

ETIOLOGY OF TRAUMATIC BRAIN INJURY:
IMPACT ON PSYCHOLOGICAL FUNCTIONING

A Dissertation

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ABSTRACT

Traumatic brain injury (TBI) due to violence has received increased research focus in recent years, with numerous studies examining the impact of violent TBI on cognitive and psychosocial outcomes. However, few studies have examined psychological outcomes following violent TBI. The present study examined the prevalence of symptoms of depression and anxiety in individuals with TBI due to violence ($n = 180$) and other causes ($n = 360$). Results indicated that while etiology was not a significant predictor of psychological status, overall there were some differences in endorsement of depressive and anxious symptoms among individuals with moderate to severe injuries compared to those with mild injuries. Implications of these findings and future directions are discussed.

INTRODUCTION

Traumatic brain injury (TBI) and violence are both serious public health problems in the United States. TBI affects an estimated 1.4 million people each year (Langlois, Rutland-Brown, & Thomas, 2006), of whom 50,000 will not survive, 235,000 will survive but be hospitalized, and 80,000 to 90,000 will develop a long-term disability (Machamer, Temkin, & Dikmen, 2003). Currently, approximately 5.3 million people are living with a TBI-related disability (Thurman, Alverson, Dunn, Guerro, & Sniezek, 1999). Injuries due to violence are also highly prevalent in the United States. An estimated 1.9 to 2.3 million persons per year receive nonfatal injuries as a result of interpersonal violence (Rosenberg & Fenley, 1991).

Individuals who suffer traumatic brain injury (TBI) may receive their injury due to a variety of causes. The most frequent causes of TBI appear to be motor vehicle accident (MVA), violence, and falls. Estimates of the rates of TBI due to various causes vary across studies and have changed over time. Historically, MVA has been the leading cause of TBI; however, recent reports from the National Center for Disease Control have indicated that falls are responsible for 28% of TBI, followed by MVA at 20%, struck by/against events at 19%, and assaults at 11% (Langlois et al., 2006). These rates vary according to additional factors such as age and population density. Kraus (1993) found that teenagers and young adults are more likely to be injured in vehicular crashes or through violence, while older adults are more likely to be injured in falls. Studies conducted in urban areas have found interpersonal violence to be the leading cause of TBI, with young, minority, urban-dwelling males at highest risk (Cooper et al., 1983; Whitman, Coonley-Hoganson, & Desai, 1984).

Estimates of the rate of TBI secondary to violence have increased over the years. The 1991 National Health Interview Survey reported that violence was responsible for 9% of all

nonfatal TBIs (Sosin, Sniezek, & Thurman, 1996). More recent studies have shown an escalation of this prevalence. Reporting data from the nationwide TBI Model Systems database, Harrison-Felix and colleagues (1998) found that 29% of injuries were attributable to a violent etiology, and Hanks et al. (2003) later placed the rate of violent TBI at 26%. This increase has led to greater attention being paid to research on outcomes from violent TBI.

TBI due to violent etiology can come in many forms. Injuries are typically thought to be either penetrating, caused by gunshot or stab wounds to the head, or blunt assault injuries. However, interpersonal violence may also result in TBI from falls, as when the victim is pushed from a height or moving vehicle, or from the victim being intentionally struck by a motor vehicle (Hart, Bogner, Whyte, & Polansky, 2003). Regardless of the exact mechanism, the common unifying theme that distinguishes violent TBI from non-violent TBI is the intention to inflict harm (Schopp et al., 2006). Blunt trauma is the leading cause of violent TBI, followed by penetrating injury. Harrison-Felix et al. (1998) placed the incidence of violent TBI due to blunt assaults at 56% (blunt trauma, unarmed fight, or jump), with 23% resulting from penetrating objects (19% firearm injuries) and 21% with the specific mechanism of violence being unknown. In a study restricted to individuals with TBI due to violence, Black et al. (2002) found that 24% of cases had penetrating injuries and the other 76% had received blunt trauma.

