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Social Bonds and Posttraumatic Stress Disorder

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Abstract

Retrospective and prospective studies consistently show that individuals exposed to human-generated traumatic events carry a higher risk of developing Posttraumatic Stress Disorder (PTSD) than those exposed to other kinds of events. These studies also consistently identify perceptions of social support both before and after a traumatic event as an important factor in the determining vulnerability to the development of PTSD. We review the literature on interpersonal traumas, social support and risk for PTSD and integrate findings with recent advances in developmental psychopathology, attachment theory and social neuroscience. We propose and gather evidence for what we term the social ecology of PTSD, a conceptual framework for understanding how both PTSD risk and recovery are highly dependent on social phenomena. We explore clinical implications of this conceptual framework.

Keywords

PTSD; social support; social neuroscience; anxiety disorders; attachment

INTRODUCTION

There is accumulating evidence that phenomena such as social support, social cognition, and attachment organization contribute to emotion regulation under conditions of traumatic stress and, more particularly, contribute to risk for or protection against posttraumatic stress disorder (PTSD). In the past few years, several articles have been published summarizing the definition, risk factors, and treatment of PTSD (e.g., Ballenger et al. 2004, Davidson et al. 2004, Foa 2006, Nemeroff et al. 2006). Increasingly, researchers and clinicians emphasize the emotional components of PTSD. Specifically, an individual's inability to adequately modulate intensely emotional memories is increasingly seen as leading to symptoms of re-experiencing, hypervigilance, and ultimately avoidance and numbing (Cahill 1997, Cahill et al. 2003, Frewen & Lanius 2006, Kazui et al. 2000, Pitman & Delahanty 2005, Quirk et al. 2006, Rauch et al. 2006, Rothbaum & Davis 2003, Shin et al. 2006), and the resolution of fear and various other associated emotions is critical to recovery from the disorder. In this review, we propose and gather evidence for what we term the social ecology of PTSD, a conceptual framework for understanding how both PTSD risk and recovery are highly dependent on social phenomena.

We review and synthesize an understanding of PTSD in its social context, drawing from the fields of epidemiology and developmental psychology, the emerging sciences of attachment and social neuroscience, and the PTSD intervention literature. In Part 1, we review the evidence that interpersonal traumas are more pathogenic for PTSD, indicating the particular salience of

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human interactions in eliciting fear and other trauma-related responses. In Part 2, we describe research linking social support as it occurs both before and after a trauma, and we note the consistency with which this social phenomenon is among the most powerful influences in both risk for and recovery from PTSD. The data suggest that social support is an effective emotion regulator where the behaviors of others can soothe or exacerbate trauma-driven fears. In Part 3, we describe the first social bond, that between caregiver and child, within the context of attachment theory and review the literature on the ways in which the adult caregiver influences the child's responses to traumatic events, particularly in the modulation of feelings of fear versus those of safety. Childhood abuse and other early life adversities are significant risk factors for PTSD in adulthood. We suggest this risk factor exerts its influence in part through enduring diminished expectations of support from others and similarly chronic and possibly related compromised capacity for emotion regulation. We highlight studies in both humans and animals that identify the influences of parental stress and traumatic reactions on the biology of stress responsiveness in offspring, suggesting that social phenomena such as attachment behaviors and other parent-child interactions have immediate and enduring influences on stress-regulating biological systems. In Part 4, we report on studies investigating the neurobiological circuitry of fear, which identify ways in which social ties and social perceptions modulate fear reactivity in the brain, and discuss the implications for understanding the “social brain” of individuals with PTSD. In Part 5, we review the neurochemistry of social bonds to suggest that in addition to a fear system that has been well described, there may be a “safety” system based upon the brain circuits responsible for social affiliation. We focus on the neuropeptides oxytocin and arginovasopressin, which appear to be integral in mediating social affiliation. In Part 6, we conclude with the clinical implications of these findings.

In summary, we propose that human social experience has a particularly salient if not central role in the way an individual responds to trauma, beginning with the first social bond, the parent-child relationship, and extending to experiences in adulthood at both the dyadic and community or group level. Evidence from neurobiology and neurochemistry is beginning to identify ways in which social phenomena modulate fear circuitry in the brain and has suggested that social experience can shape and alter brain behavior and chemistry in the context of traumatic stress. PTSD may serve as a useful model in understanding the fundamental issues in the evolution of the role of social bonds in the assessment of threat and the management of fear responses.

PART 1: INTERPERSONAL TRAUMAS ARE WORSE

Humans experience traumatic events differently from all other animals because we ascribe meaning to events that befall us. The experience of fear associated with a trauma will reflect, in some way, the meaning ascribed to the event. Supporting this claim is the observation that not all traumas are equivalent in the risk of subsequent PTSD. Traumatic injuries caused by other people are the most likely to lead to PTSD. Indeed, this is so pervasive an observation that it is included in the characterization of PTSD in the Diagnostic and Statistical Manual of Mental Disorders, i.e., that PTSD may be especially severe or long lasting when the stressor is of “human design” (Am. Psychiatr. Assoc. 2000).

Three symptoms clusters comprise the diagnosis of PTSD: (*a*) re-experiencing symptoms (e.g., flashbacks, intrusive thoughts and images) (*b*) emotional numbing and avoidance of reminders of the trauma (e.g., places, people, thoughts), and (*c*) hyperarousal (e.g., increased startle response, irritability) (Am. Psychiatr. Assoc. 2000). However, for the diagnosis of PTSD to be considered, the individual must experience an event and have a response that entails certain characteristics. The event must involve an experience of threat to one's physical well-being or witnessing (seeing, hearing about) the death, injury, or threat to physical well-being of another person. In addition, the individual's subjective reaction must include but is not limited to the

experience of fear, horror, and helplessness. If the event and the individual's reaction fulfill such criteria, the diagnosis of PTSD can be considered. The higher rate of PTSD related to events generated by “human intent” highlights the importance of subjective experience as a contributor to the development of PTSD. Although there is some room for argument, the cause of an event is often a subjective perception that varies from person to person and lends a specific meaning to the event and its long-term consequences. The appraisal of an event as human-caused appears to be particularly fear inducing. The following studies report on rates of PTSD by type of event. They support the observation of the salience of human-caused events in the generation of PTSD and allow some speculation about why this is so.

Epidemiological studies have consistently reported relatively higher rates of PTSD for events resulting from human intent. The most statistically and methodologically sound data on the incidence and prevalence of different types of mental illness in the United States comes from the National Comorbidity Survey (NCS) (Kessler et al. 1994, 2005; Kessler & Merikangas 2004). The NCS survey data indicate that among men and women who report rape as their most upsetting trauma, 65% of men and about 46% of women developed PTSD from this. For men, other traumas associated with a high rate of PTSD were combat exposure (conditional probability = 38.8%), childhood neglect (23.9%), and childhood physical abuse (22.3%), whereas among women, high rates of PTSD followed childhood physical abuse (48.5%), sexual molestation (26.5%), physical assault (21.3%), and being threatened with a weapon (32.6%). These findings are consistent with earlier studies (Kilpatrick & Resnick 1992, March 1992). Traumas of a nonpersonal nature (e.g., “accident” or “natural disaster with fire” or witnessing a trauma) had conditional probabilities for PTSD of less than 10% (Kessler et al. 1995), about half that associated with events related to human intent.

