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Does neuroticism in adolescents moderate contextual and explicit threat cue modulation of the startle reflex?

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Abstract

Background—This study evaluated the relationship between neuroticism (N), a probable risk factor for emotional disorders, and modulation of startle reflexes (SRs).

Methods—132 adolescents with varying levels of N, but without anxiety or depressive disorders, were evaluated in contextual cue and explicit threat cue paradigms.

Results—Within the explicit threat cue paradigm, N potentiated SRs more in conditions that were intermediately associated with threat of an aversive biceps contraction than conditions that were the furthest from and conditions that were the closest to the same threat. Also, N potentiated SRs across the entire experiment, regardless of experimental conditions, in males and not in females.

Discussion—These results suggest that adolescents with high levels of N show greater sensitivity to contexts intermediately associated with threat. Results are discussed in comparison to other studies of groups at risk for anxiety and depressive disorders.

Keywords

Startle reflexes; context; explicit threat; risk for anxiety and depression; adolescents

The magnitude of the human startle reflex (SR) indexes defensive responding to aversive stimuli and associated contexts, and is potentiated by fear and anxiety (1). Numerous studies of individuals with anxiety disorders have demonstrated sustained elevations in “baseline” SRs throughout experiments involving delivery of aversive electric shocks signalled by a cue (2,

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3,4). However, SRs to explicit threat cues were not more elevated in clinically anxious individuals than in non-anxious controls, and baseline SR was not elevated in clinically anxious individuals when informed that aversive stimuli would not be presented (2). Thus, sustained *contextual anxiety* (i.e., elevated baseline SR in contexts associated with aversive stimuli), but not *explicit threat cue anxiety*, is believed to differentiate individuals with and without anxiety disorders (5). The distinction between SR modulation to contextual versus explicit threat cues is supported by neuroanatomical evidence in rodents, where modulation of SRs by aversive contexts eliciting anxiety is mediated by the bed nucleus of the stria terminalis, whereas modulation of SRs by explicit threat cues eliciting fear is mediated by the central nucleus of the amygdala (6,7).

Similar SR modulation results have been documented in female (but not male) youths at risk for anxiety disorders by virtue either of parental anxiety (8) or grandparental major depressive disorder (9). The personality trait of neuroticism (N) (10) is another risk factor for anxiety as well as depressive disorders (11,12,13,14). N is sometimes called “negative affectivity” (15) and is a trait disposition to experience negative emotions across a variety of situations (16, 15). There are no studies of SR modulation to contextual or explicit threat cues as a function of N. Extant studies of N are limited to affect modulation paradigms that use emotion-arousing film clips (17) and evaluation of SR in the absence of aversive stimuli (18). If patterns of SR modulation to contextual or explicit threat cues as a function of N parallel the findings as a function of familial anxiety and depression (8), then SR modulation may represent an observable indicator of a shared mechanism underlying these two risk factors for anxiety and depressive disorders.

The present study assessed contextual and explicit threat cue modulation of SR as a function of N in adolescents. Risk for anxiety and depressive disorders increases during adolescence (19,20), making it an important developmental stage in which to study potential premorbid risk factors. We hypothesized that SR magnitude would be larger as a function of N when elicited in the presence versus the absence of a contextual cue of threat, both before and after an explicit threat cue paradigm. We hypothesized that within the explicit threat cue paradigm, there would be a stronger association between N and SR magnitude during phases that signaled safety from aversive stimuli (i.e., context) than phases that signaled immediate danger of an aversive stimulus (i.e., explicitly cued threat). Finally, we explored the role of sex in all hypothesized effects, given that context modulation of SR as a function of risk for anxiety disorders has previously been found in females (8,9).

Method

Participants

All procedures were approved by University ethical review boards. Participants (Ps), recruited over three years, were high school juniors from schools in suburban Chicago, Illinois and suburban Los Angeles, California¹ (21). 1269 students (of whom 60% scored in the top third of a screening measure of N, and 20% scored in each of the middle and lower thirds) were invited to participate in the Northwestern-UCLA (NUCLA) Youth Emotion Project. 627 were enrolled with assent and parental consent, and completed a baseline diagnostic assessment. The final sample was 69% female, in part due to the greater willingness of females to participate. Approximately one half of males and females were selected randomly and invited to participate in the startle experiment. The 185 who completed the experiment did not differ from the remaining 442 Ps in terms of age, gender, ethnicity or our composite measure of N

¹See Zinbarg, Mineka, Craske et al., submitted (21) for further details on recruitment and overall study design.

