

Attention Moderates the Fearlessness of Psychopathic Offenders

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Background: Psychopathic behavior is generally attributed to a fundamental, amygdala-mediated deficit in fearlessness that undermines social conformity. An alternative view is that psychopathy involves an attention-related deficit that undermines the processing of peripheral information, including fear stimuli.

Methods: We evaluated these alternative hypotheses by measuring fear-potentiated startle (FPS) in a group of 125 prisoners under experimental conditions that 1) focused attention directly on fear-relevant information or 2) established an alternative attentional focus. Psychopathy was assessed using Hare's Psychopathy Checklist-Revised (PCL-R).

Results: Psychopathic individuals displayed normal FPS under threat-focused conditions but manifested a significant deficit in FPS under alternative-focus conditions. Moreover, these findings were essentially unchanged when analyses employed the interpersonal/affective factor of the PCL-R instead of PCL-R total scores.

Conclusions: The results provide unprecedented evidence that higher-order cognitive processes moderate the fear deficits of psychopathic individuals. These findings suggest that psychopaths' diminished reactivity to fear stimuli, and emotion-related cues more generally, reflect idiosyncrasies in attention that limit their processing of peripheral information. Although psychopathic individuals are commonly described as cold-blooded predators who are unmotivated to change, the attentional dysfunction identified in this study supports an alternative interpretation of their chronic disinhibition and insensitive interpersonal style.

Key Words: Amygdala, attention, conditioning, emotion, fear, psychopathy

Psychopaths are infamous for using charm, manipulation, and violence to control others and satisfy their own selfish needs (1). According to Lykken (2), prototypical psychopathy reflects an innate deficiency in fearfulness that is not in itself evil or vicious but can result in a dangerous syndrome when combined with perverse appetites or an unusually aggressive temperament. Supporting this low-fear hypothesis, psychopathic offenders display poor fear conditioning (3), weak electrodermal responses in anticipation of aversive events such as loud noises or electric shocks (4), poor passive avoidance learning (3,5), and a lack of startle potentiation while viewing unpleasant versus neutral pictures (6). Moreover, there is preliminary evidence that psychopathic offenders display less amygdala activation than control subjects during aversive conditioning procedures (7). Theorists have interpreted this evidence to indicate that primary psychopathy involves "a basic deficit in fear reactivity, i.e., reduced defensive reactivity to aversive stimuli that are direct and explicit" ([8] p. 70) and that such data "strongly indicate amygdala dysfunction in individuals with psychopathy" ([9] p. 138).

Newman's response modulation theory provides an alternative view of psychopathy (10,11). According to this theory, psychopaths' fear conditioning deficits, as well as their other behavior and emotion deficits, reflect a failure to process affective, inhibitory, and other potentially important information when it is peripheral to their ongoing goal-directed behavior (10,11). Relative to the low-fear hypothesis, the response mod-

ulation hypothesis is more specific because it predicts situation-specific rather than pan-situational fear deficits and more comprehensive because the attention-moderated deficits transcend fear responses (11). Supporting this view, psychopathic offenders display poor passive avoidance and weak electrodermal responses to punishment cues while focused on earning rewards, yet show no deficits on the same measures when avoidance learning is their primary goal (5,12). Moreover, there is increasing evidence that higher-order cognitive processes, like attention and working memory, moderate amygdala-mediated responses to emotional cues (13–16). To date, however, there is no direct evidence that psychopaths' fear responses to physical threats (e.g., electric shocks) vary as a function of their goal-directed behavior.

To evaluate these competing perspectives on psychopathy, we examined fear-potentiated startle (FPS) in a sample of incarcerated psychopathic and nonpsychopathic offenders under experimental conditions that 1) focused attention directly on fear-relevant information or 2) established an alternative attentional focus under low or high cognitive load. Fear-potentiated startle has played a critical role in probing the neurocircuitry of fear (17,18). The startle response is significantly potentiated when elicited in the presence of a stimulus that has been paired with electric shock. Fear-potentiated startle is reduced by anxiolytic drugs, increased by anxiogenics, and enhanced among patients with anxiety-related psychiatric disorders (18–20). Moreover, under most circumstances, FPS is mediated via the amygdala ([18,21,22] cf. [23]).

