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University of Wisconsin—Madison

Laboratory studies of psychopathy have yielded an impressive array of etiologically relevant findings. To date, however, attempts to demonstrate the generalizability of these findings to African American psychopathic offenders have been largely unsuccessful. The fear deficit has long been regarded as the hallmark of psychopathy, yet the generalizability of this association to African American offenders has not been systematically evaluated. In this study, we used an instructed fear paradigm and fear-potentiated startle to assess this deficit and the factors that moderate its expression in African American offenders. Furthermore, we conceptualized psychopathy using both a unitary and a two-factor model, and we assessed the constructs with both interview-based and self-report measures. Regardless of assessment strategy, results provided no evidence that psychopathy relates to fear deficits in African American offenders. Further research is needed to clarify whether the emotion deficits associated with psychopathy in European American offenders are applicable to African American offenders.

Keywords: African American offenders, psychopathy, fear deficit

The scientific study of psychopathy has made great progress in multiple domains, spanning the identification of potential etiological mechanisms, development of self-report and interview-based assessments of the construct, and specification of relevant diagnostic dimensions and subtypes. Despite these advances, much of the research progress in psychopathy is based solely on European Americans. Increasingly, researchers are focusing on the generalizability of psychopathy-related constructs, but most of the research examines whether assessment tools are equally appropriate across race and ethnic groups (Cooke, Kosson, & Michie, 2001; Swogger, Walsh, & Kosson, 2008; Vitacco, Neumann, & Jackson, 2005). By contrast, research on the generalizability of laboratory-based correlates of psychopathy across races is extremely limited. Given that prior research has documented similar rates of psychopathy in African Americans and European Americans (Skeem, Edens, Camp, & Colwell, 2004), it is essential for investigators to examine the generalizability of etiologically relevant laboratory findings as well as other external correlates of psychopathy to non-European American individuals.

To date, research that explicitly evaluates race-related differences in psychopathy and the generalizability of known laboratory correlates of psychopathy to African American offenders has yielded equivocal results. On the one hand, some investigators have reported a lack of race-related differences in laboratory correlates of psychopathy on the basis of the absence of a significant race by psychopathy interaction (Epstein, Poythress, & Brandon, 2006; Kosson, Miller, Byrnes & Leveroni, 2007; Suchy & Kosson, 2005). However, the absence of a significant interaction is not the same as demonstrating that a particular finding generalizes to another group (i.e., African American offenders). Depending on sample size and within-sample variability, it is entirely possible for a psychopathy-related effect to be significant in one group, have virtually no association within the second group, and still not yield a significant interaction. For this reason, it is crucial to evaluate explicitly whether etiologically relevant correlates of psychopathy in European American samples generalize to (i.e., replicate in) African American samples before assuming their causal significance in the latter group.

On the other hand, research explicitly examining the generalizability of known correlates of psychopathy has generally failed to replicate etiologically relevant laboratory findings in African Americans. For example, attempts to replicate theoretically significant abnormalities in passive avoidance learning (Kosson, Smith, & Newman, 1990; Newman & Schmitt, 1998; Thornquist & Zuckerman, 1995), emotion facilitation on a lexical decision task (Lorenz & Newman, 2002a), and selective attention (Newman, Schmitt, & Voss, 1997) in African American offenders have all been unsuccessful.

Despite growing evidence that laboratory correlates of psychopathy are difficult to replicate in African American samples, the existing literature is limited in several respects. First, the power to observe moderate effect sizes within the African American samples in these studies varied from about .59 to .99. Thus, it is possible that some of these investigations would have yielded significant psychopathy-related effects had they included a larger

sample. Second, the number of laboratory correlates examined has been relatively few. For each etiologically relevant laboratory finding, there is an opportunity to demonstrate its relevance for African American samples or, alternatively, to establish the limits of its generalizability. Third, investigators have yet to examine whether the fear deficit, long considered the primary etiologically relevant correlate of psychopathy, replicates in African American samples. In light of its central role in conceptualizations of psychopathy, this omission represents a significant lacuna in our evaluation of psychopathy in African Americans and the extent to which key correlates of psychopathy in European American offenders extend to African American offenders.

A variety of results have contributed to the widely accepted view that fear deficits are a core feature of psychopathy (Blair, Mitchell, & Blair, 2005; Lykken, 1995). In numerous tasks, psychopathic individuals demonstrate poor fear conditioning, reduced electrodermal responses to threat stimuli, and reduced fear-potentiated startle (Birbaumer et al., 2005; Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002; Hare, 1978; Lykken, 1957; Patrick, Bradley, & Lang, 1993). For example, Patrick (1994) has reported that male psychopathic individuals fail to demonstrate normal potentiation of startle during unpleasant images.