(see below). Due to exclusions described below, final analyses were conducted on 132 Ps (47 males, 85 females).

46% were Caucasian, 21% Hispanic/Latino American, 9% African American, 7% Asian American/Pacific Islander, and 17% were 'other' or multi-ethnic. Age ranged from 16 to 18 years ($M=17.0$, $SD=0.5$). Ps received monetary compensation for their time and transportation costs.

Overall Design

The *contextual cue modulation* paradigm included an initial baseline condition, followed by a context condition in which contraction pads from an electrical muscle contraction device were placed on the biceps (i.e., the pads were the contextual cue). The next *explicit threat cue modulation* paradigm involved alternating safe and danger phases, during which an unpredictable aversive biceps contraction was threatened during the final 15 sec of danger phases only. Baseline and context conditions were then repeated.

Diagnostic and psychometric instruments

Axis I psychopathology—Lifetime Axis I psychopathology was assessed using the SCID-Non-Patient Version (22) on average 4 months (range 1–14 months) after screening. Reliability was adequate to good between primary interviewers and a reliability assessor who observed approximately 11% of SCIDs ($\kappa = .65$ – $.83$). Ps were excluded from analyses if they were diagnosed with current anxiety or mood disorders at baseline assessment ($n = 29$).²

Neuroticism—To increase the reliability and validity of the measured construct N, we used a composite of standardized values from four self-report questionnaires: EPQ-R-N³ (23), the Big-Five Mini-Markers Neuroticism scale (24), the International Personality Item Pool-NEO-PI-R (IPIP-N; <http://ipip.ori.org/newNEOKey.htm>, extracted 05/16/06), and the Behavioral Inhibition System scale (BIS; 25), with the latter representing a facet of N, namely trait anxiety, that is also included in the total scores for the EPQ (Mor et al., 2008) and IPIP. Details of the psychometrics for the composite index of N are in the on-line supplement. The EPQ-R-N was administered as part of initial screening, and remaining questionnaires were administered a median of 4 months (range 1–14 months) later.

Electrophysiological materials, equipment and data acquisition

Auditory startle stimuli (105 dB, zero rise time, 50 ms white noise bursts) were presented binaurally through stereophonic headphones (Sony, Model MDRV700). The muscle contraction, delivered by a Digital 807 Electrical Muscle Stimulation Device (Everyway Medical Instruments Co.), was a 20.4 mA peak current (i.e., equating to 50 V peak) for 0.5 sec⁴.

The SR was measured by electromyogram (EMG) activity of the orbicularis oculi and by skin conductance responses (SCR), to index both muscle responses and sympathetically mediated arousal responses to a startle stimulus (29). Details are available in the on-line supplement.

²Six Ps had a past anxiety disorder diagnosis, but were retained in the sample as they no longer met criteria for those diagnoses at baseline and did not meet criteria for anxiety disorders when re-assessed 12 months later.

³A suicide item was omitted and item 12 was excluded because it did not load well with the other items.

⁴The intensity level was pre-set based on pilot testing to represent an uncomfortable but not painful intensity, but was similar to mean voltage levels of shock intensity in studies using shock work-up procedures (27). Individualized work-up procedures were not chosen because pre-exposure to the muscle contraction might have decreased anticipatory anxiety during the explicit threat cue paradigm and/or weakened its aversiveness due to habituation (28).

Subjective Ratings—Anxiety was rated on a 10-point Likert scale (“calm and relaxed” to “really nervous or scared”) after each baseline and context condition and the explicit threat cue paradigm. After the experiment, Ps rated the intensity and unpleasantness of the startle stimulus and biceps contraction separately, on 20-point scales (higher scores reflecting higher values), developed specifically for the current study.

Procedure

The startle experiment was completed by 93 Ps at UCLA and 92 Ps at Northwestern University (NU). The two laboratories used identical hardware, software, manualized procedures, and technician training procedures.⁵

Ps were seated upright in a sound attenuated room adjacent to the experimental room, interconnected via intercom and closed-circuit cameras from two angles (UCLA) or one-way mirrors (NU). Ps were instructed to sit quietly and as still as possible. The experiment commenced with a 5 min *resting period* for adaptation, as Ps watched a silent movie, after which they were fitted with headphones and presented with a single startle stimulus to reduce initial reactivity (discarded from analyses). Figure 1 illustrates the experimental protocol and timing of delivery of auditory startle stimuli for each condition and phase.

