

Posttraumatic Stress Disorder: A Social-Cognitive Perspective

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This study offers a developmentally sensitive conceptual understanding of trauma by viewing posttraumatic stress disorder (PTSD) through the lens of social cognition. First, we justify our focus on social cognition by examining the literature on problematic interpersonal relationships associated with PTSD. Next, we link impaired social cognition to the developmental compromise of mentalizing capacity in attachment relationships. We then integrate the diverse research literature into a social-cognitive model of the development of PTSD. We finally conclude by suggesting directions for future research, as it might be shaped by trends in social-cognitive neuroscience.

Key words: attachment, mentalizing, posttraumatic stress disorder, social cognition. [*Clin Psychol Sci Prac* 19: 229–240, 2012]

While posttraumatic stress disorder (PTSD) is only one among many psychiatric sequelae of trauma, it is a characteristic phenotypical expression following exposure to extremely stressful events (Nietlisbach & Maercker, 2009). PTSD is the only trauma-specific psychiatric disorder for which a set of diagnostic criteria, including a specified etiology, has been developed. In the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev., *DSM-IV*; American Psychiatric Association, 2000), stressors potentially leading to PTSD are defined objec-

tively in criterion A1 and subjectively in criterion A2. The objective criterion focuses on physical threat (i.e., threatened death or serious injury, or threat to physical integrity) and includes not only direct experience of such threat but also indirect exposure (i.e., witnessing or learning about such threat to others). The subjective criterion focuses on the individual's emotional response to the stress exposure, namely, the extent of fear, helplessness, or horror.

These objective and subjective stress-exposure criteria have proven to be problematic. Debate regarding the objective criterion revolves in part around the extent to which the criterion is too broad (e.g., including indirect exposure, such as watching events on TV) or too narrow (Friedman & Karam, 2009; Spitzer, First, & Wakefield, 2007). We contend that the focus on threat to physical integrity is too narrow, given the evidence for the traumatic effects of psychological abuse (Bifulco, Moran, Baines, Bunn, & Stanford, 2002) and psychological neglect (Erickson & Egeland, 1996) as well as research indicating that the majority of themes in posttraumatic reexperiencing symptoms relate to psychological threat (Holmes, Grey, & Young, 2005). The subjective criteria have been criticized as focusing too narrowly on fear to the exclusion of a range of other emotions commonly experienced in the midst of traumatic stress, such as shame, guilt, anger, and disgust (Brewin, 2003; Friedman, Resick, & Keane, 2007). Consistent with this criticism, Holmes et al. (2005) tallied the frequencies of different emotions experienced in the midst of traumatically stressful events and found that, although fear was the most

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prominent emotion, fear along with helplessness and horror comprised only half the emotions; collectively, other emotions were equally prominent.

There are two additional problems in the endeavor to link the syndrome of PTSD to trauma exposure. First, despite the fact that the majority of men and women have been exposed to potentially traumatic events in their lifetime, only a small minority (5–10%) have a history of PTSD (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Second, the symptom cluster of PTSD is sometimes evident in the absence of objectively defined traumatic events. The entire PTSD syndrome has been observed in relation to common stressors such as family or romantic relationship problems, occupational stress, parental divorce, and serious illness or death of a loved one (Gold, Marx, Soler-Baillo, & Sloan, 2005). Long et al. (2008) found that, with some assessment methods, participants reported *higher* levels of PTSD symptoms in response to more ordinary stressors than to ostensibly traumatic stressors.

The seemingly futile aspiration to draw a bright line between traumatic and nontraumatic levels of stress has led some authors to make the radical proposal of dropping the stress-exposure criteria altogether from the diagnosis of PTSD (Brewin, Lanius, Novac, Schnyder, & Galea, 2009). Plainly, stress exposure occurs along a continuum, and researchers have more to gain from going beyond identifying an arbitrary cutoff point of severity to determining what kinds and combinations of stressful events lead to what kinds of symptoms and disorders (Dohrenwend, 2010). More broadly, the inclination to associate trauma with PTSD is problematic. From their developmental perspective, Sroufe, Egeland, Carlson, and Collins (2005) comment, “It is unfortunate that the consequences of trauma, and harsh experience more generally, are sequestered into such a category” (p. 275). In a similar vein, Frueh, Elhai, and Acierno (2010) make the broader point that “the assumption that exposure to life-threatening stressors is the primary cause of a unique set of stress response symptoms is highly problematic and represents a disconnection from and the failure to incorporate the larger body of general stress literature” (p. 263).

