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Facial Emotion Recognition Difficulties in Individuals with PTSD Symptoms
by

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An Abstract of
A dissertation submitted to the Faculty of the Graduate
School of Emory University in partial fulfillment
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in Psychology

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Abstract

Facial Emotion Recognition Difficulties in Individuals with PTSD Symptoms

By Mark Gapen, M.A.

The purpose of the current study was to examine the relationship of exposure to traumatic events and PTSD symptoms to facial emotion recognition difficulties. Recent studies have found hyper-responsivity of the amygdala in individuals with PTSD in response to emotional facial stimuli. The amygdala has been implicated in both the processing of emotions and facial expressions. On the behavioral side, facial emotion recognition difficulties have been found in individuals with a variety of psychiatric disorders. Finally, facial emotion recognition difficulties have been found in maltreated children. The current study tested the facial emotion recognition abilities of 162 participants from a NIMH-funded study investigating environmental and genetic risk factors for PTSD in a sample of low SES, African American men and women seeking care in the primary care clinics of a public urban hospital. Results indicated that individuals with a current or lifetime CAPS diagnosis of PTSD made more errors to faces ($p<.05$). In contrast, exposure to childhood sexual and physical abuse, and to traumatic events as an adult were associated with fewer errors to faces ($p<.05$). Thus, facial emotion recognition may be one mechanism underlying interpersonal difficulties in individuals with PTSD. This is the first study to document an association between PTSD and facial emotion recognition difficulties and implications for future research are discussed.

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Facial Emotion Recognition Difficulties in Individuals with PTSD Symptoms

One of the hallmarks of Posttraumatic Stress Disorder is an interruption of normal social ties. In fact, one focus of treatment with trauma survivors is re-establishing a sense of connection to others (Herman, 1997). Thus, understanding the mechanisms that may underlie any difficulties with interpersonal connection is of utmost importance. One new avenue of exploration is examining the ability of trauma survivors to accurately recognize the facial expressions of other people. The reason for thinking that individuals with PTSD may have differences in their facial emotion recognition abilities comes from an emerging literature on the neurobiology of PTSD, which has only recently become the subject of scrutiny by researchers. Studies have begun to document both hormonal and neurological abnormalities in individuals suffering from PTSD (Rauch, Shin & Phelps, 2006; Yehuda, Giller & Mason, 1993), and rapid advances in neuroimaging technology have made it possible to begin identifying the underlying structural characteristics of psychiatric disorders that had previously been considered "functional" (Pitman, Shin & Rauch, 2001). These new data on the underlying neural bases of PTSD have led to new directions and new hypotheses at the behavioral level of analysis. First, several studies have found abnormal functioning of the amygdala in individuals suffering from PTSD (e.g. Bremner et al., 1999; Liberzon et al., 1999; Pissiota et al., 2002; Rauch et al., 1996; Shin et al., 1994). Second, the amygdala has been implicated in emotion processing, especially fear (Dalgleish, 2004; Whalen, 1998), and the processing of facial emotions (Amaral, 2003; Haxby, Hoffman & Gobbini, 2003; Nelson, 2001). Third, researchers have found that

individuals with other disorders, including social phobia, panic disorder, and depression have displayed difficulties identifying certain facial expressions (for a review, see Kornreich & Philippot, 2006**)**. Fourth, at least one study has reported that individuals with PTSD have a recognition bias for faces that they perceive as threatening (regardless of facial expression)(Paunovic, Lundh & Öst, 2003). Given this converging evidence, it is surprising that only one published study to date has looked at the performance of individuals with PTSD in recognizing facial emotions (McClure, Pope, Hoberman, Pine & Leibenluft, 2003). Given evidence that: 1) the amygdala plays an important role in the processing of facial emotions specifically, 2) the amygdala is hyper-reactive in individuals with PTSD, and 3) individuals with other anxiety and mood disorders have displayed difficulties recognizing certain facial emotions, it seems important to explore the accuracy of individuals with PTSD in recognizing facial emotions.

The purpose of this study is to explore the ability of individuals with PTSD to accurately and quickly identify facial emotions as compared to individuals without PTSD and individuals who have not experienced trauma. While much of the literature that will be reviewed here is focused on the amygdala, this study did not directly assess neural activations. Thus, it is important to consider other developmental factors that may contribute to emotion recognition difficulties. There is evidence that maltreated children have difficulty decoding emotions compared to nonmaltreated children (Bowen & Nowicki, 2007; Hodgins & Belch, 2000). Studies have also shown that maltreating parents differ from control parents in that they tend to display less positive emotion and

more negative emotion (Camras et al., 1990; Pollak, Cichetti, Hornung & Reed, 2000). It may be the case that maltreated children have biases in their recognition abilities as a result. Thus, it is important to explore whether any difficulties with emotion recognition are a function of maltreatment and trauma per se, or whether they may be a specific function of PTSD. Additionally, PTSD in urban, low income populations remains an under-studied phenomenon (Schwartz, Bradley, Sexton, Sherry & Ressler, 2005). This is an especially vulnerable population, and it is important to consider the implications of PTSD in light of the myriad challenges low-income individuals face navigating the stresses of poverty generally. If it is the case that individuals with PTSD have more difficulty decoding facial expressions, it may be particularly detrimental to their social and relational functioning because links have been established among psychiatric disorders, nonverbal decoding and interpersonal difficulties (Kornreich & Philippot, 2006). One of the hallmarks of PTSD is avoidance of activities, places and people associated with the traumatic experience. However, it may be that this avoidance is a luxury of higher SES individuals as low-income individuals are more likely to experience trauma in their neighborhoods, and therefore may not be able to avoid situations that are reminiscent of their traumatic experiences. Additionally, low-income neighborhoods tend to be more disordered, which in turn has been linked with increased PTSD symptoms (Gapen et al., in press).

Finally, it is important to study the facial emotion recognition of individuals with PTSD because nonverbal decoding abilities have been linked to relationship well-being (Carton, Kessler & Pape, 1999). Individuals with PTSD

tend to isolate themselves and feel disconnected and out of synch with those around them. Facial emotion recognition difficulties may be a contributing factor, or at the very least may exacerbate the symptoms. This may be especially important for women in low-income, minority neighborhoods because social support has been shown to be more important for the well-being of women than men (Andrews, Brewin & Rose, 2004; Brewin, Andrews & Valentine, 2000).

This study will examine the facial emotion recognition abilities of a pseudo-random sampling of individuals presenting for care in a large, urban public hospital. This sampling ensures a wide range of individuals, men and women, with and without PTSD, and who have experienced significant traumas or not.

Background

In order to understand why there might be differences in the nonverbal decoding abilities of individuals with PTSD, it is imperative to look to evidence on a variety of fronts. First, there is emerging evidence that the amygdala is integrally implicated in the neurobiology of PTSD. Second, there is evidence that the amygdala is integrally involved in emotion processing and the processing of faces. Third, there is evidence of behavioral abnormalities in primates and humans as a result of bilateral amygdala damage. Fourth, there is emerging evidence that the amygdala differentially activates in response to trauma related stimuli and facial emotions in individuals with PTSD versus normal controls. Many of the arguments for the hypotheses presented in this study rely on the evidence from neurobiology; however, it must be noted that this study did not

directly assess amygdala activation in response to facial emotion stimuli. Thus, it was impossible to ascertain the role of the amygdala in the results. Therefore, the amygdala is hypothesized as a construct that provides a basis for the hypotheses presented. While the amygdala has been implicated in PTSD and the processing of facial emotions, it is important to examine developmental theories of facial emotion recognition. Specifically, it is important to review the evidence of facial emotion recognition difficulties in individuals who have experienced child maltreatment. Finally, many researchers have reported evidence of facial emotion recognition difficulties in other neuropsychiatric disorders. All of this leads to the hypothesis that individuals with PTSD will have difficulty recognizing certain facial emotions.

Neurobiology of PTSD

Current models of PTSD have generally implicated the amygdala in the pathogenesis and maintenance of PTSD. In reviewing the neurological data, it appears that the medial prefrontal cortex plays an important role in modulating the response of the amygdala, which may be integral to pathological states including PTSD (Davidson, 2002; McNally, 2006; Quirk & Gehert, 2003; Rauch, Shin & Phelps, 2006; Shin, Rauch & Pitman, 2006). The amygdala and prefrontal cortex are highly inter-connected, and activation of the amygdala has been implicated in fear conditioning, while activation of the medial prefrontal cortex is activated during the extinction phase of fear conditioning (Davis, 1992). Rats with lesions of their medial prefrontal cortex show deficits in the extinction of conditioned fear (Morgan, Romanski & LeDoux, 1993). These data support the contention that inhibitory signals from the medial prefrontal cortex to the amygdala

are necessary for the extinction of conditioned fear responses. Thus, failure of the medial prefrontal cortex to inhibit amygdala responding may lead to the amygdala hyper-responsivity seen in PTSD (Quirk & Gehlert, 2003). Overall, PTSD symptoms in general, and intrusive recollections specifically appear to be mediated by amygdala hyper-responsivity, pre-frontal hyporesponsivity, or both (McNally, 2006; Shin, Rauch & Pitman, 2006).

Metcalfe and Jacobs (1996) distinguished between an amygdala centered "hot" system, and a hippocampus centered "cool" system in memory. They hypothesized that the cool system is responsible for organized, cognitive, complex memories, while the hot system is responsible for emotional, fragmentary memories. Thus far, neuroimaging data have been supportive of this view of parallel processes in memory. In their review, Rauch et al. (2006) note that structural neuroimaging studies have found relatively smaller hippocampal volume, and functional neuroimaging studies have found increased amygdala reactivity. These findings match the symptom profile of PTSD because it is characterized by intrusive, emotional hot system memories. Other theorists have postulated a "fear structure" underlying anxiety disorders, and particularly PTSD (Foa, Steketee & Rothbaum, 1989; Lang, 1977). It may be the case that the "fear structure" is mediated by the amygdalocentric hot memory system. The result is that information relevant to the fear structure evokes a fear response including avoidance of fearful stimuli. Overall, Foa et al. (1989) argue that perceived threat is a better predictor of PTSD symptoms than actual threat. Thus, it may be the case that threatening facial expressions such as anger and fear activate this fear structure (or hot memory system) in individuals with PTSD, which in turn may inhibit information processing about those

faces. Studies have demonstrated interference to information processing when threat-related stimuli are presented to individuals with PTSD (Thrasher, Dalgeish & Yule, 1993; for a review, see Buckley, Blanchard & Neill, 2000). Using modified Stroop paradigms, researchers have found that individuals with high PTSD symptoms take longer to name colors when they are presented with threat-related words as compared to naming neutral words and compared to the performance of individuals with low PTSD symptoms. However, the bias was only evident in the processing of specific disaster related words and not general threat material (Thrasher, Dalgleish & Yule, 1993). Individuals with PTSD may not be reactive to general threat words, as evidenced by a recent study the reported no main effect for word type in a Stroop paradigm with a combined group of individuals with generalized anxiety or PTSD (Dalgleish et al., 2003). The question remains whether such a bias might apply to facial emotion stimuli, and what the behavioral correlates of such a bias would entail.

In summary, emerging models of PTSD generally implicate the amygdala and the medial prefrontal cortex in the pathogenesis and maintenance of PTSD symptoms. Specifically, it appears that PTSD is a dysfunction of the "hot" memory system such that the medial prefrontal cortex fails to inhibit amygdala response to threat-related stimuli. This may be part of the underlying neurobiology of the "fear structure" that becomes activated in response to stimuli that are perceived as threatening. Overall, the amygdala appears to be central to the neurobiology of PTSD, and may affect subsequent information processing once it becomes activated.

General and Specific Functions of the Amygdala

Quite a lot of attention has been paid to the functions of the amygdala (Adolphs, 2006; Amaral, 2003; Dalgleish, 2004; Haxby, Hoffman & Gobbini, 2001; Whalen, 1998), and there is universal agreement that the amygdala plays a central role in the processing of emotions, especially negative emotions. Dalgleish (2004) summarized the state of the field on the neurobiology of emotion processing, and reviewed evidence that the amygdala plays an important role in social processing, especially the processing of fear, and fearful faces. Additionally, the amygdala seems to be selective for fear in vocal expressions, and amygdala activation to fearful faces can be affected by the allocation of attentional resources, which means the activation is susceptible to top down control. Finally, he argued that the amygdala is also involved in long-term consolidation of emotional memories.

Whalen (1998) reviewed the evidence from animal studies, noting that fear states in animals are much easier to manipulate than those in humans. For this reason, much of what is known about the function of the amygdala comes from animal studies. The amygdala is a complex structure that both affects "downstream" autonomic responses and "upstream" higher order information processing systems. Thus, while the amygdala may be central to the "fight or flight" response, it also may affect how subsequent sensory information is processed once it has been activated. For example, while the amygdala is definitely involved in affective states, there is also evidence that it is active in viewing photographic facial expressions. These are subtle stimuli not likely to produce strong affective states; thus, the author argues for differentiating affective emotional stimuli processing from affect itself. Whalen conjectures that the amygdala plays a role in

modulating the vigilance level of an organism. He argues that perhaps the amygdala responds more to fearful faces than angry faces because the threat is more ambiguous. He supports this contention with evidence that amygdala activity decreases if an aversive stimulus is consistent, but re-emerges if the stimulus is taken away in subsequent trials.

The amygdala seems to be involved in the recognition of facial emotions, but not the recognition of faces themselves (Amaral, 2003; Haxby, Hoffman & Gobbini, 2003; Nelson, 2001). There are separate systems for processing variant and invariant aspects of faces, with facial expression processing activating emotion systems. Much of the evidence supporting the role of the amygdala in social functioning, and more specifically facial emotion recognition, comes from studies of primates with lesions of the amygdala (Amaral, 2003; Hamann et al., 1996). Rhesus monkeys with discrete lesions to the amygdala engage in more affiliative behavior, and show a lack of fear to objects that would normally produce a fear response (e.g. rubber snakes)(Amaral, 2003). This may mean that the amygdala is integral in evaluating threat in the environment, including from facial emotions.