During the *first baseline condition*, Ps focused on a white fixation cross on the computer and 8 startle probes were presented. For the *first context condition*, Ps were fitted with two contraction pads to the biceps muscle and told they would be informed when the contractions would happen. Eight more startle probes were presented while Ps focused on the white fixation cross. Before initiating the *explicit threat cue paradigm*, Ps were told they could be 100% sure that no muscle contractions would be delivered while the words ‘Safe: no contraction will be given’ were on the green screen, and they might receive a contraction when the words ‘Danger: contraction may be given’ were on the red screen. There were 8 safe and 8 danger phases in alternating order. Ps were also told that for both phases, they would see a progressing bar showing the time from 0 to 55 seconds, and that if a biceps contraction occurred during a Danger phase, it would occur when the bar turned from pink to red in the last 15 s. Finally, they were informed they might receive a muscle contraction up to three times, of increasing intensity each time. These instructions were designed to enhance anticipatory anxiety. In actuality, Ps received only one contraction in the final 15s of the fourth danger phase, half way through the paradigm. A total of 16 startle probes were presented in each phase (see Figure 1).

Next, the muscle contraction pads were removed for the *second baseline condition*, and reattached for the *second context condition*. Following electrode removal, startle stimuli and muscle contractions were rated, hearing was tested (all Ps passed), and Ps were debriefed.

Response definitions

Startle blink—EMG magnitudes were defined as the difference between the mean amplitude of the 200ms of EMG preceding the startle stimulus and the peak response, in microvolts (μ V). Details are presented in the on-line supplement.

Skin conductance—SCR magnitudes were defined as the difference between the trough and apex of the curve, expressed in microsiemens (μ S), and commencing within 1–4 sec following startle stimulus onset (31,27).

⁵The majority (67%) of experiments at each laboratory were run between 3pm and 8pm, with the rest before 3pm. Time of experimentation did not correlate significantly with N ($r = .06$).

Statistical analyses

Analyses were performed in SPSS version 14.0 (SPSS Inc., Chicago, Illinois) on natural log (ln) transformed eye blink data (30) and square root transformed SCR data (31), using a linear mixed model for repeated measurements with Satterthwaite's Approximation for degrees of freedom.

For contextual modulation, the factors were N (continuous), Sex (Male, Female), Condition (Baseline vs. Context), and Trial (First Half: Trials 1 to 4 vs. Second Half: Trials 5 to 8). These effects were analyzed separately, before and after the explicit threat cue paradigm (see Figure 1).

For explicit threat cue modulation, the fixed effects were N (continuous), Sex, Block (Pre vs. Post muscle contraction), Phase (Safe vs. Danger), and Probe Time (Early vs. Late). For Probe Time, "Early" referred to 5, 15, and 35 sec probe times (see Figure 1) (SRs at these three times did not differ). "Late" referred to the 45 sec probe time, when the muscle contraction could occur within danger but not safe phases.

Results

As there were no significant main or interaction effects involving N for SCR data, SCR analyses are not reported, but are available upon request. There were no site differences in age or N levels. There were site differences in ethnicity, 29.3% Caucasian at UCLA vs 66.7% at NU, $X^2(1)=18.2$, $p<.001$, and sex, 56% female at UCLA vs 73.7% at NU, $X^2(1)=4.4$, $p<.05$. Also, SRs were larger at NU than UCLA in the contextual cue ($F(1, 455)=7.02$, $p<.001$) and explicit threat cue modulation paradigm ($F(1, 519)=3.88$, $p=.05$). There were no other effects of interest involving Site.

First contextual cue modulation

$N \times \text{Sex} \times \text{Condition} \times \text{Trial}$ analyses revealed a main effect of Trial, $F(1, 1972)=16.86$, $p<.001$, reflecting reductions in SR magnitudes across baseline (first half; $M = 4.52$, $SE = .074$; last half; $M = 4.33$, $SE = .07$) and context conditions (first half; $M = 4.51$, $SE = .07$; last half; $M = 4.30$, $SE = .074$) (see Table 1). Also, the $N \times \text{Sex}$ interaction was significant, $F(1, 256)=4.81$, $p<.03$: N positively moderated SRs in males ($N = 47$; $\beta = .20$, $p<.02$), but not in females ($N = 85$; $\beta = -.03$, $p=.64$). There were no other significant effects, all F 's < 2.70 . In sum, the contextual cue did not modulate SR relative to baseline, and N did not influence SRs during baseline or context conditions, but it did influence SRs in males but not females.