The primary difference between the low-fear and response modulation theories of psychopathy relates to the pan-situational versus situation-specific nature of the fear deficit. To the extent that low fear or a weak defensive response to aversive stimuli is an inherent feature of psychopathy, psychopathic offenders should display deficient FPS in all three conditions. However, if their fearlessness is an indirect manifestation of deficient response modulation, then psychopathic offenders should display normal FPS in the threat-focused condition and deficiencies in the alternative-focus conditions.

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Methods and Materials

Participants

Participants were 125 white male prisoners between the ages of 18 and 45. Participants were excluded if they were age 45 or older; currently prescribed psychotropic medication; had clinical diagnoses of schizophrenia, bipolar disorder, or psychosis not otherwise specified (NOS); scored below the fourth grade reading level; or had estimated IQ scores of less than 70 on the Shipley Institute of Living Scale (SILS) (24).

Psychopathy was assessed using the Psychopathy Checklist-Revised (PCL-R) (25). Information for the ratings was derived from a semi-structured interview and file review. The PCL-R contains 20 items that are rated 0, 1, or 2, according to the degree to which a characteristic is present. A wealth of evidence supports the reliability and validity of the PCL-R (25). Interrater reliability for the 29 participants with dual PCL-R ratings was .917. Other relevant assessments were conducted and analyzed as noted in Supplement 1. Participants were recalled for psychophysiological testing 1 or 2 weeks after the interview.

Procedure

Presentation of stimuli and recording of responses were controlled by DMDX (<http://www.u.arizona.edu/~kforster/dmdx/dmdx.htm>) (26) and NeuroScan Synamps2 amplifiers and acquisition software (Compumedics, Charlotte, North Carolina). All participants were tested by one of four male experimenters. Prior to beginning the experiment, the intensity of shocks received during the experimental session was calibrated to a participant’s subjective shock sensitivity (see Supplement 1).

Experimental Task: Instructed Fear-Conditioning Paradigm

During the instructed fear-conditioning paradigm, participants viewed a series of letter cues. Stimuli were presented for 400 msec with a variable intertrial interval between 2 sec and 2.8 sec. Letter cues were either upper or lower case and colored 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 msec to adjacent fingers on the participant’s left hand at 1400 msec poststimulus onset on 20% of threat trials in each condition (10 shocks per condition).

To contrast the predictions of the low-fear and response modulation theories, we measured FPS under experimental conditions that made threat processing the primary focus of goal-directed behavior or peripheral to goal-directed behavior (Figure 1). In the threat-focused condition, participants’ attention was focused on the threats by requiring them to indicate whether letters indicated threat (red) or no-threat (green) by pressing one of two buttons on each trial. There were two alternative-focus conditions. In the low-load alternative-focus condition, participant responses indicated whether letters were upper or lower case. In the high-load alternative-focus condition, participants were instructed to monitor the sequence of letters and indicate whether each letter matched or mismatched the letter that appeared two letters back.

Startle Response Elicitation and Measurement

Forty-eight startle-eliciting noise probes (50 msec, 102 dB white noise bursts) were presented 1400 msec after letter onset. Probes were equally distributed across threat/no-threat trials in

