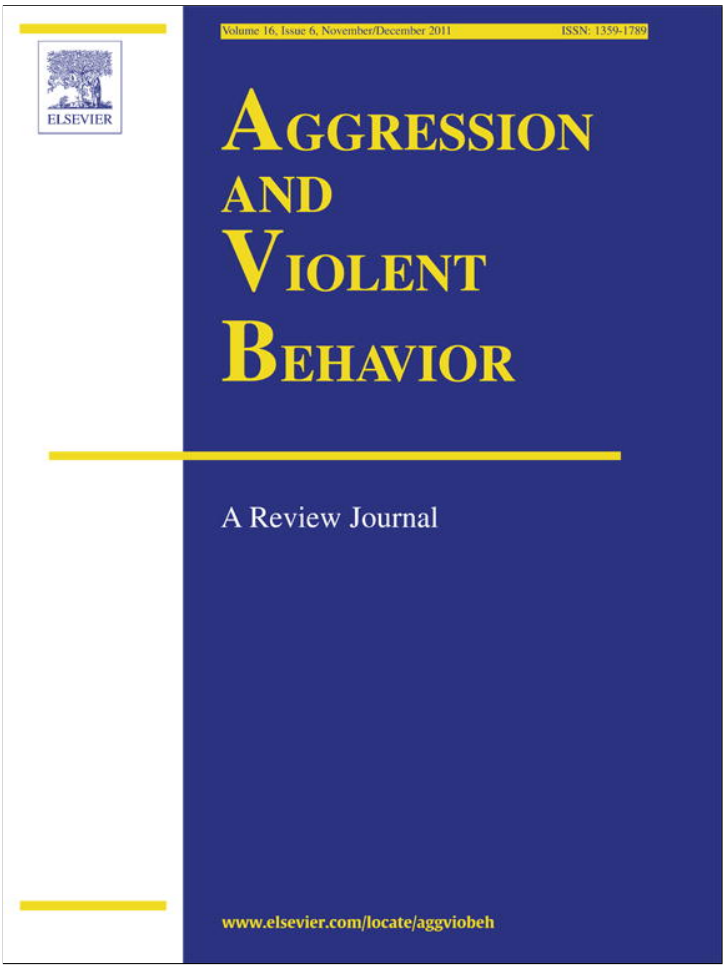

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Psychopathy, reactive aggression, and precarious proclamations: A review of behavioral, cognitive, and biological research☆

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ABSTRACT

Psychopathic personality (psychopathy) is associated with a heightened risk for physical aggression, although the nature of this link remains unclear. Despite widespread claims that psychopathy is associated with reactive aggression, the evidence for this assertion is mixed. We provide a comprehensive review of behavioral, cognitive, and biological research on the relation between psychopathy and aggression, and conclude that although psychopathy is clearly associated with instrumental aggression, its association with reactive aggression is not robust. In fact, at least some research points to a potential protective role of psychopathy against reactive aggression. We conclude that future research must clarify the differential implications of the separable components of the broad psychopathy construct before the relations between psychopathy and physical aggression can be adequately understood.

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Contents

1. Introduction

Hervey Cleckley (1941) identified lack of remorse or shame and general poverty of affect as core features of the psychopathic personality (psychopathy). Although general correlates of antisocial and deviant behavior typify common criminals, Cleckley included

☆ The findings and conclusions in this report are those of the author(s) and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

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other members of society, such as doctors, lawyers, and businessmen, under the rubric of psychopathy. According to him, psychopathy was not restricted to individuals identified by the legal system. McCord and McCord (1964) described the psychopath as "an asocial, aggressive, highly impulsive person, who feels little or no guilt, and represents a major danger to society" (p. 3). However, they warned that equating deviant behavior with psychopathy was an oversimplification and an insufficient marker of the construct. Schneider (1958), much like Cleckley, believed that psychopathy was prevalent among the general public regardless of criminality and even speculated that some psychopaths might make highly successful community leaders (for a review of adaptive manifestations of psychopathy, see Hall & Benning, 2006).

Despite these early theorists' proclamations, most studies have focused on forensic populations, fueling recent debate over the centrality of criminal behavior to the construct of psychopathy (Cooke & Michie, 2001; Hare & Neumann, 2010; Skeem & Cooke, 2010a, 2010b). Nevertheless, it is generally agreed that criminality is certainly a correlate, and may even be a consequence of the construct (Cooke & Michie, 2001; Cooke, Michie, Hart, & Clark, 2004; but see Hart & Hare, 1997, who do not see criminality as a consequence of psychopathy). Moreover, psychopathy is a potent predictor of criminal behavior, especially violence (Cooke & Michie, 2001; Hart & Hare, 1997; Porter & Woodworth, 2006). For example, although psychopaths represent only 1% of the general population and 20% of prison populations, they are responsible for nearly 50% of all violent crimes (Hare, 1993, 1996, 1999; Hare & McPherson, 1984) and are at heightened risk for violent recidivism (Hemphill, Hare, & Wong, 1998; Olver & Wong, 2006; Salekin, 2008). Psychopathy is related to some of the most severe, deleterious, and "cold-blooded" acts of violence (e.g., Porter, Woodworth, Earle, Drugge, & Boer, 2003). However, most notable among psychopaths' traits are perhaps their stunning lack of empathy, disregard for others' perspectives, and lack of remorse. These traits almost certainly distinguish the psychopath from common citizens as well as criminals and other aggressive individuals.

The literature suggests that psychopathy is distinctive in its risk for instrumental aggression (Porter & Woodworth, 2006; see "Defining aggression and violent behavior" below, for a discussion of instrumental and reactive aggression). Similarly, it is widely accepted that psychopathy is related to an increased risk of reactive aggression (e.g., Blair, 2004; Blair, Mitchell, & Blair, 2005; Cornell, Warren, Hawk, Stafford, Oram, & Pine, 1996; Harenski & Kiehl, 2010; Hart & Dempster, 1997): "*the data suggest that psychopathy is associated with increased risk of reactive aggression*" (Blair, 2010; p. 386); "*psychopathic individuals demonstrate reactive aggression in addition to instrumental aggression*" (Glenn & Raine, 2009; p. 253); "*laboratory-based research has suggested that psychopathy traits are broadly related to aggression including instrumental and reactive forms*" (Reidy, Zeichner, & Martinez, 2008; p. 320). However, existing data have proffered mixed results for this "truism" and suggest that the relation between psychopathy and reactive aggression may be weak at best. Indeed, Patrick (2001) argued that true psychopathy is associated with proactive (i.e., instrumental), but not reactive aggression. Surprisingly, despite an abundant literature on psychopathy and aggression, there is a relative dearth of literature addressing the relation between psychopathy and reactive aggression, *per se*. This is probably because reactive aggression, an arguably "less pathological" variant compared to instrumental aggression (Raine et al., 2006), is the most prevalent form of real-world aggression (Bushman & Anderson, 1998; Cornell et al., 1996). Consequently, it is logical to assume that individuals predisposed to perpetrating "cold-blooded" instrumental acts of violence (i.e., psychopaths) would also be as prone, if not more prone, than nonpsychopaths to committing this "normative" form of aggression: ostensibly, a type of aggression of which nearly anyone is capable under sufficient provocation (e.g., Berkowitz, 1988; Caprara, Renzi, Alcini, D'Imperio, & Travaglia, 1983). Additionally, much research fails to disaggregate forms of violence, in part, because pure forms may not exist (see below). Nevertheless, as we will see, the research that has directly addressed this question does not consistently support the association between psychopathy and reactive aggression. Moreover, there are theoretical and conceptual reasons to question this link.

The purpose of the present review is to clarify the association between psychopathy and physical aggression in its various manifestations. In particular, we highlight the dearth of consistent research on the relationship between psychopathy and reactive aggression, identify potential explanations for these equivocal findings, and propose new conceptual considerations and directions for research. We begin by reviewing extant definitions and debates regarding conceptualization and measurement in both the aggression and psychopathy literatures. We then lay out research on psychopathy and variants of violent behavior, beginning with the data that substantiates the risk for instrumental violence. Then, we review the equivocal findings on the association between psychopathy and reactive violence. We next review the literature on the cognitive and biological processes and substrates associated with psychopathy and reactive aggression. Finally, we propose ideas for future research and new considerations for conceptualizing the risk for aggression in psychopathy.

