

2000

Traumatic Life Events, Posttraumatic Stress Disorder, and Health Outcomes in a Low -Income, Primary Care Population.

Bradford West Applegate

Louisiana State University and Agricultural & Mechanical College

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**TRAUMATIC LIFE EVENTS, POSTTRAUMATIC STRESS DISORDER, AND
HEALTH OUTCOMES IN A LOW-INCOME, PRIMARY CARE POPULATION**

A Dissertation

**Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy**

in

The Department of Psychology

by

Bradford West Applegate

B.S., Virginia Polytechnic Institute and State University, 1992

M.S., Mississippi State University, 1995

August 2000

UMI Number: 9984309

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ACKNOWLEDGEMENTS

The dissertation is the crowning jewel in a lengthy journey of classes, research, and clinical training that culminates in the doctoral degree for psychology. As such, I will acknowledge several people, some of whom had little or no direct bearing on this particular project.

First, I would like to acknowledge my wife, Heather, who provided tremendous amounts of support, advice, and motivation throughout the past 10 years. Without your unwavering support, this project would not have been completed. Not by a long shot. It won't be long until we are the "Doctors Applegate!" Graduate school is a long and stressful process that we weathered pretty darn well.

Second, I would like to acknowledge the guidance of my major professor, Phillip Brantley, who took a chance on an unproven, underachieving student and turned him into a "finisher." Your supervision and guidance over the last six years have had a tremendous impact on my professional and personal life. You managed to see and bring out my potential as a psychologist when even I had my doubts. I will always be proud to call you my major professor as well as my friend.

Third, I would like to acknowledge the contributions of my dissertation committee members who sacrificed time and mental energy, and had excellent suggestions throughout this project. Thank you Drs. Waters, Hawkins, Gouvier, Boudreaux, and Windhauser. This project certainly is better because of your contributions.

Fourth, I would like to acknowledge the research team that made this project together. Phillip Brantley, Glenn Jones, Isabel Scarinci, Dan Mehan, Shawn Jeffries, Steve Ames, Serrhel Adams, and Melanie Boyce all put in great amounts of time and efforts in the design, data collection, and analysis of parts of this project.

A sincere thank you goes out to goes out to “The Team,” the group of Brantley students that have worked, played, supported, and helped each other throughout graduate school. Being a part of The Team made graduate school much more pleasant than without. You all have inspired, supported, ridiculed, laughed, and even cried with me. Thanks for the memories.

I would like to thank my family for all the emotional and financial support they have provided. Mom, Dad, Bill, Christine, and everybody else - thanks. Of course, I would be remiss in not thanking the pets (Raleigh, Pablo, & Virgil) that have provided a wonderful emotional outlet from the stresses of graduate school. Thanks, dudes.

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ABSTRACT

Research suggests that individuals with Posttraumatic Stress Disorder (PTSD) have significant dysregulation in a number of physiological indices, especially the sympathetic nervous system (SNS) and hypothalamic-pituitary-adrenal (HPA) axis. Biological theories of PTSD propose that prolonged SNS and HPA axis dysregulation places individuals with PTSD at risk for development of medical morbidity and impairments in health status.

The present study examined the following research questions: (a) what is the prevalence of traumatic life events and PTSD in a low-income primary care population? (b) does PTSD predict impairments in self-reported health status after controlling for age, alcohol abuse, tobacco use, and obesity? and (c) does PTSD predict the presence of physician diagnosed medical disease?

The sample included 431 randomly selected adult patients recruited from primary care clinics at a public teaching hospital in the state of Louisiana. The sample consisted predominately of uninsured, African-American, low-income females. The results indicated that prevalence of at least one traumatic life event (88%) was similar to that of community surveys. For females, the lifetime prevalence of PTSD was significantly higher than in community samples. The same was not true for males, however. Individuals with PTSD were likely to have at least one other comorbid mental disorder. The median duration of symptoms for those with a PTSD diagnosis was 12 months, but considerable variability in remission rates was found as a function of traumatic event type. Individuals who developed PTSD from some type of

interpersonal violence had a median symptom duration of 48 months, compared to 12 months for either other events directly experienced, or events experienced by a close friend or loved one.

Logistic regression analyses revealed that PTSD was predictive of impairments in self-reported health status, but only when compared to control subjects with no history of mental disorder. Logistic regression analyses also revealed that PTSD was predictive of presence of circulatory system disease, but only when compared to control subjects with no history of mental disorder. Results provided tentative support for the notion that PTSD, like other psychiatric conditions, is associated with increased risk of impaired health status and medical disease.

INTRODUCTION

There is a rich history documenting the adverse physiological and psychological effects of exposure to traumatic life events. Early accounts of adverse reactions to trauma came from reports of civilian disasters such as train wrecks, fires, and other disasters as well as war-related traumas (Kinzie & Goetz, 1996). Descriptions of these reactions carried several different labels and had differing descriptions of symptom clusters. A comparison of these symptom clusters, however, reveals similar symptom profiles. For example, Hyams, Wignall, and Roswell, (1996) examined several of the war-related conditions, and found that symptoms common among all of the 'syndromes' were sleep disturbance, nightmares, hyperarousal/irritability, heart palpitations, and shortness of breath. Thus, although adjustment to trauma was widely written about, there was little attempt to categorize these various adjustment reactions. It wasn't until 1980, with the publication of the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III, American Psychiatric Association [APA], 1980), that PTSD was made a diagnosable mental disorder. Since then, however, a tremendous amount of literature has been published about traumatic life events and their sequelae.

There is also a broad literature base exploring the physiological and psychological adjustments to a variety of stressors. This body of research began with the work of Hans Selye and the development of the general adaptation syndrome (GAS, Selye, 1958), a model used to describe the negative effects of stress and stress hormones on the body. This research initiated the current focus of sympathetic

nervous system (SNS) and hypothalamic-pituitary-adrenal (HPA) axis activity after exposure to stress. Selye examined the negative effects of prolonged and intense exposure to stress, and documented their effects on organ systems (Selye, 1958). The end organ damage found in animals exposed to stress was evidence for the so called “stress diseases.” Selye also conducted animal experimental studies that linked stress to a variety of “diseases of adaptation,” including hypertension, kidney disease, myocardial infarction, and inflammatory diseases (Selye, 1958).

The groundbreaking studies of Selye provided the foundation for extensive inquiry into stress-illness relationships in humans. Research has examined the role of major life events (e.g., divorce), minor stressors (e.g., arguments with spouse) and laboratory stressors (e.g., cold pressor task, exposure to flu virus) on physiological reactivity as well as the development and/or progression of disease. Both minor and major stressors have been linked with disease onset and progression (see Brantley & Jones, 1993; Stein & Miller, 1993 for reviews). Stress has been implicated in a number of illnesses including hypertension, infectious diseases, myocardial infarction, inflammatory diseases, and several others.

Interestingly, absent in much of the stress-illness literature has been examinations of traumatic life events and PTSD. The physiological reactivity and health outcomes of individuals exposed to traumatic stressors has only recently gained systematic attention. This may be due to the relatively recent inclusion of PTSD as a diagnosis in the DSM system. Traumatic life events were also previously thought to

happen to only a select few individuals as well. Recent research, however, suggests that traumatic life events are fairly common experiences.

The literature review in this paper will cover the following topics: 1) an overview of PTSD, 2) theories regarding the etiology of PTSD (including behavioral theory, cognitive theory, and biological theory), 3) traumatic life events and health outcomes, and 4) PTSD and health outcomes.

REVIEW OF THE LITERATURE

Overview of PTSD

PTSD is a cluster of symptoms that emerges in individuals who have been exposed to some type of traumatic life event, such as a serious car accident, natural disaster, military combat, and others (APA, 1994). Appendix A presents the diagnostic criteria for the disorder. PTSD is characterized by three types of symptoms after exposure to a traumatic life event: (a) re-experiencing, (b)arousal, and (c) avoidance symptoms (APA, 1994). Re-experiencing symptoms include recurrent and intrusive distressing recollections and/or dreams of the event, acting or feeling as if the event were recurring, and intense psychological distress and/or psychophysiological reactivity upon exposure to cues that resemble or represent the event (APA, 1994). Avoidance symptoms include efforts to avoid cues associated with the event, inability to recall important aspects of the trauma, markedly diminished interest in daily activities, feelings of detachment from others, restricted range of affect, or sense of a foreshortened future (APA, 1994). Arousal symptoms include difficulty sleeping, irritability and/or anger, difficulty concentrating, hypervigilance, and/or exaggerated startle response. Although the historical foundations of the disorder are not presented here, Appendix B presents a review of the history of the disorder and its entry into the DSM system.

Epidemiological research indicates that exposure to traumatic life events and PTSD are fairly common in the community. To date, five studies examined the prevalence of traumatic life events and PTSD symptomatology in the general

population. The first two (Helzer, Robins, and McEvoy 1987; Davidson, Hughes, Blazer, et al., 1991) studies were conducted as part of the Epidemiological Catchment Area survey ([ECA], Regier, Myers, Kramer, et al., 1984). Both studies found prevalence rates of PTSD at 1%, but neither study assessed the prevalence of traumatic life events. The diagnostic criteria and methods used to assess PTSD have since been revised, and have found substantially higher rates of the disorder. The next three community studies found traumatic life events to be fairly common in the community, with lifetime event exposure percentages ranging from 39 to 90% (Breslau, Davis, Andreski, & Peterson, 1991; Breslau, Kessler, Chilcoat, Schultz, Davis, & Andreski, 1998; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). These studies found the conditional risk of developing PTSD after traumatic event exposure ranged between 9 and 24%. The overall lifetime prevalence of PTSD ranged from 8-12% in these studies. Furthermore, women appear to be at greater risk of developing PTSD symptoms (Kessler et al., 1995). Lastly, a wide range of events are associated with the development of PTSD, with physical assault and military combat being among the most commonly associated with a PTSD reaction (Kessler et al., 1995).

Until recently, no data were available to examine the course of PTSD symptoms. However, recent military, disaster, and community surveys indicate that PTSD is a relatively chronic condition (Freedly and Donkervoet, 1995). For example, Breslau and Davis (1992) found that more than 57% of the subjects in a community sample that had PTSD reported symptom duration of greater than one year. Furthermore, 34% of the sample experienced PTSD symptoms for more than three

years. These data are similar to the Kessler et al. (1995) NCS study, which examined median remission times in persons with PTSD who had or had not received treatment. They found that the median duration of PTSD symptoms in treatment seekers to be 36 months, compared with 60 months for non-treatment seekers. Interestingly, one third of each condition reported PTSD symptom duration of at least 10 years. Other community and military studies examining the duration of PTSD symptoms have yielded similar results (Breslau et al., 1998; Kulka, Schlenger, Fairbank, Hough, Jordan, Marmar, & Weiss, 1990).

Results from several studies indicate that individuals with PTSD are likely to have at least one comorbid mental disorder (Brady, 1997). Studies with different sample types such as community (e.g., Kessler et al., 1995), Vietnam combat veterans (Kulka et al., 1990) and disaster samples (e.g., Green, Lindy, Grace, & Leonard, 1990) have shown comorbidity rates ranging from 60 to 98%. It appears that the high comorbidity rates in persons with PTSD appear to be a result of PTSD symptoms rather than a result of traumatic life event exposure. For example, both Breslau and Davis (1987) and Boudreaux, Kilpatrick, Resnick, Best, & Saunders (1998) found that PTSD status was the major predictor of other mental disorders in individuals exposed to traumatic life events. In other words, after PTSD status was controlled for, traumatic life event exposure itself was not predictive of presence of other mental disorders such as depression, panic disorder, or phobias.

Little is known about the prevalence of traumatic life event exposure or PTSD in low-income primary care populations. One recent report from Samson, Benson,

Beck, Price, and Nimmer (1999) found that 38% of those patients who endorsed psychological distress on a screening measure received a diagnosis of PTSD after a subsequent interview. Given the high rates of utilization and cost to primary care physicians associated mental disorders, it is important to effectively identify and treat those in distress after exposure to traumatic events.

Etiological Models of PTSD

Behavioral Theory

Behavioral theories of PTSD derive mainly from human and animal learning literature. Keane and colleagues (Keane, Zimering, & Caddell, 1988; Keane, Fairbank, Caddell, Zimering, & Bender, 1985) proposed a model of PTSD based on Mowrer's (1939) two-factor theory of fear and avoidance. This model proposes that traumatic life event exposure serves as a powerful unconditioned stimulus that evokes a severe fear reaction. Environmental cues present during the traumatic event become conditioned stimuli capable of generating an intense conditioned response. Subtle environmental cues such as sights, smells, tastes, sounds and other contextual factors become associated with fear. Higher order conditioning then occurs, where these environmental cues actually serve as unconditioned stimuli capable of eliciting fear in other situations. Avoidance symptoms serve as an escape mechanism for situations that have become associated with fear. This model, however, fails to account for why some people develop PTSD to a particular stimulus and others do not.

Cognitive Theory

Cognitive models of PTSD (e.g., Foa, Steketee, & Olasov-Rothbaum, 1989) are based on information processing literature and derive mainly from Lang's (1977) bioinformational processing model of fear. Bioinformational theory proposes that a 'fear network' stored in memory contains emotional, situational, and response information regarding threat and potentially harmful stimuli. It is this structure that allows one to effectively deal with potentially harmful situations. Foa et al.'s (1989) model states that a crucial element in the development of PTSD is that the trauma violates safety assumptions in memory, leading to an inability to discriminate threat from safety. Furthermore, the intensity of the fear experienced after traumatic event exposure leads to the traumatic memory being more easily accessed than other memory structures. In this context, the wide ranging fear, reexperiencing, arousal and avoidance seen in PTSD results from the constant activation of this fear structure. An important aspect of PTSD in this model is the element of unpredictability and uncontrollability of the traumatic event, which are very important to the development and maintenance of fear and avoidance (Foa, Zinbarg, & Olasov-Rothbaum, 1992).

Biological Theory

Biological theories of PTSD posit that the arousal, hypervigilance, and re-experiencing symptoms seen in the disorder are due to abnormalities in neurohormonal functioning after exposure to trauma. Biological theories propose that individuals who develop PTSD symptoms after exposure to trauma have physiological responses that

differ from those who do not develop PTSD. The abnormal responses involve both the SNS and the HPA axis.

