RELATIONSHIPS BETWEEN ANXIETY, HOSTILITY, STARTLE, AND GUILT IN VIETNAM VETERANS SUFFERING FROM PTSD: A PATH ANALYTIC STUDY

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Relationships Between Anxiety, Hostility, Startle, and Guilt in Vietnam Veterans Suffering from PTSD: A Path Analytic Study

BY

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Abstract

Anxiety, hostility, guilt, and an exaggerated startle response are common symptoms experienced by Vietnam veterans with posttraumatic stress disorder (PTSD). In the present study, several theory based path models of possible causal relationships among these symptoms and exposure to trauma (combat) were developed and assessed in two samples of Vietnam veterans with PTSD. A total of 39 Vietnam combat veterans with PTSD and 34 Vietnam combat veterans without PTSD took part in the study. All subjects completed the State-Trait Anxiety Inventory (STAI), the Buss-Durkee Hostility Inventory, and either the Legacies Combat Scale-Revised or the Combat Exposure Scale (CES). Auditory startle data was also available for 15 of the veterans with PTSD and 10 of the veterans without PTSD. Assessment of an initial model indicated that intensity of combat exposure per se is not predictive of PTSD symptomatology. Given that the latest edition of the Diagnostic and Statistical Manual states that an individual's perception of an event as traumatic is equally as important as the objective severity of the trauma, the initial model was modified to include a trauma factor that represented those aspects of combat that accounted for the variability in PTSD diagnosis. The good overall fit indices and significant paths obtained when the modified model was applied to a test sample of veterans replicated when the model was applied to a second data sample. Alternative models of the relationships among the relevant variables, with literature based rationale, were constructed and assessed in the two data samples. These alternative models differed

from the initial model in terms of the relationships predicted between trauma, state anxiety, and trait anxiety. Of the four alternative models tested, two were found to fit the two data samples as well as the hypothesized model. Overall, the results of the study suggest that the increased levels of hostility seen in veterans with PTSD may be due to increases in anxiety that result from exposure to trauma. Increases in hostility then lead to increased guilt. In addition, the models tested supported the idea that the exaggerated startle response observed in many individuals with PTSD is the result of elevated levels of state anxiety. Implications of each of the models for therapy are discussed.

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I want to express my love and thanks to my husband Keith. Thanks for always being there for me and supporting everything I've done in the last couple of years.

Dedication

I would like to dedicate my thesis to the memory of my late mother, Dr. Elizabeth Hesson, who is the inspiration for much of what I strive to achieve in life. I know she would be proud of my accomplishment and I only wish she could be here to share this with me.

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INTRODUCTION

Posttraumatic Stress Disorder: A Historical Perspective

Descriptions of the symptoms of posttraumatic stress disorder (PTSD) appear as early as the works of Homer and Cicero (Tomb, 1994). However, it is only over the course of the last one hundred years that these symptoms have been named and grouped in an effort to understand the nature of the disorder that produces them.

Two important events occurred in the last century that resulted in an explosion of interest in posttraumatic disorders (Trimble, 1985). The first of these was the American Civil War. During the war doctors noted that many soldiers suffered from a state of physical and mental exhaustion. Unable to account for this they relied on the diagnosis of "neurasthenia" (Trimble, 1981). The term "soldier's heart" also arose during this war, because many soldiers complained of heart palpitations and chest pain (Marmar and Horowitz, 1988).

At around the same time, travel by train in Europe increased. There was a corresponding rise in the number of railway accidents. As a result, many lawsuits were brought against the railway companies by individuals claiming chronic pain, anxiety, and invalidism due to trauma from the accident (Trimble, 1985). In 1882, John Eric Erichsen, a London surgeon, provided one of the earliest explanations of PTSD. In his book, Erichsen described symptoms "following (train) accidents which may assume the form of traumatic hysteria, neurasthenia, hypochondriasis, or melancholia" (Keiser, 1968). These symptoms were believed to be due to "molecular disarrangement" or

vascular changes in the spinal cord (Titchener and Ross, 1974). In 1885, Herbert Page, introduced the term "nervous shock" as an alternative explanation of the symptoms seen in people after railway accidents. Unable to find any evidence to support the idea that railway spine was the result of organic disease, Page postulated that the symptoms were psychological (Trimble, 1985).

The idea that trauma-related syndromes were due to an organic pathology re-emerged during World War I. During this conflict many soldiers displayed one or more of the following symptoms: daze, fear, trembling, nightmares, and an inability to function (Marmar and Horowitz, 1988). These symptoms were attributed to a condition known as "shell-shock", which was believed to be the result of head injuries and vascular damage caused by air blasts from high explosives (Marmar and Horowitz, 1988).

However, several observations made near the end of World War I led to the conclusion that the symptoms of "shell-shock" were not necessarily due to physical trauma. They were: (1) the symptoms of "shell-shock" were rare in individuals exposed to explosives (Glass, 1954); (2) severe brain and spinal cord injuries were not accompanied by symptoms similar to those in "shell-shock" (Glass, 1954); (3) the symptoms of "shell-shock" occurred in individuals who had not been exposed to explosive devices (Trimble, 1981); and (4) many patients believed to be suffering from "shell-shock" showed rapid improvement following brief psychological treatment at forward areas (Glass, 1954). Because of these observations the concept of "shell-shock" soon fell by the wayside. In its place emerged the diagnostic category of

"psychoneuroses" which incorporated "war neuroses" and "traumatic neuroses" (Bourne, 1978). This new terminology reflected the view that these disorders, although brought on by combat exposure, were the result of predisposing character or personality defects (Bourne, 1978). This idea was perpetuated by the military, who after dealing with the high cost of mental disorders due to combat in World War I, suggested that psychiatric screening be carried out (Glass, 1966). Thus, the notion of a premorbid personality type began to dominate the literature with the resultant dismissal of the traumatic effects of war.

According to Glass (1966), it became evident early in World War II that psychiatric screening was neither effective nor practical. Despite a pre-induction psychiatric rejection rate that was five to six times higher during World War II, the incidence of psychiatric disorders was two to three times higher than that observed in World War I (Glass, 1966). Apparently, a re-evaluation of combat-related stress symptoms was necessary. Grinker and Spiegel (1945) provided such an evaluation with the results of their study on combat reactions. They reported nineteen common symptoms that persisted long after soldiers were removed from combat. In order of frequency these symptoms were restlessness, irritability and aggressive behavior, fatigue on arising and lethargy, difficulty in falling asleep, subjective anxiety, easy fatigue, startle reaction, feeling of tension, depression, personality changes and memory disturbances, tremor and evidence of sympathetic overactivity, difficulty in concentrating and mental confusion, increased alcoholism, preoccupation with combat experiences, decreased appetite, nightmares and battle

dreams, psychosomatic symptoms, irrational fears (phobias), and suspiciousness (Grinker and Spiegel, 1945).

It was also during World War II that Kardiner provided the first systematic definition of PTSD with his diagnosis of "physioneurosis", a term that emphasized the co-existence of physiological and psychological symptoms (Tomb, 1994). The main features of Kardiner's traumatic syndrome were (1) persistence of a startle response and irritability; (2) proclivity to explosive outbursts of aggression; (3) fixation on the trauma; (4) constriction of the general level of personality functioning; and (5) atypical dream life (Kardiner, 1959).

Seven years after the end of World War II, the original Diagnostic and Statistical Manual (DSM) was published. DSM-I's (1952) "Transient Situational Personality Disorders" included the category of "Gross Stress Reaction" (GSR). GSR was described as a transient reaction in a normal individual to "conditions of great or unusual stress". It was to be distinguished from neurotic or psychotic conditions based on its reversibility, transient nature and the speed with which it cleared upon prompt and adequate treatment. However, it was possible that the condition could progress to one of the neurotic reactions. If the condition persisted, GSR was to be seen as a temporary diagnosis until a more definitive diagnosis could be established. DSM-I (1952) also required the specification of the nature of the stressor as either combat or civilian catastrophe.

DSM-II (1968) minimized reactions to trauma by reclassifying GSR into the category (DSM 307.3) "Adjustment reaction of adult life". This category gave three examples of

the reaction: (1) unwanted pregnancy accompanied by hostility, depression and suicidal gestures; (2) fear associated with military combat and manifested by trembling, running, and hiding; and (3) a Ganser syndrome associated with the death sentence.

It soon became apparent that the descriptions of trauma reactions in DSM-II (1968) were inadequate. Awareness regarding the impact of trauma was on the increase for several reasons: (1) the growing number of psychological casualties from the Vietnam War; (2) the recognition of a host of PTSD-like symptoms in victims of civilian disasters such as the Buffalo Creek dam collapse in 1972; and (3) the publication in 1978 of Horowitz's Stress Response Syndromes, a text that examined the impact of trauma in civilian populations (Tomb, 1994).

DSM-III (1980) was the first diagnostic manual to include the diagnosis of "Posttraumatic Stress Disorder". PTSD was included within the anxiety disorders and described a consistent pattern of symptoms that occurred following exposure to "a stressor that would evoke significant symptoms of distress in almost anyone" (Criterion A). To be diagnosed with PTSD, an individual had to exhibit at least four symptoms from three symptom clusters that included forms of reexperiencing the trauma (Criterion B), numbing of responsiveness or reduced involvement (Criterion C), and heightened arousal and avoidance of reminders of the trauma (Criterion D). According to Wilson (1994), the creation of PTSD as a separate diagnostic category was historically important for several reasons. It stimulated more research; promoted clarification regarding the nature of comorbid disorders; and helped clinicians avoid misdiagnosis and mistreatment.

The recognition of PTSD as an official mental disorder also led to its use in the courts (Wilson, 1994).

DSM-III-R was published in 1987 and included a revision of the diagnostic criteria for PTSD. This revision was the result of knowledge gained from research and clinical work with victims of trauma (Wilson, 1994). DSM-III-R increased the total number of diagnostic symptoms to 17 with the requirement that an individual must exhibit six symptoms from the three major clusters presented in DSM-III (1980). As well DSM-III-R (1987) required that the symptoms had been present for at least one month from the time of the trauma or had begun at least six months after the trauma (delayed onset). DSM-III-R (1987) also attempted to clarify the diagnostic criteria. With regards to Criterion "A", stressors associated with the onset of PTSD were now defined as "external events outside the usual range of daily hassles that would be markedly distressing to almost everyone." DSM-III-R (1987) also redefined Criterion "B" by specifying that the traumatic event be persistently re-experienced and that the visual imagery and emotional distress associated with the trauma be intrusive, unbidden, involuntary, and unexpected. In addition, DSM-III-R (1987) included new ways in which the event could be re-experienced and in which people could avoid its impact or numb or diminish painful emotions associated with memories of it (Criterion "C"). Finally, in response to advances in the psychobiology of PTSD, DSM-III-R (1987) reorganized the "D" diagnostic category (Wilson, 1994). Survivor guilt, memory impairment, and hyperalertness were deleted and replaced with irritability or outbursts of anger, hypervigilance, and physiologic reactivity upon

exposure to stimuli that activated memories of the traumatic event.

Thus, at the end of the 1980's, PTSD was defined as a response that occurred when an individual was exposed to a severe stressor outside the range of usual human experience. This response generated a number of consistent symptoms that were clustered into three categories: (1) intrusive or re-experiencing symptoms; (2) avoidance responses to evidence of the trauma or generalized psychological numbing and isolation; and (3) widespread psychological arousal not previously present.

Posttraumatic Stress Disorder: Current Diagnostic Criteria

The fourth edition of the diagnostic and statistical manual was published in 1994 and includes some revisions to the PTSD diagnostic criteria. Of particular importance is the redefinition of the stressor, criterion "A". In DSM-IV (1994), emphasis is shifted from the severity of the stressor to a mixture of exposure to a traumatic event combined with the patient's reaction to it. Tomb (1994) states that this change reflects the predominant idea that the individual's perception of the trauma is almost as important in determining the stressor's impact and the production of symptoms as is the objective severity of the stressor itself.

DSM-IV (1994) maintains the three clusters of symptoms in the DSM-III-R (1987). In addition, DSM-IV (1994) also requires that the duration of the disorder be specified. If the symptoms have been present for less than three months, the individual is classified as having acute PTSD. If symptoms have lasted three or more months the disorder is considered chronic. Finally, if at least six months have passed between the traumatic

event and the onset of symptoms the individual is described as having PTSD with delayed onset.

The Epidemiology of Posttraumatic Stress Disorder

Taking into consideration that prevalence rates are affected by the methods used to attain them as well as the population sampled, DSM-IV (1994) states that lifetime prevalence of PTSD in the community ranges from 1% to 14%. At-risk individuals, such as combat veterans, victims of natural disasters or crime, show prevalence rates ranging from 3% to 58%.

According to Tomb (1994), one reason for the difficulties in defining the epidemiology of PTSD is that other psychopathologies can alter the form and incidence of the disorder. It is well established that there is a high rate of psychiatric comorbidity among patients with PTSD (Blank, 1994; Davidson and Fairbank, 1993; Keane and Wolfe, 1990). Possible comorbid illnesses include generalized anxiety disorder (GAD), depression, dysthymia, obsessive-compulsive disorder, panic disorder, substance abuse, phobias, and mania (Davidson and Fairbank, 1993). However, which disorders are primary and which are secondary is still unclear. Depressive, antisocial, or substance abuse factors may place people at risk when exposed to an extreme stressor (Keane and Wolfe, 1990). Alternatively depression, substance abuse, or antisocial characteristics could develop as a function of PTSD symptomatology, and the individual's attempts to cope with distress (Keane and Wolfe, 1990).

Clinical Findings and PTSD

Recent research findings suggest that patients with PTSD have marked abnormalities in; (1) sympathetic and/or autonomic nervous system arousal; (2) hypothalamic-pituitary-adrenal axis function and; (3) the endogenous opioid system. The present section will be a brief review of the relevant psychophysiological and neuroendocrine findings in patients with PTSD.

Currently, psychophysiological assessment provides the most specific biological diagnostic test for PTSD (Friedman, 1991). It is also considered a major source of support for the validity of the diagnosis of PTSD (Pitman, Orr, Forgue, Altman, de Jong, and Herz, 1990). Psychophysiologic investigations of PTSD have been carried out since World War I. Meakins and Wilson (1918) exposed veterans with "shell-shock" to gunfire and sulfuric flame and found that, in comparison to healthy subjects, they exhibited greater increases in heart rate and respiratory rate. Similar groups of subjects also exhibited severe anxiety, heart rate, and blood pressure increases when given intravenous epinephrine (Peabody, Clough, Sturgis, Wearn, and Tompkins, 1918).

Since the 1980's, a number of psychophysiologic studies have been conducted that have further documented heightened autonomic or sympathetic nervous system arousal in individuals with PTSD. Based on the stimulus used to elicit physiological responses, these studies fall into one of three experimental paradigms; (1) responses to external stimuli reminiscent of the trauma; (2) responses to mental imagery of the trauma; and (3) responses to intense but neutral stimulations, such as auditory startle stimuli (Shalev and

Rogel-Fuchs, 1993).

Several studies have shown that combat veterans with PTSD exhibit significantly larger increases in heart rate when exposed to visual and auditory combat-related stimuli compared with combat veterans without PTSD (Malloy, Fairbank, and Keane, 1983), combat veterans with psychiatric disorders other than PTSD (Pallmeyer, Blanchard, and Kolb, 1986) or healthy controls (Blanchard, Kolb, Pallmeyer, and Gerardi, 1982). Blanchard, Kolb, Prins, Gates, and McCoy (1991) compared heart rate and blood pressure responses to combat sounds in Vietnam veterans with PTSD and combat controls. The difference in these responses correctly classified 80% of the subjects into those with or without PTSD. The discriminant function derived from this group of subjects was then applied to a second group of veterans and yielded 83% correct discrimination.

Studies of responses to mental imagery differ from those that look at response to standard stimuli in that they use subjects' own recollection of the trauma as the eliciting stimulus. Thus, these studies look at physiological response to reminiscences (Shalev and Rogel-Fuchs, 1993).

In one such study, Pitman, Orr, Forgue, de Jong, and Claiborn (1987) asked Vietnam veterans with PTSD and combat controls to listen to recorded scripts describing traumatic events. Some of the scripts were standardized while others were based on the subjects' own individual experiences. Imagery of the subjects' personal incidents provoked extreme heart rate, electromyogram, and skin conductance responses in PTSD

subjects, but not in combat controls. A discriminant function analysis, based on the size of these responses, distinguished PTSD veterans from non-PTSD veterans with a specificity of 100% and a sensitivity of 61%.

In a second study Pitman et al. (1990) compared the physiological responses of Vietnam combat veterans with PTSD and Vietnam combat veterans with non-PTSD anxiety disorders to tapes of individual combat scripts. Subjects with PTSD exhibited higher skin conductance and electromyogram responses to the individualized scripts when compared to the anxious subjects. The discriminant function derived from Pitman et al.'s (1987) study was applied to the physiological responses of these subjects. It distinguished PTSD subjects from non-PTSD anxious subjects with a sensitivity of 71% and a specificity of 100%.

Orr and Pitman (1993) have also used personalized combat experience scripts to look at the ability of non-PTSD veterans to simulate the physiologic responses of PTSD. They found that skin conductance and electromyogram responses best discriminated PTSD veterans from non-PTSD veterans and that most non-PTSD veterans were unable to simulate the physiologic response patterns of the PTSD veterans.

One of the diagnostic criteria of PTSD is an exaggerated startle response (DSM-IV, 1994). Like the symptom of physiological arousal, it is unique in the diagnosis of PTSD in that it can be confirmed without using self-report measures (Butler, Braff, Rausch, Jenkins, Sprock, and Geyer, 1990). In addition, studies using the acoustic startle response paradigm use elementary stimuli that are not associated with the traumatic event

and do not require deliberate mental activity by the subject (Shalev and Rogel-Fuchs, 1993). Despite its apparent value as a diagnostic tool, to date only a few studies have been conducted that look at the validity of the startle response in PTSD.

Butler et al. (1990) tested Vietnam veterans with PTSD and non-PTSD veterans for acoustic and tactile startle response using eyeblink electromyogram amplitudes as their dependent variable. Veterans with PTSD exhibited higher eyeblink electromyogram amplitudes than non-PTSD veterans. No significant differences were found between the two groups in tactile startle response magnitude. This may be due to stimulus specific, in this case auditory or tactile, startle reactions in individuals with combat-related PTSD (Butler et al., 1990).

Paige, Reid, Allen, and Newton (1990) measured event-related component amplitudes and heart rate to four intensities of randomly presented tones in Vietnam veterans with PTSD and combat veterans without PTSD. Measuring event related brain potentials (ERPs) provides a means of examining central nervous system responses that are sensitive to the processing of sensory input (Paige et al., 1990). Individuals can be classified as augmenters or reducers based on their ERP component amplitudes as a function of stimulus intensity (Paige et al., 1990). Augmenting is associated with a cortex tuned to seek increases in stimulus intensity. Reducing is associated with a protectively tuned sensory system that attempts to shut out increased stimulation. Paige et al. (1990) hypothesized that individuals with PTSD, when faced with intense stimuli, enter a state of protective inhibition and thus would have ERP gradients that correspond to those of

reducers. The results of the study suggested that the veterans with PTSD were more autonomically arousable than the control subjects and that they were more likely to be ERP reducers.

Shalev, Orr, Peri, Schreiber, and Pitman (1992) looked at heart rate, electromyogram responses and skin conductance responses in noncombat PTSD patients, anxiety disorder patients, mentally healthy patients with traumatic experiences, and mentally healthy subjects without traumatic experiences. They found that PTSD patients had larger heart rate and skin conductance responses and also did not show habituation of the skin conductance component of the acoustic startle response.

Orr, Lasko, Shalev, and Pitman (1995) compared the startle responses of Vietnam combat veterans with and without PTSD. They found that the veterans with PTSD exhibited larger heart rate and eyeblink responses and that skin conductance response magnitude declined more slowly across trials in veterans with PTSD compared to non-PTSD controls. However, the number of trials it took for PTSD veterans and non-PTSD veterans to reach the skin conductance nonresponse criterion did not differ. Thus, veterans with PTSD were able to habituate the skin conductance component of the startle response but at a slower rate than the nonPTSD subjects.

Given the sympathetic psychophysiological hyper-reactivity of individuals with PTSD, one would predict an associated elevation in catecholamine levels (Friedman, 1991). Kosten, Mason, Giller, Ostroff, and Harkness (1987) have found elevated urinary epinephrine and norepinephrine levels in hospitalized patients with PTSD in comparison

to inpatients with diagnoses of major depressive disorder, bipolar mania, paranoid schizophrenia, and undifferentiated schizophrenia. Yehuda, Southwick, Giller, Xiaowan, and Mason (1992) also report elevated urinary epinephrine, norepinephrine and dopamine levels in inpatient and outpatient Vietnam combat veterans with PTSD in comparison to inpatient and outpatient normal control subjects. In contrast to the findings of these two studies, Pitman and Orr (1990) report no difference in urine levels of norepinephrine and epinephrine in combat veterans with PTSD when compared to healthy nonpsychiatric combat veteran control subjects.

Yehuda, Giller, Southwick, Lowy, and Mason, (1991) suggest that methodological differences in urine sampling and the use of combat veterans for controls may account for the inconsistencies in these studies. However, the difference in these findings indicate that attention should be paid to the nature of the control group being used for comparison when interpreting the results of neuroendocrine studies of PTSD. Yehuda et al. (1991) suggest that studies looking at individuals with PTSD should include combat controls and normal controls.

Consistent with the observation of increased peripheral catecholamine levels in individuals with PTSD, Perry, Giller, and Southwick (1987) report a 40% reduction in in vitro total alpha2-adrenergic receptor binding sites in the platelets of inpatient Vietnam veterans with PTSD compared to normal control subjects without PTSD. Since the late 1970's, the platelet alpha2-adrenergic receptor has been used as a marker for the central alpha2-receptor (Perry, Southwick, Yehuda, and Giller, 1990). PTSD subjects also

exhibit an increased low to high affinity binding site ratio in comparison to controls (Perry et al., 1987). These findings suggest that the platelet alpha2-adrenergic receptor sites in PTSD patients show both down-regulation and desensitization (Perry et al., 1990). This alteration of alpha2-adrenergic receptor sites is further supported by the finding of decreased lymphocyte adenylate cyclase activity in inpatients with PTSD as a result of combat, terrorist activity and automobile accidents compared to age and sex matched nonpsychiatric controls (Lerer, Ebstein, Shestasky, Shemesh, and Greenberg, 1987). In addition, the alpha2-agonist clonidine has been shown to reduce anxiety and autonomic arousal in Cambodian refugees with PTSD (Kinzie, 1989).

Biochemical challenge studies have shown that agents such as lactate (Rainey, Aleem, Ortiz, Yeragani, Pohl, and Berchou, 1987) and yohimbine (Southwick, Krystal, Morgan, Johnson, Nagy, Nicolaou, Heninger, and Charney, 1993) elicit panic attacks and Vietnam-related flashbacks in veterans with PTSD. The anxiogenic properties of yohimbine are mediated through its ability to increase presynaptic activity by antagonizing the alpha2-adrenergic autoreceptor (Southwick, Bremner, Krystal, and Charney, 1994). The observed effects of yohimbine are consistent with the increased peripheral catecholamine excretion and down-regulation of platelet alpha2-adrenergic receptors observed by Perry et al. (1987). Precisely how lactate has its effects is unknown although central noradrenergic dysregulation has been suggested (Southwick et al., 1994).

It thus appears that elevated catecholamine levels may be biochemical markers for

the sympathetic dysregulation associated with PTSD. However, they may also reflect another abnormality, reduced monoamine oxidase (MAO) activity in individuals with PTSD (Friedman, 1991). Since MAO is a major degradative enzyme in catecholamine metabolism, reduced MAO could lead to higher systemic NE and EPI levels.

Davidson, Lipper, Kilts, Mahorney, and Hammett (1985) have reported lower platelet MAO activity in veterans with PTSD compared to age-matched normal control subjects. However, it should be noted that when the PTSD group in this study was divided into individuals with and without a history of alcohol abuse, only the former differed significantly from the control subjects (Davidson et al., 1985).

The hypothalamic-pituitary-adrenal (HPA) axis has also been investigated in PTSD because of the important role it plays in the stress response (Sutherland and Davidson, 1994). As the primary function of HPA axis activation is to rapidly produce glucocorticoids from the adrenals (Yehuda, Boisoneau, Mason, and Giller, 1993), a number of studies have looked at cortisol levels in individuals with PTSD. Mason, Giller, Kosten, Ostroff, and Podd (1986) report significantly lower mean 24 hour urinary cortisol excretion in inpatients with PTSD when compared to inpatient control subjects with diagnoses of major depressive disorder, bipolar mania, paranoid schizophrenia, and undifferentiated schizophrenia. Yehuda, Teicher, Levengood, Trestman, and Siever (1994) also report lower mean 24 hour urinary cortisol excretion in patients with PTSD in comparison to patients with major depression and normal nonpsychiatric controls. However, Pitman and Orr (1990) report finding increased 24 hour urinary cortisol

excretion in PTSD outpatients in comparison to combat veteran control subjects. Once again these differences could be accounted for by the method of urine sampling and the use of combat veterans as controls instead of normal volunteers (Yehuda et al., 1991).

Alternatively, Yehuda et al. (1990) suggest that these results may indicate that the HPA axis is dynamic in PTSD and that cortisol excretion is related to the state characteristic of severity of the illness (Yehuda et al., 1990). According to Yehuda, Resnick, Kahana and Giller (1993), whether cortisol excretion for a PTSD patient is higher or lower depends on the nature of the environmental stressors and particular symptoms being experienced at the time of sampling.

Several other findings support the idea of a dynamic HPA axis in PTSD. It has been reported that combat veterans with PTSD have an increased number of lymphocyte glucocorticoid receptors in comparison to normal control subjects (Yehuda, Lowy, Southwick, Shaffer, & Giller, 1991) and patients with major depressive disorder, bipolar mania, panic disorder, and schizophrenia (Yehuda et al., 1993). In addition a strong positive correlation has been found between lymphocyte glucocorticoid receptor number and PTSD symptoms (Yehuda et al., 1991). According to Yehuda et al. (1993), if a large number of lymphocyte glucocorticoid receptors reflects a large number of neuronal glucocorticoid receptors, then it is possible that large numbers of glucocorticoid receptors in PTSD may help to modulate transient increases in cortisol and allow for a quicker recovery from stress in individuals with PTSD.

In addition, it has been shown that individuals with PTSD show an enhanced

suppression of cortisol following administration of the steroid dexamethasone in comparison to normal age-comparable controls (Yehuda, Southwick, Krystal, Bremner, Charney, and Mason, 1993). This finding combined with those discussed above suggest the existence of an enhanced negative feedback sensitivity of the HPA axis in PTSD (Yehuda et al., 1993).

As stress-induced analgesia has been observed in animals after exposure to a variety of inescapable stressors (van der Kolk and Fisler, 1993) researchers have also investigated the endogenous opioid system in PTSD.

Pitman et al. (1990), exposed a group of Vietnam veterans with PTSD and a group of Vietnam veterans without PTSD to combat scenes from the movie Platoon. Subjects in each group received either the narcotic antagonist naloxone or a placebo. In the placebo condition, subjects with PTSD showed a 30% decrease in reported pain intensity ratings after the combat video. Subjects with PTSD in the naloxone condition and non-PTSD subjects in either condition exhibited no decrease in pain ratings. Out of a series of hormonal, autonomic and emotional variables measured in the study, pain intensity rating best differentiated the PTSD and non-PTSD subjects. Pitman et al. (1990) suggest that dysregulation of the endogenous opioid system may contribute to the avoidance and numbing symptoms of PTSD.

Models of PTSD

Yehuda and Antelman (1993) state that animal models of human disorders are valuable for several reasons. First, they allow the opportunity to simulate a human

condition in a controlled setting, with a large sample size, and in a simpler and more easily understood system. Second, animal models, unlike human disorders which can only be studied when they become clinically noticeable, can be observed as they develop. This allows the study of symptoms as they develop. Finally, pharmacological and other treatments that might be difficult to test in humans can be evaluated in animals. Because PTSD is a disorder in which the main precipitating causes are known, Yehuda and Antelman (1993) suggest that the potential exists to accurately model the disorder in animals. Identification of which variables produce which symptoms in animals may serve to generate hypotheses about the development of PTSD in humans (Foa et al., 1992). To date, several animal models of PTSD have been developed. The following section is a brief review.