2. Defining aggression and violent behavior

Anderson and Bushman (2002) defined human aggression as "any behavior directed toward another individual that is carried out with the proximate intent to cause harm, and during which, the perpetrator must believe that the behavior will harm the target, and the target is motivated to avoid the behavior (p. 28)." They further defined violence as aggression that has extreme harm as its goal (e.g., physical injury or death). All violence is aggression, but not vice-versa, as can be seen in verbal aggression. For the purposes of our discussion, we use the terms aggression and violence interchangeably to refer to acts of physical aggression and violence, because most studies have assessed violence or assessed aggression as an analogue to violence.

To understand risk factors and develop interventions aimed at reducing and preventing violence, a wealth of research has attempted to understand aggression in terms of the motivation precipitating this behavior. The motivation-based distinction between types of aggression has consistently identified two relatively distinct forms: reactive (also referred to as hostile, angry, impulsive, or affective) and instrumental (also referred to as proactive). Reactive aggression arises in response to provocation (i.e., threat or frustration) that generally induces anger and ultimately triggers the aggressive event. This type of aggression is generally an impulsive, immediate reaction to an emotionally-laden stimulus, such as perceived insult, embarrassment, or imminent physical danger (Berkowitz, 1993). The aggressive act is initiated without a secondary goal beyond the aggressive act. A spouse who murders his/her partner upon discovering an affair or a youth who assaults a peer in response to being teased would be seen as engaging in reactive aggression. In contrast, instrumental aggression is a goal-driven behavior motivated by the attainment of an external reward or reinforcement other than the aggressive act (Berkowitz, 1993). A spouse who murders his/her partner to collect life insurance or an adolescent who assaults a peer to earn gang membership would be seen as engaging in instrumental violence.

This distinction between the two forms of aggression has been criticized as misrepresenting aggressive acts as pure when, in fact, most aggressive acts may reflect mixed motives (Bushman & Anderson, 2001). Further, some experts have argued that human behavior may be too complex to classify an aggressive act as being purely instrumental in nature (Dodge & Coie, 1987; Giancola, 1995). Nevertheless, abundant data support the existence of two relatively distinguishable forms of aggression in children (Kempes, Matthys, de

Vries, & van Engeland, 2005; Raine et al., 2006; Vitiello, Behar, Hunt, Stoff, & Riccuiti, 1990), adult humans (Chase, O'Leary, & Heyman, 2001; Raine et al., 1998), and nonhuman animals (Eichelman, 1992; Gregg & Siegel, 2001). Moreover, these disparate forms of aggression bear different correlates important for the identification of targets for future treatment, prevention, and research efforts (Connor, 2002; Vitiello & Stoff, 1997). In fact, Bushman and Anderson (2001) did not argue against existence of disparate forms of aggression; instead, they argued that the key factor distinguishing them is the ultimate goal of the act. "Thus, both robbery and physical assault are acts of aggression because both include intention to harm the victim at a proximate level. However, they typically differ in their ultimate goals with robbery serving primarily profit-based goals and assault serving primarily harm-based goals" (Anderson & Bushman, 2002; p. 29). Moreover, the fact that real-world behaviors rarely reflect pure manifestations of either variant of aggression does not undermine existence of these variants.

It is not our objective to renew the debate over the existence of aggression variants or the optimal method for classifying them. The noteworthy ambiguities in the literature aside, the extant findings reveal identifiable variants of aggression with disparate underlying biological, neurological, cognitive, social, and developmental contributions.

3. Conceptualizing and assessing psychopathy

The etiology of psychopathy, like that of virtually all psychological disorders, is largely unknown. Nevertheless, there have been several major theoretical models of the causes of psychopathy; we have drawn on these models periodically in our review. The (1) fearlessness model (Lykken, 1995; see also Fowles & Dindo, 2009) posits that psychopaths are characterized by an abnormally high fear threshold. In other words, they do not experience as much fear as do nonpsychopaths in anticipation of punishment. Such fearlessness, in turn, ostensibly gives rise to the other core features of the condition. For example, children who lack adequate fear will not react strongly to criticism from their parents and other significant adult figures, and hence will not internalize a strong conscience. The (2) low arousal model (Quay, 1965) proposes that psychopaths possess an abnormally low level of autonomic arousal. As a consequence, they experience chronic "stimulus hunger" and are prone to seeking out risky activities as a means of compensating for their boredom. The (3) response modulation model (Patterson & Newman, 1993) hypothesizes that psychopaths, once engaged in a dominant response set (such as the pursuit of a desired reward), develop psychological "blinders," becoming oblivious to extraneous stimuli, including (but not limited to) cues of punishment. As a result, they do not learn readily from their mistakes, especially when presented with competing reward and punishment contingencies. The (4) integrated emotion systems model (Blair, 2004) integrates earlier models and hypothesizes that dysfunction of the amygdala precludes the development of empathy. Consequently, psychopaths cannot be morally socialized and evince a propensity for socially deviant behavior. At this point, it is unclear which, if any, of these models best captures the causes of psychopathy; moreover, each model could be accurately explaining certain features of the condition, but not others.

The Psychopathy Checklist—Revised (PCL-R; Hare, 2003) and its derivatives, the Psychopathy Checklist: Screening Version (Hart, Cox, & Hare, 1995) and the Psychopathy Checklist: Youth Version (Forth, Kosson, & Hare, 2003), are the most widely used and best validated diagnostic measures of psychopathy (Kiehl, 2006; Skeem & Cooke, 2010a). These rating scales use a semistructured interview, review of file records, and collateral information (when possible) to assess traits and behaviors of the disorder. Hare's original conceptualization of psychopathy identified two moderately correlated factors (Hare, 1991; Harpur, Hakstian, & Hare, 1988). Factor 1 (F1), *Emotional Detachment*, includes emotional and interpersonal features, such as affective shallowness, absence of empathy, lack of remorse, lack of shame, superficial charm, manipulative style, grandiosity, and lying. Factor 2 (F2), *Social Deviance*, encompasses impulsivity, aggression, substance abuse, high sensation seeking, low socialization, proneness to boredom, irresponsibility, lack of concern or plans for the future, low motivation, and early life behavioral problems and delinquency (Hare, 1991, 2003). The two factors form a superordinate psychopathy factor. Although the PCL-R yields dimensional scores, it has most commonly been used to categorize individuals dichotomously as psychopaths or nonpsychopaths. This practice has potentially obfuscated associations between core features of psychopathy and external correlates because it fails to consider the dimensional nature of the construct and the disparate contributions of the individual factors.[1]

Initial versions of the PCL and the PCL-R were intended largely to operationalize Cleckley's 16-item description of the clinical psychopath (Hare, 1991). However, the PCL-R includes several features not identified by Cleckley (e.g., antisocial behavior) and omits others that he did identify (e.g., low anxiety; Cooke, Michie, & Hart, 2006). Hare's original two-factor conceptualization of psychopathy was largely accepted by researchers for many years (Hare, 1991; Harpur, Hare, & Hatskian, 1989). But debate about the factor structure of the PCL-R and its progeny has stimulated new models of this factor structure. Cooke and Michie (2001) proposed that the PCL-R's factor structure is better understood in terms of a three-factor model tapped by only 13 of the original 20 PCL-R items. They argued that the remaining seven items were psychometrically redundant and did not validly assess psychopathy. Six of these seven items reflected criminal behavior, leading them to argue that the three-factor model is more consistent with early theorists' conceptualizations, which did not identify criminality as a central component (e.g., Cleckley, 1941; Schneider, 1950). The three factors, (1) *Arrogant Deceitful Interpersonal Style*, (2) *Deficient Affective Experience*, and (3) *Impulsive Irresponsible Behavioral Style* were similar to those in the two-factor model in that they are hierarchical in nature, underpinning a superordinate psychopathy construct. Hare (2003) later proposed a four-facet model in which the original two factors each comprised two subfacets. F1 was split into the *Interpersonal* and *Affective* facets, whereas F2 was split into the *Lifestyle* and *Antisocial* facets. Despite the emergence of new structural models, the two-factor structure has been the most widely researched conceptual model of psychopathy thus far.

