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Expressive inhibition in response to stress: Implications for emotional processing following trauma

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ABSTRACT

Expressive inhibition – the willful restriction of expressed emotion – is documented in individuals reporting trauma-related distress, but its impact on global affective functioning remains unclear. Theoretical models propose that chronic activation of negative emotion and deliberate restriction of affect operate synergistically to produce trauma-related emotional deficits. The current project examined the impact of these factors on subjective experience and physiological activation following exposure to an analog trauma. University students ($N = 192$; $M_{age} = 20$, 57% female, 42% White/Non-Hispanic) viewed a graphic film depicting scenes of a televised suicide. Participants then viewed either a sadness- or humor-eliciting film under instructions to inhibit [$n_{sadness} = 45$, $n_{humor} = 52$] or naturally express emotion [$n_{sadness} = 48$, $n_{humor} = 47$]. Expressive inhibition was associated with restricted amusement specifically among participants viewing the humor film. Inhibition also produced attenuated sympathetic and parasympathetic recovery, irrespective of film assignment. Evidence of disruptions in emotional processing supports models identifying inhibition as a possible mechanism in post-trauma affect dysregulation.

1. Implications for emotional processing following trauma

Emotional deficits associated with posttraumatic stress disorder (PTSD) have been identified as the most poorly conceptualized and least studied aspect of the disorder (Litz, 1992). This is problematically given that affective deficits, historically indicated as symptoms of emotional numbing (APA, 1980), demonstrate robust associations with negative outcomes including the prospective severity of PTSD (e.g., Harvey & Bryant, 1998), reduced quality of life (e.g., Lunney & Schnurr, 2007), interpersonal dysfunction (e.g., Kuhn, Blanchard, & Hickling, 2003), and poor response to intervention (e.g., Jaycox & Foa, 1996; Taylor et al., 2001). Existing theory identifies both automatic and volitional processes as contributing to emotional dysregulation following trauma. The deliberate restriction of expressed emotion (i.e., expressive suppression) is one mechanism having received empirical attention (e.g., Roemer, Litz, Orsillo, & Wagner, 2001) although the impact of this behavior on global affective response and its relation to broader conceptualizations of post-trauma emotional functioning remains unclear. The goal of the current project was to evaluate the impact of deliberate expressive inhibition on subjective experience and physiological response following the activation of negative, trauma-related emotion networks. For this research, an analog paradigm was used to simulate trauma-relevant processes in a diverse, non-clinical sample.

1.1. Post-trauma affective deficits

The specific mechanisms contributing to symptoms of emotional restriction and detachment have received limited attention within prominent models of PTSD (e.g., Foa, Steketee, & Rothbaum, 1989; Horowitz, 1986; Resick & Schnicke, 1992). In response, Litz (1992) provides a comprehensive framework outlining processes believed to influence affective functioning in the aftermath of trauma. Contrasting models of global emotional restriction, Litz operationalizes affective deficits as a phasic reaction to trauma-related intrusions, characterized by a restricted response to positive emotional events. Although survivors retain *the capacity* for normal emotional processing, the likelihood of an adaptive response is

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reduced by the activation of trauma-related intrusions and distress. Three hierarchical systems are outlined in Litz' model.

1.1.1. Expressive-motor programs

Unlearned, expressive-motor programs are proposed to coordinate processing at the most basic level. These programs are activated involuntarily in the presence of emotionally relevant stimuli and organize universal affective processes (e.g., stereotyped expressive behavior, physiological response). Expressive-motor programs provide the capacity for basic emotional learning and remain unaltered following trauma exposure. Evidence of similarities in emotional processing across PTSD and non-PTSD groups in the absence of explicit trauma cues suggests that basic programs for adaptive emotional responding remain intact following exposure (e.g., Amdur, Larsen, & Liberzon, 2000; Phan, Britton, Taylor, Fig, & Liberzon, 2006).

1.1.2. Schematic processes

A higher-order schematic subsystem is proposed to organize networks that coordinate learned emotional reactions (Litz, 1992). As with other models (e.g., Foa & Rothbaum, 1998), trauma networks are thought to be characterized by their accessibility and by the intensity of cued emotional responding. Activation of trauma-relevant networks serves to decrease the threshold for negative affect while increasing the threshold for positive emotional experience. Research indicating cued deficits in expressive behavior, positive emotion, physiological activity, and defensive startle provides evidence of state-dependent processing anomalies in survivors with PTSD (Casada, Amdur, Larsen, & Liberzon, 1998; Litz, Orsillo, Kaloupek, & Weathers, 2000; Miller & Litz, 2004).