A Swedish national probability sample study of 3000 participants investigated trauma type as well as gender and ethnicity as factors influencing the development of PTSD following a trauma. Of the 1824 respondents reporting a traumatic event, type of trauma independently explained 16.7% of the variance in the rate of PTSD and was the strongest predictor of PTSD symptoms/severity. Of the specifically assessed traumas (robbery, physical assault, sexual assault, tragic death, war, and traffic accident), only traffic accidents did not independently contribute to predicting PTSD diagnosis, with sexual assault being the most likely to predict PTSD (Frans et al. 2005).

It should be noted that the study of interpersonal traumas (e.g., rape or childhood physical/sexual abuse), is often complicated by the problem of retrospective self-report, which is vulnerable to memory biases or distortions. Even events that have been corroborated by multiple observers, such as large-scale disasters, suffer from the absence of independent verification regarding the presence and exact type of exposure for any individual. For this reason, prospective studies or those from the military in which there is independent corroboration of trauma exposure (e.g., documentation of soldiers assigned to combat areas) are critical (e.g., Dorhenwend et al. 2006). The results of such studies have been consistent with retrospective studies, supporting the general finding of differential rates of PTSD by type of events. Accordingly, of particular importance is a prospective study by Shalev & Freedman (2005) that assessed rates of PTSD arising from terror attacks as compared to motor vehicle accidents within the same Israeli community, interviewing survivors at one-week and four-months post trauma (Shalev & Freedman 2005). Terror attack survivors developed PSTD at twice the rate of survivors of motor vehicle accidents (37.8% versus 18.7%). This study provided a rare opportunity to assess two different types of trauma (interpersonal and noninterpersonal) for which there is documentation and that result in differential rates of PTSD. Of interest, terror survivors who developed PTSD did not statistically differ from motor vehicle accident survivors with PTSD at one-week post trauma on scores of trauma symptoms,

depression, anxiety, or dissociation. This would suggest that an as-yet unmeasured difference exists between the perceptions of victims of interpersonal and noninterpersonal traumas.

A few studies explore factors that provide some explanation for the apparent power of interpersonal violence as a risk factor for PTSD. First, appraisal of threat is higher when it is of an interpersonal nature. In a meta-analysis of PTSD risk factor studies, Ozer et al. (2003) found that in the civilian population, the predictive effect of perceived life threat on development of PTSD was greater when the traumatic event was interpersonal violence (effect size, or ES, = 0.36) than when the trauma was an accident (ES = 0.20), suggesting that fearing for one's life is more often associated with interpersonal violence. In addition, such traumas are associated with higher levels of subjective distress. Frans et al. (2005) found that perceived distress and trauma frequency explained 10 times more of the variance in the development of PTSD than did ethnicity or gender. In addition, for a given level of reported distress, women and men had similar odds of developing PTSD. This is of interest as it has been repeatedly demonstrated that, controlling for type of trauma, rates of PTSD are generally twice as high for women as for men. Thus, subjective distress seems to be a useful variable and potent mediator or moderator of risk for PTSD.

King et al. (1995) reported that in combat, the experience of seeing human beings severely maimed and killed, whether friend or foe, appears to be more subjectively disruptive than exposure to harm only. The authors reported a specific effect on PTSD risk from exposure to atrocities or episodes of extraordinarily abusive violence distinct from the effect of the perceived threat to one's own life. Thus, both the experience of threat to one's life as well as that to another share in common increased risk for PTSD. King and colleagues (1995) suggest that there is a special horror in violating basic norms of human conduct that is distinct from fearing for one's life. This may reflect the evolutionary significance of social bonding, whereby the species survival has depended on the ability to form cooperative social networks based on trust and norms of behavior. Exposure to cruelty, perversion, or betrayal may lead to a greater sense of threat or fear as this represents not just the risk of physical injury but also the breakdown of social norms as well as the sense of safety associated with being a member of a rule-guided community.

These data highlight the salience of perceptions of humans as actual or potential agents of harm, against oneself or others, in contributing to emotional distress and more specifically to the development of PTSD. The relevance of the perception of others as helpful or hurtful before or after the event—as distinct from agents of the event—is discussed in the next section.

PART 2: SOCIAL NETWORKS AND SOCIAL SUPPORT

There is a large literature regarding the role of social support in influencing the mental health consequences of stressful life events (such as chronic illness and difficulties with employment), with the general and rather robust finding that support helps buffer against psychological distress (Cohen & Wills 1985). The salience of social support as it relates to PTSD has been demonstrated in two meta-analytic studies. Brewin et al. (2000) found that social support was the strongest correlate of PTSD (ES = 0.4). Ozer et al. (2003), who included 21 studies not analyzed by Brewin et al. (2000), also found that social support was a robust predictor of PTSD, with an effect size of 0.29, making social support the second strongest predictor of PTSD risk, after peritraumatic dissociation (ES = 0.35). Studies in these meta-analyses include both retrospective and prospective designs. Recognizing the potential limitations of retrospective studies, Brewin et al. (2000, p. 752) assessed the potential moderating effect of study design on predictors of PTSD and reported that the retrospective versus prospective nature of the study design did not influence the identified relationship between social support and PTSD.

Debate about the nature of the relationship between social support and PTSD persists, however, as some studies indicate that social support exerts its influence as a protective factor against the risk of PTSD, whereas other studies suggest that the relative absence of support is salient because it creates an increased risk for PTSD. Our review of the literature leads us to conclude that both types of experiences can occur, sometimes even simultaneously, and the relative impact of each is context sensitive relative to the nature of the trauma, the individual's needs, and the nature of the social or interpersonal relationships.

Measures of social support either ask people about functional support, which refers to the individual's perception or experience of social interactions as helpful or unhelpful, or assess structural support, which refers to external aspects of the individual's social network (e.g., size and complexity of the social network, actual support provided). Notably, both measures rely on self-report; investigators rarely, if ever, can independently verify the number of friends, the nature of the relationships, or the qualities of the interactions. Studies that have directly compared the contributions of functional support with those of structural support indicate that posttrauma distress is more greatly influenced by functional support than by structural support (Kaniasty & Norris 1992, Norris & Kaniasty 1996), and measures of quantitative aspects of social networks tend to be less predictive of mental health outcomes than do measures of the qualitative perception of social network interactions (Kessler et al. 1985, Sarason et al. 1987, Shinn et al. 1984). These findings indicate that the reported subjective experience of the helpfulness of others is more relevant than any quantitative measure of the social network reinforces the central role that subjective appraisal appears to have in determining the development and course of PTSD.

Studies in combat veterans provide a first picture of how social factors affect the development of PTSD. The National Vietnam Veterans Readjustment Study (NVVRS; Kulka et al. 1990) provides the most well characterized sample of combat veterans. Importantly, a recent reanalysis of the NVVRS sample confirmed the reports of traumatic exposures using multiple independent historical records, indicating that the retrospective reports of trauma in this sample were highly accurate (Dohrenwend et al. 2006). King et al. (1998) completed a series of studies on data from the NVVRS. Their initial findings indicated that for both men and women, the most important postwar mediator of the risk for PTSD was perceived social support and that social support was negatively correlated with PTSD. The authors conclude that social support may serve to offset the deleterious consequences of traumas on PTSD risk (King et al. 1998). Several other studies show a consistent positive effect of social support on reducing risk of PTSD among prisoners of war (Engdahl et al. 1997), United Nations soldiers (Kaspersen et al. 2003), and among Albanian Kosovars after the Balkan civil war (Ahern et al. 2004).