Risk Factors for Violent and Other TBI

Many factors are thought to be related to risk for TBI. Demographic factors studied previously include age, gender, race, and socioeconomic status (SES). Population based studies have generally reported a trimodal age distribution for TBI occurrence, with incidence peaking in early childhood, late adolescence/early adulthood, and in the elderly (Bruns & Houser, 2003). Among these age groups, the highest age-specific incidence is in the adolescent/young adult age

group. The high rate of TBI in this age group is typically attributed to the higher rates of impulsivity and risk-taking behavior displayed by younger adults, which could also explain the higher rate of injuries related to vehicular crashes or violence in teenagers and young adults (Bushnik, Hanks, Kreutzer, & Rosenthal, 2003).

TBI incidence declines among middle age adults, which is thought to result from a decline in the impulsivity of the younger years (Bruns & Hauser, 2003) and before the increase in falls of the later years. A combination of sensory and motor decline, deconditioning, and cognitive or conscious impairments may lead to an increased risk of falls and MVAs in the geriatric population (Bruns & Hauser), which in turn would increase the risk for and vulnerability to TBI. In line with this thinking, Kraus (1993) found an increase in incidence of TBI occurs in the geriatric population (>75 years), with injuries resulting primarily from falls.

Gender, race/ethnicity, and socioeconomic factors are also thought to influence risk for TBI. Overall, males are about 1.5 times as likely as females to sustain TBI (Langlois et al., 2006). This ratio is highest during adolescence and early adulthood, peak times for interpersonal violence and MVAs, and can approach or exceed 3 to 4:1 (Bruns & Hauser, 2003).

Race/ethnicity may also play some role in risk for TBI. Higher incidences have been reported for blacks compared with non-blacks; however, these categorizations are thought to be confounded by SES (Bruns & Hauser). People living in areas with lower average incomes have been found to sustain more TBI than those with higher income (Wagner, Sasser, Hammond, Wiercisiewski, & Alexander, 2000).

These demographic factors can also interact to influence relative risk of TBI. Gender interacts some with age as it relates to TBI risk. Ip, Hesch, Brandys, Dornan, and Schentag (2000) found the highest rate of injury occurred between the ages of 21 to 40 years for males,

and between the ages of 31 to 40 years for females. There also appears to be an interaction between race and age, with males and ethnic minorities more at risk during adolescence and early adulthood. This is related to the increased risk of violence and MVAs in these age groups (Bruns & Hauser, 2003). Combining all these demographic factors in an attempt to identify characteristics of persons with TBI, Sosin and colleagues (1996) found those at greatest risk for brain injury are teens and young adults, males, and persons with low income who live alone.

Demographic characteristics of different etiologies of injury have been examined. Bushnik and colleagues (2003) found that people injured in MVAs are typically in their early thirties, white, involved in productive activity before injury, and have no history of substance abuse, behavioral, or psychological problems. They also found that individuals with TBI resulting from a fall tend to be older and primarily white, married, and retired. Overall, falls predominate as a cause of injury in children and the elderly, regardless of race and gender (Bruns & Hauser, 2003). By comparison, Bushnik found that individuals with TBI due to violence tended to be nonwhite (56% African American, 11% other), unemployed at time of injury, unmarried, and have a premorbid history of illegal substance use and law enforcement encounters.

Research on TBI resulting from violence has found a consistent demographic pattern. Individuals with violence-related injuries are more likely to be male, African American, of younger age, less educated, unemployed, and living alone at the time of the injury (Bogner, Corrigan, Mysiw, Clinchot, & Fugate, 2001; Hanks et al., 2003; Harrison-Felix et al., 1998; Sosin et al., 1996; Wagner, Sasser, et al., 2000). This pattern closely parallels the risk factors for interpersonal violence in general. These include young age, minority status, alcohol and drug use, low socio-economic status, and availability of a firearm (Harrison-Felix et al., 1998). The

pattern of poorer, unemployed, ethnic minorities has also been found in studies of violent TBI in rural settings (Schopp et al., 2006). Individuals with violent TBI are also more likely to have used illegal drugs and been arrested (Bushnik et al., 2003), and have higher blood alcohol levels in the emergency department (Machamer et al., 2003). It is thought that there may be an underlying factor, possibly socioeconomic in nature, associated with risk for violent TBI (Bogner et al., 2001).