While the amygdala has been implicated in the processing of facial emotions specifically, it may differentially activate depending upon the emotional valence of the expression. Phillips et al. (1998) reported that the amygdala was activated to a greater extent in response to fearful faces than to faces showing disgust in a passive viewing paradigm. Breiter et al. (1996) measured the amygdala responsivity of healthy participants as they viewed fearful, happy, and neutral faces, and found that the amygdala responded more to both fearful and happy faces than to neutral faces. However, they only found significant amygdala activation in response to happy faces in the second of

their two experiments. They noted significant habituation of the amygdala within runs and across trials, and since fearful faces were always presented first in the first experiment, they attribute the lack of response to happy faces to habituation. In contrast, Morris et al. (1996), using a similar paradigm, found that amygdala activity increased as the intensity of fearful faces increased and decreased as the intensity of happy faces increased. Amygdala activation to fearful faces may not depend upon conscious processing because it has also been found while participants performed a gender discrimination task (Morris et al., 1998). It is well established that the amygdala is responsive to fearful faces, but whether it is responsive to other faces and whether the activations change over time remain unclear (Phillips et al., 2001; Strauss et al. 2005). For example, the amygdala may become more sensitive to angry faces over time while habituating to fearful faces (Strauss et al., 2005).

The amygdala may also be differentially activated depending upon whether the processing is conscious or unconscious. In at least two studies, amygdala activation has only been reported for overtly presented faces, versus covert faces (Phillips et al., 2004; Williams et al., 2004). However, other researchers have shown amygdala activations to covert faces in individuals with PTSD (Rauch et al., 2000), which will be discussed later.

Overall, the amygdala plays an important role in social functioning, and emotional processing. It has been implicated in regulating vigilance level, and may affect the processing of subsequent information when activated. Additionally, the amygdala has been implicated in the processing of facial emotions (and not implicated in facial recognition). Specifically, it may be more integral to the processing of negative emotions, especially fear. While studies with primates indicate that lesions of the

amygdala cause profound impairments in social behavior, iot is only possible to generalize to humans with a great amount of caution. Of course, individuals with specific damage to their amygdaloid regions are hard to find, but intrepid researchers have sought out those rare individuals.

Studies of Amygdala Damage in Humans

A few studies have looked at the abilities of individuals with selective bilateral amygdala damage to recognize facial emotions as compared to individuals with other types of brain damage, and individuals with no brain damage. Due to the relative rarity of specific bilateral damage to the amygdala, all of these studies have been either a single case or a small sample size. Despite this limitation, most have found difficulties with emotion processing, especially with fear (Adolphs et al., 1994, 1995, 1999; Broks et al., 1998; Calder, Young, Rowland, Perrett & Hodges, 1996; Graham, Devinsky & LaBar, 2007; Sato et al., 2002; Young, Hellawell, van de Wal & Johnson, 1996).

One of the first studies of amygdala damage involved a woman suffering from Urbach-Wiethe disease, a condition which almost completely destroyed the amygdala while sparing other brain regions such as the hippocampus (Adolphs et al., 1994). She rated the intensity of different facial expressions, and the intensity of her ratings for fearful, angry and surprised faces was much lower than control subject's ratings. Also, she had difficulty recognizing faces with a mixture of emotions. However, she easily recognized familiar faces.

Another study of a woman with partial bilateral amygdala damage found that she had difficulty recognizing emotion in moving images (Young at al., 1995). Additionally, she had difficulty recognizing familiar faces on a simultaneous or successive matching

task when the facial expressions were discrepant. Unlike the previous study, the difficulty with facial emotions was not limited to the negative facial expressions.

Building on prior findings, researchers tested whether unilateral amygdala damage would produce the same difficulties with emotion recognition as bilateral damage, and also compared the results to individuals with other types of brain damage (Adolphs et al., 1995). They found that the participant with bilateral damage continued to show difficulty recognizing fearful faces, as well as the fearful aspects of other facial emotions. Additionally, she had trouble reconstructing the visual aspects of fearful faces, even though she could verbally describe the concept. In contrast, individuals with unilateral damage did not show any deficits in the recognition of facial emotions.

Calder at al. (1996) conducted a similar experiment with two individuals who had suffered bilateral amygdala damage as a result of surgery for epilepsy and encephalitis. It is important to note that one of the individuals was previously studied in Young et al. (1995), but they did not look for specific detriments in types of emotions. This study also expanded on previous work by "morphing" faces to form a continuum of facial emotions. They found that both participants were especially impaired at recognizing fearful faces, but also showed some difficulty recognizing angry faces. Their impairment with the morphed faces was most severe in the region of the fearful face prototype.

In contrast to the previous findings, Hamann et al. (1996) reported on two men who had survived encephalitis with complete bilateral lesions of the amygdala. They tested these men using the same procedures as Adolphs et al. (1994) and found no impairments in their ability to recognize facial expressions. They suggest that the amygdala may be important in emotion processing, but it is not integral to the recognition

of facial emotions. They speculate that the difference in findings may have resulted because the two participants in this study did not incur damage to the amygdala until late in life.

In response to Hamann et al. (1996), Brok et al. (1998) studied five post-encephalitic individuals, four of whom had suffered extensive amygdala damage, and one whose amygdala had mostly been spared. They found that three out of the four had normal functioning on every emotion except fear. The fourth had more severe difficulties which the authors attribute to more extensive damage of the temporal lobes. They analyzed the responses of those individuals and found that the most common misattribution of fearful faces was as surprise. In contrast, the individual with the relative sparing of the amygdala did not have difficulties with the recognition of facial expressions. The authors do not know how to account for the findings of Hamann et al. (1996), but note that explanations about differences in IQ and age are not sufficient to explain differences.

Because of the difficulties in comparing across studies as a result of discrepant methods and small sample sizes, Adolphs et al. (1999) conducted a collaborative study looking at nine individuals with bilateral damage to the amygdala, sixteen with other types of brain damage, and seven healthy controls. They found that overall, individuals with amygdala damage had the most problems with recognizing fearful faces; however, the extent of individual's difficulties varied widely from severely impaired to essentially normal. This was the first study with enough statistical power to conclude that amygdala damage is significantly associated with difficulty in recognizing fearful faces. Two facets stand out from this study: 1) the two individuals tested by Hamann et al. (1996)

were included, and 2) some of the individuals with amygdala damage had trouble recognizing other negative emotions besides fear.

Another case study of an individual with bilateral amygdala damage found a bias towards labeling fearful and angry faces as happy faces (Sato et al., 2002). When angry and fearful faces were morphed with happy faces, this bias was further demonstrated because the individual rated the angry and fearful faces with some happy content as happy, whereas healthy control participants rated them as angry and fearful respectively. The authors speculate that previous studies did not find a bias towards happy faces because the extent of brain damage in their participants was not limited solely to the amygdala.

In order to further elucidate the processing of facial emotions by individuals with bilateral amygdala damage, Graham, Devinsky and LaBar (2007) modified the paradigm to allow unlimited viewing time. Even given unlimited time, the participant still displayed some difficulties with neutral-to-anger and fear-to anger morphs. Surprisingly, she did not display difficulties with the neutral-to-fear morphs. Despite correct answers, her reaction times were considerably longer, which led the researchers to speculate that she was able to use heuristics in guiding her decision-making. Support for this contention came from the fact that her performance declined significantly compared to healthy controls and individuals with unilateral lobectomies when a time limit was put on the task. This is interpreted as evidence that the amygdala is involved in automatic processing of fearful and angry faces.

Overall, there is strong emerging evidence that the amygdala is integral to the automatic processing of fearful faces. Some evidence exists that the amygdala is

involved in the processing of other emotions, particularly anger, but the evidence is not as strong. Only one study did not find impairments in facial emotion processing, and the reason for this is unknown. One limitation to this area of the literature is the scarcity of individuals with bilateral damage to their amygdaloid regions. Most of the studies cited previously were single case studies or recruited participants that had been in prior studies. Despite this, it is clear that the amygdala is somehow involved in the processing of facial emotions, but it remains unclear the extent. Interestingly, one of the participants from the previously cited studies participated in an emotional music recognition task. She had difficulty recognizing sad and scary music, frequently labeling both as peaceful music. In contrast, participants without amygdala damage never labeled scary music as peaceful (Gosselin, Peretz, Johnsen & Adolphs, 2007). Thus, individuals with amygdala damage may have difficulty with more general emotion processing, and deficits may not be limited to facial expression.

Studies of Amygdala Activation in PTSD

It is well established that the amygdala plays a role in the processing of emotions, but what role the amygdala plays in regards to the genesis and maintenance of PTSD symptoms is not as clear. Individuals with PTSD show an increase in physiological reactivity and PTSD symptom expression when exposed to traumatic stimuli (Bremner et al., 1999). Thus, one way to assess the neural substrates of PTSD is to expose participants to traumatic imagery during scanning.

Studies have generally found an increase in activation in the amygdala during symptom provocation paradigms. There was an increase in blood flow to the limbic systems (including the amygdaloid region) in individuals with PTSD while they imagined

a personal traumatic script as compared to imagining a personal neutral script (Rauch et al., 1996) in a sample of men and women recruited from the trauma clinic of an urban hospital. This pattern of activations is similar to an earlier study comparing male Vietnam veterans to controls, in which they reported activations to the right amygdala that were not present in controls (Shin et al., 1994). A more recent study of Vietnam veterans (including male combat veterans and female nurse veterans) using personal script imagery reported that changes in the medial frontal gyrus were inversely correlated with changes in both the left and right amygdaloid complex. Liberzon et al. (1999) found that activation occurred in the left amygdala using generic combat sounds instead of personalized scripts. Using the same paradigm with generic combat sounds, Pissiota et al. (2002) found right amygdala activation in veterans of recent conflicts. In contrast, while Bremner et al. (1999) found decreased activation in the medial prefrontal cortex in individuals with PTSD as compared to controls, they did not find increased amygdala activations. One caveat in interpreting these findings is that only individuals with psychophysiological reactivity to trauma-related cues were included, and findings cannot be extended to individuals with PTSD but without reactivity.

There may be a "trauma specific" pathway that becomes activated in individuals with PTSD (Hendler et al., 2003). They measured responsivity of the visual cortex and amygdala to trauma-related, neutral and scrambled visual stimuli at different levels of awareness in veterans with or without PTSD and found that the visual cortex is activated in response to trauma-related stimuli at preconscious levels in individuals with PTSD, but not in others. Additionally, they found increased amygdala activation to trauma-related stimuli at all levels of awareness. They argue that this represents, "the impact of the

traumatic experience on sensory-emotion interaction processes in PTSD" (pg. 596). Thus, the experience of trauma somehow changes the processing of emotionally salient information, which may imply changes in the perception of emotional faces.

In addition to increased amygdala activity to specific trauma-related stimuli, three studies have reported increased responses to facial emotion stimuli in individuals with PTSD (Armony, Corbo, Clément & Brunet, 2005; Rauch et al., 2000; Shin et al., 2005). Rauch et al. (2000) presented masked fearful, happy and neutral faces to Vietnam veterans with and without a current diagnosis of PTSD in a passive viewing task. They found that all participants had greater activation of the amygdala to the masked fearful faces than the masked happy faces, but that the amygdala response of the individuals with PTSD was significantly higher than that of control participants. Additionally, using the masked paradigm, there was not significant activation of the medial prefrontal cortex. They concluded that the amygdala response is independent of the "top down" control of the medial prefrontal cortex. Additionally, the strength of the amygdala response was associated with the current severity of symptoms, while there was no correlation between amygdala response and combat exposure intensity. Shin et al. (2005) followed up and extended on this work by presenting overt happy and fearful faces during a passive viewing paradigm. They again found increased amygdala activations to fearful versus happy faces, and activity in the medial prefrontal cortex was negatively correlated with amygdala activations only in the PTSD group. A study of individuals diagnosed with acute PTSD reported similar exaggerated activity to masked fearful faces, but found increased amygdala activity as a function of symptom severity to unmasked happy faces compared to unmasked fearful faces (Armony et al., 2005). In contrast, a study using

event-related potentials (ERPs) reported increased ERPs to fearful faces compared to neutral faces in the non-PTSD control group, but not in individuals with PTSD (Felmingham, Bryant & Gordon, 2003). They interpret this as evidence that individuals with PTSD have difficulty discriminating non-threat and generalized threat stimuli. It is unclear why they found seemingly inconsistent results, but the differing methodology makes strong comparisons impossible.

In summary, researchers have reliably found exaggerated activation of the amygdala in response to trauma-related stimuli. Several have also reported decreased prefrontal cortex activations, which bolsters the model of the neurobiology of PTSD outlined earlier. Additionally, the idea of a "trauma specific" pathway is remarkably similar to the "fear structures" as well. Given that trauma specific stimuli activate the amygdala to a greater degree in individuals with PTSD, it is interesting that negative facial expressions have also been implicated in this regard. While there is evidence on a neurological level that negative facial stimuli have a differential impact on individuals with PTSD, it remains unclear if there might be effects at a behavioral level.

Alternative Explanations for Facial Affect Recognition Difficulty I: Child Maltreatment

One reason that individuals may have difficulty recognizing the facial emotions of others is as a result of traumatic experiences, and not because of PTSD. There is not a literature on adult trauma and facial emotion recognition; however, such a literature exists for child maltreatment. It is a tautology that children who grow up in abusive and neglectful households are treated differently than other children; however, there are differences above and beyond the abuse itself. In one study, for example, the rate of negative behaviors by abuse mothers was 77% greater than control mothers (Burgess &

Conger, 1978). Abusive parents are also more likely to ignore or react negatively to their children than non-abusive parents (Kavanagh, Youngblade, Reid & Fagot, 1988). Additionally, children with abusive parents were found to talk less (in positive conversation) than non-abused children, although both groups were just as likely to react positively to positive parent behavior.

It appears to be the case that children who are raised by abusive parents are exposed to a much more negative environment than children raised by non-abusive parents, and this extends to emotional facial expressions. Nelson (2001) reviewed the evidence for the development of facial recognition and concluded that face recognition is an experience-expectant process. This means that while recognizing faces requires learning, the brain is already primed for the learning. In short, infants learn to recognize faces much more quickly than would be expected from a pure learning model. The evidence for this conclusion comes from many studies that have demonstrated prosopagnosia (the inability of individuals to recognize other's faces). Thus, there appears to be a specific neural basis for the recognition of faces, but it is less clear whether facial emotion recognition has a specific neural basis and is an experience-expectant process. This may not be the case as several studies have documented facial emotion recognition difficulties in maltreated children.

Camras et al. (1990) observed the facial behavior of 20 maltreated children, 20 nonmaltreated children and their mothers during a laboratory interaction task and during home visits. They found that both maltreatment status and mothers' facial behavior were significant predictors of the children's facial emotion recognition scores. Specifically, they found that maltreated children performed significantly worse on a facial emotion

recognition task, and children's whose mothers were less expressive also tended to perform worse. This study did not analyze emotion recognition by specific emotion, but the authors suggest that maltreated children do not benefit in their recognition skills from intense negative interactions. They postulate that anxiety interferes with learning in strongly negative environments.