At least one prospective study has identified social support as both a risk and protective factor (Koenen et al. 2003). In a 14-year prospective study of American Legionnaires whose combat exposure was in Vietnam, veterans with PTSD who showed more community involvement were more likely to show remission of their PTSD over the course of the study. On the other hand, veterans who reported more perceived negative community attitudes at homecoming were more likely to have a chronic course of PTSD. These two variables both indicate a degree of connectedness to a social network—greater community involvement suggests an ability to reintegrate into a social network and form social bonds, whereas experiencing negative community attitudes upon homecoming suggests a feeling of ostracism, isolation, and a weakening of social bonds. This study provides an example of the differing influences of social support: Positive social network interactions can facilitate resolution of PTSD while negative interactions contribute to its maintenance.

Retrospective studies of adults who have experienced childhood physical and sexual abuse provide another perspective on the interaction between traumatic events and social support.

Positive social support appears to mitigate the negative effects of child abuse on measures of general psychological adjustment (Conte & Schuerman 1987, Everson et al. 1989, Gold et al. 1994, Runtz & Schallow 1997, Wyatt & Mickey 1987) and to attenuate PTSD symptom severity (Hyman et al. 2003). Schumm and colleagues (2006) studied a large community sample of low-income, predominantly minority women, and found that among women who were victims of both childhood abuse and adult sexual assault, social support had a large effect size in protecting against PTSD symptom severity (ES = 0.83). Similarly, in a prospective study, Bal et al. (2005) found that among adolescents, higher levels of crisis support immediately following disclosure of sexual abuse protected against the development of internalizing symptoms, which included posttraumatic stress symptoms, as measured by the Trauma Symptom Checklist for Children.

In contrast, several retrospective studies of adult rape victims have shown that the perception of blame and unsupportive social responses are strongly associated with psychological distress, whereas positive social interactions do not appear to bestow any benefit (Davis et al. 1991, Filipas & Ullman 2001, Ullman 1996a, Ullman & Filipas 2001). Similarly, a prospective study evaluating predictors of PTSD in adult survivors of sexual and nonsexual assault found that "interpersonal friction," defined as overt arguments and assessed at two-week post event in multiple social domains, predicted PTSD severity at three-month follow-up (Zoellner et al. 1999). In addition, after controlling for initial PTSD and depression symptoms, interpersonal friction was a stronger predictor of PTSD severity than was the type of assault. In contrast, initial positive social support was not predictive of later PTSD symptoms.

A second prospective study found that, controlling for baseline PTSD symptoms, negative reactions from the social network and dissatisfaction with support were predictive of both the onset and severity of PTSD symptoms at six months, whereas positive support had no effect on PTSD onset or course (Andrews et al. 2003). Compared with men, women had a higher frequency of self-reported negative responses from their social networks, but both sexes reported the same amount of positive support. These findings suggest that negative and positive reactions are distinct social processes, and that women victims have a higher risk for specifically negative social reactions.

In a study that addressed the notion that different kinds of social experiences may have different domains of influence, Borja et al. (2006) reported that among a non-clinical sample reporting histories of adult sexual assault, negatively experienced social interactions contributed specifically to the risk of developing PTSD symptoms. In contrast, positively experienced social interactions contributed to measures of posttraumatic growth (e.g., having a great appreciation for life, a greater sense of personal strength, or spiritual development). Importantly, positive social support was not correlated with PTSD symptoms, and conversely, negative support did not influence posttraumatic growth.

The study data in their totality suggest that negative and positive social support have different patterns of influence. The absence of support, or actual negative responses, appears rather consistently to exert a negative effect, while the benefit of positive social responses or interactions is less uniform. The psychological value of positive social support often depends on who gives the support (Pilisuk & Parks 1986) and whether the support offered matches a specific need (Kaniasty & Norris 1992, Punamaki et al. 2005). The buffering influence of positive social interactions on trauma response may be particularly limited by the vicissitudes of misunderstanding between the traumatized individual and his or her social network. Moreover, certain types of traumatic events are more likely than are others to elicit positive versus negative social reactions. As noted by Punamaki et al. (2005), traumas that typically mobilize positive social support are "visually distressing, unambiguous, collectively shared and ... often attribute heroic characteristics to the victims"; such as was the case with the 9/11

terrorist attack and its victims, particularly first responders. In contrast, traumatic events that elicit negative responses are often unseen and unshared, ambiguous in their acceptability, and associated with stigma and shame, as is often the case with sexual assault, sexual or physical abuse, or combat in an unpopular war. Several studies have reported the adverse impact of negative reactions from a social network on victims of marital violence and rape victims (Andrews & Brewin 1990, Campbell et al. 2001, Davis et al. 1991, Janoff-Bulman 1992). Similar effects have been described for returning Vietnam War veterans, who were harshly judged and in many ways socially rejected (Koenen et al. 2003, Summerfield & Hume 1993).

The most profound question, however, is why social support in either direction has such a potent influence on PTSD. A trauma is by definition an event that is threatening, unexpected, and uncontrollable, and from a cognitive perspective directly challenges beliefs that the world is safe, predictable, and controllable, contributing to the cardinal emotion of PTSD, namely fear (Am. Psychiatr. Assoc. 2000). In the case of interpersonal trauma, the event requires revision of the individual's fundamental “inner schemata of self in relation to the world,” changing how safe the person feels in the world (Horowitz 1986) and ultimately, as Herman (1992) noted, revision of the systems of interpersonal bonds that connect the individual to his or her community.

During the course of natural recovery, fearful feelings decrease over time and the individual regains his or her emotional and cognitive equilibrium. At a cognitive level, positive social support may facilitate this process as it conveys the message that one is taken care of and is a member of a group whose task is in part the protection of its members. In contrast, negative support may reinforce beliefs that the world is hostile, contributing to feelings of fear and lack of safety. A prospective study assessing posttrauma beliefs among adult victims of sexual and physical assault found that posttrauma beliefs such as, “People who would stand by me have let me down” and “I feel like other people are ashamed of me now,” were more predictive of PTSD than were variables related to the traumatic event itself, including perceived threat to life and objective characteristics of the assault, such as presence of a weapon and severity of assault (Dunmore et al. 2001).

The finding that negative social support is consistently associated with the development of PTSD suggests that individuals who are vulnerable to PTSD are more likely to react with greater fear to negative interactions in comparison with those who do not develop PTSD following a traumatic exposure. Biases toward the perception of threat are consistent with emerging neurobiological models that suggest that variations in subcortical emotion processing may contribute to individual differences in vulnerability to various anxiety disorders (Phelps 2006), as discussed below. The findings of several psychosocial studies suggest that social support may be a significant mediator in emotional approach and avoidance of trauma-related reminders. Several retrospective studies have reported that following trauma, low social support is associated with avoidant coping in various forms, such as behavioral withdrawal and emotional disengagement (Irwin 1996, Runtz & Schallow 1997, Ullman 1996b). Furthermore, two prospective studies have shown that positive social support predicts lower subsequent PTSD avoidance symptoms (Dalgleish et al. 1996, Joseph et al. 1993). The influence of social support on avoidant symptomatology and behavior is significant because avoidant behaviors and symptoms are the most consistent predictors of poor outcome among trauma victims. For example, several studies have shown that avoidant coping (e.g., passive isolation) is associated with subsequent PTSD (North et al. 2001, Silver et al. 2002) and that trauma avoidance symptoms are the most predictive of developing full PTSD (North et al. 2002, Silva et al. 2000). In summary, there is evidence that low social support leads to avoidant coping, and that positive support decreases PTSD avoidant behaviors. These data, taken together, suggest that social support may modulate the trauma victim's capacity to approach

and process trauma-related feelings, and this may account for some of its influence on the development of and recovery from PTSD.