SNS Dysregulation in PTSD. It is well known that stress is associated with SNS and HPA activity that prepares a fight or flight response. SNS activity, however, is time-limited and typically ends shortly after the stressful situation has been resolved. Individuals with PTSD, however, continue to show dramatic displays of physiological reactivity (e.g., increases in heart rate and blood pressure) after presentation of trauma-related stimuli (Prins, Kaloupek, & Keane, 1995). Studies that have utilized exposure-control conditions (e.g., a control group exposed to a similar traumatic life event but without PTSD), have also reliably demonstrated excessive SNS reactivity in PTSD subjects (Prins et al., 1995). Similarly, studies examining exposure to noxious but non-trauma related stimuli (e.g., acoustic startle) have found increased heart rate in PTSD subjects as compared to control subjects (Butler, Bratf, Rausch, Jenkins, Sprock, & Geyer, 1990; Pallmeyer, Blanchard, & Kolb, 1986).

Other evidence to suggest that individuals with PTSD have abnormal SNS activity comes from reports that have examined norepinephrine (NE) functioning (a major neurohormone involved in SNS activity) in PTSD and control subjects. Combat veterans with PTSD show a pattern of downregulation of alpha-2 adrenergic receptors (a common NE receptor) (Lerer, Bleich, & Kotler, 1987; Perry, Giller, & Southwick, 1987). In addition, PTSD, but not control, subjects experienced acute anxiety symptoms of increased heart rate, increased blood pressure, feelings of panic, and re-experiencing symptoms after the injection of yohimbine (an alpha-2 autoreceptor

antagonist that facilitates NE functioning) but not placebo (Southwick, Morgan, Bremner, Grillon, Krystal, Nagy, & Charney, 1998).

Research examining urinary excretion of NE and its major metabolite have been mixed. Two reports (Kosten, Mason, Giller, et al., 1987; Yehuda, Southwick, Mason, et al., 1992) showed increased urinary NE output, but others have failed to reliably show differences between PTSD and control subjects on baseline plasma NE output (Blanchard, Kolb, Prins, et al., 1991; McFall, Murburg, Ko, et al., 1990). Studies examining 24-hour plasma NE levels are similarly mixed. For example, Yehuda, Siever, Teicher, et al., (1999) found that combat veterans with PTSD had significantly greater 24 hour plasma NE excretion than normal controls, whereas Murburgh et al. (1995), using a slightly different technique, could not separate PTSD subjects from controls.

Taken in sum, these results suggest that individuals with PTSD demonstrate significant SNS hyper-reactivity to trauma-related stimuli compared to individuals with no history of PTSD. However, it is still unclear whether individuals with PTSD show higher baseline levels of catecholaminergic functioning or hyper-reactivity to more neutral stimuli.

HPA functioning in PTSD. As reported above, the HPA axis becomes activated upon exposure to stress. The degree of HPA activity has been shown to correlate with the magnitude of the stressor (Selye, 1958). That is, the more stressful the event, the greater HPA activity. Research investigating HPA activity in PTSD subjects has demonstrated significant dysfunction in HPA axis indices when compared

to normal controls. For example, individuals with PTSD show lower levels of urinary and plasma cortisol excretion (the primary stress hormone released from the adrenal medulla) than control subjects (Yehuda, 1998a). This result has been demonstrated with a number of different methodologies and in different PTSD populations (Yehuda, Giller, Levengood, Southwick, & Siever, 1995, Yehuda, 1998b). PTSD subjects also demonstrate decreased cortisol functioning in relation to psychiatric controls (Yehuda, 1998a, 1998b). Preliminary evidence also indicates that individuals with PTSD have higher concentrations of corticotropin releasing hormone (CRH) in cerebrospinal fluid (Bremner, Licinio, Darnell, et al., 1997).

Yehuda and colleagues (Yehuda 1998a, 1998b; Yehuda, Giller, Levengood, et al., 1995) have recently proposed a model to explain the pattern of HPA dysregulation seen in PTSD. The model proposes that individuals with PTSD show a sensitization of the HPA axis, with enhanced negative feedback inhibition of cortisol to the other HPA components (Yehuda 1998a). The dysregulation arises from an initial increase of CRH release from the hypothalamus, leading to decreased responsivity of the pituitary to CRH, but a *hyper-responsivity* to cortisol. This effectively explains the increased levels of CRH and decreased levels of cortisol found in many PTSD samples.

As originally posited by Selye (1958), disruptions in homeostatic functioning resulting from chronic levels of stress places individuals at risk for medical conditions later in life. Given that the pattern of HPA axis functioning in PTSD subjects is opposite of those seen in chronically stressed individuals and/or animals, it is unclear

as to which specific conditions that individuals with PTSD would be at risk. Lower levels of circulating cortisol may place individuals at risk for inflammatory processes given cortisol's role as an anti-inflammatory agent. Excessive SNS reactivity may be linked with a host of cardiovascular and other ANS end organ dysfunction including hypertension, myocardial infarction, and others. Therefore, the development and maintenance of PTSD symptoms after traumatic event exposure may be a risk factor for the development of subsequent medical morbidity.

Traumatic Life Events, PTSD, and Health Outcomes

There is a growing literature base that has examined the role of traumatic life event exposure and/or PTSD on physical health outcomes. Comparison of these studies reveals differences in methodological sophistication, outcome variables, and populations studied. The literature consists of studies that have investigated traumatic life event exposure or PTSD in community, specific exposure populations, and combat veterans. These studies have examined a number of health outcomes, including self-reported health status, quality of life, health care utilization and medical morbidity. Methodological differences in outcome variables also exist, such as the use of self-reported versus objective measures of health status and medical utilization. Nevertheless, traumatic life event exposure and PTSD are robustly associated with impairments in self-reported health status and morbidity. However, evidence regarding objective health outcomes measures is mixed.

Traumatic Life Events and Health Outcomes

Studies investigating the role of traumatic life events on physical health functioning have utilized a variety of methods. The most common and economical of these involves self-reported health impairments in samples known to have been exposed to traumatic life event (e.g., rape victims). Although these studies do not control for PTSD, the high rates of PTSD seen in these populations makes them a convenient and economic option.

Self-Reported Health Status Impairments. The most commonly used measures of health status include the Medical Outcomes Study Short Form-36 ([SF-36], Hays, Sherbourne, & Mazel, 1993) and the Behavioral Risk Factor Survey Satellite ([BRFSS], Centers for Disease Control [CDC], 1988). These instruments have been administered in a number of different trauma populations. A consistent finding across studies is that individuals exposed to traumatic life events report more health status impairments than control subjects (Friedman & Schnurr, 1995).

Only one study has reported on the role of traumatic life events in physical health functioning in a community sample. Ullman and Siegel (1996) tested whether individuals with a traumatic life event exposure history were more likely to report poorer health status and more medical problems than those without such history. Subjects were assessed for traumatic life event history via a set of interview questions. The outcome variables in this study consisted of questions developed from the Rand Medical Outcomes Study (Tarlov, Ware, Greenfield, Nelson, Perrin, & Zubkoff, 1989), and via a checklist of common chronic medical conditions. Results indicated

that traumatic life event exposure status was predictive of poorer self-rated health status. Furthermore, those subjects with a traumatic life event exposure history were almost five times more likely to report a chronic medical condition than those without a history of exposure.

The health status of violent crime victims and the sexually abused have been studied extensively (Resnick, Acierno, & Kilpatrick, 1997). Several studies have examined self-reported health perceptions, medical utilization, and prevalence of chronic illnesses in these populations. These studies have all indicated that victims of sexual assault report more health status impairments than control subjects (Golding, Stein, Siegel, Burnam, & Sorenson, 1988; Kimerling & Calhoun, 1994; Koss, Woodruff, & Koss, 1990; Resnick et al., 1997). Furthermore, the degree of health impairment correlates positively with the magnitude of assault (Koss et al., 1990; Resnick et al., 1997).

A third area of traumatic life event research that has received attention in the literature comes from studies examining the health sequelae of combat exposure. Two comprehensive studies of health status of Vietnam veterans have been published, the CDC's Vietnam Experience Survey ([VES], CDC, 1988), and the National Vietnam Veterans Readjustment survey ([NVVRS], Kulka et al., 1990). These studies compared physical health in cohorts of Vietnam theater and Vietnam non-theater veterans via the BRFSS and SF-36. In the VES, Vietnam veterans were almost twice as likely to rate their health as 'poor' or 'fair' on the BRFSS than non-theater veterans. Vietnam veterans were also more likely to report the presence of medical conditions

(in 10 out of 13 medical conditions, odds ratios were significant), an increased number of hospitalization days, more limitations in physical activities, and more use of prescription medications than their non-theater veteran counterparts (CDC, 1988).

Results of the NVVRS indicate that Vietnam-theater veterans who were exposed to high levels of war-zone stress scored significantly lower on self-reported health status than both non-theater Vietnam-era veterans and control subjects. The authors also examined the groups on self-reported chronic health problems. Results indicated that Vietnam-theater veterans with high war-zone stress exposure reported significantly more chronic health problems than both non-theater Vietnam-era veterans and civilian control subjects. Importantly, war-zone stress and not just theater service was the major risk factor for self-reported health impairments and chronic health problems in this study (Kulka et al, 1990).

The Iowa Persian Gulf Study Group (1997) examined the presence of self-reported health status in Persian Gulf War (PGW) veterans and control subjects. Physical health status was measured via questions from the SF-36 and the BRFSS. Analyses were conducted to examine if group differences in psychological and physical health status differed according to PGW exposure and/or military branch service status. Results indicated that PGW veterans reported significantly worse health status across all 8 domains of the SF-36.

Objective Health Status Impairments. Objectively-rated health status impairments (e.g., presence of disease or medical diagnosis confirmed by laboratory test or physician) are less conclusive than self-reported impairments. This is, in part,

due to methodological inconsistency in addition to equivocal results. Early studies noted a high prevalence of sexual abuse and other criminal victimization in medical populations diagnosed with chronic pelvic pain, irritable bowel syndrome, and other ob/gyn medical problems (Drossman, Leserman, Nachman, Li, Gluck, Toomey, & Mitchell, 1990; Harrop-Griffiths, Katon, Walker, Holm, Russo, & Hickock, 1988; Wurtele, Kaplan, & Keairnes, 1990). Other research has noted a high frequency of gastrointestinal and cardiovascular problems in prisoners of war (POWs) when compared to non-POW military controls (Beebe, 1975; Goulston, Dent, Chaipuis, et al., 1985).

Investigations of objective health outcomes in combat populations have generally yielded inconclusive results. In the CDC VES study (CDC, 1988), a random sample of 245 participants underwent extensive physical and laboratory testing to investigate whether Vietnam veterans experienced more objectively rated physical problems than non-theater veterans. Physical exam results did not substantiate Vietnam veterans claims of increased number of medical conditions. Vietnam theater veterans were no more likely than Vietnam non-theater veterans to have any of the 13 medical conditions examined, contrary to the self-report data. Further analysis revealed that Vietnam veterans showed physical impairments in two areas: hearing and sperm count. A notable weakness of this study is that the investigators did not control for traumatic life event exposure (potentially measured by degree of combat exposure) or PTSD status. Therefore, it is not known whether these variables may have mediated an impaired health response in this study.

PTSD and Health Outcomes

Research examining the health outcomes of individuals with PTSD has focused primarily on combat veterans. Furthermore, most of the studies conducted on this topic have utilized self-report outcome measures. Some studies have examined the relative power of PTSD to predict health outcome after controlling for traumatic life event exposure, whereas others have examined both self-report and physician rated medical conditions.

Self-Reported Health Status. Only one study exists that examines medical outcomes in a community sample of PTSD. In an extension of their earlier epidemiological study of PTSD (Breslau et al., 1991), Breslau and Davis (1992) examined differences in symptom expression and self-reported mental health in 93 HMO enrollees with either acute or chronic PTSD. Chronic PTSD was defined as duration of PTSD symptoms for greater than one year. Participants self-reported medical conditions from a checklist of 11 common medical problems. Results indicate that individuals with chronic PTSD symptoms reported almost twice as many medical conditions than the acute PTSD group.

Studies examining health outcomes of veteran populations have generally focused on self-report measures. In a reanalysis of the CDC VES study data, Zatzick, Marmar, Weiss, et al. (1997) examined the role of PTSD in the physical health of Vietnam veterans. Using logistic regression, the authors examined whether PTSD status (as measured by the Mississippi Scale) was predictive of subjective well-being and self-reported physical status (as measured by the BRFSS). After controlling for a

number of service related and demographic variables. PTSD was still predictive of impairments in well-being and physical status. Boscarino (1997) also reexamined the CDC VES database, and examined the role of PTSD in specific self-reported medical conditions. Boscarino conducted logistic regression analyses examining whether PTSD was associated with specific disease states in 10 major categories (cancer, circulatory, digestive, musculoskeletal, genitourinary, endocrine, nervous system, dermatological, respiratory, and chronic diseases). Data were obtained from a face to face interview with a physician's assistant. Data from the objective medical assessment were not used in this study. Results indicate that PTSD was associated with circulatory, digestive, musculoskeletal, endocrine, nervous system, and respiratory system diseases after several demographic and service related variables were controlled for. Boscarino concluded that this study provided positive evidence for a stress-illness relationship. The results of these studies mirror other combat studies of Vietnam (e.g., Kulka et al., 1990; Long, Chamberlain, and Vincent (1992); Wolfe, Schnurr, Brown, & Furey, 1994) and Persian Gulf War (Baker, Mendenhall, Simbartl, Magan, & Steinberg (1997) veterans that found that veterans with PTSD report worse health status and more medical problems than control veteran subjects.

Objective Health Status Impairments. There are few published reports that have examined objectively-rated health impairments in individuals with PTSD. Irwin, Falsetti, Lydiard, Ballenger, Brock, and Bremner (1996) investigated the prevalence of mental disorders in 50 continuous patients with Irritable Bowel Syndrome (IBS) in a gastroenterology practice. Irwin et al. drew upon literature (e.g., Drossman et al.,

1990) reporting that a significant number of individuals with gastrointestinal disorders such as IBS report a history of childhood physical or sexual abuse. Subjects in this study were assessed via structured interview. Results indicated that 27 out of 54 (54%) of the IBS patients in this study were diagnosed with a psychiatric disorder at some point in their lifetime. Twenty-two patients in the entire sample recorded a significant trauma history, with 18 out of the 54 (36%) subjects receiving a diagnosis of PTSD.

Recently, Beckham, Moore, Feldman, Hertzberg, Kirby, and Fairbank (1998) examined both self-reported and physician rated medical conditions in a group of 276 Vietnam veterans either with or without PTSD. Subjects filled out questionnaires inquiring about health status, and chart reviews were conducted to evaluate for evidence of medical disorders. Multivariate analyses of covariance (controlling for age, socioeconomic status, minority status, combat exposure, alcohol use, and tobacco use) indicated that individuals with PTSD had higher ratings of self-reported health complaints, lifetime physical conditions, and current physical conditions. Furthermore, individuals with PTSD had more medical conditions in more categories than those without a PTSD diagnosis. A simultaneous regression examining PTSD indicated that PTSD symptom severity was related to symptom severity, somatization, and tobacco use. Interestingly, somatization was related only to the self-reported outcome variables. The results of this study are in contrast to Shalev et al. (1990), who found that Israeli combat veterans with PTSD reported significant medical

impairments as compared to control subjects, but that their self-reported conditions were not borne out in physical examination and laboratory findings.