The Classical Conditioning Model of PTSD

Classical conditioning has been suggested as a mechanism that links the symptoms of PTSD to the original trauma (Kolb and Multipassi, 1982). According to Southwick et al. (1994), the feelings of fear and extreme anxiety an individual experiences when exposed to a life-threatening trauma can become conditioned to a number of stimuli present at the time of the trauma. Subsequently, these previously neutral stimuli are able to evoke fear and anxiety in the individual, a phenomenon that Kolb (1987) refers to as "conditioned emotional response". Both specific and nonspecific cues associated with the trauma can become conditioned stimuli (Southwick et al., 1994). Stimulus generalization and higher order conditioning can also occur with the result that the individual

experiences fear and anxiety in response to a wide variety of stimuli (Southwick et al., 1994). The role of conditioning in PTSD is supported by the psychophysiological findings discussed above which suggest that individuals with PTSD experience increased arousal to trauma-related cues in comparison to individuals without PTSD. As well, Pitman et al.'s (1990) finding of stress-induced analgesia in Vietnam veterans with PTSD exposed to trauma-related cues is accommodated within a conditioning model.

However, it has been argued that the conditioning model of PTSD does not readily account for the finding of an exaggerated startle response in many patients with PTSD (Krystal, 1990; Pitman, Orr, and Shalev, 1993; Shalev, 1993).

The Fear-Potentiated Startle Model of PTSD

The basic startle response is viewed as an unconditioned form of phasic reactivity (Orr et al., 1995). Based on the findings of their lesioning experiments, Davis, Gendelman, Tischler and Gendelman (1982) initially proposed a neural circuit for the acoustic startle response in the rat that consisted of the auditory nerve, the posteroventral cochlear nucleus, an area just medial to the ventral nucleus of the lateral lemniscus, a ventromedial region of the nucleus reticularis pontis caudalis, and spinal motor neurons. However, Lee, Lopez, Meloni, and Davis (1996) have recently suggested that the large lesion sizes and the relatively nonselective nature of the electrolytic lesions employed in the Davis et al. (1982) study did not allow the researchers to delineate the startle pathway in detail. Using fiber-sparing lesions, Lee et al. (1996), have recently proposed a more simplified acoustic startle pathway that consists of three synapses. In this circuit, cochlear

root neurons embedded in the auditory nerve synapse onto neurons in the ventrolateral part of the nucleus reticularis pontis caudalis which then synapse on spinal motor neurons.

Several authors have suggested that the animal model of fear-potentiated startle may be particularly useful for studying exaggerated startle in PTSD (Kolb, 1987; Krystal, 1990: Orr et al., 1995: Southwick et al., 1994). However, it should be noted that it is currently unclear whether the exaggerated startle seen in PTSD is associated with increased baseline startle, fear-potentiated startle, or both (Krystal, 1990).

Brown, Kalish, and Farber (1951) were the first to demonstrate that the amplitude of the rodent acoustic startle response can be increased by presenting the auditory stimulus in the presence of a cue, such as a light, that has previously been paired with a shock. The findings that startle potentiation is blocked by anxiolytic drugs, enhanced by anxiogenic drugs, and fails to occur in a nonassociative control condition suggests that fear-potentiated startle is produced by the associative conditioning of a central fear state (Cook, Hawk, Davis, and Stevenson, 1991).

Fear alters startle at a specific point on its neural pathway (Davis, 1992). Berg and Davis (1985) have shown that startle elicited from either the ventral cochlear nucleus or the ventral lateral lemniscus is potentiated by a conditioned fear stimulus. Startle elicited in the nucleus reticularis pontis caudalis or points beyond is not potentiated (Berg and Davis, 1985).

Because the central nucleus of the amygdala has been shown to have direct

projections to brain areas that may be involved in many of the symptoms of fear and anxiety (Davis, 1992) researchers have looked at the role of the amygdala in fear-potentiated startle. Low-level electrical stimulation of the amygdala has been shown to increase the amplitude of the startle response (Davis, 1992). In addition, electrolytic or ibotenic acid lesions of the central nucleus of the amygdala following fear conditioning completely eliminate fear-potentiated startle to both auditory and visual conditioned stimuli (Campeau and Davis, 1995). Campeau and Davis (1995) state that this finding is consistent with the idea that the central nucleus of the amygdala functions as a response independent, final common relay for fear conditioning. Campeau and Davis (1995) also report that electrolytic or NMDA-induced lesions of the basolateral complex of the amygdala disrupt fear-potentiated startle to conditioned stimuli of both modalities. This finding is in keeping with the notion that, in fear conditioning, the basolateral complex of the amygdala serves as a necessary relay of sensory information from subcortical and cortical sensory areas to the central nucleus of the amygdala (Campeau and Davis, 1995).

It also appears that the central gray may be a component of the neural circuitry involved in the fear enhancement of startle. Fendt, Koch, and Schnitzler (1994) have found a projection in the rat brain from the central nucleus of the amygdala to the central gray. In addition Fendt et al. (1994) have identified a projection from the dorsomedial and lateral part of the central gray to the nucleus reticularis pontis caudalis, shown by Davis et al. (1982) to be an important component of the basic startle response.

Fendt et al. (1994) report that, in rats, lesions of the dorsal and lateral parts of the central gray totally block sensitization of the acoustic startle response without affecting the amplitude of the response in the absence of the sensitizing stimuli. In their study Fendt et al. (1994) used footshock as the sensitizing stimulus. Footshocks, like conditioned fear, have been shown to increase the amplitude of the acoustic startle response in rats (Davis, 1989). These findings are also consistent with Deakin and Graeff's (1991) dual theory of anxiety involving the amygdala and the dorsal central gray. According to Deakin and Graeff (1991), the amygdala is responsible for conditioned fear and anticipatory anxiety while the dorsal central gray organizes the response to aversive unconditioned stimuli. In humans, Deakin and Graeff suggest, dysfunctional activation of the amygdala results in generalized anxiety while dysfunctional activation of the dorsal central gray leads to panic.

Researchers have looked at the startle response in healthy non-psychiatric human subjects. Vrana, Spence, and Lang (1988) have found that the acoustic startle response is enhanced when subjects view slides depicting unpleasant scenes and objects. This effect is independent of measures of orienting, arousal, and interest in the subject matter of the slides. Cook et al. (1991) have examined the generalizability of startle potentiation across a number of emotional states as well as its sensitivity to individual differences in fearfulness. High and low fear subjects were distinguished on the basis of their scores on the revised version of the Fear Survey Schedule. Cook et al. (1991) report that startle responses were larger in all aversive negative states than during pleasant imagery and that

this effect was enhanced among high fear subject. The results of these two studies suggest that potentiated startle in humans is associated with experimentally manipulated fear and negative affect as it is in animals.

Grillon, Ameli, Foot, and Davis (1993) have examined the effects of individual differences in state and trait anxiety, as measured by the State-Trait Anxiety Inventory (Spielberger, 1983), on baseline and fear-potentiated startle. State anxiety was induced by the threat of electric shock. The results showed that magnitude of fear-potentiated startle was larger in the high-anxiety group as compared to the low-anxiety group. Baseline startle did not differ between the low and high anxiety subjects. Trait anxiety did not relate to individual differences in either baseline or fear-potentiated startle. This is consistent with the finding that the anxiogenic alpha2-receptor antagonist, yohimbine increases the magnitude of the acoustic startle response in young healthy men (Morgan, Southwick, Grillon, Davis, Krystal, and Charney, 1993) and combat veterans with PTSD (Morgan, Grillon, Southwick, Nagy, Davis, Krystal, and Charney, 1995). In the latter study, yohimbine significantly increased the amplitude and magnitude of the startle response in the veterans with PTSD but not in combat veteran control subjects without PTSD. Because the startle responses of the PTSD subjects more closely resembled those of the younger healthy control subjects used in the Morgan et al. (1993) study, Morgan et al. (1995) suggest that the lack of a significant yohimbine effect on the startle response of the combat controls may be the result of a reduction in the modulatory mechanisms of the startle reflex caused by aging.

Recently, Morgan, Grillon, Southwick, Davis and Charney (1995) examined the effects of threat of shock on startle responses in Vietnam combat veterans with PTSD and age-matched, healthy controls. Fear of shock was assessed with the state portion of the State Trait Anxiety Inventory. State anxiety scores were higher at baseline and at the time of shock anticipation in subjects with PTSD. While subjects with PTSD exhibited significantly larger startle responses during baseline and during shock anticipation, the rate of habituation of startle response did not differ between PTSD subjects and controls. Morgan et al. (1995) state that their findings suggest that the higher levels of startle seen in PTSD patients may be due to their exhibiting a greater conditioned emotional reponse to threatening stimuli.

Orr et al. (1995) suggest that if the exaggerated startle response seen in PTSD is the result of anxiety or fear then reducing the anxiety associated with PTSD through psychotherapy or medication should decrease the magnitude of the startle response in this disorder. It is worth noting that substances commonly abused by individuals with PTSD include anxiolytic substances such as the benzodiazepines, ethanol and the barbiturates, all of which reduce fear-enhanced startle-response amplitude in experimental animals (Howard and Ford, 1992). This also suggests that some individuals with PTSD exhibit potentiated startle reponses as a result of their being in withdrawal at the time of testing.

In their study of Vietnam veteran twins True, Rice, Eisen, Heath, Goldberg, Lyons, and Nowak (1993) found that 32% of the variance in self-reported startle was accounted for by genetics. Thus, the issue of predisposition also has relevance for the startle issue

in PTSD. Orr et al. (1995) suggest that the constitutional versus acquired origin of abnormal responsivity to loud tones in PTSD could be looked at in a study that acquired data from subjects prior to their experiencing a traumatic event.

The Inescapable Shock Model Of PTSD

Van der Kolk, Greenberg, Boyd, and Krystal (1985) propose that the animal model of inescapable shock (IS) may be directly applicable to PTSD. IS occurs when animals are subjected to stressful events, like electric shocks, from which they are unable to escape. Animals who have experienced IS later exhibit (1) decreased initiation of behavior, (b) cognitive deficits and (c) symptoms of emotional disruption (Rosen and Fields, 1988).

According to van der Kolk et al. (1985) IS is a valid model of PTSD because the behavioral and biochemical changes that occur with IS parallel the development of PTSD. They provide several lines of evidence to support their theory. First, exposure to IS increases NE turnover, increases plasma catecholamine levels, depletes central NE and increases MHPG production (van der Kolk et al., 1985). That these neurotransmitter systems are involved in the behavioral effects of exposure to IS is supported by the finding that drugs that deplete brain catecholamines produce similar alterations in behavior (Krystal, 1990). Van der Kolk et al. (1985) state that the catecholamine-mediated behavioral alterations seen with IS in animals parallel the negative symptoms of PTSD in humans. They state that the symptoms of global constriction, social isolation, diminished motivation, and decline in occupational function are correlates of NE

depletion. Van der Kolk et al. (1985) also suggest that the positive symptoms of PTSD (exaggerated startle response, explosive outbursts, nightmares, and intrusive recollections) are the result of chronic adrenergic hypersensitivity following transient catecholamine depletion from acute trauma. Petty, Chae, Kramer, Jordan, and Wilson (1994) have recently shown that rats who have developed learned helplessness as a result of being exposed to inescapable tail shock show a significantly greater increase in NE output after exposure to a milder form of IS in comparison to nonhelpless, nonprestressed, or control animals. Petty et al. (1994) conclude that IS sensitizes the hippocampus to increase NE in response to a smaller, subsequent stressor.

Stress-induced analgesia (SIA) is another phenomenon that has been observed in IS experiments that may be relevant to PTSD in humans (van der Kolk et al., 1985). SIA refers to the finding that animals exposed to inescapable shock develop analgesia when re-exposed to a subsequent stressor within a brief period of time (van der Kolk, 1987). SIA is mediated by endogenous opioids and is reversed by naloxone (Kelly, 1982). Naloxone and termination of the stressful stimuli can produce opiate withdrawal symptoms suggesting that chronic stress can produce a physiological state similar to that of opiate dependency (van der Kolk, 1987).

It has been established that a reciprocal relationship exists between the opioid and noradrenergic systems (van der Kolk et al., 1985). It has also been suggested that the locus coeruleus mediates opiate withdrawal symptoms through noradrenergic hyperactivity (van der Kolk et al., 1985). Symptoms of opiate withdrawal include

anxiety, irritability, explosive outbursts, insomnia, hyperalertness, and emotional lability (van der Kolk et al., 1985). Given that these symptoms resemble the positive symptoms of PTSD, van der Kolk et al. (1985) postulate that opiate withdrawal and PTSD may have a common cause in that they are both due, in part, to central noradrenergic hyperactivity. Thus, exposure to a traumatic situation may give rise to an endorphin response that subjectively provides a paradoxical sense of control (van der Kolk et al., 1985). According to van der Kolk et al. (1985) subsequent withdrawal of the traumatic stimulus leads to the physiological symptoms of opiate withdrawal (anxiety, hyperactivity, and explosive outbursts of aggressiveness).

Pitman et al.'s (1990) finding that Vietnam veterans with PTSD exhibit SIA in response to trauma-related stimuli supports the SIA component of van der Kolk et al.'s (1985) model. The clinical observation that individuals exposed to traumatic events appear to experience a lifelong preoccupation with repetition of the trauma also supports this model (van der Kolk et al., 1985).

The IS model of PTSD has however been criticized for several reasons. It has been argued that the model fails to adequately explain the chronicity and delayed development of some of the symptoms of PTSD (Yehuda and Antelman, 1993; Jones and Barlow, 1990). In addition, Yehuda and Antelman (1993) state that the IS model does not account for the possibility of developing the symptoms of PTSD after a single, brief exposure to trauma. Finally, because the biochemical and behavioral alterations can only be produced in animals that are unable to escape the shock the model fails to take into consideration

the effect of stressor intensity, a factor that has been shown to be relevant to the severity of PTSD (Yehuda and Antleman, 1993).

Despite its shortcomings, Yehuda and Antleman (1993) suggest that the IS model of PTSD may be a valuable means to further the understanding of the factors that make an individual susceptible to PTSD. IS is able to account for the interindividual variability in response to a stressor in that it has been shown to produce behavioral differences in only a portion of exposed animals (Krystal, 1990). Studies in which animals have been bred for susceptibility to IS suggest that inherited biological traits may also influence vulnerability to PTSD (Krystal, 1990). In their Vietnam veteran twin study, True et al. (1993) reported that genetic analysis indicated inheritance had a substantial influence on liability for all symptoms, even after adjusting for combat experience.

The Kindling Model Of PTSD

As a result of his work with IS, van der Kolk (1987) also suggests that kindling is an animal model that may be applicable to PTSD. Kindling refers to a process by which repeated presentation of subthreshold stimuli, such as electrical or chemical stimulation, sensitizes limbic circuits and leads to lowered firing thresholds (Martin, 1991). According to the kindling model of PTSD, repeated traumatization or intrusive re-experiencing of a single trauma results in chronic central sympathetic arousal that is mediated by the locus coeruleus (van der Kolk, 1987). This chronic arousal then kindles pathways from the locus coeruleus to other limbic structures such as the hippocampus and the amygdala (van der Kolk, 1987). As was mentioned previously, it has been shown

in rats that sensitization of the hippocampus results in an increase of NE in response to subsequent, smaller stimulation (Petty et al., 1994). Nieminen, Sirvio, Teittinen, Pitkanen, Airaksinen, and Riekkinen (1992) have shown that kindling of the basolateral amygdala in rats increases anxiety in the elevated plus maze but does not affect spatial memory as evidenced by similar performance to that of control animals in the Morris water maze. Adamec (1990) and Adamec and McKay (1993) report that anxiety in the elevated plus maze is also increased in rats by kindling of the right medial amygdala. In a subsequent study Adamec and Morgan (1994) compared the effects of kindling in the medial and basolateral amygdaloid nuclei in each hemisphere. They found that kindling of the medial or basolateral amygdala in the left hemisphere decreased anxiety in the elevated plus maze for at least a week after the last kindled seizure. In contrast to this, kindling of the right hemispheric medial or basolateral amygdala tended to increase anxiety. The results of Adamec and Morgan's (1994) study are of particular interest in light of the soon to be published finding that individuals with PTSD show increased blood flow, as measured by positron emission tomography, in right-sided limbic and paralimbic areas when exposed to audiotapes of individualized traumatic event scripts (Rauch, van der Kolk, Fisler, Alpert, Orr, Savage, Fischman, Jenike, and Pitman, in press). This finding is consistent with the theory that the right hemisphere is involved in negative emotions (Sackeim, Greenberg, Weiman, Gur, Hungerbuhler, and Geschwind, 1982).

Thus, it appears that a kindling-like phenomenon may account for the generalized

anxiety experienced by many individuals with PTSD. Davis (1992) has also stated that electrical stimulation of the rat amygdala produces behavioral and autonomic effects that include changes in heart rate, blood pressure, respiration, and elevated startle. If kindling reduces the firing threshold for neurons in the amygdala or relevant structures connected to it, this could account for the increased arousability upon provocation observed in individuals with PTSD compared to individuals without PTSD .

Of additional relevance to the kindling model of PTSD, is the finding that anxiety and fear are more common in patients with epilepsy than normals (Mittan and Locke, 1982). Adamec (1990) has proposed that repeated and intense involvement of limbic tissue during epileptic seizures increases a patient's vulnerability to anxiety in response to the psychological and environmental stress created by their disorder. The demonstration that the anti-convulsant carbamazepine provides some benefit to PTSD sufferers also supports the kindling model of PTSD (Lipper, Davidson, Grady, and Edinger, 1986).

The Time-Dependent Sensitization Model of PTSD

A model related to kindling and IS is that of time-dependent sensitization (Antelman, 1988; Rosen and Fields, 1988; Yehuda and Antelman, 1993). Sensitization refers to the ability of a potentially threatening stimulus to enhance the response to the same or a weaker stimulus presented at a later time (Antelman, 1988). In the animal model of time-dependent sensitization, the animal receives one brief exposure to a stressor and is later tested with the same or another recall stressor (Yehuda and Antelman, 1993). In

comparison to animals receiving the stressor for the first time, animals previously exposed exhibit significantly altered responsivity (Yehuda and Antelman, 1993). Antelman (1988) has shown that this effect increases with time since the first exposure. According to Yehuda and Antelman (1993) this indicates that the influence of the first stressor strengthens solely as a function of the passage of time.

Intermittency is a key element of sensitization in that sensitization is more likely to occur following periodic rather than frequent exposure to an appropriate stimulus (Antelman, 1988). According to Antelman (1988) the combination of a potentially threatening stimulus and intermittency as the determinants of sensitization is accommodated within an evolutionary adaptation perspective, in that dangerous situations are more likely to be encountered on a more periodic basis than innocuous events. Thus, sensitization can be viewed as an unusual form of memory which enables an organism to make an accelerated defense response to a previously experienced threat (Antelman, 1988).

Yehuda and Antelman (1993) state that time-dependent sensitization meets all the requirements of what they believe constitutes an appropriate animal model of PTSD. First, it can occur as a result of a very brief exposure to a stressful event, as is the case with some cases of PTSD. Second, it can be induced by a variety of stressors of varying intensity which replicates Yehuda et al.'s (1992) finding of a relationship between stressor intensity and severity of PTSD symptoms. Third, the effects of time-dependent sensitization both persist for long periods of time and increase with the passage of time

which is similar to what is seen with chronic or delayed PTSD. Fourth, the effects of time-dependent sensitization can be excitatory or inhibitory which accounts for the intrusive and avoidance symptoms of PTSD. Finally, time-dependent sensitization shows interindividual variability which is consistent with the fact that not all individuals exposed to trauma will develop symptoms of PTSD (Yehuda and Antelman, 1993).

The Emotive Biasing Model Of PTSD

A final animal model of PTSD worth discussing is that of emotive biasing (Adamec, 1978). Pitman et al. (1993) have suggested that emotive biasing may account for the lasting changes in emotional disposition found in PTSD.

Emotive biasing combines elements of both the kindling and sensitization models. The hypothesis behind emotive biasing is that repeated stimulation of the limbic substrate of a specific emotional state ultimately alters the substrate and results in an enhancement of its functioning (Adamec, 1978). This idea is consistent with Kolb's (1987) theory that PTSD is the result of cortical neuronal death and synaptic changes that occur as a result of excessive and prolonged sensitization of limbic structures in response to trauma.

In support of Kolb's (1987) theory two recent studies employing magnetic resonance imaging have found reduced hippocampal volumes in individuals with PTSD. Bremner, Randall, Scott, Bronen, Seibyl, Southwick, Delaney, McCarthy, Charney and Innis (1995) report that individuals with PTSD have smaller right hippocampal volumes in comparison to age matched nonpsychiatric controls. In comparison to combat veterans without PTSD and non-PTSD, non-veterans, combat veterans with PTSD have also been

found to exhibit significantly reduced left and right hippocampi (Gurvits, Shenton, Hokama, Ohta, Lasko, Orr, Kikinis, Jolesz, McCarley, and Pitman, unpublished manuscript). While the reduced volumes observed in the individuals with PTSD may be the result of exposure to trauma, Gurvits et al. (unpublished manuscript) also suggest that individuals with pre-existing hippocampal abnormalities may be more vulnerable to developing PTSD in response to trauma.

Adamec's emotive biasing theory is based on his studies of feline defensive behavior. Feline defensive behavior meets the majority of the criteria for an animal model of anxiety (Adamec, 1990). Adamec (1978) has found that cats differ in defensive behaviour. On the basis of their response upon exposure to a rat, cats can be categorized as either "rat killers" or "non-rat killers" (Adamec, 1978). Cats that are rat killers attack the rat and kill it and exhibit little defensive behaviour. Non-rat killers exhibit a variety of defensive behaviours when exposed to the rat (Adamec, 1978). Because such differences in defensive response are seen very early in life it is possible that some animals may be born with a predisposition to respond defensively to threatening stimuli (Adamec, 1991).

Adamec (1978; 1990; 1991) has found that it is possible to alter defensive behaviour in cats. Partial kindling in the feline limbic system increases defensive responsivity in cats to a number of stimuli (Adamec, 1990; 1991). Although reversible, this change in responsivity can be very long lasting (Adamec, 1990).

It appears that in cats defensive response to threat is the result of three factors; (1)

increased excitability of limbic cells to threatening stimuli; (2) a biased routing of sensory information processed in the amygdala to the ventromedial hypothalamic circuitry of defense and (3) a biased routing of excitatory activity away from the ventral hippocampus, which has been shown to facilitate aggressive predation and reduce defensive behaviour (Adamec, 1991). Adamec (1991) has shown that partial kindling of the ventral hippocampus produces a lasting increase in defensive behaviour in cats to rats. This enhancement appears to depend on the growth of seizure activity in the ventromedial hypothalamus and the amygdala of the cat, suggesting the importance of the amygdala-ventromedial hypothalamic pathway in increased defensive response to rats (Adamec, 1991).

Further support for feline defensive behavior as a model of human anxiety comes from the finding that the beta carboline inverse agonist, FG-7142, increases feline defensive behavior in a behavioral and physiological manner almost identical to that of partial limbic kindling (Adamec, 1990; 1991; 1994). The relevance of this finding is that FG-7142 produces anxiety in humans (Adamec, 1990).

According to Pitman et al. (1993) emotive biasing can not be explained by conditioning because the rat stimulus is not presented during the electrical or pharmacologically induced acquisition. As well, the increase in defensive responding occurs across all situations (Pitman et al., 1993). Pitman et al. (1993) suggest that the concept of increased unconditional responding may explain features of PTSD such as irritability, hypervigilance, and exaggerated startle. These are all features of PTSD that

can not be explained by a conditioning model and are more accurately seen as being the result of consistent emotively biased fear (Pitman et al., 1993).

To strengthen the applicability of emotive biasing as a model of PTSD, Pitman et al. (1993) suggest that it should be demonstrated that lasting alterations in temperament can be produced by more natural stimuli than electrical or pharmacological stimulation. Adamec and Shallow (1993) have conducted such a study. In their study, rats were given a single five minute exposure to a cat. Anxiety behaviour was then measured in the elevated plus maze 1, 2, 7, 14, or 21 days after cat exposure. Increased anxiety, in comparison to controls, was found in the elevated plus maze 1 to 21 days after cat exposure. Severity of anxiety was predicted by the approach behaviour of the cat which is analogous to Foy, Sipprelle, Rueger, and Carroll's (1984) report that in Vietnam veterans, a significant portion of the variance in PTSD severity, can be accounted for by the extent and severity of combat exposure. Adamec and Shallow (1993) state that the findings of their study suggest that the increased anxiety observed in rodents in the elevated plus maze following exposure to a cat may model the acute and chronic anxiety seen in individuals with PTSD.

All of the models discussed above imply that many of the symptoms of PTSD may be the result of alterations in the specific neural systems that have been shown to be involved in the stress response and anxiety in animals. Future studies may be able to confirm these same alterations in human subjects through the sophisticated techniques of magnetic resonance imaging and positron emission topography.

The Present Study

One of the three symptom clusters included in the DSM-IV (1994) diagnostic criteria for PTSD is persistent increased arousal. Symptoms in this cluster include irritability or outbursts of anger and exaggerated startle response. Several factor analytic studies have been conducted that confirm these symptoms of increased arousal in PTSD (Keane, Caddell, and Taylor, 1988; Silver and Iacono, 1984; Watson, Kucala, Juba, Manifold, Anderson, and Anderson, 1988).

Increased levels of aggression, anger, and hostility have been found in combat veterans with PTSD in comparison to combat veterans without PTSD (Beckham, Roodman, Barefoot, Haney, Helms, Fairbank, Hertzberg, & Kudler, 1996; Chemtob, Hamada, Roitblat, and Muraoka, 1994: Lasko, Gurvits, Kuhne, Orr, and Pitman, 1994). As was discussed previously, exaggerated startle has also been found to be a prominent symptom of combat related PTSD.

The finding of increased hostility among combat veterans with PTSD (Beckham et al., 1996; Chemtob et al, 1994; Lasko et al., 1994) may have serious health implications for these individuals. Numerous studies have reported an increased risk of coronary heart disease and atherosclerosis in individuals with high levels of hostility (Lachar, 1993; Littman, 1993; Pasternac and Talajic, 1991; Shekelle, Gale, Ostfeld, and Oglesby, 1983; Suarez and Williams, 1989; Williams, Haney, Lee, Kong, Blumenthal and Whalen, 1980). Kubany, Gino, Denny, and Torigoe (1994) state that hostility in combat veterans with PTSD may increase their risk for cardiovascular disease.

While it is unknown if increased arousability, as evidenced by the exaggerated startle response, poses any major health concerns for veterans with PTSD, it is one of the most frequently occurring symptoms in these individuals (Keane, 1993).

Given the problems that these symptoms pose to individuals suffering from PTSD, a worthwhile task for PTSD research might be to determine which aspects of this disorder are related to the increased levels of hostility and startle response found in individuals with combat related PTSD. An analytic technique which may be of assistance in this endeavour is causal modeling with path analysis.

According to Fassinger (1987) causal modeling with path analysis is a useful technique for psychological research in that it enables a researcher to use correlational and nonexperimental data to test the applicability of their theoretical models to a specific sample. Path analysis requires the researcher to prepare statements that describe possible causal relationships between a number of variables (Biddle and Martin, 1987). These statements are most often theoretically based on the currently available literature in the relevant field of research. Once a path model has been constructed it is assessed against a data set that contains a correlation matrix showing the observed relationships among measures of variables collected during research (Biddle and Martin, 1987). A model is said to be confirmed if the correlations in the matrix match those that would have been predicted by the model (Biddle and Martin, 1987). Numerous statistical tests are available to assess the "goodness of fit" of a model to the data (Specht, 1975). It is also possible to compare several theory based models to determine which maximizes the goodness of

fit criteria (Biddle and Martin, 1987). Causal models tested in one sample can also be assessed by applying them to a new data sample (Biddle and Martin, 1987).

Based on what is known about anxiety, the startle response, hostility, and guilt it is possible to create several path models of possible relationships among these variables in PTSD. One such model is presented in Figure 1.