Other studies have found abnormalities in responding to angry faces in maltreated children. Pollak et al. (2000) compared the facial emotion recognition abilities of physically abused, neglected and control children. They found that neglected children had more difficulty discriminating among angry, sad and fearful faces, and physically abused children had difficulty with sadness and disgust. While physically abused children did not have difficulty recognizing angry faces, they tended to show an attention bias towards those faces. In a later study, Pollak and Tolley-Schell (2003) again found that physically abused children demonstrated a bias towards angry faces. This was a follow-up of Pollak et al. (2001), which found differential activations in P3b brainwaves in response to angry and fearful faces between maltreated and nonmaltreated children. Specifically, in the 2003 study they found that maltreated children had increased P3b activation in response to trials that the child thought would include an angry face. They conclude that this activation represents increased cognitive resources needed to disengage from a previously cued location.

Overall, there is clear evidence that child abuse and neglect can affect an individual's facial emotion recognition ability. There is also evidence that children learn to recognize facial emotions, and that this skill may not be experience-expectant. However, none of these studies assessed whether children were suffering from PTSD as a

result of the abuse and neglect they had experienced. Thus, it is possible that some of these findings are due to PTSD rather than the trauma of abuse.

Alternative Explanations for Facial Affect Recognition Difficulty II: Alexithymia

A possibility that has not been considered heretofore is that individuals with PTSD have difficulty recognizing the facial emotions of others due to alexithymia. In fact, a recent meta-analysis of studies examining the relationship between PTSD and alexithymia concluded that there is a positive correlation between the two (Frewen, Dozois, Neufield & Lanius, 2008). The term "alexithymia" was coined in the 1970's in response to observations that individuals with psychosomatic disorders had marked difficulty expressing and describing their emotions (Sifneos, 1997). Since that time, the concept of alexithymia has been refined and now generally refers to three related difficulties: 1) difficulty identifying and describing emotions, 2) difficulty describing feelings, and 3) an externally oriented cognitive style (Badura, 2003; Söndergaard & Theorell, 2004). It is noteworthy that these difficulties sound strikingly similar to the emotional numbing that is characteristic of PTSD, and it is not surprising that many studies have since found that individuals with PTSD usually have alexithymia (Frewen, Dozois, Neufield & Lanius, 2008). For example, a study of holocaust survivors with and without PTSD found that alexithymia symptoms were related to PTSD and not to trauma per se (Yehuda et al., 1997). This has led to speculation that alexithymia is not a separate construct but would be better subsumed under the emotional numbing criteria of PTSD (Badura, 2003). In sum, alexithymia has been shown to be a common symptom in individuals with PTSD. What is less clear is whether individuals' difficulty in identifying their emotional state might extend to difficulty recognizing the facial emotions of others.

Sifneos (1997) argued that the etiology of alexithymia must ultimately be answered through neuroimaging studies. Since that time several studies have reported on differential activations in individuals with high scores on a measure of alexithymia. In a study comparing fMRI activations of individuals high on alexithymia to individuals low on alexithymia, researchers reported differential activations in response to positive and negative facial emotions (Berthoz et al., 2002). Specifically, they reported that individuals high in alexithymia had lower activations of the left mediofrontal-paracingulate gyrus in response to negative facial expressions and higher activations of the anterior cingulate, mediofrontal and middle frontal gyri. Interestingly, they did not find any differential activations of the amygdala, hippocampus, or hypothalamus. They suggest that these areas may be important in the emotional response to stimuli but less relevant for their interpretation. A similar study using PET technology also found differential activations of several cortical areas but no difference in activations in the amygdala, hippocampus and hypothalamus (Kano et al., 2003). Specifically, they reported that the biggest differences occurred in response to angry faces with individuals high in alexithymia showing significantly less activation of the anterior cingulate cortex (ACC). They argue that the ACC is integrally involved in assessing emotional arousal and the attentive components of emotion. However, they note that the decreased activations were limited to negative faces and suggest that individuals with alexithymia may have particular difficulty with negative emotions. Overall, there is ample evidence of the ACC's involvement in individuals high in alexithymia (Aleman, 2005). Thus, individual's high in alexithymia have different neural activations in response to

emotional facial stimuli which could underlie difficulties in recognition. However, Berthoz et al. (2002) argued against this interpretation.

Emotion Recognition of Faces in Psychiatric Disorders

A fair amount of data has accumulated that indicate several psychiatric disorders are associated with abnormalities of facial emotion recognition. Recently, Kornreich and Philippot (2006) reviewed the evidence for facial emotion recognition abnormalities in a variety of neuropsychiatric disorders. First, the evidence for difficulties in Parkinson's is contradictory. While one study found difficulties, subsequent studies have failed to replicate that finding. They note that the results could have been due to depression, which was not well-controlled in these studies.

Second, Alzheimer patients have shown difficulty with emotional face processing, but nonverbal processing overall seems relatively intact compared to the other cognitive difficulties associated with Alzheimer's. Overall, abnormalities seem to be due to visuospatial dysfunctions.

Third, individuals with Huntington's disease (HD) and preclinical individuals have difficulty processing negative emotions. A large study of individuals with preclinical HD found a negative correlation between time to diagnosis and negative facial emotion recognition (Johnson et al., 2007). Participants underwent MRI imaging, but no association was found between striatal volume and emotion recognition. The authors conclude that the decline in emotion recognition must be due to functional changes in other areas of the brain and suggest that future studies examine activations in the amygdala, orbitofrontal cortex, right somatosensory areas, and others. One study found an association between amygdala volume and the ability to recognize happy faces (Kipps,

Duggins, McCusker & Calder, 2007). They note that the individuals with preclinical HD show atrophies to other areas of the brain including the parahippocampal region which may contribute to difficulties. Interestingly, they did not find an association of amygdala volume and fear recognition. One factor to consider is that they measured volume and not activation, which differentiates it from the majority of functional studies in this area.

Fourth, Schizophrenia is the most studied in regards to psychiatric disorders and facial emotion recognition. Evidence supports the contention that individuals with schizophrenia have difficulty recognizing facial emotions, but it is unclear whether those difficulties are more pronounced for negative emotions than positive emotions. Also, the underlying neurological bases for these difficulties have not been adequately addressed. Researchers have found that reduced right amygdala volume is associated with impaired facial emotion learning (Exner, Boucsein, Degner, Irle & Weniger, 2004). And, at least one study has directly looked at the association of amygdala volume and facial emotion recognition in individuals with Schizophrenia, and reported that decreased amygdalar volume was associated with greater difficulty recognizing sadness, surprise, disgust, and anger (Namiki et al., 2007). They conclude that amygdala dysfunction may be responsible for emotion specific processing problems in individuals with Schizophrenia.

Fifth, studies of facial emotion recognition in depression have, for the most part, postulated a negative bias in keeping with Beck's cognitive theory of depression (Kornreich & Philippot, 2006). Evidence supports difficulties with facial emotion recognition, but does not necessarily show a clear bias towards negative faces. Certain studies have shown over (Gur et al., 1992; Mandal and Bhattacharya, 1985) or under (Ekman, Friesen, Jones & Malstrom, 1969 as cited in Persad & Polivy, 1993) recognition

of sad faces, but the majority of studies has shown a general impairment. Persad and Polivy (1993) compared college students, depressed college students, and depressed psychiatric patients and found that both depressed groups made more errors in identifying facial emotions than non-depressed college students. In a task comparing whether depressed patients show a visuospatial bias or affective bias, researchers found that depressed patients showed both and emphasized that their deficits were not emotion specific (Asthana, Mandal, Khurana & Haque-Nizamie, 1997). Other studies have reported similar findings (e.g. Rubinow & Post, 1992), including in depressed patients with Major Depressive disorder or Schizotypal Personality disorder (Mikhailova, Vladimirova, Iznak, Tsusulkovskaya & Sushko, 1996). Interestingly, depressed patients manifest increased left amygdala activation in response to all facial emotions but particularly fearful faces as compared to matched control participants in a masked faces paradigm. This exaggerated activation was reduced following treatment with antidepressants (Sheline et al., 2001). On the behavioral level of analysis, it is important to test whether depressed individuals improve on a facial emotion recognition task after treatment with antidepressants. Finally, depressed patients with comorbid anxiety disorders may differ from patients without anxiety. In a face-in-the-crowd task, the only difference between depressed patients and matched control patients was that depressed patients without comorbid anxiety were slower to respond to positive faces than other groups (Suslow, 2004).

Sixth, individuals with high-trait anxiety may be better at recognizing fearful faces than others (Surcinelli, Codispoti, Montebatocci, Rossi & Baldaro, 2004), and there may be differential amygdala response associated with trait anxiety. Researchers

observed an inverse correlation between activations to happy versus neutral faces and trait anxiety (Somerville, Kim, Johnstone, Alexander & Whalen, 2004). This correlation was explained mostly by an increased activation to neutral faces in individuals with high trait anxiety. In response to fearful faces, children with anxiety disorders showed increased amygdala activation to fearful faces, while children with depression showed blunted responses to fearful faces as compared to healthy control participants. Moreover, the magnitude of the amygdala change was associated with the children's self-reports of everyday anxiety (Thomas et al., 2001). In contrast, Mullins and Duke (2004) found that accuracy on a facial emotion recognition task was not related to social avoidance. However, they reported that socially anxious individuals were faster to identify angry and fearful faces. They tested college students, and conjecture that ceiling effects may have been responsible for their lack of findings.

More specifically, studies have looked at individuals with Social Phobia and Panic disorder, and report varying difficulties with facial emotion processing. Children with Social Phobia make more errors to faces in general and have higher state anxiety when completing the task (Simonian, Beidel, Turner, Berkes & Long, 2001). Two studies looked at the abilities of individuals with social phobia to recognize critical and accepting faces, and both found a clear bias towards critical faces while one found a bias towards accepting faces among control participants (Coles & Heimberg, 2003; Lundh & Öst, 1996). Additionally, individuals with Social Phobia have shown amygdala activation to neutral faces, while control participants do not (Birbaumer et al., 1998). Stein, Goldin, Sareen, Zorilla and Brown (2002) found increased amygdala activation in individuals with social phobia for angry and contemptuous faces as compared to happy

faces, but did not find increased activation for fearful faces. Finally, severity of social anxiety symptoms has been positively correlated to increased activation of the amygdala to angry, contemptuous and fearful faces in individuals with Social Phobia (Phan, Fitzgerald, Nathan & Tancer, 2005). Results have been much the same in individuals with Panic disorder in that they have shown worse recognition overall. However, individuals with Panic disorder made more mistakes to sad and angry faces, and had a tendency to interpret non-angry faces as angry (Kessler, Roth, von Wietersheim, Deighton & Traue, 2006). Additionally, children of parents with Panic disorder take longer to identify fearful faces and report more fear when completing the task (Pine et al., 2005).

Finally, some evidence of facial emotion processing abnormalities has been presented for individuals with recently detoxified alcoholics, and patients with Bipolar, Anorexia, Obsessive Compulsive, Antisocial personality, and Borderline personality disorder; however, the results have been mixed and there is not strong evidence of any particular bias (Kornreich & Philippot, 2006). Individuals in a manic state of Bipolar I performed worse in identifying fear and disgust than healthy controls, but euthymic Bipolar I and II individuals did not differ significantly from healthy control participants (Lembke & Ketter, 2002). In contrast, adolescents with bipolar disorder made more errors in identifying children's faces, but not adults. They were more likely to identify faces as angry, which differed from both healthy and anxious participants who themselves did not differ significantly (McClure, Pope, Hoberman, Pine & Leibenluft, 2003).

Several studies have looked at aspects of face recognition bias in individuals with PTSD. Islam-Zwart, Heath and Vik (2005), for example, tested female inmates both with and without a history of sexual assault on the Weschler Memory Scale Faces I and II subtests. They found that women with a history of sexual assault performed better on both the immediate and delayed recall of faces, and those with PTSD performed better on immediate recognition but not delayed. It is unclear whether there are PTSD specific results, and this study only tested memory for faces, not facial emotion recognition, which has been more associated with amygdala activity. In contrast, in a study of crime victims with acute stress disorder, researchers reported a general memory impairment, and bias for faces that were perceived as hostile (Paunovic, Lundh & Öst, 2003). A study looking at the affective competence of recovering alcoholics (by having them rate the emotional valence of faces) also included a group of individuals with PTSD, and found that the PTSD group attributed less negativity to the negative stimuli (Clark, Oscar-Berman, Shagrin & Pencina, 2007). Whether this constitutes a recognition bias, however, remains an open question. Only one published study has reported on facial emotion recognition abnormalities in individuals with PTSD, and it was not the primary focus of the study (Masten et al., 2007). Masten et al. (2007) noted that childhood maltreatment has been associated with atypical processing of emotion and the development of PTSD, but little evidence relates the two. They found that maltreated children were faster at recognizing faces, and that this difference was most pronounced for fearful faces. However, there was no evidence that sensitivity to fearful faces was a predictor of current PTSD diagnosis because no differences emerged between groups in accuracy of labeling the emotional faces. Maltreatment may be linked to emotional

processing in a separate pathway than PTSD, or this particular study may not have had sufficient power to detect differences in the maltreatment group because 72% of maltreated children were given a probable diagnosis of PTSD.

Finally, an unpublished dissertation examined the facial emotion recognition abilities of individuals with PTSD, individuals with no trauma, and individuals with trauma but no diagnosis of PTSD (Sta. Maria, 2002). They found a trend towards more overall errors in individuals with PTSD, and significant difference in number errors to fearful faces when the PTSD group was considered separately. The sample consisted of 54 veterans, and those with a PTSD diagnosis had symptoms for more than five years.

In summary, there is emerging evidence of biases and difficulties to facial emotion recognition in many psychiatric disorders. For example, depressed individuals seem to have difficulty recognizing facial emotions in general. Trait anxiety has been linked to better recognition of fearful faces, and individuals with Schizophrenia and premorbid Huntington's disease have demonstrated decrements in facial emotion recognition. However, while studies have assessed facial recognition in PTSD as well as memory biases for specific types of faces, only one study has yet to directly test the facial emotion recognition abilities of individuals with PTSD.

Gender, the Amygdala and Facial Emotion Recognition

Animal studies have found sex differences in the structure of amygdala subnuclei, and behavior after amygdala lesions. However, sex differences in humans have been hard to evaluate because most studies have employed mixed samples of men and women. Even so, the processing of facial emotion is one of the specific domains in which there is some emerging evidence of differences (Zald, 2003). McClure et al. (2004) found

differential activations for adult men and women in response to emotional faces, but did not find differences in adolescents. Specifically, they found that adult women's frontolimbic structures activated in response to unambiguous threat stimuli, but not to ambiguous stimuli while men's frontolimbic structures activated in response to both types of stimuli. Amongst individuals with Schizophrenia, men over-attributed anger and women over-attributed sad to neutral stimuli (Weiss et al., 2006). In contrast, a study testing gender differences among healthy participants found that women performed better than men in the Caucasian sample, but not in the African American sample (Terracciano, Merritt, Zonderman & Evans, 2003).