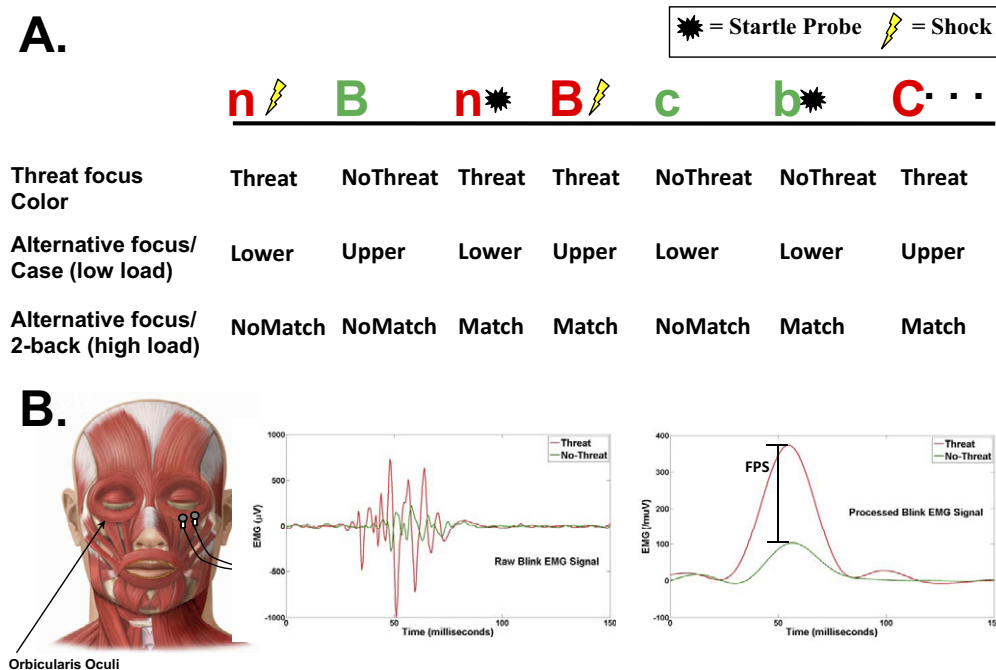


Figure 1. Schematic of task and fear-potentiated startle measurement. (A) Participants view a series of 400 msec upper/lower case letter stimuli colored red/green. In all three conditions, electric shocks are administered after some red but not green letters. In threat-focused condition, participants respond (via two buttons) to indicate letter color. In alternative focus/low load, participants respond to indicate letter case. In alternative focus/high load, participants respond to indicate letter match between current letter and letter 2-back. White noise “startle probes” are presented following letter stimuli to measure fear-potentiated startle (FPS). (B) The eyeblink component of the startle reflex is measured via electromyographic (EMG) activity in the orbicularis oculi muscle. Raw blink EMG activity is elicited by 50-msec startle probes during threat and no-threat trials. This raw signal is rectified and smoothed (30 Hz low pass filter). FPS is calculated as the difference between peak EMG response on threat versus no-threat trials.

all three conditions so that participants experienced 16 noise probes (8 threat, 8 no-threat) per condition. Probes never occurred in the same trial as shock administration. Startle eye-blink electromyographic activity was sampled at 2000 Hz with a bandpass filter (30–500 Hz; 24 dB/octave roll-off) from electrodes placed on the orbicularis oculi muscle under the right eye. Startle blink magnitude was scored as the peak response between 20 msec and 120 msec postonset of probe. Fear response to threat cues was indexed by FPS, calculated as the difference in blink-response magnitude to probes following threat versus no-threat trials in each of the three task conditions.

Results

We analyzed fear-potentiated startle in a general linear model with condition as a within-subject categorical factor and psychopathy total score as a between-subject quantitative factor. As predicted by the response modulation theory, the relationship between psychopathy and FPS was moderated by condition (i.e., psychopathy \times condition interaction), $F(2,246) = 3.77, p = .026$. Follow-up tests of orthogonal interaction contrasts indicated that psychopathy interacted with the focus of attention (i.e., threat vs. alternative focus), $F(1,123) = 5.28, p = .023, B = -5.8$. This critical interaction contrast demonstrating strikingly different psychopathy effects on fear across threat-focused versus alternative-focus conditions is displayed in Figure 2. A second interaction contrast examining the effects of high versus low cognitive load on psychopathy was not significant (Table 1 shows raw startle responses as a function of cue type, condition, and psychopathy).

Follow-up simple effect tests for the significant attentional focus interaction contrast indicated that the relationship between psychopathy and FPS was not significant in the threat-focused condition, $B = .7, p = .800$. In fact, prisoners with high psychopathy scores (point estimate at 1.5 SD above mean) displayed descriptively greater FPS ($M = 27.2 \mu\text{V}$) than prisoners with low psychopathy scores (1.5 SD below mean; $M = 25.2 \mu\text{V}$). Thus, results for the threat-focused condition provided no evidence that psychopaths displayed reduced defensive system reactivity when their attention was focused on threat-relevant cues.