In summary, there is sufficient evidence to indicate that both traumatic life event exposure and PTSD are associated with self-reported impairments in health status. Indeed every study reviewed in both the traumatic life event exposure and PTSD literature reveals that these individuals rate their health poorer than control subjects. This is consistent across community, specific event, and combat veteran populations. In addition, there is evidence indicating that PTSD mediates the self-reported health status impairments in exposed populations. There is also some evidence to suggest that severely traumatizing events such as rape and PTSD reactions are associated with increased medical utilization. Lastly, there is some evidence to suggest that PTSD is associated with increased risk of objectively diagnosed medical disorder, although the literature is decidedly mixed on the topic, and is in need of clarification.

Recently, Resnick, et al. (1997) offered a path model describing the potential impact that criminal victimization may have on women's health. The model suggests that health impairments can arise from several different mechanisms, including injury during assault, impairment in immune functioning, and increased health risk behaviors. This model can be extended to other PTSD-provoking events as well. It is true that some traumatic life events are associated with physical injury, but Friedman & Schnurr (1995) note that this is a fairly rare phenomenon in the published literature. Health risk behaviors risks such as alcohol, tobacco, and drug use are significantly

more prevalent among individuals with PTSD, and need to be controlled for in studies examining health status impairments (Breslau et al., 1991).

The Present Study

Summary and Rationale

There is sufficient epidemiological evidence to suggest that traumatic life events are highly prevalent in the community (Breslau et al., 1998; Kessler et al., 1995). Furthermore, PTSD is one of the most prevalent DSM-IV anxiety disorders. However, little research has documented the prevalence of traumatic life events and PTSD in lower-income populations who may be at particular risk for traumatic life event exposure. Sociodemographic variables such as poverty or minority status may lead to increased criminal victimization, hazardous living or working conditions, and increased mortality, all of which may place low-income and minority populations at higher risk for traumatic event exposure and PTSD. Recognition and treatment of mental disorders has been associated with increased quality of life, decreased symptom expression, and decreased medical utilization (Borus and Olendzki, 1985; Kessler, Steinwachs, and Hankin, 1982).

Individuals with history of PTSD appear to have significant ANS and HPA axis dysregulation. Studies have consistently shown that individuals with PTSD show increased levels of NE metabolites and other aspects of NE functioning that are indicative of impaired SNS functioning. This dysregulation has the potential to affect numerous physiological systems, including the cardiovascular system and many other organ systems (van der Kolk, 1997).

In addition, recent evidence has shown that the PTSD response is associated with decreased levels of cortisol functioning and a proposed decreased ability of the HPA axis to terminate ANS arousal (Resnick et al., 1997). Because ANS and HPA axis dysregulation have been associated with increased risk of medical sequelae, individuals with chronic PTSD may be at risk for medical conditions, impaired health status, and increased medical utilization (Blanchard, 1990; van Der Kolk, 1997).

Research Questions and Data Analytic Strategy

1.) Since there is little data regarding the prevalence of traumatic events and PTSD in low-income, primary care settings, the first goal of the current study was to identify those participants who have experienced traumatic life events, report the lifetime and point prevalence of PTSD, and identify other parameters such as patterns of comorbidity and symptom course. It was hypothesized that this low-income population would be at greater risk for traumatic event exposure and prevalence of PTSD when compared to a recent community survey using DSM-IV criteria (Breslau et al., 1998). In order to test the hypothesis that this low-income, primary care sample is at greater risk for both traumatic life event exposure and PTSD, chi-squares and t-tests were conducted.

2.) Does PTSD predict impairments in self-reported health status after risk factors of age, alcohol abuse, tobacco use, and obesity status are controlled for? Based on the literature, it was hypothesized that PTSD status would be an independent predictor of impaired health status when compared to control subjects who have no history of mental disorder. A secondary goal was to determine whether the PTSD status would

be as or more predictive of self-reported health status in comparison to a psychiatric control group with depression but no history of PTSD. This hypothesis was tested by using hierarchical logistic regression analysis, with self-reported health status from the BRFSS as the dependent variable, and age, tobacco use history, alcohol abuse history, obesity status, and PTSD status entered as independent predictors.

3.) Does PTSD predict presence of physician-rated medical disease after the risk factors of age, alcohol abuse, tobacco use, and obesity are accounted for? PTSD is marked by persistent ANS system dysregulation, which has been associated with the development of a wide variety of medical illnesses. Therefore, it is predicted that PTSD status will serve as an independent predictor of medical illness. Similar to aim 2, a secondary goal was to determine whether PTSD status would be as or more predictive of medical illness presence as a psychiatric control group. This aim was tested by using hierarchical logistic regression analysis, with presence of physician-rated medical disease as the dependent variable, and age, tobacco use history, alcohol abuse history, obesity status, and PTSD status entered as independent predictors.

MATERIALS AND METHODS

Participants

The sample consisted of 433 adult male and female primary care patients randomly recruited from the Family Practice and Internal Medicine clinics of Earl K. Long Medical Center (EKLMC) in Baton Rouge, Louisiana. EKLMC is a public teaching hospital that provides health care to predominantly low-income and minority patients.

Measures

Demographics Questionnaire

This is a 16-item questionnaire designed for this study. It includes questions assessing age, gender, ethnicity, marital status, educational level, occupation, income, and insurance coverage (see Appendix A).

Diagnostic Interview Schedule for DSM-IV

The DIS-IV (Robins, Cottler, Bucholz, & Compton, 1996) is a structured diagnostic interview designed to assess the history and presence of DSM-IV mental disorders. It was developed as a research tool to provide a reliable and valid determination of the presence of mental disorders in a variety of research settings. For example, previous versions of the DIS have been used to determine the prevalence of mental disorders in the community in both the NCS (Kessler et al., 1995) and ECA studies (Davidson et al. 1991). At the time of the present study, there was no data regarding the reliability and validity of the DIS-IV, although earlier editions of the DIS have been shown to demonstrate adequate reliability. Previous test-retest reliability

coefficients have ranged .37 to .59, and concurrent validity kappas (between the DIS and independent psychiatrists) have ranged from .47 to 1.0 (Helzer, Spitznagel, & McEvoy, 1987).

The DIS-IV was designed to be used by trained lay interviewers for the purposes of cost containment and manpower utilization. The interviewers in the present study included 4 advanced doctoral students in clinical psychology and one masters' level clinician. All were trained to give the DIS-IV to ensure reliable and valid administration. Each participant's responses to the questions were double-entered into the scoring program provided by the publisher. For the purposes of the present study, only certain modules were administered. Modules pertaining to disorders arising in childhood (i.e., Attention Deficit Hyperactivity Disorder, Separation Anxiety Disorder) were not administered, although modules pertaining to PTSD, tobacco, alcohol, drug use, and other adult DSM-IV axis I mental disorders were administered to determine comorbidity between PTSD and other mental disorders.

Patient's Global Health Status Rating (GHSR)

The GHSR (Barsky, Wyshak, & Klerman, 1986) is a physician-rated scale of health status on a seven point Likert scale, ranging from zero (patient in good physical health, no illnesses) to six (patient has a terminal illness) (see Appendix B). Previous research has utilized the GHSR to objectively assess illness severity (Barsky et al., 1986; Jones Mabe, & Riley, 1989; Mabe, Hobson, Jones, & Jarvis, 1988). The GHSR has an adequate inter-rater reliability coefficient of .76 (Jones et al., 1989). In the

present study. an attending primary care physician reviewed the medical charts during the index year of the study (i.e., the 12 months before the administration of the DIS-IV) and rated the health status of each patient according to the Likert scale. The physician also provided a list of medical diagnoses each participant had on the form. Medical diagnoses were then assigned their corresponding International Classification of Diseases (ICD-9, 1998) codes in order to classify diseases into ICD-9 categories.

1994 Behavioral Risk Factor Surveillance System (BRFSS)

The BRFSS (Centers for Disease Control, 1994) is a survey schedule that has been widely used to identify high-risk behaviors among noninstitutionalized American adults (18 years of age or older). Currently, the data obtained through the BRFSS are a central component of federal and state activities designed to monitor progress toward achieving the health objectives for the year 2000 (U.S. Department of Health and Human Services [Public Health Service], 1992). The core sections covered by the 1994 BRFSS include: demographics, health status, health care access, diabetes, leisure-time physical activity, cigarette smoking, nutrition, weight control, women's health, and AIDS knowledge and testing. The present study included only the section on health status.

Methodology

The present study was part of a larger project examining the roles of stress and psychopathology in medical utilization that was funded by the National Institutes of Health (NIMH grant # 1RO1 MH51194-01A1). The study was approved by the Institutional Review Board of Louisiana State University.

Recruitment of subjects for the larger study occurred at the primary care clinics at EKLMC. Patients were randomly approached in the clinic patient rooms prior to their appointments. Potential subjects were given an explanation of the study and were asked if they would like to participate. Any questions potential participants had about the study were answered and informed consent was then obtained. Potential participants then filled out an Informed Consent form (see Appendix C) indicating that they understood the procedures involved in the study and their rights and privileges as research participants. After informed consent was obtained, baseline measures consisting of the demographics and other questionnaires were obtained. Participants were first given an option of participating in a brief study consisting of the demographics questionnaire and a brief measure of psychological distress. Participants were paid \$15 for participation at this level and were given the opportunity to participate in the larger part of the study, and were paid an additional \$20 to complete the rest of the baseline study measures. The design of this study included bimonthly phone calls to assess stress and medical utilization, and subjects were paid \$10 for each completed assessment. Participants were removed from the study if they missed more than two of the bimonthly assessments. After participating in the study for one year, participants were brought to EKLMC where the BRFSS questionnaire and the DIS interview were administered. Participants were paid \$50.00 for completing this phase of the experiment. Therefore, participants in the present study: a.) agreed to participate in the entire study, b.) completed at least three of five

bimonthly phone calls assessing stress and medical utilization, and c.) showed up for the yearly assessment where the BRFSS and DIS were administered.

Medical assessment via chart review and physician rating of health were conducted after the first year of participation. Chronic and acute medical illnesses were assigned appropriate ICD -9 codes. Participants were classified as having a particular “category” of disease if their GHSR had an ICD-9 code matched to the included categories of diseases. Qualifying medical conditions were modeled after Boscarino’s (1997) study, but expanded somewhat to be broader in range. Table 1 presents the ICD-9 categories used in the study.

Table 1: Proposed ICD-9 Chronic Illnesses Categories

1. NEOPLASMS (140-239)
2. ENDOCRINE, NUTRITIONAL AND METABOLIC DISEASES, AND
IMMUNITY DISORDERS (240-279)
*exclude Obesity
3. DISEASES OF BLOOD AND BLOOD FORMING ORGANS (280-289)
4. DISEASES OF THE NERVOUS SYSTEM AND SENSE ORGANS (320-389)
5. DISEASES OF THE CIRCULATORY SYSTEM (390-496)
*exclude Hemorrhoids
6. DISEASES OF THE RESPIRATORY SYSTEM (460-519)
7. DISEASES OF THE DIGESTIVE SYSTEM (520-579)
8. DISEASES OF THE GENITOURINARY SYSTEM (580-629)
9. DISEASES OF THE SKIN AND SUBCUTANEOUS TISSUE (680-739)

(Table 1, cont.)

**10. DISEASES OF THE MUSCULOSKELETAL SYSTEM & CONNECTIVE
TISSUE (710-759)**

RESULTS

Demographic Data

Four hundred thirty one participants had sufficient data to be included in the statistical analyses. The average age of the sample was 46.4 (+/- 13.8) years. Eighty one percent of the sample were females, 74.2 percent were African-American, and 33% were married. The average yearly family income for participants was \$11,756 (+/- 8680), the average education level was 11.1 years (+/- 2.74), and 74% of the sample was uninsured. Table 2 presents the demographic statistics for the sample. The demographic statistics are similar to those of the patient population of EKLMC (Carroll, 1995).

Table 2: Demographic Data

<i>Variable</i>	<i>N</i>	<i>%</i>	<i>Mean (S.D.)</i>	<i>Range</i>
Gender				
Female	350	81		
Male	81	19		
Race				
African American	320	74		
Caucasian	108	25		
Other	3	1		
Marital Status				
Single	133	31		
Married	142	33		
Separated	26	6		
Divorced	84	20		

(Table 2. cont.)

Widowed	44	10		
Age (years)			46.4 (13.8)	19-79
Education Level (years)			11.1 (2.7)	0-16
< High School	193	45		
High School Grad	146	34		
Some College /Technical School	77	18		
College and/or Beyond	15	3		
Household Income, yearly (\$)			11,756 (8689)	0-50,000
0 - 14,999	259	60		
15,000 - 24,999	58	13		
25,000 - 34,999	14	3		
35,000 or more	4	1		
unreported	96	22		
Health Insurance				
None	318	74		
Medicare/Medicaid	79	18		
Private	13	8		

Aim 1: Prevalence and Associated Features of Trauma and PTSD

Traumatic Life Events

A series of descriptive statistics was conducted to evaluate the demographic variables involved in trauma and PTSD prevalence. Eighty-eight percent of the sample reported at least one traumatic life event. The mean number of events endorsed by the sample was 3.1 (*SD* = 2.3). Table 3 presents the prevalence of traumatic life events across the eighteen different probes of the PTSD module of the DIS. Table 3 also

presents the 18 traumatic events collapsed into three event categories: interpersonal violence, other trauma to self, and trauma to others. Prevalence of traumatic life event exposure varied widely, with some traumas being endorsed by very few participants, whereas other events were widely endorsed by the sample. Military combat-related traumas were reported the least (<2%), whereas unexpected/sudden death of a friend or relative was endorsed the most (71%). Participants were asked to list the traumatic event that upset them the most if they reported experiencing more than one traumatic life event. These “most upsetting” categories are also listed in Table 3.

Table 3: Traumatic Life Events Endorsed by the Sample

<i>Event</i>	<i>N</i>	<i>%</i>	<i>N as worst event</i>	<i>%</i>
Interpersonal Violence	173	40	69	19
Held captive or tortured (military combat)				
Wounded (military combat)	3	.7		
Seen someone injured or killed (military combat)	6	1		
Shot or stabbed	44	10		
Mugged, threatened, or robbed	114	26		
Raped or sexually assaulted by a relative	30	7		
Raped or sexually assaulted by non-relative	53	12		
Other Trauma to Self	285	66	130	36
Natural Disaster	151	35		
Exposure to dangerous materials	27	6		

(Table 3, cont.)