In addition to sex differences in the processing of facial emotions, a recent meta-analysis examined gender differences in the development of PTSD and found sex differences in the rates of development of PTSD after controlling for types and level of trauma exposure. First, women are more likely than men to experience sexual assault and child sexual abuse, whereas men were more likely to experience accidents, nonsexual assaults and other types of disasters. Overall, females were more likely to meet criteria for PTSD, and this remained after controlling for types of exposure to trauma. Thus, it appears that women may be more susceptible to the development of PTSD than men (Tolin & Foa, 2006).

Statement of the Problem

Overall, there is ample evidence of facial emotion processing abnormalities in psychiatric disorders, although for many the trends remain unclear. In depression and social phobia, the data have shown clear links between viewing emotional faces and amygdala activation, and in turn difficulties with emotion recognition tasks. However,

no links have yet been established between PTSD and facial emotion recognition abnormalities, except for one study with a relatively small sample size. This is a striking gap in the current knowledge given the weight of the evidence: 1) the amygdala is implicated in general emotion processing, as well as facial emotion processing specifically, 2) individuals with lesions to the amygdaloid regions have shown considerable difficulties with recognizing certain facial emotions, 3) the amygdala has increasingly been seen as a crucial mechanism in the pathogenesis of PTSD, and 4) facial emotion recognition difficulties, as well as increased amygdala responses have been identified in other psychiatric disorders. PTSD fundamentally affects the interpersonal functioning of individuals, and it is important to begin to identify the processes contributing to those difficulties. The current study proposes to examine the nonverbal decoding abilities of individuals in a primary care setting using a standardized facial emotion recognition task (DANVA-II AAAF; Nowicki, Glanville & Demertzis, 1998). Unlike any studies to date, the use of the DANVA provides for a more ecologically valid and fine-grained analysis of facial emotion recognition. First, unlike any other test employing facial emotion stimuli, the DANVA has a specific African American version. This may be important given that the sample for the current study will be recruited from the primary care setting of an urban hospital whose patients are almost exclusively African American. For example, a recent study on trauma exposure that recruited individuals from a similar large urban hospital reported a sample that was 96% African American (Alim et al., 2006). In-group advantages for cultural and ethnic groups in the recognition of facial expressions have been established according to a meta-analysis on group differences in emotion recognition (Elfenbein & Ambady, 2002). Specifically,

researchers have reported differences in performance among Caucasians and African Americans on the different DANVA-2 versions (Weathers, Frank & Spell, 2002). Thus, using stimuli from individuals of the same race as the majority of the sample will maximize the ecological validity because there remain high levels of residential segregation, especially in low-income urban areas (Logan, Stults & Farley, 2004). For this reason, it is likely that the majority of individuals in the sample will interact with other African Americans the majority of the time. The second advantage of using the DANVA is that it provides greater specificity in identifying facial emotion recognition difficulties because it has both high intensity and low intensity emotional stimuli. Other studies have used computer morphing techniques to manipulate the intensity of the emotions expressed by the Ekman faces (Ekman & Friesen, 1976), but the DANVA differs in that the intensity ratings come from a normative sample. The following hypotheses were tested:

1. *Facial Emotion Abilities will be associated with current PTSD symptoms*—Sta. Maria (2002) found a trend towards more errors overall in individuals with PTSD. However, her sample size was quite small. The current study employed a larger sample, and thus it is predicted there will be a significant effect such that individuals with a PTSD diagnosis will make more overall errors on the DANVA.
2. *Individuals with PTSD will make more errors to fearful faces than other emotions*—Following the findings of Sta. Maria (2001) and given the weight of the evidence regarding the amygdala's involvement in the maintenance of PTSD and emotion processing, it is predicted that

individuals with PTSD will make more errors to fearful faces than other types of faces.

3. *PTSD symptoms and symptoms of depression will differentially predict patterns of errors*—PTSD and depression are often comorbid disorders, and some studies have found a general impairment to facial emotion recognition in depression (Kornreich & Philippot, 2006, Persad & Polivy, 1993). However, it is predicted that a significant effect for PTSD on errors to overall faces will remain after controlling for symptoms of depression. In accordance with Beck's cognitive theory of depression, it is predicted that levels of depression as measured by the BDI will correlate with fewer errors to sad faces specifically, and will predict faster responding to negative emotion pictures (Gur et al., 1992; Kornreich & Philippot, 2006; Mandal and Bhattacharya, 1985).

4. *History of physical abuse will predict faster responding to angry faces*—Previous studies have found that physically abused children orient more quickly to angry stimuli (Pollak et al., 2000; Pollak et al., 2001; Pollak & Tolley-Schell, 2003). Thus, it is predicted that individuals with a history of physical abuse will make fewer errors to angry faces and have faster response times to those faces.

5. *History of neglect will predict more errors*—Neglected children have been found to be worse at differentiating facial emotions (Camras et al., 1990; Pollak et al., 2000). Thus, it is predicted that individuals with a history of neglect will make more errors to faces overall.

6. *PTSD and child maltreatment will interact to predict errors*—Given the evidence that individuals who experience child maltreatment may have difficulty discriminating facial emotions (Camras et al., 1990; Pollak et al., 2000), and the prediction that individuals with PTSD have the same difficulty (Sta. Maria, 2002), it is predicted that they will interact such that individuals with both PTSD and child maltreatment will make significantly more errors than other groups.
7. *There will be gender differences in error patterns*—There is evidence of differential amygdala activity in men and women (McClure et al., 2004; Zald, 2003), as well as evidence of differential responses to facial emotion stimuli (Weiss et al., 2006). Thus, it is predicted that men and women with high PTSD symptoms will show differential error patterns. Specifically, it is predicted that women will make significantly more errors to angry faces (unambiguous threat) than women without PTSD, and men will make significantly more errors to angry and fearful faces (unambiguous and ambiguous threat) than men without PTSD (McClure et al., 2004).

Method

Sample

The sample consisted of 162 individuals with 52 males and 110 females. The ethnic make-up of the sample is 85.8% African American, 8.0% Caucasian, 1.2% mixed, 0.6% other, and 4.3% not reported. Participant's ages ranged from 18 to 74 years old.

Additionally, types of trauma exposure along with gender differences in rates of exposure are presented in Table 1.

Procedure

Participants were recruited from the General Medical and Obstetric/Gynecological Clinics at a publicly funded, not-for-profit healthcare system that serves the low-income and homeless population in Atlanta, Georgia. The Clinic population is overwhelmingly minority (>80% African American and 5-10% Hispanic) and poor (87% with monthly household income < $1000). Data were collected as part of the Grady Trauma Project, an ongoing 5-year NIH-funded study of risk and resilience to PTSD at Grady Hospital.

Participants were approached while waiting for appointments and asked if they would like to participate in a study. Of those approached, about 58% agreed to participate. Once participants agreed, they were read a consent form and asked to sign. Participants were then read each question by a trained interviewer who recorded their responses onto a tablet PC. The deomgraphics questionnaire, Traumatic Events Inventory (TEI), the Beck Depression Inventory (BDI), the Modified Posttraumatic Stress Scale (MPSS-SR), and the Childhood Trauma Questionnaire (CTQ) were administered during this interview, as well as several other measures that were not analyzed in the present study. A subset of participants were selected to return for further interviews. The selection was based first on individuals who indicated that they would like to continue further, and they were then randomly selected to be scheduled. Participants then met with a member of the team for further assessment that included cognitive and other measures that were not analyzed for this study. Once the participants completed this second assessment, they were scheduled for a third meeting with our team, and the

Clinician Administered PTSD Scale (CAPS), the Diagnostic Assessment of Nonverbal Accuracy (DANVA), and the Structured Interview for DSM Disorders (SCID) were administered during that meeting. If participants were called for their appointment before the first interview was finished, they were paid the full amount for their time, scheduled for their second appointment with the team, and the interview was completed at that time. During the time of data collection, 1093 participants completed the screening, while 162 participants completed the third interview with the team. Overall, the data for this sample were collected from Spring, 2007 to Summer, 2008.

During their third visit with the team, participants completed a computerized acoustic startle procedure, which included collecting physiological reactivity data and were administered the DANVA on the same laptop immediately afterward. Trained interviewers then completed a battery of instruments (which included the CAPS and SCID) with participants.

Measures

Assessment of PTSD Symptoms

Modified Posttraumatic Stress Scale (MPSS-SR; Falsetti et al., 1993)–This is a brief 17 item measure that assesses frequency of PTSD according to DSM-III-R criteria. The major change from the PTSD Symptom Scale (PSS; Foa, Riggs, Dancu & Rothbaum, 1993) is that items are not keyed to a specific trauma. Since many of the individuals recruited for this study had multiple traumas that meet criterion A, it was important to capture PTSD symptoms regardless of the trauma. Frequency items are rated on a 4-point scale (ranging from 0 = “not at all” to 3 = “5 or more times per week”). The MPSS-SR can be used to make a preliminary diagnosis according to DSM-III-R

criteria, or can be used as a continuous score for PTSD symptoms. For the purposes of this study, it will be used as a continuous score. See Appendix A for this measure.

Clinician Administered PTSD Scale (CAPS, Blake et al., 1995)—This is considered the gold standard for the assessment of PTSD (National Center for PTSD Research). It is a clinician administered 30-item interview that corresponds to DSM-IV criteria for PTSD. It can be used to assess PTSD symptoms over the past week, month or lifetime. For the purposes of this study, PTSD symptoms were assessed for the past month and lifetime. The current study differed from the standard administration protocol, which stipulates that the interviewer ask about lifetime symptoms only if criteria for current PTSD are not met, by asking about current and lifetime symptoms simultaneously. Each item of the CAPS has two parts, frequency and intensity, which are both scored on a 5-point scale from 0 to 4. A general cut-off rule of frequency greater than or equal to 1 and intensity greater than or equal to 2 for a symptom to count towards diagnosis was employed in assigning PTSD diagnosis.

Assessment of Traumatic Experiences

Traumatic Events Inventory (TEI; Schwartz et al., 2006; Schwartz et al., 2005). The Traumatic Events Inventory (TEI) is a 14-item screening instrument for lifetime history of traumatic events. For each traumatic event, the TEI assesses experiencing and witnessing separately, and it also assesses confrontation of traumatic events where appropriate. In addition, the TEI also asks the number of times that each event has occurred; age at self-perceived "worst" instance for a given traumatic event; and feelings of helplessness or horror for each traumatic event. For the purposes of this study, only traumatic events that occurred during adulthood were considered. While simply summing

the number of traumatic experiences is not ideal because it does not take into account when the trauma occurred and severity of trauma, previous work with this population has indicated that it is the best way to estimate overall trauma exposure (Binder et al., 2008; Davis et al., 2008; Schwartz et al., 2006). This is because the same event does not affect every individual in the same way. However, adult trauma exposure was significantly correlated with PTSD symptoms as measured by the MPSS (r = .36). Thus, summing traumas appears to be an acceptable approximation for overall trauma exposure.

See Appendix B for this measure.

The Childhood Trauma Questionnaire **(**CTQ**)** (Bernstein, Ahluvalia, Pogge, & Handelsman, 1997)— This is a 28-item, self-report inventory assessing three domains of childhood abuse (sexual, physical, and emotional), and two domains of childhood neglect (physical and emotional). Cutoff scores for each category have shown excellent sensitivity and specificity in correctly classifying cases of abuse and neglect in psychiatric patients (Bernstein et al., 1997; Bernstein & Fink, 1998; Bernstein et al., 1994; Bernstein et al., 2003). The CTQ applies to early childhood trauma that occurred at or before 12 years of age. The variables produce both present and absent (above and below cutoff scores for no or minimal abuse) and severity scores for each type of abuse. The current study employed 5 data points derived from the CTQ subscales: physical abuse, sexual abuse, emotional abuse, physical neglect and emotional neglect. These five data points were derived in the following manner: on each subscale a score of less than 10 was considered absence of abuse, and 10 or higher was considered presence of abuse. Additionally, a variable was created that considered the presence of one or more types of abuse as positive for abuse, and no abuse of any kind as absent. The Cronbach's alpha

for the five subscales for this sample are 0.83 (physical abuse), 0.96 (sexual abuse), 0.84 (emotional abuse), 0.70 (physical neglect) and 0.88 (emotional neglect). See Appendix C for this measure.

Assessment of Facial Recognition

Diagnostic Analysis of Nonverbal Accuracy, Form 2, African American Adult Facial Expressions *(DANVA2-AAAF; Nowicki & Carton, 1992; Nowicki & Duke, 1994; Nowicki et al., 1998)*—The DANVA2-AAAF is made up of 32 photographs of 12 African American adults portraying low- and high-intensity happy, sad, angry, and fearful faces. The participants were undergraduates who had volunteered to model facial expressions, and photographs were taken during brief conversations about what made posers happy, sad, angry, and fearful. The pictures were then presented to African American college students and fourth graders, and 84 of the photos were identified by at least 80% of the judges as displaying the selected emotion. Of those, an equal number of men and women and high- and low-intensity photographs were randomly selected to form the final pool of 32 photographs. The scoring is similar to other DANVA-2 subtests, with higher error scores indicating greater difficulty.

Evidence of construct validity and reliability of the DANVA2-AAAF has been obtained from Nowicki et al. (1998), who found that 32 middle-class, African American fifth grade students scored significantly lower than 36 African American undergraduate students attending a predominantly African American college, $t(67) = 7.86$, $p < .01$. Test-retest reliabilities over a four-week period were also sufficient for both groups (fifth grade, $r(28) = .70$, $p < .05$; college $r(27) = .81$, $p < .05$). Regarding construct validity, Nowicki et al. (1998) also found that DANVA2-AAAF accuracy was positively

associated with self-esteem scores for fifth graders, $r(31) = .41$, $p < .05$, and college students, $r(34) = .70$, $p < .05$.

The computerized DANVA has been modified from the standard administration for the purposes of this study. In the standard administration, faces are shown for a maximum of two seconds. This study was interested in individuals' reaction times to facial stimuli; thus, the procedure was modified such that the facial emotion stimulus was presented until the respondent made a choice. This procedure has been successfully employed in at least one other study (Mullins & Duke, 2004). See Figure 1 for stimuli.