In addition to clinical measures, a growing literature supports the use of multiple self-report measures for the assessment of psychopathy traits in adults. The Self-Report Psychopathy Scale (SRP-III; Paulhus, Neumann, & Hare, in press) and the Levenson Self-Report Psychopathy Scale (LSRP; Levenson, Kiehl, & Fitzpatrick, 1995) are patterned after the PCL-R, with the former possessing the four-facet structure and the latter comprising two factors. The Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996) and its successor Psychopathic Personality Inventory—Revised (PPI-R; Lilienfeld & Widows, 2005), unlike other self-report measures, were constructed based on the full spectrum of traits proposed by early theorists (i.e., Cleckley, 1941/1976; Karpman, 1941, 1948; Lykken, 1957; McCord & McCord, 1964). Initially, factor analyses identified eight replicable factor scales; however, more recent research suggests that seven of the eight factors may reflect two largely orthogonal higher-order factors termed *Fearless Dominance* and *Impulsive*

[1] In our review, we address the role of the factors and the subfacets in relation to forms of aggression when they have been presented. However, much research has used a dichotomous classification of participants based solely on the total score. As such, we do not refer to the subcomponents of the psychopathy indices in these instances.

Antisociality (Benning, Patrick, Blonigen, Hicks, & Iacono, 2005; Benning, Patrick, Hicks, Blonigen, & Krueger, 2003; Lilienfeld & Widows, 2005; but see Neumann, Malterer, & Newman, 2008, for a competing factor structure of the PPI). The two factors are conceptually related, but not entirely analogous to, the two major factors of the PCL-R.

Whereas, the PCL:YV is used to assess psychopathy traits in adolescents ages 12–17, the Antisocial Process Screening Device (APSD; Frick & Hare, 2001) is a 20-item informant rating scale used to measure conduct and psychopathic traits in children ages 6–13. Trait indices are derived from parents, teachers, or a combination of ratings. This instrument was designed to be a downward extension of the Hare's PCL-R. Much like its parent measure, the APSD has proffered both a two-factor and a three-factor model (Frick, Bodin, & Barry, 2000; Frick, O'Brien, Wootton, & McBurnett, 1994); the latter is better supported by large-scale factor analyses (Fite, Greening, Stoppelbein, & Fabiano, 2009; Frick et al., 2000; Frick & Hare, 2001). Data generally suggest moderate correlations among psychopathy measures (e.g., Brinkley, Schmitt, Smith, & Newman, 2001; Edens, Skeem, Cruise, & Cauffman, 2001; Gaughn, Miller, Pryor, & Lynam, 2009; Poythress, Edens, & Lilienfeld, 1998; Reidy, Zeichner, & Foster, 2009; Reidy, Ziechner, & Seibert, 2011). These findings suggest that studies using different psychopathy measures must be interpreted with some caution; nevertheless, to the extent that findings replicate across studies based on different psychopathy measures, they should be regarded as especially robust.

4. Psychopathy and instrumental aggression

4.1. Adult forensic populations

The relationship between psychopathy and instrumental violence has been well substantiated. For example, Williamson, Hare, and Wong (1987) investigated elements of offenders' index offense (i.e., crime for which they were currently incarcerated). In their sample, 45.2% of the psychopaths committed violence motivated by material gain compared with 14.6% of nonpsychopaths.[2] Similarly, Woodworth and Porter (2002) and Porter and Woodworth (2007) examined the motivational elements of index homicides. Recognizing the potential for mixed motive (i.e., elements of instrumental and reactive) acts of violence, they created a rating continuum ranging from 'purely instrumental' to 'purely reactive.' In both studies, approximately 90% of the homicides perpetrated by psychopaths were 'purely' or 'primarily instrumental'. These authors further reported correlations for F1 that were significant and moderate in size, whereas correlations for F2 were negligble and nonsignificant. Dempster et al. (1996) similarly found that the positive correlations between psychopathy and ratings of instrumentality on index offenses were due solely to F1.

Rather than examining the index offense, Walsh, Swogger, and Kosson (2009) rated offenders' most violent historical act on degree of instrumentality. Regression analyses indicated that PCL-R total scores were associated with instrumentality after controlling for both IQ and history of violence. Further, facet level analyses revealed unique positive associations between the interpersonal and antisocial facets and ratings of instrumentality. Surprisingly, regressions also revealed a unique inverse relationship between instrumentality and affective facet scores.

Whereas the aforementioned studies looked at a single violent act, several research groups have investigated propensity for violence by examining the cumulative history of such behavior. Serin (1991) reported that among a sample of incarcerated men, psychopaths endorsed significantly greater histories of instrumental violence than violent nonpsychopaths. Cornell et al. (1996) identified criminal offenders as either instrumental or reactive aggressors based on their history of violence from official institutional records. In two separate samples, instrumental aggressors were significantly more psychopathic than reactive aggressors. However, there was a discrepancy between the samples when examining the factor contribution to group differences. In the first sample, instrumental offenders were differentiated from reactive offenders by F2, but not F1, of the PCL-R. In the second sample, instrumental and reactive offenders were significantly different on both F1 and F2 of the PCL-SV, and the effect size for F1 ($d = 0.95$) was larger than for F2 ($d = 0.66$). Chase et al. (2001) used a similar methodological approach to classify a group of partner-violent men. In their sample, instrumental aggressors were more frequently classified as psychopathic (17%) on the MCMI–II than were reactive aggressors (0%).

Cima and Raine (2009) administered the PPI and Reactive/Proactive Aggression Questionnaire (RPQ: Raine et al., 2006) to 121 male inmates in multiple prisons throughout the Netherlands. Using residualized scores for aggression, they found an association between the PPI total score and proactive aggression. In addition, the PPI's two largely orthogonal factors were equally related to proactive aggression ($r = .23$ and $r = .27$). However, as noted earlier, the two factors of the PPI are not directly analogous to those of the PCL-R.

4.2. Adult nonforensic populations

There is a much smaller of body of research on aggression and psychopathy in college samples and no studies that we could identify in community samples (in reference to this topic). Reidy, Zeichner, Miller, and Martinez (2007) randomly assigned collegiate men to an instrumental or reactive condition in a modified version of the Taylor Aggression Paradigm (TAP; Taylor, 1967). Participants assigned to the instrumental condition were informed that they would earn $1 for each trial they won and forfeit $1 for each trial lost. To reinforce the instrumental nature of the task, participants were told that they could punish their opponent by shocks after each trial, which could interfere with their opponent's performance, thereby helping the participant win money. Self-reported psychopathy scores on the LSRP predicted more physical aggression by electric shock ($r = .36$). Simultaneous regression analysis indicated that prediction of instrumental aggression was accounted for by F1 ($\beta = .39$; $p < .01$), but not F2 ($\beta = -.03$; *ns*).

4.3. Child and adolescent populations

In children and adolescents, Callous-Unemotional (CU) traits have been identified as a potential developmental marker of psychopathy (Frick, Cornell, Barry, Bodin, & Dane, 2003; Viding, Blair, Moffit, & Plomin, 2005) and identify a severe and chronic subgroup of antisocial youth (Frick et al., 2000; Frick & Ellis, 1999). In child and adolescent samples, CU traits (as endorsed by self-report, teacher-report, or caregiver-report) have repeatedly correlated positively with self-report measures of instrumental aggression (Fanti, Frick, & Georgiou, 2009; Fite, Stoppelbein, & Greening, 2009; Frick, Cornell, Barry, et al., 2003; Marsee & Frick, 2007; Munoz, Frick, Kimonis, & Aucoin, 2008; Raine et al., 2006).

Several research teams have adapted methods used with adults in the previously discussed research to assess the association of psychopathy to instrumental aggression in adolescent populations. Murrie, Cornell, Kaplan, McConville, and Levy-Elkon (2004) adapted the methods of Cornell et al. (1996) to identify juveniles as instrumental or reactive offenders. Total scores on the PCL:YV indicated a greater likelihood of being an instrumental offender ($r = .36$).[3] Similarly, Flight

[2] For the sake of brevity, we use the term psychopath(s) in our review of published studies to refer to those participants who were classified as such by the authors determined by a cut score on the PCL, PCL-R, or PCL-SV. When alternative measures were employed, we use the term psychopathy and/or reference the specific measure used.

[3] However, scores on the ASPD were not associated with the likelihood of being an instrumental offender.

and Forth (2007) classified adolescents as instrumental or reactive based on their history of violent offenses. They found that youthful offenders scoring high on the PCL:YV were more likely than low scorers to be classified as instrumental ($r = .59$). Moreover, psychopathy traits were positively and strongly correlated with the frequency of prior instrumental violence ($r = .69$). Simultaneous logistic regression indicated that F1 was the only significant predictor of instrumental violence.