Cognitive and Psychosocial Impact of TBI

The impact of TBI can be great, depending on the severity of the injury and the nature of the injury (i.e., location and extent of lesions), as well as the cause of injury. Injury severity is an important factor with regards to outcome from TBI. Severity of injury has been shown to be predictive of acute disposition as well as long-term disability and community integration in survivors of TBI (Wagner, Hammond, Sasser, & Norton, 2000; Wagner, Hammond, Grigsby, & Norton, 2000). The more cognitive deficits an injured individual experiences the more likely that individual will have functional and psychosocial difficulties. Persons with TBI commonly report poor functioning on post-injury psychosocial variables including alcohol and drug abuse, limited financial resources and social support, emotional disturbances, and low rates of vocational rehabilitation (VR) services (Schopp et al., 2006).

Outcomes can also differ based on the etiology of the injury. Bushnik and colleagues (2003) compared outcomes in individuals with TBI due to MVA, falls, violence and other causes. They found individuals injured in MVA to be more severely injured than any other cause. However, despite having more severe initial injuries, persons in vehicular crashes reported the best functional and psychosocial outcomes, with persons with violent injuries

having the worse outcomes, and individuals in the falls and other group having outcomes lying between the MVA and violence groups.

Cognitive and Psychosocial Impact of Violent TBI

Findings have been inconsistent with regards to severity of injury in violent TBI compared to other causes. Some studies have found that survivors of violent TBI have less severe initial injuries than those with nonviolent TBI (Harrison-Felix et al., 1998; Machamer et al., 2003). This is thought to be due to the more focal nature of injuries from assault, as compared to the more diffuse nature of other causes of injury. In contrast to these findings, one study found a higher proportion of severe head injuries resulting from assault (Wenden, Crawford, Wade, King, & Moss, 1998). Bushnik et al. (2003) found injury severities in the moderate to severe disability range for a violent TBI group. Other researchers have found no significant difference in injury severity between those with violent and nonviolent TBI (Hanks et al., 2003; Schopp et al., 2006). When comparing penetrating and blunt trauma intentional injuries, some differences in injury severity have been observed. Zafonte et al. (1997) found a trend for longer duration of posttraumatic amnesia (PTA) in patients with penetrating injury. Black and colleagues (2002) found penetrating injuries are more likely than blunt trauma to be moderate to severe based on Glasgow Coma Scale scores (GCS; Teasdale & Jennet, 1976), although their study did not show a significant difference in length of PTA.

The effects of violence-related TBI can be extensive. Such injuries can significantly impact social, vocational, financial, emotional, and functional independence (Hanks et al., 2003). The immediate impact of violent injury is seen with an increased chance of mortality. Individuals with intentional TBI are more likely to die before discharge (Wagner, Sasser, et al., 2000), particularly with penetrating injuries (Benzel et al., 1991; Kaufman, 1993). For this

reason, long-term outcome from violent TBI has only recently begun to be examined. Some early studies reported worse neuropsychological and psychosocial outcome from violent TBI (Hanlon, Demery, Martinovich, & Kelly, 1999; Wenden et al., 1998). However, these studies did not consider the contribution of premorbid characteristics such as sociodemographic factors to outcome.

Studies that have examined the relative contribution of sociodemographic factors have found these factors account for much of the observed difference in outcomes from violent versus nonviolent TBI. Results of multivariate analyses performed by Harrison-Felix et al. (1998) showed that intentional etiology of injury did not predict functional outcomes, with most of the variance being explained by injury severity and length of unconsciousness. Bogner et al. (2001) similarly failed to find any difference in outcomes due to etiology of injury, with substance abuse history playing a more prominent role in predicting long-term outcomes. Examining neuropsychological and psychosocial outcomes, Machamer et al. (2003) found that injury severity and personal characteristics such as age, gender, race, and education were more important to outcome than the circumstances of the injury. More specifically, they found people with more severe injuries, lower education, older age, male gender, and nonwhite race had worse neuropsychological outcome.