Held captive, tortured, or kidnapped	11	2		
Diagnosed with life-threatening illness	75	17		
Serious Accident	101	23		
Saw someone seriously hurt or killed	128	30		
Unexpectedly discovered a body (military combat)	2	.5		
Unexpectedly discovered a dead body	50	11		
Other terrible or frightening event to self	67	15		
Learning of Terrible Event to Another	328	76	164	45
Unexpected, sudden death of friend or relative	308	71		
Learned of terrible thing to friend or relative	148	34		
Any Event	380	88		

A series of chi-square analyses and T-tests were conducted to test for demographic differences in trauma prevalence of the sample. Men reported more traumatic life events than women [$t(429) = 2.73, p = .007$]. No other significant differences were revealed (see Tables 4 and 5).

Table 4: Chi-Square Analyses of Demographic Differences Between Participants Exposed or Non-Exposed to Traumatic Life Events

<i>Variable</i>	<i>Exposed</i>	<i>Not Exposed</i>	X^2	<i>DF</i>	<i>P</i>
Race			2.4	2	.48
Black	34	286			
White	17	91			

(Table 4, cont.)

Other	0	3				
Sex			.36	1	.54	
Female	43	307				
Male	8	73				
Marital Status			3.5	4	.47	
Single	20	113				
Married	15	127				
Separated	1	25				
Divorced	11	73				
Widowed	4	40				

Table 5: T-Test Analyses of Demographic Differences Between Participants Exposed or Non-Exposed to Traumatic Life Events

<i>Variable</i>	<i>M</i>	<i>(SD)</i>	<i>M</i>	<i>(SD)</i>	<i>T</i>	<i>DF</i>	<i>P</i>
Age	45	(14.5)	45	(14.2)	.18	429	.85
Education Level	11	(2.8)	10	(3.1)	.64	427	.52
Household Income	11000	(7423)	11360	(7423)	.29	333	.77

Next, analyses were conducted to test the hypothesis that participants in this low-income primary care population would be at greater risk for traumatic event exposure than has been demonstrated in the community. Breslau et al's (1998) prevalence data were used as the comparison group because it was the only published study using similar methods and DSM-IV criteria. A chi-square analysis was conducted to examine whether the prevalence of at least one trauma in this sample

(88%) was significantly different than the Breslau et al. (1998) prevalence of 89%. Results of the chi-square analysis indicated that participants in this sample did not experience traumatic life events at a higher frequency than the Breslau et al. (1998) study, [$X^2(1) = .305, p = .58$.] Next, t-tests were calculated to examine whether subjects in this sample experienced more traumatic life events than the Breslau et al. (1998) study. Analyses were conducted separately for females and males due to the large discrepancy in sample size, and because of known gender differences in trauma and PTSD prevalence (Breslau et al., 1998; Kessler et al., 1995). Results of the one-sample T-tests indicated male participants in this sample experienced significantly fewer events than in the Breslau et al. (1998) study (3.7 to 5.3, respectively, [$t(431) = -5.78, p < .001$]). In addition, female participants in this study also reported significantly fewer events than females in the Breslau et al., 1998 study (2.9 to 4.3, respectively, [$t(431) = -11.68, p < .001$]).

PTSD Prevalence

Ninety-three of the 431 participants in this study met criteria for PTSD at some point in their lives, yielding a lifetime prevalence of 21.6%. At the time of interview, 43 of the 431 participants in this study met criteria for PTSD, yielding a point prevalence of 10%. The conditional risk for PTSD for the worst event encountered was 24.4%. Since the DIS PTSD module assesses PTSD symptoms for the single worst trauma experienced, conditional risk for PTSD could not be calculated for specific events. Events endorsed as the “most traumatic” by participants were

categorized into three categories, and the conditional risk of PTSD from these categories was calculated and are presented in Table 6.

Table 6: Conditional Risk of PTSD from Worst Event Categories

	<i>PTSD Diagnosis</i>		<i>X²</i>	<i>DF</i>	<i>P</i>
	<i>No</i>	<i>Yes</i>			
Event Category			.63	2	.73
Interpersonal Violence	71	29			
Other Trauma to Self	76	24			
Trauma to Other	74	26			

Note: Values presented as percentages

Chi-square analyses were also conducted to compare the prevalence of PTSD in this sample to that of Breslau et al. (1998) study. Again, statistics for males and females were conducted separately given the higher prevalence of PTSD in females and larger number of females in this study. Results indicated that females showed significantly higher prevalence compared to the Breslau et al. (1998) study (23.98% vs. 17.7%, respectively, [$X^2(1) = 9.4, p = .002$]). Males in this study did not show significantly higher prevalence rates than males in the Breslau et al. (1998) study (12% to 9.5%, respectively, [$X^2(1) = .627, p = .429$]).

Comorbidity of PTSD

Chi-square analyses were conducted to test whether participants with current PTSD diagnoses were more likely to have other concurrent Axis I psychiatric diagnoses than participants without current PTSD diagnoses. Results indicated that participants were more likely to have at least one comorbid psychiatric disorder than

participants without a current PTSD diagnosis (61.9% versus 25.1%, [$X^2(1) = 1.61, p = .205$]). Participants with current PTSD were significantly more likely to have comorbid major depressive disorder, panic disorder, generalized anxiety disorder, and agoraphobia (without panic) than those subjects without current PTSD (all p 's < .05). Participants with current PTSD were no more likely to have drug abuse/dependence, alcohol abuse/dependence, or bipolar disorders than those without current PTSD.

Table 7 presents the percentages and chi-square analyses for each disorder.

Table 7: Comorbidity Between Current PTSD and Other Disorders

<i>Disorder</i>	<i>Overall Prevalence</i>	<i>PTSD Diagnosis</i>		<i>X²</i>	<i>DF</i>	<i>P</i>
		<i>No</i>	<i>Yes</i>			
Major Depression	16	13	45	27.7	1	.00
Bipolar Disorder	46	4	7	.6	1	.42
Panic Disorder	3	3	10	4.3	1	.04
Generalized Anxiety D/O	7	6	19	9.7	1	.00
Obsessive-Compulsive D/O	2	1	7	5.7	1	.01
Agoraphobia (no panic)	5	4	19	15	1	.00
ETOH Abuse/Dependence	1	1	2	.3	1	.56
Drug Abuse / Dependence	4	4	2	.3	1	.58
Any Disorder	35	25	612	25	1	.00

Note: Values are expressed as percentages.

Duration of PTSD Symptoms

Duration of PTSD symptoms was examined by using Kaplan-Meier survival curve methods (Kaplan & Meier, 1958). The median remission time for participants

the sample reported symptom duration of 48 months or more, and 20% of those with a PTSD diagnosis reported symptom duration of over 60 months. Log-rank chi-square analyses were conducted to test for the presence of gender or event type differences. Results indicated that males and females did not have significantly different remission rates of PTSD, [$X^2(1) = 1.6, p = .21$]. Significant differences were found in remission time based on event type, [$X^2(1) = 8.1, p = .01$]. Participants with PTSD from interpersonal violence had a median remission time of 48 months, compared to 12 months for participants with PTSD from either other traumatic event to self or traumatic event to other. Figure 1 presents the survival curves of the three event types.

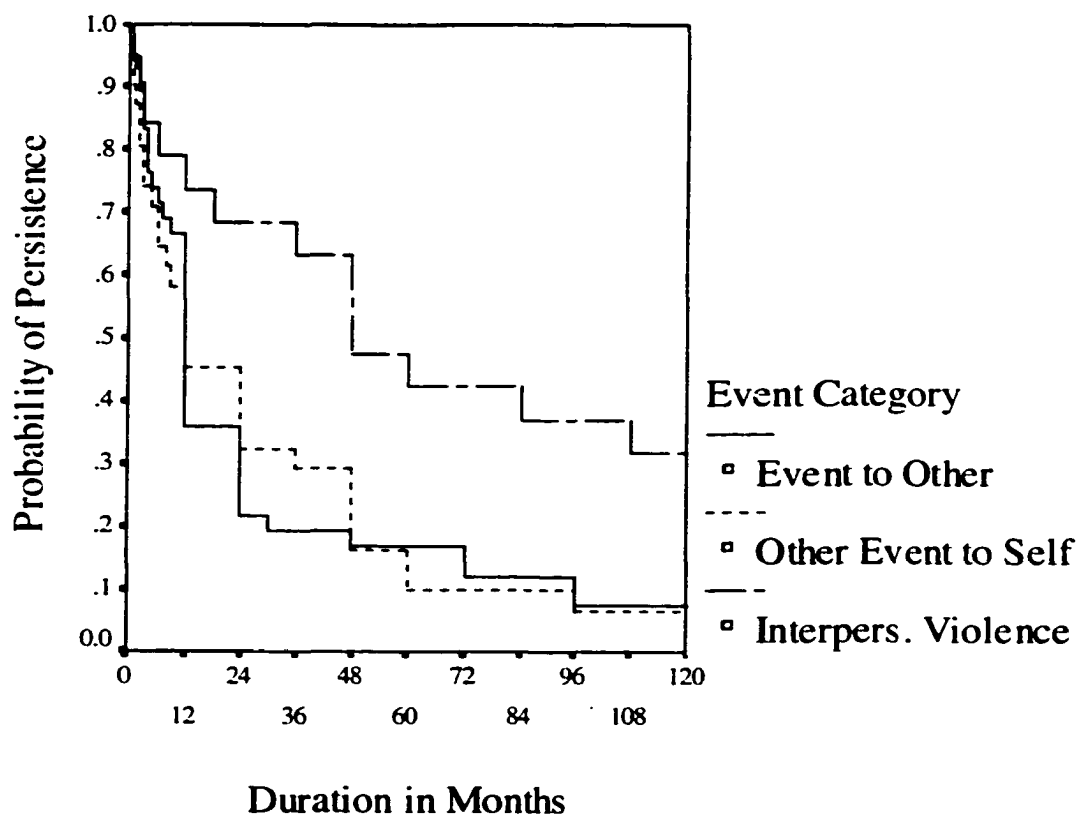


Figure 1: Survival Curve Function for PTSD Cases

Comparisons of “Clean,” “PTSD,” and “Psychiatric Control” Cases

Chi-square analyses and one-way ANOVA’s were used to test for demographic differences between individuals with no history of mental disorder (“clean cases”), those participants with depression but no history of PTSD (“psychiatric controls”), and those participants with a history of a PTSD diagnosis (“PTSD cases”). Results indicated that PTSD cases were significantly younger than clean cases [$F(2,317) = 4.44, p = .01$]. No significant differences were found for race, marital status, or education level (Tables 8 and 9).

Table 8: Chi-Square Analyses of Demographic Differences Between Participants Exposed or Non-Exposed to Traumatic Life Events

<i>Variable</i>	<i>Clean Cases</i>	<i>Psychiatric Controls</i>	<i>PTSD Cases</i>	X^2	<i>DF</i>	<i>P</i>
Race				9.1	4	.06
Black	124	45	67			
White	31	26	25			
Other	0	1	1			
Gender				5.1	2	.08
Female	121	60	83			
Male	34	12	10			
Marital Status				7.4	8	.49
Single	43	18	27			
Married	59	20	34			
Separated	12	7	6			

(Table 9, cont.)

Divorced	27	21	21
Widowed	15	5	4

Table 10: One-Way ANOVA's of Demographic Differences Between Participants Exposed or Non-Exposed to Traumatic Life Events

	<i>Clean Cases</i>		<i>Psychiatric Controls</i>		<i>PTSD Cases</i>		<i>F</i>	<i>P</i>
	<i>M</i>	<i>(SD)</i>	<i>M</i>	<i>(SD)</i>	<i>M</i>	<i>(SD)</i>		
Age	47*	(14.5)	44	(14.2)	42*	(11.7)	4.44	.01
Ed Level	11	(2.8)	11	(3.1)	11	(2.5)	2.02	.13

Note: * denotes significant difference at .05 level via Fisher's LSD.

Aim 2: Prediction of Self-Reported Health Status

Logistic regression equations were hierarchically created to test the hypothesis that PTSD status would be predictive of impaired health status in relation to normal and psychiatric control cases. The first regression equation tested whether PTSD cases were more likely than control cases to report their health status as impaired. If so, a secondary regression equation was created to examine whether PTSD cases were more likely than psychiatric control cases to report their health status as impaired.

The possible confounding factors of age, tobacco use, and alcohol use, were entered in the first step in the logistic regression. Furthermore, because of the high prevalence of obesity (62.7%) in the sample, it was also entered in step one to control for its potential confounding effect on health outcome. Psychiatric status (clean, psychiatric control, & PTSD cases) was entered as a categorical variable in step 2.

The outcome variable in the equation was the health status question from the BRFSS. dichotomized into either “impaired” or “not impaired.”

Using a forced entry procedure with a significance level of .05 as the criterion for entrance into the equation, age, obesity, and psychiatric status were the only factors that were significant predictors of impaired health status (Table 10). Both psychiatric control and PTSD cases were significantly more likely to view their health status as impaired than clean cases. Examining the odds ratios revealed that the presence of PTSD increased the odds of having impaired health status by a factor of 2.9 ($p = .01$, 95% CI = .10, 1.34).

Table 10: Logistic Regression Results for Prediction of Impaired Health Status

<i>Variable</i>	<i>Beta</i>	<i>S.E.</i>	<i>Wald</i>	<i>Df</i>	<i>P</i>	<i>R</i>	<i>OR</i>	<i>95% CI</i>
Age	.04	.01	12.2	1	.001	.18	1.04	1.01, 1.06
Pack Years			2.6	4	NS	.00		
1-10	-.27	.49	.30	1	NS	.00	.76	.28, 2.007
11-19	-1.12	.93	1.6	1	NS	.00	.30	.04, 1.88
20-29	.40	.69	.3	1	NS	.00	1.5	.38, 5.82
>29	.17	.49	.1	1	NS	.00	1.18	.45, 3.1
ETOH Dx	-.27	.54	.2	1	NS	.00	.76	.26, 2.21
Obesity	.62	.31	3.8	1	.04	.07	1.85	1.002, 3.44
Psych Hx			0.7	2	.004	.15		
Dep	.778	.37	4.3	1	.037	.08	2.17	1.04, 4.53
PTSD	1.09	.35	9.6	1	.01	.16	2.98	1.49, 5.9

Because psychiatric control and PTSD cases were significantly more likely to report their health status as impaired than control cases, a second regression equation was created to test whether PTSD differed significantly from psychiatric controls on the likelihood of rating their health status as impaired or not. Similar to the first equation, the control variables of age, tobacco use history, alcohol use history, and obesity were entered into the equation at step 1. Psychiatric status, (dichotomized into psychiatric control or PTSD case) was entered at step 2. The outcome variable of health status (not impaired vs. impaired) remained the same.