Vitacco, Neumann, Caldwell, Leistico, and Van Rybroek (2006) created a dimensional coding scheme to assess instrumental violence based on Cornell et al. (1996). Using structural equation modeling with a group of juvenile offenders, they demonstrated that psychopathy, as measured by the PCL:YV, explained 20% of the variance in the instrumentality of the youths' violence. Like Flight and Forth (2007), these authors found that F1, specifically the interpersonal facet, was positively associated with instrumental violence. Additionally, the antisocial facet of F2 was negatively related to instrumental violence. Interestingly, when the antisocial facet was removed from the model, the interpersonal facet was no longer significantly associated with instrumental violence. These results suggest the potential presence of suppressor effects among the facets of psychopathy and forms of violence.

Stafford and Cornell (2003) asked case managers at a psychiatric hospital to rate adolescents on separate 4-point scales indicating their correspondence to a prototype of instrumental and reactive offenders. The prototypes were derived from earlier research with adult inmates (Cornell et al., 1996). Additionally, PCL-R scores were derived from separate raters who did not communicate any information to clinical staff members. Psychopathy total scores were significantly and positively correlated with ratings of similarity to instrumental aggression prototypes ($r = .49$). Moreover, these correlations were significant even after controlling for demographic variables, impulsivity, and conduct behaviors.

In sum, the data yield a rather consistent finding across forensic and nonforensic populations of adults, children, and adolescents: psychopathy is a risk factor for instrumental violence. Moreover, a general pattern seems to appear when the subfactors are considered: the traits of F1 (i.e., the affective and interpersonal) are often identified as solely responsible for this association.

5. Psychopathy and reactive aggression

5.1. Adult forensic populations

In one of the first studies to speak to the question of the relation between psychopathy and reactive aggression, Williamson et al. (1987) reviewed police reports detailing the offenses of 101 incarcerated men. When they examined the motives of their violent offense, they found that only 9.5% of the homicides perpetrated by psychopaths were in response to some provocation (i.e., revenge motivated) and only 2.4% were committed during emotional arousal (i.e., jealousy, rage, heated argument). Of note, nonpsychopaths committed significantly more homicides than psychopaths and did so during a period of heightened emotional arousal. In contrast, psychopaths committed their homicides without the emotional arousal common to reactive violence.

Woodworth and Porter (2002) created a Likert-type coding scale that allowed for the presence of mixed motives to classify homicides perpetrated by a sample of 125 incarcerated men. Homicides were classified as: (1) purely reactive, (2) primarily reactive, (3) primarily instrumental, or (4) purely instrumental. Of the homicides perpetrated by psychopaths, only 6.7% were purely reactive and none were primarily reactive. However, 33.3% of psychopaths' murders were primarily instrumental with elements of reactivity. Interestingly, 71.8% of nonpsychopaths' homicides were purely or primarily reactive, with another 10.3% being primarily instrumental with elements of reactivity. Nonpsychopaths committed significantly more homicides with evidence of some reactivity (82.1%) compared with psychopaths (40%). In a follow-up study, Porter and Woodworth (2007) utilized the same classification system with a sample of 50 incarcerated men. Only 11.1% of psychopaths committed homicides that were purely or primarily reactive compared with 57.9% of nonpsychopaths. The authors noted that "psychopaths had almost always perpetrated premeditated homicides (89% of the time) that did not contain any substantial reactive, impulsive component" (p. 103).

These studies with incarcerated adult psychopaths suggest a tenuous link between psychopathy and reactive violence, and potentially even a decreased risk of reactive violence. However, it is plausible that psychopaths in these studies also had a significant history of reactive violence. Because the authors did not have access to sufficient records of previous violent acts, they were able to assess only one severe act of violence (i.e., offenders' most recent murder). Psychopaths may have had a significantly greater history of reactive homicides (and other nonfatal violence) that went undetected by the legal system. Yet it seems implausible that unplanned reactive homicides would be less likely to be detected by the forensic system than would premeditated homicides.

Alternatively, psychopaths may commit more acts of reactive aggression than nonpsychopaths, but only perpetrate more severe acts such as murder under premeditated instrumental circumstances and with much less frequency. Woodworth and Porter (2002) proposed that psychopaths might display a "selective impulsivity" that reflects a decision *not* to inhibit aggressive behavior in less severe contexts. However, when the consequences of the violent act are more extreme (e.g., life in prison, death penalty), they are able to inhibit their impulsively violent inclinations. This would admittedly belie theory and data suggesting that psychopathy relates to a deficit in the processing of punishment relevant information (e.g., Blair et al., 2004; Budhami, Richell, & Blair, 2006; Lykken, 1957; Patrick, 1994). Additionally, prospective data challenge this theory of selective impulsivity. For example, Veit et al. (2010) used a modified Taylor Aggression Paradigm (TAP) to assess reactive aggression in a small sample of criminal psychopaths. Perhaps surprisingly, dimensional scores on F1 of the PCL-SV were strongly and inversely related to average intensity of reactive aggression ($r = -.67$) despite incrementally increasing provocation.[4] Relatedly, Dempster et al. (1996) found that F1 correlated negatively with the presence of provocation in the violent acts of psychiatric offenders, but correlated positively with ratings of instrumentality, planning, and goal-directedness. Importantly, F2 was associated with greater intoxication and less planning of these acts, suggesting a link between the antisocial trait spectrum and reactive aggression. These diverging relationships suggest possible suppressor effects between the two factors and reactive violence.

Researchers have argued that examining a single sample of behavior generally proffers less reliability and validity than a history of violent acts (Chase et al., 2001; Cornell et al., 1996). For example, Cornell et al. (1996) suggested that individuals who engage in instrumental violence (i.e., psychopaths) are also more likely than other individuals to have a history of reactive violence as well. Based on violence in institutional records, these authors classified incarcerated men as instrumental, reactive, or nonviolent offenders.[5] Instrumental offenders were those who had committed at least one act of instrumental violence. Nearly all instrumental offenders had committed acts of reactive aggression. Conversely, reactive offenders

[4] These authors also administered the LSRP to assess psychopathic traits and found a nearly identical association between F1 and mean aggression intensity ($r = -.68$). However, this correlation was due largely to the effect of a single outlier (R. Veit; personal communication, September 23, 2010).

[5] Only one study of Cornell et al. (1996) contained a group of nonviolent offenders; the second smaller sample contained only participants categorized as either instrumental or reactive offenders.

had committed *only* acts of reactive violence. Group comparisons indicated that reactive offenders and nonviolent offenders were less psychopathic than their instrumental counterparts.[6] However, these results do not allow for the determination of a relationship between psychopathy and reactive aggression. To identify an increased risk between the two, comparisons of the frequency (i.e., quantity) of reactively violent acts are necessary. It may be that instrumental offenders commit less overall reactive aggression than those who would be identified as reactive offenders. In fact, because aggression was rated based on a review of official records, we cannot verify that psychopaths committed more reactive aggression than those persons identified as nonviolent offenders, as logical as it seems. Rather, we can conclude only that psychopaths committed more violent acts for which they were caught.

5.2. Adult nonforensic populations

Falkenbach, Poythress, and Creevy (2008) adapted Cornell and colleagues' classification system for a group of collegiate men, and assessed psychopathy using the LSRP. Participants reported past incidents of aggression and were categorized as either reactive aggressors or combined (instrumental) aggressors based on a history of at least one instrumental act. Instrumental aggression was rare, but all participants who reported a history of instrumental aggression also endorsed a history of reactive aggression. Using cluster analytic procedures, the authors identified a "primary psychopathic subtype" that had the highest scores on F1, above average scores on F2, and near average scores on measures of behavioral inhibition, behavioral activation, and anxiety. In contrast, the "secondary psychopathic subtype" had average scores on F1 and the highest scores on F2, behavioral inhibition, behavioral activation, and anxiety. Individuals in the primary psychopathic cluster were more likely to be in the combined aggressor group, whereas those in the secondary psychopathic cluster were more likely to be in the reactive aggressor group.

Lotze, Veit, Anders, and Birbaumer (2007) split a sample of community participants into high and low psychopathy groups based on their F1 scores from the LSRP. Participants competed in a laboratory aggression paradigm that was a modified version of the TAP. The high F1 group showed significantly greater retaliatory aggression in response to incrementally increasing provocation. Notably, the authors found strikingly different results with the same paradigm in a sample of clinical psychopaths (Veit et al., 2010). In that sample, F1 was strongly and inversely related to reactive aggression. The reasons for this discrepancy are unclear but may suggest differential relationships in disparate samples (i.e., criminal vs. subcriminal psychopaths).