Excessive focus on objectively defined levels of stress, even when including subjective emotional

responses, seriously underplays the profound role of individual differences in stress vulnerability as well as the developmental origins of these individual differences. Numerous factors that predispose individuals to develop PTSD in the aftermath of exposure to stress have been identified. Shalev (1996) reviewed the results of 38 studies revealing a wide array of factors that increase the risk of developing PTSD: pretrauma vulnerability (e.g., family history of mental disorders, gender, genetic and neuroendocrine factors, personality traits, early traumatization, negative parenting experiences, and lower education); the magnitude of the stressor, preparation for the event, and immediate reactions to the trauma (e.g., dissociation and coping responses); and posttrauma factors (e.g., emerging symptoms, social support, and other life stress). Prospective research in this area is rare and especially valuable, as the Dunedin longitudinal study exemplifies. Koenen, Moffitt, Poulton, Martin, and Caspi (2007) assessed participants at multiple intervals from birth to age 32 to identify risk factors for PTSD. One set of factors was associated with the likelihood of exposure to traumatic stress (difficult temperament, antisocial behavior, hyperactivity, maternal distress, and loss of a parent in childhood), whereas another partially overlapping set of risk factors was associated with the likelihood of developing PTSD after stress exposure (low intelligence, difficult temperament, antisocial behavior, being unpopular, changing parental figures, multiple changes of residency, and maternal distress). Moreover, a developmental cascade was evident insofar as an accumulation of different categories of risk factors most powerfully predicted PTSD.

The sheer diversity of factors contributing to vulnerability to developing PTSD calls for a unifying theoretical framework to guide future research. This study offers a developmentally sensitive conceptual understanding of trauma by viewing PTSD through the lens of social cognition. Our review proceeds as follows: (a) we justify our focus on social cognition by examining the literature on problematic interpersonal relationships associated with PTSD; (b) we link impaired social cognition to the developmental compromise of mentalizing capacity in attachment relationships; (c) we integrate the diverse research literature into a social-cognitive model of the development of PTSD; and

(d) we conclude by suggesting directions for future research, as it might be shaped by trends in social-cognitive neuroscience. While we recognize that an extensive literature linking attachment trauma to dissociative defenses and symptoms (Barlow & Freyd, 2009; Freyd, 1996; Liotti, 2009) also has implications for compromised mentalizing, we confine the present review to research bearing on impaired social cognition in PTSD, the implications of which have not previously been fully addressed.

IMPAIRED INTERPERSONAL FUNCTIONING IN TRAUMA SURVIVORS WITH PTSD

Several theoretical models of PTSD development emphasize interpersonal and social factors (Brewin, 2005; Brewin, Andrews, & Valentine, 2000; DePrince, 2005; Nietlisbach & Maercker, 2009). These theories have in common the proposition that the pathway from traumatic stress to PTSD may be mediated by interpersonal factors. Previous research reviewed in detail by Nietlisbach and Maercker (2009) has focused on three interpersonal constructs associated with PTSD: (a) poor quality of intimate relationships (Cook, Riggs, Thompson, Coyne, & Sheikh, 2004; Johnson et al., 1996; Solomon, Mikulincer, Freid, & Wosner, 1987); (b) limited disclosure and social acknowledgment of traumatic experience (Maercker & Müller, 2004; Müller et al., 2008); and (c) lack of social support (Brewin et al., 2000; Guay, Billette, & Marchand, 2006; Jankowski et al., 2004; Olf, Langeland, & Gersons, 2005; Schnurr, Lunney, & Sengupta, 2004; Solomon, Waysman, & Mikulincer, 1990).