Still, some research findings have been mixed. While failing to observe any difference in functional outcomes due to etiology, Bushnik et al. (2003) found individuals with TBI related to violence tended to have higher unemployment rates and higher rates of separation or divorce within the first year post-injury, though these differences only approached significance. Hanks et al. (2003) found evidence of higher levels of caregiver burden and disability, as well as decreased productivity and community reintegration in the violent TBI group. Overall,

functional outcomes do not appear to differ based purely on the cause of injury. However, trends have been identified toward higher rates of post-injury unemployment/productivity loss, divorce, and lower community integration scores in persons with violent etiology TBI. Also, the interaction of these pre-injury characteristics and the etiology of the TBI may have an adverse impact on outcome. Schopp et al. (2006) concluded persons injured through violence have poorer premorbid functioning and are likely to have less favorable outcomes than the general population.

Emotional Impact of TBI

Traumatic brain injuries can have a profound emotional impact, possibly leading to adverse effects on cognitive and psychosocial functioning. Mood and anxiety disorder in particular appear to be frequent psychiatric complications among TBI patients (Levin, Goldstein, & Mackenzie, 1997; Silver, Kramer, Greenwald, & Weissman, 2001). Risk factors for developing a mood disorder after TBI are prior psychiatric history and impaired social support (Jorge & Robinson, 2003). Multiple factors may contribute to the development of psychiatric problems following TBI. Alterations of personality, mood, and behavior may be organically based, reactive to the trauma and its sequelae, or a combination of both processes (Lucas, 1998). Mood change in moderate to severe injuries is more often organic in nature, although reactive and compensatory changes are possible (Lezak, 1995; Lucas, 1998). With severe TBI, emotional consequences generally involve either exaggeration or muting of affective experience and response, with both the excitable and apathetic patterns of behavioral and emotional alterations having their organic bases primarily in damage to the frontal lobes or underlying structures, with damage to temporal limbic structures also affecting emotionality (Lezak, 1995).

Emotional disturbances are also common with mild head injury. Postconcussive sequelae such as fatigue, irritability, and physical discomfort are often sufficient to produce dysphoric mood (Lucas, 1998). With milder injuries, individuals are more likely to be acutely aware of their deficits, which can contribute to the development of psychopathology. Symptoms of anxiety and depression are frequently reactive to a patient's appreciation of their physical and cognitive disabilities and social limitations (Lezak, 1995). These reactions can take the form of an adjustment disorder following head injury. With mild head injury, adjustment reactions are common and may develop into clinical syndromes of depression and anxiety if not addressed early (Lucas, 1998).

Mood disorders occur with greater frequency following TBI than in the general public. A significant association has been observed between poor psychosocial and activities of daily living outcome and the presence of major depression (Jorge, Robinson, Starkstein, & Arndt, 1994). Depression typically does not occur in isolation following TBI. Jorge and Robinson (2003) point out that major depression is significantly associated with the presence of co-morbid anxiety disorders, particularly generalized anxiety disorder (GAD). The combined impact of these disorders can lead to even greater functional impairment. According to Jorge, Robinson, Starkstein, and Arndt (1993), anxious depressions tend to have a more prolonged clinical course and a greater impact on the patient's psychosocial outcome. Just as cognitive and interpersonal deficits from TBI can lead to emotional problems, symptoms of depression and anxiety can influence cognitive outcomes. Patients with depression or anxiety may be more functionally disabled and consequently perceive their injury and cognitive impairment as more severe (Fann et al., 2002). This interaction between cognitive and emotional factors can greatly impact

functioning after TBI, making assessment of emotional functioning just as important as evaluation of cognitive functioning.

Emotional Impact of Violent TBI

Of the numerous studies on psychological outcomes in TBI, few have systematically examined the influence of cause of injury on subsequent psychological symptoms. Machamer and colleagues (2003) examined responses of individuals with violent and nonviolent TBI to the Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983), focusing on symptoms of depression, anxiety, hostility, and phobic anxiety. They found a significant difference between the violent and nonviolent groups on the composite BSI measure, along with a tendency for those injured violently to report more depression and anxiety than those injured nonviolently. However, once demographics were considered along with nature of injury in the prediction of composite BSI outcome, gender was the only significant predictor in the model, with males reporting more distress than females (Machamer et al., 2003). It may be important to note that the authors did not explicitly state whether or not the trend for more depression and anxiety in the violent group became nonsignificant once demographics were considered. The only reported comparison where demographics were considered was for the composite BSI score, which is comprised of many items in addition to those specific to depression and anxiety.