In this model, it is hypothesized that the trauma of combat exposure leads to an increase in trait anxiety. Several studies have reported increased levels of trait anxiety, as measured by Spielberger's State-Trait Anxiety Inventory (STAI), in veterans with PTSD when compared to non-PTSD combat controls (Hovens, Falger, Op den Velde, De Groen, and Van Duijn, 1994: Hovens, Op den Velde, Falger, Schouten, De Groen, and Van Duijn, 1992: Orr et al., 1995). Adamec's (1978; 1991) emotive biasing studies with cats and Adamec and Shallow's (1994) studies in rats suggest that this increase in trait anxiety may be due to the trauma of combat lastingly altering the neural substrate of anxiety/fear in these individuals and thus making them more anxious/fearful. As indicated in the model, this increase in the trait component of anxiety may then also increase an individual's state anxiety. Scores on the trait scale of the STAI have been found to be positively correlated, in normative samples, with scores on the state scale (Spielberger, 1983). Similar findings have been reported for college students (Martin, Blair, and Hatzel, 1987) and high school students (Layton, 1987). Thus, in the present model the path from trait to state anxiety suggests that individuals who have high levels of trait anxiety will experience high levels of anxiety in response to any situation or

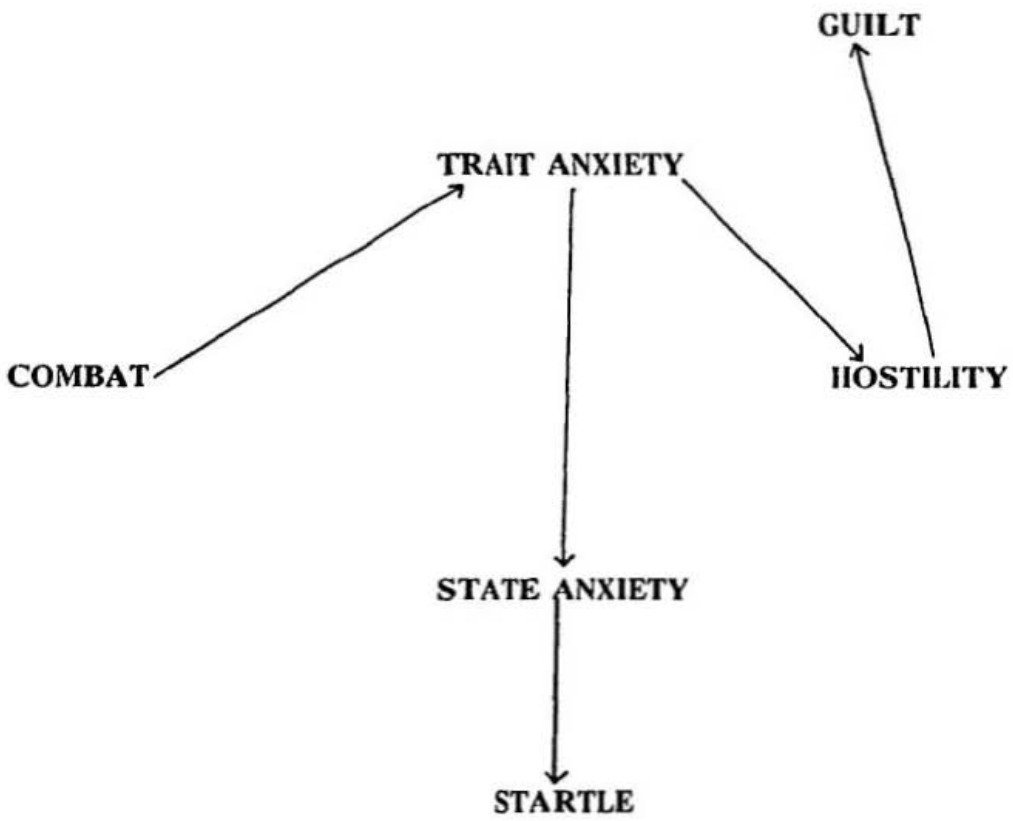

Figure 1. A possible model of the relationship between combat exposure, anxiety, startle, hostility, and guilt in PTSD.

object they perceive as threatening.

The previously discussed findings regarding the effects of high anxiety/fear on the startle response in animals and humans suggest that anxiety in PTSD may lead to increased startle response magnitude in sufferers of this disorder. This idea is shown in the model by a path leading from state anxiety to startle. Thus, increased startle in PTSD may be a form of the fear-potentiated startle seen in animals.

Lasko et al. (1994) state that the increased hostility in their subjects with combat related PTSD is not explained by the amount of combat exposure experienced by the individual. Beckham et al. (1996) report similar findings in their study of Vietnam combat veterans with PTSD. Thus, while combat exposure increases the risk for PTSD, hostility is a part of PTSD rather than the result of combat exposure (Lasko et al., 1994).

One possible cause of increased hostility in combat related PTSD may be anxiety. Evidence exists to support the idea that anxiety increases hostility. Bourne (1971) describes members of a Special Forces team in Vietnam who would engage in externally directed hostile behaviour to alleviate feelings of vulnerability in response to any environmental threat. Deffenbacher, Demm, and Brandon (1986) report that subjects in their study who were found to be high in general anger were also high in trait anxiety. To reduce anger, Deffenbacher et al. (1986) suggested using interventions aimed at reducing general anxiety. In addition, Katz, Wetzler, Cloitre, Swann, Secunda, Mendels, and Robins (1993) report that while anxiety in depressed women is related to motor retardation, anxiety in depressed men is highly related to hostility. They suggest that

while their findings only apply to anxious depression they may be applicable to other anxiety disorders (Katz et al., 1993). In terms of PTSD, Hovens et al. (1992) have found that anxiety and anger are highly related to each other in this disorder. Hovens et al. (1992) suggest that their findings indicate that uncontrollable anxiety makes one angry. In a recent study, Dutton (1995) reports that abusive men with a PTSD-like profile experience increased levels of chronic anger in a wide range of situations. Dutton (1995) suggests that, for these men, aggression/hostility may serve to dissipate anxiety. Thus, in men with PTSD, hostility may be a reaction to the increased levels of anxiety/fear they experience as a result of the disorder.

While guilt is no longer included as a symptom of PTSD in DSM-IV (1994), a number of Vietnam veterans experience guilt over their behavior (Watson et al., 1991; Glover, Pelesky, Bruno, & Sette, 1990). It is possible that veterans with PTSD experience guilt as a result of their increased levels of hostility. Paltiel (1981) has proposed a causal pathway for the violence that occurs in spousal abuse. According to Paltiel (1981), for a spouse abuser, the perception of threat leads to an increase in anxiety. Increased anxiety leads to hostility and/or aggression which then leads to guilt. This same cycle may be applicable to Vietnam veterans.

The previously discussed information supports the viability of the above model of the relationship between trauma, anxiety, exaggerated startle, hostility, and guilt. However, as previously mentioned, it is possible to compare a number of theory based models to determine which maximizes the goodness of fit criteria (Biddle and Martin, 1987). In

fact, many researchers state that testing of alternative models should be undertaken especially when knowledge in the area of interest is not complete enough to provide a single model specification (Bollen & Long, 1992; Hull, Lehn, & Tedlie, 1991; Raykov, Tomer, & Nesselroade, 1991).

Thus, it is the aim of the present study to test the applicability of the above hypothesized model and several plausible alternative models, which will be discussed in later sections, to a sample of Vietnam veterans with PTSD. A comparison of the models will allow us to determine which, if any, best represent the relationships between anxiety, hostility, guilt, and startle in PTSD.

Byrne (1994) states that a rare but valuable practice in the area of modeling is to test the generalizability of a model by cross-validating it over independent samples. Therefore, the models in the present study will be assessed and modified as necessary and then applied to a second data set.

Method

Subjects.

A total of 39 Vietnam combat veterans who met DSM-III-R (1987) criteria for PTSD and 34 Vietnam combat veterans without PTSD took part in the study.

Subjects in Sample 1 included 24 Vietnam combat veterans with PTSD and 24 non-PTSD Vietnam combat veteran controls who participated in the Chemtob et al. (1994) study. All data was collected by Chemtob et al. (1994). Veterans with psychoses, organic mental disorder, current alcohol or substance abuse or dependence, or anti-social personality disorder were excluded from the study.

Subjects in Sample 2 included 15 Vietnam combat veterans with PTSD and 10 non-PTSD Vietnam combat veteran controls. Subjects were participants in both the Lasko et al. (1994) and the Orr et al. (1995) studies. All data was collected solely by the researchers involved in these two studies. Excluded from the studies were individuals with a DSM-III-R (1987) diagnosis of a current organic mental, bipolar manic, schizophrenic, paranoid, delusional, or other psychotic disorder, or with alcohol or other substance dependence or abuse within the past year. Also excluded were individuals with a history of gross trauma or diagnosable neurologic disorder. None of the subjects had used psychotropic or other medications with potentially confounding neurologic or cognitive effects for at least two weeks prior to examination.

14 PTSD veterans in Sample 1 had a concurrent disorder: 10 had mood disorders, 1 had social phobia, and 3 had both a mood disorder and an anxiety disorder other than

PTSD (of these 3, 2 veterans had panic disorders and 1 had agoraphobia without panic disorder). One non-PTSD control subject had a mood disorder. Comorbid axis I disorders in PTSD subjects in Sample 2 included 4 current and 5 past major depression, 4 current dysthymia, and 2 current generalized anxiety disorders. Some subjects had more than one comorbid axis I disorder. Axis I disorders in the non-PTSD control subjects included one current and one past major depression and one current dysthymia disorder.

Materials.

Anxiety was measured by the State-Trait Anxiety Inventory (STAI; Speilberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983; see Appendix A). The STAI is a 40 item self-administered test of two aspects of anxiety. The State anxiety component of the test asks the subject to indicate how they feel at the present moment. Trait anxiety is assessed by having the subject indicate how they "generally" feel.

Hostility was measured with the Hostility Inventory (Buss & Durkee, 1957; see Appendix B). The Hostility Inventory consists of 75 true-false items each representing one of seven types of hostility including assault, indirect hostility, irritability, negativism, resentment, suspicion, and verbal hostility. The sum of these seven hostility scales yields a total scale on the instrument. In addition to the hostility subscales, the inventory includes a guilt subscale.

The combat exposure of subjects in Sample 1 was assessed with the Combat Exposure Scale (CES; Lund, Foy, Sipprelle, & Strachan, 1984; see Appendix C). The

CES is a Guttman scale in which eight items describing stressful events related to military experience in Vietnam are hierarchically arranged to reflect increasingly more intense combat exposure.

Combat exposure was assessed for subjects in Sample 2 with the Legacies Combat Scale-Revised (Egendorf, Kadushin, Laufer, Rothbart, & Sloan, 1981; see Appendix D). The Legacies consists of 10 hierarchical self-report items that deal with a number of combat situations a subject may have experienced in Vietnam. The lower six items are scored "1" if applicable; the remaining four items are scored "2". A score of 1 to 4 is considered light combat exposure, 5 to 9 moderate, and 10 to 14 heavy combat exposure. Foy, Siprelle, Rueger, and Carroll (1984) have found the Legacies Combat Scale to be highly correlated with the CES. Fontana and Rosenbeck (1993) report similar findings. Thus, after rescaling the scores on the CES, the Legacies and CES were taken to be equivalent measures of combat exposure for subjects in this study.

The stimuli, dependent physiologic measures, and procedure employed by Orr et al. (1995) to measure the physical responsivity (startle) of subjects in Sample 2 were the same as those employed by Shalev et al. (1992). Stimuli consisted of 15 95-dB, 1000 Hz, 500 ms pure tones with 0 ms rise and fall times. Stimuli were presented binaurally over headphones with intertrial intervals randomly selected by a computer and ranging from 27s to 52s. While data for skin conductance and heart rate were available from the Orr et al. (1995) study, the only dependent physiologic measure of interest for the present study was the left orbicularis oculi electromyogram (EMG). This measure was chosen

because, in humans, the acoustic startle reflex is measured as the magnitude of the eyeblink, EMG response, component of the reflex (Morgan et al., 1995).

Subjects were seated comfortably in a humidity- and temperature-controlled, sound-attenuated room. After the recording electrodes were attached, subjects were given the following instructions:

> You are going to hear a series of tones. Please sit quietly and listen to the sounds as they come. Keep your eyes open throughout the entire procedure, which will not last more than twenty minutes. There will be a five-minute resting period before the tones begin. Do you have any questions?

A technician then placed earphones on the subject and left the room. Participation was monitored through closed circuit television. EMG response was sampled at the rate of 2 Hz during the five-minute rest period after which the first tone was presented without warning. Sampling frequency was increased to 50 Hz at 4s prior to each tone presentation and continued at this rate until 8.5s after each tone onset.

An EMG response score for each of the 15 tone trials was calculated by subtracting the mean EMG level during the 1 second immediately preceding tone onset from the highest EMG level measured within 40 to 200 milliseconds after tone onset. The EMG responses were then averaged across the 15 trials.

Scores on the Mississippi Scale for Combat-Related Post-Traumatic Stress Disorder (Keane, Caddell, & Taylor, 1988; see Appendix E) were also available for all subjects in both samples. The Mississippi Scale is a 35 item, Likert scale, self-report questionnaire specifically designed to assess PTSD symptoms in Vietnam combat

veterans. Subjects' scores on this measure were used solely in the present study as predictor variables in regression equations employed to replace missing values of other variables.

Statistical Analysis

As previously discussed, causal modeling with path analysis is a multivariate analytic technique that allows a researcher to test for possible causal relationships among a number of variables. Given the difficulties encountered in treating the symptoms of PTSD and the potentially life threatening nature of symptoms such as hostility, path analysis was employed in the present study in an attempt to elucidate the nature of the relationships between anxiety, hostility, guilt, and increased startle reactivity in Vietnam veterans with PTSD. By better understanding these relationships, it may be possible to determine the most efficient and successful means of treating these symptoms. In the following section the hypothesized model and several alternative models are presented along with the results of the path analyses conducted on these models.

Results

Data Screening

Prior to analysis all variables for both samples were examined through several NCSS, BMDP, and EQS programs for accuracy of data entry, missing values, and fit between their distributions and the assumptions of multivariate analysis. The issue of kurtosis is discussed later within the model construction section. The variables were examined separately for each sample and for the subjects with PTSD and the non-PTSD combat control subjects within each sample.

Missing Values

In both Sample 1 and Sample 2, four cases were found to have missing values. State anxiety and education were the only variables to have missing values. Due to the small sizes of the samples it was decided to estimate the missing data. An acceptable method for estimating missing values is to use regression equations (Tabachnik and Fidell, 1989). In the present study, the BMDP (1990) program 9R was used to compute regression equations employing the best set of predictors from each data sample for each variable with missing values. These equations were then used to compute scores to replace the missing data points where necessary. Table 1 shows the variables and the R^2 for the regression equation used to compute missing values of state anxiety in Sample 1. Table 2 shows the same information for the regression equation used to compute missing values of state anxiety in Sample 2. Finally, Table 3 shows the variables and the R^2 value for the regression equation used to compute missing values of education in Sample 2.

Table 1. BMDP best predictor equation for state anxiety in Sample 1 (N=48)

Variable	Regression Coefficient
Mississippi Scale	0.451533
Buss Durkee Assault	1.75992
Buss Durkee Indirect	3.38975
Buss Durkee Resentment	3.67336
Buss Durkee Total	-1.00922
Intercept	-23.04430

*R^2=0.79411, p<.0001

Table 2. BMDP best predictor equation for state anxiety in Sample 2 (N=25)

Variable	Regression Coefficient
Mississippi Scale	0.485810
Intercept	0.510772

R^2=0.60534, p<.0001

Table 3. BMDP best predictor equation for education in Sample 2 (N=25)

Variable	Regression Coefficient
Legacies Combat Scale	0.208117
Mississippi Scale	-0.057005
Buss Durkee Total	0.455551
Buss Durkee Assaultive	-0.639633
Buss Durkee Irritable	-0.443894
Buss Durkee Negative	-1.063333
Buss Durkee Suspicion	-0.924394
Intercept	14.594800

*R^2=0.67992, p<.01

Outliers

Path analysis is highly sensitive to the inclusion of univariate outliers, cases with extreme values on one variable, and multivariate outliers, cases with extreme values on a combination of variables (Tabachnik and Fidell, 1989). While the NCSS screening program showed there to be no multivariate outliers in either Sample 1 or Sample 2, histograms revealed that several variables with univariate outliers existed in both samples.

As the variables found to have univariate outliers were an integral part of the analysis, steps were taken to reduce the influence of the outliers. Tabachnik and Fidell (1989) suggest that one way of dealing with outliers is to change the score on the relevant variable for the outlying case so that it is deviant but not as deviant as it was. This can be done by assigning the outlying case a score on the variable that is one unit larger or smaller than the next most extreme score in the distribution (Tabachnik and Fidell, 1989).

In the non-PTSD control group of Sample 1, one case was identified as an outlier due to an extreme score on the Buss Durkee assault subscale. The score for this case was changed from 8 to 4, one above the next lowest score. Two cases with high scores on the Buss Durkee suspicion subscale had their scores changed from 6 and 9 to 3, one above the next highest score.

Examination of the data for the non-PTSD control group from Sample 2 revealed several univariate outliers. Two cases were identified as having extreme scores on the

trait anxiety measure. Their scores was changed from 8 to 22 and 77 to 46 respectively. Two cases were outliers due to high state anxiety scores. Their scores were changed from 35 and 39 to 23, one above the next highest score. One case was an outlier due to a high score on the Buss Durkee suspicion subscale. Their score was changed from 8 to 5.

One case in the PTSD group of Sample 2 was identified as an outlier due to a low score on the Buss Durkee irritability subscale. The score for this subject was changed from 3 to 5.

Group Demographic, Psychometric, and Physiologic Means

Group differences on the demographic, psychometric, and physiologic measures were assessed within the two data samples for two reasons. Firstly, the data set compiled from Lasko et al (1994) and Orr et al. (1995) was based on a reduced sample size and thus it was important to report the descriptive statistics for the reduced data set. Secondly, reporting the group differences within each sample provides a general picture of the characteristics of the two samples. Having a picture of the two samples can aid in the interpretation of the path analysis. For example, comparing where differences lie within the two samples may help in accounting for possible noninvariances in path coefficients when a model is tested on the two samples.

Table 4 shows the means and standard deviations of the demographic, combat exposure, anxiety, hostility, guilt, and physiologic measures for subjects in Sample 1. Table 5 shows the means and standard deviations of the same variables for subjects in

Sample 2.

The PTSD and non-PTSD subjects in both Sample 1 and Sample 2 did not differ significantly in age or amount of education. Nor did the two samples differ on these measures.

There was no significant group difference in Sample 1 with regards to amount of combat exposure, although the mean exposure scores were slightly higher for the combat veteran control subjects. In contrast to Sample 1, the veterans with PTSD in Sample 2 reported experiencing significantly more combat exposure than the veterans without PTSD.

Veterans with PTSD in Sample 1 and Sample 2 had significantly higher scores than control subjects on the state component of the STAI. Veterans with PTSD in Sample 1 also had significantly higher scores than control subjects on the trait component of the STAI. In a similar fashion, veterans with PTSD in Sample 2 had higher scores than controls on the trait component of the STAI, though the group difference was weaker than in Sample 1, $F(1,23)=3.88$, $p<.07$: equivalent $t(23)=1.97$, $p<.031$, 1-tailed. A one-tailed t-test is justified in this instance given the numerous studies reporting that veterans with PTSD have significantly higher levels of trait anxiety than combat veteran controls without PTSD (Chemtob et al., 1994; Hovens et al., 1994; Hovens et al., 1992; Hyer, O'Leary, Saucer, Blount, Harrison, & Boudewyns, 1986; Orr et al., 1995).

Veterans with PTSD in both Sample 1 and Sample 2 had significantly higher total scores on the Hostility Inventory than control subjects. Veterans with PTSD in both

Table 4. Group demographic, psychometric, and physiologic means and standard deviations for Sample 1 (N=48)

	PTSD(n=24)	Non-PTSD(n=24)	F(1,46)
Age (years)	43.38+/- 6.30	44.63+/- 5.03	0.58
Education (years)	15.92+/- 3.44	15.00+/- 2.77	1.04
Combat Exposure	9.10+/- 3.43	10.27+/- 2.13	2.00
Hostility Inventory			
Assault	7.17+/- 2.28	4.54+/- 2.78	12.79***
Indirect	6.25+/- 1.92	3.25+/- 2.03	27.75***
Irritability	9.17+/- 1.49	3.79+/- 2.62	76.20***
Negativism	3.58+/- 1.25	2.00+/- 1.41	16.91***
Resentment	5.25+/- 1.87	1.63+/- 1.84	45.87***
Suspicion	6.21+/- 2.55	1.67+/- 1.79	50.99***
Verbal	8.42+/- 2.39	6.29+/- 2.60	8.69*
Guilt	5.88+/- 1.85	2.63+/- 2.14	31.64***
Total	46.33+/- 8.12	23.67+/-11.21	64.36***
STAI			
State Anxiety	37.63+/-14.51	7.67+/- 8.06	78.13***
Trait Anxiety	54.04+/-12.23	30.42+/- 7.19	66.56***

*= p<.01, two-tailed
**= p<.001, two-tailed
***= p<.0001, two-tailed

Table 5. Group demographic, psychometric, and physiologic means and standard deviations for Sample 2 (N=25).

	PTSD (n=15)	nonPTSD (n=10)	F(1,23)
Age (years)	44.00+/- 2.04	46.10+/- 4.25	2.76
Education (years)	14.40+/- 1.88	15.50+/- 2.88	1.35
Combat Exposure	12.07+/- 1.44	9.40+/- 4.03	5.60^{+}
Hostility Inventory			
Assault	6.47+/- 2.90	3.10+/- 1.29	11.79*
Indirect	5.67+/- 2.64	5.00+/- 1.89	0.47
Irritability	8.40+/- 1.92	5.60+/- 3.24	7.41^{+}
Negativism	3.13+/- 1.60	2.70+/- 2.00	0.36
Resentment	5.40+/- 1.80	2.00+/- 1.83	21.10^{+}
Suspicion	5.93+/- 1.98	1.80+/- 1.40	32.50***
Verbal	10.20+/- 1.90	7.20+/- 2.04	14.11**
Guilt	5.27+/- 1.62	3.7+/- 1.70	5.37^{+}
Total	45.00+/- 9.72	28.70+/-12.11	13.87**
STAI			
State Anxiety	57.47+/-15.51	36.20+/-12.81	12.88*
Trait Anxiety	48.20+/-13.15	37.30+/-14.16	3.88
Electromyogram R.A.	1.08+/- 0.55	0.43+/- 0.33	11.07*

$+$ = $p<.05$, two-tailed
$*$ = $p<.01$, two-tailed
$**$ = $p<.001$, two-tailed
$***$ = $p<.0001$, two-tailed

samples also had significantly higher scores than controls on the assault, irritability, resentment, suspicion, verbal, and guilt subscales of the Hostility Inventory. While veterans with PTSD in Sample 1 had significantly higher scores than controls in Sample 1 on the indirect and negativism subscales, no significant group differences existed on these subscales for subjects in Sample 2.

With regards to physiological responsivity, veterans with PTSD in Sample 2 had, on average, a significantly larger electromyogram response average than combat veterans without PTSD. Physiological data were not available for the subjects in Sample 1.

The Buss Durkee Hostility Inventory: The Issue of Employing the Total Score Versus Scores on the Individual Subscales

The total score on the Buss Durkee Hostility Inventory is a composite score made up of scores on the individual hostility subscales. Thus, an issue that needed to be addressed before constructing any models, was whether to use the total score on the Buss Durkee Hostility Inventory to represent hostility within our model or whether to consider employing individual hostility subscales.

To determine which alternative was most appropriate it was decided to run two discriminant analyses on the anxiety and hostility measures; one including the Hostility Inventory total score by itself and one which included both the total score and the individual subscale scores. A comparison of the predictor variables selected for and the accuracy of classification provided by the resulting discriminant functions of these two analyses would then be possible. From these comparisons it could be determined which

aspect of the hostility scale, i.e which subscales or the total score, most reliably represented hostility as a variable which discriminated PTSD from non-PTSD persons. For this reason, analyses were run on both of the data samples.

Discriminant Analysis: Normality of Distributions

The D'Agostino-Pearson omnibus K^2 test was used to assess the normality of the distributions of the relevant variables in the discriminant analyses. The only variable found to have a nonnormal distribution was the Buss Durkee assaultive subscale ($K^2=6.36$, $p<.042$) for the PTSD subjects in Sample 1. However, as discriminant analysis is robust with regards to nonnormal distributions in which the smallest group in the analysis is larger than 20 (Tabachnik & Fidell, 1989) it was decided to include this variable in the analyses and not transform it.

Discriminant Analysis: Method

As there was no reason to assign any of the variables higher priority than others, a stepwise discriminant analysis was used to produce the reduced set of predictors (Tabachnik & Fidell, 1989). Wilks' Lambda was used to direct the progression of entry of predictors in the analysis. This method produces the smallest values of Wilks' Lambda i.e. the largest multivariate F values (Tabachnik & Fidell, 1989).

In order to reduce classification bias and to ensure that the discriminant functions derived from the analyses were valid, the jackknife, or leave one out, method of cross-validation was used (Betz, 1987; Huberty, 1994; Tabachnik & Fidell, 1989).

Both analyses were performed using the BMDP7M (1990) stepwise jackknife

discriminant analysis procedure. Prior probabilities of group membership were set at .50 and .50 for Sample 1 with equal group size and .40 and .60 for sample 2 with unequal numbers in the groups.

Discriminant Analysis I: Hostility Inventory Total Score (No Hostility Subscales)

Stepwise discriminant function analysis tested the accuracy of inclusion in the predetermined PTSD and non-PTSD groups based on state anxiety, trait anxiety, and the Hostility Inventory total score.

Table 6a shows the two variables used in the discriminant function and the associated standardized (by pooled with-in group variances) canonical coefficients based on the data from Sample 1. The variables, state anxiety and Hostility Inventory total score, are shown in order of their inclusion in the discriminant function. State anxiety, as measured by the STAI, was included in the function first as it was the variable that contributed the most (i.e. had the smallest Wilks' Lambda and hence the largest F value) to the separation of the groups with respect to the discriminant function.

As can be seen from Table 6b, the discriminant function procedure correctly classified 91.7% of the PTSD patients and 91.7% of the non-PTSD subjects in Sample 1.

A stepwise discriminant analysis was run on the data from Sample 2 using the same variables as were entered in the first analysis with the data from Sample 1. Table 7a shows the two variables, in order of inclusion, that were used in the discriminant function and the associated standardized canonical coefficients. As was the case with

Table 6a. Stepwise discriminant function analysis on Sample 1 (no Hostility Inventory subscales): canonical variables*

Variable	Standardized Coefficient
State Anxiety	-0.66845
Hostility Inventory Total	-0.54686
Eigenvalues	2.30435
Cumulative Proportion of Total Dispersion	1.00000
Canonical Correlations	0.83509
Constant	3.24486

* The remaining variable did not have a high enough F value to be included in the discriminant function.

Table 6b. Stepwise discriminant function analysis on Sample 1 (no Hostility Inventory subscales): jackknifed classification.

	Percent Classified As:	
Diagnostic Group	PTSD	Non-PTSD
PTSD	91.7	8.3
Non-PTSD	8.3	91.7

Table 7a. Stepwise discriminant function analysis on Sample 2 (no Hostility Inventory subscales): canonical variables*

Variable	Standardized Coefficient
Hostility Inventory Total	-0.66580
State Anxiety	-0.63053
Eigenvalues	0.97760
Cumulative Proportion of Total Dispersion	1.00000
Canonical Correlations	0.70309
Constant	4.51594

* The remaining variable did not have a high enough F value to be included in the discriminant function.

Table 7b. Stepwise discriminant function analysis on Sample 2 (no Hostility Inventory subscales):jackknifed classification.

	Percent Classified As:	
Diagnostic Group	PTSD	Non-PTSD
PTSD	93.3	6.7
Non-PTSD	40.0	60.0

Sample 1, the Hostility Inventory total score and state anxiety were significant predictors of group membership. However, for Sample 2, the Hostility Inventory total score, rather than state anxiety, contributed the most to the group separation based on the discriminant function.

The discriminant function procedure correctly classified 93.3% of subjects with PTSD and 60.0% of non-PTSD subjects in Sample 2 (Table 7b). The classification rate for PTSD subjects is comparable to that obtained with Sample 1. However, there is considerably more misclassification of control subjects in Sample 2.

Discriminant Analysis II: Hostility Inventory Total Score and Hostility Subscale Scores

The total score on the Hostility Inventory was a significant discriminating variable on the analyses for both data samples. As the total score is comprised of the scores on the seven individual hostility subscales, a second discriminant analysis was run on the two samples this time with both the hostility subscales and the Hostility Inventory total score included. From this analysis it would be possible to determine if one specific aspect of hostility, as measured by the subscales, best differentiated the PTSD and non-PTSD groups in place of the total score. If this should be the case then that subscale or set of subscales would be chosen to represent hostility in our models.

Table 8a shows, in order of their inclusion, the two variables that were used in the discriminant function and the associated canonical coefficients based on the data from Sample 1. As with the first analysis, state anxiety is a significant predictor of group

Table 8a. Stepwise discriminant function analysis on Sample 1 (Hostility Inventory subscale scores and total score included):canonical variables*

Variable	Standardized Coefficient
State Anxiety	-0.77065
Buss Durkee Suspicion	-0.61423
Eigenvalues	2.72601
Cumulative Proportion of Total Dispersion	1.00000
Canonical Correlations	0.85535
Constant	2.58418

* The additional 8 variables did not have high enough F values to be included in the discriminant function.

Table 8b. Stepwise discriminant function analysis on Sample 1 (Hostility Inventory subscale scores and total scores included):jackknifed classification.

	Percent Classified As:	
Diagnostic Group	PTSD	Non-PTSD
PTSD	91.7	8.3
Non-PTSD	8.3	91.7

Table 9a. Stepwise discriminant function analysis on Sample 2 (Hostility Inventory subscale scores and total scores included):canonical variables*

Variable	Standardized Coefficient
Buss Durkee Suspicion	-1.00000
Eigenvalues	1.41324
Cumulative Proportion of Total Dispersion	1.00000
Canonical Correlations	0.76526
Constant	2.41012

* The additional 9 variables did not have high enough F values to be included in the discriminant function.

