

# Clarifying the Role of Defensive Reactivity Deficits in Psychopathy and Antisocial Personality Using Startle Reflex Methodology

Uma Vaidyanathan  
Florida State University

Jason R. Hall  
University of South Florida

Christopher J. Patrick and Edward M. Bernat  
Florida State University

Prior research has demonstrated deficits in defensive reactivity (indexed by potentiation of the startle blink reflex) in psychopathic individuals. However, the basis of this association remains unclear, as diagnostic criteria for psychopathy encompass two distinct phenotypic components that may reflect differing neurobiological mechanisms—an *affective–interpersonal* component and an *antisocial deviance* component. Likewise, the role of defensive response deficits in antisocial personality disorder (APD), a related but distinct syndrome, remains to be clarified. In the current study, the authors examined affective priming deficits in relation to factors of psychopathy and symptoms of APD using startle reflex methods in 108 adult male prisoners. Deficits in blink reflex potentiation during aversive picture viewing were found in relation to the affective–interpersonal (Factor 1) component of psychopathy, and to a lesser extent in relation to the antisocial deviance (Factor 2) component of psychopathy and symptoms of APD—but only as a function of their overlap with affective–interpersonal features of psychopathy. These findings provide clear evidence that deficits in defensive reactivity are linked specifically to the affective–interpersonal features of psychopathy and not to the antisocial deviance features represented most strongly in APD.

*Keywords:* psychopathy, antisocial personality disorder, startle, defensive reactivity, fear

The etiologic basis of antisocial behavior has long been a focus of intensive study. Two diagnostic entities commonly associated with persistent antisocial behavior are psychopathy and antisocial personality disorder (APD). While these two diagnoses share several features, they also differ in important ways, both phenotypically and neurobiologically. Whereas both are marked by the presence of chronic antisocial deviance beginning in childhood and persisting into adulthood, psychopathy additionally entails a constellation of affective–interpersonal features, including lack of normal affective reactivity, callousness, and interpersonal charm. Though psychopathy has traditionally been regarded as a unitary syndrome, recent research suggests that its observable symptoms reflect two distinct underlying

processes—one related to the deficient behavioral inhibition characteristic of APD and the other to deficits in defensive reactivity (Fowles & Dindo, 2006; Levenston, Patrick, Bradley, & Lang, 2000; Patrick & Bernat, 2009). Evidence of defensive response deficits in psychopathy, particularly in association with the distinctive affective–interpersonal features that distinguish it from APD, have emerged from studies of the startle response—a reflex that is reliably potentiated in the context of aversive or threatening cues. The aim of the present study was to replicate and extend this line of research by more systematically evaluating relations of the two factors of psychopathy and symptoms of APD with deficits in aversive potentiation of the startle blink reflex.

## Psychopathy and Antisocial Personality Disorder

The most widely used instrument for assessing psychopathy over the past two decades has been the Psychopathy Checklist—Revised (PCL–R; Hare, 2003). Although the PCL–R was developed to index psychopathy as a unitary disorder, its 20 items coalesce around two distinctive factors that exhibit diverging relations with external criteria from multiple domains (Harpur, Haktian, & Hare, 1988), suggesting that two separate processes contribute to the disorder. Factor 1 encompasses the affective and interpersonal features of psychopathy and is characterized by tendencies toward narcissism, superficial charm, conning/manipulation, callousness, shallow affect, and lack of remorse. Factor 2 indexes general antisocial deviance through items reflecting irresponsibility, impulsivity, aggression, and chronic rule- and law-breaking behavior. Prior research has indicated that it is the second

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Uma Vaidyanathan, Christopher J. Patrick, and Edward M. Bernat, Florida State University; Jason R. Hall, University of South Florida.

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Correspondence concerning this article should be addressed to Christopher J. Patrick, Florida State University, Department of Psychology, 1107 West Call Street, Tallahassee, FL 32306. E-mail: cpatrick@psy.fsu.edu

factor that shows the greatest overlap with APD (Harpur et al., 1988; Patrick, Hicks, Krueger, & Lang, 2005).