Another study to examine emotional functioning following violent TBI looked specifically at appraisal of threat and symptoms of avoidance (Riley, Brennan, & Powell, 2004), finding more reports of avoidance in males and those with TBI resulting from assault. The positive result for etiology of injury remained even once gender was accounted for in the analysis. Riley et al. (2004) further argued that greater use of avoidant coping strategies is associated with higher levels of anxiety and depression. Past research on TBI in general has

shown that frequent use of avoidant strategies, such as escape and denial, is associated with greater psychological distress (Moore & Stambrook, 1994).

Several potential reasons for greater psychological impairment in individuals with TBI due to violence have been proposed. According to Riley et al. (2004), TBI arising from assault may present particular challenges in terms of psychological adjustment because it leads to a general loss of confidence and trust in others. Another factor thought to increase the risk of adverse psychological outcome is attribution of blame to others (Hart et al., 2003). Their study found that individuals injured by violent means apportioned significantly more of the blame for their injuries to other people, compared to an accident group.

Another possibility is socioeconomic factors could influence psychological outcomes following violent TBI. As noted previously, risk for violence in general as well as violence resulting in TBI is higher for those of lower SES (Bogner et al., 2001; Harrison-Felix et al., 1998). Numerous studies have examined the relationship between SES and risk for psychiatric illness, with many finding higher rates of psychiatric disorders among those of lower SES (Holzer et al., 1986; Kessler et al., 1994; Ostler et al., 2001; Weich & Lewis, 1998), including symptoms of depression and anxiety (Bruce, Takeuchi, & Leaf, 1991; Gallo & Matthews, 1999; Ross, 2000).

Explanations for the difference in rates of psychopathology among SES groups have largely focused on exposure to environmental stressors in lower SES areas. It has been noted that those with lower SES tend to experience higher levels of stressful life events (Dohrenwend, 1973; McLeod & Kessler, 1990; Turner & Lloyd, 2004). Gallo and Matthews (1999) argue that since lower SES individuals encounter stressors more often, an increased demand is placed on their coping resources, possibly depleting their reserve capacity to deal with future stressors.

There are numerous theoretical explanations behind this process, including stress-diathesis or vulnerability-stress models such as the learned helplessness (Miller & Seligman, 1982) and hopelessness theory (Abramson, Metalsky, & Alloy, 1989), and social causation hypotheses (e.g., Dohrenwend & Dohrenwend, 1969).

In general, stress-diathesis models acknowledge individual differences in vulnerability to stress by claiming stressful life events and underlying individual vulnerabilities are required for the onset or recurrence of disorders (Slavik & Croake, 2006). According to Miller and Seligman's (1982) reformulated Learned Helplessness Model, experiences within an environment in which one has no control over outcomes may result in specific self-attributions that can lead to thoughts and feelings of helplessness and hopelessness and often to depression. According to hopelessness theory (Abramson et al., 1989), individuals who have a tendency to attribute negative events to stable and global causes, and infer both negative consequences and negative self-characteristics following the occurrence of those events, should be vulnerable to developing symptoms of depression in the presence, but not absence, of negative life events (Gibb, Beevers, Andover, & Holleran, 2006).

Social causation theories reason low-status social groups show higher rates of disorder because members of these groups disproportionately encounter difficult, harsh, or traumatic life conditions and also have restricted access to social, economic, or personal resources that could be used to combat these difficult life circumstances (Aneshensel, 1992). Because their resources are already taxed by the stresses of daily living, lower SES individuals are forced to confront unexpected negative stimuli with an already exhausted system, making these noxious stimuli more likely to persist or escalate, potentially causing negative emotional states (Gallo & Matthews, 1999). Ross (2000) argues the daily stress of living in a neighborhood where social

order has broken down is associated with depression, and that all of the impact on psychological well-being is mediated by perceptions of disorder in the neighborhood.