Explicit threat cue modulation

$N \times \text{Sex} \times \text{Block} \times \text{Phase} \times \text{Probe Time}$ analyses revealed main effects of: Block, $F(1, 1039)=26.77$, $p<.001$, with SRs larger pre- ($M = 4.31$, $SE = .049$) than post-muscle contraction ($M = 3.98$, $SE = .048$); Phase, $F(1, 2600)=8.28$, $p<.01$, with SRs larger during danger ($M = 4.40$, $SE = .045$) than safe phases ($M = 3.88$, $SE = .044$); and Trial, $F(1, 3107)=131.12$, $p<.001$, with SRs larger during late ($M = 4.30$, $SE = .041$) than early trials ($M = 3.99$, $SE = .036$) (see Table 1). Significant two-way interactions were found between Block and Probe Time, $F(1, 3235)=9.47$, $p<.01$, Phase and Probe Time, $F(1, 2987)=170.49$, $p<.001$, and Sex and N, $F(1, 530)=16.18$, $p<.001$. The latter reflected that N positively moderated SRs in males ($N = 47$; $\beta = .29$, $p=.01$) but not females ($N = 85$; $\beta = .02$, $p=.67$). Also, there was a significant three-way interaction between N, Phase, and Probe Time, $F(1, 2990)=6.49$, $p<.02$.

Figure 2 shows the scatter plots of mean SRs regressed on N scores in safe phases (upper panels) and danger phases (lower panels) for early trials (left panels) and late trials (right panels). N had opposite effects on SRs at late and early trials within safe versus danger phases.

In safe phases, higher levels of N tended to be associated with larger SRs during late trials ($\beta = .15, p = .06$) than early trials ($\beta = .08, p = .11$). In danger phases, the regression of SRs on N were larger for early trials ($\beta = .13, p = .02$) than late trials ($\beta = .06, p = .31$). In sum, SRs were enhanced as a function of N in late safe and early threat trials, and overall, N influenced SRs in males but not females.

Second contextual cue modulation

$N \times \text{Sex} \times \text{Condition} \times \text{Trial}$ repeated measures analyses revealed significant main effects of: Condition, $F(1, 905) = 19.35, p < .001$, with SRs larger during context ($M = 3.87, SE = .07$) than baseline ($M = 3.49, SE = .07$); and Trial, $F(1, 1886) = 15.97, p < .001$, with SRs larger during the first ($M = 3.81, SE = .065$) than last half of trials ($M = 3.55, SE = .065$) (see Table 1). Moreover, there was a $N \times \text{Sex}$ interaction, $F(1, 292) = 7.20, p < .01$, with higher N related to larger SRs in males ($N = 47; \beta = .35, p < .001$) but not females ($N = 85; \beta = .05, p = .50$). In sum, the contextual cue modulated SRs relative to baseline, and N did not influence these effects. However, N influenced SRs in males but not females.

Subjective Rating

Anxiety—Analysis of the first baseline and context conditions (see Table 2) revealed main effects of Condition, $F(1, 127) = 11.58, p < .001$, with higher anxiety for baseline ($M = 3.49, SE = .19$) than context ($M = 2.88, SE = .21$), and for N, $F(1, 127) = 8.14, p < .01$, with higher N associated with higher anxiety in both conditions ($\beta = .54, p < .001$, and $\beta = .43, p < .03$). For the explicit threat cue modulation paradigm, the $N \times \text{Sex}$ analysis revealed only a main effect of N, $F(1, 129) = 10.03, p < .002$, with higher N associated with higher anxiety ($\beta = .66, p < .002$). For the second baseline and context conditions, there was a main effect of Condition, $F(1, 128) = 21.23, p < .001$, due to higher anxiety for context ($M = 2.40, SE = .20$) than baseline ($M = 1.64, SE = .16$). The main effect and interactions with N and Sex were not significant, F 's < 2.59 . In sum, anxiety to the first (but not second) baseline and context conditions and the explicit threat cue modulation paradigm were enhanced as a function of N.

Muscle contraction—There was a main effect of Sex on intensity ratings of the muscle contraction (see Table 2), $F(1, 129) = 9.85, p < .01$, with higher ratings by females ($M = 13.72; SE = .41$) than males ($M = 11.40; SE = .56$). However, neither the main effect of N nor the $\text{Sex} \times N$ interaction was significant. For unpleasantness, there was a main effect of N, $F(1, 129) = 4.79, p < .02$, with higher N associated with higher unpleasantness ($\beta = .70, p < .05$). There was also a main effect for Sex, $F(1, 129) = 14.41, p < .001$, with females ($M = 9.69; SE = .40$) rating the contraction as more unpleasant than males ($M = 7.16; SE = .53$), but there was no $\text{Sex} \times N$ interaction. In sum, unpleasantness but not intensity ratings were enhanced as a function of N.

