Toward a Psychobiology of Posttraumatic Self-Dysregulation

Reexperiencing, Hyperarousal, Dissociation, and Emotional Numbing

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ABSTRACT: In this article we propose a psychobiological model that construes PTSD fundamentally as a disorder of affect arousal regulation. Neuroimaging studies of emotion regulation in psychologically healthy populations are initially reviewed as a framework for interpreting the results of previously published investigations of the neural correlates of PTSD reexperiencing and dissociation. We then apply the emotion regulation framework toward understanding other perturbed affective states in PTSD. We conclude by discussing the clinical significance of this framework for psychological assessment and treatment of posttrauma psychopathology.

KEYWORDS: posttraumatic stress disorder; dissociation; emotion regulation; emotional numbing; script-driven imagery; neuroimaging

INTRODUCTION

Under the current Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV rubric, posttraumatic stress disorder (PTSD) is classified as an instance of anxiety disorder. PTSD is diagnosed when individuals report intrusively reexperiencing memories of a past traumatic event(s), exhibit cognitive and behavioral avoidance, and report symptoms of psychophysiological arousal, mood disturbance, and functional impairment. A key clinical feature of PTSD, however, and a universal mark of the anxiety disorders as a class, is that not only are symptoms of marked anxiety and fear present, but these symptoms are perceived by the individual as beyond their capacity

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to control. Specifically, the individual with PTSD is characteristically unable to manage or *down* regulate his or her level of psychophysiological aversive arousal and distress. Comparatively, at other times individuals with PTSD appear to be unable to *up* regulate their level of arousal, for example, during periods of hypoarousal, such as anhedonia and felt "emotional numbness." That individuals with PTSD often report a generalized lack of control over their emotional responding is of clinical and theoretical interest, as it gives credence to the notion that a deficiency in the ability to adaptively regulate levels of affective arousal and distress in the context of stressors may be central to this disorder. This may be particularly true of individuals exposed to long-term interpersonal and developmental attachment trauma, such as in cases of recurrent domestic violence and childhood sexual abuse, in comparison with single-incident adult-onset traumatic events, such as motor vehicle or workplace accidents, natural disasters, and even one-time acts of violence including physical and sexual assault by a stranger. Because of the severe, recurrent, and early-onset nature of the traumatic events to which the former group of individuals has been exposed, their neural capacities for managing-regulating stress arousal levels may be seriously compromised.

Accordingly, in this article we propose a psychobiological model that delineates PTSD principally as a disorder of generalized affect arousal dysregulation. First, we define emotion regulation processes as a class of primarily medial–frontal/paralimbic executive functions that hierarchically modulate the activity of lower-level emotional appraisal processes. We then briefly review the results of an emerging number of neuroimaging studies of emotion regulation in the psychologically healthy population, and apply the results of these studies as a framework toward understanding our previously published investigations of the fMRI–BOLD neural correlates of reexperiencing[1] and dissociative[2] reactions during trauma script recall imagery. Therein we conceive of the PTSD reexperiencing response as an instance of deficient neuroregulatory control over emotional arousal, whereas at the other extreme, we consider the PTSD dissociative response to be an *enhanced* neuroregulatory suppression or inhibition of traumatic memory-related emotional arousal. We then apply the emotion regulation framework toward understanding other perturbed affective states in PTSD, specifically sadness and generalized anxiety,[3] and conjecture on the model's relevance for understanding PTSD-associated anger, guilt, shame, and pain processing, as well as *hypo* arousal states, such as anhedonia and "emotional numbing" in PTSD. In the concluding section, we discuss issues pertinent to psychological assessment and treatment of posttraumatic event psychopathology.

NEUROIMAGING OF ADAPTIVE AFFECT AROUSAL REGULATION

Phillips *et al.*[4] define a theoretical organizational framework for understanding the psychobiological processes involved in healthy human emotion.

