas Factor 2 shows preferential positive relations with reactive aggression, substance use problems, and suicidal behavior. Factor 2 also accounts for the moderate-level relationship between the PCL-R and ASPD diagnoses or symptoms; controlling for overlap with Factor 1, scores on PCL-R Factor 2 are unrelated to ASPD (Verona et al. 2001).

Factor analyses of the PCL-R's main childhood counterpart, the APSD, have also revealed distinct callous–unemotional (CU) and impulsive/conduct problems (I/CP) subdimensions. Children who score high on both appear average or above average in intellect and show reduced reactivity to stressors, failure to learn from punishment, and high levels of both reactive and proactive aggression, whereas those high on the I/CP factor alone tend to be below average in intellect and show heightened stress reactivity and emotional liability along with increased reactive

[1] PPInv is used here in place of the more standard abbreviation PPI to avoid confusion with the psychophysiological phenomenon of prepulse inhibition, also abbreviated PPI.

(but not instrumental) aggression (Frick and Marsee 2006; Frick and White 2008). These findings served as the impetus for inclusion of a "low prosocial emotions" specifier for the diagnosis of conduct disorder in DSM-5—allowing for designation of a callous–unemotional (i.e., "psychopathic") variant of this child behavior disorder.

Distinct subdimensions are also evident in all contemporary self-report inventories for psychopathy. Like the PCL-R itself, inventories patterned after it have correlated factors (e.g., Andershed et al. 2002; Levenson et al. 1995; Williams et al. 2007). By contrast, the PPInv—which was developed to index basic trait dispositions associated with psychopathy without specific requirements for convergence—has two higher-order factors that are largely uncorrelated. These factors, labeled fearless dominance and impulsive antisociality by Benning (2005a), show differential relations with criterion variables in domains of self-report, interview based assessment, and physiology (for a review, see Patrick and Bernat 2009). Notably, the PPInv contains one subscale, Coldheartedness, which fails to load appreciably on either of these factors—instead emerging as a separate subdimension in structural analyses (Benning et al. 2003). As discussed further below, this subscale appears to index callous–unemotionality or meanness more exclusively than the other subscales of the PPInv.

*Subdimensions of ASPD*: The childhood criteria for ASPD in Section II of DSM-5 mirror those for conduct disorder (CD) and include aggressive and destructive behaviors along with theft/deceptiveness and non-aggressive rule-breaking acts. Factor analyses of the CD diagnostic criteria (e.g., Frick et al., 1991; Tackett et al. 2003) have shown that the aggressive and rule-breaking symptoms define separate, albeit correlated factors. Follow-up twin studies have demonstrated differing sources of genetic and environmental influence for these factors (Tackett et al. 2005; Kendler et al. (2013), with the proportion of symptom variance attributable to genes higher for the aggressive than the rule-breaking factor (see review by Burt 2009). Evidence for two distinct factors underlying the adult symptoms of ASPD—a disinhibition factor encompassing tendencies toward impulsivity, irresponsibility, and deceitfulness and an aggressive-disregard factor reflecting irritability/aggressiveness, reckless behavior, and lack of concern for self or others—has also been reported Kendler et al. (2012). Paralleling findings for the subdimensions of CD, these adult ASPD factors appear to reflect differing sources of genetic influence.

*Clarifying relations among differing psychopathy measures and ASPD*: *The Triarchic model*. The Triarchic model of psychopathy (Patrick et al. 2009) was advanced as a framework for integrating alternative conceptions, organizing findings pertaining to psychopathy subdimensions, and guiding research on neurobiological correlates and etiologic influences. The model characterizes psychopathy as encompassing three distinct but intersecting symptomatic (phenotypic) constructs: disinhibition, boldness, and meanness. *Disinhibition* entails impulsiveness, weak restraint, hostility and mistrust, and difficulties in regulating emotion; *meanness* entails deficient empathy, lack of affiliative capacity, contempt toward others, predatory exploitativeness, and empowerment through cruelty or destructiveness; and *boldness* encompasses tendencies toward confidence and social

assertiveness, emotional resiliency, and venturesomeness. The Triarchic model provides a frame of reference for relating subdimensions of psychopathy to those of ASPD. In addition, the constructs of the model have biobehavioral referents and show replicable associations with physiological variables and thus can be helpful for relating psychopathy and ASPD to neurobiology (cf. Patrick et al. 2012).

The Triarchic Psychopathy Measure (TriPM; Patrick 2010) was developed to index the three constructs of this model. Its disinhibition and meanness subscales index disinhibitory externalizing and callous aggression factors of the externalizing psychopathology spectrum (Krueger et al. 2007); the TriPM's Boldness scale indexes fearless–dominant tendencies associated with the common factor among scale measures of fear and fearlessness (Kramer et al. 2012). The TriPM has been used as a referent to evaluate coverage of the Triarchic model facets in differing psychopathy inventories. The PPInv provides balanced coverage of boldness, meanness, and disinhibition as indexed by the TriPM (Drislane et al. 2014a; Sellbom and Phillips 2013). Subscales that demarcate the PPInv's fearless dominance factor relate very strongly to TriPM Boldness, scales demarcating PPInv impulsive antisociality relate very strongly to TriPM Disinhibition (particularly carefree non-planfulness and blame externalization/alienation), and moderately to meanness (mainly due to Machiavellianism Egocentricity), and the PPInv Coldheartedness scale relates specifically to TriPM Meanness. By contrast, other psychopathy inventories index meanness and disinhibition either more so than boldness (e.g., SRP, YPI) or to the exclusion of boldness (e.g., LSRP; Drislane et al. 2014a; Sellbom and Phillips 2013; Hall et al. 2014).

The TriPM has also been used to clarify similarities and differences between PCL-R psychopathy and ASPD in terms of the Triarchic model (Venables et al. 2014; Wall et al. in press). This work shows that (a) overall scores on the PCL-R contain variance associated with all three Triarchic model constructs, whereas ASPD indexes the meanness and disinhibition constructs only; (b) PCL-R Factor 1 is associated with boldness and meanness but not disinhibition; and (c) Factor 2 is associated with disinhibition and meanness but not boldness. These findings serve to clarify the relationship between PCL-R psychopathy and ASPD: The two overlap in terms of Factor 2, which includes common elements of disinhibition and meanness, but differ in elements of boldness and meanness that are represented exclusively in PCL-R Factor 1 (cf. Patrick et al. 2007).

## *2.2 ASPD and Psychopathy: Associations with Aggression and Violence*

Disorders such as ASPD and substance abuse/dependence co-occur at high rates (Krueger 1999) and show relationships in common with disinhibitory personality traits (i.e., impulsivity, sensation seeking, nonconformity; Krueger et al. 2002). The variance in common among disorders within this externalizing spectrum has been shown to reflect a highly heritable liability factor (Krueger et al. 2002; Young et al.

2000), labeled externalizing proneness (Krueger et al. 2002, 2007), or disinhibition (Patrick et al. 2009, 2013a). By contrast, the variance specific to each disorder appears to be attributable more to non-shared environmental influences. From this perspective, aggressive behavior associated with ASPD in part reflects high levels of externalizing liability, shaped toward violent criminal expression by adverse physical and social experiences encountered by individuals across time.

However, research directed at modeling externalizing problems and traits more comprehensively (Krueger et al. 2007) demonstrates factors distinct from the general disinhibitory-externalizing factor, reflecting callous–aggressive tendencies and proneness to abuse various substances. The finding of distinct disinhibitory and callous–aggression factors coincides with aforementioned evidence for distinct etiologic influences contributing to aggressive versus rule-breaking subdimensions of CD and adult ASPD, and with evidence for separable callous–unemotional and impulsive–disruptive subdimensions to child psychopathy (Frick and Marsee 2006). Other above-noted work that has directly evaluated relations between PCL-R psychopathy and ASPD from the perspective of the Triarchic model (Venables et al. 2014; Wall et al. in press; see also Patrick et al. 2005, 2007; Venables and Patrick 2012) shows that PCL-R Factor 2 reflects externalizing proneness (disinhibition) and elements of callous–aggression (meanness) in common with ASPD, whereas PCL-R Factor 1 reflects boldness and other elements of callous–aggression separate from ASPD.

Considering these findings, a key question is whether documented predictive relations for PCL-R psychopathy with violent offending and criminal recidivism are accounted for by the features it shares in common with ASPD, or by tendencies that distinguish it from ASPD. As reviewed by Kennealy et al. (2010), the answer appears to be that PCL-R psychopathy is predictive of violent behavior largely as a function of features encompassed by Factor 2 (i.e., disinhibition and affiliated aspects of meanness). Using a meta-analytic, regression-based approach in which scores on the two PCL-R factors were evaluated as concurrent predictors, these authors found that the antisocial deviance features associated with PCL-R Factor 2 were substantially predictive of violence (effect size $d = 0.40$), whereas the affective–interpersonal features associated with Factor 1 were only mildly predictive ($d = 0.11$). Further analyses were undertaken to examine whether affective–interpersonal (Factor 1) features might interact with impulsive–antisocial (Factor 2) features to predict elevated risk for violence in a non-additive fashion (cf. Hare and Neumann 2009). Results indicated that these two components of PCL-R psychopathy did not contribute interactively to violence prediction.

Similarities and distinctions between psychopathy and ASPD, and relations of each with violent behavior, are important to consider in reviewing the literature on physiological correlates of these conditions. In particular, it can be expected that physiological correlates will be more similar among impulsive violence, ASPD, and Factor 2 of psychopathy than between impulsive violence or ASPD and Factor 1 of psychopathy or psychopathy as a whole.

# 3 Physiological Correlates of Psychopathy, Antisocial Personality, and Aggression

## *3.1 Peripheral Measures: Cardiovascular, Electrodermal, and Startle Blink Responses*

Studies of children and adolescents exhibiting antisocial behavior have yielded consistent evidence of lower resting levels of autonomic activity—most notably heart rate (HR), but also to some extent skin conductance (SC)—in comparison with control youth (Lorber 2004; Ortiz and Raine 2004). The finding of low resting HR in particular is especially robust among children with aggressive behavioral tendencies (Scarpa and Raine 1997). Findings for autonomic *reactivity* to noxious or threatening stimuli have been more mixed, but as a whole the available evidence points to *enhanced* HR and SC response to stressors in children exhibiting aggressive conduct problems specifically (Lorber 2004). This is particularly the case for children exhibiting *reactive* aggression; proactively aggressive children if anything tend to show attenuated reactivity to stressors compared with control children (Hubbard et al. 2002).

Other studies focusing on parasympathetic versus sympathetic mediation of cardiovascular activity have yielded evidence of weaker vagal–parasympathetic regulation in children and adolescents with aggressive conduct problems—reflected in enhanced HR variability under circumstances involving stressors or challenges (Beauchaine et al. 2001; Mezzacappa et al. 1997). It has been hypothesized that this lack of vagal control combines with chronic underarousal and weak inhibitory capacity (reflected in lower resting HR and reduced spontaneous SC responses, respectively) to lower the threshold for impulsive aggressive behavior (Beauchaine et al. 2001). Other research has identified weak vagal control as a variable associated with the development of both internalizing (emotional dysregulation) and externalizing problems in at-risk children (e.g., El-Sheikh et al. 2001). Taken together, these findings are consistent with the idea that aggression in children entails difficulties regulating anger and other emotional reactions—with consequent enhancement of defensive reactivity under conditions of threat.

