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Heterogeneity of defensive responses after exposure to trauma: Blunted autonomic reactivity in response to startling sounds

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ABSTRACT

Research on threat responses, particularly among trauma-exposed individuals, has traditionally focused on increased autonomic arousal and reactivity. However, clinical features associated with trauma exposure, such as dissociation (e.g., shutting down or "spacing out") manifest as the opposite pattern: non-reactivity and blunted arousal. These clinical features suggest that the possibility of threat responses other than fight/flight, namely, immobilization may be undergirded by hyper- or hypo-arousal. The goal of this paper is to examine autonomic responses to a stressful stimulus (acoustic startle) using analytic approaches which have been previously used to examine defensive responses before: heart rate acceleration, heart rate deceleration, and skin conductance response. We examined these responses in relation to symptoms (Posttraumatic Stress Disorder, or PTSD, and dissociation) and trauma exposure (cumulative exposure, age of onset) in a sample of trauma-exposed college students. We found evidence of blunted reactivity, with decreased acceleration and skin conductance, but with increased deceleration, particularly among individuals who had significant symptoms and early exposure to multiple types of trauma. However, individuals with sub-clinical symptoms and more attenuated exposure had large heart rate acceleration and skin conductance responses during the task. Taken together, these findings suggest that moderate symptoms and trauma exposure are related to exaggerated autonomic responses, while extreme symptoms and trauma exposure are related to blunted autonomic responses. These findings further suggest heterogeneity of stress responses within individuals with PTSD and with trauma exposure.

1. Introduction

While research on cardiovascular threat responses typically reports increases in heart rate following threat, several studies in recent years have found blunted cardiovascular responses among the very individuals who might most be expected to have exaggerated responses: namely, chronically-traumatized people (Cuthbert et al., 2003; McTeague et al., 2010). To date, existing studies on the cardiovascular reactivity of individuals exposed to traumatic stressors have rarely parsed components of defensive responses, such as orienting and fight/flight. Research with traumatized samples may be a useful avenue for studying the entire repertoire of threat responses, as traumatized individuals report a diverse range of responses, from fight/flight to immobilization (Adenauer et al., 2010; Griffin et al., 1997). Several variables, including reactions to trauma exposure and features of the exposure itself (e.g., chronicity) may help clarify defensive responses. The goals of this study are to 1) examine the range of autonomic defensive responses to stressful stimuli in a trauma-exposed sample and 2) examine factors that may contribute to autonomic response variance which may underlay behavioral threat strategies.

1.1. Defensive responses: orienting, preparation for action, and freezing

Bradley et al. (2001) suggest the Defense Cascade Model for delineating stress responses as proximity to threat changes over time. This response occurs over two phases: orienting, which allows the individual to take in information and ascertain threat; and preparation for action, which, if needed, facilitates a fight or flight response. The orienting phase is characterized by a moderate heart rate (HR) deceleration (typically of 2–3 beats per minute [bpm]) in the 3–4 s post-stimulus, and a moderate increase in skin conductance. The heart rate deceleration in this phase is interpreted as parasympathetically-driven: modest increases in parasympathetic activity (Thayer, 2000) and sympathetic activity (Andreassi, 2007) facilitate information processing. In contrast, the fight/flight phase is characterized by heart rate (HR) acceleration, typically under 5 bpm (Bradley et al., 2001; Cuthbert et al., 2003), and a large skin conductance response (SCR). Defensive responses change with repeated exposure to a threatening stimulus. If a stimulus occurs repeatedly (without signals of escalating threat), deceleration and

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SCRs quickly diminish. However, if a stimulus increases in proximity/intensity, deceleration responses diminish, but acceleration and SCRs escalate. Thus, the presence of a modest deceleration response and a modest and rapidly-diminishing SCR reflect the processing of novel stimuli, whereas HR acceleration and increasing SCR reflect preparation for action.

Cuthbert et al. (2003) posit that chronic anxiety may prime defensive responding. Rather than deceleration followed by acceleration, anxious individuals show immediate HR acceleratory responses and large SC responses. However, in contrast with data from anxiety disorders, Cuthbert et al. (2003) notes that some individuals who would be expected to show increased arousal in response to threat, namely, some individuals with Posttraumatic Stress Disorder (PTSD), do not show this response. Instead, these individuals have blunted reactivity.

1.2. Trauma responses: hyper- and hypo-arousal in PTSD and dissociation

One of the defining features of Posttraumatic Stress Disorder (PTSD) is physiological hyperreactivity to trauma cues (APA, 2000), including increased acoustic startle responses (Pole, 2007). However, several studies have reported paradoxical autonomic hyporeactivity. In such studies, individuals with PTSD show no difference in reactivity relative to controls, or responses that decline from baseline (Cuthbert et al., 2003; Limberg et al., 2011; McTeague and Lang, 2012). Epidemiological studies (van der Kolk et al., 2005; Zucker et al., 2006) as well as conflicting physiological findings have generated the proposal of two types of PTSD: (1) the hyper-reactive type described in the DSM (i.e., avoidance, hyperarousal, and re-experiencing); and 2) a dissociative type, with blunted emotional and physiological responses (Ebner-Priemer et al., 2005; Koopman et al., 2004; Lanius et al., 2012). Taken together, the literature suggests that PTSD is associated with increased HR and SCR in response to startle probes; however, the presence of dissociation may attenuate or reverse this effect.