Specifically, Phillips *et al*. distinguish between three mechanisms purportedly involved in the processing of emotional stimuli and situations: (*a*) emotion perception-appraisal (detection of an emotionally relevant stimulus), (*b*) affect generation (production of a bodily state in response to the first process, as well as the conscious awareness of that state), and (*c*) regulation of the affective state. Emotion regulatory processes refer to the manner by which people volitionally influence how they experience and express emotions (e.g., Ref. 5). Whereas the first two mechanisms are regarded as stimulus-driven and automatic, emotion regulation is viewed by theory as a "top-down" (attention-controlled, executive) process.[4,5] Although contemporary theoretical models often portray emotional processes as unfolding in linear sequence from perception to regulation, we would submit that each of the emotional subprocesses is more aptly represented as a state in flux in accordance with the principles of a nonlinear dynamical system (see Ref. 6). For example, preexisting affective states may bias perceptive processing in a manner favoring emotional appraisals that are consistent with the current affective state, and emotion regulatory processes may modulate emotional appraisal prior to affect generation. It is also important to note that emotion regulation processes can be used in the service not only of attenuating but also of accentuating the intensity of generated affects.[7]

The neural substrates of emotion perception and affect generation have been the subject of rigorous study, and a central role for the amygdala in these processes has been established.[8,9] A number of studies have now confirmed that emotional perception appraisal structures are hyperresponsive to threat-relevant stimuli in individuals with PTSD, such as the amygdala response to facial expressions of fear (e.g., Ref.10). In contrast, the brain basis of emotion regulation has only recently become a topic of neuroscientific study (see Ref. 11 for review). A growing number of neuroimaging studies have shown, however, that willfully attempting to "reappraise" negatively valenced pictorial stimuli via one's verbal analytical faculties in such a way as to decrease their affect-arousing properties recruits dorsolateral and ventromedial prefrontal cortex (PFC), anterior cingulate cortex (ACC), and orbitofrontal cortex (OFC) relative to passive viewing of the same stimuli, and that activation in these structures negatively correlates with response in the amygdala.[12–14] Similarly, attempted "suppression" or inhibition of film-elicited sadness or fear associated with mild-electric shock via actively "decentering and detaching" oneself was associated with activity in an overlapping set of brain regions.[15–18] Finally, Critchley and Dolan and their colleagues have observed increasing ventromedial PFC, OFC, and ACC activation during biofeedback relaxation, suggesting a role for these structures in volitional autonomic control.[19–21] These findings strongly suggest designations for the ACC, dorsolateral and ventromedial PFC, and OFC in the executive volitional top-down regulatory control of negative affects, impulses, and autonomic arousal states. Another area of the brain that

appears to be involved in interoceptive awareness of negative affective feeling states is the insula, particularly the anterior insula of the right hemisphere.[22,23]

NEUROIMAGING OF MALADAPTIVE AFFECT AROUSAL REGULATION IN PTSD DURING TRAUMATIC MEMORY RECALL

A number of previously published studies have investigated the neural correlates of script-driven recall imagery of traumatic memories in individuals with PTSD (reviewed in Ref. 24). In this paradigm research participants who have previously encountered a traumatic event listen to second-person-narrated scripts of the event while imagining that the event is happening in the present, and while attending to their emotional response. We have observed two unique psychological profiles of responses to the trauma script imagery paradigm: individuals who report *reliving* their traumatic events in the form of flashbacks and experience-associated psychophysiological hyperarousal,[1] and individuals who report *dissociative* reactions including derealization, depersonalization, and a feeling of emotional decenteredness and detachment.[2] These differing phenomenological responses were associated with a distinctive set of neural and cardiovascular correlates.[1,2,24] Individuals with PTSD who reported reliving experiences during trauma script imagery displayed increased heart rate and reduced BOLD response relative to control subjects in the medial PFC, ACC, and thalamus[1]; see FIGURE 1 for a reanalysis of these data with replication using a mixed effects model (i.e., participants as random factor). Reduced activation in medial PFC and ACC is consistent with less regulatory control over affective arousal states on the part of individuals with PTSD, and parallels these individuals' reports of feeling *overwhelmed* by fear and other negative affects during traumatic memory recall. In addition, not only did individuals with PTSD demonstrate less overall activation in ACC in comparison with healthy controls, but they also evidenced a different pattern of functional connectivity of the right ACC (Brodmann Area 32; "cognitive–division") in comparison with controls.[25] In brief, whereas healthy controls demonstrated significantly greater activation in multiple areas of the left hemisphere in concert with right ACC response during traumatic memory recall, consistent with a verbally mediated pattern of recall, individuals with PTSD evidenced significantly greater coactivation with right ACC primarily in structures of the right hemisphere, including the inferior frontal gyrus and posterior cingulate gyrus, consistent with a negatively valenced and primarily nonverbal pattern of recall (see Refs. 24 and 25 for further discussion).