APD is defined in the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association, 2000)* as “a pervasive pattern of disregard for, and violation of, the rights of others that begins in childhood or early adolescence and continues into adulthood” (p. 701). Though intended to capture the psychopathy construct, APD is diagnosed predominantly on the basis of antisocial and/or aggressive behaviors corresponding to the antisocial deviance (Factor 2) component of psychopathy. This emphasis on overt antisocial behaviors, in addition to the fact that prevalence rates for APD are several times higher than psychopathy in forensic settings (Hare, 2003), has led to criticisms of APD as an operationalization of psychopathy (Hare, Hart, & Harpur, 1991). However, it should be noted that the adult criteria for APD in particular include items dealing with lack of remorse, externalization of blame, and conning/deceitfulness—which are included among the affective–interpersonal (Factor 1) features of PCL–R psychopathy. From this standpoint, the adult criteria for APD can be viewed as providing partial coverage of these essential features of psychopathy. Additionally, previous research on this topic (see Kosson, Lorenz, & Newman, 2006, for a review) suggests that individuals who meet diagnostic criteria for both APD and psychopathy exhibit affective deficits and criminal behaviors not evident in those with APD alone. Similarly, a recent study by Poythress et al. (2010) used model-based cluster analysis to examine subtypes of APD among incarcerated individuals and found evidence for distinct and homogenous clusters of offenders with APD, including both psychopathic and nonpsychopathic variants. Nonetheless, the distinction between psychopathy and APD remains an active matter of debate, particularly as efforts proceed toward development of criteria for *DSM-5*.

### Psychopathy Factors and Defensive Startle Reactivity

Prior research dealing with the distinct correlates of the two psychopathy factors can potentially clarify the nature of the association between psychopathy and APD. Although the two PCL–R factors are moderately intercorrelated, they demonstrate diverging relations with a host of criterion-related variables spanning multiple assessment domains, particularly when controlling statistically for their overlap. Of particular relevance to the present study, the unique variance in PCL–R Factor 1 exhibits negative relations with measures of trait negative affect, including indices of dispositional fear (Hicks & Patrick, 2006), whereas variance unique to PCL–R Factor 2 shows positive associations with measures of trait anxiety, neuroticism, depression, and suicidality (Hicks & Patrick, 2006; Patrick, 1994; Verona, Patrick, & Joiner, 2001; Widiger & Lynam, 1998).

Based on evidence of this sort, it has been posited that the affective–interpersonal features of psychopathy indexed by PCL–R Factor 1 reflect an underlying impairment in emotional responsiveness to aversive/fearful stimuli (cf. Fowles & Dindo, 2006; Patrick & Bernat, 2009). One well-validated physiological index of fear reactivity is potentiation of the defensive startle reflex. Previous research in normal populations has shown that the amplitude of the blink response, elicited by an acoustic noise probe and measured via electromyographic (EMG) recording of the *orbicularis oculi* muscle, is reliably enhanced during viewing of aversive or threat-

ening foreground stimuli, relative to neutral (cf. Vrana, Spence, & Lang, 1988). Basic neuroscience research has demonstrated that this effect is mediated by the amygdala, a centerpiece of the brain’s defensive (fear) system (Davis, 1998).