The present study proposes to examine the prevalence of specific symptoms of depression and anxiety in individuals with TBI due to violence compared to those with TBI due to other causes (e.g., MVA, falls). SES will be controlled for due to its relationship with risk for developing psychological disorders. The primary hypothesis is that a greater proportion of those in the violent TBI group will endorse symptoms of depression and anxiety, as measured by the factors yielded by factor analysis. Secondary analysis will look at the relative contribution of etiology and demographic factors such as gender, race, age, and injury severity to the prediction of symptoms of depression and anxiety. The secondary hypothesis is that while the demographic factors will attenuate the relationship between etiology and psychological symptomology to some extent, etiology will remain a significant predictor of level of depressive and anxious symptomology.

METHODS

Participants

Participants for this study were obtained through the South Carolina Traumatic Brain Injury Follow-up Registry (SCTBIFR, Selassie et al., 2006). The structure of the sample has been described in previous research (Horner et al., 2005; McCarthy et al., 2006). This earlier study was approved by the Institutional Review Boards of the Medical University of South Carolina and the Centers for Disease Control and Prevention (CDC). The present study was reviewed and exempted by the Institutional Review Board of Louisiana State University. A representative sample of persons with TBI was randomly selected from a South Carolina statewide hospital discharge data set and recruited to participate in a larger follow-up study. State law mandates that all non-federal hospitals report uniform, abstracted billing data to the South Carolina State Budget and Control Board; additional information on pre-existing health conditions and injury variables was acquired through medical record review. All persons, aged 15 years and older at the time of injury, who were hospitalized with a TBI between January 1, 1999, and June 30, 2002, were eligible for the larger study. TBI was defined as any discharge with a primary or secondary diagnosis of injury to the head associated with decreased consciousness, amnesia, neurological abnormalities, skull fracture, or intracranial lesion, in accordance with the CDC case definition of TBI (Thurman, Snieszek, Johnson, Greenspan, & Smith, 1995).

After excluding out-of-state residents and children younger than 15, a stratified random sample was selected based on injury severity. By design, the sample was constructed to include 67% moderate to severe TBI [defined as Abbreviated Injury Scale (AIS; Association for the Advancement of Automotive Medicine, 1990) head region scores of 3-6] and 33% mild TBI

(AIS head region score = 2). Further information on the AIS scoring system is presented in the Measures section.

7,613 individuals were eligible for the larger study. Of these, 4,519 were selected for the study using the stratified random sampling protocol. From this group, 713 (15.8%) were not eligible to participate because they were deceased, had moved out of state, did not speak English, were medically unable to participate and had no proxy, or were prisoners. Another 875 (19.4%) could not be located, and 813 (18.0%) chose not to participate, leaving 2,118 participants who completed interviews. The overall response rate was 55.7%. Comparison of participants with non-participants indicated that non-participants tended to be minorities, uninsured, and older than age 65.

Among the individuals in the participant sample, 183 (8 %) received their TBI as the result of an assault. A comparison sample (n = 366) of individuals with TBI due to other etiologies (e.g., motor vehicle accident, fall) was then selected from the remaining interview participants. In an effort to control for SES, members of the control group were selected to match the violence group as closely as possible based on income before their injuries. Nine individuals (3 from the violence group and six from the control group) were excluded from the study due to having more than one missing response to the relevant survey questions. This resulted in a total sample size of 540, with 180 individuals in the violence group and 360 individuals in the control group.

Procedures

Participants were recruited and interviewed by telephone approximately 1 year after hospital discharge. In addition to mood questions, the interview included standardized measures and other questions to assess general health, post-injury symptoms, employment, life

satisfaction, and demographic and other variables. The interview was approximately 45 minutes long. The data collected through medical record abstraction and interview were validated for internal consistency.