In studies of adults, one prominent focus has been on autonomic (particularly cardiac) reactivity differences in individuals high on aggression-related traits such as hostility, anger expression, and Type A personality. A meta-analysis of relations between trait hostility and cardiovascular reactivity by Suls and Wan (1993) reported that although effects in studies of this kind were generally small, positive results were especially evident in studies that examined relations between dispositional hostility (particularly when defined by overt expressions of anger such as verbal and physical aggression) and cardiac (particularly blood pressure) reactivity in situations involving interpersonal stress or provocation as opposed to physical stressors. Studies published since this meta-analysis have not yielded positive findings in all cases (see, e.g., Gallo et al. 2000), but significant effects when obtained have generally been in the direction of heightened autonomic reactivity for

high trait-aggressive individuals during interpersonal stress (e.g., Smith and Gallo 1999; Peters et al. 2003).

Studies comparing autonomic reactivity in adults with and without a history of violent behavior have yielded less consistent results. For example, physiological studies of men who have assaulted their romantic partners have not revealed consistent differences in relation to non-assaultive men. Gottman et al. (1995) suggested a possible explanation for this in terms of two distinct subgroups of male batterers: one exhibiting decreases in HR activity during a marital interaction (Type 1) and the other exhibiting increases in HR (Type 2). Type 1 batterers scored higher on antisocial traits and were more hostile and contemptuous toward their spouses and more assaultive toward other people in general, whereas Type 2 batterers scored higher in social dependency. However, this pattern of results has not been replicated in subsequent studies (Babcock et al. 2004; Meehan et al. 2001).

Most published studies examining autonomic response have focused on detecting simple reactivity differences between aggressive and non-aggressive individuals. Only a few studies have sought to assess underlying psychological processes contributing to such differences. One of these was a study by Verona et al. (2002) that examined the mediating role of negative emotional activation in enhancing punitive behavior among aggression-prone individuals. These investigators used increased magnitude of reflexive startle responding to index unpleasant activation associated with threat versus absence of threat in a laboratory aggression paradigm. Individuals high on traits of anxiousness, alienation, and aggressiveness showed enhanced unpleasant activation during shock-threat periods (as evidenced by heightened startle reactivity to unwarned noise probes), and in conjunction with this, enhanced aggressive behavior (i.e., delivery of stronger shocks to a putative co-participant). This was interpreted as supporting the perspective that negative emotional activation operates to prime aggressive behavior (Berkowitz's 1990). Subsequent work (e.g., Verona and Curtin 2006) has shown this facilitative effect of negative emotion on aggression to be stronger in men than women, in line with prior research findings (cf. Hokanson 1970).

In sum, research to date has generally revealed lower baseline levels of autonomic arousal, but increased autonomic reactivity to stressful events, in aggressive children and adolescents. Findings for adults have been less consistent, but in general have indicated enhanced autonomic reactivity to stressors (interpersonal stressors in particular) in hostile–aggressive individuals. Notably, the general finding that aggression-prone individuals show enhanced autonomic reactivity to stressful events fits with the hypothesis that violent behavior entails a breakdown in normal affective regulatory capacity (Davidson et al. 2000).

In contrast, markedly different results are evident in the psychophysiological literature on adult psychopathy. Most of these studies have relied on diagnoses based on Cleckley's criteria or Hare's PCL-R. Adult psychopathic offenders, relative to non-psychopathic offenders, show *reduced* electrodermal response to aversive cues and during anticipation of stressful events (Arnett 1997; Hare 1978; Lorber 2004). However, psychopathy is not consistently associated with differential HR reactivity to aversive or stressful stimuli (Lorber 2004), or with differential

baseline levels of either HR or electrodermal arousal (Arnett 1997; Hare 1978; but see Hansen et al. 2007). These contrasting results are noteworthy because higher levels of PCL-R psychopathy in offender samples are reliably associated with increased violence and violent recidivism (Porter and Woodworth 2006). However, as discussed earlier, it is the impulsive–antisocial (Factor 2) component of the PCL-R that is most predictive of violent offending. This factor reflects disinhibitory–externalizing tendencies along with elements of callous aggression (meanness), and a subset of offenders who score very high on the PCL-R exhibit personality profiles and behavioral tendencies characteristic of extreme externalizing individuals (i.e., high negative affectivity along with low behavioral restraint; Hicks et al. 2004). The other, affective–interpersonal factor of the PCL-R reflects boldness along with elements of meanness that cohere more with boldness than with disinhibition (Patrick et al. 2009; Venables et al. 2014; Wall et al. in press), and another subset of individuals who score as psychopathic on the PCL-R shows personality profiles characteristic of high fearless dominance or boldness (i.e., low anxiety, high social potency, low harm avoidance; Hicks et al. 2004). This factor of the PCL-R tends to be associated more with instrumental/proactive aggression than impulsive/reactive aggression, both in adult offenders (Porter and Woodworth 2006) and clinic-referred youth (Frick and Marsee 2006).

While at odds with results for impulsive–aggressive individuals, the finding of reduced autonomic (in particular electrodermal) reactivity to stressors in psychopathic individuals is consistent with theories that have emphasized insensitivity to punishment or diminished fear capacity in psychopathy (e.g., Lykken 1995)—especially in relation to its affective–interpersonal features (Patrick 1994; Patrick and Bernat 2009). Direct evidence for reduced electrodermal reactivity to stress in relation to affective–interpersonal features in offenders was provided by Patrick (1995), who reported opposing correlations (positive and negative, respectively) for scores on PCL-R Factors 1 and 2 with amplitude of skin conductance response during anticipation of aversive noise. In more recent work with non-offenders, Dindo and Fowles (2011) reported reduced electrodermal activation during stressor anticipation as a function of high scores on PPInv fearless dominance (akin to boldness), but not PPInv impulsive antisociality, which reflects disinhibition to a substantial degree along with lesser representation of meanness. Additionally, evidence for a selective association of PPInv fearless dominance with electrodermal reactivity to aversive picture stimuli was reported by Benning et al. (2005b).

Complementing these findings for electrodermal reactivity deficits is another body of literature demonstrating a lack of startle reflex potentiation during viewing of aversive picture stimuli in offenders rated high on PCL-R Factor 1 (Patrick et al. 1993; Vaidyanathan et al. 2011; Vanman et al. 2003; see also Patrick 1994) or community participants scoring high on PPInv fearless dominance (Benning et al. 2005b). This deficit in startle potentiation is not seen for individuals scoring high on PCL-R Factor 2 or PPInv impulsive antisociality alone. The absence of startle potentiation during aversive cuing provides strong evidence for deficient fear because startle potentiation is directly indicative of defensive motivational priming (Lang et al. 1990) and as such covaries with individual differences in dispositional

fear (Kramer et al. 2012; Vaidyanathan et al. 2009). Extending this literature on startle modulation during picture viewing, Newman and colleagues have tested for psychopathy-related differences in startle reactivity to noise probes during exposure to shock threat versus safety cues, under conditions of concurrent distraction or no distraction (Baskin-Sommers et al. 2011; Dvorak-Bertsch et al. 2009; Newman et al. 2010). Findings from these shock threat studies indicate that deficits in startle potentiation for offenders high on the PCL-R or non-offenders high on PPInv fearless dominance occur mainly under conditions of concurrent distraction. This result has been interpreted as indicating that reduced reactivity to fear cues in individuals with affective–interpersonal features of psychopathy reflects "idiosyncrasies in attention that limit their processing of peripheral information" (Newman et al. 2010, p. 66). However, the finding can be also interpreted as evidence for deficient fear reactivity, insofar as threat cues normally exert an automatic "pull" on attentional resources (Bradley 2009; LeDoux 1995).

Regardless of interpretation, these findings for electrodermal reactivity and startle modulation indicate that individuals exhibiting core-affective features of psychopathy need to be considered separately from other types of violent offenders in attempting to understand physiological processes in aggressive behavior. A similar conclusion has emerged in the literature on antisocial behavior in youth, where it has been shown that children or adolescents who exhibit callous–-unemotional tendencies in conjunction with conduct problems show distinctly different behavioral responses to laboratory stressors and (as discussed further below) differential brain reactivity to fear-relevant cues.

## *3.2 Electrocortical Measures: EEG and ERP*

Early investigations of brain differences in violent criminal offenders focused on abnormalities in electroencephalographic (EEG) activity. A relatively consistent finding in these early studies was enhanced cortical slow-wave activity, particularly in the delta (<4 Hz) frequency range (cf. Volavka 1990). While much of this early literature suffered from notable methodological weaknesses, subsequent research using improved designs and procedures has successfully replicated this finding, with some work demonstrating prediction of antisocial behavior later in life (i.e., official criminal convictions) from increased slow-wave EEG activity in adolescence (Raine et al. 1990). Theoretical interpretations of the association between slow-wave EEG and violent offending have focused on cortical immaturity resulting in impaired inhibitory control (Volavka 1990), and cortical underarousal that predisposes toward compensatory stimulation seeking (Raine et al. 1990).

Associations with aggression have also been reported for various components of the cortical event-related potential (ERP)—reflecting average changes in voltage at scalp recording sites across time following the presentation of a stimulus or the emission of a response. The most consistent finding has been reduced amplitude of the P300 response component in oddball tasks where participants respond to

intermittent target stimuli interspersed with more frequently occurring nontargets. Diminished P300 has been reported especially among individuals exhibiting aggression of the impulsive variety (e.g., Barratt et al. 1997; Branchey et al. 1988; Gerstle et al. 1998). In view of theoretic models that interpret P300 response as reflecting brain activity associated with post-perceptual processing of salient stimuli within a task (Donchin and Coles 1988; Polich 2007), reduced P300 amplitude in impulsively aggressive individuals implies some impairment in higher cognitive–elaborative processing of stimulus events.

Reduced P300 response has also been reported in individuals with ASPD (Bauer et al. 1994) and other impulse control disorders—most notably alcohol dependence (cf. Polich et al. 1994), but also drug dependence, nicotine dependence, child conduct disorder, and attention-deficit hyperactivity disorder (Iacono et al. 2002). Given aforementioned evidence for a common liability factor underlying these various conditions (Krueger et al. 2002), Patrick et al. (2006) tested the hypothesis that reduced P300 amplitude reflects this shared liability factor and found clear supportive evidence. Subsequent research has corroborated this finding and demonstrated that the association between externalizing proneness (disinhibition) and reduced P300 reflects common genetic influences (Hicks et al. 2007; Yancey et al. 2013).