Using the same forced entry procedure and same significance level of .05 to be included into the equation, only age was a significant predictor of impaired health status (Table 11). PTSD cases were not significantly more likely to rate their health status as impaired than psychiatric control cases. Examining the odds ratios revealed that the presence of PTSD increased the odds of reporting impaired health status by a factor of 1.39 (95% CI = .60, 3.22).

Table 11: Logistic Regression Results for Predictors of Impaired Health Status: PTSD vs. Depressed Cases

<i>Variable</i>	<i>B</i>	<i>S.E.</i>	<i>Wald</i>	<i>df</i>	<i>Sig</i>	<i>R</i>	<i>O.R.</i>	<i>95% CI</i>
Age	.04	.01	6.62	1	.01	.18	1.04	1.01, 1.08
Pack Years			2.52	4	NS	.00		
1-10	-.54	.75	.51	1	NS	.00	.58	.13, 2.55
11-19	-.72	1.04	.49	1	NS	.00	.48	.06, 3.71
20-29	.54	.94	.33	1	NS	.00	1.72	.26, 11.00
> 29	.72	.49	.87	1	NS	.00	2.07	.44, 9.54

(Table 11, cont.)

ETOH DX	-.99	.65	2.27	1	NS	-.04	.37	.10, 1.34
Obesity	.42	.45	.88	1	NS	.00	1.53	.62, 3.73
PTSD	.33	.42	.59	1	NS	.00	1.39	.60, 3.22

Aim 3: Prediction of Medical Illness

The same technique used to test for differences in self-reported health status was applied to the prediction of ICD-9 medical diagnoses. That is, the same hierarchical analysis procedure first testing for difference between PTSD and clean cases was utilized, with a second regression equation to test for differences between PTSD and psychiatric control cases if differences between PTSD and control cases were revealed. Similar to aim two, the same control variables of age, tobacco use history, alcohol use history, and obesity were entered at step one. Each of the ICD-9 medical disease categories (neoplasm: endocrine / metabolic / immunity; blood; nervous system; circulatory system; respiratory system; digestive system; genitourinary system; and musculoskeletal system) were dichotomized into either “not present” or “present.”

Neoplasm

Only the presence of tobacco use (pack year history between 20-29 years) was significantly associated with the presence of neoplasm at the .05 level (Table 12). Examining the odds ratios revealed presence of a 20-29 pack-year tobacco use history increased the odds of having a neoplasm by a factor of 6.6 (95% CI = 1.1, 40.5). Presence of a PTSD diagnosed decreased the odds of having a neoplasm by a factor of .39 (95% CI = .05, 2.7).

Table 12: Logistic Regression Results for Predictors of Neoplasm Category

<i>Variable</i>	<i>B</i>	<i>S.E.</i>	<i>Wald</i>	<i>df</i>	<i>Sig</i>	<i>R</i>	<i>O.R.</i>	<i>95% CI</i>
Age	.02	.02	.52	1	NS	.00	1.01	96, 1.08
Pack Years			4.49	4	NS	.00		
1-10	-7.2	33.76	.04	1	NS	.00	.007	.00, 4.5
11-19	1.13	1.32	.73	1	NS	.00	3.11	.23, 41.6
20-29	1.88	.92	4.15	1	.04	.18	6.6	1.1, 40.5
> 29	-7.44	33.1	.05	1	NS	.00	.006	.00, 8.7
ETOH DX	1.81	1.01	3.19	1	NS	.13	6.13	.83, 44.7
Obesity	.03	.76	.001	1	NS	.00	1.03	.23, 4.6
Psych Status			.92	2	NS	.00		
Depressed	-.45	.93	.232	1	NS	.00	.63	.10, 3.9
PTSD	-.93	.99	.89	1	NS	.16	.39	.05, 2.7

Endocrine / Nutritional / Metabolic / Immunity Disease

Only age was significantly associated with the presence of endocrine system diseases at the .05 level (Table 13). Examining the odds ratios revealed that chances of receiving an endocrine system disease increased by a factor of 1.05 for each year of age (95% CI = 1.02, 1.07). Presence of PTSD increased the odds of having an endocrine disorder by a factor of 1.5 (95% CI = .78, 3.04).

Table 13: Logistic Regression Results for Predictors of Endocrine / Nutritional /Metabolic / Immunity Disease

<i>Variable</i>	<i>B</i>	<i>S.E.</i>	<i>Wald</i>	<i>df</i>	<i>Sig</i>	<i>R</i>	<i>O.R.</i>	<i>95% CI</i>
Age	.05	.01	17.8	1	.001	.23	1.05	1.02, 1.07
Pack Years			.91	4	NS	.00		
1-10	.21	.48	.19	1	NS	.00	1.23	.48, 3.1

(Table 13, cont.)

11-19	-.03	.90	.001	1	NS	.00	.96	.16, 5.6
20-29	-.58	.72	.65	1	NS	.00	.55	.13, 2.3
> 29	-.01	.47	.001	1	NS	.00	.98	.38, 2.5
ETOH DX	-.04	.54	.006	1	NS	.00	.95	.32, 2.7
Obesity	.35	.31	1.2	1	NS	.00	1.42	.76, 2.6
Psych Status			2.2	2	NS	.00		
Depressed	-.13	.7	12	1	NS	.0	.87	.2, 1.8
PTSD	.43	.34	1.6	1	NS	.00	1.54	.78, 3.04

Blood and Blood Forming Organ Disease

No variables in the regression equation were significantly associated with the presence of blood diseases at the .05 level (Table 14). Examining the odds ratios revealed that presence of PTSD increased the odds of having a blood disorder by a factor of 1.11 (95% CI = .30, 4.1).

Table 14: Logistic Regression Results for Predictors of Blood and Blood Forming Organ Disease

<i>Variable</i>	<i>B</i>	<i>S.E.</i>	<i>Wald</i>	<i>df</i>	<i>Sig</i>	<i>R</i>	<i>O.R.</i>	<i>95% CI</i>
Age	-.02	.02	.91	1	NS	.00	.97	.93, 1.02
Pack Years			.96	4	NS	.00		
1-10	-.20	1.08	.36	1	NS	.00	.81	.09, 6.8
11-19	-6.72	62.9	.01	1	NS	.00	.001	.00, 4.2
20-29	-7.3	50.7	.02	1	NS	.00	.007	.00, 1.1
> 29	.81	.88	.83	1	NS	.00	2.2	.39, 12.8
ETOH DX	-7.44	38.2	.03	1	NS	.00	.006	.00, 2.1
Obesity	-.34	.5	.35	1	NS	.00	.70	.22, 2.2

(Table 14, cont.)

Psych Status			.03	2	NS	.00		
Depressed	.09	.72	.01	1	NS	.00	1.1	.26, 4.5
PTSD	.11	.66	.02	1	NS	.00	1.1	.30, 4.1

Nervous System Disease

No variables in the regression equation were significantly associated with the presence of nervous system disease at the .05 level (Table 15). Examining the odds ratios revealed that presence of PTSD increased the odds of having a nervous system disorder by a factor of 1.42 (95% CI = .07, 6.0).

Table 15: Logistic Regression Results for Predictors of Nervous System Disease

<i>Variable</i>	<i>B</i>	<i>S.E.</i>	<i>Wald</i>	<i>df</i>	<i>Sig</i>	<i>R</i>	<i>O.R.</i>	<i>95% CI</i>
Age	.02	.01	1.7	1	NS	.00	1.02	.98, 1.06
Pack Years			1.69	4	NS	.00		
1-10	.32	.81	.15	1	NS	.00	1.38	.27, 6.8
11-19	-5.89	24.2	.05	1	NS	.00	.002	.00, 1.1
20-29	1.02	.85	1.43	1	NS	.00	2.79	.52, 14.9
> 29	.41	.72	.32	1	NS	.00	1.5	.36, 6.2
ETOH DX	-.36	1.1	.10	1	NS	.00	.69	.07, 6.0
Obesity	-.73	.49	2.2	1	NS	-.04	.47	.18, 1.2
Psych Status			.48	2	NS	.00		
Depressed	-.05	.63	.007	1	NS	.00	.94	.27, 3.2
PTSD	.35	.57	.38	1	NS	.00	1.42	.46, 4.3

Circulatory System Disease

Age, alcohol use history, obesity status, and PTSD status were all significantly associated with the presence of circulatory system disease at the .05 level (Table 16).

Examining the odds ratios revealed that chances of having a circulatory system disease increased by a factor of 1.08 each year of age (95% CI = 1.05, 1.11). Presence of an alcohol abuse/dependence disorder decreased the odds of a circulatory disorder by a factor of .28 (95% CI = .08, .89). Presence of obesity increased the odds of having a circulatory system disorder by a factor of 2.4 (95% CI = 1.2, 4.7). Presence of PTSD increased the odds of having a circulatory system disorder by a factor of 2.1 (95% CI = 1.004, 4.48).

Table16: Logistic Regression Results for Predictors of Circulatory System Disease

<i>Variable</i>	<i>B</i>	<i>S.E.</i>	<i>Wald</i>	<i>df</i>	<i>Sig</i>	<i>R</i>	<i>O.R.</i>	<i>95% CI</i>
Age	.08	.01	35.14	1	.001	.36	1.08	1.05, 1.11
Pack Years			2.65	4	NS	.00		
1-10	.56	.54	1.05	1	NS	.00	1.75	.60, 5.1
11-19	1.07	1.29	.69	1	NS	.00	2.9	.23, 36.8
20-29	.53	.78	.47	1	NS	.00	1.7	.37, 7.94
> 29	-.29	.54	.30	1	NS	.00	.74	.25, 2.1
ETOH DX	-1.2	.58	4.6	1	.03	-.10	.28	.08, .89
Obesity	.88	.33	6.8	1	.009	.13	2.4	1.2, 4.7
Psych. Status			5.5	2	NS	.07		
Depressed	.79	.41	3.6	1	NS	.07	2.2	.97, 4.98
PTSD	.75	.38	3.8	1	.04	.08	2.1	1.004, 4.48

Because the PTSD was a significant predictor of circulatory system disease at the .05 level, a secondary analysis was conducted to examine whether PTSD cases were more likely than psychiatric control cases to have a circulatory system disorder.

Using the same forced entry procedure and same significance level of .05 to be included into the equation, PTSD was not a significant predictor of circulatory system disease compared to psychiatric control cases. Examining the odds ratios revealed that presence of PTSD decreased the odds of having a circulatory system disorder by a factor of .77 (95% CI = .29, 2.0; Table 17).

Table 17: Logistic Regression Results for Predictors of Circulatory System Disease: PTSD Versus Psychiatric Control Cases

<i>Variable</i>	<i>B</i>	<i>S.E.</i>	<i>Wald</i>	<i>df</i>	<i>Sig</i>	<i>R</i>	<i>O.R.</i>	<i>95% CI</i>
Age	.09	.02	18.3	1	.001	.38	1.09	1.05, 1.14
Pack Years			4.68	4	NS	.00		
1-10	-.72	.81	.79	1	NS	.00	.48	.09, 2.39
11-19	1.22	1.38	.78	1	NS	.00	3.39	.22, 50.8
20-29	1.94	1.33	2.12	1	NS	.03	7.07	.51, 95.8
> 29	1.14	.95	1.44	1	NS	.01	3.14	.48, 20.3
ETOH DX	-1.5	.74	4.39	1	.03	-.14	.21	.04, .90
Obesity	1.14	.55	6.4	1	.01	.19	4.12	1.3, 12.3
PTSD	-.25	.49	.26	1	NS	.00	.77	.29, 2.0

Respiratory System Disease

Only presence of >29 pack-year history of tobacco use was associated with a respiratory system disease at the .05 level (Table 18). Heavy smokers were 3.4 times as likely as nonsmokers to have a respiratory system disease (95% CI = 1.2, 10.4). Presence of PTSD decreased the odds of having a respiratory system disorder by a factor of .66 (95% CI = .23, 1.89).

Table 18: Logistic Regression Results for Predictors of Respiratory System Disease

<i>Variable</i>	<i>B</i>	<i>S.E.</i>	<i>Wald</i>	<i>df</i>	<i>Sig</i>	<i>R</i>	<i>O.R.</i>	<i>95% CI</i>
Age	-.11	.01	.005	1	NS	.00	.99	.96, 1.03
Pack Years			5.11	4	NS	.00		
1-10	.5	.62	.65	1	NS	.00	1.65	.48, 5.57
11-19	.19	1.18	.02	1	NS	.00	1.21	.11, 12.39
20-29	-.21	1.09	.03	1	NS	.00	.80	.09, 6.92
> 29	1.23	.56	4.69	1	.03	.12	3.42	1.12, 10.42
ETOH DX	.61	.66	.83	1	NS	.00	1.84	.49, 6.85
Obesity	-.53	.41	1.66	1	NS	.00	.58	.26, 6.85
Psych Status			1.72	2	NS	.00		
Depressed	.34	.46	.55	1	NS	.00	1.41	.56, 3.52
PTSD	-.40	.53	.58	1	NS	.00	.66	.23, 1.89

Digestive System Disease

No variables were associated with presence of a digestive system disease at the .05 level (Table 19). Examining the odds ratios revealed that presence of PTSD cases decreased the odds of having a digestive system disease by a factor of .66 (95% CI = .22, 2.01).

Table 19: Logistic Regression Results for Predictors of Digestive System Disease

<i>Variable</i>	<i>B</i>	<i>S.E.</i>	<i>Wald</i>	<i>df</i>	<i>Sig</i>	<i>R</i>	<i>O.R.</i>	<i>95% CI</i>
Age	.02	.01	1.40	1	NS	.00	1.02	.98, 1.05
Pack Years			1.58	4	NS	.00		
1-10	.08	.68	.01	1	NS	.00	1.08	.28, 4.14
11-19	.39	1.17	.11	1	NS	.00	1.48	.14, 14.94

(Table 19, cont.)

20-29	-.19	1.09	.03	1	NS	.00	.81	.09, 7.02
> 29	-1.24	1.07	1.33	1	NS	.00	.28	.03, 2.37
ETOH DX	-.62	1.08	.33	1	NS	.00	.53	.06, 4.48
Obesity	.44	.50	.79	1	NS	.00	1.56	.58, 4.19
Psych Status			.61	2	NS	.00		
Depressed	.04	.53	.007	1	NS	.00	1.04	.36, 2.99
PTSD	-.40	.56	.51	1	NS	.00	.66	.22, 2.01

Genitourinary System Disease

No variables were associated with presence of a genitourinary system disease at the .05 level. (Table 20). Examining the odds ratios revealed that presence of PTSD increased the odds of a genitourinary system disease by a factor of 1.28 (95% CI = .42, 3.89).