Research on quality of intimate relationships, disclosure, and social support clearly points to a role for social cognition in interpersonal functioning associated with PTSD. Yet, like much research in social cognition, these constructs are based on the questionable theoretical assumption that social cognition is a property of the person and not the relationship or the interaction between two or more people (Sharp, 2012). This conceptual approach to social cognition ignores the stochastic nature of social interaction. Social interaction is intrinsically dynamic insofar as the thoughts and actions of one agent depend critically on the changing actions (and mental states) of other social agents (Rilling, King-Casas, & Sanfey, 2008). Put

another way, social cognition encompasses not only the sender's viewpoint as a necessary prerequisite for social interaction, but also the receiver's viewpoint (Moskowitz, 2005). It is therefore critical that social-cognitive research in PTSD focus on the capacity of each member of an interacting dyad to take the viewpoint of another—in short, the capacity to mentalize (Fonagy, 1998).

ORIGINS OF MENTALIZING IN ATTACHMENT RELATIONSHIPS

Mentalizing refers to the natural human capacity to interpret the behavior of others within a mentalistic framework—that is, an individual's ability to ascribe desires, feelings, thoughts, and beliefs to others and to employ this ability to interpret, anticipate, and influence others' behavior. Mentalizing and the related construct, theory of mind (Premack & Woodruff, 1978), fall under the broader rubric of social cognition, which refers to the perception, interpretation, and processing of all information relating to a person's social environment and relationships (Moskowitz, 2005; Sharp, Fonagy, & Goodyer, 2008).

A variety of measures have been developed to assess mentalizing (Luyten, Fonagy, Lowyck, & Vermote, 2012); these measures tap into deficits (see Sharp et al., 2008) and distortions (Sharp, 2006) in mentalizing that predispose to psychopathology. Over two decades of empirical research has demonstrated the role of mentalizing and theory-of-mind impairments in the interpersonal problems associated with autism (Baron-Cohen, 2000), borderline personality disorder (King-Casas et al., 2008; Sharp et al., 2011), conduct disorder (Sharp, 2008), depression (Kyte & Goodyer, 2008), schizophrenia (Langdon & Brock, 2008), psychopathy (Blair et al., 1996), and anxiety disorders (Banerjee & Henderson, 2001). This research shows that, beyond a complete lack of mentalizing, there are multiple ways in which mentalizing can go awry and that different aspects of mentalizing are reflected in the heterogeneity of different psychiatric disorders (see Sharp & Venta, 2012, for a review). We observe, for instance, hypermentalizing (hypervigilance to mental states) in patients with borderline personality disorder (e.g., Sharp et al., 2011), reduced mentalizing in psychopathic individuals (e.g., Blair et al., 2004), and distorted mentalizing in conduct disorder (e.g., Sharp, Croudace, & Goodyer,

2007). These variations in mentalizing styles have justified the need to identify factors associated with individual differences in mentalizing. Recently, emerging theory and data have lent support to the proposal that the quality of early attachment relations is an important source of individual differences in mentalizing capacity (Fonagy & Luyten, 2009).

John Bowlby's (1973, 1980) attachment theory has been one of the most influential psychological theories of the 20th century (Cassidy & Shaver, 2008). Anyone who has observed the immediate and engaged response of a mother to her distressed baby would agree with Bowlby that there is something innate in the preparedness of the infant to seek protection from attachment figures, coupled with the attachment figures' natural disposition to provide care. The reciprocity between caregiver and infant includes behaviors such as touching, holding, and soothing on the parent's side and smiling, clinging, and crying on the infant's side. This reciprocity creates an enduring bond between caregiver and infant. Bowlby referred to this enduring bond as attachment, and he saw it as the foundation for the infant to develop internal working models of self and other that function as templates for future relationships.

Recently, the internal working model has been reinterpreted in the context of the social information processing approach to function in a similar way to a cognitive schema (Dykas & Cassidy, 2011). As such, the internal working model is viewed as a cognitive schema that influences the ways in which individuals obtain, organize, and operate on attachment-relevant social information (Bowlby, 1980). The schema has three functions: It stores information about interpersonal events with attachment figures; it generates expectations about how attachment figures will behave in subsequent interactions; and it provides, in the long term, important information to the individual about the self in the context of relationships. Therefore, individuals are likely to use different (sometimes biased) rules to process attachment-relevant social information as a function of whether they have a secure or an insecure attachment schema (Dykas & Cassidy, 2011).