Table 9b. Stepwise discriminant function analysis on Sample 2 (Hostility Inventory subscale scores and total scores included):jackknifed classification.

	Percent Classified As:	
Diagnostic Group	PTSD	Non-PTSD
PTSD	86.7	13.3
Non-PTSD	0.0	100.0

membership. However, the Hostility Inventory total score has been replaced in the discriminant function by the suspicion subscale score.

As shown in Table 8b, this second discriminant function correctly classified 91.7% of the subjects with PTSD and 91.7% of the control subjects in Sample 1. These classification rates are identical to those found with the analysis that did not include the hostility subscales.

The variable used in the discriminant function based on Sample 2 and the associated standardized canonical coefficient are shown in Table 9a. In this analysis only one variable, suspicious hostility, was included in the discriminant function. As occurred with Sample 2, the suspicion subscale score replaced the Hostility Inventory total score in the discriminant function.

As per Table 9b, this discriminant function correctly classified 100% of the control subjects and 86.7% of the subjects with PTSD. These rates are similar to those found with Sample 1. However, in comparison to the analysis that did not include the Hostility Inventory susbscales, the discriminant function produced by this analysis vastly improved classification of control subjects but loses some accuracy in terms of PTSD subject classification.

Analysis of Covariance

As reported above, the suspicion subscale of the Hostility Inventory was a significant predictor of group membership for both samples and in fact provided better overall classification for subjects in Sample 2 than the Hostility Inventory total score. It was thus

possible that this subscale characterizes hostility in veterans rather than the total hostility score. To address this issue the contribution of suspicious hostility to differences between PTSD and control veterans in other aspects of hostility was examined. Analyses of covariance were used to remove the influence of suspicious hostility on the other subscales and total scores of the Hostility Inventory for both data sets.

The F values obtained with the covariance analyses are shown in Table 10 for Sample 1 and in Table 11 for Sample 2. For both Sample 1 and Sample 2 assault, resentment, and verbal subscales, group differences depended on suspicious hostility. Covarying suspicious hostility scores out of these measures removed group differences ($p > .05$). However, while covarying suspicion out of the Hostility Inventory total score and the irritable subscale for Sample 2 removed statistical group differences ($p > .05$), group differences in these variables remained significant in Sample 1 with suspicion as a covariate ($p < .005$).

Covarying suspicion from the negativism subscale for Sample 1 removed group differences while group differences remained statistically significant for the indirect subscale ($p < .01$). There were no group differences on these two subscales for Sample 2 however, so analysis of covariance was not run on these variables.

The results of these analyses of covariance suggest that while suspicious hostility may be a significant discriminating variable when classifying combat veterans with and without PTSD, it does not capture all aspects of hostility experienced by combat veterans suffering from PTSD. The present analyses suggest that hostility is expressed in different

Table 10. F and probability values of group differences on hostility measures with Buss Durkee suspicion subscale as covariate based on Sample 1 (N=48)[1]

Variable	F(1,45)	p (two-tailed)
Buss Durkee Assault	1.24	0.2718
Buss Durkee Indirect	8.79	0.0048
Buss Durkee Irritability	19.58	0.0001
Buss Durkee Negativism	2.99	0.0907
Buss Durkee Resentment	2.85	0.0985
Buss Durkee Verbal	0.42	0.5214
Buss Durkee Total Score	9.15	0.0041

[1] See Table 4 for original comparisons

Table 11. F and probability values of group differences on hostility measures with Buss Durkee suspicion subscale as covariate based on Sample 2 (N=25)[1]

Variable	F(1,22)	p (two-tailed)
Buss Durkee Assault	0.98	0.3329
Buss Durkee Indirect*		
Buss Durkee Irritability	0.19	0.6698
Buss Durkee Negativism*		
Buss Durkee Resentment	0.68	0.4182
Buss Durkee Verbal	1.44	0.2433
Buss Durkee Total Score	0.10	0.7608

*Covariance analysis not done, see text.
[1] See Table 5 for original comparisons

forms, within different populations. In this sense use of the Hostility Inventory total score is more likely to uncover hostility difference per se. Attention to individual subscales in different populations is also warranted, though replicability across samples may be less likely for individual subscales than for the total score. Thus, for the purposes of the present study the Hostility Inventory total score will be used to represent hostility.

Path Analysis - Model Construction

In order to better understand the nature of some of the symptoms of PTSD, path analysis was used to examine the relationships between trauma, anxiety, hostility, and guilt. The path diagram for the hypothesized model of these relationships is shown in Figure 2. This diagram, like those of all subsequent figures, follows the conventions of EQS version 5.1 (Bentler & Wu, 1995). Measured variables are shown in boxes and unmeasured variables are shown in circles. Unidirectional arrows between variables represent regression coefficients and indicate the influence of one variable on another. Positive coefficients indicate a positive relationship between the variables i.e. high scores on one variable predict high scores on the other variable. Negative coefficients indicate an inverse relationship between the two variables. Sourceless one-way arrows represent error terms, i.e. the residuals associated with the measurement of the observed variables. The rationales for the various paths in the model are discussed below.

A path is predicted from combat exposure to trait anxiety on the basis of animal research that indicates a relationship between traumatic events and increased fear/anxiety. Adamec's (1978; 1991) emotive biasing studies with cats have found that partial kindling

or pharmacological stimulation of brain areas associated with fear/anxiety produce a long lasting increase in feline defensive behavior which is considered analogous to anxiety in humans. In addition, Adamec and Shallow (1993) report that exposure to a cat, a natural fear stimulus for a rodent, lastingly increases anxiety like behaviors in rodents. Adamec (1996) has argued that such anxiety increases in animals model aspects of anxiety disorder following traumatic stress. These studies suggest that, in humans, the trauma of combat may lastingly alter the neural substrate of anxiety/fear in individuals with PTSD and thus make them more anxious/fearful. It could be suggested that, rather than exposure to trauma producing an increase in trait anxiety, individuals high in trait anxiety may be more predisposed to develop PTSD in response to traumatic events. However, Shalev, Peri, Canetti, and Shreiber (1996) have conducted a prospective study of possible predictors of PTSD in individuals exposed to road traffic accidents, work and other accidents, terrorist acts, and armed assaults which suggests that trait anxiety does not predispose an individual to PTSD. In their study, Shalev et al. (1996) found no significant differences in trait anxiety one week post trauma between those subjects who went on to develop PTSD and those who did not. As it has been consistently shown that individuals with PTSD experience significantly higher levels of trait anxiety than individuals without PTSD (Hovens et al., 1994; Hovens et al., 1992; Kuhne, Orr, & Baraga, 1993; Orr et al., 1995; Sutker, Bugg, & Allain, 1991) it would appear that increased trait anxiety in PTSD is the result of exposure to trauma rather than a predisposing factor for the development of PTSD.

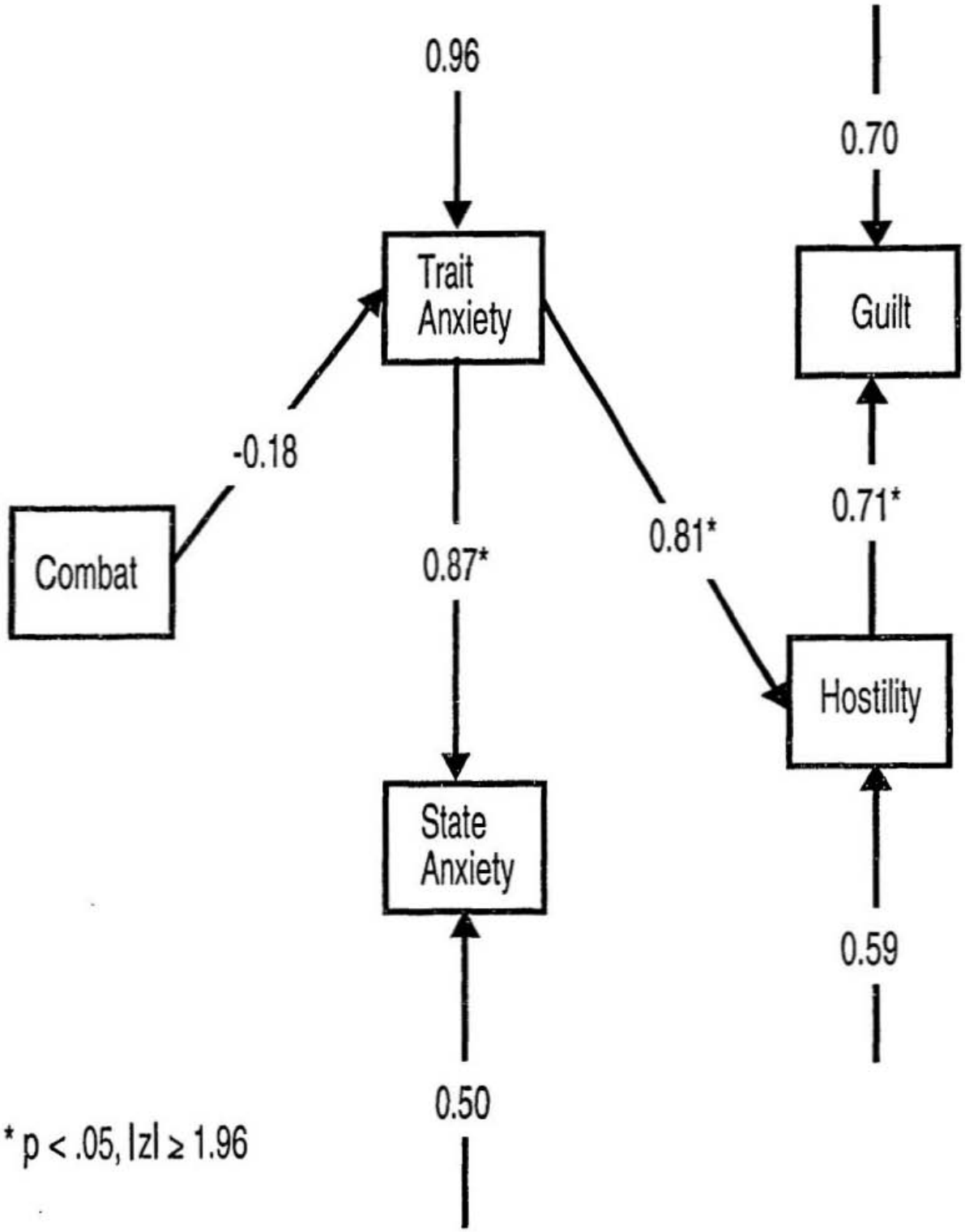

Figure 2. Standardized solution of Model 1, data from Sample 1 (N=48)

Thus, a path from trauma to trait anxiety is justified in the present model from human as well as animal data. Moreover, a positive path coefficient between trauma and anxiety is predicted given that high scores on the CES are related to higher levels of PTSD symptom intensity (Lund et al., 1984).

A strong correlation had been found between scores on the trait and state components of the STAI in normative samples (Spielberger, 1983), college students (Martin et al., 1987), and high school students (Layton, 1987). In addition, Grillon et al. (1993) reported that, in their study of anxiety and fear-potentiated startle, individuals who scored high on the trait component of the STAI also scored higher on the state component of the STAI when threatened with a shock. Based on these findings, which support the logical proposition that an anxious individual will experience increased levels of anxiety in response to situations that they perceive as threatening, a path, with a positive path coefficient, is predicted from trait anxiety to state anxiety.

A path is also predicted from trait anxiety to hostility. It should be noted that for all models assessed in this study, hostility is represented by the total score on the Hostility Inventory. This is in keeping with the results of the discriminant and covariance analyses which suggested that the total score is a more appropriate measure of hostility than any of the individual subscales. Increased hostility does not appear to be related to the amount of combat an individual experiences (Lasko et al., 1994; Beckham et al., 1996). Thus, a path from combat exposure to hostility would not be predicted. There is considerable evidence to support the idea that increased hostility in PTSD is the result

of increased levels of anxiety. Deffenbacher et al. (1986) have found that subjects who score high on measures of anger also score high in trait anxiety. Katz et al. (1993) suggest that hostility in depressed men may be related to high trait anxiety. Bourne's (1971) studies with Vietnam combat soldiers suggest that hostility may be a means of dealing with anxiety provoking situations. This corresponds to Hovens et al.'s (1992) suggestion that the high correlation they found in their study between anger and trait anxiety in veterans with PTSD is the result of uncontrollable anxiety making one angry. Dutton (1995) also suggests that, in abusive men who exhibit a PTSD profile, aggression/hostility may function to alleviate anxiety. Thus, individuals with PTSD who are high in trait anxiety may become more hostile in response to their increased feelings of anxiety. A positive path coefficient is therefore expected between trait anxiety and hostility.

Glover et al. (1990) report that many Vietnam veterans experience considerable guilt for having participated in aggressive acts during the war. This is consistent with Paltiel's (1981) proposition that for male spouse abusers, hostility and/or aggression leads to feelings of guilt. Thus, individuals with PTSD who find themselves feeling and acting more hostile as a result of their disorder may experience considerable guilt as a response. Thus, a path is predicted in this model from hostility to guilt. A positive path coefficient is expected, indicating that individuals who exhibit more hostility experience more guilt.

Path Analysis: Method and Goodness of Fit Indices

The hypothesized model appears in Figure 2. This model was tested in the subjects

in Sample 1 (N=48) using EQS version 5.1 (Bentler & Wu, 1995). Mardia's coefficient generated by the EQS program and shown in Table 12 confirmed that none of the variables being tested exhibited significant multivariate kurtosis.

All of the beta weights in this model were assessed as free parameters by the maximum likelihood method. The correlation matrix for this model is shown in Appendix F.

Following Bollen and Long (1992), several indices of overall fit were used. In accordance with Hoyle and Panter's (1995) recommendations, several fit indices were chosen to assess the model. These are presented in Table 12. The chi-square for the hypothesized model is reported. The value of this X^2 should be nonsignificant indicating a good fit of the data to the hypothesized model. Hoyle and Panter (1995) recommend reporting Joreskog and Sorbom's (1981) goodness-of-fit index (GFI) in addition to chi-square. The GFI indicates the relative amount of observed variance and covariance jointly accounted for by the model (Hoyle and Panter, 1995). Finally, Bentler's (1990) comparative fit index (CFI) was included. The CFI is the index of choice when dealing with small samples (Bentler, 1990). Values for the CFI are derived from a comparison of the hypothesized model with the null model, the model which assumes all correlations between variables equal zero (Byrne, 1994). The CFI reflects the reduction in X^2 associated with the hypothesized model versus the null model. For the GFI, values can range between 0 and 1. For the CFI, values are 0 and forced to a ceiling of 1. Values greater than .90 on either index are taken to indicate an acceptable fit of the model to

Table 12. Table of indices of fit for Model 1 based on the data from Sample 1 (N=48)

Sample	N	Maximum Likelihood X^2	GFI	CFI	Mardia's Coefficient	Normalized Estimate[1]
1	48	$X^2(6)=4.151$, $p=.65627$	.965	1.00	-1.8908	-0.7829*

[1] The normalized estimate represents a z score which tests the significance of the coefficient based on the expected value as per the assumption of a normal distribution.

* $p > .05$ two-tailed test ($[z] > 1.96$)

the data (Byrne, 1994; Hull, Lehn, & Tedlie, 1991: Kline, 1991).

Individual path coefficients were tested for significance by the EQS program and considered significant if $p < .05$, two-tailed (z-test).

Results of the Path Analysis

In terms of overall fit, the model in Figure 2 fits the data from Sample 1 quite well. As shown in Table 12, the indices of overall fit were very good. X^2 for the hypothesized model was small and non-significant, $X^2(6, N=48)=4.151$, $p=.65627$. The GFI was .965 and the CFI was 1.00.

The path coefficients between variables are shown in Figure 2. The paths between trait anxiety and state anxiety, trait anxiety and hostility, and hostility and guilt were significant ($p < .05$). However, the path between combat exposure and trait anxiety was nonsignificant ($p > .05$) and the sign of the coefficient was also in the opposite direction to that predicted.

EQS provides two modification indices that can suggest ways of improving the fit of a model. The multivariate LaGrange Multiplier Test tells you how much the X^2 for the model will be improved by freeing each of the fixed parameters, the zero paths between variables, in the model. Thus, the multivariate LaGrange Multiplier Test suggests possible paths to add to the model to improve its fit. The Wald Test assesses whether sets of free parameters in the model can be simultaneously set to zero without substantial loss in model fit (Byrne, 1994) i.e. it tests multivariately for redundant paths in the model.

While the LaGrange Multiplier Test did not indicate adding any paths to the model

and the Wald test did not suggest dropping any paths, findings from previous research suggested reasons that could account for the lack of significance in the path from combat exposure to trait anxiety. On the basis of these findings, which will be discussed in the next section, it was decided to modify Model 1 and to assess the fit of this second model.

Model Respecification

In Model 1, the coefficient for the path from combat exposure to trait anxiety was not significantly different from zero and was opposite in direction to that predicted. A negative sign for this path coefficient was consistent with the data from Sample 1, given that there was a nonsignificant trend for PTSD subjects, who were significantly higher in trait anxiety than control subjects, to report experiencing less combat exposure than control subjects.

DSM-IV (1994) redefines the nature of the stressor responsible for PTSD. In this latest edition of the Diagnostic and Statistical Manual, emphasis is shifted away from the severity of the stressor to a mixture of exposure to a traumatic event and an individual's reaction to it. This new definition indicates that the person's perception of the trauma is equally important in determining the stressor's impact and the production of PTSD symptoms as the objective severity of the stressor itself (Tomb, 1994).

The Combat Exposure Scale, which was used in Model 1 to represent exposure to a traumatic event, requires the subject to indicate whether they experienced particular events during the Vietnam War. The scale does not directly assess the subject's reaction to the events nor does it directly assess their perception of what happened.

In addition to the issue of perception of traumatic events, Friedman, Schnurr, and McDonagh-Coyle (1994) state that the type of combat experiences an individual has, as well as the amount of combat experience they have are important risk factors for PTSD. Indeed, a number of studies indicate that certain aspects of the combat experience are more directly related to the development of PTSD than others. Being a member of a unit patrol in Vietnam (Green, Grace, Lindy, Gleser, & Leonard, 1990), being exposed to grotesque and mutilating death (Green, Lindy, Grace, & Gleser, 1989), and being wounded or injured (Friedman et al., 1994) are all significant risk factors for the development of PTSD.

To take these findings into account, a second model, as shown in Figure 3, was constructed. In this model, factor analysis was used to create a latent variable of trauma from PTSD diagnosis, a categorical variable, and CES score. This factor represents a reaction to combat exposure and captures the variance in PTSD accounted for by combat exposure. Thus, an event can be seen as traumatic by some but not by others. By creating such a factor it is possible to account for situations in which individuals high in combat exposure are low in PTSD symptoms and individuals low in combat exposure are high in PTSD symptoms.

In Model 2, a path from the trauma factor to trait anxiety is predicted based on the same justifications for the combat exposure to trait anxiety path in Model 1. A positive coefficient is predicted for this path. The other paths in Model 2 are identical to those presented in Model 1. Model 2 was tested with the data from Sample 1 (N=48).

Mardia's coefficient, shown in Table 13, indicated an absence of multivariate kurtosis.

All of the beta weights in this model were assessed as free parameters except for the factor loading of PTSD diagnosis onto trauma. The value of this factor loading was set at 1.0 to establish the scale of the latent variable. Given that PTSD diagnosis was a categorical variable, with values of either 0 or 1, Model 2 was assessed with the EQS program using polychoric correlation and an arbitrary distribution generalized least squares (AGLS) method as per Lee, Poon, & Bentler (1990). The correlation matrix for the variables in this model is shown in Appendix F.

The indices of goodness of fit for Model 2 are shown in Table 13. They included the Yuan-Bentler corrected AGLS chi-square for the hypothesized mode. This index is a test of the deviation of the predicted covariance matrix for the variables from the observed covariance matrix and is derived from the estimated parameters of the model. Nonsignificant values of the Yuan-Bentler corrected chi square indicate a good overall fit of the model to the data. Also included are the AGLS adjusted fit index and the CFI. The adjusted AGLS fit index is comparable to an overall multivariate R^2. Its values range from 0 to 1, with values close to 1 suggesting a good fit of the model to the data.

Individual path coefficients were tested for significance by the EQS program and considered significant if $p < .05$, two-tailed and $[z] > 1.96$.

The Modified Model: Results of Path Analysis and Testing for Replication in a Second Sample

In terms of overall fit, the model in Figure 3 fits the data from Sample 1 quite well.

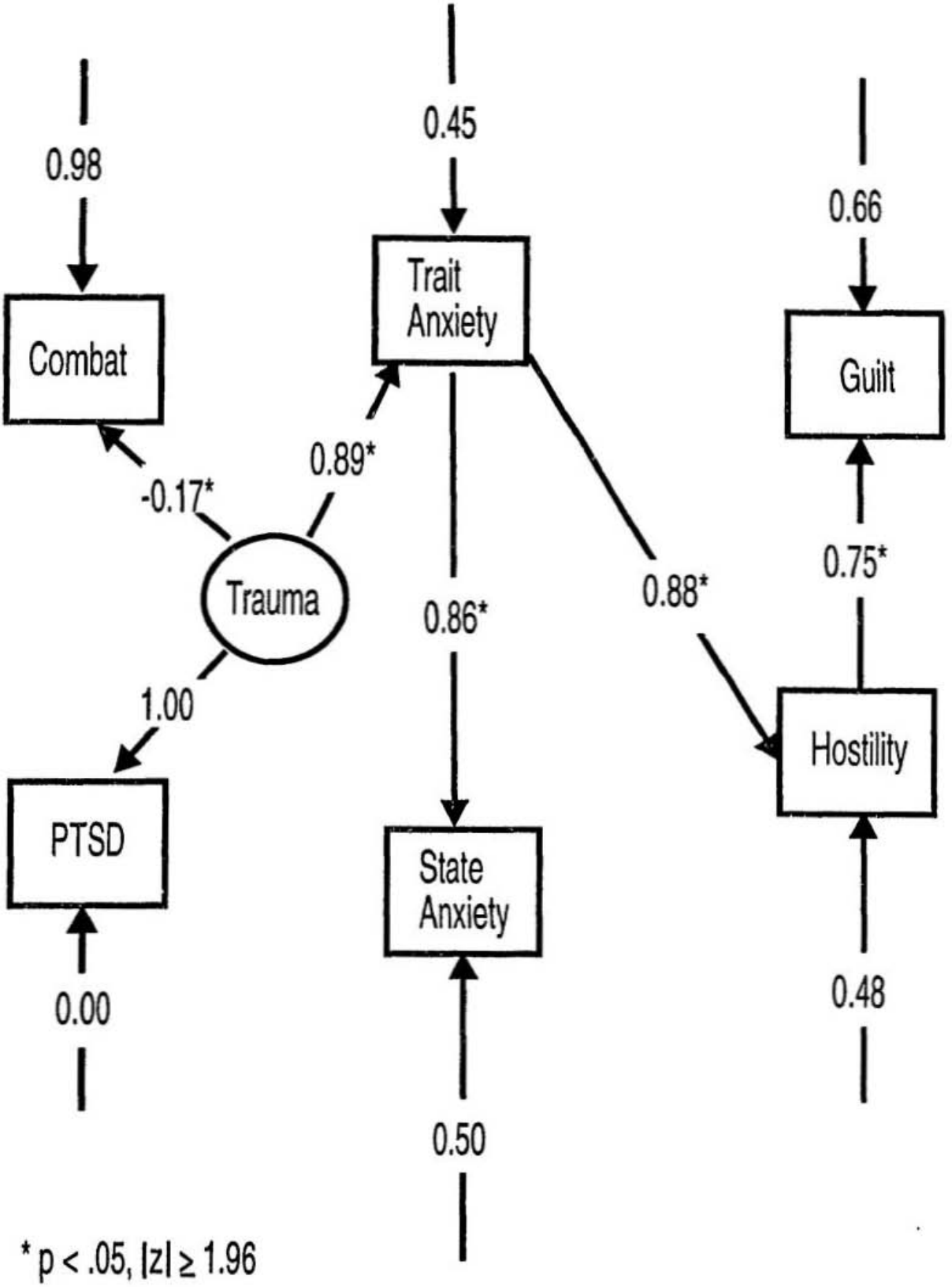

Figure 3. Standardized solution of Model 2, data from Sample 1 (N=48)

Table 13. Table of indices of overall goodness of fit for all models.

Model	Sample	N	Yuan Bentler Corrected AGLS X^2	AGLS Adjusted Fit	CFI	Mardia's Coefficient	Normalized Estimate[1]
2	1	48	$X^2(10)=1.379$, p=.99927	1.000	1.000	-2.3736	-0.8392*
	2	25	$X^2(10)=9.218$, p=.51159	0.999	1.000	-2.7512	-0.7020*
AM2a	1	48	$X^2(10)=1.950$, p=.99671	1.000	1.000	-2.3736	-0.8392*
AM2a	2	25	$X^2(10)=7.712$, p=.65697	1.000	0.999	-2.7512	-0.7020*
AM2b	1	48	$X^2(10)=1.716$, p=.99809	1.000	1.000	-2.3736	-0.8392*
AM2b	2	25	$X^2(10)=7.751$, p=.67064	1.000	0.999	-2.7512	-0.7020*
AM2c	1	48	$X^2(9)=1.350$, p=.99811	1.000	1.000	-2.3736	-0.8392*
AM2c	2	25	$X^2(9)=6.629$, p=.67652	1.000	0.999	-2.7512	-0.7020*
AM2d	1	48	$X^2(9)=1.350$, p=.99811	1.000	1.000	-2.3736	-0.8392*
AM2d	2	25	$X^2(9)=6.629$, p=.67572	1.000	0.999	-2.7512	-0.7020*
3	2	25	$X^2(10)=7.192$, p=.70712	0.999	1.000	-2.1123	-0.5390*
AM 3a	2	25	$X^2(10)=7.712$, p=.65697	0.999	1.000	-2.1123	-0.5390*
AM 3b	2	25	$X^2(10)=10.098$, p=.43194	0.998	1.000	-2.1123	-0.5390*

[1] The normalized estimate represents a z score which tests the significance of the coefficient based on the expected value as per the assumption of a normal distribution.
* p>.05 two-tailed test ([z]>1.96)

Table 14. Indices of fit for the simultaneous model comparisons.

Model	Yuan-Bentler Corrected AGLS X^2	CFI
2	$X^2(10,N=73)=21.092$,p=.68746	1.00
AM 2a	$X^2(10,N=73)=21.212$,p=.68072	1.00
AM 2b	$X^2(10,N=73)=21.357$,p=.67256	1.00

As shown in Table 13, the indices of overall fit were very good. The hypothesized model chi-square was small and nonsignificant, $X^2(10,N=48)=1.420$, $p=.99916$. The adjusted AGLS fit index was 1.00 and the CFI was 1.00. In comparison to Model 1, these indices suggest that Model 2 fit the data from Sample 1 much better. If one calculates a CFI using Model 1 as the null model and Model 2 as the test model, the CFI equals 1.00, also indicating a better fit of Model 2 to the data.

The path coefficients between variables are shown in Figure 3. All of the path coefficients were significantly different from zero ($p<.05$ and $[z]>1.96$). The LaGrange Multiplier test did not suggest adding any additional paths nor did the Wald test suggest dropping any of the paths in the model.

Hoyle and Panter (1995) note that post-hoc modifications of a model should not be taken seriously with sample sizes under 800, unless they replicate in an independent sample. Therefore, Model 2 was tested with the data from Sample 2 in order to assess the replicability of the model. The correlation matrix for the variables in this model is shown in Appendix F. Mardia's coefficient, shown in Table 13 (Model 2, Sample 2), indicated that none of the variables exhibited significant multivariate kurtosis.

The resulting indices of fit for Model 2, based on the data from Sample 2, are shown in Table 13. All indices were very good, indicating an adequate fit of the model to the data from Sample 2. The Yuan-Bentler corrected AGLS chi-square was small and nonsignificant ($X^2(10,N=25)=9.22$, $p=.5116$. The adjusted AGLS fit index was .9999 and the CFI was 1.00.