In a recent investigation of fear-potentiated startle in psychopathy, Newman, Curtin, Bertsch, and Baskin-Sommers (2010) found that the fear deficit of psychopathic individuals is moderated by focus of attention (see also Dvorak-Bertsch, Curtin, Rubinstein, & Newman, 2009). In both studies, fear-potentiated startle was measured as participants performed a task under three conditions. Each condition placed different demands on attention and working memory. One condition required participants to respond on the basis of the threat-relevant aspect of the stimuli. Two other conditions required participants to perform an alternative primary task (e.g., discrimination of letter case or a two-back working memory task), so that threat was no longer relevant to their goal-directed behavior. Psychopathy scores were significantly and inversely related to fear-potentiated startle under conditions that required participants to focus on a threat-irrelevant dimension of stimuli. However, they were positively, though nonsignificantly, correlated with fear-potentiated startle under conditions that involved focusing on the threat-relevant dimension. Such findings are particularly important because they suggest that the fear deficits of psychopathic individuals may not be absolute. Rather, they may be a product of specific attentional demands. To date, however, there is no explicit replication of the fear deficit or the potential importance of attention in moderating this deficit in an African American sample.

Toward this end, we investigate whether African American individuals display a fear deficit and, if so, whether it is moderated by attention, as has been demonstrated in European Americans (Newman et al., 2010). We measured psychopathy in three ways. First, we assessed psychopathy with the Psychopathy Checklist—Revised (PCL–R; Hare, 2003). Second, we used the Hare Self-Report Psychopathy–III (SRP-III; Paulhus, Hemphill, & Hare, in press) to evaluate whether the correlates of psychopathy in African Americans may be different for interviewer-based compared with self-report measures of psychopathy.

Last, some researchers have advocated parsing psychopathy into two components (i.e., interpersonal–affective and impulsive–antisocial) so that the unique correlates of the factors, which otherwise could be obscured, can be identified (Patrick, 2007). The interpersonal and affective symptoms of psychopathy (i.e., PCL–R Factor 1) have been suggested to correspond to an amygdala-related deficit in emotion processing (Patrick, 1994, 2007). Conversely, the impulsive and antisocial symptoms of psychopathy (i.e., PCL–R Factor 2) have been attributed to a deficit in higher order processes that interferes with a person's ability to inhibit approach behavior or focus on threat cues and, thus indirectly, results in weak defensive system functioning (Patrick, 1994, 2007). Here, too, we used both an interview-based measure (Hare, 2003) and a self-report measure (Benning, Patrick, Blonigen, Hicks, & Iacono, 2005; see also Dvorak-Bertsch et al., 2009, for a discussion of the Multidimensional Personality Questionnaire—Brief [MPQ–B] factors)[1] to examine whether the association between the psychopathy factors and fear-potentiated startle would replicate in African American offenders. Although, there are differences in content between these assessments, and at times the correlations between the PCL–R and other measures of psychopathic traits are small, the inclusion of these various measures allowed for comparisons with previously reported results.

Method

Participants

Participants were 92 male inmates from a maximum security prison in southern Wisconsin. A priori power analysis conducted with the pwr package in R (R Development Core Team, 2009) confirmed that the current sample size provided 96% power to detect moderate effect size ($f^2 = .15$; Cohen, 1992) with a two-tailed alpha of .05. Participants were excluded according to the same criteria used by Newman et al. (2010) with the European American sample. First, participants who were 46 years of age or older were eliminated because the expression of psychopathy has been found to change with advancing age (Hare, Harpur, & Hakstian, 1990). Second, to increase the likelihood that participants had the intellectual aptitude to complete self-report measures and laboratory tasks, we excluded those with scores below 70 on the Shipley Institute of Learning (Zachary, 1986) estimate of intelligence. Third, we disqualified individuals who, on the basis of institutional records, had a clinical diagnosis of schizophrenia, bipolar disorder, or psychosis not otherwise specified, because traditional definitions of psychopathy rule out psychoses (e.g., Cleckley, 1976) and, consistent with this view, individuals diagnosed with psychopathy on the PCL–R are unlikely to receive one of these Axis I diagnoses (Hart & Hare, 1989). Last, we excluded anyone who was currently using psychotropic medications because such use could interfere with startle reactivity.