In contrast with individuals from the general population and incarcerated controls, offenders high in psychopathy exhibit diminished or absent startle potentiation in the face of negative emotional cues such as aversive images (Levenston et al., 2000; Patrick, Bradley, & Lang, 1993). While some studies have found that this deficit in aversive startle modulation is specifically associated with PCL–R Factor 1 (Patrick, 1994; Patrick et al., 1993), others have reported interactive effects of Factors 1 and 2 on startle potentiation (Sutton, Vitale, & Newman, 2002; Vanman, Mejia, Dawson, Schell, & Raine, 2003). However, given distinctive aspects of samples in these prior studies (i.e., incarcerated female participants in Sutton et al., 2002; community participants in Vanman et al., 2003), the replicability of these findings in incarcerated male offenders remains to be determined. Thus, one of the aims of the current study was to replicate and extend prior research by examining the separate and interactive effects of PCL–R Factors 1 and 2 on affective modulation of startle in a sample of incarcerated offenders. Additionally, no prior work has directly addressed the overlap between APD and the two psychopathy factors with respect to affective startle modulation. The current study addressed this gap in the literature by examining defensive reactivity as indexed by aversive startle potentiation in relation to *DSM-IV* APD and PCL–R psychopathy within the same participant sample. Our major hypotheses were that (a) deficits in aversive startle potentiation would be selectively related to scores on PCL–R Factor 1, and (b) scores on Factor 1 of the PCL–R would mediate any associations of PCL–R Factor 2 or APD symptoms with aversive startle potentiation.

### Method

#### Participants

Participants were 108 male prisoners recruited from a medium-security state prison in Minnesota. Informed consent was obtained prior to testing. Subjects were screened via questionnaire to be free of visual or hearing impairments and received \$20, deposited to their institutional accounts, for participating. Data for three additional subjects were dropped due to equipment malfunction. Data for 31 others were excluded from analyses due to unusable startle response data (see below).

#### Measures

**Psychopathy Checklist—Revised.** Subjects were rated on the PCL–R based on a semistructured diagnostic interview and information derived from prison file records. Primary diagnostic ratings were assigned by the interviewer. Separate independent ratings were provided by another diagnostician based on a video of the interview together with file information. Interviewers were advanced undergraduate or graduate-level psychology students trained in the use of the PCL–R. Their training included completion of ratings for multiple sample cases to establish reliability with experienced raters. Following training, in connection with ratings of new cases, primary and secondary raters met with project investigators an

ongoing basis to protect against rater drift. PCL-R total and factor scores for each of the two raters were averaged for each case. Consistent with prior research, scores on Factor 1 and Factor 2 were positively correlated ( $r = .57, p < .001$ ). Subjects were diagnosed as psychopathic if their mean PCL-R total score exceeded a value of 30 ( $n = 35$ ) and as nonpsychopathic if their mean PCL total was 20 or less ( $n = 26$ ).

**Antisocial personality disorder.** Subjects were rated for child and adult symptoms of *DSM-IV* APD based on structured interview questions phrased specifically to assess for these symptoms. Information from prison file records was also used in the ratings of APD symptom criteria; conduct disorder symptoms were assessed retrospectively, using information gathered from the diagnostic interview and from collateral file records. As with psychopathy ratings, primary diagnostic ratings for APD were assigned by the interviewer, and secondary ratings were assigned by an independent diagnostician. A participant was considered to have met criteria for APD if both independent raters assigned a diagnosis of APD. One subject could not be diagnosed due to missing diagnostic information from one rater, leading to a total of 107 subjects with diagnoses for APD, though data regarding symptoms were available for all subjects. Sixty-six participants were diagnosed with APD, and 41 were not. A cross-tabulation of psychopathy and APD diagnoses is presented in Table 1. For analyses involving APD symptom scores, the mean of scores assigned by the two raters was used for each case; separate scores for adult and child (i.e., conduct disorder) symptoms of APD were calculated for each subject.

## Procedure

The affective picture-viewing task incorporated 66 pictures from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 1999), presented for 6 s each.

Startle probes, consisting of 50-ms, 105-dB white noise tone bursts with abrupt ( $<10 \mu\text{s}$ ) rise time, were generated by an S81-02 Coulbourn white noise generator and presented binaurally through insert earphones (Etymotic Research Inc., Elk Grove Village, IL). To familiarize subjects with the noise probe stimulus, habituation probes (excluded from analyses) were presented during the first three pictures. Of the remaining 63 picture trials, 54 included noise probes occurring between 3 and 5 s after picture onset. To reduce predictability of the noise stimulus, probes were presented at variable points during 10-s intertrial intervals for the other nine picture trials. Data for these intertrial probe stimuli were excluded from analyses.