Assessment of Other Functioning

Beck Depression Inventory *(BDI-II)*(Beck, Steer & Garbin, 1988)—The BDI is a 21-item self-report measure that assesses symptoms of depression. The BDI-II has been employed in countless research studies, and generally has shown good reliability and validity. In published research, internal consistency ranged from 0.73 to 0.92 with a mean of 0.81 for nonpsychiatric samples (Beck et al., 1988). The split-half reliability coefficient for the measure is .93. The BDI-II provides cut-off scores for clinically significant depression; however, the appropriateness of those scores is variable. The Cronbach's α for this sample is 0.94.

Structured Clinical Interview for DSM-IV (SCID-I)(First, Spitzer, Gibbon & Williams, 2002)—The SCID-I is a semi-structured interview that assesses current and lifetime diagnoses for the major axis I disorders. The reliability of the SCID modules employed in this project is generally good, with most studies reporting reliability statistics above 0.60 (Psychometrics "SCID website," n.d.). Additionally, several studies

have found the SCID-I to be superior to a standard clinical interview in making diagnoses (Psychometrics "SCID website," n.d.).

Results

In order to test the hypotheses, there are several sources of information for trauma history, mood symptoms, and PTSD symptoms. Data on trauma histories are contained in the CTQ and the TEI, data on mood symptoms are contained in the BDI and the SCID, and data on PTSD symptoms are contained in the MPSS and the CAPS. This means that for both PTSD and depressive symptoms, there are categorical and continuous measures of symptoms. If the effects of trauma and PTSD symptoms on facial emotion recognition are continuous (such that difficulties with recognition increase as symptoms or exposure increase), then the continuous measures may be more sensitive to these effects. However, the continuous measure were administered during the initial meeting with our team, were conducted in the hospital waiting room, and were designed as screening measures. The categorical measures were administered during the third meeting with our team, and the measures are designed to make accurate diagnoses. For these reasons, both ANCOVA's and regressions will be used to test each hypothesis where appropriate. The comparison groups for the ANCOVA's employing the SCID and CAPS were created in the following manner: for depression, the SCID was used to create groups of individuals with current depression versus those who do not have current depression, while the CAPS was used to create groups of individuals who have no diagnosis, a lifetime diagnosis, or a current diagnosis of PTSD. Further, amongst the no diagnosis group, individuals who had never experienced a criterion A stressor were separated from individuals who had experienced such a trauma but had no diagnosis; thus, four groups were created from the

CAPS: no trauma, trauma but no diagnosis of PTSD, a lifetime only diagnosis of PTSD, and a current diagnosis f PTSD. The means and standard deviations for the continuous measures are presented in Table 2, and the number of individuals meeting criteria for current and lifetime PTSD, current depression, and who experienced childhood physical, sexual or emotional abuse (as defined by the cutoff score presented in the method section) are presented in Table 3. The correlations amongst age, trauma exposure, childhood maltreatment, depression, PTSD symptoms, and DANVA errors are presented in Table 4.

Since only a subset of individuals continued to the third interview, during which time the CAPS and SCID were conducted, a comparison of individuals who did not continue with those who did was conducted. A one way ANCOVA was conducted comparing the two groups on demographic characteristics, BDI scores, TEI scores, CTQ scores, and PSS scores. It was found that individuals who did not continue differed in several ways from those that continued. First, participants who continued were significatly older than those that did not, $F(1, 1{,}057) = 17.35$, $p < 0.000$. Second, individuals who continued endorsed significantly more symptoms on the BDI than those that did not continue, $F(1, 877) = 9.22$, $p < 0.05$. Additionally, chi square tests were conducted for several of the categorical variables. Individuals who continued were significantly more likely to be unemployed, χ^2 $(1, N = 697) = 7.29$, $p < 0.05$. Additionally, those who continued were more likely to have a lower household income, χ^2 $(4, N = 1{,}030) = 12.13$, $p < 0.05$. There were no significant differences for childhood trauma, adult trauma, or PTSD symptoms.

Overall, tests were preformed looking at the relation of PTSD, depression, and child maltreatment to errors to emotional faces and reaction times to those same faces. First, it was hypothesized that PTSD symptoms would be related to more overall errors and more errors to fearful faces, and it was predicted that these effects would remain after controlling for depression. Second, it was predicted that depression would predict a different pattern of errors than PTSD, specifically that higher BDI scores would be associated with fewer errors to sad faces and faster response times to negative faces in general. Third, it was predicted that childhood physical abuse and neglect would be related to overall errors and faster reaction times, and that maltreatment would interact with PTSD to predict more errors. Finally, it was predicted that men and women would have differential patterns of errors with men having more difficulty with both angry and fearful faces, and women having more difficulty with angry faces.

Hypothesis 1

The first hypothesis was that overall errors on the DANVA would be higher in individuals with PTSD. This hypothesis was supported when using a categorical diagnosis of PTSD. As a first test of this hypothesis, individuals with a current or past diagnosis of PTSD as assessed by the CAPS were compared to a combined group of individuals who had no trauma or had never met criteria for PTSD. The ANCOVA was significant such that individuals who had ever had a diagnosis of PTSD made more overall errors on the DANVA after controlling for childhood (CTQ total score) and adult trauma (TEI total score), $F(1, 116) = 10.47$, $p = 0.020$. It is possible that increased errors to facial emotion recognition may resolve when an individual no longer meets criterion for PTSD. Thus, an ANCOVA comparing individuals with a CAPS diagnosis of current

PTSD to a combined group of all other individuals was performed. Individuals with current PTSD made significantly more errors than others, $F(1, 116) = 4.12$, $p = 0.045$.

As a second test of this hypothesis, a regression was performed with the total score on the MPSS predicting overall errors on the DANVA while controlling for CTQ total score and TEI total score. Using these continuous variables, PTSD symptoms did not significantly predict errors to the DANVA, $\beta = 0.11$, $t(129) = 1.13$, $p = 0.261$.

Hypothesis 2

The second hypothesis was that individuals with PTSD would make more errors to fearful faces in particular; this prediction was partially supported. In order to test this hypothesis, the same analyses were performed as in the first hypothesis with errors to fearful face in place of overall errors. In the ANCOVA comparing lifetime PTSD diagnosis to individuals with no trauma or no PTSD diagnosis while controlling for trauma, errors to fearful faces were not significantly different, $F(1, 116) = 0.104$, $p = 0.748$. When using MPSS total score to predict errors to fearful faces after controlling for childhood and adult trauma, MPSS did not significantly predict errors to fearful faces on the DANVA although there appeared to be a trend towards significance, $\beta = 0.16$, $t(129) = 1.70$, $p = 0.092$. However, when examining the correlations among measures, it was noted that errors to fearful faces were significantly correlated with participant's age, while errors to other types of faces were not. For this reason, the ANCOVA and regression were repeated while controlling for age. The ANCOVA remained nonsignificant, $F(1, 114) = 0.217$, $p = 0.642$, while the regression equation became significant, $\beta = 0.213$, $t(127) = 2.21$, $p = 0.029$.

Hypothesis 3

The third hypothesis has two parts; the first is that error patterns for PTSD will remain after controlling for depression, and the second that high depression scores would be associated with fewer errors to sad faces and faster responding to negative faces. The first part of the hypothesis was supported, and the regression predicting errors to fearful faces continued to be significant. The second part of the hypothesis, however, was not supported. In order to test the first part of the hypothesis, the ANCOVA's and regressions for total errors and fearful errors were repeated while controlling for depression symptoms as measured by the BDI. First, in the ANCOVA testing differences in total errors by PTSD diagnosis, the effect remains significant, $F(1, 112) = 4.136$, $p = 0.044$. Additionally, the regression with PTSD symptoms predicting total errors continues to be non-significant, $\beta = 0.06$, $t(126) = 0.66$, $p = 0.616$. Second, again since errors to fearful faces were significantly associated with age, the following analysis controlled for age as well as depression symptoms. The ANCOVA testing for differences in errors to fearful faces by PTSD diagnosis continues to be non-significant, $F(1,110) = 0.194$, $p = 0.660$. Additionally, the regression using PTSD symptoms to predict errors to fearful faces continued to be significant after controlling for depression, $\beta = 0.321$, $t(124) = 2.58$, $p = 0.011$. Also, ACOVA's and regressions were performed for errors to sad faces using SCID current diagnosis of depression and BDI total score to predict errors on the DANVA. The ANCOVA using the SCID diagnosis of depression to test for differences in errors to sad faces was not significant, $F(1, 110) = 0.35$, $p = 0.556$. Additionally, the regression predicting errors to sad faces using the BDI total score was not significant, $\beta = 0.06$, $t(130) = 0.58$, $p = 0.560$. Finally, to test whether depressive

symptoms were associated with faster reaction times to negative emotional faces, reaction times to sad, angry and fearful faces were combined, and an ANCOVA was performed testing whether individuals with a current diagnosis of depression had faster reaction times. This was not significant, $F(1, 110) = 1.65$, $p = 0.202$. Additionally, bivariate correlations were performed for angry, sad, and fearful faces and BDI scores. Depressive symptoms were not significantly correlated with angry, $r(145) = -0.05$, $p = 0.528$, sad, $r(145) = 0.02$, $p = 0.809$, or fearful faces, $r(145) = -0.04$, $p = 0.635$.

Hypothesis 4

The next hypothesis was that individuals with a history of physical abuse would make fewer errors and orient more quickly to angry faces; however, this was not supported. An ANCOVA was performed looking for differences in error rates to angry faces by physical abuse history while controlling for adult trauma on the TEI. No significant difference was found for physical abuse, $F(1,137) = 2.49$, $p = 0.117$. As a follow-up, a regression was performed using the continuous CTQ physical abuse variable to predict errors to angry faces while controlling for adult trauma exposure. The regression equation was not significant, $\beta = 0.06$, $t(136) = 0.64$, $p = 0.525$. To test whether there was a difference in reaction times to angry faces, an ANCOVA was performed with reaction times to angry faces in place of errors to angry faces. This was not significant, $F(1, 137) = 2.55$, $p = 0.059$; however, it appeared to be trending towards significance. The follow-up regression using the continuous variable for physical abuse was not significant, $\beta = -0.13$, $t(136) = -1.40$, $p = 0.165$.

Hypothesis 5

It was predicted that individuals with a history of childhood neglect would make more errors to all faces, but this was not supported. In order to test this hypothesis, the same analyses were performed as above using emotional neglect instead of physical abuse and overall errors to faces instead of angry errors. First, an ANCOVA was performed that looked for differences in error rates to faces overall in individuals with or without a history of neglect while controlling for adult trauma exposure. This was not significant, $F(1, 136) = 1.20$, $p = 0.313$. The follow-up regression using the continuous variable for emotional neglect was also not significant, $\beta = -0.02$, $t(136) = -0.21$, $p = 0.836$.

Hypothesis 6

It was predicted that childhood maltreatment and PTSD would interact such that individuals with a history of child maltreatment and PTSD would make more errors to all faces than other groups; however, this was not supported. An ANCOVA was performed with current PTSD diagnosis and child maltreatment (combining all types of abuse such that participants with any type of abuse were included in the maltreatment group) as the independent variable and total errors to faces as the dependent variable while controlling for adult trauma exposure from the TEI. The ANCOVA showed a significant main for current PTSD, $F(1,116) = 3.91$, $p = 0.050$, but not for child maltreatment or the interaction. However, the interaction appeared that it might be trending towards significance, $F(1,116) = 2.45$, $p = 0.121$, although not in the direction expected. It appears that child maltreatment may be a protective factor against increased errors to emotional faces seen with a current PTSD diagnosis (see Figure 2). Only 16 individuals

met criteria for current PTSD; thus, there is not likely adequate power for detecting an interaction effect.

Hypothesis 7

First, it was predicted that women with PTSD would make more errors to angry and than those who did not have a diagnosis of PTSD. Second, it was predicted that men with PTSD would make more errors to angry and fearful faces combined than those without PTSD. Neither of these predictions were supported. To test the hypothesis for women, an ANCOVA was performed looking for differences in errors to angry faces by PTSD diagnosis on the CAPS and gender while controlling for trauma exposure on the CTQ and TEI. There were not a significant main effects for PTSD, $F(1, 116) = 2.05$, $p = 0.155$, or gender, $F(1, 116) = 0.005$, $p = 0.823$ nor was the interaction significant, $F(1, 116) = 0.88$, $p = 0.350$. To test the hypothesis for men, a variable was created that combined errors to fearful and angry faces, and the same ANCOVA was repeated substituting the combined angry and fearful errors for errors to angry faces. There were not significant main effects for PTSD, $F(1, 116) = 1.57$, $p = 0.213$, or gender, $F(1, 116) = 0.24$, $p = 0.627$, nor was the interaction significant, $F(1, 116) = 0.06$, $p = 0.803$.

Supplementary Analyses

Thus far, all analyses have collapsed across high intensity and low intensity faces and have found relatively small effects. It may be the case that there is a ceiling effect in identifying high intensity faces. To test for this, the analysis testing for differences to overall errors by lifetime PTSD diagnosis was repeated substituting high intensity and low intensity errors for overall errors. For high intensity errors there was not a significant difference for lifetime PTSD diagnosis, $F(1, 116) = 1.82$, $p = 0.180$. In contrast, for low

intensity errors there was a significant difference by lifetime PTSD diagnosis, $F(1, 116) = 5.33$, $p = 0.023$. This suggests that the effects found above may be due to errors to low intensity faces.

The effect for PTSD diagnosis being associated with more errors to low intensity faces strengthened slightly, and in several of the analyses above, especially hypothesis 6, trauma exposure and abuse appeared to be associated with fewer errors to faces, while PTSD symptoms appeared to be associated with more errors to faces. Thus, regressions were performed to examine the relation of trauma exposure to errors on low intensity faces. First, after controlling for symptoms of depression, total adult trauma exposure on the TEI significantly predicted fewer errors to low intensity faces, $\beta = -0.20$, $t(137) = -2.33$, $p = 0.021$. Additionally, of types of childhood abuse, sexual abuse significantly predicted fewer errors to low intensity faces, $\beta = -0.218$, $t(137) = -2.52$, $p = 0.013$, as did physical abuse, $\beta = -0.17$, $t(137) = -1.98$, $p = 0.049$.