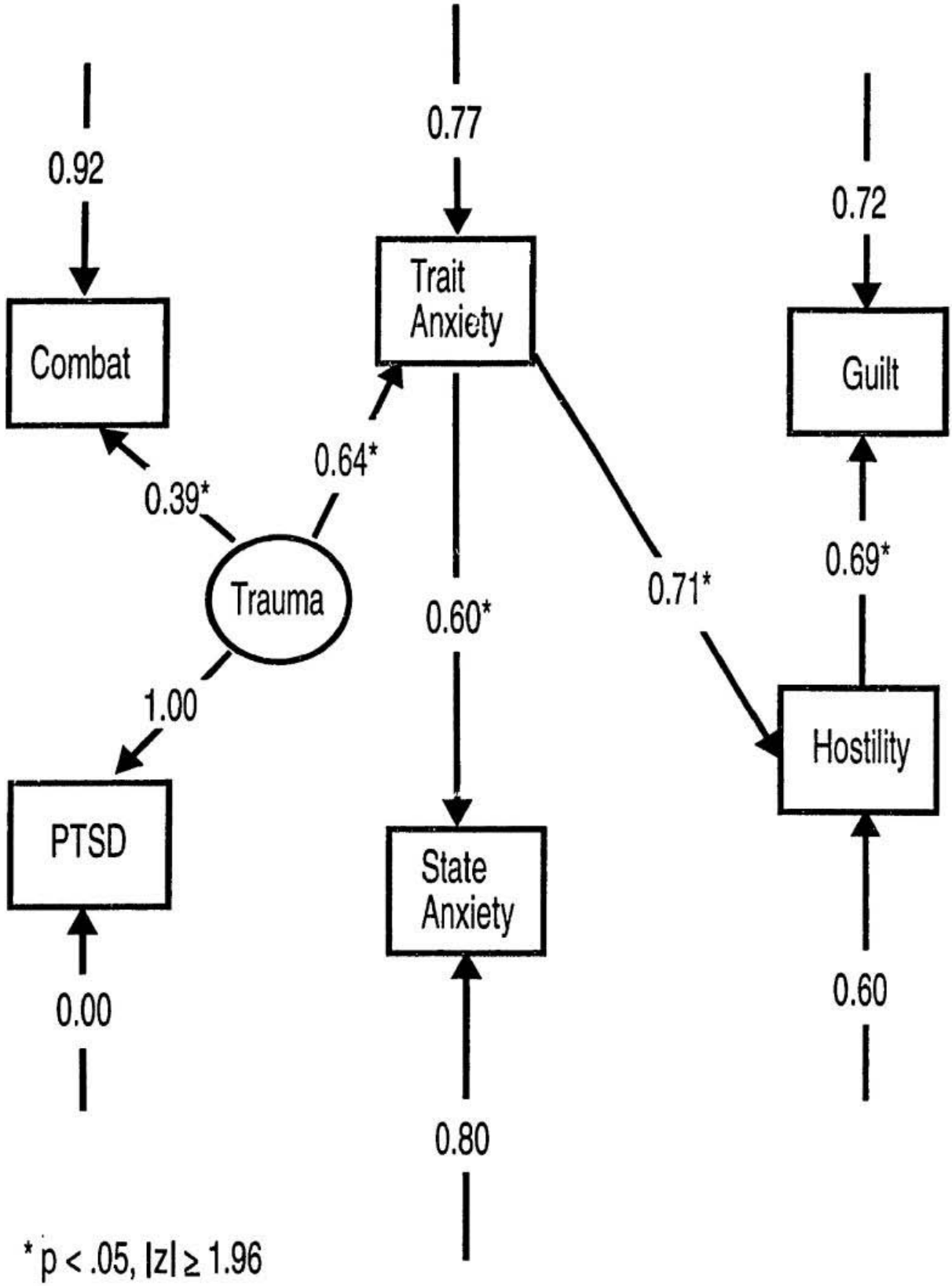

Figure 4. Standardized solution of Model 2, data from Sample 2 (N=25)

As can be seen from Figure 4, all path coefficients between variables were significant ($p < .05$). The LaGrange Multiplier and Wald tests did not indicate adding or dropping any parameters to improve the fit of Model 2.

The EQS program also allows the simultaneous comparison of a model in two or more data samples. In this type of analysis, the EQS program uses the LaGrange Multiplier Test to test whether significant differences exist between the path coefficients in the model based on Sample 1 versus the model based on Sample 2. The LaGrange Multiplier Test tests the constraint hypothesis that the observed path coefficients in the model based on Sample 1 are equal to those in the model based on Sample 2.

A Yuan-Bentler corrected chi-square and a CFI can be calculated for a multigroup model analysis. These are shown in Table 14. They represent a comparison of a model based on the model estimates for each of the respective data sets and the relevant equality constraints with the null model. The Yuan-Bentler corrected chi-square for the present multigroup model was small and nonsignificant ($X^2(10, N=73)=21.092$, $p=.68746$ and the CFI was 1.000, indicating a very good fit of the model to the two data sets.

Examination of the LaGrange Multiplier chi-squares showed that all but two of the constraints held across the two groups. This reflected the noninvariance, across the two samples, of the factor loading of combat onto the trauma factor and the coefficient for the path from trait to state anxiety.

Alternate Model 2a

As previously stated, the testing of alternative models is considered an integral part

of the modeling process (Bollen & Long, 1992; Hull et al., 1991; Raykov et al., 1991). We have thus reviewed the relevant research and created several alternative models that may also represent the relationships between anxiety, hostility, and guilt in PTSD. The first of these models, Alternate Model 2a, is shown in Figure 5.

In Alternate Model 2a, a path is predicted from trauma to state anxiety. This path is predicted from Shalev et al.'s (1996) study on individuals exposed to a variety of civilian traumas. In their study, Shalev et al. (1996) report that, at one week posttrauma, subjects who developed PTSD as a result of their exposure to trauma had significantly higher levels of state anxiety, but not trait anxiety, than subjects who did not develop PTSD. Subjects who developed PTSD also had higher levels of state anxiety at 6 months posttrauma than subjects who did not develop PTSD. Unfortunately, Shalev et al. (1996) did not assess trait anxiety at 6 months posttrauma. This finding of increased state but not trait anxiety would suggest that exposure to trauma has a direct influence on state anxiety. It would not be unreasonable to suggest that experiencing high levels of state anxiety over an extended period of time might result in an individual becoming more anxious in general, i.e having a higher level of trait anxiety. This would account for the high levels of trait anxiety reported in studies that tested individuals with PTSD many years after their exposure to a traumatic event (Hovens et al., 1994; Hovens et al., 1992; Kuhne, et al., 1993; Orr et al., 1995; Sutker, et al., 1991). Thus, a path from state to trait anxiety is predicted in this model.

Alternate Model 2a includes the trauma factor. The paths from trait anxiety to

hostility and hostility to guilt have been left the same as in the hypothesized model as we were unable to find any evidence in the current literature to suggest alternative relationships among these variables.

Alternate Model 2a was tested with the data from Sample 1 (N=48). Mardia's coefficient, shown in Table 13 (AM 2a, Sample 1), indicated the absence of multivariate kurtosis.

All of the beta weights in this model were assessed as free parameters except for the factor loading of PTSD diagnosis on to trauma which was set at 1.0. The correlation matrix for the variables in this model is shown in Appendix F.

The indices of fit for Alternate Model 2a are shown in Table 13. Individual path coefficients were tested for signficance by the EQS program and considered significant if $p < .05$ (z-test).

In terms of overall fit, the indices in Table 13 show that Alternate Model 2a fits the data from Sample 1 quite well. The Yuan-Bentler adjusted chi-square was small and nonsignificant, $X^2(10, N=48) = 1.950$, $p = .99671$. The adjusted AGLS fit index was 1.00 and the CFI was 1.00. These fit indices are very similar to those obtained with Model 2. A CFI of .07 is obtained when using Model 2 as the null model and Alternate Model 2a as the test model. This indicates that, in terms of overall fit, the two models are equivalent.

The path coefficients between variables are shown in Figure 5. All of the path coefficients were signficantly different from zero ($p < .05$ and $[z] > 1.96$). The LaGrange

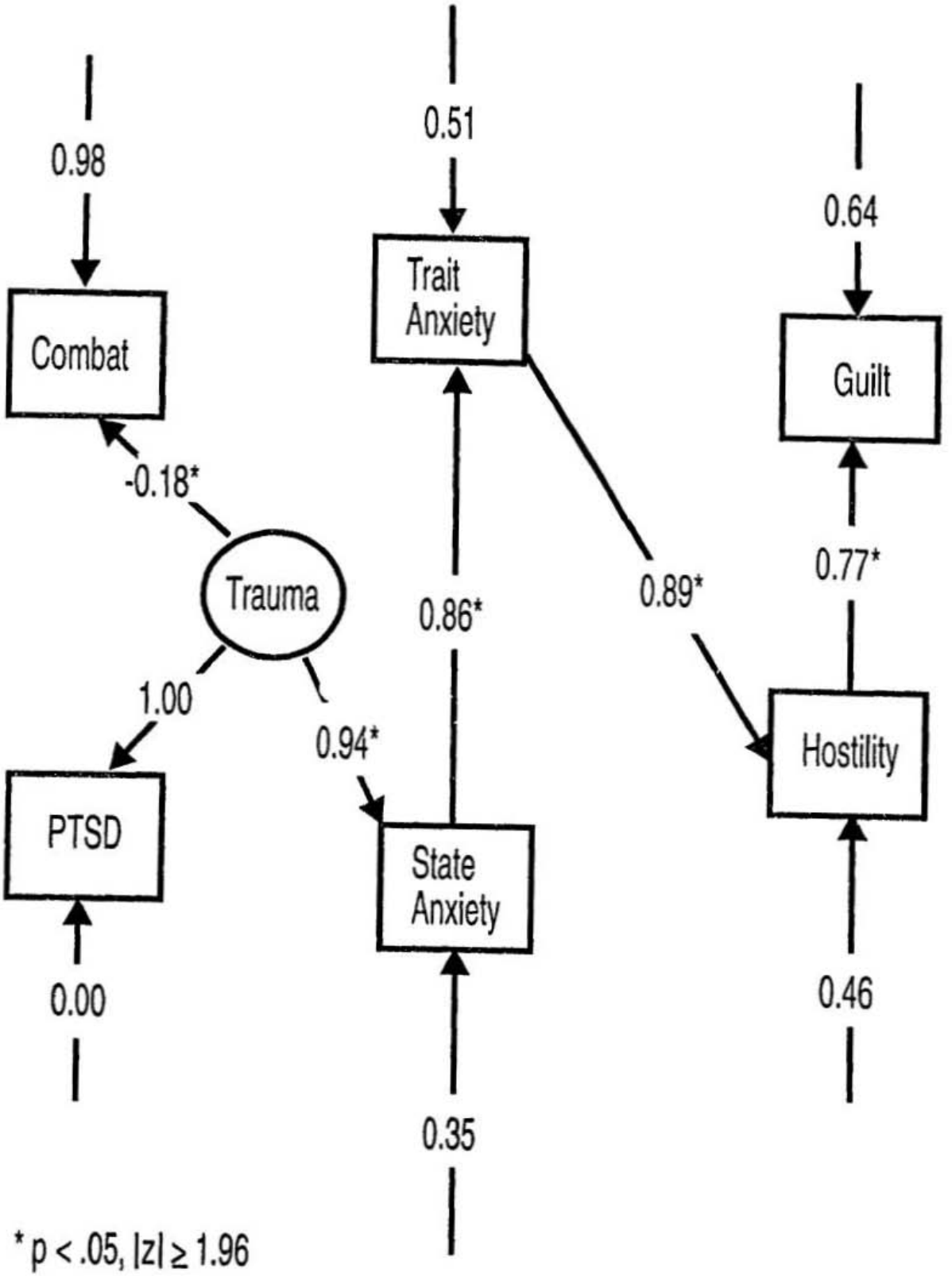

Figure 5. Standardized solution of Alternate Model 2a, data from Sample 1 (N=48)

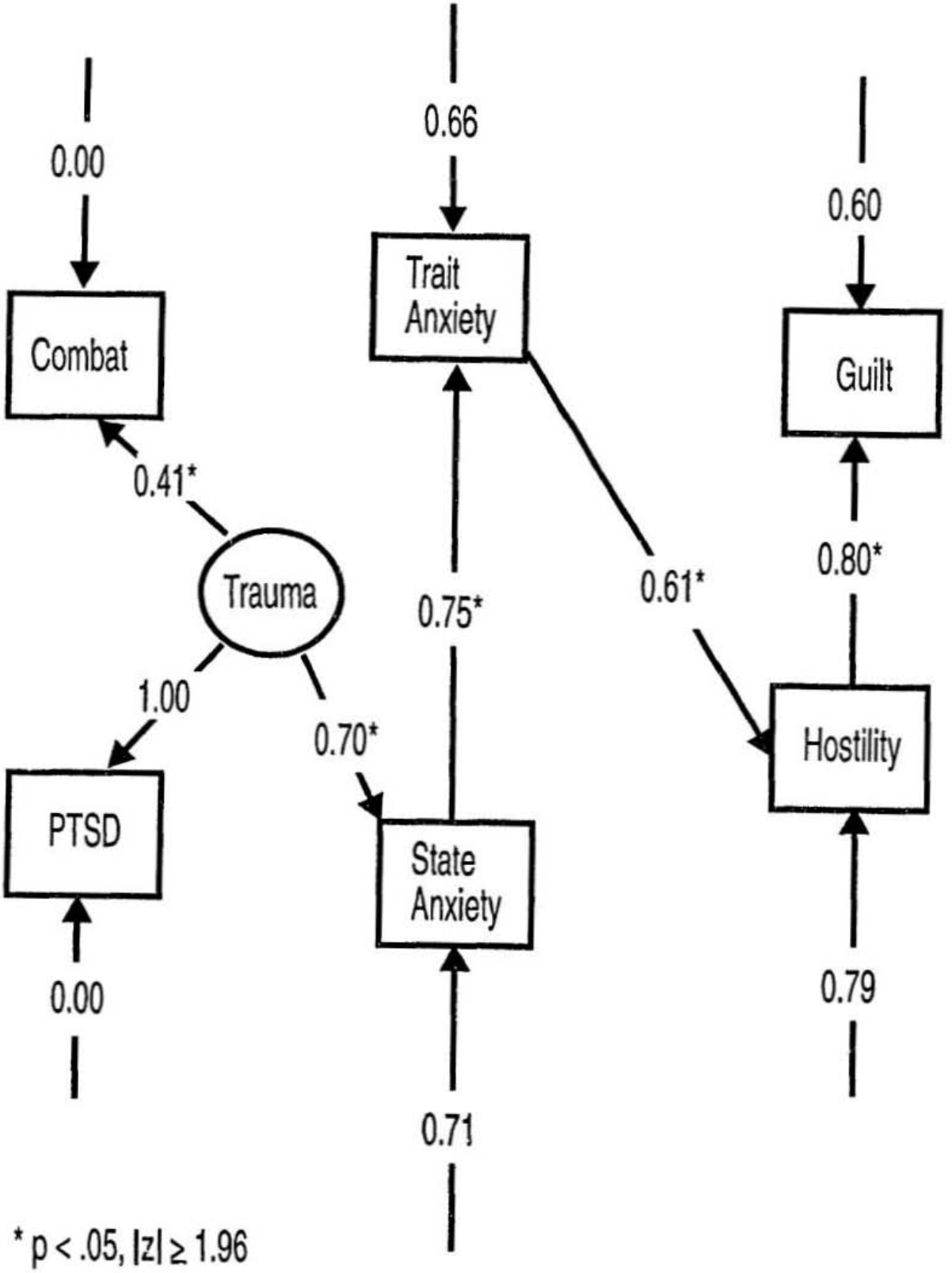

Figure 6. Standardized solution of Alternate Model 2a, data from Sample 2 (N=25)

Multiplier test did not suggest adding any additional paths nor did the Wald test suggest dropping any of the paths in the model.

Alternate Model 2a was also tested with the data from Sample 2. The correlation matrix for the variables in this model is shown in Appendix F. Mardia's coefficent, as shown in Table 13 (AM 2a, Sample 2), indicated the absence of multivariate kurtosis.

Table 13 shows the resulting indices of fit for Alternate Model 2a based on the data from Sample 2. All indices of fit are quite good, indicating that Alternate Model 2a adequately fits the data from Sample 2. The Yuan-Bentler adjusted chi-square was small and nonsignificant (X^2 (10,N=25)=7.712, p=.65697. The adjusted AGLS fit index was 0.999 and the CFI was 0.999. A CFI of .311 is obtained when using Model 2 as the null model and Alternate Model 2a as the test model. This suggests that the two models fit the data equally well.

As shown in Figure 6, all path coefficients between variables were significant ($p < .05$). The LaGrange Multiplier and Wald tests did not suggest adding or dropping any parameters to improve the fit of Alternate Model 2a.

A simultaneous comparison of Alternate Model 2a in Samples 1 and 2 resulted in a small and nonsignificant Yuan-Bentler corrected chi-square (X^2 (10, N=73) = 21.212, p=.68072) and a CFI of 1.00. These indices are shown in Table 14 and indicate that Alternate Model 2a fits the data from both samples very well.

Examination of the LaGrange Multiplier chi-squares showed that all but two of the constraints held across the two groups. This reflected the noninvariance, across the two

samples, of the factor loading of combat onto the trauma factor and the coefficient for the path from state anxiety to trait anxiety.

Alternate Model 2b

In Alternate Model 2b, as shown in Figure 7, a path goes directly from trauma to trait anxiety and a path goes from trauma to state anxiety. This model suggests that trauma increases both state anxiety and trait anxiety. An immediate increase in state anxiety as a result of trauma is predicted from Shalev et al. (1996) as discussed in our first alternative model. Although Shalev et al. (1996) did not find elevated levels of trait anxiety in their subjects at one week posttrauma, high trait anxiety levels have been consistently found in combat veterans who have suffered from PTSD for almost twenty years (Hovens et al., 1994; Hovens et al., 1992; Orr et al., 1995). Thus, it is possible that while exposure to trauma has an immediate effect on an individual's level of state anxiety, its effect on trait anxiety is slower and thus increases in trait anxiety may not appear until the individual has had PTSD for some time. Indeed, Rothbaum, Foa, Riggs, Murdock, and Walsh (1992) have shown that the symptom profile that is observed shortly after the trauma may be quite different from that observed later on. This model differs from Alternate Model 2a in that it suggests that the increases in trait anxiety are the direct result of exposure to trauma and not the result of the increased levels of state anxiety that occur with exposure to trauma.

As is the case with the first alternative model, the paths between the other variables are the same as those predicted in the original hypothesized model.

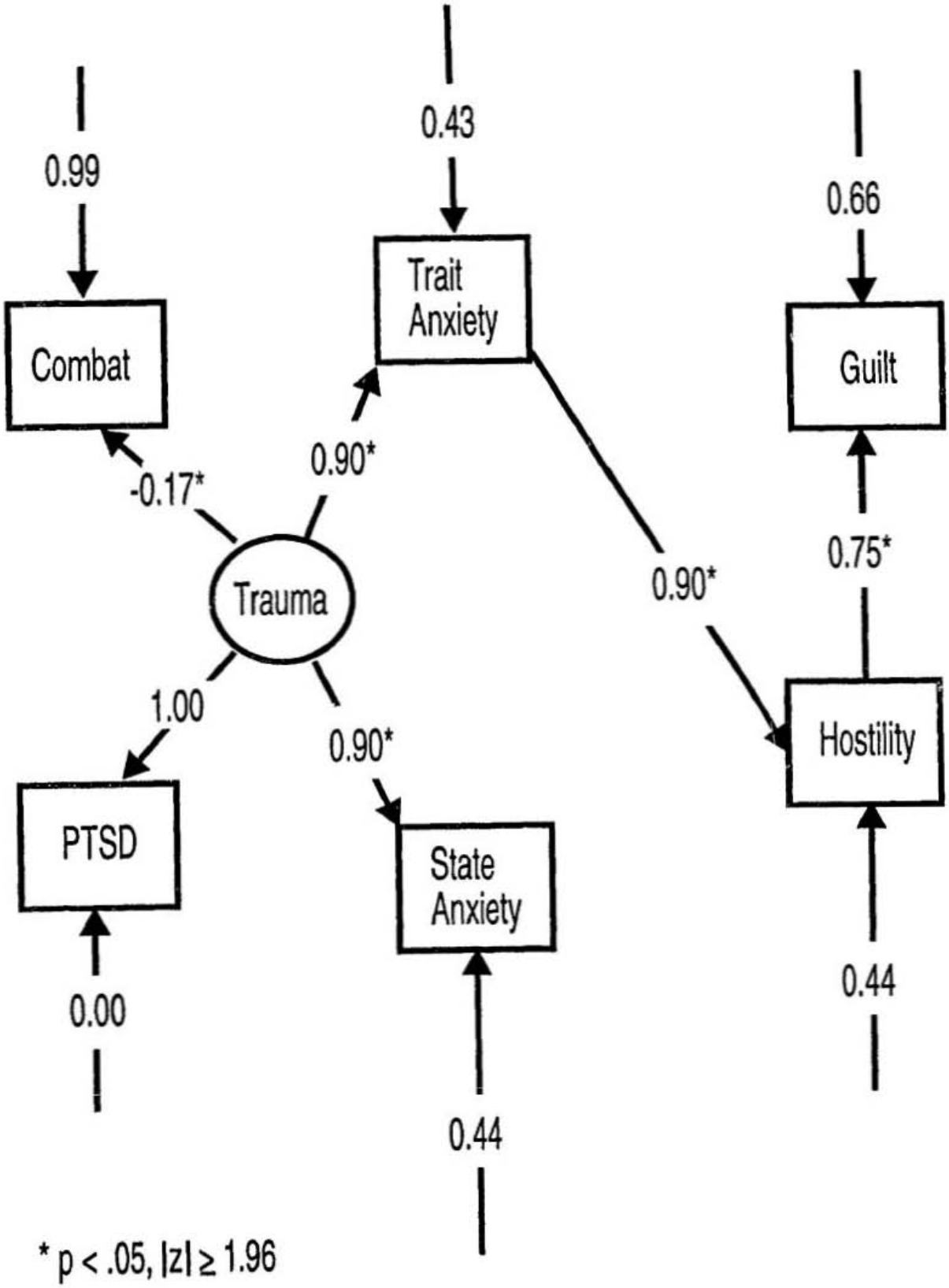

Figure 7. Standardized solution of Alternate Model 2b, data from Sample 1 (N=48)

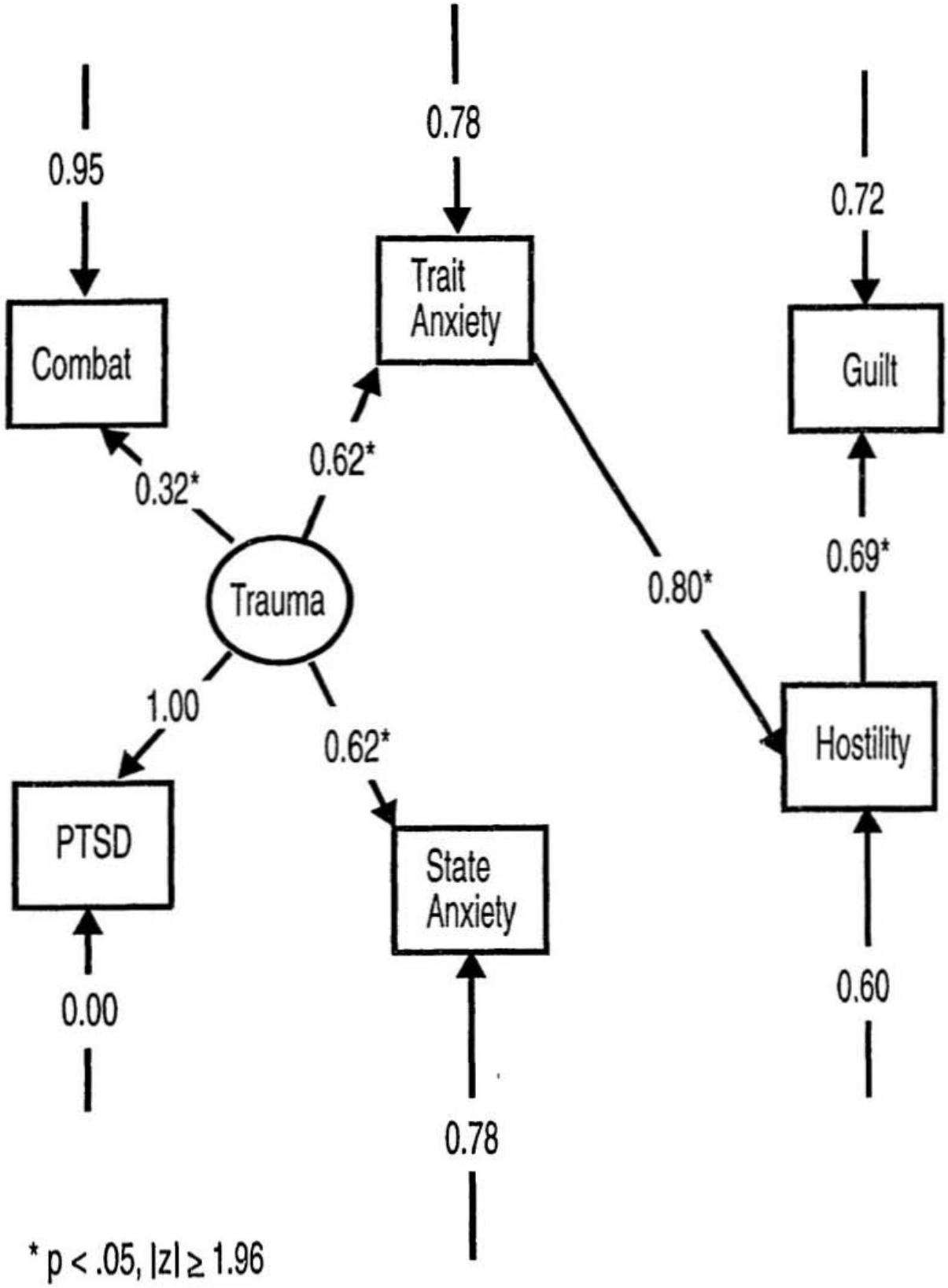

Figure 8. Standardized solution of Alternate Model 2b, data from Sample 2 (N=25)

Alternate Model 2b was tested with the data from Sample 1 (N=48). Mardia's coefficient, shown in Table 13 (AM 2b, Sample 1), indicated the absence of multivariate kurtosis.

All of the beta weights in this model were assessed as free parameters except for the factor loading of PTSD diagnosis on to trauma which was set at 1.0. The correlation matrix for the variables in this model is shown in Appendix F.

The indices of fit for Alternate Model 2b are shown in Table 13. Individual path coefficients were tested for signficance by the EQS program and considered significant if $p < .05$ (z-test).

In terms of overall fit, the indices in Table 13 show that Alternate Model 2b fits the data from Sample 1 quite well. The Yuan-Bentler adjusted chi-square was small and nonsignificant, $X^2(10, N=48) = 1.716$, $p = .99809$. The adjusted AGLS fit index was 1.00 and the CFI was 1.00. These fit indices are very similar to those obtained with Model 2. A CFI of .04 is obtained when using Model 2 as the null model and Alternate Model 2b as the test model. This indicates that the two models fit the data equally well.

The path coefficients between variables are shown in Figure 7. All of the path coefficients were signficantly different from zero ($p < .05$ and $[z] > 1.96$). The LaGrange Multiplier test did not suggest adding any additional paths nor did the Wald test suggest dropping any of the paths in the model.

Alternate Model 2b was also tested with the data from Sample 2. The correlation

matrix for the variables in this model is shown in Appendix F. Mardia's coefficent, as shown in Table 13 (AM 2b, Sample 2), indicated the absence of multivariate kurtosis.

Table 13 shows the resulting indices of fit for Alternate Model 2b based on the data from Sample 2. All indices of fit are quite good, indicating that Alternate Model 2b adequately fits the data from Sample 2. The Yuan-Bentler adjusted chi-square was small and nonsignificant (X^2 (10,N=25)=7.751, p=.67064. The adjusted AGLS fit index was 0.999 and the CFI was 0.999. A CFI of .323 is obtained when using Model 2 as the null model and Alternate Model 2b as the test model. This suggests that the two models fit the data from Sample 2 equally well.

As shown in Figure 8, all path coefficients between variables were significant ($p < .05$). The LaGrange Multiplier and Wald tests did not suggest adding of dropping any parameters to improve the fit of Alternate Model 2b.

A simultaneous comparison of Alternate Model 2b in Samples 1 and 2 resulted in a small and nonsignificant Yuan-Bentler corrected chi-square (X^2(10,N=73)=21.357, p=.67256) and a CFI of 1.00. These indices are shown in Table 14 and indicate that Alternate Model 2b fits the data from both samples very well.

Examination of the LaGrange Multiplier chi-squares showed that all but two of the constraints held across the two groups. This reflected the noninvariance, across the two samples, of the factor loading of combat onto the trauma factor and the coefficient for the path from the trauma factor to state anxiety.

Alternate Model 2c

Alternate Model 2c combines the information from Model 2 and Alternate Model 2a. In this model, as shown in Figure 9, a path goes from trauma to state anxiety and trauma to trait anxiety. In addition a path goes from trait anxiety to state anxiety. Thus, this model suggests, following the rationales presented with Alternate Model 2a, that trauma increases both trait and state anxiety. Increased levels of trait anxiety can then increase state anxiety, as suggested in Model 2.