Expanding on the concept that relationships within a social network affect how an individual processes thoughts and feelings, we suggest that a functional social network provides a sense of safety to an individual through the presence of stable, reliable interpersonal connections. Certain kinds of social interactions in this network may help trauma survivors regulate their emotions, particularly emotions of fear, anxiety, and mistrust. A consideration of the first social environment all individuals experience, the family, provides further evidence for this view of how social bonds affect PTSD.

PART 3: TRAUMATIC EVENTS IN CHILDHOOD AND THE FAMILY CONTEXT

The family is the first social network human beings experience. When children feel overwhelmed by fear or anxiety, they turn to salient attachment figures to restore their sense of safety. The theory of attachment as developed by Bowlby (1969, 1973) and Ainsworth et al. (1978) proposes that humans are born with an innate psychobiological system (the attachment behavioral system) that motivates them to seek proximity to significant others in times of danger, stress, or novelty. Proximity to the caretaker protects the child from threat, relieves distress through the receipt of comfort and soothing, and provides direction about how to negotiate the world at large so that the child can return to and be successful in exploration. Over time, such experiences result in the organization of an “internal working model” that specifies relational or interpersonal contingencies that guide expectations about when and from whom an individual can expect help during times of distress and difficulty. From an attachment theory perspective, effective adaptation over the course of development derives from evolving self-generated capacity for emotion regulation interacting with continuing reliance on the support of others when demand exceeds the individual's capacity (Ainsworth 1991, Bowlby 1988).

An emerging literature indicates that a child's proximity to its caretaker is a critical modulator of the child's sense of safety following a trauma. The first naturalistic observations concerning the impact of trauma on children were published during World War II, where it was noted that children exposed to trauma did not have a fear response proportional to the severity of the trauma. Rather, their response was proportional to the distance from their caretaker (Freud & Burlingham 1943). More recent work has indicated that the loss of parental presence, both threatened and actual, contributes to PTSD symptomatology. In a case series describing 41 traumatized preschool children, Scheeringa & Zeanah (1995) reported that of six proposed predictors, including age, gender, proximity to the event, duration, and injury to self, the only variable associated with significantly higher rates of PTSD was the child's observation of physical threat to the parent. Such traumas can signify to a child the loss of its “secure base” and the perception of a parent's lesser ability to respond to the child's needs, further contributing to a child's sense of separation and subsequent lack of safety (Scheeringa & Zeanah 1995).

More recently, a prospective study of children hospitalized for burns demonstrated the influence of loss of physical proximity to the parent as a contributor to PTSD symptomatology. Saxe et al. (2005) followed 72 children with acute burns and found that the degree of physical trauma sustained, as measured by total body area burned, and reported pain were linked to severity of PTSD symptoms through two factors: acute dissociation and, more strongly, through separation anxiety. Separation anxiety provides a measure of distress when the child is physically separated from its parent. This study gives some indication of the psychological processes involved in the disruption of the physical bond between child and caretaker and more particularly the presence of a significant relationship between separation anxiety and PTSD symptoms (Saxe et al. 2005).

When both parent and child are exposed simultaneously to the same stressor, the child and adult responses are highly related, suggesting that parental emotional and cognitive appraisal influences the child's response. Scheeringa & Zeanah (2001) reviewed 17 studies of children ranging from preschool to teen years who were exposed to clearly defined traumatic events. They reported that higher rates of diagnosed PTSD in parents were associated with higher rates in their children, although this relationship appears to be much more pronounced in infant and preschool-age children than in older children, consistent with the view that younger children are more dependent on parents to modulate emotional experience (Scheeringa & Zeanah 2001). At least four studies of a prospective nature have found that parental PTSD symptom status predicts children's PTSD symptoms at a later time (Koplewicz et al. 1994, Landolt et al. 1998, Laor et al. 1997, McFarlane 1987). These studies suggest a pattern of co-occurrence of posttraumatic symptoms between an adult caregiver and a young child and the risk of subsequent exacerbation of the child's symptoms related to the symptom status of the adult caregiver.

A child's PTSD appears to be influenced not only by parental PTSD symptoms but also by other parental behaviors, which suggests the role of parental behaviors as a modulator of emotional experience. These included avoidant behaviors in the parents with respect to the trauma as well as the child's perceived rejection by parents or feelings of guilt and anxiety caused by parents (Deblinger et al. 1999). Parental avoidant behavior can prevent the traumatic event from being discussed or processed within the relationship as may be needed by the child, or these behaviors can place constraints on exploratory activities that might allow the child to encounter and process traumatic reminders (Laor et al. 1997, 2001; Pynoos et al. 1995). In addition, parental behaviors of distancing or rejection, owing to a parent's own negative emotional reactions to the trauma or to the child's symptom expression, can disrupt the capacity of the child to regain equilibrium. In these situations, the social bond for the child will no longer be experienced as safe and durable, and this may be the most pernicious effect of a traumatic stress on a child and its family.

The final circumstance of trauma in the parent-child dyad is when the parent is the direct source of the child's trauma, as in the case of physical or sexual abuse and other forms of maltreatment. From the organization of care described by attachment theory, abuse describes a circumstance in which the child's source of safety is also a source of danger. Numerous studies in which the child's “safe harbor” or secure base is disturbed by maltreatment demonstrate the deleterious effects on emotional regulation and interpersonal ability when the relationship with a parent is unsafe.

Similar to findings in the adult literature, the rates of PTSD in children are higher for interpersonal traumas than for other kinds of trauma, such as accidents. Most of the PTSD data on the consequences of childhood trauma are retrospective because of the challenges of conducting research on victims of unpredictable events. Again, however, the findings from studies of children converge with findings in adults from prospectively and retrospectively obtained data. At least one study reported on the varying effects of diverse traumas among treatment-seeking children using the same measures and sampling methods across the traumas. Rates of PTSD for accidents were 11%; for war, 29%−33%; and for physical and sexual abuse, 65% (Saigh et al. 1999). When a child experiences physical or sexual abuse from a parent, the consequences are significant because the child not only experiences a traumatic event, with the parent as the agent of the trauma, but also loses an important source of soothing, comfort, emotional repair, and cognitive reorganization. At times, the parent is both agent of the trauma and an effective agent of comfort, creating a paradoxical or confusing state that further undermines a child's sense of safety.

Beyond the development of PTSD, the experiences of assault or abuse by a parent have a profound effect on developing capacities for modulation of affect and the use of others as support in times of need. A traumatizing parent clearly influences a child's ability to interact effectively with a social network. Children with histories of maltreatment show rigid and situationally inappropriate affective displays (Shields & Cicchetti 1998), diminished emotional self-awareness, difficulty modulating excitement in emotionally arousing situations, and difficulty recovering from episodes of upset or distress (Shields & Cicchetti 1997). Such children are more likely to isolate themselves or withdraw under conflictual circumstances (Sroufe et al. 1983) and are less likely to initiate social engagement with adults (Karrass & Walden 2005) and with their peers (Contreras et al. 2000). They expect little help under stressful circumstances and tend to interpret the ambiguous or even supportive efforts of others as hostile (Suess et al. 1992). Thus, the very ability to join and benefit from a social network is impaired by the effects of early childhood maltreatment, suggesting one of the ways in which early childhood traumas and their effects on attachment have such a profound influence on later risk of PTSD in adulthood.