Finally, there were not gender differences revealed by the specific hypotheses. However, when the data were split by gender some interesting findings emerged. First, when ANCOVA's were performed separately for men and women looking for differences in errors to angry faces by lifetime PTSD diagnosis while controlling for trauma exposure on the TEI and CTQ, there was a significant effect for women, $F(1, 77) = 4.87$, $p = 0.030$, but not for men, $F(1, 39) = 0.10$, $p = 0.760$. When the same analyses were run substituting sad faces for angry faces, there appeared to be a trend towards significance for women, $F(1, 77) = 3.73$, $p = 0.057$, but not for men, $F(1, 39) = 5.50$, $p = 0.200$. For fearful faces, the results were not significant for either women, $F(1, 77) = 0.16$, $p = 0.689$, or men, $F(1, 39) = 0.98$, $p = 0.329$.

Discussion

This study is the first to find that a diagnosis of PTSD is assocaited with more errors on a facial emotion recognition task. Additionally, there was partial support for the hypothesis that PTSD symptoms are associated with errors to fearful faces specifically. However, a present or past diagnosis of PTSD was not significantly associated with errors to fearful faces; thus, this finding should be interpreted with caution. Additionally, the secondary hypotheses were not supported. There were no specific effects for a current diagnosis of depression or depressive symptoms, or specific effects for childhood neglect. Since this is the first study to address child maltreatment (albeit retrospectively) and PTSD simultaneously, additional analyses were performed guided by observations from the primary analyses. These secondary analyses revealed that adult trauma exposure, childhood sexual abuse, and childhood physical abuse were all predictive of fewer errors to low intensity faces. This indicates that exposure to traumatic events may make individuals more attuned to the emotional facial expressions of others, while those who develop PTSD have more difficulty discriminating the facial expressions of others. Finally, while gender differences did not emerge from the specific hypotheses, it was found that women with a lifetime diagnosis of PTSD made more errors to angry faces than men with a lifetime diagnosis of PTSD. Additionally, there appeared to be a trend for women with a lifetime diagnosis of PTSD to make more errors to sad faces, while this did not hold for men with a lifetime diagnosis of PTSD. However, there were three times as many women with a lifetime diagnosis of PTSD than men in the sample; thus, this last finding may be due to unequal sample size and should be interpreted with caution.

The overall hypotheses were supported in that individuals who had ever had a diagnosis of PTSD made more errors to identifying facial emotions. Two aspects of the findings were particularly interesting. First, while strong evidence is emerging that the amygdala is hyper-responsive in individuals with PTSD (Armony, Corbo, Clément & Brunet, 2005; Bremner et al., 1999; Rauch et al., 2000; Shin et al., 1994; Shin et al., 2005), and the amygdala is implicated in processing fearful faces (Adolphs, 2006; Amaral, 2003; Dalgleish, 2004; Haxby, Hoffman & Gobbini, 2001; Whalen, 1998), individuals with PTSD did not seem to have a specific deficit in recognizing fearful faces. Only when using a continuous measure of PTSD symptoms and controlling for age was a significant effect found for fearful faces. This finding may be due to chance because there was not a significant effect when using the categorical diagnosis. Instead, individuals with PTSD showed a general pattern of difficulty in recognizing all facial emotions. Second, the effect did not seem to be limited to individuals with a current diagnosis of PTSD; rather, individuals with a lifetime diagnosis of PTSD appeared to make more errors to emotional faces than individuals who had never experienced a criterion A trauma and individuals who had experienced a criterion A trauma but had never developed PTSD.

Much of the argument that individuals with PTSD would show specific deficits to recognizing fearful faces was based on the assumption that the amygdala is involved in the maintenance of PTSD. However, the current study did not measure amygdala activation; thus, there is no way to know if individuals with PTSD showed differential amygdala activation in response to the facial stimuli. While it cannot be shown that the amygdala activated more in response to the stimuli in individuals with a PTSD diagnosis

than in others, the results do not rule out that possibility. While there is evidence that the amygdala is involved in processing negative emotions specifically, its role may not be limited only to emotions with a negative valence. Whalen (1998) argued that once the amygdala becomes activated, it affects both "downstream" autonomic responses, and "upstream" higher order information processing. Thus, once the amygdala becomes activated, it may affect information processing generally rather than to fearful faces specifically. Additionally, at least one study has reported increased amygdala activation to both fearful and happy faces as compared to neutral faces in psychiatrically healthy individuals (Breiter et al., 1996). Specific to PTSD, Armony et al. (2005) measured amygdala activity in response to unmasked happy and fearful faces in individuals with PTSD and found increased amygdala activation in response to both as compared to individuals without PTSD. For these reasons, whether the amygdala activates in response to positive as well as negative emotional faces remains unclear, especially in individuals with PTSD (Phillips et al., 2001; Strauss et al. 2005). If it is the case that the amygdala activates in response to facial stimuli more generally, the results of the current study could still result from hyper-responsivity of the amygdala in individuals with PTSD.

Despite the fact that it is unclear whether the amygdala was involved as a mechanism contributing to the current results, it is clear that these results are consistent with evidence of facial emotion recognition difficulties in other psychiatric disorders. Facial emotion recognition difficulties have been documented in a variety of psychiatric and medical disorders (Kornreich & Philippot, 2006) including Huntington's disease, schizophrenia, depression and anxiety. Most relevant to the current findings are the studies that have examined the facial emotion recognition abilities of individuals with

anxiety disorders. One study of socially anxious individuals reported that they were faster to identify angry and fearful faces (Mullins & Duke, 2004), a result that was not replicated in this study. However, their population included college students with social anxiety, while the population in this study consisted of low-income, predominately minority individuals. The differing results may also be a result of the failure of individuals in this study to respond as quickly as possible, which is indicated by problems with the reaction time data. In contrast, a study of children reported that those with Social Phobia had higher trait anxiety and made more errors to emotional faces in general (Simonian, Beidel, Turner, Berkes & Long, 2001). Another study reported that individuals with Panic Disorder made more errors to emotional faces overall, but that this effect was driven by errors to sad and angry faces (Kessler, Roth, von Wietersheim, Deighton & Traue, 2006). When these previous results are interpreted in conjunction with the current finding that individuals with PTSD made more errors to facial emotions, this suggests that anxiety may be the important factor inhibiting individual's recognition abilities.

Another important finding is that individuals with a lifetime diagnosis of PTSD that did not meet criteria currently were more similar in their facial emotion recognition abilities to individuals with a current diagnosis of PTSD than to individuals who had never had a diagnosis. One possibility is that facial emotion recognition difficulties may not resolve even after the symptoms of PTSD have abated; however, another possibility is that individuals with facial emotion recognition difficulties are more prone to develop PTSD. In considering the former possibility, the converging evidence from neuroimaging studies may point to reasons that individuals with PTSD continue to have

difficulty with facial emotion recognition even after symptoms no longer meet criteria for the disorder. A neural model of fear conditioning and extinction has emerged from the animal literature, and is convergent with current neurobiological models of PTSD (Rauch, Shin & Phelps, 2006). Neural models of fear extinction postulate that it is not the same as "forgetting;" rather, it involves new learning to inhibit the initial fear response. Thus, animals do not unlearn fear, but instead learn not to respond to the initial fear-eliciting stimuli.

Exposure therapy has been empirically shown to be the most effective treatment for PTSD (Foa, Keene & Friedman, 2000). Exposure therapy is very similar to the concept of extinction that was first described by Pavlov, and extinction models fit the neuroimaging evidence in regards to PTSD (Myers & Davis, 2007). One of the important facets of extinction is that it is not the same as forgetting. Instead, fear extinction seems to involve the formation of new memories that counteract the hyperresponsivity to the trauma-related memory. As outlined earlier, current neurological models of PTSD focus on three areas: the amygdala, the medial prefrontal cortex and the hippocampus. Basically, the amygdala-centered fear system activates in response to fearful stimuli, and is down-regulated by both the prefrontal cortex and the hippocampus. However, in individuals with PTSD, the prefrontal cortex and hippocampus do not seem to down-regulate the amygdala, leading to many of the symptoms of PTSD (Shin, Rauch & Pitman, 2006). This model is supported by the fear extinction literature because it has been reported that the medial prefrontal cortex and hippocampus are integrally involved with fear extinction (Milad et al., 2007; Quirk, Garcia & González-Lima, 2006). Not only have activations of the medial prefrontal cortex been implicated in fear extinction,

but the magnitude of activation has been correlated to the strength of the extinction memory (Quirk, Garcia & González-Lima, 2006). Thus, individuals who recover from PTSD have likely learned not to respond to trauma-related stimuli, and this learning is mediated through the medial prefrontal cortex. However, it is important to note that extinction learning is not general, rather it is cue-specific (Myers & Davis, 2007). Thus, while individuals may learn to inhibit amygdala responding to cues that are directly reminiscent of their traumas, they may not learn to inhibit responses to cues not directly related to trauma. As facial expressions of strangers are not directly linked to an individual's trauma, they may activate the amygdala-centered fear system which in turn would not be down-regulated by the new learning of fear extinction. For this reason, it is possible for individuals whose acute PTSD symptoms have resolved to evidence difficulties with facial emotion recognition.

Additionally, individuals with a lifetime diagnosis of PTSD who do not meet current criteria are not necessarily asymptomatic. In fact, in this sample individuals with a lifetime diagnosis of PTSD reported more current symptoms than individuals who had never experienced a criterion A trauma and individuals who had never met criteria for PTSD. Thus, individuals with a lifetime diagnosis of PTSD have recovered to a point that they no longer meet criteria: however, many still have residual symptoms. Difficulty with facial emotion recognition may be a symptom that remains even after acute PTSD symptoms have remitted.

Thus far, the results have been interpreted assuming that the emotion recognition difficulties were associated with PTSD. However, whether the results may be associated with alexithymia must be considered. Alexithymia has been linked to PTSD and

differential activations of the brain in response to facial emotion stimuli. In fact, several researchers have argued that alexithymia may interfere with emotional interpretation and attention (Berthoz et al., 2002; Kano et al., 2003). Given this evidence, it is possible that alexithymia and not PTSD per se is responsible for the results reported in this study. However, two factors seem to be arguing against that interpretation.

First, in the two neuroimaging studies that have specifically looked at emotional faces, researchers have found decreased activations in response to negative faces but *increased* activations in response to positive faces. This suggests that if facial emotion recognition difficulties are due to alexithymia, the difficulties should be limited to negative facial emotions. In contrast, individuals' performance in recognizing positive faces could be expected to be better than those low in alexithymia. The data presented in this paper do not support this interpretation as the findings were strongest for overall errors to faces, and were not limited to the negative faces. The only specific effect for PTSD on angry faces was for women, and the effects of alexithymia would not be expected to be gender specific.

Secondly, the differences in activation in response to angry faces were more pronounced as the intensity of the angry emotion increased (Kano et al., 2003). Thus, if the results were due to alexithymia, individuals with PTSD should make more errors to high intensity faces. In contrast, the results presented here demonstrated that individuals with PTSD made significantly more errors to low intensity faces rather than the opposite. These two factors seem to contradict the argument that the findings here are due to alexithymia, but there is still the need to evaluate the role of alexithymia in PTSD as it relates to the recognition of facial emotions.

Most of the secondary hypotheses of the current study were not supported, and there may be multiple reasons for this. First, there were no significant findings regarding reaction times to faces. In order to obtain reaction time data under the constraints of the Superlab software, the standard administration of the DANVA had to be modified. The standard presentation is to show the stimuli for a maximum of two seconds. However, in order to get reaction time data, the stimuli were presented until the participant responded. While the participants were instructed to respond as quickly as possible, it is unclear whether that was the case. Despite the instructions, the median time for responding to most items was just over two seconds with some respondents taking as long as 30 seconds for a single item. Even when responses over 10 seconds were removed, there were still no significant findings for reaction time. Given that individuals seemed to be taking longer than the standard administration time to respond to the stimuli, it is unsurprising that no significant effects were found for reaction time. In contrast, it may illustrate the robustness of the recognition findings as even when taking their time individuals with PTSD still evidenced difficulties. The results presented here may underestimate the magnitude of difficulties. When interacting with people in the world, they do not tend to have static facial expressions. Thus, individuals do not have unlimited time to evaluate the expressions of others. If given time constraints, individuals with PTSD may show more severe difficulties than reported in this study.

Depression was examined separately due to evidence of facial emotion recognition difficulties in depressed individuals (Asthana, Mandal, Khurana & Haque-Nizamie, 1997; Mikhailova et al., 1996; Persad & Polivy, 1993) and because depression is often comorbid with PTSD. The effects for PTSD remained after controlling for

depression, but no specific decrements to facial emotion recognition were found to be related to symptoms of depression. One possibility is that the current study did not have enough power to detect effects due to depression because only 15 participants met SCID criteria for current depression. Unlike for PTSD, there is some evidence that specific difficulties to facial emotion recognition may resolve when depression remits (Sheline et al., 2001). Another possibility is that depressed individuals in this sample also tended to have comorbid anxiety. Suslow (2004) only found differences between depressed and healthy individuals when those with depression did not have comorbid anxiety. A final possibility is that there are not specific effects due to depression. As reviewed earlier, studies have been somewhat equivocal in their findings with some researchers reporting over-recognition of sad faces (Gur et al., 1992; Mandal and Bhattacharya, 1985) and others reporting under-recognition (Ekman, Friesen, Jones & Malstrom, 1969 as cited in Persad & Polivy, 1993). Thus, it may not be the case that Beck's cognitive theory of depression applies to biases in the recognition of facial emotions.

The hypotheses concerning the association of child maltreatment to errors to emotional faces were not supported. It was hypothesized that a history of physical abuse would predict faster responding to angry faces, and that neglect would predict more overall errors to faces. The first hypothesis may not have been supported due to problems with the data concerning reaction times. Another possibility is that these hypotheses may not have been supported because they were based on data collected from abused children. The current study employed retrospective reports of abuse and tested the facial emotion recognition abilities of adults. However, despite these limitations, associations were found for childhood sexual and physical abuse such that each was

associated with *fewer* errors to faces. Another related unexpected finding was that adult trauma exposure was also related to *fewer* errors to faces. These surprising findings are not completely unexpected, and intuitively make sense from a social learning perspective. Several studies have found an attention bias towards angry faces in physically abused children (Pollak et al., 2000; Pollak & Tolley-Schell, 2003). Additionally, in another study, researchers reported that physically abused children were able to identify angry facial expressions on the basis of less sensory input than control children (Pollak & Sinha, 2002). Children who are abused may become hyper-sensitive to facial emotion cues as they may signal when a parent is likely to abuse them. Regardless, all of the evidence from the child maltreatment literature suggests that the recognition of the facial emotions of others is not a static phenomenon. Abused children seem to learn to recognize the facial emotions of others, especially negative emotions, more quickly and accurately than children who were not abused.