1.1.3. Conceptually driven deficits

Whereas schematic deficits in response to cued reactions are largely involuntary, processing in Litz's (1992) conceptual subsystem is both deliberate and effortful. The conceptual system enables volitional, top-down regulation and is influenced by an individual's expectations and attributional style. Deficits at the conceptual level are the result of deliberate regulatory strategies intended to restrict emotional response, particularly during the activation of trauma-relevant networks. Examination of conceptual-level processing has primarily targeted the willful inhibition of expressive behavior.[2] Research with combat survivors suggests those with PTSD deliberately restrict expressive response to positive and negative events to a greater degree than veterans with no diagnosable disorder (Roemer et al., 2001). Associations between inhibition and post-trauma symptoms also have been noted in student and community survivor samples (Marx & Sloan, 2002; Moore, Zoellner, & Mollenholt, 2008). Evidence of indiscriminant and habitual withholding in this literature is consistent with top-down conceptual processes believed to influence dysregulation in the aftermath of trauma (Litz, 1992).

1.2. Consequences of expressive inhibition

Assuming survivors with PTSD routinely inhibit expression, what are the potential consequences of this behavior? Clearly, deliberate and pervasive inhibition could enhance the appearance of flattened affect characteristic of many individuals with PTSD. Inhibition may also selectively attenuate the experience of positive emotion. Gross and Levenson (1997) found that women instructed to inhibit expression during a humorous film reported less positive emotional experience than those permitted to naturally express. Inhibition in response to negative stimuli, by contrast, was unrelated to negative emotional experience. Whereas successful replication of these effects has been limited (i.e., Bonanno, Papa, Lalande, Westphal, & Coifman, 2004; Bush, Barr, McHugo, & Lanzetta, 1989; Davis, Senghas, Ochsner, 2009; Schmeichel, Volokhov, & Demaree, 2008; Zuckerman, Kiorman, Larrance, & Spiegel, 1981), the possibility of a targeted impact of inhibition on positive emotion – particularly during the activation of trauma-relevant networks – could account for conflicting symptoms of numbness and intense negative affect observed in survivors with PTSD (Amdur et al., 2000).

Data also suggest inhibition may impact elements of physiological response. Relative to natural expression, inhibition is shown to produce reliable elevations in sympathetic activity as indexed via skin conductance and other sympathetic-mediated indices (e.g., Gross & Levenson, 1993). Sympathetic dysregulation is proposed as a biological substrate for hyperarousal (Kolk, Greenberg, Boyd, & Krystal, 1985) which, in turn, demonstrates unique associations with posttraumatic numbing (e.g., Litz et al., 1997). The relation of post-trauma dysregulation with parasympathetic responding is less clear although some research provides evidence of parasympathetic nonresponse among individuals with PTSD (Cohen et al., 2000; Sahar, Shalev, & Porges, 2001). While preliminary, these effects are interesting in that parasympathetic reactivity is proposed to be instrumental to adaptive emotional functioning (Friedman, 2007; Porges, 1995). Research evaluating the impact of expressive regulation on parasympathetic activity remains equivocal (e.g., Demaree et al., 2006; Hagemann, Levenson, & Gross, 2006) although no study to date has examined these processes as they are likely to occur within the context of trauma-related distress.

1.3. Overview of the current research

Existing research indicates anomalies among trauma survivors at the schematic-level of processing, but studies examining the impact of conceptual-level regulation remain limited. The aim of the current project was to evaluate the consequences of expressive inhibition in a non-clinical sample following exposure to an analog trauma. Analog methodologies have proven useful for assessing a variety of trauma-relevant processes including memory coding, thought suppression, and the maintenance of intrusions (e.g., Brewin & Saunders, 2001; Davies & Clark, 1998; Krans, Näring, Holmes, & Becker, 2009). As Litz's (1992) model posits affective deficits to be a state-dependent reaction to negative emotional priming, the analog paradigm provides a unique opportunity to model trauma-relevant processes in a non-clinical sample.

University students in this study were shown a series of affectively valenced films. Upon establishing baseline levels of behavioral, subjective, and physiological response, participants viewed commercially available scenes of a televised suicide intended to prime trauma-relevant emotion networks. Finally, participants viewed either a sadness- or humor-eliciting film under instructions to inhibit or naturally express emotion. Inhibit instructions were designed to model deliberate regulatory processes thought to influence post-trauma emotional deficits. Expressive restriction was expected to uniquely produce attenuation in positive emotional experience while elevating measures of sympathetic response. Parasympathetic reactivity also was assessed although analyses were considered exploratory given conflicting effects within the trauma and expressive manipulation literatures (e.g., Cohen et al., 1998, 2000; Roberts, Levenson, & Gross, 2008).

[2] *Expressive inhibition* as used in this research is synonymous with Gross and Levenson's (1993, 1997) conceptualization of *expressive suppression*. The term expressive inhibition is used to highlight the targeted regulation of expressive behavior as opposed to more general suppression of affective response (see Campbell-Sills, Barlow, Brown, & Hofmann, 2006).