Table 20: Logistic Regression Results for Predictors of Genitourinary System Disease

<i>Variable</i>	<i>B</i>	<i>S.E.</i>	<i>Wald</i>	<i>df</i>	<i>Sig</i>	<i>R</i>	<i>O.R.</i>	<i>95% CI</i>
Age	-.02	.02	1.42	1	NS	.00	.97	.93, 1.01
Pack Years			2.84	4	NS	.00		
1-10	.41	.82	.25	1	NS	.00	1.51	.29, 7.72
11-19	-6.33	39.55	.02	1	NS	.00	.001	.00, 8.3
20-29	-6.36	30.79	.04	1	NS	.00	.001	.00, 2.79
> 29	1.187	.71	2.74	1	NS	.07	3.28	.80, 13.38
ETOH DX	.93	.77	1.47	1	NS	.00	2.54	.56, 11.54
Obesity	.81	.67	1.44	1	NS	.00	2.25	.59, 8.51
Psych Status			1.67	2	NS	.00		

(Table 20. cont.)

Depressed	-.87	.83	1.07	1	NS	.00	.41	.08, 2.17
PTSD	.25	.56	.19	1	NS	.00	1.28	.42, 3.89

Skin and Subcutaneous Tissue Disease

No variables were associated with presence of a skin and subcutaneous disease at the .05 level (Table 21). Examining the odds ratios revealed that presence of PTSD increased the odds of having a skin disease by a factor of 1.09 (95% CI = .23, 5.05).

Table 21: Logistic Regression Results for Predictors of Skin and Subcutaneous Tissue Disease

<i>Variable</i>	<i>B</i>	<i>S.E.</i>	<i>Wald</i>	<i>df</i>	<i>Sig</i>	<i>R</i>	<i>O.R.</i>	<i>95% CI</i>
Age	.004	.02	.02	1	NS	.00	1.004	.95, 1.05
Pack Years			1.78	4	NS	.00		
1-10	-.06	1.12	.003	1	NS	.00	.93	.10, 8.44
11-19	-6.4	40.1	.02	1	NS	.00	.001	.00, 2.30
20-29	-6.08	31.28	.03	1	NS	.00	.002	.00, 9.74
> 29	1.02	.80	1.60	1	NS	.00	2.78	.57, 13.53
ETOH DX	.83	.91	.81	1	NS	.00	2.29	.37, 13.91
Obesity	.63	.82	.58	1	NS	.00	1.88	.37, 9.46
Psych Status			.20	2	NS	.00		
Depressed	.35	.79	.19	1	NS	.00	1.42	.30, 6.70
PTSD	.08	.78	.01	1	NS	.00	1.09	.23, 5.05

Musculoskeletal System Disease

Age and obesity status were both significantly associated with the presence of musculoskeletal system disease at the .05 level (Table 22). Examining the odds ratios revealed that chances of receiving a musculoskeletal system disease increased by a

factor of 1.05 for each year (95% CI = 1.02, 1.08). Presence of obesity was positively related to presence of a musculoskeletal disease, with obese participants being 2.27 times as likely as non-obese subjects to have a musculoskeletal system disease (95% CI = 1.003, 5.19). Although not significant at the .05 level, PTSD was positively related to presence of a musculoskeletal system disorder, with PTSD cases being 1.9 times as likely as control cases to have a musculoskeletal system disorder (95% CI = .86, 4.22).

Table 22: Logistic Regression Results for Predictors of Musculoskeletal System Disease

<i>Variable</i>	<i>B</i>	<i>S.E.</i>	<i>Wald</i>	<i>df</i>	<i>Sig</i>	<i>R</i>	<i>O.R.</i>	<i>95% CI</i>
Age	.05	.01	13.1	1	.001	.22	1.05	1.02, 1.08
Pack Years			3.25	4	NS	.00		
1-10	-.54	.67	.64	1	NS	.00	.58	.15, 2.19
11-19	-.79	1.16	.46	1	NS	.00	.45	.04, 4.44
20-29	.94	.70	1.78	1	NS	.00	2.56	.64, 10.24
> 29	-.18	.54	.11	1	NS	.00	.83	.28, 2.41
ETOH DX	-.20	.68	.08	1	NS	.00	.81	.21, 3.14
Obesity	.82	.42	3.84	1	.04	.09	2.27	1.003, 5.19
Psych Status			2.76	2	NS	.00		
Depressed	.06	.46	.02	1	NS	.00	1.07	.42, 2.68
PTSD	.64	.40	2.57	1	.10	.05	1.91	.86, 4.22

Because the regression equation indicated a tendency for PTSD cases to be associated with musculoskeletal disease presence, a secondary analysis was conducted to examine whether PTSD cases were more likely than psychiatric control cases to

have a musculoskeletal system disorder. Using the same forced entry procedure and same significance level of .05 to be included into the equation, PTSD was not a significant predictor of musculoskeletal system disease compared to psychiatric control cases, although examining the odds ratios revealed that presence of PTSD increased the odds of a musculoskeletal disorder by a factor of 2.0 (95% CI = .67, 5.95; Table 23).

Table 23: Logistic Regression Results for Predictors of Musculoskeletal System Disease: PTSD Cases Versus Psychiatric Control Cases

<i>Variable</i>	<i>B</i>	<i>S.E.</i>	<i>Wald</i>	<i>df</i>	<i>Sig</i>	<i>R</i>	<i>O.R.</i>	<i>95% CI</i>
Age	.07	.02	9.8	1	.001	.27	1.08	1.02, 1.13
Pack Years			.94	4	NS	.00		
1-10	.03	.91	.001	1	NS	.00	1.03	.17, 6.25
11-19	-7.2	25.13	.08	1	NS	.00	.007	.00, 1.70
20-29	.92	1.01	.82	1	NS	.00	2.51	.34, 18.56
> 29	.23	.79	.09	1	NS	.00	1.27	.26, 6.02
ETOH DX	-.36	.87	.18	1	NS	.00	.69	.12, 3.80
Obesity	1.59	.72	4.9	1	.02	4.95	.01	1.20, 20.31
PTSD	.69	.55	1.56	1	NS	.00	2.00	.67, 5.95

Prediction of Any Medical Disorder

Age and obesity status were both significantly associated with the presence of any ICD-9 disease at the .05 level (Table 24). Examining the odds ratios revealed that chances of receiving an ICD-9 increased by a factor of 1.06 each year (95% CI = 1.03, 1.1). Presence of obesity was positively related to presence of an ICD-9 disease, with obese participants being 2.27 times as likely as non-obese subjects to have an ICD-9

disease (95% CI = 1.22, 5.62). Examining the odds ratios revealed that PTSD cases were 1.37 times as likely as control cases to have an ICD-9 medical disorder (95% CI = .54, 3.43).

Table 24: Logistic Regression Results for Predictors of Any Medical Disorder

<i>Variable</i>	<i>B</i>	<i>S.E.</i>	<i>Wald</i>	<i>df</i>	<i>Sig</i>	<i>R</i>	<i>O.R.</i>	<i>95% CI</i>
Age	.06	.01	17.26	1	.001	.29	1.06	1.03, 1.10
Pack Years			.33	4	NS	.00		
1-10	.26	.69	.15	1	NS	.00	1.30	.33, 5.10
11-19	5.77	23.95	.05	1	NS	.00	323	.00, 7.98E
20-29	6.50	18.42	.12	1	NS	.00	667	.00, 3.19E
> 29	-.004	.83	.000	1	NS	.00	.99	.19, 5.08
ETOH DX	.67	1.08	.38	1	NS	.00	1.95	.23, 16.55
Obesity	.96	.38	6.12	1	.01	.15	2.62	1.22, 5.62
Psych Status			.48	2	NS	.00		
Depressed	.18	.49	.13	1	NS	.00	1.19	.45, 3.15
PTSD	.31	.46	.45	1	NS	.00	1.37	.54, 3.43

DISCUSSION

This study was the first to examine PTSD and health outcomes in a low-income primary care population. It was found that the prevalence of exposure to a single traumatic life event were commonly experienced in this population, but its occurrence was no higher than in community samples. Despite similar prevalence, it was found that PTSD was highly prevalent in this population, with low-income female primary care patients demonstrating higher lifetime prevalence rates of PTSD than females in a community sample (Breslau et al., 1998). Females in this study were also more likely than community samples to develop PTSD after exposure to a traumatic life event.

Consistent with previous studies, an association was found between PTSD status and self-reported health status impairments. Participants with a diagnosis of PTSD in this study were more likely to endorse self-reported health status impairments than control participants who had no history of mental disorder, after controlling for age, alcohol abuse, tobacco use, and obesity. However, participants with PTSD did not differ from psychiatric control cases who had a history of depression, but not PTSD.

Participants with PTSD were twice as likely as control participants with no history of mental disorder to have a circulatory system disease after potential confounds of age, alcohol abuse, tobacco use, and obesity status were controlled for. Participants with PTSD, however, were no more likely than psychiatric control cases to have a circulatory system disease.

The prevalence of traumatic life events in this sample was consistent with previous studies investigating the prevalence of trauma in a community sample (Breslau et al., 1998). However, both male and female participants reported fewer traumatic events than the only other study using DSM-IV criteria (Breslau et al., 1998). These results were contrary to the hypothesis that low-income patients would be at higher risk for exposure to trauma. However, it should be noted that the comparison study from Breslau et al. (1998) does not represent a “gold standard” for trauma and PTSD prevalence; it was selected because it is the only DSM-IV based population study of trauma. As larger scale studies with wider catchment areas become available, further comparisons can be made.

One possible explanation for this may be because the changes in the DSM-IV stressor criterion. The DSM-IV stressor criterion focuses on the reaction to the stressor rather than the stressor itself. Therefore, any event that involves significant fear or helplessness can qualify as a trauma. This change has led to more events qualifying as “traumatic.” Indeed, the results of both this and the Breslau et al., 1998 study (the only other study using DSM-IV criteria for traumatic events) found that traumatic life events were highly prevalent (88 and 89%, respectively). This is significantly higher than the prevalence of trauma in community studies using DSM-III or DSM-III-R criteria, which range from 40-60%. Interestingly, both this study and the Breslau et al. (1998) investigation found higher incidence rates in one category of traumatic life event (sudden / traumatic death of loved one, a new qualifying event in DSM-IV) than for the aggregate of DSM-III and DSM-III-R investigations. Therefore, the lack of

significant results may represent some type of “ceiling effect” due to the high prevalence of traumatic events reported in this and other studies. However, the fact that participants in this study reported fewer traumatic events than other studies is also contrary to the hypothesis that lower-income primary care patients experience traumatic events at a higher rate than community samples.

The types of traumatic events endorsed in this sample were similar to those of community samples, with exposure to the sudden or traumatic death of a loved one being the most common traumatic event experienced (71%). Other commonly experienced events included exposure to a natural disaster (35%), witnessing someone being seriously injured or killed (29%), and being mugged, robbed, or threatened with a weapon (27%). These rates were similar to community studies of trauma (Breslau et al., 1998, Kessler, 1995). Furthermore, the percentage of events listed as the “most upsetting” (i.e., the worst event of all traumatic events experienced by a participant in this study) was very similar to the Breslau et al. (1998) report.

The conditional risk for PTSD in females after exposure to trauma was significantly higher in this sample than in a community sample. This finding may be the result of several factors. First, the physiological arousal symptoms associated with PTSD may be misinterpreted as a medical rather than psychiatric problem and lead to physician visits. A second reason may be lack of access to specialty mental health care. Primary care settings have been labeled the “de facto” providers of mental health care due to the high number of patients with psychiatric problems and amount of psychotropic medications prescribed (Regier, Goldberg, & Taube, 1978). This is

especially true for low-income populations who often lack insurance and financial resources to obtain specialty mental health care.

In addition, low-income populations have higher levels of psychological distress in general (Perez-Stable et al., 1990), which may lower their ability to cope with traumatic stressors, possibly leading to PTSD symptomatology. This is supported by the theory of Post, Weiss, and Smith (1995), who theorized that individuals who experience intermittent, unpredictable minor stressors may be “sensitized,” and therefore less able to cope with traumatic stressors. Additional studies examining the combination of minor stress, exposure to trauma, and PTSD may help clarify this possible relationship.

Another consideration in the high rates of PTSD seen in this sample may be the change in criteria for DSM-IV. The introduction of PTSD resulting from traumatic death to family members / loved ones may be at least partially responsible for the increased prevalence of lifetime and current PTSD in this population. Sudden or traumatic death of a loved one was both the most commonly reported traumatic event and PTSD-provoking event in this study. When this event is not included in the data analysis, the prevalence rates of both traumatic life events and PTSD are similar to community studies using DSM-III-R criteria (Kessler et al., 1995, Breslau et al., 1992).

Results from this study indicate that a diagnosis of PTSD was highly associated with comorbid axis I mental disorders such as depression and other anxiety disorders. Participants with a history of PTSD consistently showed increased psychiatric comorbidity across virtually all diagnostic categories examined. This replicates both

community samples (e.g., Kessler et al., 1995) as well as specific trauma populations such as crime victims (e.g., Boudreaux et al., 1998), disaster victims (Green et al., 1990), and combat veteran samples (Kulka et al., 1990).

Interestingly, the only diagnostic categories not associated with increased comorbidity were alcohol and drug use disorders. The data from this study are in contrast to other studies with community (Kessler et al., 1995), combat veteran (Kulka et al., 1990), and disaster samples (Green et al., 1990) that have demonstrated significant comorbidity patterns with alcohol/drug use disorders. One potential explanation for this result is that the sample consisted of mainly African American females, a population that has been documented to have lower alcohol and tobacco use rates (U.S. Department of Health and Human Services, 1991). Indeed, the overall prevalence of alcohol and drug use disorders in this study was minimal (<1%).

Results from this study found that the median duration of PTSD symptoms varied according to trauma type, with PTSD from interpersonal violence having a median duration twice as long as other events directly experienced by the participant and events happening to others (48 vs. 12 and 12 months, respectively). Interestingly, the median duration of PTSD symptoms in individuals experiencing sudden/traumatic death of a loved one in this study was almost exactly the same as the only other study including traumatic death (12.1 months, Breslau et al., 1998). Similar to this study, the Breslau et al. (1998) study found that directly experienced events resulted in significantly longer duration of symptoms than traumatic death or other indirectly experienced events. Breslau et al. (1998) did not specifically examine the duration of

symptoms in subjects reporting interpersonal and other directly experienced events. however.