Measures

Psychopathy Checklist—Revised. Ninety-two participants were assessed with file information and a semistructured interview

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[1] We opted to use the MPQ–B as the self-report measure to assess the factors because the SRP-III does not map well onto the two-factor model (Williams, Paulhus, & Hare, 2007) and because Dvorak-Bertsch et al. (2009) used the MPQ–B to operationalize the dual-deficit model.

that lasted approximately 60 min. The PCL–R (Hare, 2003) contains 20 items that are rated 0, 1, or 2 according to the degree to which a characteristic is present: *significantly* (2), *moderately* (1), or *not at all* (0). Early work with the PCL–R revealed a replicable two-factor structure (Hare et al., 1990), with Factor 1 items assessing interpersonal–affective characteristics (e.g., glibness, callousness) and Factor 2 items relating to impulsive–antisocial behavior (e.g., irresponsible behavior, criminality). Factors 1 and 2 of the PCL–R are moderately intercorrelated. In this study, for the 18 participants randomly selected during the interview process for reliability ratings, the interrater reliability for the PCL–R total score, Factor 1, and Factor 2 was .96, .80, and .95, respectively. The internal consistency (Cronbach's alpha) of the PCL–R total score, Factor 1, and Factor 2 was .73, .70, and .57, respectively.

Hare Self-Report Psychopathy–III. Although the PCL–R is the gold standard in psychopathy assessment, self-report measures of psychopathy are also widely used and have yielded promising results (e.g., Brinkley, Schmitt, Smith, & Newman, 2001; Epstein et al., 2006; Lynam, Whiteside, & Jones, 1999; Miller & Lynam, 2003). Ninety-one participants completed the SRP-III, a 64-item self-report questionnaire that is intended to measure features of psychopathy similar to those assessed by the PCL–R. Items are scored on a 5-point Likert scale ranging from 1 (*disagree strongly*) to 5 (*agree strongly*). The SRP-III (Paulhus et al., in press) consists of four subscales: Criminal Tendencies, Erratic Life Style, Interpersonal Manipulation, and Callous Affect. In the present study, the SRP-III displayed good internal consistency (Cronbach's alpha = .85).

Multidimensional Personality Questionnaire—Brief. Ninety-one participants completed the MPQ–B (Patrick, Curtin, & Tellegen, 2002), a 155-item self-report questionnaire that consists of 11 primary trait scales. Designed to parallel the primary factors of the Psychopathic Personality Inventory (Lilienfeld & Andrews, 1996), the Fearless Dominance and Impulsive Antisociality scores of the MPQ–B are orthogonal to each other and correlate with Factors 1 and 2 of the PCL–R, respectively, in multiethnic samples (Benning et al., 2005; Benning, Patrick, Hicks, Blonigen, & Krueger, 2003). The Fearless Dominance and Impulsive Antisociality dimensions of psychopathy are calculated as linear combinations of specific standardized (i.e., z-scored) MPQ–B primary trait scales. Specifically, Fearless Dominance is calculated as $(0.34 \times z\text{Social Potency}) + (-0.42 \times z\text{Stress Reaction}) + (-0.21 \times z\text{Harm Avoidance})$. Impulsive Antisociality is calculated as $(0.16 \times z\text{Aggression}) + (0.31 \times z\text{Alienation}) + (-0.13 \times z\text{Traditionalism}) + (-0.29 \times z\text{Control}) + (-0.15 \times z\text{Social Closeness})$ (Benning et al., 2003). The internal consistency for each of the primary trait scales that contribute to Fearless Dominance are .71 (Social Potency), .79 (Stress Reaction), and .65 (Harm Avoidance). The internal consistency for each of the primary trait scales that contribute to Impulsive Antisociality are .84 (Aggression), .74 (Alienation), .61 (Traditionalism), .68 (Control), and .71 (Social Closeness).

Procedure

Shock sensitivity evaluation. The shock sensitivity protocol, which helped control for individual differences in sensitivity level, was conducted immediately prior to the start of the experimental task. Participants were given a series of electric shocks of increasing intensity to the fingers of their nondominant hand.[2] Participants reported two intensity anchors: the first was the intensity that they considered uncomfortable and the second was the maximum intensity level that they could tolerate. The series of electric shocks was terminated when the participant reached his maximum intensity level. The shock intensity administered during the experimental task was calibrated to the midpoint between each participant's discomfort level and their maximum intensity level.