The 54 pictures during which startle probes were presented consisted of 18 pleasant, 18 neutral, and 18 unpleasant scenes selected on the basis of IAPS normative ratings of valence and arousal. Pleasant pictures included erotic scenes and adventure-related ("action") scenes (e.g., bungee-jumping, skydiving); aversive pictures included images of directly threatening objects ("threat" scenes), such as menacing figures or guns pointed toward the viewer, and depictions of vicarious attack ("victim" scenes). Neutral scenes included pictures of nonaffective stimuli such as buildings, kitchen utensils, and other commonplace objects (e.g., truck, fire hydrant). Pleasant and aversive picture sets were selected to be equivalent in average rated arousal and more arousing on average (to a comparable degree) than neutral pictures. Picture stimuli were presented in 12 different orders, counterbalanced according to the following rules: No two pictures of the same content appeared consecutively; pictures were rotated across stimulus orders to ensure they appeared in both probed and nonprobed trials; the order of pictures and probes were rotated to offset effects of serial position.<sup>1</sup>

## Physiological Data Acquisition and Reduction

Participants sat in a padded recliner at a distance of 100 cm from a 52-cm computer monitor on which picture stimuli were displayed. Data collection was performed using two computers, one configured with E-Prime software (Version 1.1) for stimulus control and the other with Neuroscan Acquire (Version 4.2) software for physiological data acquisition. Blink EMG responses to noise probes were recorded from a pair of 0.25 cm Ag-AgCl electrodes (Med Associates, St. Albans, VT) filled with electrolyte gel and positioned over the *orbicularis oculi* muscle under the left eye. Responses were recorded at a sampling rate of 2000 Hz using a Neuroscan SynAmps amplifier, with a 500-Hz low-pass and 0.05 Hz high-pass analog filter applied before digitization to prevent aliasing. Data were then digitally high-pass filtered at 10 Hz to remove artifacts due to movement and rectified and integrated using a digital single-pole recursive filter (implemented using Matlab Version 2006b) to simulate a Coulbourn contour-following filter with a 30-ms time constant.

Following this procedure, the magnitude of the startle blink response to each probe stimulus was scored using an algorithm in Matlab. The peak of the blink response was quantified as the highest point occurring between 30 and 120 ms after noise probe onset, relative to the median activity during the 50-ms period preceding the probe. Next, all trials were visually inspected to

Table 1

*Cross-Tabulation of Participant Diagnoses of Psychopathy and Antisocial Personality Disorder*

Disorder	Diagnostic status	Antisocial personality disorder	
		Yes	No
Psychopathy	Yes	33	2
	No	33	39

<sup>1</sup> The following IAPS pictures were used: Pleasant: 2381, 4000, 4233 (4617), 4274, 4230, 4653 (4750), 4690, 4687, 4290 (4651), 4533(8032), 8041, 8033, 5622 (8250), 5626, 5623, 8370 (8180), 8080, 8042. Neutral: 2190, 2210, 2214, 2372, 2480, 2495, 2850, 2890, 9700, 7002, 7030, 7034, 7040, 7050, 7150, 7205, 7705, 7710. Aversive: 6010, 2520, 9594 (4621), 6571, 9400, 6530 (3550), 9250, 3400, 6350 (3500), 2100 (6241), 2682, 2130, 6242 (6244), 6370, 6243, 6510, (6250), 6260, 6230. Habituation: 4650, 7080, 9252. Picture sets for the current study matched those used in an earlier study (Bernat, Patrick, Benning, & Tellegen, 2006) that examined effects of picture content, valence, and intensity on physiological reactivity. Pictures in parentheses are alternate exemplars from the same content category that were substituted within some stimulus orders to achieve counterbalancing of conditions across run orders.

identify responses with unstable baselines and zero-amplitude responses. Trials with unstable baselines were defined as those in which (a) blink onset occurred earlier than 20 ms, (b) an apparent startle response overlapped with a preceding spontaneous eye-blink, or (c) EMG activity during the preprobe baseline period was highly variable. Zero amplitude response trials were defined as trials in which no discernible blink response occurred within the 30- to 120-ms peak window. Subjects for whom 25% or more of the trials were rejected or scored as zero response trials were omitted from the analyses.