Results from this study found that individuals with PTSD view their physical health less favorably than do control subjects with no history of mental disorder after the variables of age, alcohol abuse, tobacco use, and obesity were controlled for. These findings replicate several other studies that have found impaired self-reported health status in a variety of trauma populations, including combat veterans and victims of sexual assault (Beckham et al., 1998; Koss, Woodruff, & Koss, 1990; Long, Chamberlain, & Vincent, 1992). This study extends these findings to a heterogeneous group of trauma victims, few of which were in any type of specialty mental health treatment.

Self-reported health status has been identified as an important variable in determining the severity of psychiatric symptomatology (Wells et al., 1989). It has also been related to service use and treatment outcome (Wilson & Cleary, 1992). This is true of PTSD as well, where reports have found that self-reported health status impairments are well predicted by the magnitude of a stressor (Koss et al., 1991). In addition, Malik, Conner, Sutherland, Smith, Davison, and Davidson (1999) found that successful treatment of PTSD symptoms via fluoxetine was associated with increases in perceived health.

The impairments in self-reported health status by PTSD cases may be related to several factors. First, the impairments in health status may be a reflection of the intense physiological arousal experienced when patients are reminded of their

traumatic event. The arousal could be interpreted as a medical symptom and may prompt them to rate their health as more impaired than control subjects. Second, the rated impairments may be representative of medical conditions brought on by chronic disruptions in ANS and HPA axis functioning. In this regard, the reported impairments in health status may reflect true medical symptomatology. A third possibility is that the stress of mental illness itself may be reflected in poorer rated health. Thus, the poorer ratings may be a 'cry for help' resulting from psychological distress. Further research into the physiological effects of PTSD and other mental illness may clarify these issues.

Posttraumatic stress disorder was significantly related to the presence of physician rated ICD-9 circulatory system disease. Participants with a history of PTSD were more than twice as likely than clean cases to have a circulatory system disease after age, tobacco use, alcohol use, and obesity were controlled for. However, these results were not found when PTSD participants were compared to psychiatric control cases. Thus, both PTSD and psychiatric control cases had roughly twice the incidence of circulatory system disease as clean cases. Associations for other medical disorders including neoplasms, respiratory disorders, skin disorders, blood disorders, genitourinary disorders, digestive disorders, and nervous system disorders were not demonstrated.

Although not significant, there was a trend that associated a PTSD diagnosis with the presence of a musculoskeletal system disease. PTSD cases were 1.9 times as likely as clean cases to have a musculoskeletal system disease after age, alcohol abuse

history, tobacco use, and obesity status were controlled for. In addition, the odds ratio was nearly identical (2.0) when PTSD cases were compared to psychiatric control cases.

These results provide initial support for an association between PTSD and medical outcomes. This study replicates results indicating higher rates of self-reported medical impairments in PTSD samples, including Boscarino (1997), Breslau and Davis (1992), and Litz, Keane, Fisher, Marx, & Monaco (1992), and extends them to objective disease outcomes completed via medical chart review. In addition, this work compliments that of Buckley (2000) who demonstrated via meta-analysis that individuals with PTSD were more likely than control subjects to have higher resting heart rate and blood pressure in psychophysiological reactivity studies.

The failure to detect significant differences in both self-reported and physician-rated health outcomes between PTSD and depression control participants is not surprising. Several reports have documented self-reported health status impairments in depressed individuals (Wells et al., 1989). In addition, there is literature that indicates that individuals with depression show abnormal HPA axis functioning and altered catecholaminergic functioning (Arborelius, Owens, Plotsky, & Nemeroff, 1999; Plotsky, Owens, & Nemeroff, 1998). Furthermore, there are also reports indicating an association between depression and cardiovascular disease (Rosanski, Blumenthal, & Kaplan, 1999). Subjects with depression often report a number of physiological symptoms such as decreased appetite, insomnia, and increased pain complaints. Thus, it is unclear whether PTSD and depression have their effects by

similar means. Complicating this is the fact that 45% of the participants in the sample with PTSD also had a comorbid diagnosis of depression. Unfortunately, sample size restrictions prevented secondary analyses examining the role of a comorbid depression diagnosis on medical outcomes.

There are a number of weaknesses to the current study. First, this study was cross-sectional and correlational in nature, and was only able to determine the presence of an association between PTSD and health outcomes. Cross-sectional data prevents inferences in causality, and it is possible that participants with PTSD and depression may have had impaired health outcomes prior to their onset of PTSD or depression. In addition, participants were required to retrospectively report on traumatic events, onset and duration of symptoms, and severity of symptoms. It is entirely possible that participants misjudged, misrated, overestimated, or underestimated a number of variables related to psychiatric symptomatology. Although retrospective studies are commonly utilized in psychiatric research, future studies should attempt to prospectively examine the development of medical outcomes in trauma populations.

A second weakness of this study relates to the heterogeneous nature of the PTSD group. Although this study is unique in that it draws from a number of traumatic events, utilizing a homogeneous trauma population such as combat veterans or disaster samples may eliminate potential confounds making a Type II error less likely. For example, the use of a homogeneous trauma population such as disaster victims eliminates the potentially confounding effects of symptom duration in outcome. Another way to control for this confound is to utilize a prospective design.

The high rate of comorbid depression in the PTSD group limits the conclusions that can be made about the results. It is impossible to determine whether the medical outcomes were due to PTSD, depression, a combination of both, or another variable. Small sample size of PTSD cases prevented further analyses that could have controlled for comorbid depression. Furthermore, the use of a depression psychiatric control group may not have been the most appropriate choice. The known HPA and catecholaminergic dysfunction seen in depression limits the conclusions that can be drawn from nonsignificant differences between the two groups. The choice of another psychiatric condition with less pervasive HPA and SNS dysfunction such as generalized anxiety disorder may have been a more appropriate choice.

A fourth weakness of this study concerns the population studied. Low-income populations provide a challenge to psychiatric research given the low literacy rates and lack of tailored instrument development. Although every attempt was made to use instruments that were clear and easy to understand, a number of difficulties in communicating with participants were noted. Given the level of intimacy and disclosure required in answering questions about psychological distress and disorder, participants may have responded in subtle ways that affected the validity of the diagnostic interviews. Other studies using geographically similar primary care populations have shown a tendency for participants to respond in a socially desirable manner (Boudreaux, 1997).

Last, although medical diagnoses were objectively reported via a physician-generated chart review, no lab or diagnostic testing occurred to validate the presence

of medical disease in this study. The only study to date that utilized lab and diagnostic testing (CDC, 1988) had nonsignificant results. Thus future studies should have corroborative laboratory and diagnostic testing as a more rigorous outcome measure.

The high rates of trauma and PTSD seen in this study indicate that primary care physicians may have significant numbers of traumatized patients in their waiting rooms. This underscores the need for physicians to assess for trauma and its sequelae. Furthermore, primary care physicians should be aware of the proper specialty mental health care practitioners to which to refer patients undergoing significant distress after trauma. In particular, it may benefit primary care practitioners to have a referral source trained in the practice of Critical Incident Stress Debriefing (CISD). CISD is a technique used to assist individuals to cope with the emotional aspects of trauma (Everly, 1995) and is utilized by a number of state agencies such as police officers, firefighters, and others.

The medical morbidity associated with PTSD further underscores the need for better identification and treatment of trauma. Untreated PTSD may drive up practitioner costs with higher medical utilization rates, laboratory testing, and other costs related to medical morbidity. Early identification and treatment of the psychiatric complications of trauma may beneficially affect this. For example, studies examining the treatment of depression in primary care have demonstrated reduced costs associated with better identification and treatment (Panzarino, 1998; Revicki, Simon, Chan, Katon, & Heiligenstein, 1998; Von Korff, Katon, Bush, et al., 1998). This may be achievable in the case of PTSD, and may have an added benefit of

improving medical morbidity. Further studies are needed to examine the physiological profiles of individuals who have recovered from PTSD symptomatology to demonstrate this.

In addition, the finding that the sudden or traumatic death of a loved one was the primary PTSD-provoking event in this study warrants further attention. Although there are several recent reports documenting PTSD symptomatology after exposure to this event (e.g., Murphy, Braun, Tillery, Cain, Johnson, & Beaton, 1999; Prigerson, Shear, Frank, & Beery, 1997; Sigman & Wilson, 1999), there has been little research investigating the physiological reactivity patterns of individuals experiencing PTSD from the sudden or traumatic death of a loved one. Thus, future studies should be aimed at examining the comorbidity, duration, and other factors involved in traumatic grief in order to validate the inclusion of this broader event description in the DSM.

In summary, the present study found a high prevalence of traumatic life events and PTSD symptomatology in a low-income, primary care population. This study also supported previous findings that PTSD, like other psychiatric conditions, is an important predictor of self-reported health status impairments. It was found that PTSD is also associated with increased incidence of circulatory system disease, after risk factors of age, alcohol abuse, tobacco use, and obesity status were controlled for.

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APPENDIX A: DIAGNOSTIC CRITERIA FOR PTSD

A. The person has been exposed to a traumatic event in which both of the following were present:

(1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others.

(2) the person's response involved intense fear, helplessness, or horror.

NOTE: In children, this may be expressed instead by disorganized or agitated behavior.

B. The traumatic event is persistently reexperienced in one (or more) of the following ways:

(1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. NOTE: In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.

(2) recurrent distressing dreams of the event. NOTE: In children, there may be frightening dreams without recognizable content.

(3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated). NOTE: In young children, trauma-specific re enactment may occur.

(4) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event

(5) physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:

(1) efforts to avoid thoughts, feelings, or conversations associated with the trauma

(2) efforts to avoid activities, places, or people that arouse recollections of the trauma

(3) inability to recall an important aspect of the trauma

(4) markedly diminished interest or participation in significant activities

(5) feeling of detachment or estrangement from others

(6) restricted range of affect (e.g., unable to have loving feelings)

(7) sense of a forshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:

(1) difficulty falling or staying asleep

(2) irritability or outbursts of anger

(3) difficulty concentrating

(4) hypervigilance

(5) exaggerated startle response

E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than one month.

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Source: *DSM-IV* (APA, 1994, pp. 427-9).

APPENDIX B: HISTORY OF PTSD

Although PTSD was not formally recognized as a mental disorder until the publishing of the DSM-III in 1980, there is a rich history documenting the adverse physiological and psychological effects of exposure to traumatic life events. Early accounts of adverse reactions to traumatic life event exposure come from reports of civilian disasters such as train wrecks, fires, and other disasters as well as war-related syndromes (Kinzie & Goetz, 1996). These reactions carried several different labels and had descriptions of symptom clusters. For example, Railroad Spine Syndrome was used to describe the psychological and physiological aftermath of train wreck victims (Erickson, 1867). War-related reactions carried labels such as Da Costa's Syndrome, irritable heart, shell shock, effort syndrome, soldier's heart, and combat stress reaction (Hyams, Wignall, & Roswell, 1996).

Comparisons of these various reactions reveal similar symptom profiles. For example, Hyams et al., (1996) examined several of the war-related conditions, and found that symptoms common amongst all of the 'syndromes' were sleep disturbance, nightmares, hyperarousal/irritability, heart palpitations, and shortness of breath. Psychosis and/or dissociation were present in some of these syndromes as well. Thus, it appears that several early researchers were cataloging similar symptoms from very different situations with one common thread: exposure to some type of traumatic life event.

Also similar in the descriptions of the various syndromes were the explanations of the development of symptoms. The authors of these studies generally tended to

attribute the symptoms to some organic cause such as spine damage as a cause of the adverse reactions seen in Railroad Spine Syndrome: infectious disease as the etiology of DaCosta's Syndrome, as well as others (Kinzie & Goetz, 1996). Later, in World War I and World War II, there was a speculation of a psychogenic component to the military combat syndromes such as shell shock and combat stress reaction. For example, Salmon and Fenton (1929, cited in Kentsmith (1986)) found that patients with shell shock showed better and faster treatment rates when treated near the war front than those that had been evacuated and treated away from front lines. Even through World War II, however, there was still some discussion as to whether there was a physiologic or psychogenic basis to these reactions (Hyams et al., 1986).

Efforts to categorize the adjustment difficulties to traumatic life events were described in the psychiatry literature, and fell under the dominant school of thought at the time: psychoanalytic theory. Freud's theory conceptualized these "war neuroses" as a conflict between the aversive state of war atrocity and aggressive instincts, with the driving factor of the neurosis being repression (Wilson, 1994). Two important contributions came from Freud's conceptualization of the disorder. First, he was amongst the first to ascribe a functional versus organic cause of these reactions. This opened the concept to further study from psychiatry and psychology. Second, he continually refined the clinical description of the syndrome, which was essentially written into the first edition of the DSM in 1952 (Wilson, 1994).

DSM-I

In the first edition of the DSM (APA, 1952), no formal diagnosis of PTSD was listed. Instead there was the diagnosis of “Gross Stress Reaction.” which fell in the category of “Transient Situational Personality Disorders.” The disorder was marked by a change in personality function in reaction to situations in which an individual has been subjected to severe physical demands or extreme emotional stress. Specifically listed were combat and civilian disasters such as fire, earthquake, and explosion. Emphasized in this diagnosis was that the disorder was viewed as transient, and that treatment was associated with ‘cure.’

DSM-II

The second edition of the DSM, (DSM-II, APA, 1968) changed the nature of the stress reaction. The diagnosis of Gross Stress Reaction disappeared, and was replaced with a category of disorders called “Transient Situational Disturbances.” This diagnosis carried little description to it, except for examples of stress reactions (such as, “Fear associated with military combat and manifested by trembling, running, and hiding.” [APA, 1968, p. 49]). Some reported the second revision of the DSM actually represented a step backwards in the description and conceptualization of reactions to trauma (Wilson, 1994).

DSM-III

The publication of the third edition of the DSM (DSM-III, APA, 1980) marked the official entry of PTSD into the psychiatric nomenclature. The DSM-III represented a major departure in the conceptualization and description of mental disorders from

prior editions of the DSM. Significantly, DSM-III was promoted as an empirical and data-driven attempt to classify mental disorders according to testable and verifiable diagnostic criteria.