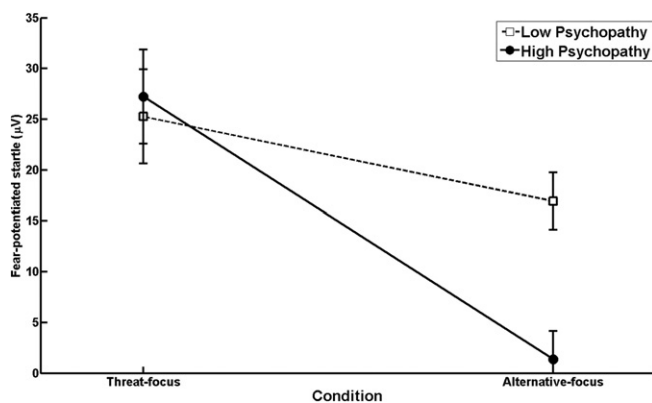


Figure 2. Fear-potentiated startle by psychopathy and condition. Focus of attention significantly moderated the psychopathy effect on fear-potentiated startle (FPS). High psychopathy prisoners displayed significantly lower FPS than low psychopathy prisoners in the alternative-focus conditions. High and low psychopathy prisoners displayed comparable FPS in the threat-focus condition. FPS was calculated as startle response during red/threat minus green/neutral letter trials. FPS means displayed for low and high psychopathy were calculated at 1.5 standard deviations below and above the sample mean on psychopathy total scores, respectively. Error bars represent the standard error for the point estimate.

Table 1. General Linear Model Point Estimates (and standard errors) for Startle Response Associated with No-Threat and Threat Cues as a Function of PCL-R Psychopathy Total Score and Task Condition

	Threat-Focused Condition		Alternative-Focus Conditions	
	No-Threat	Threat	No-Threat	Threat
Low Psychopathy	69.5 (10.0)	94.8 (12.4)	84.7 (12.2)	101.6 (12.3)
Mean Psychopathy	73.6 (5.5)	99.9 (6.8)	89.0 (6.7)	98.1 (6.8)
High Psychopathy	77.7 (10.0)	104.9 (12.4)	93.2 (12.2)	94.6 (12.3)

Point estimates are provided for low psychopathy (1.5 standard deviations below sample mean PCL-R total score), mean psychopathy, and high psychopathy (1.5 standard deviations above sample mean PCL-R total score).

PCL-R, Psychopathy Checklist-Revised.

Furthermore, as predicted by the response modulation theory, psychopathy was significantly inversely related to FPS in the alternative-focus conditions, $B = -5.2, p = .001$. Prisoners with high psychopathy scores displayed significantly lower FPS ($M = 1.4 \mu\text{V}$) than those with low scores ($M = 16.9 \mu\text{V}$). In fact, FPS for participants with high psychopathy scores was essentially eliminated in the alternative-focus conditions (Figure 2). That is, FPS was no longer significantly greater than zero ($p = .628$). Overall, the results provide clear evidence that psychopathy is associated with deficits in defensive system reactivity but such deficits are observed only when their attentional and cognitive resources are allocated elsewhere (Supplement 1).

Because past research links FPS differences in psychopathy specifically to the interpersonal and affective symptoms that distinguish psychopathy from other antisocial syndromes (i.e., PCL-R factor 1 [6]), we reanalyzed the attentional focus interaction contrast using factor 1 rather than psychopathy total scores. Paralleling results for psychopathy total scores, the factor 1 \times attentional focus interaction contrast was also significant, $F(1,123) = 3.96, p = .049$ (Figure 3). The inverse relationship between factor 1 scores and FPS was significant in the alternative-focus conditions ($B = -4.0, p = .013$) and not significant in