Using a similar paradigm to the one above, Reidy et al. (2007) found that psychopathy (measured with the LSRP) was strongly predictive of physical aggression toward a confederate ($r = .51$) in the reactive condition (i.e., no incentive to shock the opponent) of the TAP. However, psychopathy was equally predictive of physical aggression ($r = .36$) in the instrumental condition (i.e., monetary incentive to shock the opponent). Moreover, when controlling for covariance between the factors, F1 was the sole predictor of aggression in both conditions. This finding may suggest that high psychopathy individuals were engaging in aggression indiscriminately. That is, these men may have been aggressive not because of, but in spite of, provocation or incentive. Indeed, follow-up studies have shown that psychopathy traits, specifically F1, predict the commission of unprovoked aggression with no incentive (Reidy et al., 2008; Reidy et al., 2011). Moreover, using the modified TAP, Jones and Paulhus (2010) found, as did Reidy et al. (2007, 2008, 2011), that high psychopathy individuals were indiscriminately aggressive. In their sample, high psychopathy men engaged in aggression regardless of provocation and became more aggressive when provocation was added.

5.3. Child and adolescent populations

Stafford and Cornell (2003) administered PCL-R total to a group of inpatient adolescent boys and girls. Additionally, case managers rated the youths' use of reactive and instrumental aggression to correspond with ratings of a prototypical reactive offender and a prototypical instrumental offender. Total scores on the PCL-R indicated that psychopathy correlated with both prototypes of aggression. Ratings of the instrumental prototype were slightly stronger than for the reactive offender prototype ($r = .49$ vs. $r = .36$). However, the authors did not examine the individual predictive contribution of the psychopathy subfactors. In a related vein, using the APSD to measure psychopathy, Fanti et al. (2009) found that adolescents characterized by high levels of CU traits were more likely to exhibit combined forms of aggression rather than pure forms of either type.

Frick, Cornell, Barry, et al. (2003) administered the ASPD to a sample of children from grades 3, 4, 6, and 7. Participants were assessed again at a 1-year follow-up for aggression and self-reported delinquency. CU traits predicted increased reactive aggression in the cohort of 6th and 7th graders. However, the authors reported that the association between CU traits and reactive aggression was accounted for by differences in the initial level of conduct problems. Children high in CU traits at the initial assessment had higher rates of conduct problems than other children, and this difference accounted for the differential rates of reactive aggression at the follow-up assessment.

Barry et al. (2007) measured parent and teacher ratings of children's reactive aggression. CU traits were positively correlated with ratings of reactive aggression ($r = .55$). However, the other subscales of the APSD were more strongly correlated with reactive aggression (narcissism, $r = .81$; impulsivity, $r = .72$). Moreover, when all factors were entered into a simultaneous regression, the relation between CU traits and reactive aggression became negligible and nonsignificant ($\beta = .02$). Using the same measure and similar analytical procedures, Fite, Stoppelbein, and Greening (2009) found a nearly identical pattern. Narcissism ($\beta = .52$) and impulsivity ($\beta = .43$) were better predictors of reactive aggression than were CU traits ($\beta = .20$), which were not significantly associated with reactive aggression. Moreover, the adolescents' self-reports on the ASPD indicated that all indices were negatively but nonsignificantly associated with reactive aggression. Raine et al. (2006) found that maternal ratings of psychopathy on the Childhood Psychopathy Scale (Lynam, 1997) correlated with both forms of aggression on the RPQ. However, when they removed the shared variance from the two aggression scores, psychopathy was not associated with residualized reactive aggression.

Flight and Forth (2007) used a coding system similar to Cornell et al. (1996) to classify youthful offenders. Additionally, they rated the frequency with which youths had engaged in reactively violent behaviors. Correlations between scores on the PCL:YV and reactive aggression were significant and large ($r = .55$). Simultaneous logistic regressions revealed that the relation of F2, but not F1, was significant, increasing the odds (OR = 1.55) of reactive violence. In the four-facet model, only the antisocial factor was significant in increasing the odds (OR = 1.85).

Munoz et al. (2008), reported that CU traits were significantly correlated with a measure of reactive aggression ($r = .31$) in a group of juvenile adolescent males. Interestingly, CU traits were negatively correlated ($r = -.23$) with skin conductance response (SCR) to high provocation (i.e., verbal taunts from a peer). The SCRs to high or low provocation did not correlate significantly with aggression in any

[6] Reactive offenders were not more psychopathic than nonviolent offenders. In fact, reactive offenders were nearly three T scores lower than nonviolent offenders on all indices of psychopathy. These differences were nonsignificant, although group comparison of F1 was not conducted because the omnibus F test was not significant.

form. Marsee and Frick (2007) assessed psychopathy traits and aggression in a sample of detained female adolescents. Reactive aggression was uniquely associated with emotion dysregulation and perceived provocation, but not with CU traits.

In full, the data on psychopathy and reactive violence are much less consistent. In forensic populations the relationship appears to be the weakest. In fact, in the criminal populations the affective deficits of psychopathy (i.e., F1) suggest a potential protective effect against this form of violence. In nonforensic samples, where levels of psychopathy traits are likely less extreme, the relation to reactive violence is more consistent.

6. Cognition

Notably, psychopaths tend to describe their violence as provoked ("he started it") even when they were not. That is, although both psychopaths and nonpsychopaths exaggerate the degree of reactivity of their violence, psychopaths do so to a significantly greater degree (Porter & Woodworth, 2007). This finding may indicate that psychopaths are more likely to lie to exculpate themselves, or, alternatively, that they may be more likely to interpret ambiguous cues as provocative and/or attribute hostile intent in ambiguous situations. The link between psychopathy and hostile-attribution-bias (HAB; Nasby, Hayden, & dePaulo, 1979) is pertinent because HAB appears to be specific to reactive aggression and account for increased rates of violence in certain groups of offenders. For example, children and adolescents who exhibit HAB display high rates of reactive aggression with their peers and commit more violent crime (Dodge & Coie, 1987; Dodge, Price, Bachorowski, & Newman, 1990).

Several investigations have assessed the tendency of psychopaths to perceive provocation in ambiguous situations and to respond with violence. Serin (1991) presented vignettes depicting ambiguous provocative situations to a sample of incarcerated men, and assessed them for impulsivity, trait anger, and hostility, all of which are risk factors for reactive aggression (Bettencourt, Tally, Benjamin, & Valentine, 2006; Raine et al., 2006). Compared with nonpsychopaths, psychopaths endorsed significantly more impulsivity but did not endorse different levels of trait anger or hostility. They did endorse more anger in response to provocative scenarios, but they did not differ in the attibution of hostile intent or their intended responses to the provocation. Importantly, the veracity of psychopaths' reports must be interpreted with caution (e.g., Cooper & Yuille, 2007; Porter & Woodworth, 2007). Although they denied HAB and aggressive responses to provocation, they may have been more likely to engage in violent tactics in a real-life scenario. Notably, when Serin looked only at vignettes deemed to be "very provocative," psychopaths demonstrated a small propensity to attribute greater hostile intent ($r = .22$). In an attempt to extend this study, Vitale, Newman, Serin, and Bolt (2005) assessed the mediating effect of HAB on the relationship between psychopathy and violence in a male forensic population. They found an effect size for the relationship between psychopathy and HAB ($r = .20$) similar to that detected by Serin (1991); however, HAB did not mediate relationship of psychopathy to violent crime. In fact, the relationship between HAB and violent crime was negligible and nonsignificant ($r = .05$). Vitale and colleagues speculated that this negative finding might have been due to the lack of specificity in their measure of violence, which did not allow differentiation of reactive from instrumental violence.

Cale and Lilienfeld (2006) clarified this issue by parsing aggression that was in response to provocation from aggression with no apparent provocation. They assessed psychopathy, anger, and the tendency to interpret interpersonal exchanges as ego-threat (i.e., provocation) using two self-report measures in a sample of incarcerated men. Additionally, they assessed aggression in response to provocation using disciplinary reports and informant reports by the Department of Corrections staff members. Similar to Serin (1991), psychopathy correlated significantly and positively with self-reported tendencies to become angry in response to provocation ($r = .34$). Moreover, psychopathy was positively correlated with a tendency to assign more provocative intent to interpersonal interactions ($r = .43$). F2 but not F1 was responsible for the association with HAB ($r = .47$) and anger in response to provocation ($r = .51$), respectively. However, the relationship between both psychopathy factors and physical aggression in response to provocation was nonsignificant for both disciplinary reports and informant reports of aggression.