Empirical studies across all developmental periods have established the link between quality of attachment and social information processing in general, and theory of mind or mentalizing in particular (for a full and recent

review, see Dykas & Cassidy, 2011). Here, we highlight some key contributions. Bretherton, Bates, Benigni, Camaioni, and Volterra (1979) were the first to demonstrate a relation between infant attachment and early social understanding. They found that infants who were securely attached at 12 months used more protodeclarative pointing at 11 months than other infants. In addition, a number of studies have reported associations between the quality of children's primary attachment relationships and the passing of standard theory-of-mind tasks (e.g., de Rosnay & Harris, 2002; Fonagy, Redfern, & Charman, 1997; Fonagy, Steele, Steele, & Holder, 1997; Fonagy & Target, 1997; Harris, 1999; Meins, Fernyhough, Russel, & Clark-Carter, 1998; Ontai & Thompson, 2002; Raikes & Thompson, 2006; Steele, Steele, Croft, & Fonagy, 1999; Symons, 2004; Thompson, 2000). For example, the Separation Anxiety Test, a projective test of attachment security, predicted belief-desire reasoning capacity in 3½-year-old to 6-year-old children, controlling for age, verbal ability, and social maturity (Fonagy et al., 1997a, 1997b). In this task, the child is asked what a character would feel, based on his or her knowledge of the character's belief. Quality of belief-desire reasoning was predicted from attachment security in infancy: 82% of babies classified as secure with mother at 12 months passed the belief-desire reasoning task at 5½ years, whereas 46% of those who had been classified as insecure failed (Fonagy et al., 1997a, 1997b). Infant-father attachment (at 18 months) also predicted the child's performance.

In summary, the extant literature on attachment and mentalizing suggests that mentalizing capacity is either delayed or impaired in the case of insecure attachment. Reddy (2008) offered a comprehensive account of factors that contribute to the emergence of mentalization, proposing that mentalizing emerges through facilitation by a second person. Reddy critiques literature that construes the development of mentalizing as an individual rather than a social process, despite the evident and profound social function that mentalization plays in human behavior. She suggests that we come to know other minds only through interacting with other persons and by observing their responses to us and our responses to them. This process requires engagement with other persons, such that the starting point for understanding other minds is attachment relationships.

In a similar vein, Sharp and Fonagy (2008) have argued that evolution may have selected the attachment relationship for conveying knowledge about minds to the human infant, and that the quality of the relationship with the attachment figure will therefore impact profoundly the rate of development and the child's competence in mentalizing.

Beyond mentalizing or theory of mind, attachment security also has been shown to relate to other aspects of social cognition. In adolescents, attachment security has been shown to relate to attention to positive social feedback (Cassidy, Ziv, Mehta, & Feeney, 2003) and positive memories of social interactions with attachment figures (Dykas, Woodhouse, Ehrlich, & Cassidy, 2010). Insecure adolescents also have been shown to perceive and generate expectations and attributions about others in a negatively biased schematic manner, whereas their secure counterparts process such information in a positively biased schematic manner (Zimmermann, 1999). The same negative bias in social information processing has been demonstrated for adults' attention to social information (Atkinson et al., 2009) and memory for social information (Mikulincer, 1998), as well as expectations and attributions of relationships with romantic partners (Crowell et al., 2002) and offspring (Slade, Belsky, Aber, & Phelps, 1999).

Insecure attachment not only impairs the development of mentalizing capacity and engenders negative biases in other aspects of social information processing as reviewed earlier; it also adversely affects an individual's capacity for engaging with distressing attachment-related experience. In particular, a large body of literature has shown suppression-related attentional processes in relation to insecure attachment. For instance, Main, Kaplan, and Cassidy (1985) showed that 6-year-old children with insecure attachment during infancy showed greater difficulty attending to family photographs than secure children. Children with insecure attachment styles also suppress memories of attachment-related experimental stimuli (Dykas & Cassidy, 2011; Kirsh & Cassidy, 1997).