## Data Analysis

Since individuals varied considerably in levels of raw blink magnitude, and because we were primarily interested in patterns of blink reactivity across pleasant, neutral, and aversive pictures, raw blink response data were standardized across picture-probe trials for each participant.<sup>2</sup> Responses for each participant (in microvolts) were converted to *z* score and then *T* score units as follows: *z* score value = (raw magnitude value -  $M_{\text{all raw values}}$ )/ $SD_{\text{all raw values}}$ ; *T* score value = (*z* score value × 10) + 50. This yielded standardized blink magnitude scores with a mean of 50 and a standard deviation of 10 for each participant; these scores were used in the analyses reported here.

To confirm basic modulatory effects of emotion on startle reactivity in the sample as a whole, we performed a repeated measures analysis of variance in which startle blink amplitude served as the dependent variable and picture valence (pleasant, neutral, unpleasant) served as the within-subjects independent variable. The effect of psychopathy diagnosis on affect–startle modulation was assessed by including group membership in the analysis as a between-subjects factor (i.e., psychopath vs. nonpsychopath). A parallel two-way analysis was conducted to the effect of APD diagnosis on startle modulation.

To evaluate continuous effects of each PCL–R factor on startle modulation across participants, we computed aversive minus neutral potentiation scores and pleasant minus neutral inhibition scores for each participant.<sup>3</sup> To account for overlap between scores on the two PCL–R factors ( $r = .57$ ), continuous scores on both PCL–R factors along with an interaction term indexing the combined effect of the two factors were included in a regression model predicting startle modulation scores. Continuous associations with startle modulation scores were also examined for overall APD symptoms and for child and adult symptoms of APD separately. Lastly, regression analyses were performed to evaluate unique predictive associations for APD symptom scores in relation to scores on the two PCL–R factors.

## Results

### Basic Emotion Modulation Effects

Replicating prior research findings for unselected participants from the community, prisoners within the current sample as a whole demonstrated the expected linear pattern of startle modulation across picture categories (i.e., aversive > neutral > pleasant; omnibus  $F(2, 214) = 48.54, p < .001$ ; linear contrast  $F(1, 107) = 84.59, p < .001$ ).

### Psychopathy Groups, APD Diagnosis, and Aversive Startle Modulation

A significant Group × Picture Category (pleasant, neutral, aversive) interaction,  $F(2, 118) = 4.91, p < .01$ , indicated that psychopathic and nonpsychopathic groups differed in patterns of affective startle modulation. Consistent with prediction, follow-up tests revealed that this interaction was attributable to a difference in startle modulation for aversive pictures—specifically, Group × Aversive/Neutral  $F(1, 59) = 9.19, p < .01$ . Whereas nonpsychopathic offenders showed robust startle potentiation for aversive pictures relative to neutral,  $t(25) = 6.65, p < .001$ , psychopathic offenders showed no such effect,  $t(34) = .80, p > .05$ . In contrast, psychopathic and nonpsychopathic groups did not differ in degree of startle inhibition for pleasant scenes, Group × Pleasant/Neutral  $F(1, 59) = .87, p > .05$ . A corresponding analysis for APD diagnosis revealed no evidence of a moderating effect of this dichotomous variable on startle modulation across the three picture valence categories,  $F(2, 210) = .34, p > .05$ , or on startle potentiation specifically for aversive scenes,  $F(1, 105) = .61, p > .05$ .