In contrast with findings from studies of impulsive aggressive individuals, Stanford et al. (2003) reported no difference in P300 amplitude to auditory target stimuli in psychiatric outpatients characterized as "premeditated aggressors" compared with controls. Similarly, Barratt et al. (1997) found no evidence of a relationship between premeditated aggression and P300. Results from these studies indicate that the association between reduced P300 and aggression may be specific to individuals who manifest aggression of an impulsive nature. Studies examining the relationship between psychopathy and P300 amplitude have yielded mixed results, with some showing a negative association, others a positive association, and still others no association (Gao and Raine 2009). As noted earlier, these inconsistent findings could reflect the fact that a diagnosis of psychopathy includes affective–interpersonal features in addition to impulsive–antisocial symptoms; the affective–interpersonal features, which tend to be associated more with proactive rather than impulsive aggression (Porter and Woodworth 2006), may moderate the relationship between psychopathy and P300 response in some samples. In support of this, Venables and Patrick (2014) examined effects for the two PCL-R factors separately and found the relationship with P300 amplitude to be specific to PCL-R Factor 2. Parallel results were reported by Carlson et al. (2009) for the two factors of the PPInv: Reduced P300 amplitude was associated significantly with scores on the Impulsive Antisocial factor, whereas no relationship was evident for the Fearless Dominance factor.

Although P300 response amplitude is the most widely studied ERP correlate of antisocial–externalizing conditions, some other brain potential correlates have been reported in the literature. One is the error-related negativity (ERN), a negative-polarity response occurring approximately 50 ms after the commission of errors in speeded performance tasks, believed to reflect early "endogenous" error processing

associated with the neural signaling function of the anterior cingulate cortex. Reduced ERN has been reported for individuals high in impulsive–antisocial tendencies (Dikman and Allen 2000; Pailing and Segalowitz 2004) and in relation to externalizing proneness or disinhibition (Hall et al. 2007; Patrick et al. 2013b).

However, as with P300, findings for ERN and psychopathy have been mixed. Munro et al. (2007) examined relations between psychopathy as indexed by overall scores on the SRP (Williams et al. 2007) in two variants of a "flanker" task (Eriksen and Eriksen 1974): one involving discrimination of letter strings and the other discrimination of fearful versus angry faces. Task performance and ERN amplitude in the letter discrimination version of the task were comparable between high and low psychopathic participants, but high-psychopathy participants were less accurate and exhibited reduced ERN amplitude in the emotional face flanker task. In a subsequent study, Brazil et al. (2009) reported relatively intact amplitude of the ERN within a letter discrimination flanker task in high-PCL-R psychopathy forensic patients as compared to controls. However, a reduction in the psychopathic group was evident for amplitude of the post-error positivity (Pe), an ERP component considered similar to P3 and thought to reflect later evaluative stages of performance monitoring. The psychopathic group also evidenced decreased behavioral recognition of errors (i.e., reduced ability to signal via a button press when they noticed an error had occurred). A subsequent study by von Borries et al. (2010) reported reduced amplitude of ERN response along with increased error rates and impaired learning of task contingencies in psychopathic forensic patients during a probabilistic learning task that included feedback (either a monetary gain or loss) regarding performance accuracy on each trial.

Notably, studies of ERN response in psychopathy have not systematically evaluated effects for distinguishable factors or facets known to exhibit differential relations with measures of various types—including physiological response measures (e.g., aversive startle potentiation, P300). The more consistent finding of reduced ERN as well as P300 in relation to impulsive–externalizing tendencies, together with evidence for a selective association of P300 with Factor 2 of psychopathy (whether assessed via the PCL-R or the PPInv), suggests that more consistent evidence of reduced ERN is likely to be found in relation to this symptomatic component of psychopathy. To the extent this component of psychopathy is more related to impulsive–aggressive tendencies, it would be expected that reductions in ERN (indicative of impairments in online monitoring of behavior and recognition of incorrect or inappropriate responses) would also be associated with proclivities toward angry/reactive aggression. A study by Kramer et al. (2011) found no difference in ERN between groups scoring high versus low (upper/low quartile) on an aggression questionnaire, but the participants were college students rather than offenders or clinic patients, and the questionnaire measure focused on aggression of various types rather than angry/reactive aggression specifically.

### 3.3 Neuroimaging Studies of Psychopathy, ASPD, and Aggressive Behavior

Different neuroimaging methods have been used in studies of psychopathy, ASPD, and aggression. Many recent studies have used magnetic resonance imaging (MRI), which quantifies variations in the alignment of endogenous subatomic particles within a magnetic field to index anatomic details of the brain (structural MRI) or variations in blood flow and blood oxygenation (i.e., hemodynamic, or blood-oxygen-level-dependent [BOLD] response) associated with neuronal activity in specific brain regions (functional MRI, or fMRI). Computerized tomography (CT), a structural imaging method that measures regional density of neural tissue using X-ray beams passed through the brain, was used in some older studies of individuals identified as violent or antisocial. Other functional imaging techniques that have been used in studies of psychopathic and antisocial–aggressive individuals are single-photon emission computerized tomography (SPECT) and positron emission tomography (PET). Both rely on the injection of radioactive tracer isotopes into the blood in small amounts, with particles emitted by the isotope from brain regions of interest (photons in the case of SPECT, positrons in the case of PET) used to index either neuronal activity or neurotransmitter function in those regions.

*Structural imaging studies*: Two older studies by Tonkonogy (1991) and Wong et al. (1994) that tested for brain anatomic differences in psychiatric patients with violent behavior using CT reported evidence for abnormalities in temporal lobe regions. A third study by Blake et al. (1995) found evidence of abnormalities in frontal as well as temporal brain regions in a sample of 31 homicide offenders. More recent studies have used structural MRI to investigate neuroanatomic differences associated with impulsive–aggressive behavior and ASPD. Tiihonen et al. (2008) reported gray matter volume reductions in bilateral regions of frontal cortex (frontopolar, orbitofrontal) in persistently violent offenders. Two other studies—one involving temporal lobe epilepsy patients with aggressive–assaultive behavior (Woermann et al. 2000) and the other female patients diagnosed with borderline personality disorder (van Elst et al. 2003)—reported evidence of reduced gray matter volume in regions of prefrontal cortex, and the latter of these also reported volume reductions in anterior cingulate cortex (ACC), hippocampus, and amygdala. However, another study by Dolan et al. (2002) that compared impulsive–aggressive patients with controls reported a significant reduction in temporal but not frontal lobe volume, and two other studies that examined subcortical structures (Laakso et al. 2000; van Elst et al. 2000) found no difference between violent and nonviolent patient groups in hippocampal or amygdala volume. Studies reporting reduced gray matter volume in prefrontal regions in individuals diagnosed with ASPD include Raine et al. (2000), Laakso et al. (2002), and Narayan et al. (2007). The latter two of these studies focused on ASPD individuals exhibiting salient violent behavior.

Evidence for neuroanatomic abnormalities in individuals scoring high in psychopathy as defined by the PCL-R has emerged from more recent studies utilizing structural MRI. Reported findings include reduced volume of gray matter in frontal

and temporal regions of cortex (Müller et al. 2008a; Yang et al. 2005), reduced volume bilaterally of the amygdala (Yang et al. 2009) and posterior hippocampus (particularly in relation to scores on PCL-R Factor 1; Laakso et al. 2001), other hippocampal abnormalities in the form of left/right volume asymmetry (Raine et al. 2004) or deviations in shape (Boccardi et al. 2010), increased volume of white matter in the corpus callosum (Raine et al. 2003), and increased volume of the striatum—with enhanced size of the lenticular nucleus in particular predicted by overall PCL-R scores, and increases in caudate body and caudate head volumes associated, respectively, with scores on PCL-R Factors 1 and 2 (Glenn et al. 2010a). In addition, a study by Craig et al. (2009) that used the MRI-based method of diffusion tensor imaging reported evidence for reduced structural integrity of the uncinate fasciculus, a neural pathway connecting the ortbitofrontal cortex and the amygdala, in a sample of nine forensic patients scoring high (>25) on the PCL-R compared with a non-forensic control group. Notably, the one study to date that tested specifically for differences in the ACC and its dorsal and ventral subregions (Glenn et al. 2010b) found no associations with PCL-R psychopathy, either in comparisons of high- versus low-PCL-R total score groups or in correlational analyses utilizing continuous PCL-R total and factor scores.

In sum, the foregoing structural MRI studies of psychopathic individuals have yielded evidence for reduced volume of limbic structures (hippocampus, amygdala) and impaired structural connectivity between amygdala and orbitofrontal cortex, along with isolated indications of increased volume of corpus callosum and striatal structures and mixed evidence for volume reductions in prefrontal and temporal brain regions. Thus, some overlap is evident in findings for psychopathic and violent/antisocial samples (i.e., volume reductions in frontal and temporal regions), along with some divergence (i.e., subcortical–limbic volume reductions mainly in psychopathic samples). The question of how psychopathic participants compare neuroanatomically with violent–antisocial individuals was directly addressed in a recent structural MRI study by Gregory et al. (2012), who tested for gray matter volume differences in predefined brain regions across the following groups: (1) violent offenders meeting diagnostic criteria for both ASPD and PCL-R psychopathy ($n$ = 17), (2) violent offenders meeting criteria for ASPD but not PCL-R psychopathy ($n$ = 27), and (3) healthy non-offenders from the community at large ($n$ = 22). The psychopathic/ASPD offender group showed significant gray matter reductions bilaterally in anterior rostral prefrontal cortex and temporal poles relative to both violent offenders with ASPD (group 2) and healthy controls, along with reductions in bilateral insula compared to violent/ASPD-only offenders.

These findings help to clarify the nature of brain structural anomalies in psychopathic offenders, while also raising questions about the pervasiveness of brain differences in individuals exhibiting violent behavior or ASPD per se. For psychopathic offenders, reduced volumes were evident in brain structures important for emotional processing as related to aversive learning, moral reasoning, and social interchange. While consistent with the notion of psychopathy as entailing deficits in affective sensitivity and social relatedness that give rise to behavioral deviance, this study did not report group comparisons for anterior brain regions involved more in

cognitive control than affective processing (e.g., dorsal–lateral regions of prefrontal cortex), and thus, it remains unclear whether frontal anomalies in this psychopathic offender group were limited to affective processing regions. The fact that no differences were found between violent/ASPD-only offenders and healthy controls could also reflect the choice of brain regions for analyses. It may be the case that regions important for cognitive control and performance monitoring would have shown up as anomalous in this offender subgroup. However, it is important to bear in mind that cognitive and affective impairments can reflect deviations in brain function not necessarily reflected in structural–anatomic anomalies.

*Functional imaging studies*: Three SPECT imaging studies that assessed neuronal activity at rest in aggressive psychiatric patients (Amen et al. 1996; Hirono et al. 2000) and impulsive violent offenders (Soderstrom et al. 2000) found evidence of reduced blood flow in both the prefrontal cortex and the temporal lobes (the left temporal lobe, specifically, in two of the three studies). In conjunction with reductions in these brain regions, Amen et al. (1996) reported evidence of *increased* activity in basal ganglia and subcortical (limbic) regions in their aggressive patient sample. One other SPECT study (Kuruoglu et al. 1996) reported reduced blood flow in frontal brain regions in alcoholic individuals with comorbid ASPD relative to non-alcoholic controls. SPECT was also used in some earlier studies to examine differences in neurotransmitter function in impulsively violent offenders. Studies of this kind revealed evidence of abnormal dopaminergic neurotransmission in the striatum and diminished serotonin transporter density in the midbrain (Tiihonen et al. 1995, 1997).