Measures

The dependent variables, symptoms of depression or anxiety, were assessed using items from the telephone survey that fit into DSM-IV-TR diagnostic criteria (APA, 2000) for either a Major Depressive Episode (MDE) or Generalized Anxiety Disorder (GAD). These criteria and the questions assessing them are shown in Appendix A for MDE and Appendix B for GAD. These questions had one of two response formats: (a) yes/no, or (b) Likert-type scale (5 points = All of the time, 4 points = Most of the time, 3 points = A good bit of the time, 2 points = Some of the time, 1 point = A little bit of the time, 0 points = None of the time). Reverse-scored items are noted on Appendices A and B by asterisks.

Distress or impairment in social, occupational, or other areas of functioning (MDE Criterion C, GAD Criterion E; APA, 2000) was assessed with selected questions from the Short Form-36 (SF-36; Ware and Sherbourne, 1992). These questions are listed in Appendix C. Each of these questions was yes/no in format. If a positive response was given to any of these questions, then criteria were considered to be met for distress/impairment secondary to depression and/or anxiety. Finally, whether or not respondents reported seeking treatment for a psychiatric disorder was assessed with the following questions: (T1) “Since your injury, have you been treated for a psychiatric disorder?” and (T2) “Since your injury, have you taken any medicine for a mood disorder?” Both questions were answered in a yes/no format and were non-standard questions developed for the epidemiological study. A response of yes to either question

was considered indicative of the individual receiving treatment for some form of psychiatric disorder.

Demographic characteristics such as gender, race, and age, as well as severity of injury, were collected for the purpose of analyzing their relative contribution along with etiology to the prediction of the dependent variables. Severity of injury was determined by translating ICD-9-CM codes into AIS scores using ICDMAP-90 software (Center for Injury Research Policy, 1997). The AIS is an anatomical scoring system that ranks injury severity for various body regions on a scale of 1 (minor) to 6 (unsurvivable). AIS scores have previously been used to relate TBI severity to longer-term outcome (e.g., Massagli, Michaud, & Rivara, 1996). For this study, only the AIS score for the head region was considered, with a score of 3 – 6 (typically corresponding to loss of consciousness exceeding 30 min) classified as moderate to severe TBI and a score of 2 classified as mild TBI (Horner et al., 2005). As noted in the preceding section, the sample was constructed by design to include 67% more severe TBI and 33% mild TBI.

While Glasgow Coma Scale (GCS; Teasdale and Jennett, 1976) scores might have been a preferable index of TBI severity, it was not available for 43.6% of the total study sample, precluding its use (Horner et al., 2005). Unfortunately, GCS and AIS are not directly comparable since they use different indicators to estimate injury severity; GCS using physiological functions (which can change over time) and AIS assessing anatomical damage (which is static). Further, GCS is usually measured before or on admission while AIS scores are based on discharge diagnosis. Thus, scores on the two measures are not expected to be strongly correlated (Association for the Advancement of Automotive Medicine, 1990). For the total epidemiological sample from which this study was derived, Horner and colleagues (2005) reported the intraclass correlation (weighted kappa) between GCS and AIS scores to be .27.

GCS scores were available for 312 (58%) of the individuals selected for this study. For these respondents, there was a significant negative correlation between AIS scores and GCS scores, Pearson $r = -.26, p < .001$.

Statistical Analyses

Based on individual responses to the phone interview, exploratory factor analysis was used to examine patterns in the relationship among the various questions used to assess depression and anxiety. The goal of this factor analysis was to combine these questions into a few factors (e.g., depression, anxiety), which could be measured as continuous dependent variables. Attempts were made to load each variable onto only one factor to prevent multicollinearity in subsequent analyses. Standardized canonical correlation coefficients were used to weight importance of each variable to its factor. Using exploratory factor analysis to develop fewer factors for analysis decreases the risk of a Type I error and increase the ability to understand the relationship between facets of psychopathology in TBI. This method also provides the most options for analyzing the resulting data.