As is the case with the other alternative models, the paths between the other variables are the same as those predicted in the original hypothesized model.

Alternate Model 2c was tested with the data from Sample 1 (N=48). Mardia's coefficient, shown in Table 13 (AM 2c, Sample 1), indicated the absence of multivariate kurtosis.

All of the beta weights in this model were assessed as free parameters except for the factor loading of PTSD diagnosis on to trauma which was set at 1.0. The correlation matrix for the variables in this model is shown in Appendix F.

The indices of fit for Alternate Model 2c are shown in Table 13. Individual path coefficients were tested for signficance by the EQS program and considered significant if $p < .05$ (z-test).

In terms of overall fit, the indices in Table 13 show that Alternate Model 2c fits the data from Sample 1 quite well. The Yuan-Bentler adjusted chi-square was small and nonsignificant, $X^2(9, N=48) = 1.350$, $p = .99811$. The adjusted AGLS fit index was 1.00

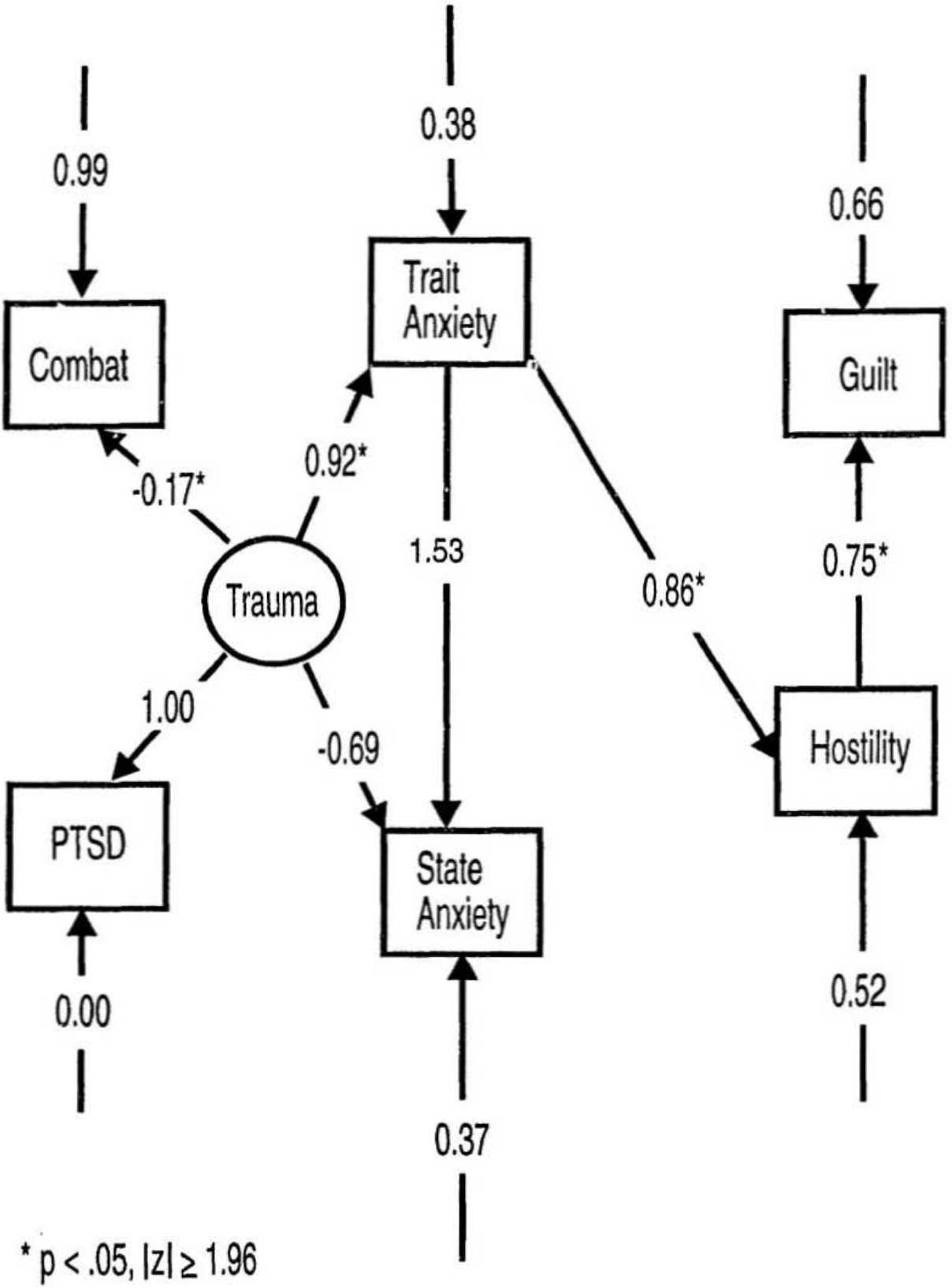

Figure 9. Standardized solution of Alternate Model 2c, data from Sample 1 (N=48)

and the CFI was 1.00. These fit indices are very similar to those obtained with Model 2. A CFI of .04 is obtained when using Model 2 as the null model and Alternate Model 2c as the test model. This indicates that the two models fit the data equally well.

The path coefficients between variables are shown in Figure 9. All of the path coefficients were signficantly different from zero ($p < .05$) except for the path from trauma to state anxiety and the path from trait anxiety to state anxiety. As, for the purposes of the present study, significant path coefficients are an estimate of the goodness of fit of the model, Alternate Model 2c was considered to be an inappropriate model of the relationships among the relevant variables and was not tested in Sample 2.

Alternate Model 2d

Alternate Model 2d combines the information presented in Alternate Model 2a and Alternate Model 2b. In this model, a path goes from trauma to state anxiety and a path goes from trauma to trait anxiety. In addition, a path runs from state to trait. This model suggests that exposure to trauma increases trait and state anxiety. It also suggests that increased levels of state anxiety contribute to the increase in trait anxiety. This is consistent with the research by Shalev et al. (1996) presented in Alternate Model 2a.

As is the case with the other alternative models, the paths between the other variables are the same as those predicted in the original hypothesized model.

Alternate Model 2d was tested with the data from Sample 1 ($N=48$). Mardia's coefficient, shown in Table 13 (AM 2d, Sample 1), indicated the absence of multivariate kurtosis.

All of the beta weights in this model were assessed as free parameters except for the factor loading of PTSD diagnosis on to trauma which was set at 1.0. The correlation matrix for the variables in this model is shown in Appendix F.

The indices of fit for Alternate Model 2d are shown in Table 13. Individual path coefficients were tested for signficance by the EQS program and considered significant if $p < .05$ (z-test).

In terms of overall fit, the indices in Table 13 show that Alternate Model 2d fits the data from Sample 1 quite well. The Yuan-Bentler adjusted chi-square was small and nonsignificant, $X^2(9,N=48)=1.350$, $p=.99811$. The adjusted AGLS fit index was 1.00 and the CFI was 1.00. These fit indices are very similar to those obtained with Model 2. A CFI of .04 is obtained when using Model 2 as the null model and Alternate Model 2d as the test model. This indicates that, in terms of overall fit, the two models are equivalent.

The path coefficients between variables are shown in Figure 10. All of the path coefficients were signficantly different from zero ($p < .05$) except for the path from trauma to trait anxiety and the path from state anxiety to trait anxiety. As, for the purposes of the present study, significant path coefficients are an estimate of the goodness of fit of the model, Alternate Model 2d was considered to be an inappropriate model of the relationships among the relevant variables and was not tested in Sample 2.

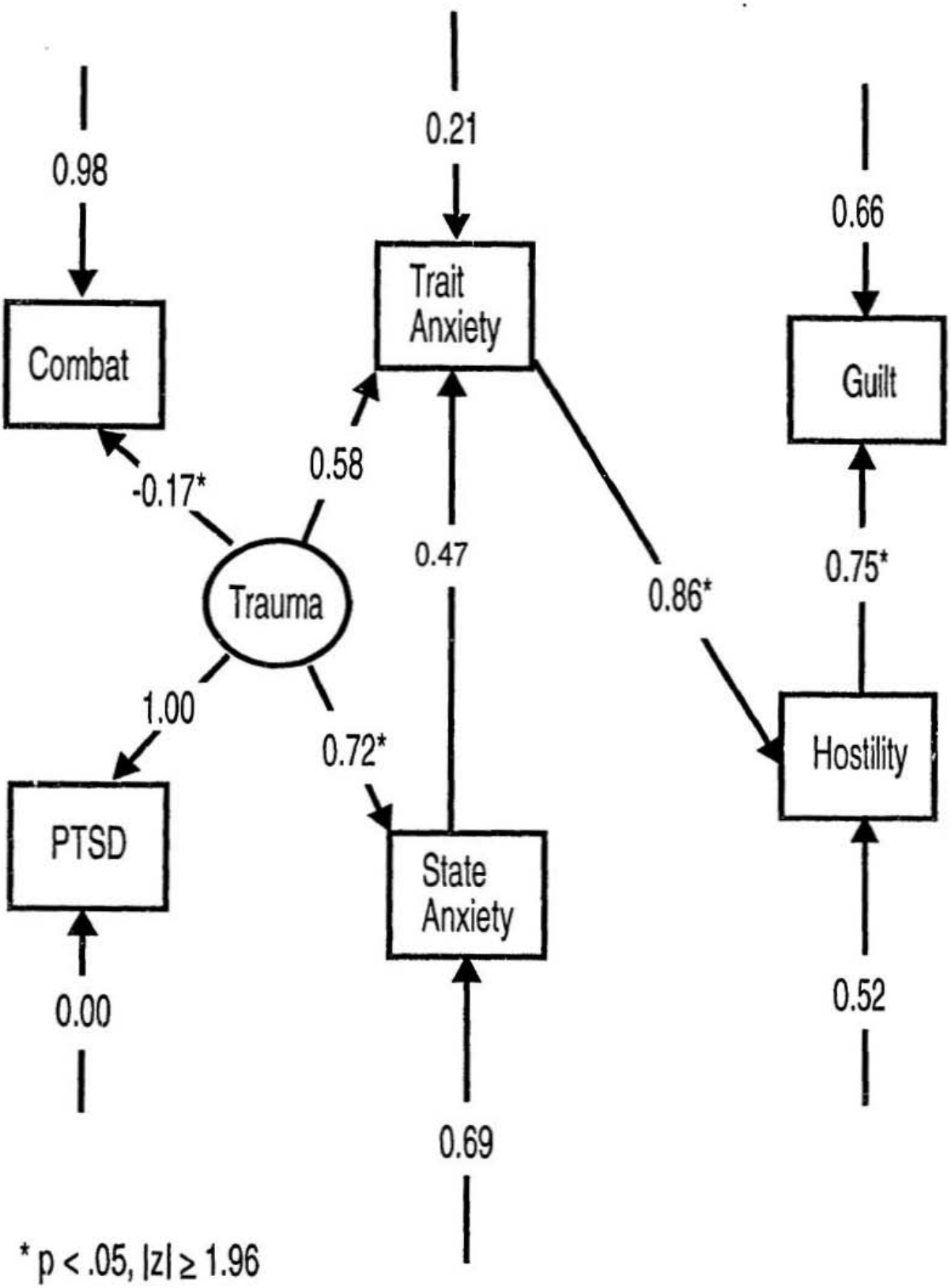

Figure 10. Standardized solution of Alternate Model 2d, data from Sample 1 (N=48)

Construction of Model 3: The Relationship of Startle To Anxiety in PTSD.

As an exaggerated startle response is a frequent symptom of PTSD in veterans (Keane, 1993), path analysis was used to examine the relationship between trauma, anxiety, hostility, and startle. The path diagram for this second hypothesized model is shown in Figure 11.

Model 3 is quite similar to Model 2 shown in Figures 3 and 4 with two exceptions. First, a path from state anxiety to EMG response average has been added. In humans, the acoustic startle reflex is measured as the magnitude of the eyeblink/ EMG response component of the reflex (Morgan et al. ,1995). Thus, in Model 3, startle response is represented by the average, across fifteen trials, of a subject's EMG response to a loud tone. The path from state anxiety to startle, EMG response, is predicted on the basis of a number of recent research findings which suggest that the exaggerated startle response observed in many patients with PTSD is analogous to the fear-potentiated startle response seen in rodents. Fear-potentiated startle in rodents occurs when the amplitude of the startle response is increased by the startle stimulus being presented in the presence of a cue, such as a light, that has previously been paired with a shock. It is believed that fear-potentiated startle is produced by the associative conditioning of a central fear state (Cook et al., 1991).

In humans, it has also been shown that fear "potentiates" the startle response. Cook et al. (1991), report that startle responses were enhanced in high fear healthy human subjects in comparison to low fear subjects. Fear was manipulated in the subjects by

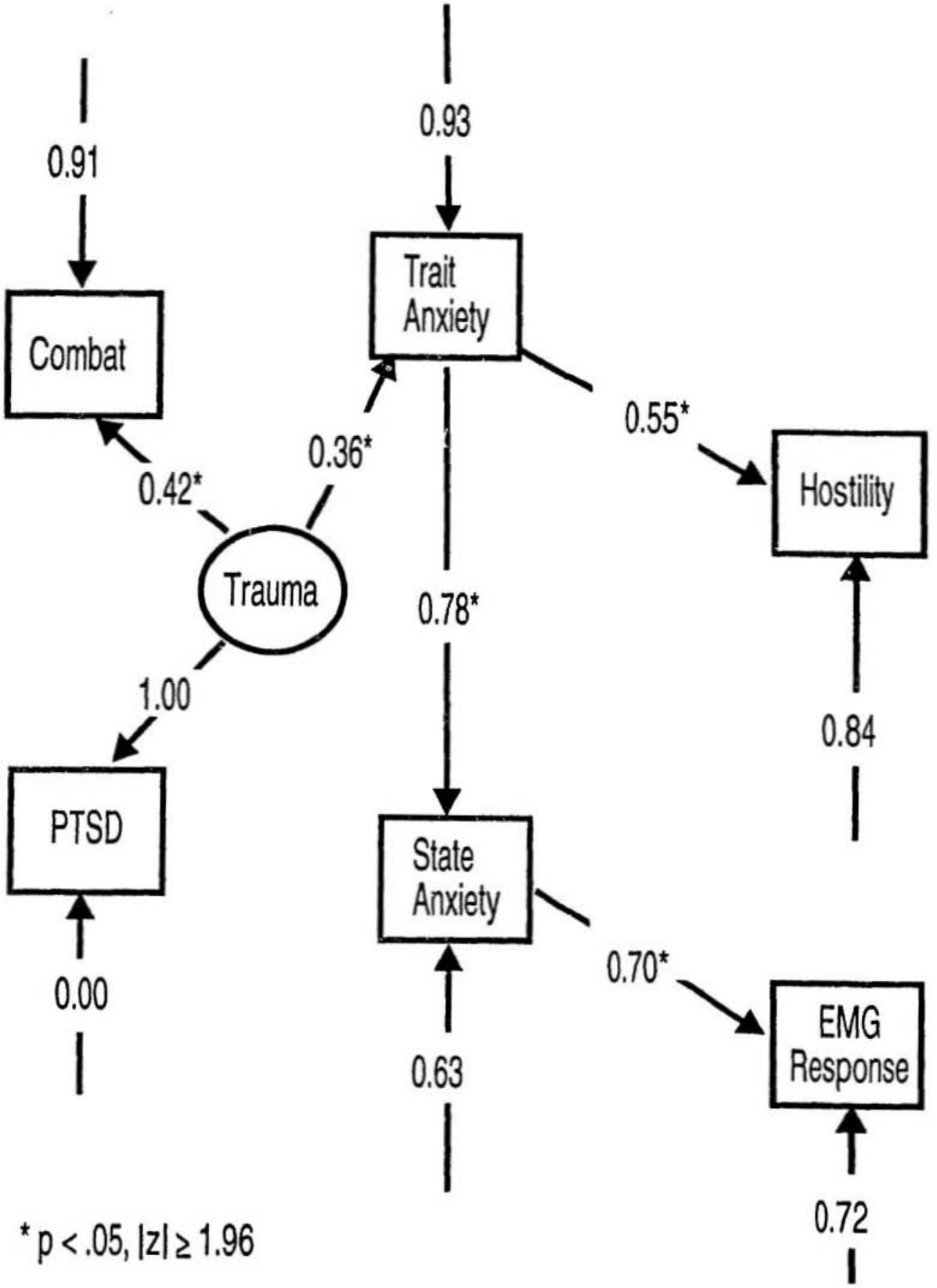

Figure 11. Standardized solution of Model 3, data from Sample 2 (N=25)

having them view negative and positive imagery. Grillon et al. (1993) assessed the role of individual differences in state and trait anxiety on baseline and fear potentiated startlein normal subjects. They found that baseline startle did not differ between high and low anxiety subjects but the magnitude of fear-potentiated startle was significantly larger in high anxiety subjects than low anxiety subjects. Trait anxiety did not account for individual differences in either baseline or fear-potentiated startle.

Morgan et al. (1995) have looked at the startle response and its relationship to trait and state anxiety in Vietnam veterans with PTSD. They report that veterans with PTSD had higher state anxiety scores and larger startle responses at baseline and during threat of a shock than age-matched, healthy controls. Morgan et al. (1995) suggest that the elevated levels of baseline startle found in their subjects with PTSD result from a greater conditioned emotional response in this group, triggered by the anticipation of shock, generalizing to the unfamiliar context in which the testing took place. Thus, the increased levels of startle are not the result of a chronic elevation of startle but rather are a result of the increased levels of state anxiety experienced by the subjects with PTSD .

Morgan, Grillon, Southwick, Davis, & Charney (1996) have also found significantly higher levels of acoustic startle response in Gulf War veterans with PTSD when compared to Gulf War veterans without PTSD and civilian controls. They state that the exaggerated startle responses seen in these veterans with PTSD may be the result of the experimental situation in which the subjects are tested producing higher levels of anxiety in the subjects with PTSD. Morgan et al. (1996) suggest that this increase in anxiety then

elevates startle magnitude in the PTSD subjects.

Based on the above findings, a path is predicted in Model 3 from state anxiety to startle, as represented by EMG response. This path supports the idea that increases in the magnitude of the startle response are the result of elevated levels of state anxiety, i.e., a fear-potentiated startle response. A positive coefficient is predicted for the path from state anxiety to EMG response.

Model 3 retains the trauma factor from Model 2 as well as the path from it to trait anxiety. It also keeps the path from trait to state anxiety and trait anxiety to hostility. However, the path from hostility to guilt has been dropped in Model 3. This path was dropped due to the fact that retaining it in the new model would result in the new model having too many parameters for analytic purposes.

Model 3: Method and Goodness of Fit Indices

Model 3 was tested with the data from Sample 2 (N=25). Unfortunately, physiological variables were unavailable for Sample 1, and it was not possible to assess the fit of Model 3 to that data sample. Mardia's coefficient, shown in Table 13 (Model 3, Sample 1), indicated the absence of multivariate kurtosis.

As with Model 2, all of the beta weights in Model 3 were assessed as free parameters except for the factor loading of PTSD diagnosis onto trauma, which was set at 1.0. Given that PTSD diagnosis was a categorical variable, with values of either 0 or 1, Model 3 was assessed with the EQS program using an arbitrary distribution generalized least squares (AGLS) method. The correlation matrix for the variables in this

model is shown in Appendix F.

The indices of goodness of fit for Model 3 are shown in Table 13. They included the AGLS chi-square for the hypothesized model, the AGLS adjusted fit index and the CFI.

Individual path coefficients were tested for significance by the EQS program and considered significant if $p<.05$, two-tailed and $[z]>1.96$.

Model 3: Results of Path Analysis

In terms of overall fit, Model 3, shown in Figure 11, fits the data from Sample 2 quite well. As shown in Table 13, the indices of overall fit were very good. The Yuan-Bentler adjusted chi-square was small and nonsignificant, $X^2(10,N=25)=7.192$, $p=.70712$. The adjusted AGLS fit index was .999 and the CFI was 1.00.

The path coefficients between variables are shown in Figure 11. All of the path coefficients were significantly different from zero ($p<.05$ and $[z]>1.96$). The LaGrange Multiplier test did not suggest adding any additional paths nor did the Wald test suggest dropping any of the paths in the model.

Alternate Model 3a

Given that two equivalent models were found for Model 2, it was decided to test similar models with Model 3.

Alternate Model 3a, as shown in Figure 12, incorporates the paths from Alternate Model 2a. Thus, there is a path from trauma to state anxiety and a path from state anxiety to trait anxiety. All other paths are the same as in Model 3, shown in Figure 11.

Alternate Model 3a was tested with the data from Sample 2 ($N=25$). As was the case

with Model 3, it was not possible to assess the fit of Alternate Model 3a in Sample 1. Mardia's coefficient, shown in Table 13 (AM 3a, Sample 1), indicated the absence of multivariate kurtosis.

As with Model 3, the beta weights in Alternate Model 3a were assessed as free parameters except for the factor loading of PTSD diagnosis onto trauma, which was set at 1.0. Given that PTSD diagnosis was a categorical variable, with values of either 0 or 1, Alternate Model 3a was assessed with the EQS program using an arbitrary distribution generalized least squares (AGLS) method. The correlation matrix for the variables in this model is shown in Appendix F.

The indices of goodness of fit for Alternate Model 3a are shown in Table 13. They included the Yuan-Bentler corrected AGLS chi-square for the hypothesized model, the AGLS adjusted fit index and the CFI.

Individual path coefficients were tested for significance by the EQS program and considered significant if $p < .05$, two-tailed and $[z] > 1.96$.

In terms of overall fit, Alternate Model 3a, shown in Figure 12, fits the data from Sample 2 quite well. As shown in Table 13, the indices of overall fit were very good. The Yuan-Bentler adjusted chi-square was small and nonsignificant, X^2 (10, $N=25$) $=7.712$, $p=.65697$. The adjusted AGLS fit index was .999 and the CFI was 1.00. A relative CFI of .164 was calculated using Model 3 as the null model and Alternate Model 3a as the hypothesized model. This indicates that Model 3 and Alternate Model 3a fit the data from Sample 2 equally well.

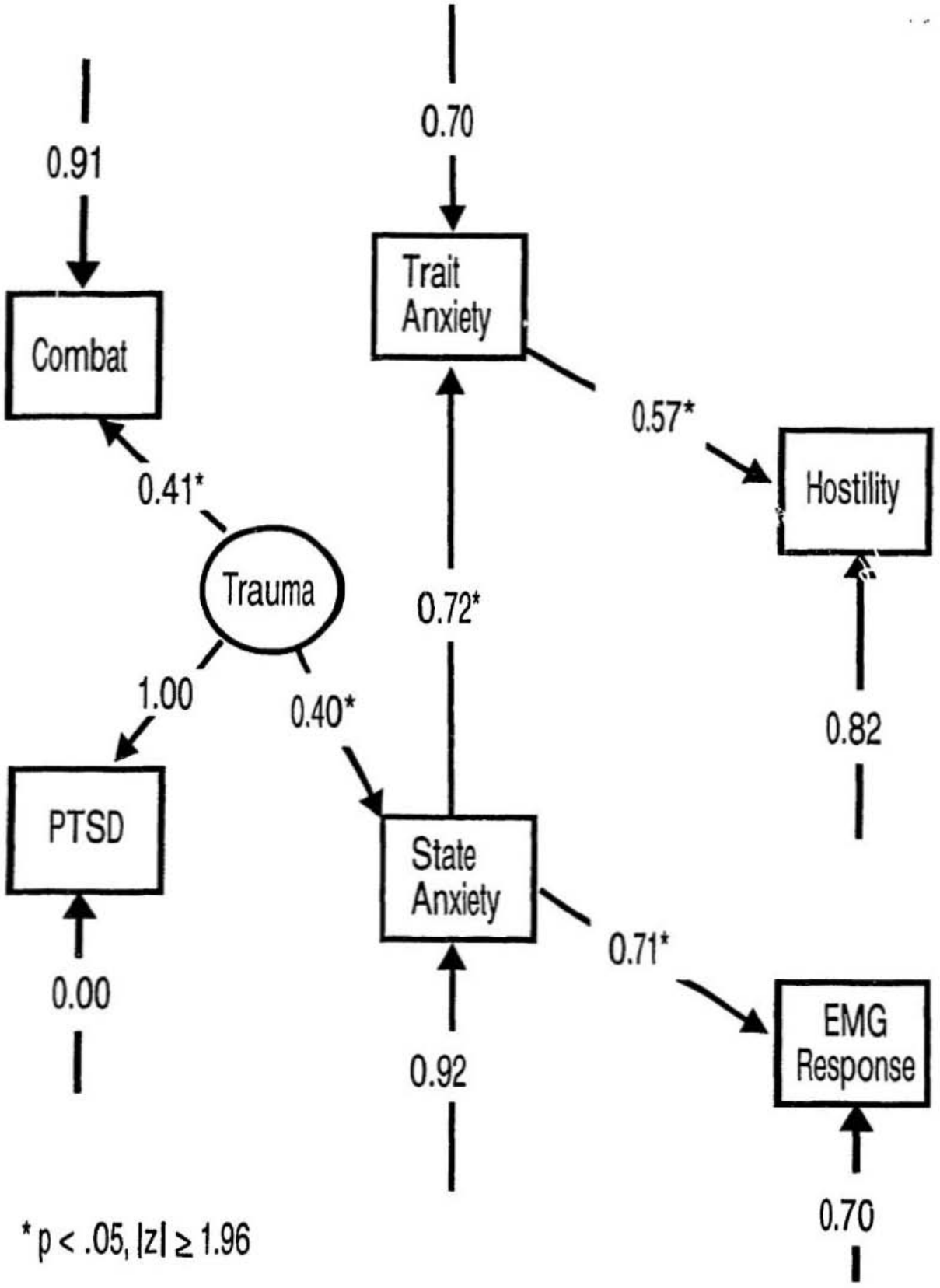

Figure 12. Standardized solution of Alternate Model 3a, data from Sample 2 (N=25)

The path coefficients between variables are shown in Figure 12. All of the path coefficients were significantly different from zero ($p < .05$ and $[z] > 1.96$). The LaGrange Multiplier test did not suggest adding any additional paths nor did the Wald test suggest dropping any of the paths in the model.

Alternate Model 3b

Alternate Model 3b, as shown in Figure 13, was constructed following the rationales used to build Alternate Model 2b. In this model, a path goes from trauma to state anxiety and a path goes from trauma to trait anxiety. All other paths are the same as those in Model 3.

Alternate Model 3b was tested with the data from Sample 2 ($N=25$). Once again, it was not possible to assess the fit of Alternate Model 3b in Sample 1. Mardia's coefficient, shown in Table 13 (AM 3b, Sample 1), indicated the absence of multivariate kurtosis.

As with Model 3, all of the beta weights in Alternate Model 3b were assessed as free parameters except for the factor loading of PTSD diagnosis onto trauma, which was set at 1.0. Given that PTSD diagnosis was a categorical variable, with values of either 0 or 1, Alternate Model 3b was assessed with the EQS program using an arbitrary distribution generalized least squares (AGLS) method. The correlation matrix for the variables in this model is shown in Appendix F.