A number of studies have used PET imaging to test for functional brain abnormalities in violent individuals compared with controls. The majority have reported evidence of prefrontal dysfunction. Some of these studies focused on brain activity at rest (e.g., Volkow and Tancredi 1987), others on activity during tasks designed to activate the prefrontal cortex (e.g., Raine et al. 1994, 1997). Raine et al. (1998) subdivided violent participants from the Raine et al. (1997) study, consisting of 41 convicted murderers, into predatory (proactive) and affective (impulsive) subgroups based on the nature of their crimes, and found that prefrontal dysfunction was specific to the affective subgroup. Other studies have used PET imaging to investigate brain reactivity to drugs that activate the serotonergic system in aggressive and non-aggressive individuals (e.g., New et al. 2002; Siever et al. 1999). These studies have reported blunted reactivity to serotonin agonists (as evidenced by lower levels of glucose metabolism) among impulsive–aggressive patients compared with controls in regions of prefrontal cortex, particularly orbitofrontal and ventromedial regions. Other brain regions implicated with some consistency in these and other PET imaging studies include temporal cortex, ACC, and, to a lesser degree, hippocampus and amygdala.

Findings of abnormal ACC activity in conjunction with prefrontal anomalies are of interest in view of earlier described work demonstrating reduced ERN in impulsive–aggressive individuals and a recent study by Aharoni et al. (2013) reporting significant prediction of post-release recidivism among criminal offenders from degree of ACC activation in a laboratory inhibitory control task. With regard

to the amygdala and hippocampus, Raine et al. (1997) found evidence of abnormal asymmetry (i.e., decreased functioning on the left side and increased functioning on the right) in both these structures in murderers compared to controls. In a PET study of serotonin binding potential, Parsey et al. (2002) reported a significant negative relationship between reported lifetime aggression and binding in brain regions including the amygdala (but not hippocampus). George et al. (2004), in a study of domestic abusers with comorbid alcoholism, reported decreased correlations between glucose activity in the amygdala and glucose activity in various cortical structures compared with nonviolent controls. The authors postulated that these decreased associations reflected a lack of cortical input to the amygdala associated with increased sensitivity to environmental stressors among impulsively violent individuals.

Some studies of violent individuals have been also conducted using functional MRI. Raine et al. (2001) examined brain activation during a working memory task in small groups of community participants with histories of serious violent behavior and/or early abuse (*n*s = 4–5) relative to a healthy control group ($n = 9$). Compared to controls, violent individuals who had been abused as children showed reduced right hemisphere activation (particularly in right temporal regions), whereas abused individuals without violence showed lower left, but higher right activation of the superior temporal gyrus. In addition, both of these groups showed generally reduced cortical activation during task processing, particularly in the left hemisphere. The authors interpreted these findings as indicating a unique role of right hemisphere dysfunction, when combined with exposure to early abuse, in violent behavior. However, the findings of this study were quite tentative given the small sample sizes.

Several studies have used SPECT or fMRI to examine brain activation differences in individuals diagnosed as psychopathic using Hare's (2003) PCL-R, with a smaller number focusing on psychopathy as defined by self-report in adult samples or the informant-rated APSD in younger samples. Of studies that have focused on PCL-R psychopathy, most have examined brain reactivity in emotional processing paradigms entailing viewing of affective and neutral visual stimuli, aversive conditioning, anticipation of punishment to oneself or another person, processing of moral dilemmas entailing more or less emotion provocation, or performance of a cognitive task following manipulation of mood. Although no two of these PCL-R studies have used the same experimental task, some have used similar procedures. Both Intrator et al. (1997) and Kiehl et al. (2001) examined reactivity to emotional versus neutral words, within discrimination (word vs. nonword) and memory (encoding, rehearsal, recall) contexts, respectively. Using SPECT, Intrator et al. found *increased* bilateral activation for emotional versus neutral words in high-PCL-R participants within frontal–temporal cortex and adjacent subcortical regions. Kiehl et al. (2001) reported *decreased* activation in multiple a priori-defined limbic–subcortical regions, along with (in post hoc analyses) increased activation in right and left inferior lateral–frontal regions of cortex. Both Schneider et al. (2000) and Birbaumer et al. (2005) examined brain reactivity to CS+ and CS− stimuli in a differential aversive condition procedure, using foul odor and painful tactile

pressure stimuli as USs, respectively. The first of these studies reported *increased* activation in amygdala and dorsolateral prefrontal cortex regions to the CS+ versus the CS− for psychopathic participants during the latter part of acquisition, whereas the second reported *decreased* differential activation for high-PCL-R participants in left amygdala and ventromedial prefrontal cortex regions, as well as in right insula, rostral anterior cingulate, and secondary somatosensory cortex.

Two other studies by Müller et al. (2003, 2008b) used emotional and neutral picture stimuli, but in quite different ways. Müller et al. (2003) examined reactivity to pictures as primary stimuli and reported a complex pattern of differences for psychopathic as compared to non-psychopathic participants (i.e., decreased activation in some cortical and subcortical brain regions, but increased activation in others, for both pleasant and unpleasant pictures relative to neutral—with specific regions of decrease and increase for unpleasant pictures overlapping only partly with regions of decrease/increase for pleasant pictures). Müller et al. (2008b) used unpleasant picture viewing as a mood induction and found that high-PCL-R offenders, in contrast to low-PCL-R controls, exhibited no impact of this induction on responding in a subsequent "cognitive" reaction-time task, either behaviorally or in terms of activity in distinct brain regions (R medial and L inferior frontal gyri, R superior temporal gyrus) during this task.

Converging results across these differing emotion-processing studies include increased activation in regions of frontal/prefrontal cortex (Intrator et al. 1997; Schneider et al. 2000; Kiehl et al. 2001; Müller et al. 2003), increased activation in temporal–subcortical regions including the amygdala in some studies (Intrator et al. 1997; Müller et al. 2003; Schneider et al. 2000) along with decreased amygdala activation in others (Kiehl et al. 2001; Birbaumer et al. 2005), decreased activation in anterior cingulate (Kiehl et al. 2001; Müller et al. 2003; Birbaumer et al. 2005) and posterior cingulate, hippocampal, and frontal gyrus regions (Kiehl et al. 2001; Müller et al. 2003), and decreased activation in inferior frontal and superior temporal gyri (Kiehl et al. 2001; Müller et al. 2008b). An additional four fMRI studies of high-PCL participants used emotional processing tasks of other types. Findings from these studies that converge with results from the six above-mentioned studies include the following: (1) increased activation in regions of prefrontal cortex (dorsolateral, evaluated post hoc (Glenn et al. 2009b); dorsal and ventral medial, selectively in relation to higher PCL-R Factor 2 (Veit et al. 2010), and (2) decreased activation in anterior cingulate (Veit et al. 2010), posterior cingulate (Glenn et al. 2009a), amygdala (Glenn et al. 2009a; Veit et al. 2010), and right fusiform gyrus (Deeley et al. 2006; also reported by Müller et al. 2003). The two studies from among this overall group that included conditions entailing receipt of physical punishment (Birbaumer et al. 2005; Veit et al. 2010) also converged in finding decreased activation of the insula—a region implicated in pain perception.

However, some clear opposing findings are also evident across these different emotion-processing studies, including the following: (1) *decreased* activation of frontal/prefrontal cortex in some studies (i.e., ventromedial orbitofrontal cortex in Birbaumer et al. 2005; post-central gyrus in Deeley et al. (2005); right medial and left inferior frontal gyri in Müller et al. (2008b); medial frontal cortex, selectively in

relation to higher PCL-R Factor 1, in Glenn et al. (2009a) versus *increased* frontal/prefrontal activation in others (i.e., bilateral frontal/temporal cortex in Intrator et al. 1997; bilateral inferior lateral frontal cortex in Kiehl et al. 2001; bilateral precentral, bilateral inferior frontal, and right medial frontal gyri in Müller et al. 2003; right dorsolateral prefrontal cortex in Glenn et al. 2009b; dorsal and ventral medial prefrontal cortex, selectively in relation to PCL-R Factor 2, in Veit et al. 2010); and (2) *decreased* activation of the amygdala specifically in some studies (Kiehl et al. 2001; Birbaumer et al. 2005; Glenn et al. 2009a; Veit et al. 2010) versus *increased* amygdala activation in others (Müller et al. 2003; Schneider et al. 2000).

Other fMRI studies have investigated college or community adults varying in levels of psychopathy as assessed by self-report inventories (PPInv, four studies; TriPM, one study). All of the PPInv studies examined reactivity in affective processing or provocation tasks (i.e., affective picture viewing; affective face discrimination; anticipation of monetary reward; Prisoner's Dilemma). One study by Harenski et al. (2009) examined brain reactivity to unpleasant pictures, including depictions of moral dilemmas, under conditions of simple viewing and instructed emotion suppression in relation to scores on the PPInv as a whole and its Coldheartedness subscale. During simple viewing of moral-violation scenes, participants with high overall PPInv scores showed *decreased* activation in medial prefrontal cortex (Birbaumer et al. 2005; Müller et al. 2008b; Glenn et al. 2009a), and those specifically high in PPInv Coldheartedness showed decreased activation of the amygdala. Additionally, in the instructed suppression condition, high overall PPInv scorers showed *increased* activation in specific subdivisions of prefrontal cortex reported to be hypoactive in a number of PCL-R/emotion-processing studies (Intrator et al. 1997; Glenn et al. 2009b; Kiehl et al. 2001; Müller et al. 2003; Veit et al. 2010).

The other three PPInv studies evaluated effects for the inventory's distinctive fearless dominance and impulsive antisociality factors. Two of these studies (Gordon et al. 2004; Rilling et al. 2007) found higher scores on PPInv fearless dominance to be associated with differential activation in certain brain regions. However, no overlap was evident between effects observed by Gordon et al. (i.e., decreased activation in right amygdala, medial prefrontal cortex, right inferior temporal cortex, and increased activation in visual cortex and right dorsolateral prefrontal cortex) and the single effect reported by Rilling et al. (i.e., decreased activation in rostral anterior cingulate cortex). Additionally, Gordon et al. (but not Rilling et al.) reported evidence of *increased* activation in the right amygdala for participants classified as high versus low on PPInv impulsive antisociality. The other study that presented results separately for the two PPInv factors (Buckholtz et al. 2010) focused primarily on reactivity in the nucleus accumbens and found effects exclusively for PPInv impulsive antisociality—with higher scorers showing increased dopamine release in the accumbens both during anticipation of monetary reward and following administration of a dopamine agonist (amphetamine).

One other fMRI study tested for brain reactivity differences in an economic decision-making task as a function of overall scores on the TriPM (Vieira et al. in press). The major finding was that high TriPM scorers exhibited a different pattern

of brain response when rejecting unfair offers, entailing enhanced activation of ventromedial prefrontal cortex relative to dorsolateral prefrontal cortex, compared to low TriPM scorers. The authors' interpretation was that economic decision making may be more strongly driven by frustration than perceived fairness in high psychopathic individuals. These findings are interesting in light of other recent work by Drislane et al. (2014b) demonstrating distinct subgroups among high overall scorers on the TriPM—namely, a classically low-neurotic, high-bold ("primary") subtype and a high-neurotic, high-disinhibited ("secondary") subtype. This work raises intriguing questions about the representation of these distinct variants in the Vieira et al. study and the contribution of one versus the other to reported differences in brain activation.