In comparison with individuals with PTSD who reported reliving experiences and associated hyperarousal during the trauma script paradigm, individuals with PTSD who reported a dissociative reaction did not show a significant

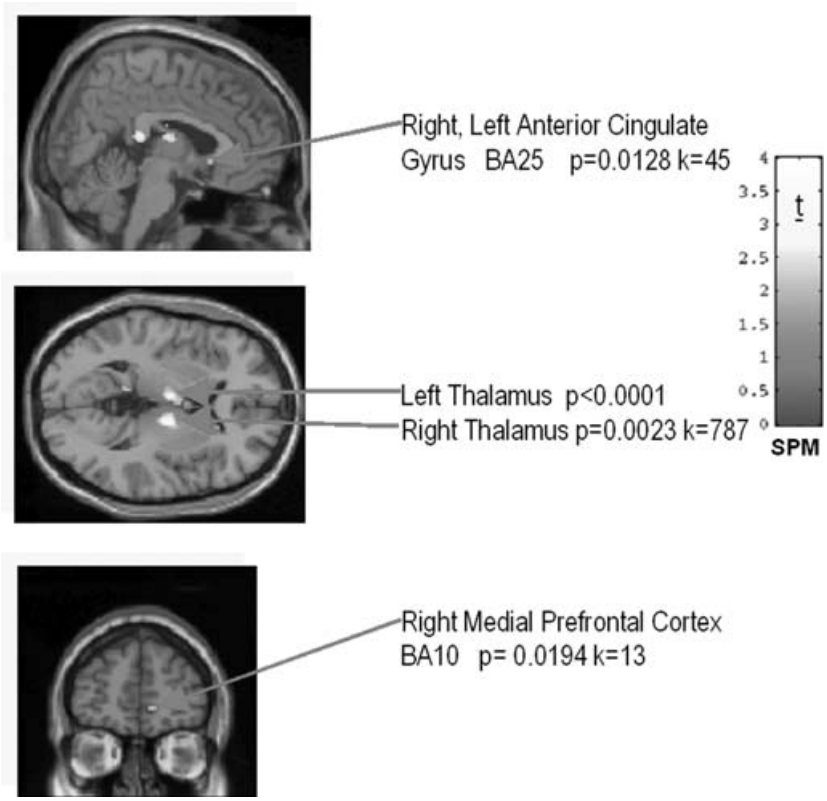

FIGURE 1. fMRI mixed effects analysis (participants as random factor), showing differences in BOLD response during trauma script-driven imagery where control participants ($n = 13$) show greater activation than do PTSD reliving/hyperarousal participants ($n = 11$). $P < 0.05$, extent cluster threshold = 5.

increase in heart rate. In fact, heart rate increase relative to the base line was observed only in a minority of this sample, with the majority showing either a lack of robust heart rate response to the script, or a deceleration of heart rate, consistent with other studies.[26,27] Additionally, individuals with PTSD who dissociated during trauma script recall displayed *increased* activation in the inferior frontal gyrus, medial PFC, and ACC relative to control subjects[2]; see FIGURE 2 for reanalysis of these data with replication using a mixed effects model (i.e., participants as random factor). These neural correlates are consistent with a "super suppression" or robust inhibition of affective arousal during dissociation on the part of these individuals with PTSD, and parallel their self-reported detached, emotionally numb, and out-of-body experiences (depersonalization) during traumatic memory recall. Indeed, the phenomenological reports of individuals who experienced dissociative reactions in this study