Experimental task. During the task, participants viewed a series of letter cues. These stimuli were presented for 400 ms with a variable intertrial interval between 2.0 s and 2.8 s. The stimuli consisted of either upper- or lowercase letters and were colored either red or green. Participants were told that in all conditions, electric shocks might be administered on some trials following red letters (threat) but that no shocks would follow green letters (no threat). Shocks were administered for 200 ms to adjacent fingers on the participant's left hand at 1,400 ms after letter onset on 20% of threat trials in each condition, for a total of 30 shocks (10 shocks per condition).

The focus of attention for the participant varied depending on which of the three conditions he was performing. In the threat-focused condition, participants were instructed to attend to the color of the letter cue and to press one of two buttons using their right hand according to whether the letters indicated threat (red) or no threat (green). This condition was designed to focus participants on the feature of the letter cue (i.e., color) that indicated threat of shock. In the alternative-focus/low-load condition participants were asked to determine whether the letter cue was upper- or lowercase. The purpose of this condition was to make the threat information irrelevant and secondary to the primary task. In the alternative-focus/high-load condition, participants performed a two-back task (Jonides, Schumacher, Smith, & Lauber, 1997), where they had to attend to each letter in a series and press one of the two buttons to indicate whether that letter matched the letter presented two trials back in the series. As in the alternative-focus/low-load condition, letter color (threat information) became peripheral and was not necessary to perform this two-back task.

As noted above, in all three conditions, participants were instructed that electric shocks might be administered following letter cues colored in red but that no shocks would follow green letters. Participants performed two consecutive blocks of each of these three conditions, and condition order was fully counterbalanced across participants. To further enhance attention to the task-relevant features and to increase task motivation, we informed participants that speed and accuracy would influence the amount of shocks they received in the threat-focused condition and the amount of monetary reward earned in the alternative-focus/low-load and alternative-focus/high-load conditions. However, the number of shocks and earnings that participants actually received was not influenced by their behavioral performance.

These three task conditions were designed to provide discrete manipulations of attentional focus and working memory load. One of the conditions required the participant to focus on the threat information (threat focused), and the other two required an

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[2] With each analysis, we examined the effect of handedness. In all models, there was no main effect or interaction of handedness with psychopathy on fear-potentiated startle magnitude.

alternative focus of attention (i.e., threat-irrelevant focus: alternative focus/low load and alternative focus/high load). To the extent that a general fear deficit is an innate feature of psychopathy, psychopathic offenders should display deficient fear-potentiated startle in all three conditions. However, if their fearlessness is an attention-related deficit, then psychopathic offenders should display normal fear-potentiated startle in the threat-focused condition and deficiencies in the alternative-focus conditions.

Startle response elicitation and measurement. A total of 48 startle-eliciting noise probes (50-ms, 102-decibel white noise burst with near instantaneous rise time) were presented 1,400 ms after stimulus onset. The noise probes were equally distributed across threat/no-threat trials in all three task conditions so that each participant experienced 16 noise probes: eight red (threat) and eight green (safety) per condition. The average time between noise probes was 28.8 s, with a minimum of 13.7 s. Additionally, probes never occurred in the same trial as a shock administration. Startle eyeblink electromyographic activity was sampled at 2,000 Hz with a bandpass filter (30–500 Hz; 24 decibel/octave roll-off) from electrodes placed on the orbicularis oculi muscle under the right eye. Offline processing included epoching (-50 ms to 250 ms surrounding noise probe), rectification and smoothing (30-Hz low-pass filter following rectification), and baseline correction. Startle blink magnitude was scored as the peak response between 20 and 120 ms postprobe onset. Fear response to threat cues was indexed by fear-potentiated startle, calculated as the difference in blink response magnitude to probes following red (threat) compared with green (no-threat) trials in each of the three task conditions.

Results

As in the Newman et al. (2010) study, there was a significant main effect for condition, $F(2, 182) = 17.45$, $p < .01$, $\eta^2_{partial} =$

.161. Following Newman et al. (2010), we decomposed the omnibus interaction using two orthogonal (Helmert) interaction contrasts. The first examined the attention manipulation (threat focused vs. alternative focus conditions). This contrast revealed that fear-potentiated startle was significantly greater in the threat-focused condition than in the two alternative-focus conditions, $F(1, 91) = 24.72$, $p < .001$, $\eta^2_{partial} = .21$. The second interaction contrast compared the two alternative-focus conditions and revealed that fear-potentiated startle was significantly greater under low-load than under high-load conditions, $F(1, 91) = 7.67$, $p = .007$, $\eta^2_{partial} = .08$. Sample characteristics and descriptive statistics are presented in Table 1.