A further set of fMRI studies has focused on psychopathy in children or adolescents as indexed by the APSD (Frick and Hare 2001). Two of these studies used affective face processing procedures, and two examined brain reactivity in reward/punishment learning paradigms. The first of the two face processing studies (Marsh et al. 2008) compared young adolescent participants meeting criteria for psychopathy on both the APSD and the youth version of the PCL-R with two other age-matched groups: (1) participants who met criteria for attention-deficit hyperactivity disorder (ADHD) but scored low on APSD callous–unemotional features and (2) a non-disorder ("healthy comparison") group. Relative to these comparison groups, psychopathic participants showed decreased right amygdala activation for fearful versus neutral faces, along with decreased covariation of activity between the right amygdala and interconnected structures including ventromedial prefrontal cortex, anterior and posterior cingulate gyrus, insula, and inferior temporal/fusiform gyrus. Using a very similar task with younger participants, and employing somewhat different selection criteria for psychopathy (i.e., ASPD ratings in conjunction with ratings on a separate measure of conduct problems) and a single non-clinical control group, Jones et al. (2009) replicated Marsh et al.'s finding of decreased amygdala activation during processing of fearful versus neutral faces and also reported a concomitant reduction in activity of the anterior cingulate cortex. The latter of these findings coincides with results from a number of PCL-R/imaging studies (Kiehl et al. 2001; Müller et al. 2003; Birbaumer et al. 2005; Veit et al. 2010) and one of four PPInv/imaging studies (Rilling et al. 2007).

The other two studies that focused on psychopathy in young participants used the same dual-diagnostic criterion (ASPD + PCL:YV) employed by Marsh et al. (2008), but examined brain reactivity in reward/punishment learning tasks. Finger et al. (2008) used a probabilistic reversal-learning task and reported increased activation in relation to punished reversal errors in bilateral medial frontal gyrus and right caudate regions in high-psychopathy participants as compared to ADHD and healthy comparison groups. Within the high-psychopathy group, scores on the callous–unemotional factor of the APSD selectively predicted degree of enhanced activation for punished errors. Finger et al. (2011) compared brain reactivity during a passive avoidance learning task in psychopathic (APSD + PCL:YV) youth and health controls (no ADHD comparison group was included). Relative to controls, psychopathic youth showed decreased reactivity in right orbitofrontal cortex and

caudate regions to earlier (as compared to later) occurrences of reinforced outcomes in the task, along with decreased reactivity in orbitofrontal cortex for correct rewarded response trials overall. A main effect of group was also evident for particular brain regions across the task as a whole, reflecting generally decreased activation for the psychopathic group in regions including the amygdala, caudate, and insula, and regions characterized by the authors as components of an "attention network" (i.e., prefrontal and parietal cortex).

## 4 Summary, Implications, and Future Directions

A number of consistent findings have emerged from psychophysiological studies of aggression and aggressive individuals. One is the finding of low resting HR, which has been interpreted as reflecting low dispositional arousal associated with tendencies toward impulsive stimulation seeking (Raine 1993, 2002; Ortiz and Raine 2004). However, this interpretation remains speculative, as no research to date has directly assessed the functional role of low cardiac arousal in the disinhibited behavior of antisocial–aggressive individuals. Two findings of related interest are enhanced EEG slow-wave activity in antisocial–aggressive individuals and reduced P300 brain response in individuals with externalizing problems more broadly. Reduced P300 response has also been reported specifically in relation to Factor 2 of psychopathy, whether indexed by the PCL-R (Venables and Patrick 2014) or the PPInv (Carlson et al. 2009). Enhanced EEG slow wave, like low resting HR, has been theorized to reflect low dispositional arousal that motivates stimulation seeking (Eysenck 1967; Zuckerman 1979). Differing explanations have been proposed for the finding of reduced P300 response. One that fits with findings of low resting HR and enhanced EEG slow wave is that anticipatory and preparatory activities are reduced in such individuals, resulting in a more stimulus-driven processing style (Malone et al. 2002; Taylor et al. 1999).

In contrast with these findings, other research has demonstrated *enhanced* phasic reactivity to stressful or aversive stimuli in hostile, aggressive, and abusive individuals—including enhanced cardiac and skin conductance reactivity to stressors, poor regulation of autonomic activity during anticipation of aversive events, and reduced cardiac vagal tone. Furthermore, some evidence exists to indicate that this pattern of heightened reactivity to aversive cues or events, like reduced P300 brain response, may be generally characteristic of individuals with impulse control problems, rather than specific to impulsive aggressive individuals (Taylor et al. 1999). Although the finding of enhanced reactivity to phasic stressors might seem inconsistent with data indicating low resting activation levels, the hypothesis that externalizing proneness (including proclivities toward impulsive aggression) entails a reactive, stimulus-driven processing style provides a framework for interpreting this overall configuration of results. From this perspective, high externalizing individuals are more reactive to immediate stressors or challenges because they anticipate and prepare for them less effectively (cf. Davidson et al. 2000).

These findings for impulsive aggression, and externalizing conditions more broadly, are clearly at odds with findings for psychopathy as defined by differing inventories. Adult psychopathic offenders do not show reliable differences in resting autonomic activity levels or P300 brain response (Raine 1993), but do show consistent reductions in phasic reactivity to aversive cues, including diminished SC response (cf. Hare 1978; Arnett 1997) and startle reflex potentiation (cf. Patrick 1994, 2007). The explanation for this divergence in findings almost certainly lies in the distinction between the affective–interpersonal versus the antisocial deviance features of psychopathy: It is the latter features that reflect heightened externalizing tendencies, including aggression and impulsiveness (Patrick 2007; Patrick et al. 2005). However, EEG/ERP and brain imaging studies have only recently begun to investigate effects for these two components of psychopathy separately. This is a key issue that should continue to be systematically addressed in future research.

However, it is important to keep in mind when considering findings from studies of these types that differing instruments for psychopathy index affective–interpersonal and impulsive–antisocial components of psychopathy differently. For example, whereas the two factors of the PCL-R are correlated and overlap in coverage of callous–aggressive tendencies (Patrick et al. 2009; Venables and Patrick 2012), the PPInv's two factors are uncorrelated, with PPInv fearless dominance indexing bold–fearless tendencies and PPInv impulsive–antisociality indexing disinhibition and to a secondary degree callous aggressiveness or meanness—and the PPInv's Coldheartedness scale indexing elements of meanness not captured by PPInv impulsive antisociality (Drislane et al. 2014a; Hall et al. 2014; Sellbom and Phillips 2013). Additionally, child and adult symptoms of ASPD reflect separable aggressive and non-normative/rule-breaking factors (Kendler et al. 2012, 2013; Tackett et al. 2003, 2005) that differentially reflect meanness versus disinhibition (Venables and Patrick 2012)—consistent with evidence from research demonstrating separable callous–unemotional and impulsive/conduct problem factors to child psychopathy. In light of this growing body of evidence, it seems likely that greater precision can be obtained in identifying reliable physiological correlates of psychopathy and related diagnostic conditions by routinely assessing distinct boldness, meanness, and disinhibition facets of these conditions in research studies. As an example of this, considerable progress has been made in indexing robust, replicable brain correlates of the disinhibition facet of psychopathy that exhibit correlations with one another, and thus can be combined to form composite brain-based indices of disinhibition or externalizing proneness (Nelson et al. 2011; Patrick et al. 2012). Through work of this kind, it will be possible in future brain electrophysiology and neuroimaging studies to characterize individuals along distinct dimensions of psychopathy or antisociality through combined use of physiological and clinical or psychometric measures (Patrick et al. 2013b). Phenotypes operationalized in this way would be more likely to exhibit consistent, meaningful biological correlates than phenotypes operationalized exclusively through diagnostic ratings or self-report (Patrick et al. 2012, 2013b).

Related to this, it will be important in future research to systematically examine alternative forms of aggression associated with differing underlying motives (e.g., proactive–instrumental versus reactive–impulsive) in relation to these two psychopathy factors in order to clarify relations with neurobiological measures. In particular, it is the impulsive–reactive subtype that appears to be most related to externalizing proneness and to impairments in brain systems that govern emotion regulation. It will also be valuable in future studies to include multiple measures of physiological response (peripheral–autonomic along with electrocortical; EEG together with structural or functional neuroimaging) so that findings for different measures can be directly compared within the same task procedures (cf. Nelson et al. 2011; Patrick et al. 2012, 2013b).

As a final point, it is important to note that most published psychophysiological studies of aggressive individuals to date (including neuroimaging studies) have focused either on differences in brain structure or differences in physiological activity at rest or in simple stimulus tasks. A pressing need exists for studies aimed at elucidating differences in online cognitive and affective processing with functional relevance to aggression—including cortical psychophysiology studies that capitalize on the fine-grained temporal and frequency information afforded by EEG/ERP, and functional neuroimaging studies that capitalize on the fine-grained spatial information provided by MRI. Along these lines, key questions for future research include the following: (1) What are the distinctive functional roles of brain regions that have been implicated in electrocortical and neuroimaging studies of aggression and how do these regions interact to achieve regulatory control over emotional states? Basic cognitive and affective neuroscience research is needed to elucidate this issue. (2) What specific impairments in the functioning of these brain systems predispose individuals toward aggressive behavior? To address this question, more EEG/ERP and functional neuroimaging studies are needed that examine online processing and brain reactivity within aggression-relevant task procedures, such as interpersonal provocation paradigms. (3) Do different types of brain dysfunction underlie impulsive–reactive and callous–proactive manifestations of aggressive behavior? The work of Raine et al. (1997), Marsh et al. (2008), and others suggests that these manifestations of aggression may reflect separate neuropathologies. Thus, an important challenge for future research will be to delineate the nature of processing impairments or deviations that underlie impulsive aggression associated with externalizing conditions compared with more callous–instrumental forms of aggression associated with psychopathic personality.