The indices of goodness of fit for Alternate Model 3b are shown in Table 13. They included the Yuan-Bentler corrected AGLS chi-square for the hypothesized model, the

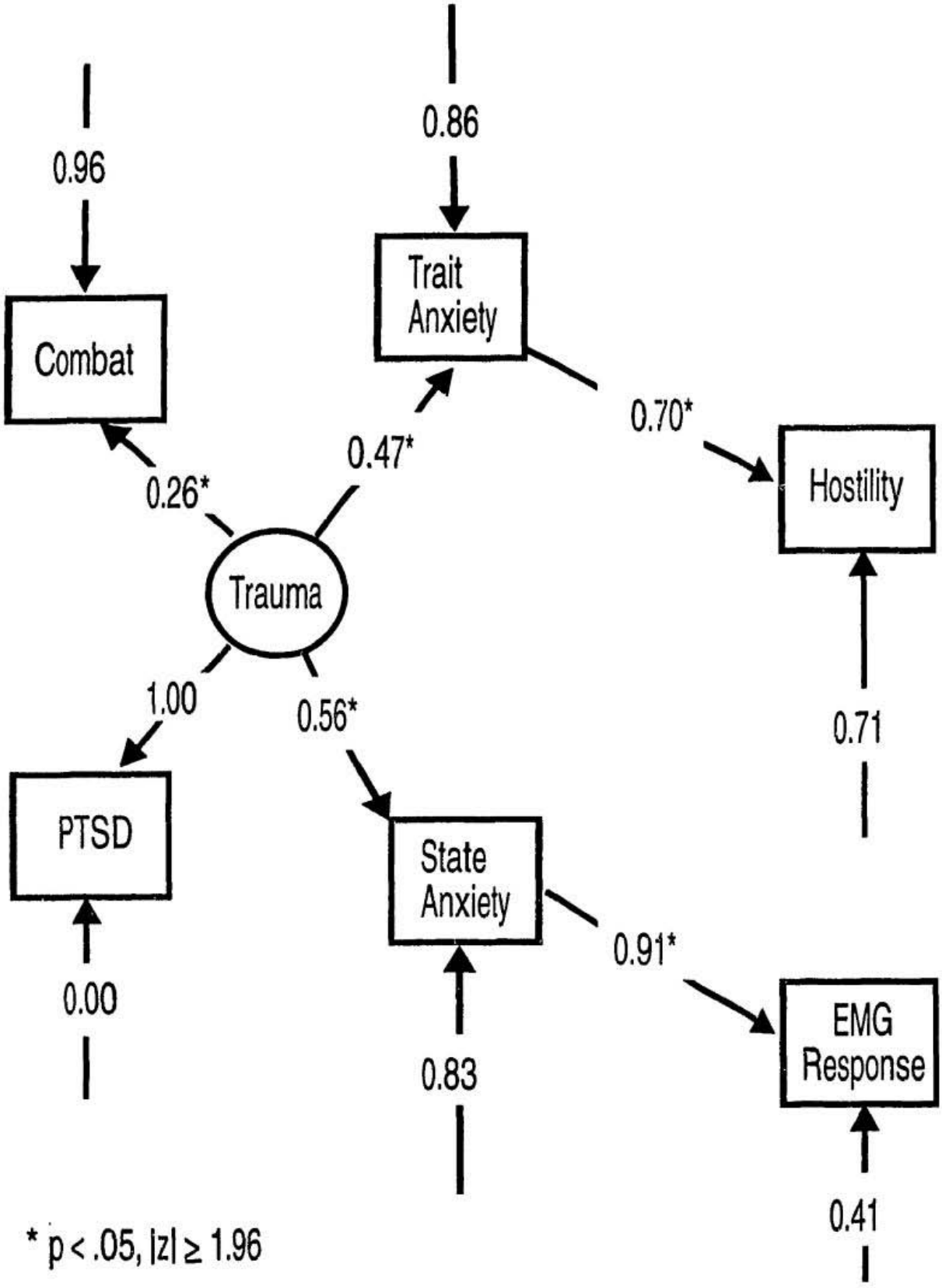

Figure 13. Standardized solution of Alternate Model 3b, data from Sample 2 (N=25)

AGLS adjusted fit index and the CFI.

Individual path coefficients were tested for significance by the EQS program and considered significant if $p<.05$, two-tailed and $[z]>1.96$.

In terms of overall fit, Alternate Model 3b, shown in Figure 13, fit the data from Sample 2 relatively well. As shown in Table 13, the indices of overall fit were acceptable. The Yuan-Bentler adjusted chi-square was small and nonsignificant, $X^2(10,N=25)=10.098$, $p=.43194$. The adjusted AGLS fit index was .998 and the CFI was 1.00. However, a relative CFI of 1.00 was calculated using Alternate Model 3b as the null model and Model 3 as the hypothesized model. This indicates that Model 3 fits the data from Sample 2 much better than Alternate Model 3b. In addition, a relative CFI of 1.00 was calculated using Alternate Model 3b as the null model and Alternate Model 3a as the hypothesized Model. This suggests that Alternate Model 3a also fits the data from Sample 2 much better than Alternate Model 3b.

The path coefficients between variables are shown in Figure 13. All of the path coefficients were significantly different from zero ($p<.05$ and $[z]>1.96$). The LaGrange Multiplier test did not suggest adding any additional paths nor did the Wald test suggest dropping any of the paths in the model.

Summary

A comparison of the overall fit indices and significance of parameters indicated that Model 2, Alternate Model 2a, and Alternate Model 2b fit the data from the two samples equally well.

Model 3 and Alternate Model 3a were found to be essentially equivalent in terms of fit and significance of parameters when applied to the data from Sample 2. Alternate Model 3b did not fit the data from Sample 2 as well as either of these models.

Discussion

Overview of Findings

PTSD is thought to be a common disorder, affecting individuals of all ages and cultures. While PTSD is an exception to other psychiatric disorders in that its initial cause is known, the nature of the symptoms that constitute this disorder remains to be firmly established in order that more effective means of treatment can be developed.

The present study is, to the best of our knowledege, the first attempt to use path analysis to test for possible interrelationships between the symptoms of anxiety, hostility, guilt, and startle within PTSD. Our hypothesized model, as shown in Figure 3, fits the data from two separate samples of Vietnam veterans very well both in terms of overall fit and the significance of the individual parameters. This suggests that the model may be capturing the relationships and directions of influence between anxiety, hostility, and guilt for these individuals with PTSD. As two of the four alternative models we tested fit the data from both samples just as well as our hypothesized model, it is equally likely that they reflect possible relationships among the variables.

Our hypothesized model of the relationship between startle and anxiety in PTSD fit the data very well. Unfortunately, we did not have a second data sample with which to test the replicability of this model. As was the case with the anxiety, hostility, guilt model, we found an alternative model for the startle model that fit the data from our sample equally well.

The present study also shows that veterans with and without PTSD can be accurately classified on the basis of their scores on the Buss Durkee suspicious hostility subscale and that our two samples of veterans differ from each other in terms of how they express hostility.

Population Differences in Style of Expressing Hostility

The analyses conducted to determine how best to represent hostility in our path models produced several findings of empirical interest.

The results of the discriminant analyses indicate that, in the present study, Vietnam Combat veterans with PTSD can be best distinguished from veterans without PTSD on the basis of their scores on the suspicious hostility subscale. This finding suggests that suspicious hostility is an important aspect of the elevated hostility levels of the veterans with PTSD. It is quite probable that the increases in trait anxiety associated with PTSD create an individual who constantly feels threatened. As a result these individuals may be very suspicious in nature and react to unknown people or situations with hostility as a means of defence.

While suspicious hostility is a significant discriminating variable, the results of the covariance analyses suggest that suspicious hostility on its own does not capture all aspects of hostility experienced by combat veterans with PTSD. Covarying suspicious hostility out of the total hostility score and the subscale scores does not eliminate significant group differences on all these measures. Moreover, there is no consistent pattern between the two samples with regard to which variables are affected by the

covariance analysis and which are not. Thus, the veterans in our two samples express hostility differently. This is consistent with previous research. In non-PTSD populations, expression of hostility measured with the Buss Durkee hostility subscales has been found to differ in Native versus non-Native Americans (Young, 1992), anabolic steroid users versus non-users (Yates, Perry, & Murray, 1992), assaultive versus suicide attempting males (Maiuro, O'Sullivan, Michael, & Vitaliano, 1989), suicide attempting versus nonviolent males (Maiuro et al., 1989) and assaultive versus nonviolent males (Maiuro et al., 1989).

The results of the discriminant analyses and the analyses of covariance do suggest however, that when using the Buss Durkee Hostility Inventory, researchers should pay close attention to differences on the individual subscales as well as on the Hostility Inventory total score. With respect to the present study, it would be interesting to see whether the suspicious subscale is a significant discriminating variable in other samples of Vietnam combat veterans. If it did turn out to be such a variable, it would suggest that suspicion is indeed a significant problem for this population of PTSD sufferers and while it may not account for all aspects of their hostility, treating suspicion may help alleviate some of their hostile feelings. It would also be worthwhile investigating whether suspicious hostility is a significant discriminating variable for individuals from populations other than male combat veterans, such as female assault victims, victims of natural disasters, or sexually and/or physically abused children with and without PTSD.

Path Analysis: The Role of Intervening Variables in the Pathogenesis of PTSD

Bollen and Long (1992) state that the best guide to assessing the fit of a model is strong substantiative theory. Following this maxim our first model, shown in Figure 2, should fit the data very well and the overall fit indices indicate that it does. However, in constructing this model we have failed to take into account one important aspect of PTSD; that not every individual exposed to a traumatic event, such as military combat, will go on to develop PTSD. Thus, it is not surprising that in applying Model 1 to the data from Sample 1, we find that the path from combat exposure to trait anxiety is not statistically significant.

Tomb (1994) states that the stressor an individual is exposed to is the most important risk factor in the development of PTSD. According to Tomb (1994) a stressor is more likely to result in PTSD if it is ".. severe, sudden, unexpected, prolonged, repetitive, or intentional; does physical damage to oneself or a loved one; is life-threatening; is isolating; conflicts with one's sense of self; is physically or psychologically demeaning; or does damage to one's community or support systems" (Tomb, 1994). Participation in combat can be considered to possess all of these characteristics. Indeed research has found that specific combat experiences that involve threat to one's life or the life of a friend or are considered to represent intense and severe combat exposure result in a greater likelihood of an individual developing PTSD (Davidson & Fairbank, 1993; Green et al., 1990; Foy et al., 1984; Foy, Carroll, & Donahue, 1987; Friedman et al., 1994; Schlenger, Kulka, Fairbank, Hough, Jordan, Marmar, & Weiss, 1992).

However, a number of the above stressor characteristics purported to increase the risk of PTSD are very subjective in nature and thus depend largely upon how an individual perceives a situation. Reich (1990) states that the meaning an individual attributes to a traumatic event must be involved in the development of PTSD. DSM-IV (1994) has also recognized this fact and emphasizes that an individual's perception of and reaction to a stressor is equally important in the development of PTSD as the objective severity of the stressor. This implies that an event considered traumatic by one individual may not be considered traumatic by another. This change in the definition of a stressor in DSM-IV (1994) helps to explain why only a portion of individuals exposed to a traumatic event will develop PTSD.

How an individual perceives a traumatic event is most likely the result of their personality and their experiences prior to the event. Schlenger et al. (1992), in a study that examined the prevalence of PTSD in Vietnam combat veterans, report that the characteristics individuals bring with them to combat have a significant role in determining who develops PTSD when exposed to high levels of combat stress. Examples of pretrauma experiences that may increase an individual's risk of developing PTSD upon exposure to a traumatic event include negative childhood experiences such as parental poverty, parental separation, and abuse (Davidson, Hughes, Blazer, & George, 1991); low intelligence, poor education, limited coping abilities, youth, and low socioeconomic status (Tomb, 1994).

Pretrauma experiences are not the only factors that may be relevant to the

pathogenesis of PTSD. Reich (1990) points out, that personality variables must also be involved in the development of PTSD as it is unlikely that personality would not play a part in the subjective meaning an individual attributes to an event. In fact there is research that suggests that certain personality styles may predispose an individual to PTSD. Neuroticism and introversion are two examples (McFarlane, 1989). In addition, personality disorders such as childhood conduct disorder may also increase the risk of developing PTSD (Helzer, Robins, & McEvoy, 1987).

Given the high comorbidity of other psychiatric disorders with PTSD (Davidson et al., 1991; Keane & Wolfe, 1990), it has been suggested that a personal history of psychiatric illness may predispose an individual to develop PTSD (DSM-III-R, 1987; Friedman, 1989). However, while two studies have linked previous mental illness to an increased risk of developing PTSD (Green et al., 1990; McFarlane, 1989), others have found it to be unrelated to PTSD (Friedman, 1989). In this regard, Keane and Wolfe (1990) have cautioned inferring any causal direction to the relationship between PTSD and other disorders.

It is interesting to note however, that a number of studies report that a family history of psychiatric illness may be a risk factor for PTSD (Davidson, Swartz, Storck, Krishnan, & Hammett, 1985; Davidson et al., 1991; Foy et al., 1987; Kulka et al., 1990). In their study, Foy et al. (1987) report that high levels of combat were a significant risk factor for PTSD regardless of whether an individual had a family history of psychiatric illness. However, veterans who were exposed to low levels of combat, but

who had a family history of psychiatric illness, were more at risk of developing PTSD than individuals exposed to similar levels of combat but who did not report having a history of psychiatric illness in their family. These findings suggest that there may be a genetic component that predisposes people to PTSD. True et al. (1993) report, in a twin study on PTSD, that even after adjusting for differences in combat exposure, genetic factors account for a significant portion of the variance in PTSD symptom liability. The concept of a genetic predisposition to develop PTSD is consistent with Adamec's (1980) observation that very early in life cats differ in terms of their defensive response with some cats exhibiting much more defensive behavior in response to a threat than others. Adamec (1991) suggests that some animals may be born with a predisposition to respond defensively to threatening stimuli. The same may be true of humans (Kagan & Snidman, 1991).

In addition to pretrauma risk factors there are also a number of posttrauma risk factors. PTSD has been associated with lack of post-trauma social support (Davidson et al., 1991; Green et al., 1990) and impaired social functioning (Davidson et al., 1991; Stretch., 1985).

The rationale for the path from combat exposure to trait anxiety in Model 1 is justified given that there is substantial research to suggest that intense trauma alters the neural substrate for fear/anxiety. However, given the information presented above, it is highly unlikely that a direct path exists between a traumatic event, such as combat exposure, and anxiety. An individual's reaction to the trauma determines the

severity/intensity of the trauma. Our modified model, which is discussed in the next section, takes this fact into account.

While the present study does not examine individual risk factors for PTSD, future testing of PTSD models which include some of these risk factors is a valid exercise for researchers interested in preventing PTSD. Identifying individuals who are predisposed to develop PTSD as a result of exposure to trauma, whether mild or severe, can result in these individuals receiving immediate intervention, in the form of counselling or medication, that may help prevent the development of this disorder.

Model 2: A Possible Representation of the Relationships Between Anxiety, Hostility, and Guilt Within PTSD

As previously discussed, for any given situation, the nature of a traumatic stressor will only account for a portion of the variability of which individuals develop PTSD. An individual's reaction to the situation, which is determined by aspects of their personality and their previous experiences, is also a determining factor.

To take the above into account, the initial model, Model 1, was modified to include a latent variable called trauma which represents an individual's reaction to combat as a traumatic experience.The trauma factor in Model 2, as shown in Figure 3, captures the variability in PTSD diagnosis that is accounted for by combat exposure. Thus, it is pulling out those aspects of combat that are most related to the development of PTSD. The trauma factor incorporates the idea that an individual's perception of their combat experience as traumatic is equally, if not more, important than any objective measure of

the severity of their combat exposure.

As shown in Table 13, the overall fit of Model 2 to the data from Sample 1 is very good. In addition, all path coefficients between the variables in Model 2 are significant. This suggests that Model 2, as a whole, may be capturing some aspects of what is occurring between trauma, anxiety, hostility, and guilt within PTSD. This is supported by the fact that the good overall fit indices and significant paths obtained for Model 2 with the data from Sample 1 replicate when the model is applied to the data from Sample 2.

In Model 2, the path from the trauma factor to trait anxiety is statistically significant when the model is tested on both samples of veterans. The fact that this path is significant and the path from combat exposure directly to trait anxiety in Model 1 is nonsignificant supports the idea, as suggested above, that the perception of combat as traumatic is perhaps more important to the development of PTSD than the objectively measured intensity of an individual's combat exposure. Thus, Model 2, suggests that when an individual perceives an event as traumatic and reacts to it as such, they become inherently more anxious. Such a concept is consistent with Adamec's (1978) emotive biasing model concept which has been applied to epileptic anxiety and PTSD (Adamec, 1996; Pitman et al., 1993). According to Adamec, sensitization of neural structures which process threatening stimuli can alter connections between these neural structures and others involved in the expression of fear/anxiety, ultimately producing a long lasting increase in the fear/anxiety expressed in response to future threatening situations.

While the LaGrange Multiplier Test indicates that the path coefficient from the trauma factor to trait anxiety is significant and equal in the two samples, it also shows that the factor loading of combat onto the trauma factor is noninvariant across the two samples. In Sample 2, the factor loading for combat onto trauma is positive while in Sample 1 the factor loading is negative. The difference in the factor loading of combat onto trauma is readily accounted for within the definition of a traumatic stressor as per DSM-IV (1994). As previously discussed, an individual's perception of an event as stressful or threatening plays a large role in determining its impact. Therefore, it would be expected that the loading of combat exposure onto trauma might vary between samples given that combat experiences are interpreted on an individual basis. Thus, in some populations, individuals with low combat exposure may nevertheless be highly reactive and develop PTSD. The opposite of this may be true in other populations. This may have occurred in the two samples studied. In Sample 1, the veterans with PTSD reported lower combat exposure scores than non-PTSD veterans, whereas the opposite was the case in Sample 2.

The path from trait to state anxiety is significant when Model 2 is applied to both of the data sets. Such a finding is not surprising in that it is only reasonable to expect that individuals high in trait anxiety or anxiety-proneness will exhibit elevated levels of state anxiety in situations they perceive as threatening (Barker, Wadsworth, & Wilson, 1976). However, the LaGrange Multiplier Test indicates that the coeffcient for the path from trait to state anxiety differs across the two data samples. The coefficient for the path

from trait to state anxiety in Sample 1 is much larger than the coefficient for the same path in Sample 2. This noninvariance of the path coefficient from trait to state anxiety may be the result of differences in the levels of state anxiety reported by subjects in the two samples at the time of testing. Subjects with PTSD in Sample 2 had significantly higher scores on the state component of the STAI than subjects with PTSD in Sample 1 ($F(1,37)=16.37$, $p<.001$). Control subjects in Sample 2 also had significantly higher state anxiety scores than the control subjects in Sample 1 ($F(1,37)=35.12$, $p<.00001$). Of particular relevance is the finding that control subjects in Sample 2 did not differ significantly in state anxiety from PTSD subjects in Sample 1 ($F(1,32)=.62$, $p>.05$). Thus, control subjects in sample 2 were experiencing levels of state anxiety at the time of testing that were comparable to those experienced by subjects with PTSD in Sample 1. The higher levels of state anxiety in Sample 2 subjects may be the result of their state anxiety being measured immediately prior to their participating in a laboratory experiment designed to assess their startle response. Uncertainty or even knowledge regarding the nature of this testing could result in the subjects in Sample 2 experiencing increased levels of anxiety at the time of completing the state component of the STAI. Subjects in Sample 1 were not tested under such conditions.

Thus, a possible explanation for the noninvariance in the coefficient for the trait to state anxiety path is that the conditions under which subjects in Sample 2 were tested produced a larger increase in state anxiety in the controls than in the subjects with PTSD in Sample 2. PTSD subjects may not have increased as much as controls because they

were near their maximum anxiety level in a basal state. This interpretation is supported by the finding that the mean difference in state anxiety between control subjects in Sample 1 and Sample 2 was 28.53 compared to a mean difference of 19.85 between PTSD subjects in the two samples. In addition, the mean difference between state anxiety scores for PTSD subjects versus controls in Sample 1 was 29.96 compared to a mean difference of 21.27 for control and PTSD subjects in Sample 2. Given that the two samples did not differ in terms of trait anxiety, i.e. neither sample was more anxious than the other in general, one would expect a lower value of the slope of the relationship between trait and state anxiety in the subjects from Sample 2. As a path coefficient is an index of this slope, the path coefficient between trait and state anxiety would be smaller in Sample 2 than in Sample 1, hence the noninvariance of this path coefficient across the two samples. This is consistent with Martin et al. (1987) who state that correlations between state anxiety and trait anxiety, as measured by the STAI, are lower when subjects are placed in threatening situations.

A particularly relevant finding in Model 2, is the significance of the path from trait anxiety to hostility which replicates in the second data sample and is invariant across the two samples. Model 2 supports the idea that, in our samples, high levels of anxiety lead to increased hostility. This is consistent with Hovens et al.'s (1992) suggestion that the high correlation between anger and trait anxiety in their sample of veterans with PTSD is the result of uncontrollable anxiety making one angry and Dutton's (1995) suggestion that, in abusive men with a PTSD-like condition, hostility/aggression serves to reduce

anxiety . Model 2 may also explain why previous studies have also found a relationship between hostility and trait anxiety in subjects other than veterans with PTSD (Deffenbacher et al., 1986; Katz et al., 1993). Anxiety may be a natural precursor of hostility for some individuals regardless of the initial cause of the anxiety.

Thus, as previously suggested by Lasko et al. (1994), it is highly unlikely that the increased levels of hostility observed in combat veterans with PTSD are the result of combat exposure per se. Rather, it appears that hostility is related to the increased levels of anxiety experienced by these individuals as a result of a traumatic stressor.

Model 2 also suggests that the veterans in our two samples experience considerable guilt in relation to their increased hostility. This would be expected in that their hostility is most likely a spontaneous, protective response to uncontrollable feelings of anxiety that are generated by any situation the individual perceives as threatening. Van der Kolk (1987) states that individuals with PTSD often go from stimulus to response without making a psychological assessment of the situation. Thus, an individual with PTSD may misinterpret an event as threatening, experience heightened levels of anxiety in response to the threat, and become hostile as a means of protecting themselves from the perceived threat. Later on, the individual may realize that their reaction was not warranted by the situation and they feel considerable guilt for their actions. It would also not be surprising if these feelings of guilt might cause further anxiety, in that the individual is able to realize that they are frequently making inappropriate responses to situations but they are unable to control these responses. This may account for the number of veterans with

PTSD who self-medicate with drugs and alcohol in an attempt to gain some control over their anxiety and heightened hostility (Friedman, 1991).

Alternative Models of the Relationship Between Anxiety, Hostility, and Guilt in PTSD

An examination of Table 13 shows that two of the alternative models we tested fit the data from our two samples of Vietnam veterans with PTSD as well as Model 2, our hypothesized model. This suggests that the paths between the variables in these models represent plausible alternative relationships between anxiety, hostility, and guilt.

(i) Alternate Model 2a

In Alternate Model 2a the path from the trauma factor to state anxiety is significant and invariant in both of our samples. This suggests that, for individuals who develop PTSD, exposure to a traumatic event such as combat may produce increases in their level of state anxiety i.e. how they react to situations they perceive as threatening. This is consistent with Shalev et al.'s (1996) finding of increased state anxiety at 1 week posttrauma and 6 months posttrauma in individuals who develop PTSD when compared to individuals who do not develop PTSD.

The path from state anxiety to trait anxiety is also significant in both of the samples. This suggests that individuals with PTSD who experience increased state anxiety to threatening situations may ultimately become more anxious in general, i.e., have higher levels of trait anxiety. This would account for Shalev et al.'s (1996) finding of no increase in trait anxiety at 1 week posttrauma in individuals who go on to develop PTSD

and the consistent finding of increased trait anxiety in individuals with PTSD who are being tested many years after the original traumatic event. The mechanism through which state anxiety may increase trait anxiety might be similar to kindling. Van der Kolk (1987) states that individuals with PTSD respond to emotional stimulation with an intensity appropriate to the original trauma.Van der Kolk (1987) also suggests that repeated traumatization may produce a kindling phenomenon which may result in behavioral (characterological) changes. Thus, it is possible that individuals who respond to subsequent stressors with the same level of state anxiety as they did with the original trauma may, through a mechanism similar to kindling, become permanently more anxious i.e have a higher level of trait anxiety than they did prior to the trauma.

The results of Alternate Model 2a have an interesting implication for the role of hostility in PTSD. If, as the model suggests, trait anxiety is a delayed symptom in PTSD then one would not expect to observe increased levels of hostility in individuals with PTSD until the disorder has progressed. It would be of interest to determine whether increased hostility is observed in individuals at the time of the trauma or sometime after. Moreover, increased hostility should follow or be coincident with increased trait anxiety.

It should be noted that the path from state to trait anxiety varied across the two samples. This can be accounted for by differences in the conditions under which state anxiety was assessed in the two samples as discussed in the previous section on Model 2.

(ii) Alternate Model 2b

In Alternate Model 2b the paths from the trauma factor to state anxiety and the trauma factor to trait anxiety are significant in both samples. The noninvariance of the path from trauma to state anxiety across the two samples can be accounted for by the sample differences in the levels of state anxiety that result from the conditions under which state anxiety was assessed in the two samples.

The finding of a significant path from the trauma factor to state anxiety and from the trauma factor to trait anxiety suggests that trauma has a direct impact on state anxiety and a direct impact on trait anxiety. These findings are consistent with Shalev et al.'s (1996) findings on state anxiety and PTSD and all the studies that have found increased levels of trait anxiety in individuals with PTSD (Hovens et al., 1994; Hovens et al., 1992; Kuhne et al., 1993; Orr et al., 1995; Sutker et al., 1991). This model suggests that while trauma has an immediate impact on state anxiety, its effect on trait anxiety may be delayed. This is consistent with Rothbaum et al. (1992) who report that the symptoms observed in individuals who develop PTSD shortly after the trauma are different than the symptoms observed later on. This model also implies that the effects of trauma on state anxiety and trait anxiety are not interdependent.

A phenomenon that may be related to the increases in anxiety observed in individuals with PTSD is dissociation. Several studies have reported that individuals who experience dissociation at the time of the trauma are more likely to develop PTSD than individuals who do not experience dissociation (Bremner, Southwick, Brett, Fontana, Rosenheck, &

Charney, 1992; Marmar, Weiss, Schlenger, Fairbank, Jordan, Kulka, & Hough, 1994; Orr, Claiborn, Altman, Forgue, De Jong, Pitman, & Herz, 1990; Shalev et al., 1996). Relationships between dissociation and anxiety have also been found (Cardena & Spiegel, 1993).

The relevance of dissociation to anxiety in PTSD comes from studies that show many individuals with epilepsy who also suffer from anxiety experience auras that resemble aspects of dissociation prior to their seizures. These auras may indicate activation of limbic structures associated with fear anxiety (Adamec, 1990). Auras such as time changes, and derealization, have been found to distinguish epileptics with anxiety and depression from epileptics without these psychopathologies (Stark-Adamec & Adamec, 1986; Adamec et al., 1990). Time changes and derealization have also been reported in phobic anxiety patients without epilepsy (Harper & Roth, 1962). Mark, Erwin, and Sweet (1972) report that a feeling of strangeness or unreality can be produced by limbic stimulation. Time change has been found to cluster with derealization (Adamec et al. 1990).

Time change and a sense of unreality are two aspects of dissociation experienced at the time of the trauma by individuals who develop PTSD (Marmar et al., 1994). Thus, it is possible that these two types of dissociation may indicate limbic activation at the time of trauma in individuals who develop PTSD. As previously discussed, Adamec and Morgan (1994) have found that kindling of the right hemisphere amygdala increases anxiety in rodents. It is possible that a kindling-like activation of the amygdala occurs in

individuals who develop PTSD in response to trauma which may produce the increases in anxiety associated with PTSD. Indeed, it has been shown that individuals given procaine hydrochloride intravenously, exhibit, via positron emission tomography (PET), increased glucose uptake in the amygdala (Parekh, Spencer, George, Gill, Ketter, Andreason, Herscovitch, & Post, 1995). Procaine hydrochloride has been shown to increase fear and feelings of dysphoria in some individuals (Kellner, Post, Putnam, Cowdry, Gardner, Kling, Minichiello, Trettau, & Coppola, 1987; Stark-Adamec, Adamec, Graham, Bruun-Meyer, Perrin, Pollock, & Livingston, 1982). Ketter, Andreason, George, Lee, Gill, Parekh, Willis, Herscovitch, and Post (1996) have found that individuals who experience intense procaine-induced fear exhibit greater increases in amygdalar cerebral blood flow after intravenous procaine injection than individuals who do not exhibit procaine-induced fear. Moreover, Rausch et al. (1995) have also found that in comparison to a neutral state, individuals with PTSD show an increased blood flow, as measured by PET, in right-sided limbic and paralimbic areas when exposed to audiotapes of individualized traumatic event scripts. These findings suggest that the amygdala may be involved in the genesis of anxiety and other negative emotions in humans. More importantly, excessive activation of the right amygdala is associated with PTSD. It is possible that some form of temporolimbic sensitization, as suggested by van der Kolk (1984) and Adamec (1990; 1996), occurs following traumatic stress and appears neurally as excessive right amygdala activation and behaviorally as increased anxiety.

If the increases in anxiety observed in PTSD are a result of trauma-related changes to the amygdala it is unclear whether these alterations produce increases in trait anxiety or state anxiety. If the trauma alters the basal level functioning of the amygdala this might indicate an increase in trait anxiety. However, if the nature of the trauma is to hypersensitize the amygdala to subsequent input, it might suggest an increase in state anxiety only. Rausch et al.'s (1995) PET study suggests that the latter may be the case. However, much more research in this area is required before any definitve conclusions can be reached.

The issue of dissociation as a possible marker of limbic activation in PTSD is one that deserves investigation. If such a relationship does exist, self-reports of dissociation may be a simple, inexpensive means of determining which individuals are most likely to develop PTSD as a result of exposure to trauma.

The Relationship of State Anxiety to Startle in PTSD

The results of the three models that examined the relationship between state anxiety and startle in PTSD suggest that the increased startle responses seen in individuals with PTSD are the result of elevated levels of state anxiety. In each of the three models, the path from state anxiety to startle, as measured by EMG response, was significant. The only differences in the three models was the way in which this increase in state anxiety was produced, which was discussed above.