Psychopathy Checklist—Revised Total

Using SPSS (Version 16; SPSS, 2007), we analyzed fear-potentiated startle within a general linear model in which condition was a within-subject categorical factor and PCL–R total score was a between-subjects quantitative factor. As discussed above, we also decomposed the omnibus interaction using two orthogonal (Helmert) interaction contrasts designed to identify which task conditions might reveal relationships between psychopathy and fear-potentiated startle. Whereas the first interaction contrast examined the effects of the attention manipulation (threat focused vs. alternative focus conditions; first contrast) on fear-potentiated startle as a function of psychopathy, the second interaction contrast compared the two alternative-focus conditions to examine the effects of low compared with high cognitive load on fear-potentiated startle in psychopathy (alternative focus/low load vs. alternative focus/high load; second contrast). In opposition to findings obtained with European American offenders (see Newman et al., 2010), the Psychopathy $\times$ Condition interaction did not approach

Table 1
Means and Standard Deviations for Relevant Descriptive Variables

Variable	M	SD
Demographic		
Age	29.76	7.35
Education (no. of years)	10.24	1.45
Psychopathy assessment		
PCL–R total score	24.18	6.30
SRP-III total score	180.21	29.91
PCL–R Factor 1	8.70	2.95
PCL–R Factor 2	12.92	3.41
Fearless Dominance	−0.11	0.48
Impulsive Antisociality	0.01	0.57
Startle		
Threat focus: Green (no threat)	80.56	89.29
Threat focus: Red (threat)	110.81	110.50
Alternative focus/low load: Green (no threat)	86.79	98.42
Alternative focus/low load: Red (threat)	100.88	103.97
Alternative focus/high load: Green (no threat)	101.50	98.80
Alternative focus/high load: Red (threat)	103.16	96.74
Threat focus: Fear-potentiated startle	30.25	45.08
Alternative focus/low load: Fear-potentiated startle	14.09	37.72
Alternative focus/high load: Fear-potentiated startle	1.66	29.01

Note. PCL-R = Psychopathy Checklist—Revised; SRP-III = Hare Self-Report Psychopathy–III.

statistical significance in this study, $F(2, 180) = 0.30$, $p = .74$,[3] $\eta^2_{partial} = .003$.[4] Additionally, neither of the interaction contrasts was significant: first contrast: $F(1, 90) = 0.39$, $p = .53$, $\eta^2_{partial} = .004$; second contrast: $F(1, 90) = 0.19$, $p = .67$, $\eta^2_{partial} = .002$; that is, there was no difference between the threat-focused condition and the two alternative-focus conditions, nor was there a difference between the two alternative-focus conditions. In order to assess whether a general fear deficit was present, we examined the main effect of PCL–R psychopathy across conditions. This effect also was not significant, $F(1, 90) = 0.11$, $p = .75$, $\eta^2_{partial} = .001$. For the sake of completeness, we present the simple effects related to the PCL–R scores for each condition in Table 2.

Hare Self-Report Psychopathy–III

Similar to the results obtained with the PCL–R scores, the SRP-III × Condition interaction did not approach statistical significance, $F(2, 178) = 0.20$, $p = .82$, $\eta^2_{partial} = .002$. Additionally, neither of the interaction contrasts was significant: first contrast, $F(1, 89) = 0.03$, $p = .87$, $\eta^2_{partial} < .001$; second contrast, $F(1, 89) = 0.43$, $p = .51$, $\eta^2_{partial} = .005$. The main effect also was not significant, $F(1, 89) = 0.36$, $p = .55$, $\eta^2_{partial} = .004$. The simple effects for all three conditions are presented in Table 2.