Several retrospective studies and meta-analyses have identified childhood abuse as a strong predictor of PTSD following adult onset stressors of all kinds, including military/combat exposure (Andrews et al. 2000, Bremner et al. 1993, Breslau et al. 1999, Brewin et al. 2000). Data from the National Vietnam Veterans Readjustment Study reveal a link between childhood abuse and low negative social support in adulthood, which in turn is linked to risk for combat-related PTSD (King et al. 1999). Pretrauma risk factors from childhood appear to exert part of their influence on PTSD via their corrosive effects on social network formation over the course of an individual's lifetime.

It is assumed that the affective and social disturbances seen in maltreated children also occur in adults, negatively affecting function and in turn increasing risk for psychiatric disorders such as PTSD. However, scant data exist to support this assumption. In response to this gap, we investigated the status of emotion-regulation capacity and expectations of support and their relationship to current attachment status among treatment-seeking individuals with PTSD. We found that emotion regulation and social disturbances, so well documented among maltreated children, are also salient problems for adults with histories of childhood maltreatment. As in the developmental literature, we found that compromised attachment contributed directly to problems with emotion regulation and expectations of support, which in turn contributed separately and significantly to functional impairment (Cloitre et al. 2007). The study results identify parallel patterns of problems in emotion regulation and social relatedness in adults, suggesting some uniformity in the points of vulnerability in functioning as a result of abuse across the lifespan. The data, however, do not identify the degree to which such patterns are stable within any individual. Longitudinal prospective studies are needed to identify factors that contribute to developmental trajectories of impaired functioning and, more importantly, individual and environmental factors that support positive change and resilience.

Recent studies have suggested both the immediate and enduring biological effects of the parent-child dynamic with regard to safety and threat exposure. Infant stress-regulatory responses are substantially dependent on the infants' relationship to their primary caretaker. Experimental manipulation of caregiver responsiveness in the face of threat to the infant has been shown to be related to cortisol secretion. For example, experiences of separation during which the infants were left with substitute caretakers were associated with increased cortisol levels when the caretaker expressed negative affect (Schechter et al. 2004). Studies of holocaust survivors and their adult children provide biological data showing that parental trauma has a chronic and enduring influence on their offspring. The bonds between holocaust survivor-parents and their children were frequently disrupted by emotional distress, intense and terrifying memories, and other PTSD symptoms. Yehuda and colleagues (2001) found that adult offspring whose parents

had PTSD were more likely to develop PTSD than those whose parents did not have PTSD (Yehuda et al. 2001). Additionally, having parents with PTSD contributed to a greater negative feedback inhibition of the hypothalamo-pituitary-adrenocortical (HPA) axis (i.e., greater dexamethasone suppression of cortisol), an effect that was independent of childhood traumatic stress or current PTSD symptoms (Yehuda et al. 2007). Yehuda has reported that PTSD-positive offspring reported more emotional abuse (Yehuda et al. 2001) and more over-protective parenting (personal communication, R. Yehuda), which suggests that disturbances in the parent-child relationship and parenting behaviors may mediate the observed PTSD relationship between parent and child. Moreover, the data suggest that parental PTSD symptoms can influence the parent-child relationship in ways that alter the child's HPA function in an enduring way. That is, it appears that disruptions to the safety of the parent-child bond may have lifelong effects on the response to traumatic stress and such disruptions imprint themselves into the biological organization of the stress response.

The fact that parental behavior can profoundly influence offspring emotional behavior and even organize the offspring biology has been demonstrated in several animal models. Early variations in maternal care produce lifelong alterations in the HPA axis and stress response, and such models demonstrate how sensitivity to stress may be transmitted across generations through behavioral interactions. These rodent and primate studies demonstrate some of the biological mechanisms by which emotional and social behavior become pervasively organized by early parent-child interactions and illustrate how variations in social experiences are essential to understanding gene-environment interactions that result in later behavioral phenotypes (Maestripieri 2005; Maestripieri et al. 2006a,b, 2007; Meaney & Szyf 2005a,b; Seckl & Meaney 2006; Shannon et al. 2005; Suomi 2005).

These recent discoveries in the developmental biology of stress response are consistent with Bowlby's view that the attachment system is active throughout the life cycle in times of stress. Attachment theory predicts that when an individual feels threatened, there will be an instinct to seek out safety in important relationships, whether these relationships are with the caregiver, as in the case of children, or within a selected social network, as in the case of adults. We have reviewed the data consistent with this proposal indicating that, following a trauma, parental response or the response of the social network influences the risk of developing PTSD in children and adults, respectively. We have also reviewed data suggesting that the impact of early traumatic experiences on the bonds between a child and its parents (in particular when the trauma comes from the parents) influences how an individual will later organize his social life, in the form of social bonds within a network, and his inner life, in terms of his ability to utilize social bonds to assist in coping with emotionally distressing events. The following sections describe how neurobiological insights into social bonding and social cognition provide the beginnings of a mechanistic account of the ways in which social bonds may contribute to a sense of safety and function to help an individual with emotion regulation.

PART 4: SOCIAL COGNITION—THE NEUROCIRCUITRY OF SOCIAL BONDS

With regard to PTSD, nearly all of the neurobiological research has focused on individual-level factors, such as neural circuits responsible for fear processing, modulation, and extinction (Phelps & LeDoux 2005, Rauch et al. 2006); alterations in the HPA axis (Marmar et al. 2006, Pitman et al. 2006, Yehuda 2006); or the nature of emotional memory (Cahill et al. 2003). Researchers have begun to identify the neural pathways underlying social affiliation, and emerging evidence shows the influence of social affiliations and evaluations on stress and fear systems in the brain. There have yet to be clinical studies examining the effect of stress or interpersonal trauma on brain regions implicated in social cognition and bonding. However, we believe that understanding at the neurobiological level how social information is processed and connected to states of fear, anxiety, safety, and reward will provide an important

perspective on the social and psychological data reviewed above. In particular, considering the sizable impact that social factors have on the development of PTSD, it is important to connect the social and psychological paradigms to the rapidly advancing biological paradigms that have, so far, overlooked social interactions as they influence anxiety, fear, and stress responses. Conversely, a richer understanding of how social information affects the development of anxiety disorders, in this case PTSD, may suggest new ways to examine brain function in relation to stress and threat. For example, the feeling of safety may require not only an absence of fear but also an awareness of social connection and support, suggesting an interaction between subcortical fear-processing regions and higher cortical regions implicated in social knowledge.

Attachment theory posits the existence of an “internal working model” of attachment relationships that evolves with each significant attachment relationship and that guides an individual's use of attachment figures for emotional regulation and other interpersonal needs. Social neuroscience experiments have provided the first glimpses of the neural substrates of such an internal working model. Imaging research implicates certain regions or networks in the brain for representing the intentions of others within one's mind (so-called theory of mind), while other networks are responsible for comparing past experiences (memories) with present stimuli and context to make decisions about risk and reward seeking (Adolphs 2003, 2006; Adolphs et al. 1998). Similarly, other imaging data suggest that the feeling of trust involves an interaction between the amygdala and the prefrontal cortices (Adolphs 2002, Winston et al. 2002).

Coan et al. (2006) recently published the first functional magnetic resonance imaging (fMRI) study that demonstrates neural processes linking social support to emotion regulation. Sixteen highly satisfied married couples were selected, and the wife in each couple was exposed to a simple threat while the kind of social support she received was varied. When the wife was in the fMRI scanner, she was able to hold her husband's hand, the hand of a male experimenter (stranger), or no one's hand. The wife faced either the threat of receiving a mild but uncomfortable electric shock while in the scanner or a nonthreatening condition of knowing no shock would be delivered.