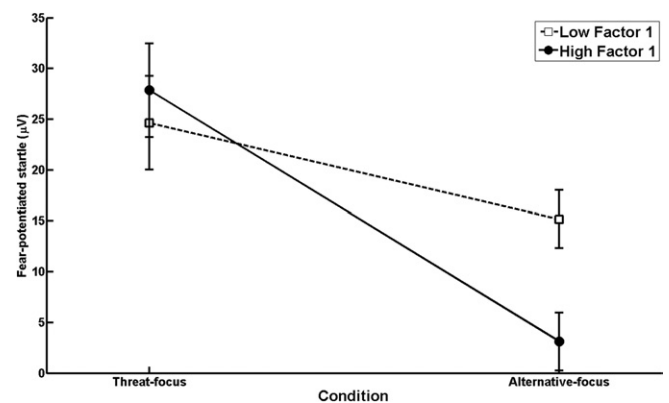


Figure 3. Fear-potentiated startle by factor 1 of the psychopathy checklist and condition. Focus of attention significantly moderated the factor 1 effect on fear-potentiated startle (FPS). High factor 1 prisoners displayed significantly lower FPS than low factor 1 prisoners in the alternative-focus conditions. High and low factor 1 prisoners displayed comparable FPS in the threat-focus condition. FPS was calculated as startle response during red/threat minus green/neutral letter trials. FPS means displayed for low and high factor 1 were calculated at 1.5 standard deviations below and above the sample mean on factor 1 scores, respectively. Error bars represent the standard error for the point estimate.

the threat-focused condition ($B = 1.1, p = .676$). Notably, results for factor 2 were highly similar to those for factor 1. Thus, regardless of whether we analyzed psychopathy total scores or its primary factors, the apparent conclusion is the same—psychopaths display deficient fear responses when they are engaged in threat-irrelevant goal-directed behavior, though not when their attention is focused on threat-relevant information.

Supplementary Analyses for the Threat-Focused Condition

Some psychopathy researchers are skeptical about findings for PCL-R total scores, especially when those findings appear to refute deficits in fear or anxiety, because the multifactorial nature of psychopathy may obscure such associations (e.g., when the associations between the potential correlate and psychopathy subfactors are opposite in direction [8]). To address such concerns with regard to the absence of psychopathy-related differences in the threat-focused condition, Table 2 presents FPS data for all facets of the two- and four-factor models. Only one of the 14 variables examined (i.e., facet 2a: impulsive lifestyle) approached statistical significance ($p = .062$).

Discussion

The innate fearlessness of psychopathic individuals is arguably the most sacrosanct assumption in the field of psychopathy. Moreover, psychopathy-related deficits in FPS are generally regarded as the most compelling evidence for this longstanding supposition (2,27). In light of the fact that psychopathy-related differences in FPS were moderated by higher-order processes and no deficit in FPS was observed under threat-focused condition, the present findings call to question these central tenets in the field of psychopathy.

Regarding the lack of psychopathy-related differences in the threat-focused condition, it is worth noting that this is the first study to use electric shocks to elicit fear with PCL-assessed participants. Thus, it may be that the fear deficits associated with psychopathy are most apparent under low threat conditions and disappear when threat is sufficiently strong. However, the same shock intensity was used in the threat-focused and alternative-focus conditions with dramatically different results. Such findings strongly suggest that attentional focus rather than the intensity of

the unconditioned stimulus is responsible for the observed differences. Supporting this conclusion, similar findings have been obtained using loss of money rather than electric shocks. For instance, psychopathic offenders display normal response inhibition when avoiding monetary punishments is their only goal, even though they fail to inhibit punished responses while focused on earning monetary rewards (5). Similarly, psychopathic offenders display normal electrodermal responses to the onset of red lights signaling increased risk of monetary punishment when focused on avoiding punishments, even though their electrodermal responses to the same lights are significantly smaller than those of nonpsychopathic control subjects when focused on earning rewards (12). Combined, these findings suggest that psychopaths' normal response to fear cues in the threat-focused condition stems from the fact that the threat information in this condition was performance-relevant and thus intrinsic to their ongoing goal-directed behavior.

There is rapidly growing evidence that higher-order processes like those associated with our alternative focus conditions moderate emotion responses, even at the level of amygdala activation (13–16). Moreover, the results of this study demonstrate that the fear responses of psychopathic offenders are moderated by higher-order processes to an abnormal degree. However, the implications of the present findings for amygdala functioning in psychopathy are limited by the fact that we studied instructed fear conditioning rather than the acquisition of fear conditioning. While there is strong evidence that intact amygdala functioning is crucial for the acquisition of conditioned fear responses, recent research with nonhuman primates shows that it is possible to display normal FPS to previously acquired conditioned fear stimuli despite essentially complete damage to the amygdala (23). Thus, the normal FPS shown by psychopathic individuals in our threat-focused condition is not incompatible with proposals regarding amygdala dysfunction in psychopathy.