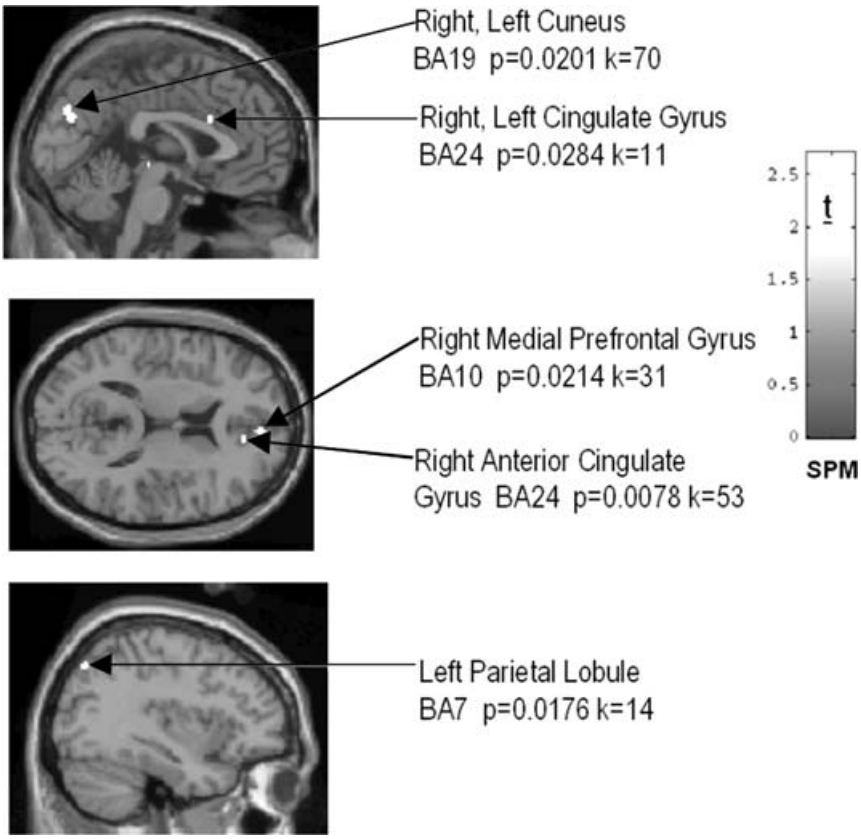

FIGURE 2. fMRI mixed effects analysis (participants as random factor), showing differences in BOLD response during trauma script-driven imagery where dissociative PTSD participants ($n = 10$) show greater activation than do control participants ($n = 13$). $P < 0.05$, extent cluster threshold = 5.

are strikingly similar to the set of instructions given to subjects in Lévesque and Bearegard's studies in order for their subjects to suppress feelings of sadness in response to viewing sad films:

> "Subjects were instructed to reappraise the stimuli by taking a *distance* from these stimuli, that is, to become a *detached* observer. . .. To do so, each subject was told to mentally imagine herself sitting in a movie theatre, watching herself reacting emotionally on the big screen and then feeling *dissociated*, that is, like if the person seen on the screen was not related to her anymore." (Ref. 17, p. 362, italics added; see also Refs. 16 and 18.)

Additionally, individuals with dissociative PTSD demonstrated an altered pattern of functional connectivity of left ventrolateral thalamus during trauma

recall when compared with healthy controls,[28] which included greater coactivation of the right insula, an area noted to be involved in awareness of bodily arousal, and right middle frontal and superior temporal gyri, possibly involved in episodic recall of affect-laden memories (see Refs. 24 and 28 for further discussion).

Historically, psychological theories of dissociation have focused primarily on its functional role in protecting the conscious psyche or the explicit episodic memory system from the horror and terror of traumatic events, not unlike the psychodynamic concept of "repression." In comparison, emerging psychobiological models increasingly focus on the role that dissociative mechanisms may play in the protection of the body and of physical survival during imminent and particularly inescapable threat. Our current view is that the dissociative state that is observed in a subset of individuals with PTSD when prompted by trauma recall may be closely aligned with the defensive strategy of behavioral immobilization observed characteristically in animals situated within the context of inescapable predation, as lucidly described by Nijenhuis and colleagues.[29,30] In a previous article, we hypothesized that the PTSD reexperiencing and hyperarousal response plausibly overlaps with animal defensive flight and pre-predator encounter freezing.[30] In contrast, PTSD dissociation may be more closely aligned with defensive immobilization in the context of acute impending attack by an unequivocally more powerful predator, specifically in situations where a more active defensive strategy, such as fight or flight would predictably entail an increasingly aggressive behavior on the part of the predator.[29,30] An example from the trauma literature would include the frozen, immobile, dissociative state that might be engendered in a child threatened by the provoking drunken rage of his or her physically violent or sexually abusive father. Therefore, whereas we posit that pre-encounter behavioral freezing should be characteristically associated with vigilance, *hyper* arousal, and action readiness, postencounter defensive immobilization should be associated with decreased sensory acuity and *hypo* arousal, consistent with the subjective reports of dissociative PTSD subjects in the fMRI study.[2,28–31]