The concept of "compulsive compliance" has been postulated to occur in children with abusive parents (Crittendon & DiLalla, 1988) and may explain the process through which children become more attuned to the facial expressions of others. Basically, this pattern originates during infancy and is related to the infant's changing cognitions about the world. Initially, the infant reacts to the caregiver's abuse by becoming upset and resitant. But, as children begin to understand causal links between their behavior and their caregiver's response, the abused child begins to comply with parental behavior (Jacobsen & Miller, 1998). In order to comply with the caregiver quickly, the child must become constantly vigilant for cues to the caregivers emotional state and desires (Mash & Barkley, 2003). In this light, it is not surprising that individuals with a history of abuse

have become more skilled at identifying the facial emotions of others. Taking into account compulsive compliance, it also makes sense that the findings held for physical and sexual abuse, but not for emotional abuse. Both sexual and physical abuse entail a threat to physical integrity while emotional abuse does not necessarily. While undoubtedly emotional abuse is detrimental to the developing child, it does not present a physical threat in the environment in the same way that physical and sexual abuse present clear dangers. Thus, the child may better be able to watch for cues from the caregiver, including facial expressions, which predict abuse in the case of physical and sexual abuse.

The experience of traumatic events during adulthood was also associated with fewer errors to faces overall. This finding is more difficult to make sense of because there is not a large literature on the effects of trauma on facial emotion recognition in adults. This finding might be explained by the compulsive re-exposure hypothesis that postulates that individuals with PTSD may, in a sense, seek out trauma to temporarily modify their symptoms (van der Kolk, Green, Boyd & Krystal, 1985). In this light, individuals who were abused as children may end up experiencing more trauma as an adult. However, the compulsive re-exposure hypothesis was developed in an attempt to begin understanding the neurobiology of PTSD. Thus, compulsive re-exposure would only apply to individuals with a diagnosis of PTSD. Given that trauma exposure and PTSD appear to operate in opposite directions, it seems unlikely that the finding that adult trauma exposure is related to fewer errors can be explained through compulsive re-exposure. Instead, the most common traumatic events of the individuals in this study involved interpersonal violence. While the majority of studies assessing PTSD have

relied on a veteran population, this study drew from a civilian population. Thus, common traumatic experiences included being robbed, being raped, and experiencing intimate partner violence. For this reason, it seems more likely that the explanation for adult traumatic experiences being associated with fewer errors is similar to the compulsive compliance seen in children. The majority of individuals in this study live in neighborhoods where violence and crime are commonplace. Given this, it seems probable that developing the ability to read other's facial expressions is adaptive.

Finally, there were minor gender differences found such that women with a lifetime diagnosis of PTSD made significantly more errors to low-intensity fearful faces while men with a lifetime diagnosis of PTSD did not make significantly more errors to low intensity fearful faces. This finding is in accordance with the findings of McClure et al. (2004) because she reported that women's frontolimbic structures activated more in response to angry faces. However, she also reported that men's frontolimbic structures activated in response to both angry and fearful faces. However, the study was not specifically examining individuals with PTSD. Given evidence that women are more likely to develop PTSD, there may be two reasons for the lack of findings for men. The first is simply that there were more women in the study than men which means there was more power to detect effects in women. The second is that women may be more likely to have developed PTSD as a result of interpersonal trauma. Thus, angry faces may be particularly arousing for women, and this may lead to specific difficulty with recognizing them.

Implications

Affective social competence, or the ability to interact effectively with others, depends on three components: 1) sending affective information, 2) receiving affective information, and 3) experiencing affect (Halberstadt, Denham & Dunsmore, 2001). It is already established that the symptoms of PTSD involve an interruption of the ability to experience affect through emotional numbing and alexithymia. This study is the first to find evidence that individuals with PTSD have difficulties receiving affective information from emotional faces. Thus, interpersonal difficulties engendered by PTSD may not be limited to experiencing affect, but may extend to receiving affective information as well. It has been postulated that successful affective social competence depends upon the successful integration of the three areas (Halberstadt, Denham & Dunsmore, 2001), which may be interrupted to an extent in individuals with PTSD. Thus, difficulty with recognizing the facial emotions of others may be one of the mechanisms underlying the interruption of social functioning often seen in PTSD. This is particularly important as one of the most important aspects in the treatment of PTSD involves reconnecting with community (Herman, 1997).

The finding that the experience of trauma in and of itself is associated with fewer errors to emotional faces, while PTSD is associated with more errors is particularly important because it may suggest that something about the mechanism of PTSD is interfering with individuals' ability to recognize the facial emotions of others. On the other hand, it may suggest that individuals who have facial emotion recognition difficulties are more likely to develop PTSD. Regardless of the causal relationship, an inability to recognize the emotions of others could lead to re-traumatization through not

recognizing potentially threatening situations. In this respect, individuals with PTSD or individuals with facial emotion recognition difficulties may be a particularly vulnerable population.

While these findings have clinical implications for the treatment of PTSD in general, they are particularly important for the low-income, minority population who comprise this study's population. One reason is that PTSD remains an under-recognized phenomenon in urban, low-income populations. In one study of low-income, urban individuals seeking mental health services, 94% had experienced at least one criterion A stressor on their lifetime, 42% met criteria for PTSD within the last 12 months, and 69% met criteria in their lifetime. Additionally, individuals with PTSD sought more services, and had lower satisfaction with those services (Switzer at al., 1999). More recently, another study of low-income, urban individuals seeking mental health services reported that 83% had experienced a severe trauma, and about 40% met criteria for PTSD. This is contrasted with the fact that only 11% of those diagnosed with PTSD via the SCID-I had a chart-based diagnosis of PTSD (Schwartz et al., 2005). Thus, while PTSD is a major problem in these settings, the need for treatment has not been recognized. The findings presented here provide additional insight into the underlying processes that may be exacerbating social difficulties in this heavily traumatized population. Intimate partner violence is more prevalent in lower socioeconomic strata, which may lead to increased incidence of PTSD. Additionally, low social support has been shown to account for battered women's distress (Thompson et al., 2000). Thus, individuals with PTSD as a result of intimate partner violence may be more likely to have difficulties maintaining social relations as a result of nonverbal decoding difficulties, which in turn may lead to

higher levels of distress. Nonverbal processing difficulties may be one of the important links in exacerbating the symptoms of PTSD, especially in women who have PTSD as a result of interpersonal violence.

Future Directions

This study is the first to identify facial emotion recognition difficulties in individuals with PTSD. Thus, the most obvious need is for replication of the results reported here. Additionally, much of the argument for individuals with PTSD having these difficulties is based on current neurobiological conceptions of PTSD including involvement of the amygdala. However, the current study did not assess neural activations. Thus, future studies should assess the underlying neural activations occurring while individuals view the facial affect stimuli. This knowledge will further the understanding of the etiology and maintenance of PTSD.

Another important question regards causality as this study cannot address whether PTSD leads to facial emotion recognition difficulties or vice versa. While much of the argument for these difficulties in individuals with PTSD came from neurobiological evidence, it may be just as likely that difficulties reading the facial emotions of others lead to the development of PTSD. One way to address this problem would be to follow a large cohort over time while measuring their trauma exposure, PTSD symptoms and facial emotion recognition abilities. Thus, it would be possible to determine whether individuals' facial emotion recognition abilities remain relatively stable over time (and therefore individuals' with more difficulties would likely be more prone to PTSD) or whether facial emotion recognition abilities decline after the development of PTSD.

While the current study found statistically significant differences in emotion recognition ability between those with and without PTSD, it remains unclear if these differences are clinically significant. It will be important to begin to assess the functional impact of these difficulties on interpersonal relationships. This will likely entail longitudinal studies to assess whether and how the ability to recognize other's facial expressions changes over time. If it is a relatively stable difficulty, then it will be important to assess whether this difficulty has a detrimental affect on interpersonal functioning.

Limitations

This study had several limitations that limit its generalizability. First and foremost, all the data were collected concurrently. Thus, all data on trauma exposure and childhood abuse are retrospective and subject to recall biases. It is possible, though unlikely, that individuals with PTSD have significantly altered perceptions of their experiences. It could be the case that individuals with PTSD view their experiences as more traumatic or selectively remember more traumatic experiences than individuals without PTSD. However, given that trauma exposure was associated with facial emotion errors in the opposite direction as PTSD, this particular recall bias seems unlikely. As mentioned previously, the issue of causality is important as well given that all data were collected over a short period of time. Thus, while facial emotion recognition abilities and PTSD have been shown to be associated, there is still the problem of which came first.

A second important limitation is that there were some significant differences between the individuals who completed the first interview, and those who went on to complete the full study (the sample employed for this study). The individuals included in

this sample were more likely to be unemployed and also have a lower household income. They also endorsed significantly more symptoms on the BDI. For these reasons there may be important differences between those included in the study and the general hospital population. In contrast, there were not significant differences in trauma exposure or reported PTSD symptoms. Still, these findings underscore the importance of replicating the reported findings in other populations.

The participants in this study were heavily traumatized and oftentimes it became difficult to assess PTSD as related to a single trauma. There is an emerging literature on the concept of "complex PTSD" that is different from PTSD as currently outlined in the DSM-IV (Herman, 1992; van der Kolk, 2001). Complex stress reactions result from situations of prolonged abuse and powerlessness, which can be found in prisoners of war and abused children. Childhood abuse and chronic trauma exposure have the potential to fundamentally affect the personality development of the individual. For this reason, many of the symptoms of complex trauma reactions do not appear in the DSM-IV. This study employed both the MPSS and the CAPS which are measures that assess the 17 symptoms subsumed under the current rubric of PTSD. The majority of individuals in the study had experienced multiple traumas, and therefore may have complex stress reactions. However, the ability of the measures to capture that complexity was limited. For this reason, some individuals with complex stress may not have been included in the PTSD group, and some individuals in the PTSD group may have better fit in the no PTSD group.

Substance abuse was very prevalent in the population from which this sample was drawn. While individuals were asked about substance use, again the data were self-

report. Thus, the data may not be reliable, and incorporating substance abuse was beyond the scope of this study. However, it is possible that individuals under-reported PTSD symptoms due to substance abuse. Overall, substance abuse can significantly mask the symptoms of PTSD, and this study had to rely on the self-reported symptoms of individuals. Thus, some individuals who might have met criteria for PTSD may not have reported symptoms because of current substance abuse.

Finally, due to the constraints of the software, it was not possible to employ the standard two second presentation time on the DANVA. While participants were instructed to respond as quickly as possible, it is unclear that this occurred. Thus, some participants took up to 30 seconds to respond to a single item. It is likely that this affected the results as individuals had more time to examine the stimuli and would likely make fewer mistakes. Facial emotions are not static in the real world, and it is unlikely that someone would display a facial expression for 30 seconds. For this reason, the current study may underestimate the extent of difficulties that some individuals may face.

Conclusion

This is the first study to document receptive facial emotion difficulties in individuals with PTSD, and it joins a host of studies documenting such difficulties in other psychiatric disorders. The current study is unique in that it examined the association of trauma exposure, PTSD and facial emotion recognition abilities. It appears that exposure to traumatic events makes individuals more able to identify the facial expressions of others, while individuals with PTSD have more difficulty. This is a particularly important finding because it highlights the possibility that something about PTSD itself interferes with individuals' ability to recognize the facial expressions of

others. This paper makes the argument that the amygdala becomes hyper-responsive in individuals with PTSD, and the hippocampus and medial prefrontal cortex fail to down-regulate that activation. It may be this hyper-responsivity of the amygdala that is responsible for the observed difficulties with recognizing facial emotions because the evidence has implicated the amygdala in both emotion and facial processing. The data presented in this study indicate clearly that the facial emotion recognition difficulties are a function of PTSD and not a function of trauma. Thus, amygdala involvement would be a parsimonious interpretation. However, the current study did not assess neural activations. For this reason, it will be particularly important to examine the underlying neural activations of individuals with PTSD in response to overtly presented emotional faces.

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Appendix A—Modified PTSD Symptom Scale

The purpose of this scale is to measure the frequency and severity of symptoms in the past two weeks. Using the scale listed below, please indicate the frequency of symptoms to the left of each item. Then indicate the severity beside each item by circling the letter that fits you best.

FREQUENCY

0 Not at all
1 Once per week or less/ a little bit/ once in a while
2 2 to 4 times per week/ somewhat/ half the time
3 5 or more times per week/ very much/almost always

FREQUENCY

____1. Have you had recurrent or intrusive distressing thoughts or recollections about the event(s)?

____2. Have you been having recurrent bad dreams or nightmares about the event(s)?

____3. Have you had the experience of suddenly reliving the event(s), flashbacks of it, acting or feeling as it were re-occurring?

____4. Have you been intensely EMOTIONALLY upset when reminded of the event(s) (includes anniversary reactions)?

____5. Have you persistently been making efforts to avoid thoughts or feelings associated with the event(s) we've talked about?

____6. Have you persistently been making efforts to avoid activities, situations, or places that remind you of the event(s)?

____7. Are there any important aspects the event(s) that you still cannot recall?.

____8. Have you markedly lost interest in free time activities since the event(s)?

____9. Have you felt detached or cut off from others around you since the event(s)?

____10. Have you felt that your ability to experience emotions is less (e.g., unable to have loving feelings, do you feel numb, can't cry when sad, etc.)?

____11. Have you felt that any future plans or hopes have changed because of the event(s)? (e.g., no career, marriage, children, or long life?

____12. Have you been having persistent difficulty falling or staying asleep?

____13. Have you been continuously irritable or having outburst of anger?

____14. Have you been having persistent difficulty concentrating?

____15. Are you overly alert (e.g., check to see Who is around you, etc) since the event(s)?

____16. Have you been jumpier, more easily startled, Since the event(s)?

____17. Have you been having intense PHYSICAL reactions (e.g., sweaty, heart palpitations) when reminided of the event(s)?

____18. How long have these symptoms bothered you?
Score 0= < 1 month, 1= 1-3 months, 2= 3 months- 1 yr, 3= > 1 yr

Appendix B—Traumatic Events Inventory

(To the patient) These questions are related to traumatic or stressful events that you might have experienced, witnessed, or been confronted with(i.e.- someone told you the event happened).