Frick et al. (2003) !assessed the relationship between CU traits and HAB in a community sample of third, fourth, sixth, and seventh grade students. HAB was associated with conduct problems, but only in boys and only in the absence of CU traits. In fact, children high on CU traits made significantly fewer hostile attributions than children low on CU traits. This finding is slightly different but consistent with the results of Cale and Lilienfeld (2006), who found no significant relationship between F1 (which is conceptually similar to CU traits) and HAB.

Collectively, studies suggest that psychopathy is not significantly related to the commission of reactive aggression despite a possible tendency to display HAB. Nevertheless, these findings must be interpreted with caution due to the reliance on psychopaths' self-reports, lack of specificity in criterion measures, and use of retrospective reports of violence.

7. Neurobiology

As noted earlier, many authors have argued that the unique feature of psychopathy is a deep-seated emotional deficit. The data we have reviewed suggest that abnormally frequent and intense use of instrumental aggression may additionally be a feature unique to psychopathy. The neuroscience of instrumental aggression is perhaps more complex than that of reactive aggression, largely because of the cognitive demands the process requires. Planning and execution of instrumental acts is a more complicated and temporally drawn-out process than the immediate reaction to a threat stimulus.

Blair et al. (2005; see also Blair, 2004, 2005, 2006a, 2006b; Blair et al., 2005; Crowe & Blair, 2008) have laid out in detail the Integrated Emotion System (IES); a model of the neurocognitive developmental processes that may engender instrumental violence. Blair et al. (2005) described a mechanism by which abnormal classical and operant conditioning lead to a selective increase in antisocial and instrumentally aggressive behaviors. They argued that because the amygdala is critically involved in the formation of connections between conditioned stimuli and unconditioned responses (classical conditioning), as well as between conditioned responses and reinforcement contingencies (operant conditioning), it is the major substrate for the development and maintenance of instrumental aggression. The ventromedial frontal, rostral insular, and rostral temporal cortex all have connections to the amygdala, especially its basolateral nucleus (Etkin, Egner, Peraza, Kandel, & Hirsch, 2006; Johnston, 1923; Kilpatrick & Cahill, 2003). These connections appear to be largely reciprocal, and according to the IES, form the core of the functional architecture responsible for instrumental aggression. People who evince psychopathy do so, in part, because their underactive amygdala fails to signal the full value of aversive stimuli, while operating more or less normally for rewarding stimuli. As such, according to Blair and his colleagues, the instinctively punishing distress cues on others' faces fail to dissuade children with this neural dysfunction from aggression. Instead, these children are reinforced for aggression and fail to learn the correct associations between the mood states of others and their behaviors. This process, in turn, disrupts normal socialization.

In contrast, reactive aggression occurs in response to an environmental trigger that provokes strong emotion. The provocation may represent an impending threat, which elicits fear. Consequently, the

evinced aggression would be a defensive act. Alternatively, the provocation may offend or frustrate individuals, inciting anger and motivating them to aggress in a retaliatory manner. Importantly, perceived presence of a provocative stimulus is sufficient to elicit a violent response even if it is not legitimate. The neural circuitry that mediates reactive aggression originates in the central nucleus of the amygdala and connects to the medial hypothalamus and dorsal periaqueductal gray via the stria terminalis. This circuitry is modulated by the anterior cingulate cortex (ACC), and regions of the frontal cortex, including the orbital (OFC), ventrolateral prefrontal cortex (vlPFC), and the medial frontal cortex (Blair, 2006a; Miller, Collins, & Kent, 2008).

Some have suggested that individuals who frequently demonstrate reactive aggression show pronounced deficits in frontocortical functioning. For example, Soderstrom, Tullberg, Wikkelsoe, Elkholm, and Forsman (2000) found that reactively violent offenders demonstrate diminished frontotemporal cerebral blood flow. Similarly, in a sample of people with personality disorders, Goyer et al. (1994) found that the frequency of reactive aggression correlated negatively with glucose metabolism in the lateral orbital frontal cortex. In healthy individuals, the OFC and associated regions of the frontal cortex work in unison with the ACC to inhibit anger and aggression in response to provocation. Consistent with this finding, patients with lesions to the OFC, medial frontal cortex, or ACC exhibit more acts of reactive aggression than do other patients (Blair, 2004; Foster, Hillbrand, & Silverstein, 1993).

In contrast, those who perpetrate homicides for instrumental reasons appear to demonstrate relatively intact prefrontal functioning (Raine et al., 1998). Similarly, psychopaths who, as previously discussed, tend to commit primarily instrumental homicides, show enhanced activity in the frontotemporal regions during affective tasks (Intrator et al., 1997; Kiehl et al., 2001; Muller et al., 2003). However, these tasks may lack precision in their ability to localize dysfunction, as other research suggests that neuroaffective deficits in psychopathy are more specific to the ventromedial PFC (vmPFC), OFC, and ACC (Blair, 2007; Blair et al., 2006; Kiehl, 2006; LaPierre, Braun, & Hodgins, 1995), which inhibit anger and response to provocation. Dysfunction of these frontal regulatory areas results in disinhibition of responding in threat circuitry, which predisposes individuals to reactive aggression.

As such, psychopaths should be at increased risk for reactive aggression due to a failure of the frontal regulatory systems to inhibit the threat response, leading to threat hypersensitivity. Yet most research indicates that psychopathy is related to a *hypo*sensitivity of response to threat provocation (Kiehl, 2006). For example, psychopaths demonstrate deficits in passive avoidance learning (Lykken, 1957; Newman & Schmitt, 1998) and aversive conditioning (Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002; Lykken, 1957) as well as diminished fear potentiated startle reflexes (Herpertz et al., 2001; Levenston, Patrick, Bradley, & Lang, 2000; Vanman, Mejia, Dawson, Schell, & Raine, 2003) and autonomic responses to threat (Lorber, 2004; Olgoff & Wong, 1990; Patrick, Cuthbert, & Lang, 1994; Patrick & Verona, 2007). In fact, Blair (2006b) noted that "Individuals with psychopathy are unlikely to display heightened reactive aggression because of overactive brainstem threat circuitry. Indeed, in line with the hypothesis of amygdala dysfunction, this population is less responsive to environmental threat rather than more responsive" (p. 300).

Instead, Blair (2004, 2005, 2006a, 2006b) suggested that purported heightened levels of reactive aggression in psychopathy are due to frustration caused by the inability to discontinue an unrewarded behavior (e.g., Mitchell, Colledge, Leonard, & Blair, 2002; Newman, Patterson, & Kosson, 1987). The OFC and vmPFC are responsible for coding the expectancy of reward and the identification of discrepancy between expected and actual reward. The vlPFC regions modulate response choices during a detected change in reinforcement contingency. As such, intact functioning of the OFC, vmPFC, and vlPFC is necessary for reward discrepancy calculations and consequent determination of frustration (Blair, 2004, 2006a). Blair (2010) reported that persons susceptible to frustration show greater activity in these regions during frustrating events (e.g., failure to obtain monetary reward; Siegrist et al., 2005). Nevertheless, as previously discussed, a growing body of literature identifies dysfunction of these areas in psychopaths. This deficit thereby impedes their ability to identify reward expectation violations (Blair, 2006a). From this perspective, highly psychopathic individuals would be *less* likely than other individuals to experience frustration and, in turn, be less likely to engage in reactive aggression. In line with this argument, a recent study indicates that lesions of the vmPFC preclude experience of regret during a gambling task (Camille, 2005). Moreover, it seems unlikely that the majority of reactive aggression committed by violent offenders during social exchanges is due to frustration from failed response reversal (e.g., continuing unrewarded responses in a gambling task). For example, Cornell et al. (1996) reported that "typical reactive offenses involved arguments with estranged spouses or disputes with neighbors or co-workers" (p. 785). Harenski and Kiehl (2010) in fact note that few if any models of research even address the issue of risk for frustration in psychopathy: "none to our knowledge have considered whether psychopaths show increased levels of any type of emotion" (p. 402).