It is important to note, however, that whereas the dissociative state itself appears to represent a form of *hypo* arousal instigated perhaps distinctly via an appraisal of uncontrollability, its instantiation requires a preceding appraisal of threat presence and fear, which may be associated with transitory *hyper* arousal. In fact, we speculate that the acute hyperarousal preempting the dissociative hypoaroused state may exist on a plane equivalent to or even *higher* in arousal than the hyperarousal response associated with reexperiencing and peritraumatic panic states. It may therefore be instructive to note that, whereas the majority of individuals who experience dissociative symptoms at the time of their trauma will likely also report experiencing some form of peritraumatic panic or hyperarousal, the reverse is not necessarily the case. Accordingly, we would predict that panic hyperarousal states occurring in the context of initial trauma exposure will be better predictors relative to peritraumatic dissociative

experiences in prospective studies of the development of PTSD following trauma exposure (i.e., the extent of peritraumatic panic hyperarousal would be expected to explain a greater amount of variance in PTSD vulnerability relative to peritraumatic dissociation, since individuals who dissociate during trauma exposure may not may be in the majority, and who themselves will likely also report experiencing some form of panic hyperarousal). However, this clinical and statistical observation in itself would in no way deemphasize the theoretical significance of the psychobiological study of peritraumatic dissociative states in PTSD, nor invalidate the possible clinical significance that might be afforded in distinguishing between individuals who demonstrate peritraumatic dissociative responses from those who do not. For example, the hypoaroused state that may ultimately be engendered during peritraumatic dissociation may be related to accentuated activity in the parasympathetic system, directly opposite to what has been found in PTSD hyperarousal.[32] For example, Schore[33] has speculated that activity of the dorsal vagal complex in the medulla may increase dramatically during dissociation, culminating in decreased blood pressure, metabolic activity, and heart rate despite increased circulating adrenaline (see Ref. 31).

Although the script recall imagery paradigm used in extant PTSD studies does not specifically instruct individuals to regulate their affective responses to the scripts, its relatively open-ended format may permit a certain level of regulatory behavior on the part of subjects during the script listening and imagery periods, whether consciously or unconsciously performed. For example, in a yet unpublished study, we observed that participants' self-reported extent of effortful avoidance of fearful hyperarousal symptoms was positively correlated with the degree to which they reported dissociative experiences during the script imagery paradigm.[34] Future studies in our lab will specifically investigate the neural correlates of willful emotion regulation during recall of traumatic memories in individuals with PTSD in comparison with individuals who have demonstrated resilience to the psychopathological effects of traumatic experiences. We predict that resilient individuals will show an increased ability to regulate their emotional responses during both traumatic and nontraumatic stress, corresponding with increasing activity in medial PFC, ACC, and ventral and dorsal lateral PFC.

NEUROIMAGING OF OTHER DYSREGULATED AFFECTS IN PTSD

Whereas the inability to regulate fearful distress associated with exposure to traumatic events is definitional to PTSD, clinical studies have observed that individuals with PTSD typically not only display a dysregulated pattern of the fear–panic–alarm system, but often exhibit a more generalized disturbance in affect arousal regulation, which may include extreme bouts of

irritability-anger, overpowering guilt, loss, and shame, and a disturbed response to physical pain.[35,36] Accordingly, neuroimaging studies have begun to move beyond the analysis of PTSD-case responses toward fear-specific stimuli and situations alone to an investigation of the generalized affective disturbances often present in complex yet clinically representative cases of PTSD. For example, we investigated responses to recall imagery of *non* traumatic sad and anxious memories in PTSD and observed reduced activation in ACC and thalamus similar to what was found during recall imagery of traumatic events.[3] Other preliminary studies have observed altered ACC activation in response to painful thermal stimulation in PTSD, suggesting another complex outcome of chronic exposure to traumatic events may be a disturbance in affective components of the pain system.[37]Finally, although states of guilt, loss, shame, and anger have thus far not received the attention of psychobiological researchers, these symptoms are often as clinically prominent as are fear–anxiety symptoms in certain cases of PTSD,[35,36] and disturbances in a common functional neuroanatomical system, the emotion regulation system, may underlie their pronounced expression in PTSD as well. Future neuroimaging analyses of these complex negative emotions will require ingenuity in research design (e.g., Refs. 38 and 39).