Both spouse and stranger handholding attenuated the neural response to threat in multiple brain areas implicated in the visceral and musculoskeletal responses to affect related arousal (e.g., ventral anterior cingulate, posterior cingulate). However, spousal handholding had a powerful and specific effect distinguishing it from stranger handholding. The women showed less activation in regions associated with emotion regulation (dorsolateral prefrontal cortex and caudate) and emotion-related homeostatic functions (superior colliculus) when holding their spouse's hands, compared with holding a stranger's hand, indicating that social knowledge influences emotion-regulation processes. The diminished activation in emotion-regulation areas correlated with subjective measures that under the threat condition, spouse handholding was less unpleasant than was stranger handholding. This suggests that there was less unpleasant affect to regulate in the spouse handholding condition, consistent with the observation of diminished neural activation. However, without more data, in particular pertaining to subcortical threat-processing activity (e.g., amygdala activity) and connectivity between the proposed emotion-regulation areas and the stress response areas, it is difficult to give a more precise interpretation of this specific finding.

Notably, the social support was nonverbal and of a sensory nature, namely touch, an elementary but perhaps universal form of comfort. Coan et al. (2006) also found that brain structures associated with more evaluative, attentional, and affective components of the threat response were attenuated more specifically by spousal handholding, suggesting that attachment figures influence both cognitive and emotion regulation processes in ways that strangers do not. Most

interestingly, threat-related activations of the right anterior insula, superior frontal gyrus, and hypothalamus were sensitive to marital quality, with higher marital satisfaction predicting greater attenuation only during spousal handholding. Individuals with higher-quality relationships appear to benefit from a greater regulatory effect of attachment relationships on neural systems supporting the brain's stress response. Regulation of the hypothalamus suggests these benefits may be pervasive, as the hypothalamus regulates multiple systems within the brain and the body, including the HPA axis. The specific effect of marital quality on the spousal handholding condition is consistent with attachment theory's prediction that specific attachment experiences influence the quality of emotional regulation provided by contact with attachment figures. Furthermore, these effects are only apparent under conditions where the attachment system, or one of the members of the system, is under threat.

In addition to the above study, which suggests the role of intimate social bonds in attenuating fear reactions, data from other studies suggest that social distance can strengthen fear responses. Specifically, race bias has been shown to impede fear extinction. Olsson et al. (2005) assessed whether individuals of another race are more readily associated with an aversive stimulus than are individuals of one's own race, and whether these effects may be moderated by attitudes, beliefs, or contact with members of the racial outgroup. Using a standard fear-conditioning and fear-extinction paradigm with images of black and white faces as the conditioned stimuli, the investigators demonstrated that whereas all subjects acquired conditioned fear to faces of either race equally, subjects extinguished fearful associations only to faces from their own racial group. That is, white subjects could extinguish the fearful association with white faces but not black faces, and black subjects could extinguish the fearful association with black faces but not white faces. Of interest, the conditioning bias to racial outgroup faces was attenuated among those with more interracial dating experience, consistent with a large body of evidence that positive intergroup contact reduces negative reactions to outgroups. Social information appears to modulate very basic aspects of the fear system in humans, and positive social interactions can attenuate certain kinds of fear learning (Olsson et al. 2005). If fear extinction proves to be essential to the pathophysiology of PTSD, these findings suggest that social information and social experiences are essential modulators of fear extinction. Such findings also suggest new methods to investigate the effects of social support on PTSD. For example, certain types of social encounters may contribute to beliefs or attitudes that impair fear extinction.

Finally, emotional numbing has been identified as a symptom associated with significant impairment from PTSD (Breslau et al. 2005), and preliminary evidence links a diminished neural capacity to respond to social rewards to emotional numbing among PTSD sufferers. Elman et al. (2005) hypothesized that emotional numbing is related to deficits in the brain reward circuits, leading to altered responses to sources of pleasure. The investigators examined reactions to social and monetary reward using a validated reward paradigm for fMRI, and found that compared with controls, PTSD patients had smaller signal changes in the nucleus accumbens, cingulate gyrus, insula, and prefrontal cortex in response to reward stimuli. Thus, a decreased ability to respond to social reward may be either a risk factor for PTSD or a consequence of PTSD. These data suggest that failure of those with PTSD to seek out or use offers of social support (Norris & Kaniasty 1996) may reflect not only a desire to avoid fear reactions or hyperarousal, but also the absence of rewards associated with social contact. Such findings suggest that neural substrates for basic social responses may be implicated in the pathogenesis of PTSD and that these may be distinct from fear-related neural circuitry.

A separate line of inquiry shows that social events and information are processed by the brain at a subconscious level, before conscious cognitive activity comes online. Such stimuli can be conveyed by a variety of nonverbal cues in the social environment, including body language. In one study, normal subjects were presented with pictures of people (with the faces blurred)

in nonfearful or fearful body postures. Although the test subjects were not making conscious appraisals, fMRI revealed increased amygdala and fusiform gyrus activity during viewing of the fearful body postures, consistent with the idea that the fear system processes such information early and automatically (Hadjikhani & de Gelder 2003). Furthermore, studies of social context processing show that cognitive appraisal can be influenced by emotional reactions to a social context. In a study of normal subjects discriminating fearful from surprised faces, the social context was varied prior to stimuli exposure. Facial expressions of surprise have been found to be somewhat ambiguous in regard to their affective "message" and were purposefully used to assess the potential influence of additional verbal information. When a surprised face was preceded by an emotionally congruent social context (e.g., conveyed verbally by being told "She just won 500 dollars"), there was less activation in the amygdala than when the social context preceding the surprised face suggested loss or threat (e.g., "He just lost 500 dollars") (Kim et al. 2004). These results suggest that information about social context regulates the fear system response to discrete stimuli and that social context may be particularly important in regulating the response to more ambiguous stimuli.

In summary, the fear system responds to and is modulated by a range of social information, including sensory information (touch), representations of bodily cues, stored social knowledge or assumptions (race), and contextual cues. Furthermore, such information has influence at both conscious and nonconscious levels of awareness (Phelps 2006). These findings suggest some of the mechanisms whereby discrete social experiences may influence fear processing, acquisition, and extinction. Consistent with the literature on social support and parent-child transmission of fear, these data suggest that positive information from socially relevant others can attenuate fear responses and that socially negative or even neutral information can heighten and maintain fear responses. It remains to be determined how such processes are different in people with PTSD and how such processes maintain or exacerbate PTSD symptoms, particularly with regard to victims of interpersonal traumas.

PART 5: SOCIAL COGNITION—THE NEUROCHEMISTRY OF SOCIAL BONDS

Investigation of the neuropeptides oxytocin (OT) and arginine vasopressin (AVP) suggests an intersection between the biology of social bonding and the biology of fear response that may parallel the intersection between social support and PTSD described above. Animal studies demonstrate that OT and AVP are involved in the neurocircuitry of fear. In rodents, OT acts on the amygdala to reduce fear (Amico et al. 2004, Gulpinar & Yegen 2004) and to modulate aggression (Bosch et al. 2005). Huber et al. (2005) recently demonstrated that receptors for OT and AVP are located within the central nucleus of the amygdala. The central nucleus appears to be a place where the expression of fear is modulated (Pare et al. 2004), suggesting that these neuropeptides may be related to distinct aspects of the fear response. Stimulation of OT receptors should lead to inhibition within the amygdala, suggesting a mechanism for the way in which OT downregulates fearful responses (Debiec 2005, Huber et al. 2005, LeDoux 2000).