Our findings suggest that the increased startle seen in veterans with PTSD may be a form of fear-potentiated startle. The results of the present study are consistent with

previous research by Morgan et al. (1995) who found an increased fear-potentiated startle in their sample of veterans with PTSD, but no increase in baseline startle.

In animals, it has been shown that low-level electrical stimulation of the amygdala increases the amplitude of the startle-response (Rosen & Davis, 1990). Campeau and Davis (1995) have suggested that the basolateral complex of the amygdala serves as a necessary relay of sensory information from the cortical and subcortical sensory areas to the central nucleus of the amygdala which they believe to be a response independent, final common relay for fear conditioning. Rausch et al.'s (1995) finding of increased blood flow in the right amygdala of individuals with PTSD when exposed to audiotapes of individual trauma events suggests that, in humans, activation of the amygdala by fear serves to potentiate startle. It is interesting that Rausch et al. (1995) find increased blood flow in the right amygdala, as this is in the hemisphere believed to be involved with negative emotions (Sackheim et al., 1982). In keeping with this idea, Vrana et al. (1988) have found that, in normal individuals, the startle response is potentiated in response to negative but not positive visual images. Vrana et al. (1988) suggest that the startle response may be a new means of measuring emotion and assessing pathological anxiety.

Implications of the Present Findings for Therapeutic Intervention in PTSD

In all of the models we tested, the path from trait anxiety to hostility is significant. This would suggest that one possible way of reducing hostility in veterans with PTSD might be to reduce anxiety. Indeed, it has been shown that PTSD patients treated with anxiolytic medications such as propanolol, a beta-blocker, do experience a reduction in

hostility/aggression (Kolb, Burris, & Griffiths, 1984; Silver, Sandberg, & Hales, 1990).

However, it may also be equally efficacious to use cognitve training to reduce hostility. Reilly, Clark, Shopshire, Lewis, & Sorensen (1994) have found that PTSD patients who undergo a cognitive-behavioral anger management program are able to control their anger more effectively. Teaching individuals with PTSD more appropriate means of dealing with their feelings of anxiety should also help to alleviate hostility.

An important issue that arises from the present study is the best way in which to deal with the increased levels of anxiety experienced by individuals with PTSD. If, as Alternate Model 2a suggests, increases in state anxiety produce later increases in trait anxiety then it would be expected that early intervention therapies directed at reducing state anxiety might be effective in preventing the development of the anxiety based symptoms of PTSD entirely. In fact, researchers have suggested that early intervention might prevent PTSD or its chronic course (Davis & Breslau, 1994; Friedman et al., 1994). Animal studies indicate that this may be the case. Adamec (1996) has found that administering CCK_b blockers to rodents after they have been exposed to a stressor prevents lasting increases in anxiety like behaviors.

Early intervention studies have been conducted with indivduals exposed to trauma. It should be noted that Davis and Breslau (1994) state that none of the studies to date have been randomized or controlled. However, Van der Kolk (1988) states that acute post-traumatic anxiety in adults is amenable to verbal therapies and suggests that pharmacological intervention can help to decrease physiological arousal which facilitates

the individual's ability to retrieve traumatic memories. Blake (1986) describes three patients with acute PTSD as a result of an accident who responded favorably to treatment with tricyclic antidepressants. Blake (1986) suggests that tricyclics, such as imipramine, may be effective in diminishing or even preventing delayed symptoms in individuals with acute PTSD. These findings are consistent with the path from trauma to state anxiety in Alternate Model 2a in that they suggest reducing anxiety in individuals with acute PTSD can prevent the development of the more chronic symptoms of PTSD.

Alternate Model 2b suggests that treating state anxiety will not reduce trait anxiety. If the increased levels of trait anxiety are a delayed symptom of PTSD, then early interventions designed to treat anxiety in general may not neccessarily be effective in reducing trait anxiety. This model suggests that individuals should be treated for elevated state anxiety levels immediately following traumatic exposure and also be provided some form of therapeutic support, whether pharamcological or psychological, in order to prevent or reduce later increases in levels of trait anxiety.

One form of therapy that may reduce trait anxiety in sufferers of PTSD is eye movement desensitization (EMD). EMD is a technique developed by Shapiro (1989) in which the patient is asked to visualize the traumatic event while simultaneously tracking the therapist's finger as it moves quickly back and forth across the patient's visual field. A number of studies have reported the effectiveness of EMD in treating the symptoms of PTSD (Forbes, Creamer, & Rycroft, 1994; Kleinknecht & Morgan, 1992; Vaughn, Armstrong, Gold, O'Connor, Jenneke, & Tarrier, 1994: Silver, Brooks, & Obenchain,

1995; Vaughn, Wiese, Gold, & Tarrier, 1994). Kleinknecht and Morgan (1992) report the case of a 40- year old man who had developed PTSD after being shot and left to die. When the subject was initially assessed eight years after the shooting, his trait anxiety score on the STAI corresponded with the 64th percentile of psychiatric patients. Four months after being treated with EMD, his trait anxiety score was at the 26th percentile for psychiatric patients and at eight months posttreatment his trait anxiety score was at the 36th percentile for psychiatric patients. While these results suggest that EMD may help alleviate increased trait anxiety in PTSD, this study, like many other studies reporting the benefits of EMD, is based on one subject. Indeed, Herbert and Mueser (1992) have recommended that statements regarding the efficacy of EMD should not be made until the results of more methodologically sound studies are available.

Eppley, Abrams, and Shear (1989) have conducted a meta-analysis of the effects of various relaxation techniques on trait anxiety in non-PTSD populations and conclude that, even after controlling for possible confounding variables, transcendental meditation produces a significantly larger reduction in trait anxiety than other forms of relaxation such as progressive relaxation, EMG feedback, and other meditation techniques. Whether transcendental meditation would be effective in reducing the elevated levels of trait anxiety that occur in PTSD remains to be determined.

With respect to the startle models of PTSD, the results imply that increased startle reactions in individuals could be reduced by strategies designed to reduce state anxiety. In fact, it has been shown in animal studies that anxiolytic drugs block fear-potentiated

startle (Davis, Falls, Campeau, & Kim, 1993). While it has been shown that anxiogenics such as yohimbine increase startle in normal humans (Morgan et al., 1993) and individuals with PTSD (Morgan et al., 1995), it still remains to be determined whether anxiolytic compounds have the potential to reduce startle in humans. The results of the present study suggest that they would.

In summary, our models do imply different approaches for the treatment of anxiety, hostility, guilt, and startle in PTSD. An extensive examination of possible treatments for PTSD is not the intention of the present study. However, it is hoped that our models suggest rationales for different approaches to the treatment of PTSD.

Limitations of the Present Study

A common problem in many studies of PTSD is small sample sizes. In the present study, our sample sizes are fairly small for the type of analyses we undertook. Bentler (1985) suggests that 5 subjects per parameter is a minimum requirement for path analytic studies. Based on this our first sample is adequate, but it does suggest that the results based on Sample 2 may be questionable.

Given that all of our models fit the data equally well and each has different implications with regards to the treatment of PTSD it is essential that they be tested in other, larger samples of veterans. This will allow researchers to reach more definitve conclusions about the nature of anxiety, hostility, guilt, and startle in Vietnam veterans with PTSD.

A second limitation is that our samples consisted solely of male subjects with

combat- related PTSD. It has been suggested that PTSD is not a homogenenous disorder (Kolb, 1989; Ciccone, Burstein, and Greenstein, 1989). Ciccone et al. (1989) state that it may not be feasible to construct generalizations about PTSD based on only one treatment group. Thus, it it would certainly be of empirical interest to determine which, if any, of our models replicate in other populations of PTSD patients such as female rape victims or sexually and/or physically abused children.

Finally, it should be pointed out that, as is the case with any correlational study, our results can not be taken to imply causation. As Biddle and Martin (1987) suggest, the strongest conclusion that can be reached when assessing a model is that one has correctly predicted the patterning of observed, associational relationships. Thus, the models in the present study suggest causal relationships among anxiety, hostility, guilt, and startle in PTSD. In addition our models only include a small sample of the many symptoms experienced by individuals with PTSD. A valid exercise for future researchers might be to construct path models that include other symptoms of PTSD such as depression, and symptoms from the DSM-IV (1994) avoidance category.

Summary

In the present study we were able to produce several models that suggest relationships between anxiety, hostility, guilt, and startle in PTSD. Given that the models fit the data from our samples equally well and are all supported by the relevant literature, it remains unclear at this point which of these models best represents the relationships among these variables. It is possible that the different models may be more applicable

to different stages of the disorder. Alternate Models 2a and 3a, which suggest that trauma increases state anxiety which then increases trait anxiety, may be more applicable to the early stages of PTSD. Models 2 and 3 may be more applicable to individuals who have suffered from the disorder for several years. Alternate Model 2b differs from these models in that it suggests that state and trait anxiety are both affected by trauma and that neither one impinges upon the other.

While we are unable to make any conclusive statements about a unique best fitting model, the results of the present study suggest testable hypotheses about the etiology of anxiety, hostility, guilt, and startle symptoms in PTSD. Moreover, the models suggest different paths of treatment.

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Appendix A

State-Trait Anxiety Inventory (STAI)
Form Y-1

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you feel **right now**, that **is, at this moment**. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

	Not at all	Moderately So	Somewhat	Very Much So
1. I feel calm	1	2	3	4
2. I feel secure	1	2	3	4
3. I am tense	1	2	3	4
4. I feel strained	1	2	3	4
5. I feel at ease	1	2	3	4
6. I feel upset	1	2	3	4
7. I am presently worrying over possible misfortunes	1	2	3	4
8. I feel satisfied	1	2	3	4
9. I feel frightened	1	2	3	4
10. I feel comfortable	1	2	3	4
11. I feel self-confident	1	2	3	4
12. I feel nervous	1	2	3	4
13. I am jittery	1	2	3	4
14. I feel indecisive	1	2	3	4
15. I am relaxed	1	2	3	4
16. I feel content	1	2	3	4
17. I am worried	1	2	3	4
18. I feel confused	1	2	3	4
19. I feel steady	1	2	3	4
20. I feel pleasant	1	2	3	4

State-Trait Anxiety Inventory (STAI)
Form Y-2

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you **generally** feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

	Not at all	Moderately So	Somewhat	Very Much So
21. I feel pleasant.....	1	2	3	4
22. I feel nervous and restless.....	1	2	3	4
23. I feel satisfied with myself.....	1	2	3	4
24. I wish I could be as happy as others seem to be.....	1	2	3	4
25. I feel like a failure.....	1	2	3	4
26. I feel rested.....	1	2	3	4
27. I am calm, cool, and collected.....	1	2	3	4
28. I feel that difficulties are piling up so that I cannot overcome them....	1	2	3	4
29. I worry too much over something that really doesn't matter.....	1	2	3	4
30. I am happy.....	1	2	3	4
31. I have disturbing thoughts.....	1	2	3	4
32. I lack self-confidence.....	1	2	3	4
33. I feel secure.....	1	2	3	4
34. I make decisions easily.....	1	2	3	4
35. I feel inadequate.....	1	2	3	4
36. I am content.....	1	2	3	4
37. Some unimportant thought runs through my mind and bothers me.....	1	2	3	4
38. I take disappointments so keenly that I can't put them out of my mind.....	1	2	3	4
39. I am a steady person.....	1	2	3	4
40. I get in a state of tension or turmoil as I think over my recent concerns and interests.....	1	2	3	4

Appendix B

Buss-Durkee Inventory

Listed below are a number of statements concerning personal attitudes and traits. Read each item and decide whether the statement is TRUE or FALSE as it pertains to you personally. Cross 'T' for TRUE or 'F' for FALSE.

1. I seldom strike back, even if someone hits me first.	T	F
2. I sometimes spread gossip about people I don't like.	T	F
3. Unless somebody asks me in a nice way, I won't do what they want.	T	F
4. I lose my temper easily but get over it quickly.	T	F
5. I don't seem to get what is coming to me.	T	F
6. I know that people tend to talk about me behind my back.	T	F
7. When I disapprove of my friends' behavior, I let them know.	T	F
8. The few times I have cheated, I have suffered unbearable feelings of remorse.	T	F
9. Once in a while I cannot control my urge to harm others.	T	F
10. I never get mad enough to throw things.	T	F
11. Sometimes people bother me just by being around.	T	F
12. When somebody makes a rule I don't like, I am tempted to break it.	T	F
13. Other people always seem to get breaks.	T	F
14. I tend to be on my guard with people who are		

somewhat more friendly than I expected. T F

15. I often find myself disagreeing with people. T F

16. I sometimes have bad thoughts which make me feel ashamed of myself. T F

17. I can think of no good reason for ever hitting anyone. T F

18. When I am angry, I sometimes sulk. T F

19. When someone is bossy, I do the opposite of what he asks. T F

20. I am irritated a great deal more than people are aware of. T F

21. I don't know any people that I downright hate. T F

22. There are a number of people who seem to dislike me very much. T F

23. I can't help getting into arguments when people disagree with me. T F

24. People who shirk on the job must feel very guilty. T F

25. If somebody hits me first, I let him have it. T F

26. When I am mad, I sometimes slam doors. T F

27. I am always patient with others. T F

28. Occasionally when I am mad at someone I will give him the silent treatment. T F

29. When I look back on what's happened to me, I can't help feeling mildly resentful. T F
30. There are a number of people who seem to be jealous of me. T F
31. I demand that people respect my rights. T F
32. It depresses me that I did not do more for my parents. T F
33. Whoever insults me or my family is asking for a fight. T F
34. I never play practical jokes. T F
35. It makes my blood boil to have somebody make fun of me. T F
36. When people are bossy I take my time just to show them. T F
37. Almost every week I see someone I dislike. T F
38. I sometimes have the feeling that others are laughing at me. T F
39. Even if my anger is aroused, I don't use strong language. T F
40. I am concerned about being forgiven for my sins. T F
41. People who continually pester you are asking for a punch in the nose. T F
42. I sometimes pout when I don't get my own way. T F
43. If somebody annoys me, I am apt to tell him what I think of him. T F
44. I often feel like a powder keg ready to explode. T F
45. Although I don't show it, I am sometimes eaten up with jealousy. T F

46. My motto is never trust strangers. T F

47. When people yell at me, I yell back. T F

48. I do many things that make me feel remorseful afterward. T F

49. When I really lose my temper, I am capable of slapping someone. T F

50. Since the age of ten, I have never had a temper tantrum. T F

51. When I get mad, I say nasty things. T F

52. I sometimes carry a chip on my shoulder. T F

53. If I let people see the way I feel, I'd be considered a hard person to get along with. T F

54. I commonly wonder what hidden reason another person may have for doing something nice for me. T F

55. I could not put someone in his place, even if he needed it. T F

56. Failure gives me a feeling of remorse. T F

57. I get into fights about as often as the next person. T F

58. I can remember being so angry that I picked up the nearest thing and broke it. T F

59. I often make threats I don't really mean to carry out. T F

60. I can't help being a little rude to people I don't like. T F

61. At times I feel I get a raw deal out of life. T F

62. I used to think that most people told the truth but

now I know otherwise. T F

63. I generally cover up my poor opinion of others. T F

64. When I do wrong my conscience punishes me severely. T F

65. If I have to resort to physical violence to defend my rights, I will. T F

66. If someone doesn't treat me right, I don't let it annoy me. T F

67. I have no enemies who really wish to harm me. T F

68. When arguing, I tend to raise my voice. T F

69. I often feel that I have lived the right kind of life. T F

70. I have known people who pushed me so far that we came to blows. T F

71. I don't let a lot of unimportant things irritate me. T F

72. I seldom feel that people are trying to anger or insult me. T F

73. Lately I have been kind of grouchy. T F

74. I would rather concede a point than get into an argument about it. T F

75. I sometimes show my anger by banging on the table. T F

Appendix C

Combat Exposure Scale

These questions concern the nature and extent of your exposure to combat if any, including all experiences that took place on land, in the air, or at sea. There is a scale ranging from 0 to 3 or 0 to 4 under each question.

Please choose the answer on the scale underneath each question that comes closest to describing your combat experiences, and circle the number from 0 to 3 or 4 for that choice. Ask the interviewer for clarification of any question you may have.

1. How many times did you ever go on combat patrols, participate in amphibious invasions, or have other very dangerous duty.

0	1	2	3	4
None	1-2 times	3-12 times	13-50 times	more than 50 times

2. How many months were you ever under enemy fire.

0	1	2	3	4
Never	Less than 1 month	1 - 3 months	4 - 6 months	more than 6 months

3. How many times were you ever surrounded by the enemy.

0	1	2	3
None	1-2 times	3-12 times	more than 12 times

4. What percentage of the men in you unit were killed (KIA), wounded, or missing (MIA) in action.

0	1	2	3
No one	Between 1 - 25%	Between 26 - 50%	More than 50%

5. How many times did you ever fire rounds at the enemy.

0	1	2	3	4
None	1-2 times	3-12 times	13-50 times	more than 50 times

6. How many times did you ever see someone hit by incoming or outgoing rounds.

0	1	2	3	4
None	1-2 times	3-12 times	13-50 times	more than 50 times

7. How many times were <u>you</u> ever in danger of being injured or killed (i.e., shot at, bombed, torpedoed, pinned down, ambushed, near miss).

0	1	2	3	4
None	1-2 times	3-12 times	13-50 times	more than 50 times

Appendix D

Legacies Combat Scale-Revised

Now I would like to ask you some questions about the nature and extent of your exposure to combat if any, including experiences that took place on land, in the air, or at sea.

1. Were you part of an artillery unit which fired on the enemy.....___
2. Were you on a ship or aircraft that passes through hostile waters or air space.....___
3. Were you stationed at a forward observation post or base camp (i.e. close to enemy lines).....___
4. Did you receive friendly or hostile incoming fire from small arms, artillery, rockets, mortars, or bombs.....___
5. Did you encounter mines or booby traps while on patrol or at your duty station.....___
6. Did your unit receive sniper or sapper fire.....___
7. Was your unit ambushed or attacked.....___
8. Did your unit engage the enemy in a firefight.....___
9. Did you see either Americans or other troops killed or wounded.....___
10. Were you wounded or injured in combat.....___

Appendix E

Mississippi Scale for Combat-Related PTSD

Circle the number that best describes how you feel about each statement:

1. Although I do not have many close personal friends now, before I entered the military I had many friends.

1	2	3	4	5
Not at all true		Somewhat true		Very True

2. I have no guilt over things that I did in the military.

1	2	3	4	5
Not at all true		Somewhat true		Very True

3. If someone pushes me too far, I am likely to become violent.

1	2	3	4	5
Very		Somewhat likely		Very likely

4. If something happens that reminds me of the military, I become very distressed and upset.

1	2	3	4	5
Never		Sometimes		Very Frequently

5. The people who know me best are afraid of me.

1	2	3	4	5
Never		Sometimes		Very Frequently

6. I am able to get emotionally close to others.

1	2	3	4	5
Never		Sometimes		Very Frequently

7. I have nightmares of experiences in the military that really happened.

1	2	3	4	5
Never		Sometimes		Very Frequently

8. When I think of some of the things I did in the military, I wish I were dead.

1	2	3	4	5
Never		Sometimes		Very Frequently

9. It seems as if I have no feelings.

1	2	3	4	5
Never		Sometimes		Very Frequently

10. Lately, I have felt like killing myself.

1	2	3	4	5
Never		Sometimes		Very Frequently

11. I fall asleep, stay asleep and awaken only when the alarm goes off.

1	2	3	4	5
Never		Sometimes		Most of the time

12. I wonder why I am still alive when others died in the military.

1	2	3	4	5
Never		Sometimes		Very Frequently

13. Being in certain situations makes me feel as though I am back in the military.

1	2	3	4	5
Never		Sometimes		Very Frequently

14. My dreams at night are so real that I waken in a cold sweat and force myself to stay awake.

1	2	3	4	5
Never		Sometimes		Very Frequently

15. I feel like I cannot go on.

1	2	3	4	5
Never		Sometimes		Most of the time

16. I do not laugh or cry at the same things other people do.

1	2	3	4	5
Not at all true		Somewhat true		True most of the time

17. I still enjoy doing things that I used to enjoy.

1	2	3	4	5
Never		Sometimes		Always

18. Daydreams are very real and frightening.

1	2	3	4	5
Never		Sometimes		Very Frequently

19. I have found it easy to keep a job since my separation from the military.

1	2	3	4	5
Never		Sometimes		Very Frequently

20. I have trouble concentrating on tasks.

1	2	3	4	5
Never		Sometimes		Very Frequently

21. I have cried for no good reason.

1	2	3	4	5
Never		Sometimes		Very Frequently

22. I enjoy the company of others.

1	2	3	4	5
Never		Sometimes		Always

23. I am frightened by my urges.

1	2	3	4	5
Never		Sometimes		Very Frequently

24. I fall asleep easily at night.

1	2	3	4	5
Never		Sometimes		Very Frequently

25. Unexpected noises make me jump.

1	2	3	4	5
Never		Sometimes		Very Frequently

26. No one, not even my family, understands how I feel.

1	2	3	4	5
Not at all true		Somewhat true		Completely true

27. I am an easy-going, even-tempered person.

1	2	3	4	5
Not at all		Somewhat		Very much so

28. I feel there are certain things that I did in the military that I can never tell anyone about because no one would ever understand.

1	2	3	4	5
Never		Sometimes		Very Frequently

29. There have been times when I used alcohol (or other drugs) to help me sleep or to make me forget about things that happened while I was in the military.

1	2	3	4	5
Never		Sometimes		Very Frequently

30. I feel comfortable when I am in a crowd.

1	2	3	4	5
Never true		Sometimes true		Always true

31. I lose my cool and explode over minor everyday things.

1	2	3	4	5
Never		Sometimes		Very Frequently

32. I am afraid to go to sleep at night.

1	2	3	4	5
Never		Sometimes		Most of the time

33. I try to stay away from anything that will remind me of things which happened while I was in the military.

1	2	3	4	5
Never		Sometimes		All the time

34. My memory is as good as it ever was.

1	2	3	4	5
Never		Sometimes		Most of the time

35. I have a hard time expressing my feelings even to the people I care about.

1	2	3	4	5
Not at all true		Sometimes		Most of the time

Appendix F

Table 1. Correlation matrix analyzed in Model 1 based on the data from Sample 1 (N=48).

	COMBAT	TRAIT ANXIETY	STATE ANXIETY	HI TOTAL	HI GUILT
COMBAT	1.000				
TRAIT A.	-0.182	1.000			
STATE A.	-0.158	0.869	1.000		
HI TOTAL	-0.146	0.806	0.700	1.000	
HI GUILT	-0.104	0.574	0.499	0.713	1.000

Table 2. Correlation matrix analyzed for Model 2 (modified model) based on the data from Sample 1 (N=48).

	PTSD	Combat	Trait Anxiety	State Anxiety	HI Total	HI Guilt	F1
PTSD	1.000						
Comba t	-0.173	1.000					
Trait A.	0.895	-0.155	1.000				
State A.	0.774	-0.134	0.864	1.000			
HI Total	0.784	-0.136	0.876	0.757	1.000		
HI Guilt	0.588	-0.102	0.657	0.568	0.750	1.000	
F1	1.000	-0.173	0.865	0.774	0.784	0.588	1.000

Table 3. Correlation matrix analyzed for Model 2 (modified model) based on the data from Sample 2 (N=25)

	PTSD	Combat	Trait Anxiety	State Anxiety	HI Total	HI Guilt	F1
PTSD	1.000						
Combat	0.394	1.000					
Trait A.	0.637	0.251	0.997				
State A.	0.385	0.151	0.602	1.000			
HI Total	0.456	0.180	0.714	0.431	1.000		
HI Guilt	0.317	0.125	0.496	0.299	0.695	1.000	
F1	1.000	0.394	0.637	0.385	0.456	0.317	1.000

Table 4. Correlation matrix analyzed for Alternate Model 2a based on the data from Sample 1 (N=48).

	PTSD	Combat	Trait Anxiety	State Anxiety	HI Total	HI Guilt	F1
PTSD	1.000						
Combat	-0.176	1.000					
Trait A.	0.804	-0.141	1.000				
State A.	0.937	-0.165	0.858	1.000			
HI Total	0.714	-0.125	0.887	0.762	1.000		
HI Guilt	0.549	-0.096	0.682	0.585	0.769	1.000	
F1	1.000	-0.176	0.804	0.937	0.714	0.549	1.000

Table 5. Correlation matrix analyzed for Alternate Model 2a based on the data from Sample 2 (N=25)

	PTSD	Combat	Trait Anxiety	State Anxiety	HI Total	HI Guilt	F1
PTSD	1.000						
Combat	0.406	1.000					
Trait A.	0.523	0.212	1.000				
State A.	0.700	0.284	0.746	1.000			
HI Total	0.317	0.129	0.606	0.453	1.000		
HI Guilt	0.253	0.103	0.484	0.361	0.798	1.000	
F1	1.000	0.406	0.523	0.700	0.317	0.253	1.000

Table 6. Correlation matrix analyzed for Alternate Model 2b based on the data from Sample 1 (N=48).

	PTSD	Combat	Trait Anxiety	State Anxiety	HI Total	HI Guilt	F1
PTSD	1.000						
Combat	-0.168	1.000					
Trait A.	0.902	-0.152	1.000				
State A.	0.896	-0.151	0.808	1.000			
HI Total	0.811	-0.137	0.899	0.727	1.000		
HI Guilt	0.609	-0.103	0.675	0.546	0.751	1.000	
F1	1.000	-0.168	0.902	0.896	0.811	0.609	1.000

Table 7. Correlation matrix analyzed for Alternate Model 2b based on the data from Sample 2 (N=25)

	PTSD	Combat	Trait Anxiety	State Anxiety	HI Total	HI Guilt	F1
PTSD	1.000						
Combat	0.321	1.000					
Trait A.	0.620	0.199	1.000				
State A.	0.620	0.199	0.385	1.000			
HI Total	0.496	0.159	0.799	0.308	1.000		
HI Guilt	0.344	0.110	0.554	0.213	0.693	1.000	
F1	1.000	0.321	0.620	0.620	0.496	0.344	1.000

Table 8. Correlation matrix analyzed for Alternate Model 2c based on the data from Sample 1 (N=48).

	PTSD	Combat	Trait Anxiety	State Anxiety	HI Total	HI Guilt	F1
PTSD	1.000						
Combat	-0.173	1.000					
Trait A.	0.924	-0.159	1.000				
State A.	0.722	-0.125	0.892	1.000			
HI Total	0.791	-0.136	0.856	0.763	1.000		
HI Guilt	0.593	-0.102	0.641	0.572	0.749	1.000	
F1	1.000	-0.173	0.924	0.722	0 891	0.593	1.000

Table 9. Correlation matrix analyzed for Alternate Model 2d based on the data from Sample 1 (N=48).

	PTSD	Combat	Trait Anxiety	State Anxiety	HI Total	HI Guilt	F1
PTSD	1.000						
Combat	-0.173	1.000					
Trait A.	0.923	-0.159	1.000				
State A.	0.723	-0.125	0.891	1.000			
HI Total	0.791	-0.136	0.856	0.763	1.000		
HI Guilt	0.593	-0.102	0.642	0.572	0.749	1.000	
F1	1.000	-0.173	0.923	0.723	0.791	0.593	1.000

Table 10. Correlation matrix analyzed for Model 3 based on the data from Sample 2 (N=25)

	PTSD	Combat	Trait Anxiety	State Anxiety	HI Total	EMG R.A	F1
PTSD	1.000						
Combat	0.423	1.000					
Trait A.	0.363	0.154	1.000				
State A.	0.283	0.120	0.778	1.000			
HI Total	0.200	0.085	0.550	0.428	1.000		
EMG R.A.	0.197	0.083	0.543	0.697	0.299	1.000	
F1	1.000	0.423	0.363	0.283	0.200	0.197	1.000

Table 11. Correlation matrix analyzed for Alternate Model 3a based on the data from Sample 2 (N=25)

	PTSD	Combat	Trait Anxiety	State Anxiety	HI Total	EMG R.A	F1
PTSD	1.000						
Combat	0.407	1.000					
Trait A.	0.285	0.116	1.000				
State A.	0.397	0.162	0.716	1.000			
HI Total	0.163	0.066	0.572	0.410	1.000		
EMG R.A.	0.283	0.116	0.511	0.713	0.292	1.000	
F1	1.000	0.407	0.285	0.397	0.163	0.283	1.000

Table 12. Correlation matrix analyzed for Alternate Model 3b based on the data from Sample 2 (N=25)

	PTSD	Combat	Trait Anxiety	State Anxiety	HI Total	EMG R.A	F1
PTSD	1.000						
Combat	0.264	1.000					
Trait A.	0.475	0.125	1.000				
State A.	0.559	0.147	0.265	1.000			
HI Total	0.333	0.088	0.702	0.186	1.000		
EMG R.A.	0.509	0.134	0.242	0.911	0.170	1.000	
F1	1.000	0.264	0.475	0.559	0.333	0.509	1.000