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## References

Aharoni E, Vincent GM, Harenski CL, Calhoun VD, Sinnott-Armstrong W Gazzaniga MS, Kiehl KA (2013) Neuroprediction of future arrest. In: Proceedings of the New York Academy of Sciences

Amen DG, Stubblefield M, Carmichael B, Thisted R (1996) Brain SPECT findings and aggressiveness. Ann Clin Psych 8:129–137

American Psychiatric Association (1980) Diagnostic and statistical manual of mental disorders, 3rd edn. Author, Washington, DC

American Psychiatric Association (2000) Diagnostic and statistical manual of mental disorders, 4th edn. Author, Washington, DC

American Psychiatric Association (2013) Diagnostic and statistical manual of mental disorders, 5th edn. Author, Washington, DC

Andershed H, Kerr M, Stattin H, Levander S (2002) Psychopathic traits in non-referred youths: initial test of a new assessment tool. In: Blaauw E, Philippa JM, Ferenschild KCMP, van Lodensteijn B (eds) Psychopaths: current international perspectives. Elsevier, The Hague, pp 131–158

Anderson JL, Sellbom M, Wygant DB, Salekin RJ, Krueger RF (in press) Examining the associations between DSM-5 Section III antisocial personality disorder traits and psychopathy in community and university samples. J Pers Disord

Arnett PA (1997) Autonomic responsivity in psychopaths: a critical review and theoretical proposal. Clin Psychol Rev 17:903–936

Babcock JC, Green CE, Webb SA, Graham KH (2004) A second failure to replicate the Gottman et al. (1995) typology of men who abuse intimate partners…and possible reasons why. J Family Psychol 18:396–400

Barratt ES, Stanford MS, Kent TA, Felthous AR (1997) Neuropsychological and cognitive psychophysiological substrates of impulsive aggression. Biol Psychiatry 41:1045–1061

Baskin-Sommers AR, Newman JP, Sathasivam N, Curtin JJ (2011) Evaluating the generalizability of a fear deficit in psychopathic African American offenders. J Abnorm Psychol 120:71–78

Bauer LO, O'Connor S, Hesselbrock VM (1994) Frontal P300 decrements in antisocial personality disorder. Alcohol Clin Exp Res 18:1300–1305

Beauchaine TP, Katkin ES, Strassberg Z, Snarr J (2001) Disinhibitory psychopathology in male adolescents: discriminating conduct disorder from attention-deficit/hyperactivity disorder through concurrent assessment of multiple autonomic states. J Abnorm Psychol 110:610–624

Benning SD, Patrick CJ, Hicks BM, Blonigen DM, Krueger RF (2003) Factor structure of the psychopathic personality inventory: validity and implications for clinical assessment. Psychol Assess 15:340–350

Benning SD, Patrick CJ, Blonigen DM, Hicks BM, Iacono WG (2005a) Estimating facets of psychopathy from normal personality traits: a step toward community-epidemiological investigations. Assessment 12:3–18

Benning SD, Patrick CJ, Iacono WG (2005b) Psychopathy, startle blink modulation, and electrodermal reactivity in twin men. Psychophysiology 42:753–762

Berkowitz L (1990) On the formation and regulation of anger and aggression: a cognitive-neoassociationistic analysis. Am Psychol 45:494–503

Birbaumer N, Veit R, Lotze M, Erb M, Hermann C, Grodd W et al (2005) Deficient fear conditioning in psychopathy: a functional magnetic resonance imaging study. Arch Gen Psychiatry 62:799–805

Blake PY, Pincus JH, Buckner C (1995) Neurological abnormalities in murderers. Neurology 45:1641–1647

Boccardi M, Ganzola R, Rossi R, Sabattoli F, Laakso MP, Repo-Tiihonen E et al (2010) Abnormal hippocampal shape in offenders with psychopathy. Hum Brain Mapp 31:438–447

Bradley MM (2009) Natural selective attention: orienting and emotion. Psychophysiology 46:1–11

Branchey MH, Buydens-Branchey L, Lieber CS (1988) P3 in alcoholics with disordered regulation of aggression. Psychiatry Res 25:49–58

Brazil IA, de Bruijn ER, Bulten BH, von Borries AK, van Lankveld JJ, Buitelaar JK et al (2009) Early and late components of error monitoring in violent offenders with psychopathy. Biol Psychiatry 65:137–143

Buckholtz JW, Treadway MT, Cowan RL, Woodward ND, Benning SD, Li R et al (2010) Mesolimbic dopamine reward system hypersensitivity in individuals with psychopathic traits. Nat Neurosci 13:419–421

Burt SA (2009) Are there meaningful etiological differences within antisocial behavior? Results of a meta-analysis. Clin Psychol Rev 29:163–178

Carlson SR, Thái S, McLaron ME (2009) Visual P3 amplitude and self-reported psychopathic personality traits: frontal reduction is associated with self-centered impulsivity. Psychophysiology 46:100–113

Cleckley H (1976) The mask of sanity, 5th edn. Mosby, St. Louis, Mo. (Original edition published in 1941)

Craig MC, Catani M, Deeley Q, Latham R, Daly E, Kanaan R et al (2009) Altered connections on the road to psychopathy. Mol Psychiatry 14:946–953

Davidson RJ, Putnam KM, Larson CL (2000) Dysfunction in the neural circuitry of emotion regulation—a possible prelude to violence. Science 289:591–594

Deeley Q, Daly E, Surguladze S, Tunstall N, Mezey G, Beer D et al (2006) Facial emotion processing in criminal psychopathy. Preliminary functional magnetic resonance imaging study. Br J Psychiatry 189:533–539

Dikman ZV, Allen JJ (2000) Error monitoring during reward and avoidance learning in high- and low-socialized individuals. Psychophysiology 37:43–54

Dindo L, Fowles DC (2011) Dual temperamental risk factors for psychopathic personality: evidence from self-report and skin conductance. J Pers Soc Psychol 100:556–566

Dolan MC, Deakin JFW, Roberts N, Anderson IM (2002) Quantitative frontal and temporal structural MRI studies in personality-disordered offenders and control subjects. Psychiatry Res Neuroimaging 116:133–149

Donchin E, Coles MGH (1988) Is the P300 component a manifestation of context updating? Behav Brain Sci 11:355–372

Drislane LE, Patrick CJ, Arsal G (2014a) Clarifying the content coverage of differing psychopathy inventories through reference to the Triarchic Psychopathy Measure. Psychol Assess 26:350–362

Drislane LE, Patrick CJ, Sourander A, Sillanmäki L, Aggen SH, Elonheimo H, Parkkola K, Multimäki P, Kendler KS (2014b) Distinct variants of extreme psychopathic individuals in society at large: evidence from a population-based sample. Pers Disord Theory Res Treat 5:154–163

Dvorak-Bertsch JD, Curtin JJ, Rubinstein TJ, Newman JP (2009) Psychopathic traits moderate the interaction between cognitive and affective processing. Psychophysiology 46:913–921

El-Sheikh M, Harger J, Whitson SM (2001) Exposure to interparental conflict and children's adjustment and physical health: the moderating role of vagal tone. Child Dev 72:1617–1636

Eriksen B, Eriksen C (1974) Effects of noise letters upon the identification of a target letter in a non-search task. Percept Psychophysics 16:143–149

Eysenck HJ (1967) The biological basis of personality. Charles C. Thomas, Springfield

Finger EC, Marsh AA, Mitchell DGV, Reid ME, Sims C, Budhani S et al (2008) Abnormal ventromedial prefrontal cortex function in children with psychopathic traits during reversal learning. Arch Gen Psychiatry 65:586–594

Finger EC, Marsh AA, Blair KS, Reid ME, Sims C, Ng P et al (2011) Disrupted reinforcement signaling in the orbitofrontal cortex and caudate in youths with conduct disorder or oppositional defiant disorder and a high level of psychopathic traits. Am J Psychiatry 168:152–162

Forth AE, Kosson DS, Hare RD (2003) The psychopathy checklist: youth version manual, 2nd edn. Multi-Health Systems, Toronto (1st edition published in 1996)

Frick PJ, Hare RD (2001) Antisocial process screening device. Multi Health Systems, Toronto
Frick PJ, Marsee MA (2006) Psychopathy and developmental pathways to antisocial behavior in youth. In: Patrick CJ (ed) Handbook of psychopathy. Guilford Press, New York, pp 353–374
Frick PJ, White S (2008) The importance of callous-unemotional traits for developmental models of aggressive and antisocial behavior. J Child Psychol Psychiatry 49:359–375
Frick PJ, Lahey BB, Loeber R, Stouthamer-Loeber M, Green S, Hart EL, Christ MAG (1991) Oppositional defiant disorder and conduct disorder in boys: patterns of behavioral covariation. J Clin Child Psychol 20:202–208
Gallo LC, Smith TW, Kircher JC (2000) Cardiovascular and electrodermal responses to support and provocation: Interpersonal methods in the study of psychophysiological reactivity. Psychophysiology 37:289–301
Gao Y, Raine A (2009) P3 event-related potential impairments in antisocial and psychopathic individuals: A meta-analysis. Biol Psychol 82:199–210
George DT, Rawlings RR, Williams WA, Phillips MJ, Fong G, Kerich M et al (2004) A select group of perpetrators of domestic violence: evidence of decreased metabolism in the right hypothalamus and reduced relationships between cortical/subcortical brain structures in position emission tomography. Psychiatry Res Neuroimaging 130:11–25
Gerstle JE, Mathias CW, Stanford MS (1998) Auditory P300 and self-reported impulsive aggression. Prog Neuropsychopharmacol Biol Psychiatry 22:575–583
Glenn AL, Raine A, Schug RA (2009a) The neural correlates of moral decision-making in psychopathy. Mol Psychiatry 14:5–6
Glenn AL, Raine A, Schug RA, Young L, Hauser M (2009b) Increased DLPFC activity during moral decision-making in psychopathy. Mol Psychiatry 14:909–911
Glenn AL, Raine A, Yaralian PS, Yang Y (2010a) Increased volume of the striatum in psychopathic individuals. Biol Psychiatry 67:52–58
Glenn A, Yang Y, Raine A, Colletti P (2010b) No volumetric differences in the anterior cingulate cortex of psychopathic individuals. Psychiatry Res Neuroimaging 183:140–143
Gordon HL, Baird AA, End A (2004) Functional differences among those high and low on a trait measure of psychopathy. Biol Psychiatry 56:516–521
Gottman JM, Jacobson NS, Rushe RH, Shortt JW, Babcock J et al (1995) The relationship between heart rate reactivity, emotionally aggressive behavior, and generaln violence in batterers. J Fam Psychol 9:227–248
Gregory S, Ffytche D, Simmons A, Kumari V, Howard M, Hodgins S, Blackwood N (2012) The antisocial brain: psychopathy matters. Arch Gen Psychiatry 69:962–972
Hall JR, Bernat EM, Patrick CJ (2007) Externalizing psychopathology and the error-related negativity. Psychol Sci 18:326–333
Hall JR, Drislane LE, Murano M, Patrick CJ, Lilienfeld SO, Poythress NG (2014) Development and validation of Triarchic construct scales from the psychopathic personality inventory. Psychol Assess 26:447–461
Hansen AL, Johnsen BH, Thornton D, Waage L, Thayer JF (2007) Facets of psychopathy, heart rate variability and cognitive function. J Pers Disord 21:568–582
Hare RD (1978) Electrodermal and cardiovascular correlates of psychopathy. In: Hare RD, Schalling D (eds) Psychopathic behavior: approaches to research. Wiley, Chichester, pp 107–143
Hare RD (1980) A research scale for the assessment of psychopathy in criminal populations. Pers Individ Differ 1:111–119
Hare RD (2003) Manual for the hare psychopathy checklist-revised, 2nd edn. Multi-Health Systems, Toronto
Hare RD, Neumann CS (2009) Psychopathy: assessment and forensic implications. Can J Psychiatry 54:791–802
Harenski CL, Kim S, Hamann S (2009) Neuroticism and psychopathic traits predict brain activity during moral and non-moral emotion regulation. Cogn Affect Behav Neurosci 9:1–15
Hicks BM, Markon KE, Patrick CJ, Krueger RF, Newman JP (2004) Identifying psychopathy subtypes based on personality structure. Psychol Assess 16:276–288

Hicks BM, Bernat EM, Malone SM, Iacono WG, Patrick CJ, Krueger RF, McGue M (2007) Genes mediate the association between P300 amplitude and externalizing disorders. Psychophysiology 44:98–105

Hirono N, Mega MS, Dinov ID, Mishkin F, Cummings JL (2000) Left fronto-temporal hypoperfusion in associated with aggression in patients with dementia. Arch Neurol 57:861–866