Pertinently, certain regions of the limbic area have been directly implicated in the experience of anger and anger rumination (Denson, 2011). In particular, the dorsal anterior cingulate cortex (dACC) appears to play a special role in the subjective experience of anger. For example, Denson, Pedersen, Ronquillo, and Nandy (2009) reported that activity in the dACC following provocation was correlated with self-reported anger in response to the provocation ($r = .56$) and not with other emotions. Moreover, the dACC activity was correlated with scores of trait aggression on the Buss Aggression Questionnaire ($r = .61$). Inversely, patients who had portions of their bilateral ACC removed exhibited less anger following the cingulotomy (Cohen et al., 2001). As such, psychopaths who, as previously discussed, demonstrate impaired function of the ACC (inclusive of dACC) and associated limbic areas (see Kiehl, 2006) would seemingly be less likely to experience anger in response to provocation/frustration. Importantly, Blair (2010) highlights the existence of at least one study suggesting intact functioning of the dACC in children with psychopathic traits (Finger et al., 2008). Nonetheless, Harenski and Kiehl (2010) suggest that this finding of intact functioning in this group of children may have been confounded by the relatively low level of psychopathy traits. They contend that impaired function of the dACC and general paralimbic areas is probable only at the highest levels of psychopathy. As such, only at the highest levels of psychopathy may an individual be immune to anger/frustration from provocation.

Taken as a whole, these data suggest that despite frontal dysregulation that predisposes some individuals to reactive aggression, the most emotionally detached psychopaths may be protected against reactive aggression. In particular, deficient function in the threat circuitry (i.e., amygdala, hypothalamus, periaqueductal gray, and stria terminalis) and the dACC suggest that psychopaths are less likely to experience the prerequisite fear and anger for reactive violence.

8. Neuroendocrinology and neurochemistry

Because of its role in the fight-or-flight process, the noradrenergic system is a useful biological marker of emotional reactivity. When a threatening stimulus activates the neurons of the central nucleus of the amygdala, neurons projecting to the locus coeruleus stimulate the release of noradrenaline (Blair, 2006a). However, the noradrenergic system is sparsely studied in relation to aggression (Minzenberg & Siever, 2006), and even less so in relation to psychopathy. In fact, we

found only one study that assessed noradrenaline in relation to psychopathy. Lidberg, Levander, Schalling, and Lidberg (1978) reported lower levels of urinary noradrenaline in a group of high psychopathy men awaiting trial relative to a group of low psychopathy men. Moreover, the authors measured noradrenaline levels at two weeks, one week, and immediately before trial under the assumption that the final measurement would reflect greater levels of stress than preceding periods. Low psychopathy men demonstrated increasing levels of noradrenaline excretion, whereas high psychopathy men did not. However, the authors measured psychopathy using the Gough Delinquency scale (Gough & Peterson, 1952). This scale reflects nonspecific antisocial behavior rather than the affective and interpersonal components of psychopathy (Harpur et al., 1989) suggesting that it is a poor marker of the core features of psychopathy.

Much like noradrenaline, cortisol is a promising biological measure of emotional reactivity that has been relatively overlooked in samples with psychopathic traits (Glenn, 2009; Loney, Butler, Lima, Counts, & Eckel, 2006). Cortisol is a hormone released as part of the hypothalamic–pituitary–adrenal (HPA) axis in response to stressful events or cues. Moreover, cortisol acts in the amygdala to potentiate fear (Glenn, 2009). Accordingly, psychopaths, who demonstrate hyporesponsiveness of the autonomic nervous system and deficits in fear (e.g., Benning, Patrick, & Iacono, 2005; Patrick, 1994; Patrick, Bradley, & Lang, 1993) may be likely to manifest low cortisol levels. Indeed, compared with nonpsychopaths, clinical psychopaths show low resting cortisol levels (Cima, Smeets, & Jelicic, 2008; Holi, Auvinen-Lintunen, Lindberg, Tani, & Virkkunen, 2006). Loney et al. (2006) noted that in adolescents, low cortisol is not a correlate of general antisocial behavior, but rather appears to be unique to a subgroup with the most severe and chronic manifestations of conduct disorder. In their community sample, adolescent males with CU traits displayed decrements in baseline cortisol levels, much like adult psychopaths.

In a related vein, O'Leary, Loney, and Eckel (2007) measured cortisol *reactivity* in response to an environmental stressor. Using a collegiate sample, they measured psychopathy traits via the F1 scale of the LSRP. They found that high F1 males demonstrated reduced cortisol reactivity to a social stressor compared with low F1 males. They replicated this finding in men and extended it to women (O'Leary, Taylor, & Eckel, 2010). O'Leary et al. (2010) suggested that these findings indicate that high psychopathy (i.e., F1) individuals display altered cognitive appraisals of stress and, consequently, feel less threatened by stressors.

Glenn, Raine, Schug, Gao, and Granger (2011) tested the ratio of testosterone to cortisol in a sample of community adults (88% male). They found no significant relationship between psychopathy and baseline testosterone, baseline cortisol, or cortisol reactivity. However, Facet 2 (Affective) and Facet 3 (Lifestyle) of the PCL-R correlated significantly with the ratio of baseline testosterone to cortisol reactivity. That is, high psychopathy (Facets 2 and 3) individuals were more likely than low psychopathy individuals to have a high baseline testosterone and *relative* low cortisol stress response.

Broadly inclusive of these aforementioned data, the Triple Balance Model of Emotion (Terburg, Morgan, & van Honk, 2009; van Honk & Schutter, 2006) proposes that a high testosterone-to-cortisol ratio presumably reflects a motivational imbalance in which the individual is hyposensitive to threat/punishment and hypersensitive to reward through mutually opposing processes. This imbalance, as we will recall, is a motivational state consistent with psychopathy (Blair et al., 2005). Testosterone inhibits the function of HPA axis and associated autonomic systems, reducing sensitivity to punishment. In contrast, cortisol is thought to increase sensitivity to fear through suppression of the hypothalamic–pituitary–gonadal (HPG) axis (Glenn et al., 2011). van Honk, Harmon-Jones, Morgan, and Schutter (2010) speculated that this imbalance is associated with both reactive and instrumental aggression as a consequence of the lessening of serotonin levels. Specifically, low serotonin, which is associated with impulsivity and aggression (Siever, 2008; Spoont, 1992), combines with a high testosterone to cortisol ratio to produce reactive aggression in secondary psychopaths (i.e., those with characteristic traits of APD). However, with normal or heightened levels of serotonergic transmission, impulsive behavior abates (Terburg et al., 2009). These heightened levels, combined with a high testosterone-to-cortisol ratio, increase the risk for instrumental aggression in primary psychopaths (i.e., those marked by elevations on F1).

This hypothesis is supported by evidence that serotonergic deficits in psychopathy are linked to F2 but not F1 (Minzenberg & Siever, 2006).[7] Moreover, F1 may be associated with heightened levels of serotonin and consequently be protective against impulsive behavior. Dolan and Anderson (2003) examined the relationship between scores on the PCL-SV and serotonin function in a sample of forensic patients. Using the three-factor model, they found that serotonergic function correlated negatively with the antisocial-impulsive behavioral factor ($r = -.31$, $p < .05$), but positively with the arrogant-deceitfulness ($r = .34$, $p < .05$) and shallow-affect factors ($r = .27$, $p = .06$). As such, based on the degree of the core psychopathic traits, highly psychopathic individuals would be less likely to evince reactive violence, but would still exhibit risk for instrumental violence. These results dovetail with research indicating that serotonergic function is positively associated with social competence and dominance in primates (Young & Leyton, 2002), children (Kruesi et al., 1990), and adults (Moskowitz, Pinard, Zuroff, Annable, & Young, 2001). Because the affective and interpersonal components of psychopathy include the ability to charm, manipulate, con, and dominate others, a positive association between these traits and serotonergic functioning would be expected.

Overall, the core affective deficit of psychopathy (i.e., F1) appears to be linked to a deficit in both the hormones and neurotransmitters that potentiate emotional reactivity associated with reactive violence. In contrast, the social deviance component (i.e. F2) appears to be associated with a heightened responsivity in these neurochemical systems. The sum of these findings again suggests the potential for suppression among the underlying psychopathy factors.