ATTACHMENT, MENTALIZING, AND PTSD: A SOCIAL-COGNITIVE MODEL

Extensive research links attachment security to PTSD and stress responses in humans. This research is

reviewed in more detail elsewhere (Fonagy, Gergely, & Target, 2007; Mikulincer & Shaver, 2007). In short, individuals scoring high on attachment preoccupation or anxiety tend to engage in hyperactivation strategies (intensifying negative emotional states) in response to distress. In contrast, individuals who score high on avoidant or dismissing attachment tend to engage in deactivation strategies (distancing themselves from emotional situations). Avoidant individuals will be likely to restrict the acknowledgment of distress, dismiss its importance, and erect barriers against their own stressful affects and thoughts (Besser & Neria, 2009). Consequently, they appear to be less sensitive to stress (see Mikulincer & Shaver, 2007, for a review).

Research on adults echoes the findings in infants, which support the link between maltreatment in attachment relationships and the most profound form of attachment insecurity, namely, disorganized attachment (see Schuengel, Bakermans-Kranenburg, & Van Ijzendoorn, 1999, for a review). For example, in Sroufe and colleagues' meticulous longitudinal study (Carlson, 1998; Sroufe et al., 2005), attachment disorganization was associated with physical abuse (e.g., intense and frequent spanking, angry parental outbursts resulting in serious injuries), psychological unavailability (e.g., parental unresponsiveness or detachment), and neglect (e.g., failure to provide physical care or emotional care). Moreover, attachment disorganization in infancy increased the likelihood of developing PTSD symptoms in response to trauma later in childhood (MacDonald et al., 2008). Sroufe and colleagues' study also demonstrated that the impact of attachment disorganization and early trauma on personality functioning was mediated by social cognition in adolescence (age 12; Carlson, Egeland, & Sroufe, 2009). Specifically, the authors conclude from the mediation analysis that "representations and related mentalizing processes are viewed as the carriers of experience that link early attachment to later psychopathology" (p. 1328).

In sum, research we have reviewed suggests that impaired mentalizing, with roots in trauma-related attachment insecurity, may play a role in the development of subsequent trauma symptomology, including PTSD. Yet little research has examined directly mentalizing capacity in trauma survivors with PTSD. Preliminary work with combat veterans suggests that

individuals suffering from PTSD showed deficits in social cognition involving emotional numbing rather than clinical symptoms such as anxiety and depression (Mazza et al., 2012). A small-scale study of trauma survivors showed that the PTSD group manifested lower empathic resonance but revealed few clear indications of other impairments in social cognition (Nietlisbach, Maercker, Rossler, & Haker, 2010). From the little empirical work in this regard, two social-cognitive approaches to PTSD have been developed. First, Maercker (2008) developed the Social Facilitation Model of PTSD, suggesting that perceived aspects of the self, others, and the world interact to increase or decrease the symptoms and course of PTSD. Central to this model are symptom facilitation or recovery processes that affect the fear network of trauma memories and the ability to integrate traumatic experiences into one's life through social relationships (Nietlisbach & Maercker, 2009). Second, DePrince (2005) developed a model of revictimization risk for persons with PTSD, based on the proposition that prolonged exposure to trauma results in social-cognitive impairments that, in turn, increase the risk for future victimization. In particular, trauma survivors with PTSD are thought to lack the social-cognitive capacity to accurately detect violations in social contracts; ordinarily, this capacity enables people to avoid or withdraw from relationships in which they are at risk to be harmed.