Hokanson JE (1970) Psychophysiological evaluation of the catharsis hypothesis. In: Megargee EI, Hokanson JE (eds) The dynamics of aggression. Harper-Collins, New York, pp 74–86

Hubbard JA, Smithmyer CM, Ramsden SR, Parker EH, Flanagan KD, Dearing KF, Relyea N, Simons RF (2002) Observational, physiological, and self-report measures of children's anger: Relations to reactive versus proactive aggression. Child Dev 73:1101–1118

Iacono WG, Carlson SR, Malone SM, McGue M (2002) P3 event-related potential amplitude and risk for disinhibitory disorders in adolescent boys. Arch Gen Psychiatry 59:750–757

Intrator J, Hare R, Stritzke P, Brichtswein K (1997) A brain imaging (single photon emission computerized tomography) study of semantic and affective processing in psychopaths. Biol Psychiatry 42:96–103

Jones AP, Laurens KR, Herba CM, Barker GJ, Viding E (2009) Amygdala hypoactivity to fearful faces in boys with conduct problems and callous-unemotional traits. Am J Psychiatry 166:95–102

Karpman B (1941) On the need of separating psychopathy into two distinct clinical types: the symptomatic and the idiopathic. J Crim Psychopathol 3:112–137

Kendler KS, Aggen SH, Patrick CJ (2012) A multivariate twin study of the DSM-IV criteria for antisocial personality disorder. Biol Psychiatry 71:247–253

Kendler KS, Aggen SH, Patrick CJ (2013) Familial influences on conduct disorder criteria in males reflect two genetic factors and one shared environmental factor: a population-based twin study. JAMA Psychiatry 70:78–86

Kennealy P, Skeem J, Walters G, Camp J (2010) Do core interpersonal and affective traits of PCL-R psychopathy interact with antisocial behavior and disinhibition to predict violence? Psychol Assess 22:569–580

Kiehl KA, Smith AM, Hare RD, Mendrek A, Forster BB, Brink J et al (2001) Limbic abnormalities in affective processing by criminal psychopaths as revealed by functional magnetic resonance imaging. Biol Psychiatry 50:677–684

Kramer UM, Kopyciok RPJ, Richter S, Rodriguez-Fornells A, Münte TF (2011) The role of executive functions in the control of aggressive behavior. Front Psychol 2:152. doi:10.3389/fpsyg.2011.00152

Kramer MD, Patrick CJ, Krueger RF, Gasperi M (2012) Delineating physiologic defensive reactivity in the domain of self-report: phenotypic and etiologic structure of dispositional fear. Psychol Med 42:1305–1320

Krueger RF (1999) The structure of common mental disorders. Arch Gen Psychiatry 56:921–926

Krueger RF, Hicks B, Patrick CJ, Carlson S, Iacono WG, McGue M (2002) Etiologic connections among substance dependence, antisocial behavior, and personality: modeling the externalizing spectrum. J Abnorm Psychol 111:411–424

Krueger RF, Markon KE, Patrick CJ, Benning SD, Kramer M (2007) Linking antisocial behavior, substance use, and personality: an integrative quantitative model of the adult externalizing spectrum. J Abnorm Psychol 116:645–666

Kuruoglu AC, Arikan Z, Vural G, Karatas M (1996) Single photon emission computerised tomography in chronic alcoholism: antisocial personality disorder may be associated with decreased frontal perfusion. Br J Psychiatry 169:348–354

Laakso MP, Vaurio O, Savolainen L, Repo E, Soininen H, Aronen HJ, Tiihonen J (2000) A volumetric MRI study of the hippocampus in type 1 and 2 alcoholism. Behav Brain Res 109:177–186

Laakso MP, Vaurio O, Koivisto E, Savolainen L, Eronen M, Aronen HJ (2001) Psychopathy and the posterior hippocampus. Behav Brain Res 118:187–193

Laakso MP, Gunning-Dixon F, Vaurio O, Repo-Tiihonen E, Soininen H, Tiihonen J (2002) Prefrontal volumes in habitually violent subjects with antisocial personality disorder and type 2 alcoholism. Psychiatry Res Neuroimaging 114:95–102

Lang PJ, Bradley MM, Cuthbert BN (1990) Emotion, attention, and the startle reflex. Psychol Rev 97:377–398

LeDoux JE (1995) Emotion: clues from the brain. Ann Rev Psychol 46:209–215

Levenson MR, Kiehl KA, Fitzpatrick CM (1995) Assessing psychopathic attributes in a noninstitutional population. J Pers Soc Psychol 68:151–158

Lilienfeld SO, Andrews BP (1996) Development and preliminary validation of a self report measure of psychopathic personality traits in noncriminal populations. J Pers Assess 66:488–524

Lilienfeld SO, Widows MR (2005) Psychopathic personality inventory—revised professional manual. Psychological Assessment Resources, Odessa

Lorber MF (2004) Psychophysiology of aggression, psychopathy, and conduct problems: a meta-analysis. Psychol Bull 130:531–552

Lykken DT (1957) A study of anxiety in the sociopathic personality. J Abnorm Clin Psychol 55:6–10

Lykken DT (1995) The antisocial personalities. Erlbaum, Hillsdale

Lynam DR (1997) Pursuing the psychopath: capturing the fledgling psychopath in a nomological net. J Abnorm Psychol 106:425–438

Malone SM, Bernat E, Patrick CJ, Iacono WG (2002) P300 and prestimulus EEG power: relationship to externalizing psychopathology in adolescent males. Psychophysiology 39:S54

Marsh AA, Finger EC, Mitchell DG, Reid ME, Sims C, Kosson DS et al (2008) Reduced amygdala response to fearful expressions in children and adolescents with callous-unemotional traits and disruptive behavior disorders. Am J Psychiatry 165:712–720

Meehan JC, Holtzworth-Munroe A, Herron K (2001) Maritally violent men's heart rate reactivity to martial interactions: a failure to replicate the Gottman et al. (1995) typology. J Family Psychol 15:394–408

Mezzacappa E, Tremblay RE, Kindlon D, Saul JP, Arseneault L, Seguin J et al (1997) Anxiety, antisocial behavior and heart rate regulation in adolescent males. J Child Psychol Psychiatry 38:457–469

Müller JL, Sommer M, Wagner V, Lange K, Taschler H, Roder CH et al (2003) Abnormalities in emotion processing within cortical and subcortical regions in criminal psychopaths: evidence from a functional magnetic resonance imaging study using pictures with emotional content. Biol Psychiatry 54:152–162

Müller JL, Ganssbauer S, Sommer M, Dohnel K, Weber T, Schmidt-Wilcke T et al (2008a) Gray matter changes in right superior temporal gyrus in criminal psychopaths. Evidence from voxel-based morphometry. Psychiatry Res 163:213–222

Müller JL, Sommer M, Dohnel K, Weber T, Schmidt-Wilcke T, Hajak G (2008b) Disturbed prefrontal and temporal brain function during emotion and cognition interaction in criminal psychopathy. Behav Sci Law 26:131–150

Munro GE, Dywan J, Harris GT, McKee S, Unsal A, Segalowitz SJ (2007) ERN varies with degree of psychopathy in an emotion discrimination task. Biol Psychol 76:31–42

Narayan VM, Narr KL, Kumari V, Woods RP, Thompson PM, Toga AW et al (2007) Regional cortical thinning in subjects with violent antisocial personality disorder or schizophrenia. Am J Psychiatry 164:1418–1427

Nelson LD, Patrick CJ, Bernat EM (2011) Operationalizing proneness to externalizing psychopathology as a multivariate psychophysiological phenotype. Psychophysiology 48:64–72

New AS, Hazlett EA, Buchsbaum MS, Goodman M, Reynolds et al (2002) Blunted prefrontal cortical superscript 1-sup-8 fluorodeoxyglucose positron emission tomography response to meta-chlorophenylpiperazine in impulsive aggression. Arch Gen Psychiatry 59:621–629

Newman JP, Curtin JJ, Bertsch JD, Baskin-Sommers A (2010) Attention moderates the fearlessness of psychopathic offenders. Biol Psychiatry 67:66–70

Ortiz J, Raine A (2004) Heart rate level and antisocial behavior in children and adolescents: a meta-analysis. J Am Acad Child Adolesc Psychiatry 43:154–162
Pailing PE, Segalowitz SJ (2004) The error-related negativity as a state and trait measure: motivation, personality, and ERPs in response to errors. Psychophysiology 41:84–95
Parsey RV, Oquendo MA, Simpson NR, Ogden RT, Van Heertum R, Arango V, Mann JJ (2002) Effects of sex, age, and aggressive traits in man on brain serotonin 5-HT-sub(1A ) receptor binding potential measured by PET using [C-11]WAY-100635. Brain Res 954:173–182
Patrick CJ (1994) Emotion and psychopathy: startling new insights. Psychophysiology 31:319–330
Patrick CJ (1995) Emotion and temperament in psychopathy. Clin Sci 5–8
Patrick CJ (2007) Getting to the heart of psychopathy. In: Hervé HF, Yuille JC (eds) Psychopathy: theory, research, and social implications. Erlbaum, Hillsdale, pp 207–252
Patrick CJ (2010) Operationalizing the Triarchic conceptualization of psychopathy: Preliminary description of brief scales for assessment of boldness, meanness, and disinhibition. Unpublished test manual, Florida State University, Tallahassee, FL. Test is available on-line at: https://www.phenxtoolkit.org/index.php?pageLink=browse.protocoldetails&id=121601
Patrick CJ, Bernat EM (2009) Neurobiology of psychopathy: a two-process theory. In: Berntson GG, Cacioppo JT (eds) Handbook of neuroscience for the behavioral sciences. Wiley, New York, pp 1110–1131
Patrick CJ, Bradley MM, Lang PJ (1993) Emotion in the criminal psychopath: startle reflex modulation. J Abnorm Psychol 102:82–92
Patrick CJ, Hicks BM, Krueger RF, Lang AR (2005) Relations between psychopathy facets and externalizing in a criminal offender sample. J Pers Disord 19:339–356
Patrick CJ, Bernat EM, Malone SM, Iacono WG, Krueger RF, McGue M (2006) P300 amplitude as an indicator of externalizing in adolescent males. Psychophysiology 43:84–92
Patrick CJ, Hicks BM, Nichol PE, Krueger RF (2007) A bifactor approach to modeling the structure of the psychopathy checklist-revised. J Pers Disord 21:118–141
Patrick CJ, Fowles DC, Krueger RF (2009) Triarchic conceptualization of psychopathy: developmental origins of disinhibition, boldness, and meanness. Dev Psychopathol 21:913–938
Patrick CJ, Durbin CE, Moser JS (2012) Conceptualizing proneness to antisocial deviance in neurobehavioral terms. Dev Psychopathol 24:1047–1071
Patrick CJ, Kramer MD, Krueger RF, Markon KE (2013a) Optimizing efficiency of psychopathology assessment through quantitative modeling: development of a brief form of the externalizing spectrum inventory. Psychol Assess 25:1332–1348
Patrick CJ, Venables NC, Yancey JR, Hicks BM, Nelson LD, Kramer MD (2013b) A construct-network approach to bridging diagnostic and physiological domains: application to assessment of externalizing psychopathology. J Abnorm Psychol 122:902–916
Peters ML, Godaert GLR, Ballieux RE, Heijnen CJ (2003) Moderation of physiological stress responses by personality traits and daily hassles: less flexibility of immune system responses. Biol Psychol 65:21–48
Polich J (2007) Updating the P300: an integrative theory of P3a and P3b. Clin Neurophysiol 118:2128–2148
Polich J, Pollock VE, Bloom FE (1994) Meta-analysis of P300 amplitude from males at risk for alcoholism. Psychol Bull 115:55–73
Porter S, Woodworth M (2006) Psychopathy and aggression. In: Patrick CJ (ed) Handbook of psychopathy. Guilford Press, New York, pp 481–494
Raine A (1993) The psychopathology of crime. Academic Press, San Diego
Raine A (2002) Biosocial bases of antisocial and violent behavior: a review. J Abnorm Child Psychol 30:311–326
Raine A, Venables PH, Williams M (1990) Relationships between N1, P300, and CNV recorded at age 15 and criminal behavior at age 24. Psychophysiology 27:567–575
Raine A, Buchsbaum MS, Stanley J, Lottenberg S, Abel L, Stoddard J (1994) Selective reductions in pre-frontal glucose metabolism in murderers. Biol Psychiatry 36:365–373