9. Summary and conclusions

Although data strongly indicate a high risk for instrumental violence in psychopathy, the data for psychopathy and reactive violence remain equivocal. In fact, some data suggest that, perhaps paradoxically, psychopathy may be a protective factor against reactive violence in criminal populations (e.g., Dempster et al., 1996; Veit et al., 2010). Scrutiny of the cognitive/affective and biological data pertinent to reactive aggression similarly yields evidence of a potential protective effect of psychopathy (e.g., Benning, Patrick, Blonigen, et al., 2005; Cima et al., 2008; Dolan & Anderson, 2003; Holi et al., 2006; Kiehl, 2006; O'Leary et al., 2007; 2010; Patrick, 1994; Patrick et al., 1993). However, this finding may not be unexpected when

[7] Among the serotonin receptors, 5HTR1B and 5HTR2A are perhaps the most frequently referenced in regard to antisocial spectrum disorders. The research on 5HTR1B has been largely conducted with patients suffering from alcoholism, and thus it is difficult to discern findings for psychopathy. However, recent research has demonstrated a link between 5HTR2A activity and impulsivity, aggression, and antisocial behavior (Burt & Mikolajewski, 2008; Mik et al., 2007). The most frequently studied mechanism of action within the serotonergic system is the serotonin transporter linked polymorphic regions (5HTTLPR), which are a transporter protein that removes serotonin from synaptic spaces into presynaptic neurons. Located on Chromosome 17 (17q11.2), the serotonin transporter (5HTT or SLC6A4) encodes 5HTTLPR. A variable number tandem repeat length polymorphism (VNTR) in the promoter of this gene (5HTTLPR) has been shown to affect the rate of serotonin uptake and may play a role in behavioral illness, with the short alleles in this mostly diallelic (either short or long) polymorphism being associated with lower levels of gene transcription (Gunter, Vaugn, & Philibert, 2010).

considering the opposing associations between the subcomponents of psychopathy. For instance, Dempster et al. (1996), Flight and Forth (2007), and Walsh et al. (2009) all found opposing relations among the factors or facets of psychopathy and elements of reactive violence. Moreover, F1 has been linked empirically to diminished threat reactivity, autonomic response, and sensitivity to fear, as well as heightened serotonergic function suggestive of low impulsivity. Conversely, F2 psychopathy, primarily reflecting a disposition toward antisocial behavior, often demonstrates relationships in direct opposition to F1 for these biological indicators.

These findings suggest the potential for suppressor effects among the psychopathy factors in relation to forms of violence and their associated internal processes. Indeed, Hicks and Patrick (2006) identified a suppressor effect pertaining to anger/hostility in which F1 showed a weak negative association and F2 showed a strong positive association. Similarly, in our own laboratory we identified suppression between the factors in the prediction of anger responses to provocative vignettes. Whereas F1 was strongly and negatively related to anger activation, F2 was strongly and positively predictive of anger activation (Zeichner et al., in preparation). Furthermore, Blonigen et al. (2010) found cooperative suppression between the two PCL-R factors for both internalizing and externalizing behaviors.

The differential effects of the factors/facets indicate a need to better understand the constellation of disparate traits that underlie psychopathy. Different combinations of these traits may engender significant differences in behavioral manifestations. For example, Patrick (1994) demonstrated that high scores on F1 predicted diminished fear responding regardless of the level of F2. Pertinently, associations between psychopathy and reactive violence appear to be most tenuous in clinical samples of violent psychopaths and strongest in nonclinical/nonforensic populations. This finding may indicate that those who are the most emotionally detached, reflecting amygdalar dysfunction, may be the least susceptible to provocation as a consequence of hypoactive threat circuitry and associated negative affect. Hence, psychopathic individuals may be relatively unlikely to perpetrate reactive violence, despite a high likelihood of perpetrating some of the most severe and gratuitous unprovoked violence (e.g., Raine et al., 2006).

Understanding how the core affective, interpersonal, and behavioral components of psychopathy combine and perhaps interact may elucidate variations in the manifestations of this construct. For instance, the affective "deficits" of psychopathy, by themselves, may facilitate success in such occupations as surgery or the military, whereas the interpersonal traits, by themselves, may facilitate success in business, politics, or chicanery. However, a potent mix of facet traits may often yield a severely violent criminal, immune to negative affect and able to conceal his or her socially deviant behavior. This could bear significant implications for the detection and prevention of frequent and severe acts of violence. For example, Dennis Rader (the BTK killer) perpetrated 10 predatory murders while evading discovery by police for 31 years; Peter Sutcliffe murdered 13 women and attempted to kill seven more over a five year period before he was identified by police. Perhaps these individuals escaped detection for such long periods because they were able to control their behavior by operating within the confines of legal society, only to deviate when they perpetrated meticulous and controlled but severe violence. We believe that the sum of these data point to a need to develop and test models of psychopathy that consider multifactorial etiologies; varying combinations of biological predispositions and constellations of personality traits; and the interactive manifestations of such factors.

Our conclusions must be tempered by several limitations. For example, as we have noted, the distinction between instrumental and reactive aggression is at times unclear. It is likely that the same violent act could be categorized in different classes by different researchers. Measures of instrumental and reactive aggression probably vary in both the method and the degree to which they assess various aspects of distinction between the two constructs (e.g., emotional arousal, planning, provocation). Indeed, several researchers have created and used rigorous methods for parsing these forms of aggression (e.g., Cornell, 1993; Cornell et al., 1996; Falkenbach et al., 2008; Porter & Woodworth, 2007; Stafford & Cornell, 2003; Woodworth & Porter, 2002).

In addition, there is significant method variance in the measurement of both predictor and criterion variables. Whereas some researchers relied on detailed reports of one violent act, others relied on cumulative histories of violence collected from records, self-reports, and/or collateral reports. The latter practice is inherently problematic in youth populations, as children and adolescents have had less opportunity to accumulate aggressive histories, which may restrict range and limit the ability to detect genuine relationships. Moreover, measures of aggressivity for children and adolescents are often based on global and informal judgments about behavioral tendencies rather than actual recorded events.

Likewise, methods of assessing and conceptualizing psychopathy differ across studies, populations, and time. Early psychopathy research tended to treat the construct as a taxon in which the precipice of pathology was demarcated by a largely arbitrary cut score on the PCL measures. This practice is in opposition to the growing literature that substantiates the dimensional nature of psychopathy (e.g., Edens, Marcus, Lilienfeld, & Poythress, 2006; Guay, Ruscio, Knight, & Hare, 2007; Murrie et al., 2007). Moreover, artificial dichotomization of quantitative measures may result in loss of statistical power, measurement reliability, and information regarding individual differences (MacCallum, Zhang, Preacher, & Rucker, 2002). Perhaps most important, it ignores the influences of varying trait constellations (i.e., two factors, three factors, or four facets) within the global construct of psychopathy.

Lastly, although most research on adult forensic populations has used a similar set of measures (i.e. PCL, PCL-R, and PCL-SV), much of the research on college and child/adolescent populations has relied on a diverse mixture of self-report measures and rating inventories. The correlations among these measures tends to be moderate at best (Lilienfeld, 1994), rendering the interpretation of discrepant findings across studies unclear. In future work, it will be important to examine the potential role of measurement factors in delineating the links between psychopathy and both instrumental and reactive aggression. For example, some indicators of psychopathy, such as F1 of the PPI, appear to be saturated substantially with the personality trait of boldness, whereas others, such as the F1 scales of the LSRP and perhaps PCL-R, appear to be saturated substantially with the personality trait of meanness or callousness (Patrick, Fowles, & Krueger, 2009). Moreover, the correlation between F1 of the PPI and F1 of the PCL-R is only modest in magnitude (Malterer, Lilienfeld, Neumann, & Newman, 2010), suggesting that these two measures are assessing quite different features of psychopathy. It is not known how these differences across measures may impact the nature and magnitude of the associations between psychopathy and instrumental and reactive aggression. We therefore encourage meta-analytic investigators to examine the role of measurement factors in these associations.

Despite these considerations, it is clearly premature to conclude that psychopathy represents a heightened risk for reactive violence. There can be little doubt that psychopaths (as conceptualized within the predominant nomological network surrounding this construct) are commonly violent individuals. However, the manner in which they perpetrate such acts, as well as the psychological risk factors for such acts, requires continued examination. Disparate forms of aggression bear different targets for future treatment, prevention, and research efforts (Connor, 2002; Vitiello & Stoff, 1997). Interventions that work for individuals with APD may not work for those high on psychopathy. As such, it is necessary that future research examine the unique contributions of the disparate trait constellations of psychopathy to physical aggression, reactive violence in particular.

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