Psychopathy Checklist—Revised Factors 1 and 2

Using a separate general linear model for each factor, we found that paralleling results for the psychopathy total score analyses, neither the Factor 1 × Condition interaction, $F(2, 180) = 0.84$, $p = .43$, $\eta^2_{partial} = .009$, nor the Factor 2 × Condition interaction, $F(2, 180) = 0.21$, $p = .82$, $\eta^2_{partial} = .002$, approached statistical significance.[5] Additionally, neither of the interaction contrasts was significant: for Factor 1, first contrast, $F(1, 90) = 1.48$, $p = .23$, $\eta^2_{partial} = .016$; second contrast, $F(1, 90) = 0.004$, $p = .95$, $\eta^2_{partial} < .001$; for Factor 2, first contrast, $F(1, 90) = 0.30$, $p = .57$, $\eta^2_{partial} = .003$; second contrast, $F(1, 90) = 0.08$, $p = .78$, $\eta^2_{partial} = .001$. Moreover, the main effects for Factor 1 and Factor 2, respectively, were not significant: for Factor 1, $F(1, 90) = 2.10$, $p = .15$, $\eta^2_{partial} = .023$; for Factor 2, $F(1, 90) = 0.00$, $p = .99$, $\eta^2_{partial} < .001$. In this analysis, we particularly targeted examination of the simple effects of the factors in each condition because of the dual-deficit model's focus on cognitive deficiencies as the crucial factor undermining emotion processing in individuals with high PCL–R Factor 2 scores (Patrick, 1994, 2007). However, none of the simple effects of Factor 2 was significant. Of the PCL–R factor comparisons presented in Table 2, only one—the association between PCL–R Factor 1 and the alternative-focus/high-load condition—was significant ($\beta_{AF/HL} = -.207$, $p = .048$, $\eta^2_{partial} = .043$; see Table 2).

Multidimensional Personality Questionnaire—Brief: Fearless Dominance and Impulsive Antisociality Factors

Neither the main effect nor the interaction involving Fearless Dominance was statistically significant, $F(1, 89) = 0.36$, $p = .55$, $\eta^2_{partial} = .004$, and $F(2, 178) = 1.31$, $p = .27$, $\eta^2_{partial} = .015$, respectively.[5] Additionally, neither of the interaction contrasts was

significant: first contrast, $F(1, 89) = 2.16$, $p = .15$, $\eta^2_{partial} = .024$; second contrast, $F(1, 89) = 0.18$, $p = .67$, $\eta^2_{partial} = .002$. Similarly, neither the main effect, the interaction, nor the interaction contrasts involving Impulsive Antisociality was significant: main effect, $F(1, 89) = 1.19$, $p = .28$, $\eta^2_{partial} = .013$; interaction, $F(2, 178) = 0.25$, $p = .78$, $\eta^2_{partial} = .003$; first contrast, $F(1, 89) = 0.12$, $p = .73$, $\eta^2_{partial} = .001$; second contrast, $F(1, 89) = 0.41$, $p = .52$, $\eta^2_{partial} = .005$. The simple effects for Fearless Dominance and Impulsive Antisociality for each condition are presented in Table 2; however, none of the effects were significant.

Discussion

The purpose of this study was to examine the generalizability of psychopathy-related fear deficits to African American offenders. Regardless of whether psychopathy was assessed by interview or by self-report or analyzed as a unitary construct or as discrete factors, the fear deficit predicted by the response modulation (Newman et al., 2010), the amygdala dysfunction (Blair et al., 2005), and dual-deficit (Patrick, 2007) models was not present in this sample of African American inmates. Though inconsistent with theory-based predictions, these results are generally consistent with previous attempts to replicate established laboratory correlates of psychopathy in African American offenders. In contrast to findings for European American offenders that document reliable deficiencies in passive avoidance learning (e.g., Blair et al., 2004; Newman & Kosson, 1986), emotion facilitation in the lexical decision paradigm (e.g., Lorenz & Newman, 2002b; Williamson, Harpur, & Hare, 1991), and fear-potentiated startle (e.g., Levenston, Patrick, Bradley, & Lang, 2000; Newman et al., 2010), these etiologically relevant findings do not appear to be psychopathy-related correlates in African American offenders (Kosson, Smith, & Newman, 1990; Lorenz & Newman, 2002a; Newman & Schmitt, 1998; Thornquist & Zuckerman, 1995).

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[3] Some researchers have suggested using standardized transformations of the startle response as opposed to the raw blink magnitude. Standardization is thought to control for individual differences in overall reactivity and habituation across trial blocks. However, there are no clear recommendations for how standardization should be done, and many laboratories do not regularly standardize startle responses. Despite this, we did reanalyze our data after applying a z-transformation to minimize individual differences, and all of these effects remain unchanged.

[4] In response to a reviewer's request, we combined the data for this African American sample with those of a European American sample (from Newman et al., 2010) in order to evaluate the Psychopathy × Race interaction for fear-potentiated startle. In fact, this interaction was not significant, indicating that the PCL–R effect in African American offenders is not significantly different from the one reported by Newman et al. (2010).