More recently, OT and AVP have been identified as essential chemical mediators of social bonding in animals (Insel 2003, Keverne & Curley 2004, Young & Wang 2004). OT released at childbirth coordinates maternal response to the infant at both the physiologic and behavioral levels, whereas AVP regulates male social behaviors of territorial marking, social aggression, social recognition, anxiety, and male parenting behavior. In adult pair bonding, OT is integral for females, whereas AVP appears to be more central for males. Elegant experiments that transgenically alter the distribution of AVP1a receptors also alter male pair-bonding behavior, but have no effect on male parenting behavior, suggesting that there are separate AVP circuits for each kind of attachment in males. Partner bonds require the formation of social memory (e.g., that a particular individual is the mate) and reinforcement of that memory. Such memory

and reinforcement depend upon an interaction between these neuropeptides and the dopamine reward system, with the release of dopamine during mating reinforcing the association between the familiar cues of the mate (e.g., odor) and reward. It appears that the neuropeptides are responsible for linking the dopamine reward system to social bonding events. From an evolutionary viewpoint, such social memories of friendly others, particularly mates, are as essential to survival as the fear memories maintained by the amygdala.

It is notable that PTSD involves a disruption of social behavior, emotional reward (particularly from social experiences), and fear response. Only one study has examined the effects of these neuropeptides in PTSD. Pitman et al. (1993) explored whether OT and AVP would have inhibiting and enhancing effects, respectively, on fear-related memories in PTSD. The investigators used a combat imagery paradigm in subjects with combat-related PTSD and found that OT attenuated and AVP augmented autonomic responses. However, this initial study focused primarily on the stress-response effects of these neuropeptides on fear-related memories, but not in relation to social variables.

Heinrichs et al. (2003) explored the relationships between OT and stress response in social settings by administering OT and using the Trier Social Stress Test. The investigators found that OT interacts with received social support to suppress both the subjective and cortisol responses to psychosocial stress. Stress response was most affected by the combination of OT and social support, whereas the effect of receiving OT alone appeared to be about equivalent to the effect of receiving social support alone with regard to measures of physiologic and subjective measures of anxiety. OT also had an anxiolytic effect that social support alone did not have.

Kirsch et al. (2005) enrolled 15 normal human male subjects and administered either OT or placebo intranasally, followed by an fMRI paradigm, to assess amygdala activity and functional connectivity in response to a task involving visual stimuli that were either interpersonally threatening (angry or afraid faces), noninterpersonally threatening (fearful or threatening scenes devoid of social interaction of facial displays), or simple shapes. Compared with placebo, OT significantly suppressed amygdala activation, with the effect more pronounced for fearful faces than fearful scenes. Kirsch et al. (2005) demonstrated that the amygdala was functionally connected to the upper brainstem during the placebo condition and that this connectivity was significantly reduced with OT administration, consistent with the anatomic data from Huber et al. (2005) suggesting that OT inhibits outgoing signals from the central amygdala to the effector regions of the fear system (e.g., brainstem regions periaqueductal gray and reticular formation). Interestingly, OT administration had no effect on self-report scales of anger, dominance, or arousal, suggesting that the emotional effect is essentially subconscious.

It is unclear whether OT simply reduces fear-reactivity or promotes a sense of trust by involving brain areas distinct from fear circuitry. An interdisciplinary group of economists and psychologists recently showed that the administration of OT can increase trust in humans (Kosfeld et al. 2005). In this study, participants were asked to play an investing game that involved being either an investor or a trustee and participation in a series of investment exchanges with the same person (i.e., two subjects were paired as investor-trustee, and the social interaction of investment and return-on-investment took place several times within the same pair). The investors had real money, which they invested with the trustee. The trustee's job was to decide how much of a return, from zero to all, the investors would receive. Participants were given either placebo or OT intranasally, in a random, double-blind fashion, one hour before the game. Compared with placebo, recipients of OT behaved in a more trusting manner when they were investing.

This effect was specific to trusting behavior, because OT administration had no effect on the behavior of the trustees, whose job did not require trusting but did require a certain kind of strategic thinking to maximize their own share of the money. Furthermore, when the game was replayed, but a computer played the trustee role and the investors knew that they were interacting with a computer rather than a person, OT had no effect on investor behavior. That is, the administration of OT only affected trusting behavior when the participants believed they were interacting with another human being, which suggests that OT does not simply lower an aversion to risk, but actually increases a certain kind of prosocial approach behavior that requires the mental representation of another human being with whom the subject is interacting. The authors postulate that this study reveals a specific system within the brain that maintains an aversion to betrayal or interpersonal rejection and that OT may be part of how this system is regulated.

Taken together, these studies suggest that OT does interact with the fear system to decrease fearful responses to stress, but that interaction with socially salient stimuli (e.g., other people) is necessary for OT to exert its modulating effect. Social bonds modulate the fear system, as demonstrated in the handholding study (Coan et al. 2006), and OT may be a neurochemical mediator of this effect. The fact that subjects in the investing game showed no response to OT when they believed they were interacting with a computer highlights the unique aspects of OT and social bonding in humans. The perception of human contact appears to be necessary for OT to influence behavior. Because PTSD often involves a disruption of interpersonal relationships, some of the social consequences of PTSD may be associated with disruptions of the neuropeptide circuitry responsible for linking social experiences with reward and fear systems in the brain.

Evidence of such disruption comes from a recent study of maltreated children. Fries et al. (2005) developed a novel method to measure the brain's output of OT and AVP using urine samples. They studied 18 adopted children who had experienced early neglect and had been in orphanages for an average of 16.6 months prior to adoption. Importantly, at the time of the study, these children had been living in stable, enriched, nurturing adoptive homes for an average of 34.6 months. Twenty-one children living in a typical home environment with their biological parents served as controls. The investigators probed the effect of a familiar social bond by having each child play an interactive computer game under two conditions: while sitting on either their mother's lap or on an unfamiliar female experimenter's lap. Through the 30-minute interaction, the mother or unfamiliar adult engaged in regularly timed physical contact (e.g., tickling, patting on the head, counting each other's fingers, whispering in ear), and a urine sample was collected about 15−20 minutes after the task. At baseline, compared with children without histories of neglect, those with a neglect history had lower levels of AVP and there was no difference in OT levels, suggesting that social deprivation may inhibit the full development of the AVP system. In response to the social interaction paradigm, children without histories of neglect showed an increase in OT secretion after interacting with their mothers, while those with histories of neglect did not. There were no group differences in OT levels after interaction with the stranger, and there were no group differences in AVP levels to either social interaction paradigm. This suggests that disruptions in early social bonding impair the OT response to active social bonding interactions with a caregiver later in life. Overall, early disruptions of attachment bonding may lead to long-term disruptions in the neural systems responsible for modulating social affiliation, including the ability to be calmed and comforted by social bonding interactions.

This experiment is the first example in humans of how early adversity programs the brain to respond in a fixed way to certain social stimuli, a phenomenon similar to that in which early maternal deprivation in rodents leads to lifelong programming of the glucocorticoid stress-response system (Seckl & Meaney 2006). This finding also suggests a possible biochemical

mechanism for how early prior traumas function as risk factors for later PTSD. Early adversity may alter neural systems for social bonding, limiting an individual's ability to use social resources and build protective social networks later in life.