Raine A, Buchsbaum M, LaCasse L (1997) Brain abnormalities in murderers indicated by positron emission tomography. Biol Psychiatry 42:495–508

Raine A, Meloy JR, Bihrle S, Stoddard J, LaCasse L, Buchsbaum MS (1998) Reduced prefrontal and increased subcortical brain functioning assessed using positron emission tomography in predatory and affective murderers. Behav Sci Law 16:319–332

Raine A, Lencz T, Bihrle S, LaCasse L, Colletti P (2000) Reduced prefrontal gray matter volume and reduced autonomic activity in antisocial personality disorder. Arch Gen Psychiatry 57:119–127

Raine A, Park S, Lencz T, Bihrle S, LaCasse L, Widom CS et al (2001) Reduced right hemisphere activation in severely abused violent offenders during a working memory task: an fMRI study. Aggressive Behav 27:111–129

Raine A, Lencz T, Taylor K, Hellige JB, Bihrle S, Lacasse L et al (2003) Corpus callosum abnormalities in psychopathic antisocial individuals. Arch Gen Psychiatry 60:1134–1142

Raine A, Ishikawa SS, Arce E, Lencz T, Knuth KH, Bihrle S, LaCasse L, Colletti P (2004) Hippocampal structural asymmetry in unsuccessful psychopaths. Biological Psychiatry 55: 185–191

Rilling JK, Glenn AL, Jairam MR, Pagnoni G, Goldsmith DR, Elfenbein HA, Lilienfeld SO (2007) Neural correlates of social cooperation and non-cooperation as a function of psychopathy. Biol Psychiatry 61:1260–1271

Scarpa A, Raine A (1997) Psychophysiology of anger and violent behavior. Psychiatric Clin North Am 20:375–394

Schneider F, Habel U, Kessler C, Posse S, Grodd W, Muller-Gartner H (2000) Functional imaging of conditioned aversive emotional responses in antisocial personality disorder. Neuropsychobiology 42:192–201

Sellbom M, Phillips TR (2013) An examination of the triarchic conceptualization of psychopathy in incarcerated and non-incarcerated samples. J Abnorm Psychol 122:208–214

Siever LJ, Buchsbaum MS, New AS, Spiegel-Cohen J, Wei T, Hazlett EA et al (1999) d, l-Fenfluramine response in impulsive personality disorder assessed with [18F]flurodeoxyglucose positron emission tomography. Neuropsychopharmacology 20:413–423

Skeem JL, Johansson P, Andershed H, Kerr M, Eno Louden J (2007) Two subtypes of psychopathic violent offenders that parallel primary and secondary variants. J Abnorm Psychol 116:395–409

Smith TW, Gallo LC (1999) Hostility and cardiovascular reactivity during marital interaction. Psychosom Med 61:436–445

Soderstrom H, Tullberg M, Wikkelsoe C, Ekholm S, Forsman A (2000) Reduced regional cerebral blood flow in non-psychotic violent offenders. Psychiatry Res Neuroimaging 98:29–41

Stanford MS, Houston RJ, Villemarette-Pittman NR, Greve KW (2003) Premeditated aggression: clinical assessment and cognitive psychophysiology. Pers Individ Differ 34:773–781

Strickland CM, Drislane LE, Lucy MD, Krueger RF, Patrick CJ (2013) Characterizing psychopathy using DSM-5 personality traits. Assessment 20:327–338

Suls J, Wan CK (1993) The relationship between trait hostility and cardiovascular reactivity: a quantitative review and analysis. Psychophysiology 30:615–626

Tackett JL, Krueger RF, Sawyer MG, Graetz BW (2003) Subfactors of DSM-IV conduct disorder: Evidence and connections with syndromes from the child behavior checklist. J Abnorm Child Psychol 31:647–654

Tackett JL, Krueger RF, Iacono WG, McGue M (2005) Symptom-based subfactors of DSM-defined conduct disorder: evidence for etiologic distinctions. J Abnorm Psychol 114:483–487

Taylor J, Carlson SR, Iacono WG, Lykken DT, McGue M (1999) Individual differences in electrodermal responsivity to predictable aversive stimuli and substance dependence. Psychophysiology 36:193–198

Tiihonen J, Kuikka J, Bergstrom K, Hakola P, Karhu J, Ryynanen OP, Fohr J (1995) Altered striatal dopamine re-uptake site densities in habitually violent and non-violent alcoholics. Nat Med 1:654–657

Tiihonen J, Kuikka JT, Bergstrom KA, Karhu J, Viinamaki H, Lehtonen J et al (1997) Single-photon emission tomography imaging of monoamine transporters in impulsive violent behaviour. Eur J Nucl Med Mol Imaging 24:1253–1260

Tiihonen J, Rossi R, Laakso MP, Hodgins S, Testa C, Perez J et al (2008) Brain anatomy of persistent violent offenders: more rather than less. Psychiatry Res Neuroimaging 163:201–212

Tonkonogy JM (1991) Violence and temporal lobe lesion: head CT and MRI data. J Neuropsychiatry Clin Neurosci 3:189–196

Vaidyanathan U, Patrick CJ, Bernat EM (2009) Startle reflex potentiation during aversive picture viewing as an index of trait fear. Psychophysiology 46:75–85

Vaidyanathan U, Hall JR, Patrick CJ, Bernat EM (2011) Clarifying the role of defensive reactivity deficits in psychopathy and antisocial personality using startle reflex methodology. J Abnorm Psychol 120:253–258

van Elst LT, Woermann FG, Lemieux L, Thompson PJ, Trimble MR (2000) Affective aggression in patients with temporal lobe epilepsy: a quantitative MRI study of the amygdala. Brain 123:234–243

van Elst LT, Hesslinger B, Thiel T, Geiger E, Haegele K, Lemieux L et al (2003) Frontolimbic brain abnormalities in patients with borderline personality disorder: a volumetric magnetic resonance imaging study. Biol Psychiatry 54:163–171

Vanman EJ, Mejia VY, Dawson ME, Schell AM, Raine A (2003) Modification of the startle reflex in a community sample: do one or two dimensions of psychopathy underlie emotional processing? Pers Individ Differ 35:2007–2021

Veit R, Lotze M, Sewing S, Missenhardt H, Gaber T, Birbaumer N (2010) Aberrant social and cerebral responding in a competitive reaction time paradigm in criminal psychopaths. NeuroImage 49:3365–3372

Venables NC, Patrick CJ (2012) Validating factors of the externalizing spectrum inventory in a criminal offender sample: relations with disinhibitory psychopathology, personality, and psychopathic features. Psychol Assess 24:88–100

Venables NC, Patrick CJ (2014) P3 brain response amplitude in criminal psychopathy: distinct relations with impulsive-antisocial versus affective-interpersonal features. Psychophysiology 51:427–436

Venables NC, Hall JR, Patrick CJ (2014) Differentiating psychopathy from antisocial personality disorder: a Triarchic model perspective. Psychol Med 44:1005–1013

Verona E, Curtin JJ (2006) Gender differences in the negative affective priming of aggression. Emotion 6:115–124

Verona E, Patrick CJ, Joiner TE (2001) Psychopathy, antisocial personality, and suicide risk. J Abnorm Psychol 110:462–470

Verona E, Patrick CJ, Lang AR (2002) A direct assessment of the role of state and trait negative emotion in aggressive behavior. J Abnorm Psychol 111:249–258

Vieira JB, Almeida PR, Ferreira-Santos F, Barbosa F, Marques-Teixeira J, Marsh AA (in press) Distinct neural activation patterns underlie economic decisions in high and low psychopathy scorers. Soc Cogn Affect Neurosci

Volavka J (1990) Aggression, electroencephalography, and evoked potentials: a critical review. Neuropsychiatry Neuropsychology Behav Neurol 3:249–259

Volkow ND, Tancredi L (1987) Neural substrates of violent behaviour: a preliminary study with positron emission tomography. Br J Psychiatry 151:668–673

von Borries AK, Brazil IA, Bulten BH, Buitelaar JK, Verkes RJ, de Bruijn ER (2010) Neural correlates of error-related learning deficits in individuals with psychopathy. Psychol Med 40:1559–1568

Wall TD, Wygant DB, Sellbom M (in press) Boldness explains a key difference between psychopathy and antisocial personality disorder. Psychiatry Psychol Law

Williams KM, Paulhus DL, Hare RD (2007) Capturing the four-factor structure of psychopathy in college students via self-report. J Pers Assess 88:205–219

Woermann FG, Van Elst LT, Koepp MJ, Free SL, Thompson PJ, Trimble MR, Duncan JS (2000) Reduction of frontal neocortical grey matter associated with affective aggression in patients with temporal lobe epilepsy: an objective voxel by voxel analysis of automatically segmented MRI. J Neurol Neurosurg Psychiatry 68:162–169

Wong MTH, Lumsden J, Fenton GW, Fenwick PBC (1994) Electroencephalography, computed tomography and violence ratings of male patients in a maximum-security mental hospital. Acta Psychiatr Scand 90:97–101

Yancey JR, Venables NC, Hicks BM, Patrick CJ (2013) Evidence for a heritable brain basis to deviance-promoting deficits in self-control. J Crim Justice 41:309–317

Yang Y, Raine A, Lencz T, Bihrle S, LaCasse L, Colletti P (2005) Volume reduction in prefrontal gray matter in unsuccessful criminal psychopaths. Biol Psychiatry 57:1103–1108

Yang Y, Raine A, Narr KL, Colletti P, Toga AW (2009) Localization of deformations within the amygdala in individuals with psychopathy. Arch Gen Psychiatry 66:986–994

Young SE, Stallings MC, Corley RP, Krauter KS, Hewitt JK (2000) Genetic and environmental influences on behavioral disinhibition. Am J Med Gen (Neuropsychiatric Genetics) 96:684–695

Zuckerman M (1979) Sensation seeking: beyond the optimal level of arousal. Erlbaum, Hillsdale