[5] To examine the unique effects of each factor, we also analyzed Factor 1 and Factor 2 in the same general linear model; neither the unique variance of Factor 1 nor that of Factor 2 significantly predicted fear-potentiated startle: for Factor 1, $F(2, 178) = 0.69$, $p = .50$, $\eta^2_{partial} = .008$; for Factor 2, $F(2, 178) = 0.06$, $p = .94$, $\eta^2_{partial} = .001$. Similarly, to examine the unique effects of Fearless Dominance and Impulsive Antisociality, we also analyzed both MPQ–B factors in the same general linear model; neither the unique variance of Fearless Dominance nor that of Impulsive Antisociality significantly predicted fear-potentiated startle: for Fearless Dominance, $F(2, 176) = 1.28$, $p = .28$, $\eta^2_{partial} = .014$; for Impulsive Antisociality, $F(2, 176) = 0.23$, $p = .79$, $\eta^2_{partial} = .003$.

Table 2
Simple Effects (Standardized Betas) of Each Psychopathy-Related Variable in All Three Conditions

Variable	Threat focused				Alternative focus/low load				Alternative focus/high load			
	ß	SE	t	$\eta^2_{partial}$	ß	SE	t	$\eta^2_{partial}$	ß	SE	t	$\eta^2_{partial}$
PCL–R total	.022	4.75	0.21	<.001	−.023	3.98	−0.22	.001	−.098	3.04	−0.93	.010
SRP-III total	.048	4.79	0.46	.002	.077	4.01	0.73	.006	−.002	3.09	−0.02	<.001
PCL–R Factor 1	−.008	4.75	−0.08	<.001	−.151	3.93	−1.45	.023	−.207*	2.99	−2.01	.043
PCL–R Factor 2	.036	4.75	0.34	.001	−.006	3.98	−0.06	<.001	−.051	3.05	−0.49	.003
Fearless Dominance	.061	4.80	0.58	.004	−.078	4.01	−0.73	.006	−.170	3.01	−1.62	.029
Impulsive Antisociality	.044	4.80	0.42	.002	.134	3.98	1.27	.018	.074	3.04	0.70	.006

Note. PCL–R = Psychopathy Checklist—Revised; SRP-III = Self-Report Psychopathy–III.
* $p < .05$.

There is increasingly compelling evidence that the major etiologically relevant correlates of psychopathy fail to replicate in African American offenders. However, such findings do not necessarily mean that the effects for African Americans and European Americans are significantly different in these studies. In fact, none of these studies, including the present one, have reported significant race by psychopathy interactions (see Footnote 4). Moreover, when investigators have explicitly examined race by psychopathy interactions, they have generally found them to be nonsignificant (e.g., Kosson et al., 2007; Suchy & Kosson, 2005; see also Epstein et al., 2006). The absence of such interactions represents an important limitation regarding the conclusions that may be drawn from this and other studies that fail to support the generalizability of psychopathy correlates in African American offenders.

Regardless of whether race-related differences are demonstrated for particular correlates, there are pragmatic reasons to evaluate the generalizability of psychopathy-related effects in African American samples explicitly. Without explicitly evaluating the generalizability of particular correlates, it is difficult to be confident when making inferences about the causal factors and treatment needs associated with psychopathy in African American offenders. In other words, the fact that studies investigating central theory-based aspects of psychopathy in African American samples have not achieved statistical significance calls into question the validity of assumptions that associate these core characteristics with psychopathy in African American offenders.

In the present study, the majority of the psychopathy-related effects were essentially nonexistent. When a relationship such as the one between psychopathy and fear-potentiated startle in African American offenders is not statistically significant, it may reflect an insufficient sample size, excessive variability, or the absence of a meaningful relationship. As noted in the Method section, the current study had adequate power to detect an effect. However, examination of the effect size and standard errors of the current sample (see Table 2) revealed substantially more variability in the African American sample than was found in the European American sample (see Newman et al., 2010). Such variability not only reduces the odds of finding significant psychopathy effects but also undermines the likelihood of finding significant psychopathy by race interactions (i.e., race-related differences). Further research is needed to evaluate the reliability of this observation and, if it is reliable, to identify methods for reducing the variability of psychopathy effects in African American offenders.