Another equally important though hitherto understudied psychobiological presentation of PTSD involves a set of experiences collectively referred to as "emotional numbing" or "affect restrictive" states. Although levels of autonomic arousal have not been studied specifically during periods of emotional numbing, the current theoretical and clinical consensus is that emotional numbing symptoms represent a form of low arousal nonagitated dysphoria or anhedonia that may oscillate with periods of hyperarousal (e.g., Ref. 40). Emotional numbing symptoms intersect with reports of alexithymia, that is, the diminished ability to identify, label, and "feel" discrete emotions often characteristic of the PTSD population (e.g., Ref. 41, this volume). Moreover, clinical parallels can be drawn between PTSD emotional numbing and alexithymia symptoms and recent empirical descriptions of depersonalization disorder subjects' lack of affective response to negatively valenced stimuli; individuals with depersonalization disorder feel disconnected from their body, and typically exhibit a blunted affective disposition.[42] For example, consistent with findings that skin conductance response to negative affective stimuli is blunted in depersonalized subjects,[43] Phillips *et al*.[44] found that individuals with depersonalization disorder failed to differentiate between aversive and neutral pictures in terms of subjective emotional responses, and demonstrated less activation in left insula, bilateral ACC, and left inferior parietal cortex when viewing affective pictures relative to controls. The comparative absence of insular and parietal response to aversive relative to neutral stimuli in depersonalization disorder may be consistent with an emotional numbing response to these stimuli. Therefore, in the context of negative emotion-eliciting stimuli, we submit that the experience of emotional numbing corresponds with *low* arousal but

nevertheless negatively valenced affective states. However, in contrast to dissociation where an initially strong affective response is suppressed, emotional numbing symptoms may signify an absence of robust primary emotional appraisals in the first place, such as observed in the depersonalization disorder subjects just described. For example, in an event-related potential (ERP) study of sustained auditory attentional vigilance, Felmingham and her colleagues[45] observed decreasing parietal P300 intensity during processing of target tones correlated with increasing emotional numbing symptoms in PTSD, consistent with an earlier report of reduced P300 amplitude correlated with increasing depressive symptoms in PTSD.[46] The P300 component of the ERP is generally accepted as an index of processing resource allocation in frontal–parietal cortex, consistent with a motivated attentional response toward the cue. Therefore, in contrast with vigilance and hyperresponsiveness to threat-relevant cues in PTSD, sensory acuity for nonthreat-relevant cues appears to be diminished according to the preponderance of tonic emotional numbing symptoms. We would predict that this effect would be even stronger for reward-relevant stimuli (e.g., Ref. 47), thereby signifying that decreasing hedonic capacity may be a plausible signature of tonic PTSD emotional numbing. We would also predict that a preponderance of state low arousal emotional numbing symptoms would be associated with reduced activity not only in parietal somatosensory cortex but also in insular cortex. Finally, future research should investigate possible associations between individual differences in emotional numbing symptoms and previous findings of stress-induced analgesia in PTSD.[48,49] In sum, we believe that future neuroimaging investigations of PTSD individuals' ability to "*up*-regulate" themselves out of the frozen, leaden apathy of emotional numbing states are critical to a future more comprehensive understanding of the psychobiology of PTSD.

In conclusion, we submit that a deficiency in the ability to adaptively recruit ACC, medial PFC, and OFC in the active service of emotion regulation in PTSD is not likely to be specific to trauma-related fear processing. Instead, this relative incapacity probably reflects a more generalized deficit that is pertinent to the multitude of complex negative affective states that are often observed clinically in individuals who have suffered long-term exposure to traumatic events in the interpersonal and developmental attachment domains of life. These symptoms plainly include trauma-related fear and intrusive re-experiencing symptoms, but may also include dissociation, marked dysphoria, generalized anxiety, guilt, loss, shame, disturbances in pain processing (e.g., increased analgesia), anhedonia, and emotional numbing. Given the complexity and heterogeneity of affective disturbances often inherent to clinical cases of posttraumatic psychopathology, the current DSM-IV classification of PTSD as an anxiety disorder may need to be reconsidered. Such a diagnostic scheme inherently situates an arguably undue clinical and research focus on symptoms of anxiety and fear, consequently emphasizing less other forms of affective disturbance that may be equally fundamental both to an understanding of the

underlying psychiatric syndrome as well as to the functional status of many individuals presenting with PTSD.