Central to both Maercker's (2008) and DePrince's (2005) work is the potential role of social cognition or mentalizing to either facilitate (Maercker) or inhibit (DePrince) socially adaptive responses to trauma. Here, we combine this central role for mentalizing with what is known about the link between mentalizing and attachment security (reviewed earlier; Dykas & Cassidy, 2011) to develop a social-cognitive model of PTSD that also fits with contemporary cognitive-behavioral, schema-based models of PTSD. Foa and colleagues (Foa & Hembree, 2007; Foa, Huppert, & Cahill, 2006) proposed an account of traumatic memory as it relates to PTSD (Foa & Hembree, 2007), suggesting that on the basis of our experience and memory, we develop cognitive fear structures (schemas) that enable us to escape danger: "The fear structure includes representations of the feared stimuli

(e.g., bear), the fear responses (e.g., heart rate acceleration), and the meaning associated with the stimuli (e.g., bears are dangerous), and the responses (e.g., fast heartbeat means I'm afraid)" (p. 12). Normal fear structures are based on realistic threats, and they serve as guides for action. PTSD is associated with maladaptive fear structures that do not accurately represent threat: Harmless stimuli (e.g., the mother berating her child in the department store) are perceived as threatening, and they evoke excessive physiological and emotional arousal (e.g., panic) as well as maladaptive escape and avoidance responses (e.g., rushing out of the store).

In our social-cognitive adaptation of cognitive schema-based models such as Foa's, we integrate the social-information processing approach to attachment-relevant experiences, such that early traumatic life experiences with caregivers lead to the establishment of maladaptive attachment-based schemas of self and other. Through repeated daily adverse experiences with caregivers, a maltreated infant begins to acquire event-based information of her attachment figure as unavailable, frightening, unresponsive, and insensitive to her needs for contact and desire for autonomous exploration. Unconscious scripts (Vaughn et al., 2006; Waters & Waters, 2006) develop, providing the infant with a causal-temporal prototype of the way attachment-related events unfold (e.g., "when I am hungry and ask for food, I am slapped"). Over time, these scripts become attachment schemas that filter the ways in which the growing child, adolescent, and adult obtain, organize, and operate on attachment-relevant social information. Especially when an individual is then confronted with a traumatic event in the interpersonal realm (e.g., loss, bereavement, rape, sexual abuse, rejection, social exclusion, bullying), the attachment-related schema is activated, leading to maladaptive social-cognitive processing at the procedural level of automatic thoughts. Impaired social cognition, in turn, prevents the individual from effectively making use of current attachment relationships or social support structures to dampen the negative impact of the trauma. The reduction in a potentially important protective factor (social support and connection) then puts the individual at risk for developing the behavioral, cognitive, and emotional symptoms of PTSD (see Figure 1).

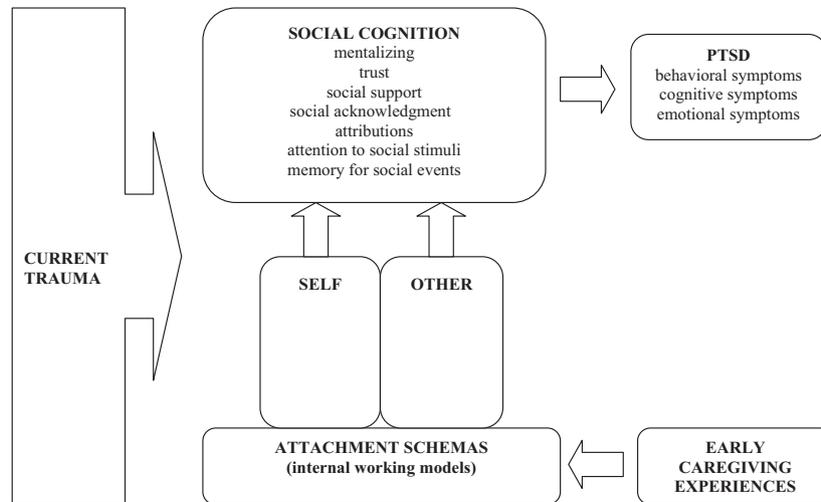


Figure 1. Social-cognitive model of PTSD.

IMPLICATIONS FOR RESEARCH ON PTSD

Originally, PTSD was conceptualized as a normal response to overwhelming psychic trauma (Brewin et al., 2000). Partly due to accumulating evidence for wide variation in the prevalence of PTSD following exposure to different kinds of stressors, there is increasing acceptance of the idea that exposure to a trauma may not always be sufficient to explain the development of PTSD and that individual vulnerability factors have a role to play in understanding this condition (e.g., Yehuda & McFarlane, 1995). In this article, along with others (DePrince, 2005; Maercker, 2008), we have suggested social cognition as a key factor that relates a certain level of traumatic stress to the syndrome of PTSD. In so doing, we have offered a developmentally sensitive conceptual model of PTSD through the lens of social cognition that may guide future research devoted to increasing our understanding of the interpersonal aspects of PTSD.