For example, the primary versus secondary psychopathy distinction has often been used with success to reduce variability in psychopathy-related correlates (e.g., Brinkley, Newman, Widiger, & Lynam, 2004; Hicks, Markon, Patrick, Krueger, & Newman, 2004; Skeem, Johansson, Andershed, Kerr, & Louden, 2007). Analogously, it may be possible to identify some variable that moderates the effects of psychopathy in African American samples and reduces the variability associated with psychopathy main effects.

An additional goal of this study was to evaluate a potential source for the failure to replicate laboratory correlates of psychopathy in African American offenders by assessing psychopathy with different methods (i.e., interview vs. self-report). To date, most studies investigating the generalizability of laboratory correlates of psychopathy in African American offenders have used the PCL–R. Thus, it is possible that the null findings reflect race differences in the efficacy of the PCL–R (Hemphill & Hart, 2003; Lorenz, Smith, Bolt, Schmitt, & Newman, 2001) rather than differences in the psychopathy construct per se. The potential validity of this concern is strengthened by the fact that PCL–R scores are based on interviews and thus the interviewers' evaluation of the construct. Of note, the internal consistency of PCL–R scores, and specifically of Factor 2 scores, was lower than those typically found for European American or racially mixed samples (Hare, 2003). Further research is required to determine whether this finding is particular to the current study or is a more general characteristic of PCL–R ratings in African American offenders. One strategy for addressing this issue was to employ self-report measures of psychopathy with the same sample to clarify whether established psychobiological correlates of psychopathy in European Americans would be replicated when alternative measures of psychopathy were used. Despite the good internal consistency of the self-report measures of psychopathy (i.e., SRP-III), we found no evidence that the SRP-III self-report measure of psychopathy performed differently from the PCL–R in terms of identifying a fear deficit in African American psychopathic individuals.

Another concern with the assessment of psychopathy relates to the unitary versus multidimensional nature of psychopathy. In past research, the interpersonal–affective features of psychopathy (Factor 1) were inversely related to startle potentiation in a picture-viewing paradigm, whereas the antisocial–impulsive features of psychopathy (Factor 2) were positively related to startle potentiation (e.g., Vanman, Mejia, Dawson, Schell, & Raine, 2003). In

cases such as this, analysis of the factor-level data may reveal significant relationships that may not be found with PCL–R total scores. Thus, we examined factor, as well as total, scores in this investigation. Here too, however, our analyses provided minimal support for predictions based on previous research with European American offenders. None of the a priori predictions for main effects or interaction contrasts were statistically significant. Neither PCL–R Factor 2 nor MPQ–B Impulsive Antisociality was associated with the pattern of fear-potentiated startle predicted by the dual-deficit model. However, in the interest of providing an exhaustive analysis of the evidence, we examined the relationship between PCL–R Factor 1 and fear-potentiated startle in a post hoc analysis involving the alternative-focus/high-load condition and found a significant association. Given the number of comparisons examined (i.e., 42) and the post hoc nature of this analysis, there is reason to be cautious in interpreting this result. Nevertheless, it is noteworthy that a similar, though only trend-level, effect was found for MPQ–B Fearless Dominance. Past investigations of fear-potentiated startle differences associated with psychopathy have highlighted the primary importance of Factor 1 traits (e.g., Patrick et al., 1993; Vanman et al., 2003), but, to our knowledge, this is the first study to show that the emergence of such effects may depend on high levels of attentional/working-memory load.

Last, previous research has suggested that African American individuals may react differently to the laboratory situation (Steele & Aronson, 1995). Specifically, stereotype threat may influence expectations, perceptions of the laboratory setting, and performance differently in African Americans than in European Americans. Therefore, we are unable to discern whether our failure to find evidence of a fear deficit in African American offenders means that psychopathy scores in this sample reflect different etiological processes or differences in the understanding of and reaction to the laboratory assessments.

In addition to the theoretical implications of failing to replicate core etiological correlates in African American samples, there are equally important implications for assessment and treatment. Because the emotion deficits explored in this investigation did not replicate in African American inmates, it seems likely that the utility of such deficits for understanding and treating the destructive behaviors associated with psychopathy in African American offenders will likewise be limited. Taken together, these findings suggest that it is crucial for future research to explore and clarify the reasons why well-replicated etiologically relevant correlates of psychopathy in European American samples are not replicated in African American samples and, if warranted by the results, to either develop better measures of psychopathy or identify the distinctive etiological mechanisms that underlie psychopathic behavior in African Americans.

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