Startle stimulus—Higher N was associated with higher intensity ratings of the startle stimulus (see Table 2) ($\beta = .68, p < .05$) and a trend for higher unpleasantness ratings ($\beta = .55, p = .06$). There were no significant effects or interactions for Sex, F 's < 1.66 .

Discussion

This study of contextual and explicit threat cue modulation of SR as a function of N in adolescence, a risk factor for anxiety and depressive disorders, yielded two main findings. First, within the explicit threat cue paradigm, the potentiating effects of N on SRs were larger during late safe and early danger trials than during early safe (furthest from threat) and late danger (closest to threat) trials. Also, N potentiated anxiety ratings for the initial baseline and context conditions, and the explicit threat cue paradigm. These findings are broadly consistent with other evidence for enhanced responding to contexts associated with threat in individuals with anxiety disorders and those at risk for such disorders due to familial anxiety or depression (4,

8,9). Second, N potentiated SRs in males but not in females across the entire experiment, regardless of phase, block or condition, even though females judged the muscle contraction as more arousing and unpleasant than males.

The electrical muscle stimulator was successful in generating explicit threat cue modulation of SRs, with larger SRs during danger than safe phases, and during late versus early trials. Also, N interacted with phase and probe time in modulating SRs, such that N tended to potentiate SRs more during late than early trials within safe phases but potentiated SRs more during early than late trials within danger phases. Conceivably, results from the safe phases represent a gradient of contexts associated with threat, with the modulating effects of N strongest in the late trials that represented closer proximity to threat by virtue of matching the timing of the contraction during danger phases. In contrast, within danger phases, the effects of N were stronger in early trials, representing the context of threat, than late trials, representing closest proximity to an explicit threat cue.

That N did not modulate SRs during closest proximity to threat (i.e., late threat trials) is consistent with previous evidence for lack of explicit threat cue modulation by risk status (8, 9). Also, this finding reflects the “strong situation” effect (32), in which anxious patients and controls respond similarly to intense threat, when anyone would be expected to show neurobiologically imperative fear responses, whereas only anxious individuals respond more strongly to less intense aversive stimuli. Moreover, our finding that N did not modulate SRs during when furthest from threat (i.e., early safe trials) parallels findings in baseline conditions when participants are reassured that no aversive stimulus will be presented (2). This may represent the other side of the “strong situation” when no-one would be expected to show neurobiologically imperative fear responses.

At least two alternative explanations exist. First is nonspecific arousal created by repeated exposure to startle probes. Second is slowed habituation as a function of higher levels of N. However, the differential effects of N across late safe and early threat trials, versus early safe and late threat trials, during which the startle stimuli were constant variables, suggests that the effects of N are not solely attributable to non-specific arousal. This same effect also rules out the slowed habituation explanation, as late versus early trials were not confounded with order effects.

In contrast to hypotheses, N did not potentiate SRs more so in the presence of a contextual cue than in its absence. However, context modulation of SR in general (i.e., larger SRs during context than baseline conditions) was not observed prior to the explicit threat cue paradigm. Conceivably, initial context modulation of SR was offset by insufficient habituation during the first baseline condition, when only nine startle probes were delivered. Additionally, threat of an ‘unknown’ aversive biceps contraction implied by attachment of the muscle contraction electrodes and instructional set may have been insufficiently aversive to generate contextual modulation; a possibility that is supported by lower anxiety ratings for the context than the baseline condition. Direct experience with the biceps contraction contributed to context modulation, since SRs and anxiety ratings were larger for the context than baseline condition following the explicit threat cue paradigm. N also modulated SR in general across the second baseline and context conditions, although main effect was accompanied by a Sex and N interaction.

Indeed, throughout the entire experiment, N potentiated SRs in males but not in females. Thus, N related to defensive reactivity in males but not females, regardless of phase, block or condition. Prior evidence showed elevated baseline SRs in female but not male offspring of anxious/depressed parents/grandparents (8,9). Conceivably, familial emotional disorders and N confer risk differently for males versus females. Additionally, the effects of N upon SRs

might be moderated by other variables in females, such as stronger family history. Moreover, sex differences in prior studies were confounded by age, as females were significantly older than males in one study (8) and high risk samples were significantly older than low risk samples in another (9). Thus, risk due to familial anxiety and depressive disorders versus N may be conferred at different developmental stages in males versus females. Clearly, replication of these findings is required before such speculations can be tested.