1. **Have you ever experienced a natural disaster (such as a tornado, hurricane, or flood)?**
   ◄Experienced?_____

   _____

   ■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

   ●Which of these is your worst incident_____

   ● At what age did it first occur?_____

   ●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

   Remember _____ Don't Remember _____

   ●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

   Remember _____ Don't Remember _____

2. **Have you ever experienced a serious accident or injury?**
   ◄Witnessed?_____

   _____

   ■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

   ◄Experienced?_____

   _____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

Remember _____ Don't Remember _____

3. **Have you ever experienced a sudden life-threatening illness?**
◄Experienced?_____

_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

Remember _____ Don't Remember _____

4. **Have you ever been in military combat or in military service in a war zone**

◄Experienced?_____

_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

Remember _____ Don't Remember _____

5. **Have you had a close friend or family member who was murdered?**

◄Witnessed?_____

_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

◄Confronted With?_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

Remember _____ Don't Remember _____

8a . Have you been attacked with a gun, knife, or other weapon by a spouse, romantic partner/boyfriend or girlfriend ?

◄Experienced?_____

_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

Remember _____ Don't Remember _____

8b. Have you been attacked with a gun, knife, or other weapon by someone other than a spouse, romantic partner/boyfriend or girlfriend?

◄Experienced?_____

_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)?_____

Remember _____ Don't Remember _____

●In the worst incident, who was the attacker?_____

8c. Have you witnessed a family member or friend being attacked with a gun, knife, or other weapon

◄Witnessed?_____

____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

●At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

Remember _____ Don't Remember _____

8d. Have you witnessed someone other than a family member or friend being attacked with a gun, knife, or other weapon?

◄Witnessed?_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

Remember _____ Don't Remember _____

●What was your relationship to the person being attacked?_____

9a. Have you been attacked without a weapon by a spouse, romantic partner/boyfriend or girlfriend?

◄Experienced?_____

_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

●At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

Remember _____ Don't Remember _____

9b. Have you been attacked without a weapon by someone other than a spouse, romantic partner/boyfriend or girlfriend

◄Experienced?_____

_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)?_____

Remember _____ Don't Remember _____

●In the worst incident, who was the attacker?_____

9c. Have you witnessed a family member or friend being attacked without a weapon?

◄Witnessed?_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

Remember _____ Don't Remember _____

9d. Have you witnessed someone other than a family member or friend being attacked without a weapon?

◄Witnessed?_____

____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

Remember _____ Don't Remember _____

●What was your relationship to the person being attacked?_____

10. Did you witness violence between your parents or caregivers when you were a child?

◄Witnessed?_____

_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

Remember _____ Don't Remember _____

11. Were you beaten or physically punished in other ways as a child (do not include spanking that interviewee considers appropriate unless it resulted in serious injury or medical attention)

◄Experienced?_____

_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16X-19x □>20x

●Which of these is your worst incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

Remember _____ Don't Remember _____

12a. Between the ages of 0 and 13 did an adult or older teenager have sexually abuse you to have any type sexual contact with you?

◄Experienced?_____

_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

Remember _____ Don't Remember _____

12b. Between the ages of 14 and 17 did an adult or older teenager have sexually abuse you to have any type sexual contact with you?

◄Experienced?_____

_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

Remember _____ Don't Remember _____

13a. After the age of 17 did someone rape you or sexually assault you (by that I mean did someone use physical force or threats of physical force to make you have some unwanted sexual contact with them)?

◄Experienced?_____

_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

●Which of these is your worst incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into account)_____

Remember _____ Don't Remember _____

14. Are there any other experiences that have been traumatic or very stressful for you that we have not covered yet?

◄Witnessed?_____

_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

◄Experienced?_____

_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-19x □>20x

◄Confronted

With?_____

■How many times? □1x □2x-3x □4x-5x □6x-8x □9x-10x □11x-15x □16x-29x □>20x

●Which of these is your worst

incident_____

● At what age did it first occur?_____

●In the worst incident, how much did you feel terrified, horrified, or

helpless?_____

Remember _____ Don't Remember _____

●In the worst incident, how much did you feel that you or someone else might die or be seriously injured or killed? (take threat into

account)_____

Remember _____ Don't Remember _____

15. Would you describe the home that you grew up in as **stable** or **unstable**?

Appendix C—Childhood Trauma Questionnaire

Directions: These questions ask about some of your experiences growing up as a child and a teenager. For each question, circle the number that best describes how you feel. Although some of these questions are of a personal nature, please try to answer as honestly as you can. Your answers will be kept confidential.

WHEN I WAS GROWING UP	Never True	Rarely True	Sometimes True	Often True	Always True
1.I didn't have enough to eat	1	2	3	4	5
2. I knew there was someone there to take care of me and protect me	1	2	3	4	5
3. People in my family called me things like "stupid","lazy", or "ugly".	1	2	3	4	5
4. My parents were usually too drunk or too high to take care of me	1	2	3	4	5
5. There was someone in my family who helped me feel important or special	1	2	3	4	5
6. I had to wear dirty clothes	1	2	3	4	5
7. I felt loved.	1	2	3	4	5
8. I thought that my parents wished I had never been born	1	2	3	4	5
9. I got hit so hard by someone in my family that I had to see a doctor or go to the hospital.	1	2	3	4	5
10. There was nothing I wanted to change about my family	1	2	3	4	5
11. People in my family hit me so hard that it left me with bruises or marks.	1	2	3	4	5
12. I was punished with a belt, a board, a cord (or some other hard object).	1	2	3	4	5
13. People in my family looked out for each other.	1	2	3	4	5
14. People in my family said hurtful or insulting things to me.	1	2	3	4	5
15. I believe that I was physically abused.	1	2	3	4	5
16.I had the perfect childhood	1	2	3	4	5
17. I got hit or beaten so badly that it was noticed by Someone like a teacher, neighbor, or doctor.	1	2	3	4	5
18. Someone in my family hated me.	1	2	3	4	5
19. People in my family felt close to each other	1	2	3	4	5
20. Someone tried to touch me in a sexual way or tried to make me touch them.	1	2	3	4	5
21. Someone threatened to hurt me or tell lies about me unless I did something sexual with them.	1	2	3	4	5
22. I had the best family in the world.	1	2	3	4	5
23. Someone tried to make me do sexual things or watch sexual things.	1	2	3	4	5
24. Someone molested me (took advantage of me sexually).	1	2	3	4	5
25. I believe that I was emotionally abused.	1	2	3	4	5

26. There was someone to take me to the doctor if I needed it.	1	2	3	4	5
27. I believe that I was sexually abused.	1	2	3	4	5
28. My family was a source of strength and support.	1	2	3	4	5

Table 1. Rates of Trauma Exposure by Gender

	Men			Women	
Trauma	Yes	No	%	Yes	No
Natural Disaster	**22**	**27**	**45**	**28**	**72**
Serious Accident or injury	**27**	**10**	**73**	**41**	**42**
Sudden life-threatening illness	14	32	30	29	68
Military Combat	**3**	**42**	**7**	**0**	**97**
Close friend or family member murdered	4	41	9	9	86
Attacked with weapon by spouse or partner	14	32	30	29	66
Attacked with weapon by other	**27**	**19**	**59**	**22**	**75**
Family member or friend attacked with weapon (witnessed)	12	34	26	22	72
Non-family member or friend attacked with weapon (witnessed)	**24**	**22**	**52**	**18**	**77**
Attacked without weapon by spouse or partner	13	31	30	41	55
Attacked by other without weapon	**20**	**24**	**45**	**24**	**70**
Family member or friend attacked without weapon (witnessed)	15	28	35	25	68
Non-family member or friend attacked without weapon (witnessed)	**22**	**22**	**50**	**23**	**72**
Sexual contact with physical force	**0**	**43**	**0**	**17**	**77**
Other experiences	3	24	1	20	56

Note. Data derived from self-reported experiences from the Traumatic Events Inventory (TEI). Bolded numbers indicate a significant difference between men and women by a chi-square test of independence, $p < .05$.

Table 2. Means and Standard Deviations of Continuous Measures

Variable	N	Mean	SD
Age	152	45.15	12.38
Childhood Trauma Questionnaire			
Total Score	144	40.80	17.18
Physical Abuse	144	8.32	4.17
Sexual Abuse	144	7.75	5.18
Emotional Abuse	144	8.71	4.51
Physical Neglect	144	7.04	3.18
Emotional Neglect	144	8.97	4.73
Traumatic Events Inventory	150	5.09	4.09
DANVA			
Total Errors	162	11.44	3.42
Happy Errors	162	1.88	1.04
Sad Errors	162	2.03	1.49
Fearful Errors	162	3.88	1.91
Angry Errors	162	3.65	1.60
High Intensity Errors	162	3.12	2.05
Low Intensity Errors	162	8.33	2.16
Reaction Time Total*	162	86631.16	25878.15
Happy Reaction Time*	162	17515.32	5912.71
Sad Reaction Time*	162	22409.39	7685.24
Fearful Reaction Time*	162	23346.68	7919.97
Angry Reaction Time*	162	23127.51	7157.07
Modified PTSD Symptom Scale	147	12.62	12.27
Beck Depression Inventory	145	14.70	12.45

* Reaction times are in thousandths of a second

Table 3. Number of Individuals in Each Group for Categorical Variables

Diagnosis	Men				Women			
	Current		Lifetime		Current		Lifetime	
	Yes	No	Yes	No	Yes	No	Yes	No
PTSD	5	39	14	30	17	75	47	45
Depression	8	31	14	30	11	79	35	57
CTQ Any abuse			17	30			42	55
CTQ Physical Abuse			8	39			21	76
CTQ Emotional Abuse			8	39			14	83
CTQ Sexual Abuse			6	41			31	66
CTQ Emotional Neglect			4	43			16	81
CTQ Physical Neglect			9	38			14	83

Note. Only individuals with either a CAPS or SCID diagnosis are included. CTQ scores are retrospective lifetime scores.

Table 4. Bivariate Correlations among Measures

Variable	*M* (*SD*)	Variable	*M* (*SD*)
1. Age	45.15 (12.38)	7. DANVA-AA-AF Happy	1.88 (1.04)
2. CTQ	40.89 (17.18)	8. DANVA-AA-AF Sad	2.03 (1.49)
3. TEI	5.09 (4.09)	9. DANVA-AA-AF Angry	3.65 (1.60)
4. MPSS	12.62 (12.27)	10. DANVA-AA-AF Fearful	3.88 (1.91)
5. BDI	14.70 (12.45)	11. DANVA-AA-AF High Intensity	3.12 (2.05)
6. DANVA-AA-AF Total	11.44 (3.42)	12. DANVA-AA-AF Low Intensity	8.33 (2.16)

	1	2	3	4	5	6	7	8	9	10	11	12
1	--	.00	.10	-.14	-.10	.20*	.04	.02	,16	.20*	.26**	.07
2		--	.41**	.32**	.37**	-.12	-.18*	-.03	-.02	-.08	-.03	-.16
3			--	.40**	.27**	-.07	-.13	-.04	-.09	.06	.04	-.14
4				--	.71**	.02	-.18*	.06	-.02	.10	.04	-.01
5					--	.06	-.07	.07	.12	-.01	.05	.05
6						--	.37**	.55**	.62**	.64**	.80**	.82**
7							--	.03	.05	.05	.19*	.40**
8								--	.17*	.04	.41**	.48**
9									--	.11	.54**	.47**
10										--	.56**	.49**
11											--	.32**
12												--

Note. For correlations involving variable 1-5, $n \geq 137$; for variables 6-12, $n \geq 144$. All Pearson correlations are two-tailed, $\alpha = .05$. CTQ = Childhood Trauma Questionnaire. TEI = Traumatic Events Inventory. MPSS = Modified PTSD Symptom Scale. BDI = Beck Depression Inventory. DANVA-AA-AF = Diagnostic Analysis of Nonverbal Accuracy, African American Adult Faces.
*$p \leq .05$. **$p \leq .01$

Figure Captions

Figure 1. African American DANVA Stimuli

Figure 2. Total DANVA Errors by Current PTSD and Combined Child Abuse


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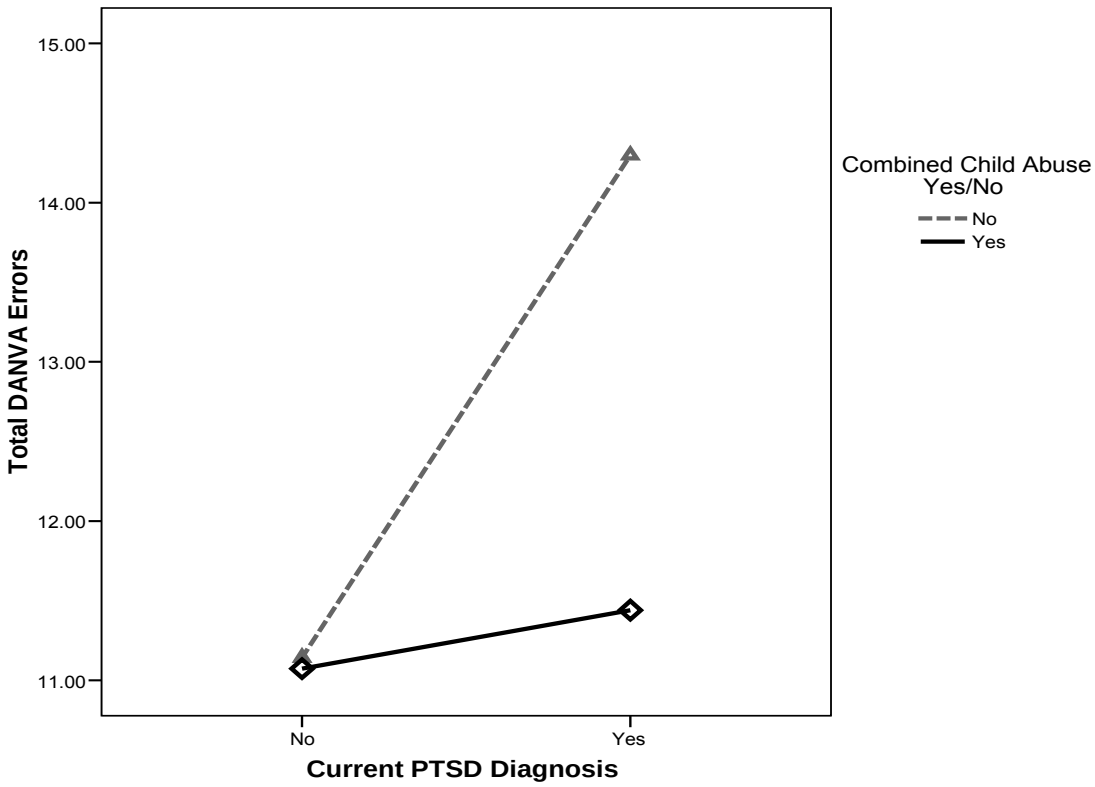
21


15.00
14.00
13.00
12.00
11.00
Total DANVA Errors
No
Yes
Current PTSD Diagnosis
Combined Child Abuse Yes/No
No
Yes