To provide more conclusive evidence that the fear-conditioning deficits associated with psychopathy reflect abnormal attentional modulation of amygdala activation rather than amygdala dysfunction per se, future research should include a fear acquisition phase rather than informing participants about the operational contingencies. Moreover, in light of the increasing evi-

Table 2. Association Between PCL-R Factors/Facets and FPS in the Threat-Focused Condition

Factors	Unique Effect			Total Effect		
	B_p	SE	p Value	B_{tot}	SE	p Value
PCL-R Total						
PCL-R	.7	2.6	.800	.7	2.6	.800
PCL-R Two Factor Model						
PCL-F1	.6	2.9	.844	1.1	2.6	.676
PCL-F2	1.1	2.9	.703	1.4	2.6	.596
PCL-R Four Facet Model						
Facet 1a	3.2	3.0	.286	1.9	2.6	.451
Facet 1b	-.3	2.8	.927	.3	2.6	.897
Facet 2a	-5.7	3.0	.062	-2.8	2.6	.282
Facet 2b	4.5	2.8	.111	3.5	2.6	.170

$n = 125$.

All predictors were standardized ($M = 0; SD = 1$). All B 's indicate the change in FPS (in microvolts) associated with a one standard deviation increase in the predictor. B_p indicates the unique/partial effect of each predictor (holding other predictors in the model constant). B_{tot} indicates the total effect of the predictor.

Scale labels from PCL-R: Factor 1: The callous, selfish, remorseless use of others. Facet 1a: Interpersonal. Facet 1b: Affective. Factor 2: A chronically unstable and antisocial lifestyle. Facet 2a: Impulsive Lifestyle. Facet 2b: Antisocial Behavior.

FPS, fear-potentiated startle; PCL-R, Psychopathy Checklist-Revised.

dence that deficiencies in brain activation are not specific to amygdala but often involve widespread deficiencies in paralimbic activation (28), neuroimaging methods should be used to examine whether attention moderates the paralimbic activation deficits associated with psychopathy more generally.

Given the use of an instructed fear paradigm, our results are not necessarily inconsistent with models of psychopathy that postulate amygdala dysfunction (e.g., [23]). It could be argued, for instance, that amygdala dysfunction would be particularly relevant for processing weak or peripheral threats as opposed to threats that are made salient by verbal communication or previous conditioning. To the extent that the fear responses of psychopathic individuals are mediated by verbal or memory processes as opposed to amygdala activation, it follows that they would be more vulnerable to disruption when psychopaths are engaged in attention-demanding tasks. Although this proposal could account for the significant condition effect observed in this study, it may not be the most parsimonious explanation for the findings. Newman *et al.* (29), Hiatt *et al.* (30), and Zeier *et al.* (31) have demonstrated that such attention manipulations also moderate psychopaths' sensitivity to affectively neutral information, which, presumably, is less dependent upon amygdala activation. To the extent that these attention-related effects reflect the same neural underpinnings in psychopathic individuals, it would be important to identify neurobiological mechanisms that could account for such phenomena in affectively neutral as well as affectively charged situations.

The results of this study provide the strongest evidence to date that a deficit in response modulation rather than reduced sensitivity to punishment cues *per se* is the crucial factor limiting fear responses in psychopathic individuals (Supplement 1). These findings reinforce the proposal that psychopaths' diminished reactivity to fear stimuli, and emotion-related cues more generally, reflect idiosyncrasies in attention that limit their processing of peripheral information (11). To the extent that such information supplies crucial context for interpreting events, decision making, interpersonal interactions, and self-regulation, psychopathic individuals would lack this perspective. Although psychopathic individuals are commonly described as cold-blooded predators that are unmotivated to change, the present findings identify an attentional dysfunction that may account for their chronic disinhibition and insensitive interpersonal style and provide a meaningful target for early clinical intervention.

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Supplementary material cited in this article is available online.

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