### Psychopathy Factor Scores, APD Symptom Counts, and Aversive Startle Modulation

Table 2 shows intercorrelations among PCL–R Total and factor scores and APD symptom count variables; correlations between these variables and startle blink modulation are presented in Table 3. Consistent with prediction, PCL–R Factor 1 showed a significant negative association with startle potentiation scores for aversive scenes as a whole ( $r = -.26, p < .01$ ). Supplemental analyses for specific aversive contents indicated that this negative association was stronger for pictures depicting threatening content ( $r = -.27$ ) than for victim scenes ( $r = -.14$ ), but the difference in these correlations (evaluated using Steiger's *t* test) was nonsignificant. Thus, remaining analyses of aversive potentiation effects were combined across these contents. Scores on PCL–R Factor 2 also evidenced a negative association with aversive modulation scores, but this relationship did not achieve significance ( $r = -.17, p > .05$ ; see Table 3). Neither PCL–R factor score was significantly associated with pleasant modulation scores.

To account for overlap between the PCL–R factors, scores for the two were entered concurrently in a regression model along with a term corresponding to their interaction, to predict aversive modulation scores. The model yielded a significant overall regression coefficient ( $R = .28, p < .05$ ) and a significant contribution of Factor 1 ( $\beta = -.25, p < .05$ ), whereas the contributions of Factor 2 and the interaction term were not ( $\beta_s = .01$  and  $.10$ , respectively,  $ps > .05$ ).

<sup>2</sup> No significant correlations were found between untransformed (i.e., raw) startle blink magnitudes recorded during pictures of any type (pleasant, neutral, or aversive) and PCL–R Total or Factor scores or APD symptom counts.

<sup>3</sup> Both group and continuous scores analyses are reported for the PCL–R and for APD to provide continuity with prior published literature and to provide alternative perspectives on the data. Regarding categorical versus dimensional approaches to psychopathy more broadly, our perspective is that these two approaches to description are complementary and have differing utility in differing contexts.

Table 2  
Correlations Between PCL-R Scores and APD Symptom Scores in Overall Sample (N = 108)

Variable	APD total symptoms	Conduct disorder symptoms	Adult antisocial behavior symptoms
PCL-R Total	.78***	.63***	.86***
PCL-R Factor 1	.60***	.45***	.71***
PCL-R Factor 2	.77***	.63***	.82***

Note. PCL-R = Psychopathy Checklist-Revised; APD = antisocial personality disorder.  
\*\*\*  $p < .001$ .

As shown in Table 3, APD symptoms as a whole (child + adult) were correlated negatively, but not significantly, with aversive startle modulation scores. A somewhat stronger, trend-level association ( $p = .08$ ) was evident for adult APD symptoms compared to child symptoms. A regression analysis incorporating adult APD symptoms together with scores on both PCL-R factors to predict aversive startle modulation scores yielded a significant overall  $R$  of .27 ( $p = .05$ ), with a unique predictive contribution for Factor 1 scores ( $\beta = -.28$ ,  $p < .05$ ), but negligible contributions for adult APD symptoms and PCL-R Factor 2 scores ( $\beta$ s = .11 and  $-.10$ , respectively,  $ps > .05$ ). This indicates that the relationship between adult APD symptoms and aversive startle modulation was entirely mediated by overlap with the affective-interpersonal features of psychopathy.

### Discussion

The findings of the present study provide compelling evidence that (a) it is specifically the affective-interpersonal (PCL-R Factor 1) component of psychopathy that is associated with deficient defensive reactivity as indexed by startle blink potentiation; (b) impulsive-antisocial tendencies reflected in Factor 2 of the PCL-R and symptoms of APD (adult symptoms specifically) are associated to a lesser degree with reduced defensive reactivity, and only as a function of their overlap with affective-interpersonal features of psychopathy. Although prior research in female prisoner (Sutton et al., 2002) and male community (Vanman et al., 2003) samples reported evidence of an interaction between PCL-R Factors 1 and 2 in the prediction of aversive startle modulation deficits, this effect was not replicated in the current male prisoner sample. Thus,

further research is needed to clarify circumstances under which the two PCL-R factors may operate in concert to moderate emotional responsiveness. Nonetheless, the current findings add to a growing body of data indicating an impact of the affective-interpersonal factor on emotional processing and reactivity in laboratory tasks.