Future research can benefit greatly from longitudinal designs to track the impact of distal experiences of caregiving on the formation of attachment-related schemas and associated social-cognitive processing as proximal stressful life events occur. While such designs are ideal, subcomponents of the model depicted in Figure 1 also may be tested. For instance, a meditational model testing the links between insecure attachment patterns, compromised mentalizing, and PTSD symptoms will help identify mentalizing as the on-line,

here-and-now expression of schema-based biases in interpersonal relatedness. Adding a neurobiological component to this model, either at the distal level of caregiver experiences (e.g., Fonagy, 2012; Strathearn, 2011; Strathearn, Fonagy, Amico, & Montague, 2009; Strathearn, Iyengar, Fonagy, & Kim, 2012; Strathearn, Li, Fonagy & Montague, 2008) or at the more proximal level of social-cognitive processing in the here-and-now in relation to PTSD, would serve to identify valuable targets for treatment at the biological endophenotypic level, in addition to cognitive and emotion-based interventions.

Adding neurobiological measures to the social-cognitive model also enables a conceptualization of PTSD in terms of the underlying neurobiology of the disorder instead of its behavioral phenotype. Such a reconceptualization will fit well with the broader recent trend in psychiatry to move away from the current nosological framework guiding research (Sharp, Monterosso, & Montague, 2012). This move is motivated by the limitations of the current nosological framework for diagnosing psychiatric disorders (National Advisory Mental Health Council [NAMHC] Workgroup, 2010), which include the limitations of current pharmacological and behavioral treatments for psychiatric disorders, as well as significant advances in brain sciences over the last 10 years. Together, these developments lend support to the view that the current nosological framework represented by the *DSM-IV* and

ICD-10 exhibits serious shortcomings with respect to validity (Insel & Cuthbert, 2009). In response, the strategic plan of the National Institute of Mental Health (NIMH) aims to “develop, for research purposes, new ways of classifying mental disorders based on dimensions of observable behavior and neurobiological measures” (National Institute of Mental Health, 2008). In addition, in 2006, the NIMH established the new discipline of translational science to inform targeted treatment development. In this approach, basic science develops models for understanding normative behavior in healthy individuals. These models are then applied to psychiatric populations to identify biomarkers or endophenotypes that point to the mechanisms of attention, memory, and other higher cognitive processes underlying the behavioral phenotypes of psychiatric disorder. Biomarkers refer to characteristics that are measured objectively as an index of a pathogenic process or as a response to treatment (Carter et al., 2011), while endophenotypes refer to well-specified physiological or behavioral measures that occupy the terrain between disease symptoms (behavioral phenotypes) and risk genotypes (Insel & Cuthbert, 2009). The final step in the translational approach involves testing the biomarker as a mechanism of change in clinical trials. For psychopathology, the modern translational goal is to explain mental phenomena at multiple levels ranging from neurobiological to psychological, but with enough detail so that consequences at one level generate testable predictions at another.

While a number of models describing the functional neuroanatomy of PTSD symptom development emerged over the past decade, these models mainly focus on PTSD as a state of heightened responsivity to threatening stimuli or as a state of insufficient inhibitory control overexaggerated threat-sensitivity (Garfinkel & Liberzon, 2009). Findings of neuroimaging studies in PTSD repeatedly converge on a number of key structures such as the amygdala, anterior cingulate cortex, medial prefrontal cortex, insula, and hippocampus. While these areas are central to threat-related processing, they are also central to social-cognitive processing in the brain (Adolphs, 2001, 2003; Montague, King-Casas, & Cohen, 2006), pointing to the potential value a social-cognitive model of PTSD holds for future research and development of treatment.

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Received July 31, 2012; accepted August 27, 2012.