The first criterion for DSM-III PTSD (criterion "A") was that an individual needed exposure to an event that was "outside the range of usual human experience," and would cause distress in "most people." DSM-III listed a number of qualifying traumatic events including rape, military combat, natural disasters, torture, car accidents, airplane crashes, and others. More common types of traumatic events such as bereavement, chronic illness, business loss, or marital conflict were specifically listed as stressors that would not qualify for a PTSD diagnosis. The second criterion (criterion "B") of the disorder included re-experiencing symptoms such as intrusive recollections, nightmares, and flashbacks of the event. The third criterion (criterion "C") of DSM-III PTSD included numbing symptoms such as reduced interest, constricted affect, and feelings of detachment. Last, in criterion "D," the individual needed at least two of six anxiety/depression symptoms, including hyper-alertness/exaggerated startle response, sleep disturbance, survival guilt, memory impairment/trouble concentrating, avoidance of events that arouse recollection of the traumatic event, and intensification of symptoms by exposure to events that symbolize or resemble the traumatic event. (APA, 1980).

DSM-III-R

The first major revision of the formal DSM diagnostic criteria for PTSD came with the publishing of the DSM-III-R (APA, 1987). In this edition, significant

restructuring of the symptom profile of the disorder occurred. The “stressor criterion” became slightly more specific, and the re-experiencing, numbing, and “other” symptoms were restructured substantially. Specifically, DSM-III-R PTSD listed criterion “B” as symptoms of avoidance versus “numbing” symptoms. Also, criterion “D” changed from a mixture of “other” symptoms to symptoms of arousal.

The publishing of the DSM-III and DSM III-R as well as a federally funded initiative to study the traumatic effects of the Vietnam War helped to stimulate tremendous amounts of research on PTSD (Kulka et al., 1991). Researchers then had the ability to study the effects of both civilian and combat-related trauma with a single set of diagnostic criteria. Other aspects of the disorder including the prevalence, course, treatment, and other crucial aspects of the disorder were opened up because of the addition of PTSD into the DSM.

The accumulation of vast amounts of data regarding PTSD also brought out the weaknesses in the DSM-III and III-R conceptualizations of the disorder. Authors such as Breslau and Davis (1987) criticized the validity of PTSD, stating that evidence was lacking to uniquely associate PTSD symptoms with catastrophic stressors, and that the same “catastrophic” amounts of stress did not bring out symptoms of PTSD to all who experienced them. Other evidence from field trials and other large scale studies provided validity for the disorder, as well as the background for yet another substantial revision in diagnostic criteria for the disorder (Kilpatrick & Resnick, 1993; March, 1993).

DSM-IV

The major criteria changes in DSM-IV related to the “A” or “stressor” criterion. In addition to the traumatic life events that would cause distress in “most anyone,” DSM-IV added several other traumatic events that were more common in the community. These included number of “witnessed events,” including: observing the serious injury or unnatural death of another person due to violent assault, accident, war, or disaster or unexpectedly witnessing a dead body or body parts. Furthermore, DSM-IV lists several traumatic events that if experienced by a close friend or family member can be associated with the development of PTSD, including: violent personal assault, serious accident, or serious injury experienced by a family member or close friend, learning about the sudden, unexpected death of a family member or a close friend, or learning that one’s child has a life-threatening disease. The change in the stressor criterion places significantly more emphasis on coping or adapting to a traumatic event as opposed to the event itself.

To summarize, although there is a vast amount of literature on the topic of trauma and its sequelae, the diagnosis of PTSD has only existed since 1980. The disorder was initially conceptualized as only occurring in only a few individuals exposed to extreme stressors. Subsequent research has changed the conceptualization to reflect individual coping abilities of those exposed to a variety of traumatic events. Hence, PTSD is now viewed more in terms of an abnormal stress response than one of extreme stressor alone. This is reflected in the significant changes to the stressor criterion and other criteria of the disorder.

APPENDIX C: DEMOGRAPHIC QUESTIONNAIRE

- 1) Subject Number: _____
- 2) Age: _____
- 3) Medical Record #: _____
- 4) Clinic: () Med Clinic () Family Prac
- 5) Sex (circle one): Male Female
- 6) Job/Occupation: _____
- 7) Marital Status (circle one):
- a. Single
 - b. Married
 - c. Separated
 - d. Divorced
 - e. Other (please specify) _____
- 8) Race (circle one):
- a. White (Non-Hispanic)
 - b. African-American
 - c. Hispanic
 - d. Asian
 - e. Other (please specify) _____
- 9) Please circle the highest grade you have completed:
- Grade school: 1 2 3 4 5 6 7 8 9 10 11 12
- College/Trade School: 1 year 2 years 3 years 4 years More than 4 years
- Have you completed high school? (circle one): Yes No
- If you have not graduated from high school, do you have a GED? (circle one): Yes No
- 10) Other education (please specify type and number of years): _____
- 11a) What is your average monthly income? \$ _____
- 11b) Where does this money come from? (circle each one that applies to you and indicate the amount of money you receive from that source each month):
- a. My job/Career \$ _____
 - b. Public assistance/Welfare \$ _____
 - c. Social Security/Disability \$ _____
 - d. Unemployment \$ _____
 - e. Child support/Alimony \$ _____
 - f. Allowance \$ _____
 - g. List other sources of income: _____ amount \$ _____
- 12a) How many people live in your home? _____
- 12b) What is the total monthly income including everyone in your home? \$ _____
- 12c) Where does this money come from? (circle each one that applies to your family and indicate the amount of money your family receives from that source each month):
- a. Jobs/Careers \$ _____
 - b. Public assistance/Welfare \$ _____
 - c. Social Security/Disability \$ _____
 - d. Unemployment \$ _____
 - e. Child support/Alimony \$ _____
 - f. List other sources of income: _____ amount \$ _____
- 13) Have you ever received treatment for a mental health problem? Yes No
- What kind of problem? _____
- 14) Have you ever received treatment for a drug or alcohol problem? Yes No
- What kind of problem? _____
- 15) Do you have any health insurance? Yes No If so, what kind? _____
- 16) Address: _____
- 17) Phone Number: _____

APPENDIX D: PATIENT'S GLOBAL HEALTH STATUS RATING

Physician: _____
EKL #: _____

Subject Number: _____
Date: _____

Does this patient have any existing medical condition(s)? If yes, list the medical conditions and indicate if they are acute or chronic.

MEDICAL CONDITION	ACUTE/CHRONIC
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____

Please rate the patient's global health status using the following scale by circling the number which best describes the patient's overall physical health:

- 0 patient is in good physical health with a history of only routine minor illnesses.
- 1 patient is largely free of serious medical problems but presents with numerous undiagnosed symptoms and complaints.
- 2 patient is largely free of serious medical problems but has one or more risk factors (e.g., smoking) which places him/her at risk for the development of future illness.
- 3 patient has a history of serious disease which is currently in remission - patient appears in good health and is compliant with medical recommendations.
OR
patient has a chronic degenerative disease (e.g., diabetes) which is well controlled and patient is compliant with medical recommendations.
- 4 patient has a history of serious disease which is currently in remission - but patient is non-compliant, engages in health risk behaviors, or has additional aggravating illnesses.
- 5 patient currently has one or more chronic degenerative disease(s) which is poorly controlled.
- 6 patient has terminal illness; death is imminent.

APPENDIX E: CONSENT FORM

1. **Study Title:** The Roles of Stress, Social Support, and Psychopathology in Primary Care Utilization.
2. **Performance Sites:** Family Practice and General Medicine Clinics, Earl K. Long Medical Center, Baton Rouge, LA.
3. **Names and Telephone Numbers of Investigators:**
For 24-hour access, please contact Isabel Scarinci or Annette Springer at 358-1105.
Phillip J. Brantley, Ph.D.....(504) 358-1105
John Howe, M.D.....(504) 358-1103
Glenn Jones, Ph.D.....(504) 358-1105
4. **Purpose of the Study:** This is a research study to determine the roles of stress, mental health, social support, and coping strategies in primary care utilization.
5. **Subject Inclusion Criteria:** Male and female volunteers ages 18 and older who are patients in the EKL Department of Family Medicine or EKL General Medicine Clinic will qualify as subjects in this research project.
6. **Subject Exclusion Criteria:** Subjects will be excluded from Phase II of the project for not having a phone at home.
7. **Description of the Study:** This is a research study to determine the role of stress, mental health, social support, and coping strategies in primary care utilization. Subjects will participate in the study in two phases. Subjects do not have to participate in Phase II to participate in Phase I. Subjects must participate in Phase I to participate in Phase II.
In Phase I, subjects will be chosen from waiting rooms at the EKL Department of Family Medicine and General Medicine Clinic. Subjects who agree to participate will complete the demographic questionnaire and the General Health Questionnaire (GHQ).
Phase II of this project will be divided into 4 tasks:
(1) Subjects will complete the following questionnaires on the same day: the Interpersonal Support Evaluation List (ISEL) (a measure of social support), the Ways of Coping Questionnaire (WOC) (a measure of coping strategies), the Weekly Stress Inventory (WSI) (a measure of minor stress), the 1994 Behavioral Risk Factor Questionnaire (a measure of health risk behaviors), and the Ways of Religious Coping Scale (a measure of religious coping).

Subject's Initials

(2) Subjects will be asked to complete the WSI and a self-report hospital utilization questionnaire (SRU) bimonthly for one year, and in the sixth month the Life Experiences Survey (LES) (a measure of major stressors) will be added to the phone interview. A research assistant will contact subjects by phone once every month in order to collect the responses to these questionnaires and to answer any questions subjects may have:

(3) One year after the initial contact, subjects will be scheduled to complete the following at the Center for Primary Care Research at EKL: (a) Composite International Diagnostic Interview (CIDI) (a mental health interview), (b) ISEL, (c) WOC, and (d) SRU.

(4) Subjects will be contacted by phone 3, 6, 9, and 12 months following the interview and asked to answer the SRU.

8. **Benefits to Subject:** At the end of the study, subjects will be provided with a summary report of findings and their relevance to primary care utilization, at their request. If needed, subjects will receive a referral to an appropriate agency.
9. **Risks to Subject:** No known physical risks. Participation in this study may involve unforeseen risks.
10. **Alternatives to Participation in the Study:** Since no treatment is involved in this study, the only alternative to participation in the study is not to participate.
11. **Subject Removal:** Subjects will be removed from the study if they fail to complete (1) Phase I; (2) part 1 of Phase II; (3) 80% or more of requested bimonthly interviews; (4) part 3 or 4 of Phase II. There is no risk involved in being removed from the study.
12. **Subject's Right to Refuse to Participate:** Study subjects may refuse to participate or withdraw from the study at any time without jeopardizing, in any way, their medical treatment at this institution in the present or future. Should significant new findings develop during the course of the research that may relate to the subject's willingness to continue participation, that information will be provided to the subject. There are no special risks involved in withdrawal from the study.
13. **Subject's Right to Privacy:** The results of the study may be released to the funding agency. The results of the study may be published. The privacy of subjects will be protected and they will not be identified in any way.

Subject's Initials

14. **Release of Information:** The medical records related to the study are available to the sponsoring agency. Information provided during the course of the study is confidential. The only exceptions are in cases where subjects indicate suicidal desires, homicidal desires, or child abuse. In these instances the researchers are ethically and legally required to inform their supervisors regarding the subject's desires.
15. **Financial Information:**
 A. Participation in this study will not result in any extra charges above and beyond those routinely incurred by patients with similar illnesses.
 B. The costs of study related and unforeseen complications must be met by subjects.
 C. Subject Payment: Subjects will be paid \$15 (fifteen dollars) for completing Phase I of the study. Subjects will be paid \$20 (twenty dollars) for completing part 1 of Phase II. Subjects will be paid \$10 (ten dollars) for each phone interview completed during part 2 Phase II. Subjects will be paid \$50 (fifty dollars) for completing part 3 of Phase II and \$10 (ten dollars) for each phone interview completed during part 4 of Phase II.
16. **Signatures:** The study has been discussed with me and all my questions have been answered. I understand that additional questions regarding the study should be directed to investigators listed on page 1 of this consent form. I understand that if I have questions about subjects' rights or other concerns, I can contact the Chancellor of LSU Medical Center, at (504) 568-4801. I agree with the terms above and acknowledge I have been given a copy of the consent form.

_____	_____
Signature of Subject	Date
_____	_____
Signature of Witness	Date

The study subject has indicated to me that the subject is unable to read. I certify that I have read this consent form to the subject and explained that by completing the signature line above the subject has agreed to participate.

_____	_____
Signature of Reader	Date

The study subject is a child and I certify that I am his/her legal guardian.

_____	_____	_____
Legal Guardian Name	Legal Guardian Signature	Date
_____	_____	_____
Child's Name and Age	Child's Signature	Date

Reason for not obtaining child assent: _____

VITA

Bradford Applegate received his bachelor's degree in psychology from Virginia Polytechnic Institute and State University in 1992. While at Virginia Tech, he participated in research with the well known researcher in child clinical psychology, Tom Ollendick. It was at Virginia Tech that he began to enjoy conducting research and desired to study psychology further. He was originally interested in the field of child clinical psychology.

Brad went on to Mississippi State University in Starkville, Mississippi, where he pursued a master's degree in experimental psychology. AT M.S.U he presented several posters at regional psychology meetings, and participated in a number of research projects in order to boost his curriculum vita to the point where doctoral caliber institutions would be interested in him. On the whole they weren't, as Brad received rejection letters from over 12 doctoral institutions. However, Phillip Brantley at Louisiana State University saw sufficient enough merit to offer Brad first an interview, and later the opportunity to study clinical psychology at L.S.U.

At L.S.U., Brad gained valuable research and clinical skill in the area of adult behavioral medicine. He had the opportunity to participate in a National Institutes of Mental Health-funded grant examining the roles of stress and psychopathology in medical utilization in low-income primary medical care patients. Brad also conducted research in the area of hypertension while at L.S.U. He presented several papers at national research conventions, and published two papers under the guidance of Phillip Brantley.

His efforts at L.S.U paid off as he was offered an internship from The University of Mississippi Medical Center / Jackson Veterans Affairs Clinical Psychology Consortium in 1998-9. While in Jackson, Brad gained valuable clinical experience in the treatment of psychopathology, nicotine addiction, trauma, and eating disorders.

Brad elected to stay in Jackson after completing internship, and is currently a postdoctoral fellow in the Department of Family Medicine at the University of Mississippi Medical Center, where he performs a variety of clinical, research, and teaching activities.

Brad was recently hired as a research coordinator for a series of primary care based tobacco cessation projects. He hopes to maintain ties to academics, especially in family or internal medicine. The degree of Doctor of Philosophy was awarded to him in August, 2000.


DOCTORAL EXAMINATION AND DISSERTATION REPORT

Candidate: Bradford West Applegate


Major Field: Psychology

Title of Dissertation: Traumatic Life Events, Posttraumatic Stress Disorder,
and Health Outcomes in a Low-Income, Primary Care
Population

Approved:





Major Professor and Chairman





Dean of the Graduate School


EXAMINING COMMITTEE:











Date of Examination:

May 12, 2000