PSYCHOLOGICAL ASSESSMENT AND INTERVENTION

The overlying thesis of this article is that PTSD is most appropriately conceptualized as a psychobiological disorder involving affect arousal dysregulation, including but not restricted to processes involved in fear and anxiety. Accordingly, we believe that psychological assessment and intervention should dutifully reflect this level of complexity.

Prior to clinical intervention, a thorough assessment of cognitive and affective functioning is indicated, so that the chief emotion regulatory difficulties of presenting PTSD patients can be effectively targeted ideographically in treatment. Briere and Spinnazola[50] recently recommended a number of psychometric scales that may assist the assessment clinician in this endeavor. Following a thorough psychological assessment, intervention proceeds in phases, where reprocessing of traumatic memories is preceded first by an emotion regulation skills training and affect arousal stabilization phase.[51–53] The sequencing of the emotion regulation/stabilization phase prior to the traumatic memory reprocessing phase has been regarded as critical in preparing PTSD patients for the psychobiological challenge that they ultimately will face during the traumatic memory exposure phase.[51–53] For example, mindfulness training, as employed in cognitive–behavioral interventions, such as Linehan's *Dialectical Behaviour Therapy,*[54] may facilitate awareness and modulation of emotions in individuals with PTSD, thereby facilitating emotional engagement (and decreasing avoidance, numbing, and dissociation) during traumatic memory exposure (e.g., Ref. 55). Additionally, research suggests that emotion regulatory skills training as well as qualities of the therapeutic relationship itself combine in assisting individuals with PTSD in tolerating and regulating their often intense levels of arousal and distress (e.g., Ref. 56). The focus on improvement in affect arousal regulatory skills in phase-based interventions exemplifies a mastery approach to trauma recovery, which is distinctive from the fear habituation model of PTSD treatment often used to explain the efficacy of single-phase, exposure-based treatments for less complex cases of PTSD.[51–53]

The clinical improvements observed with structured psychological interventions for PTSD are doubtlessly associated with concomitant alterations in neurobiological functioning. However, pre- and post-neuroimaging studies of psychological interventions for mood and anxiety disorders are only beginning to emerge (e.g., Refs. 57–59). We have currently begun a clinical trial of the neurobiological effects of Cloitre and colleagues'[52,53,56] structured psychological intervention for child abuse-related PTSD. Increased ACC, and medial PFC and ventral and dorso lateral PFC activation in the context of emotional

processing paradigms, and attendant alterations in cardiovascular and neuroendocrinological functioning are predicted to be associated with improved affect-arousal regulatory behavior and decreased symptoms in cases of child abuse-related PTSD.

CONCLUSION

This article situated the psychobiology of PTSD within the theoretical framework of affect-arousal regulation. Neuroimaging studies of emotion regulation in psychologically healthy individuals were reviewed, where it has been found that ACC, medial and dorsolateral PFC, and OFC are involved in adaptive emotion regulation. PTSD reexperiencing and hyperarousal symptoms during trauma recall were regarded as being the result of a failure of regulatory inhibitory control over fear-induced arousal and distress, whereas PTSD dissociative symptoms were regarded as being the consequence of an enhanced suppression of fear-induced arousal. Emerging evidence that emotion regulation problems in PTSD typically extend beyond emotions of anxiety and fear was then reviewed, and issues pertinent to psychological assessment and intervention were discussed. We believe that the degree to which significant progress continues to be made in the coming decades of psychobiological study of PTSD will depend upon the degree to which researchers concertedly embrace the complexity of the underlying subject matter. This will entail the psychobiological investigation of the multitude of affective disturbances often accompanying posttrauma psychopathology, especially in cases prompted by recurrent traumatic events occurring in the interpersonal and developmental attachment domains of life.

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