Extrapolating from this point, the current results are important in several respects. First, they provide insight into the biological processes underlying psychopathy and APD, lending support to a two-process model in which the affective-interpersonal features of psychopathy are theorized to reflect an etiologic mechanism distinct from that underlying the antisocial deviance features (Fowles & Dindo, 2006; Patrick & Bernat, 2009). According to this model, trademark features of psychopathy such as callousness, shallow affectivity, persuasiveness, and superficial charm reflect the overt (phenotypic) expression of an underlying (genotypic) low-fear disposition arising from a weakness in the defensive motivational system. From the perspective of this model, this weakness in defensive (fear) reactivity is one of the characteristics that distinguish offenders high in overall psychopathy from those diagnosed solely with APD.

More broadly, differences in the neurobiological correlates of these distinctive clinical phenomena have significant implications for diagnostic conceptualizations. For example, though our results show that APD as a whole is unrelated to deficits in startle blink potentiation, the adult criteria for APD do appear to index some elements of psychopathy that are predictive of startle modulation deficits. This suggests that the diagnosis of APD could be effectively refined either by specifying a distinct psychopathic variant of APD in which the affective-interpersonal features of psychopathy are evident to a marked degree or, alternatively, by defining psychopathic personality disorder as a diagnostic entity that is distinct from APD. Changes in the criteria for assessing personality disorders in the upcoming edition of the *DSM* may afford some basis for progressing in this direction.

Finally, and perhaps more intriguingly, our results suggest that variations in defensive reactivity, and affiliated trait manifestations (e.g., self-assurance, persuasiveness, empathy), are potentially dissociable from the antisocial deviance features of psychopathy. In view of this, our understanding of psychopathy would likely benefit from a focus on individuals in the general population who exhibit low levels of dispositional fear but who lack the salient aggressive-antisocial tendencies characteristic of criminal psychopaths.<sup>4</sup> Studies along these lines may be of particular relevance to the much-discussed but understudied notion of "successful" or noncriminal psychopathy, en-

Table 3  
Correlations of PCL-R Scores and APD Symptom Scores With Startle Modulation Scores for Pleasant and Aversive Pictures (N = 108)

Diagnostic variable	Aversive-neutral potentiation	Pleasant-neutral inhibition
PCL-R scores		
Total	-.23*	-.04
Factor 1	-.26**	-.07
Factor 2	-.17	-.08
APD symptoms		
Overall	-.11	-.05
Child	-.07	-.08
Adult	-.17	.02

Note. PCL-R = Psychopathy Checklist-Revised; APD = antisocial personality disorder.  
\*  $p < .05$ . \*\*  $p < .01$ .

<sup>4</sup> A striking illustration of an individual of this type is provided by bomb disposal expert William James, as portrayed by actor Jeremy Renner in the 2010 Academy Award-winning film, *The Hurt Locker*.

tailing the presence of affective–interpersonal features in the absence of severe antisocial deviance (Hall & Benning, 2006). Recent research using the self-report based Psychopathic Personality Inventory (Lilienfeld & Andrews, 1996) to investigate neurobiological correlates of psychopathic traits in community samples indicates that, similar to psychopathic offenders, individuals in the community with elevated scores on the interpersonal–affective factor exhibit diminished physiological reactivity to aversive emotional cues (Benning, Patrick, & Iacono, 2005; Gordon, Baird, & End, 2004; Vaidyanathan, Patrick, & Bernat, 2009).

Work of this kind could provide a basis for linking research on psychopathy to literatures on psychological resiliency and social competence (e.g., Masten & Motti-Stefanidi, 2009). In the absence of disinhibited-aggressive propensities, low dispositional fear might function as a psychological asset, conferring resistance to distress-related psychopathology (e.g., phobias, depression, suicide) and contributing to confidence, assertiveness, leadership, and courageous behavior. Continued research into the distinctive underpinnings of these affective–interpersonal traits (and identification of effective neurobiological indicators) is likely to be of substantial importance to a complete understanding of psychopathy in its most malignant and adaptive forms.

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