Drawing from the animal literature, Blanchard and Blanchard (1989), Bracha (2004), and others (e.g., Campell et al., 1997) have suggested a human immobilization response, occurring in contexts in which immobility is the safest threat response strategy. This freeze response (also called dissociation, fear bradychardia, tonic immobility, and shutting down) is characterized by decreased motility, rather than fight or flight.[1] The freeze response may be most relevant among trauma survivors for whom, during the traumatic situation, fight or flight was impossible (van der Kolk et al., 1989).

Little is known about the physiology that accompanies the freeze response. Some studies (Adenauer et al., 2010; Hagenaars et al., 2012; Volchan et al., 2011) have documented a heart rate deceleration and postural freeze in response to threat. Other research (Kapp et al., 1992) suggests that a freeze response reflects a physically-immobile state in which an organism is primed to flee when the opportunity arises (e.g., "response-ready freezing"). An alternative form of immobilization may manifest as a passive response, in which an organism shuts down both behavior and cardiac output to minimize risk of harm (which we will call "shut-down immobilization"). Thus, while immobilization has been described in terms of a single behavioral response—decreased motility—two potential autonomic physiological responses may underlie immobilization. The response may depend upon the co-activation or de-activation of branches of the ANS: elevated SNS alone activity may facilitate flight/flight responses; elevated activity of both SNS and PNS may facilitate response-ready freezing; and finally, decreased activity in both SNS and PNS, or elevated PNS activity, may reflect shut-down immobilization.

[1] The literature tends to use the term "freeze" to refer to both orienting and to immobile threat responses. Here, we will use orienting to refer to the early, initial phase of moderate HR deceleration and SCR describe as the first phase of the defense cascade by Bradley et al.; we will use "freeze" to refer to the immobilization observed as an alternative defensive response to fight/flight (Bracha, 2004).

1.3. Characteristics of trauma exposure that mitigate threat responses

Chronicity of exposure has been suggested as an etiological factor in the dissociative subtype of PTSD (Lanius et al., 2012; Steuwe et al., 2012). Repeated, unpredictable exposure to trauma may signal inescapability (van der Kolk et al., 1996). McTeague et al. (2010) found that while single-incident PTSD patients had heightened autonomic reactivity to startle probes compared to controls, participants with multiple-incident trauma exposure showed blunted reactivity. Cuthbert et al. (2003) found that multiply-traumatized subjects had HR deceleration, not acceleration, suggesting that multiply-traumatized individuals may not show the expected defensive response of sympathetic arousal, but instead display parasympathetic reactivity. Examining both postural freeze and heart rate deceleration, Hagenaars et al. (2012) found that while singly-exposed participants showed heart rate deceleration in response to aversive images, multiply-exposed individuals showed both heart rate deceleration and postural freeze. In McTeague's and Hagenaar's studies, self-reported emotion failed to distinguish exaggerated vs. blunted reactivity. Early developmental onset may also be related to dissociative symptoms (D'Andrea and Pole, 2012; Seng et al., in press) as well as blunted physiological reactivity (Quevedo et al., 2010). In addition to trauma exposure, other chronic stressors, such as exposure to community violence, poverty, and racism, may be related to altered cardiovascular responses (Clark et al., 1999; Lepore et al., 2006). These findings indicate that chronic, early-onset trauma may be related to 1) smaller increases in heart rate after startle probes; and 2) increased, sustained deceleratory responses to stressful stimuli.

1.4. The present study

Taken together, a growing literature suggests that several factors may differentiate between hyper- and hypoarousal. However, to date, no studies have investigated the autonomic components of freeze response a) in studies of acoustic startle; b) in relation to components of HR acceleration, HR deceleration, and skin conductance; or c) how freeze responses are distinguished from orienting responses. Because trauma survivors experience altered defensive responding, including freeze responses, we will examine their autonomic responses to threatening stimuli. We will attempt to fill gaps in the defensive response literature, first by examining both ends of the physiological spectrum: we will analyze both increased and decreased reactivity in response to startling stimuli. Second, most studies have used data analysis which collapse responses across time, effectively smoothing the data in such a way as to obscure effects. However, research on changing physiological responses throughout a task (e.g., Bradley et al., 2001) supports the prediction that averaging over an entire task may obscure patterns of habituation and sensitization. Using an analysis approach that examines reactivity over time, as used by Bradley et al. (2001), will allow us to disentangle the effects of novel (i.e., orienting response) vs. sustained reactivity. We will also distinguish between acceleratory and deceleratory components of the heart rate response, which will allow us to use the lens of the defense cascade model. Finally, we will examine how hyper-and hypo-arousal may relate to salient characteristics of trauma, namely trauma emotional response and trauma exposure. This study is also novel because it focuses on an under-studied racial minority sample; all participants were African American. This group may be particularly prone to later stress-related cardiovascular illness (Harrell and Morris, 1996). Therefore, understanding the function of trauma in cardiovascular reactivity may be important for this group. We propose that we will find evidence of blunted reactivity to startling sounds in a trauma-exposed sample, in accord with the following hypotheses: 1) We expect that predictor variables (emotional responses and trauma exposure) will not differentiate physiological variables when physiological are averaged across all trials, as effects will be obscured due to group differences in physiological responses over time, and; 2) predictor variables will differentiate physiological responses of blunted vs. elevated