Our measure of SCRs was to the startle stimulus per se (29) rather than to emotional stimuli, which may explain why we did not observe effects of N upon SCR that have been reported elsewhere (34). A limitation of this study is that an uncertain number of muscle stimulations with threat of increasing intensity (akin to threat of shock procedures, 33) was not as anxiety-provoking as intended; subjective anxiety for the explicit threat cue paradigm was only moderate. On the other hand, anxiety was rated once the paradigm was complete and may have underestimated ongoing anxiety. Ongoing measurement of anxiety, such as via a dial-and-pointer at designated times without disrupting task procedures, would be valuable in future studies. Another limitation was that order effects and habituation may have deflated SRs during danger phases (which always followed safe phases) and context conditions (which always followed baseline conditions). Furthermore, selection biases from participation rates may have impacted generalizability of the findings, although Ps in the startle experiment did not differ appreciably from remaining Ps. Finally, further investigation of the role of ethnicity and sex in SRs, that possibly contributed to overall site differences herein, is warranted.

In conclusion, N enhanced SRs to startle probes more during contexts intermediary to threat than conditions furthest from threat and conditions closest to threat within an explicit threat cue paradigm. Thus, N modulated SRs when at partial risk but not when at full risk for threat. These findings may indicate that greater defensive responding to contexts associated with threat is a pathway through which N confers risk for anxiety and depressive disorders. Longitudinal investigation will elucidate the degree to which such patterns of SRs are predictive of anxiety disorders. Finally, the finding that N modulated SRs across the entire experiment in males and not females may be indicative of sex specific effects of risk factors.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Figure 1a shows the overall experimental design. In the initial baseline and context conditions, 8 startle stimuli were delivered with a mean ISI of 22s (range 20–24s) as Ps focused on a white fixation cross. The baseline and context conditions were repeated after the explicit threat cue paradigm. The explicit threat cue paradigm included 8 safe (S) and 8 danger (D) phases presented in alternating order, with intervening rests periods of 5s, 15s or 25s showing the white fixation cross. Figure 1(b) shows that each 55 sec safe and danger phase contained two startle trials delivered at either 5 and 35 sec or 15 and 45 sec, with the order rotated every two phases. Startle trials at 45 sec were delivered during the 40–55 sec explicit threat period depicted on the progressing bar displayed on the computer screen. The muscle contraction was delivered at 50 sec during the fourth danger phase, resulting in 4 safe and 4 danger phases pre-contraction and a further 4 safe and 4 danger phases post-contraction. Grey indicates baseline and safe; black indicates context and danger.

Figure 2.

Mean SR magnitude values (+SE) as a function of N at early (left panels) and late (right panels) startle trials during safe (top panels) and danger (bottom panels) phases in the explicit threat cue paradigm.

Table 1

Overall mean SRs (+SE) during first and last half of baseline and context conditions during the contextual cue modulation paradigm and during safe and threat phases pre- and post-contraction during the explicit threat cue modulation paradigm.

Contextual Cue Modulation				Explicit Threat Cue Modulation						Contextual Cue Modulation					
				Pre-Contraction			Post-Contraction			Baseline			Context		
				Safe		Threat	Safe		Threat						
Baseline	Last Half	First Half	Context	Early	Late	Early	Early	Late	Early	Late	First Half	Last Half	First Half	Last Half	
	4.33	4.51	4.30	4.04	3.99	4.24	3.67	3.73	3.88	4.61	3.67	3.31	3.94	3.80	
	.07	.07	.074	.061	.069	.062	.065	.07	.059	.073	.088	.084	.084	.088	

Overall means (+SE) for subjective ratings of the experimental phases (scale 0–10) and the muscle contraction and startle stimulus (scale 0–20).

Table 2

Contextual Cue Modulation		Explicit Threat Cue Modulation		Contextual Cue Modulation		Biceps Contraction		Startle Stimulus	
Baseline	Context			Baseline	Context	Intensity	Unpleasant	Intensity	Unpleasant
3.49	2.88	4.98		1.64	2.39	12.56	8.42	11.43	8.74
.19	.21	.21		.13	.20	.34	.33	.32	.30