PART 6: CLINICAL IMPLICATIONS—THE THERAPEUTIC ALLIANCE AND THE CREATION OF SOCIAL NETWORKS IN THE TREATMENT OF POSTTRAUMATIC STRESS DISORDER

An individual with PTSD lives in a heightened and chronic fear state, which includes constant surveillance for and tendency toward perceiving threat in the environment, and this inevitably includes the therapist. The successful treatment of PTSD requires first and foremost providing a sense of safety to the client (Pearlman & Courtois 2005). It is a prerequisite to the treatment, and its most basic expression is realized in the ability of the client to stay in rather than flee from the treatment. The reduction of felt threat in the treatment context requires that the client experience the therapist as someone who is supportive, warm, and interested in the client, who appears to understand the meaning of the client's traumatic experiences and can identify resources (plans and interventions) to help the client. In this way, the therapeutic relationship shares some aspects in common with social support.

It may be useful to see the process of therapy as involving the creation of a social bond and to see the therapeutic alliance within therapy as a reflection of this bond. PTSD is almost inevitably associated with broken or distressed social bonds, either due to the nature of the trauma itself (e.g., sexual assault, combat), to the social repercussions of certain kinds of traumas (e.g., blaming, stigmatizing responses), or to weakened social networks (e.g., disaster victims). Consequently, the importance of the therapeutic alliance may be particularly salient in the treatment of PTSD. The client may be more sensitive to the interpersonal aspects of the therapy, particularly to lapses or imperfections in communication. This sensitivity can be attended to in the therapy, and the therapeutic relationship itself can be introduced either explicitly or implicitly as a model for the exploration of alternative and more positive relating.

A substantial empirical literature has identified that the therapeutic alliance is the most consistent predictor of psychotherapy outcome, although the relationship is often modest in size, with effect sizes ranging from 0.22 to 0.24 (Horvath & Symonds 1991, Martin et al. 2000). Alliance has proven to predict treatment outcome across different treatment modalities, including short-term cognitive-behavioral treatment (Raue & Goldfried 1994, Stiles et al. 1998), interpersonal therapy (Krupnick et al. 1996), psycho-dynamic therapy (Eaton et al. 1988, Stiles et al. 1998, Yeomans et al. 1994), gestalt therapy (Watson & Greenberg 1994), and cognitive therapy (Muran et al. 1994). In a recent study of PTSD examining the effect of therapeutic alliance, the influence of a positive relationship on PTSD symptom reduction was about twice the size typically reported in these other studies (ES = 0.46) (Cloitre et al. 2004). Thus, the strength of the patient-therapist relationship appears to be a critical common factor across treatment modalities and may be of particular importance in PTSD.

Formally, the therapeutic alliance has been defined as a collaboration that has three distinct dimensions: the presence of a feeling of mutual warmth and understanding, agreement on the goals of the treatment, and agreement on the means by which these goals will be attained (e.g., Raue & Goldfried 1994). Among individuals presenting for treatment of PTSD, the relative effectiveness of the treatment may vary according to the individual's sense of safety, essentially the antidote (or counterexperience) of the sense of fear that is the core emotional disturbance in PTSD. In a recent study, the strength of the therapeutic alliance established in the first three sessions of treatment was, among several early treatment indicators, the most powerful predictor of treatment outcome as measured by PTSD symptom reduction (Cloitre et al.

2002). Although all three components of the therapeutic alliance were predictive of this positive outcome, the strongest was the sense of a warm and understanding bond with the therapist, suggesting the importance of the positive emotional experience with the therapist.

The client's experience of felt safety is a prerequisite to the successful implementation of the various activities in the treatment. One of the essential components of treatment is the “emotional processing” of the memories of the trauma, with the goal of diminishing and resolving feelings of fear associated with memories. Psychological medicine has recognized the idea of therapeutic remembering since at least the early twentieth century (Freud 1914/1963). More recently, cognitive behavioral paradigms have framed PTSD treatment in terms of conditioning and habituation to feared internal or mental stimuli. Trauma memories are treated as the feared “objects,” and if the client can bring forward the memory of the trauma with sufficient fidelity, intensity, and duration, the client's fearful reactions to the memory will diminish and PTSD will resolve (see Shalev 1997). The therapist must be sufficiently skilled in creating a safe enough context to allow a client to be persuaded to engage in such a process. A similar recovery process may occur for traumatized individuals who experience safety such that they engage in the processes of recollection and habituation on their own.

Increasingly, effective treatments address ways in which feelings of safety can be exported to a variety of aspects of the individual's life, and these include interventions to resolve fear responses in the individual's larger physical and social environment (e.g., Rothbaum & Foa 1999). Evidence relating social support to PTSD suggests the importance of incorporating interventions that take into account social support phenomena including the benefit to PTSD sufferers of positive social support, the risk to health and recovery when social support is low, and the need to effectively manage negative social support. More recently, interventions have considered that rather than simply reducing fear associated with traumatic interpersonal events by exposure to fear-eliciting people and places, treatment could additionally target improvement of interpersonal relationships.

In light of this evidence, we have added to traditional exposure-based treatments components that target the development of interpersonal skills and enhanced affect regulation abilities (Cloitre et al. 2002) through the use of role play and substantial practice both in and out of the treatment session. In addition to substantially reduced PTSD symptoms, the treatment led to the resolution of impairments in emotion regulation, significant increases in perceived social support, and improved positive relationships with significant others and individuals in the work, home, and social environments. The treatment was adapted to a real-world setting following the World Trade Center attack on September 11, 2001. The nature of this trauma was such that many survivors lost members of their social network through death or relocation. Moreover, many of those with PTSD associated with the World Trade Center attack suffered from diminished perceptions of social support despite its evident availability. The treatment was successful in resolving PTSD as well as improving interpersonal functioning and improving social support perceptions to normative levels (Levitt et al. 2007).

Finally, there has been exploration of treatment that focuses solely on interpersonal functioning without the use of any explicit fear-reduction intervention. In a pilot study of 14 individuals with PTSD, Bleiberg & Markowitz (2005) adapted interpersonal psychotherapy, an evidence-based standardized treatment, used initially for depression, that focuses on improving relationships. The treatment was successful in substantially reducing PTSD symptom severity in the majority of the study participants. Although the durability of these effects is unknown, the results suggest that specific efforts to improve the quality of social bonds can lead to improvement in individual PTSD symptoms, even in the absence of exposure-based work, potentially through self-initiated efforts to engage in emotional processing of the trauma.

Future studies are also needed to explore insights into the neurochemistry of feelings of trust and connectedness, how these relate to feelings of fear and anxiety, and their implications for improving treatments. Oxytocin has been widely used in humans already, albeit for nonpsychological purposes, and an efficient delivery system to the central nervous system already exists. Explorations of the therapeutic potential of this agent for PTSD and disorders of anxiety seem inevitable.

In conclusion, social bonds exert a powerful influence on the development and maintenance of PTSD as the location of important processes that influence how an individual responds to a traumatic event. Most crucially, it is within social bonds that individuals may receive or develop a sense of safety, which appears to be essential to the prevention of or recovery from PTSD. The process of therapy may by considered one type of social bonding, and measures of the therapeutic alliance begin to describe the social bond within therapy. Finally, insights into the neurobiology of social bonding may illuminate aspects of the neurobiology of PTSD and more generally may provide insights into the neurobiology of therapeutic change.

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