Multiple regression was used to analyze the hypotheses that (1) a greater proportion of those in the violent TBI group will endorse criteria of depression and anxiety, and (2) etiology will significantly contribute to the prediction of psychological status in excess of the contribution of the demographic factors and severity of injury. To answer the first hypothesis, etiology was coded as a dummy variable in the regression analysis. Violent etiology was coded as 1 and nonviolent as 0. To avoid perfect multicollinearity in the analysis, nonviolent etiology was omitted as the reference category, allowing the beta coefficients, or semi-partial coefficients, to be interpreted with reference to it. The beta coefficient represents the difference in means between the two values of the dummy variable and, if significant, would indicate a significant

difference between the included group, violent TBI, and the reference group, nonviolent TBI, on the response variable, the factors reflecting depression and anxiety symptoms.

This analysis was also used to determine the relative contribution of each of the independent variables (etiology of injury, gender, race, age, injury severity) to prediction of the dependent variables (factors reflecting depression and anxiety symptoms). A separate analysis was performed for each outcome factor with adjustment of the alpha level for the number of analyses performed, and was done in two stages. In the first step, severity of injury and demographics were entered into the model, allowing for control of the influence of that information before looking at the contribution of etiology to the model. Etiology of injury was then entered, with a significant change in the F value and the magnitude of the change in R^2 being used to determine if etiology provides a unique contribution to the prediction of psychological outcomes following TBI or if it is redundant with the other factors.

Additional Analyses

Taking into consideration the impact of severity of injury on outcomes following TBI, Multivariate Analysis of Variance (MANOVA) was performed with the anxiety and depression factors yielded by the factor analysis as dependent variables and etiology and injury severity as independent variables. Results from the MANOVA were followed up with one-way ANOVAs on each dependent variable factor as an exploratory analysis to further scrutinize the findings. First, the total sample was split between those with mild and moderate to severe injuries to look for differences between the violence and control groups on the dependent variables. Next, the procedure was reversed, splitting the total sample between members of the violence and control groups to look for differences between the two injury severity groups on the dependent variables.

Self-reports of distress/impairment and treatment seeking by the participants were then examined, looking for differences in these variables among the study groups as well as within the study groups. This was done by looking at frequencies of responses to these items in the violent compared to nonviolent groups, and endorsement of these items by those reporting psychological symptoms within each group. Chi-square analysis was used to assess for significance of any differences in these frequencies. The purpose of this analysis was to examine the question of whether those who are reporting symptomology and resultant impairment in functioning are receiving any form of treatment for their symptoms.

A priori power analyses were performed using G*Power 3 (Faul, Erdfelder, Lang, & Buchner, 2007) and effect sizes recommended by Cohen (1998). For the multiple regression procedure, with alpha set at .017 and Power at .80, with five predictors the sample size needed to detect a small effect size (Cohen's $f^2 = .02$) was 821 and for a medium effect size (Cohen's $f^2 = .15$) was 116. Thus, the available sample size ($N = 540$) should be adequate to detect an effect somewhere in size between a small and medium effect. For the MANOVA procedure, with alpha set at .05, Power at .90, examining 2 IVs and 3 DVs, the sample size need for a small effect size (Cohen's $f^2 = .10$) was 146, meaning the available sample size is sufficient to detect small global effects. For the follow-up ANOVA analyses, setting alpha at .05 and Power at .80, the sample size needed to detect a small effect size (Cohen's $f = .10$) was 788 and for a medium effect size (Cohen's $f = .25$) was 128. Thus, the available sample sizes for the violence group ($n = 180$), control group ($n = 360$), mild injury group ($n = 161$), and moderate to severe injury group ($n = 379$) were all sufficient to detect a medium effect size, but not to detect a small effect size.

RESULTS

The mean age for participants in the study was 42.13 years ($SD = 21.54$). See Table 1 for a distribution of subjects by age groups. Additional demographic characteristics of the participants are provided in Table 2. Student t-tests and Pearson Chi-squares were used to compare the violence and control groups on the demographic variables. Individuals in the violence group (mean age = 36.47 yrs, $SD = 16.01$) were significantly younger than those in the control group (mean age = 44.96 yrs, $SD = 23.34$), $t(538) = -4.39, p < .001$. This same difference was observed for the age range distributions, $X^2(6) = 35.92, p < .001$. Overall, more males (64.4%) than females (35.6%) received TBI due to any cause. This difference was even more pronounced in the violence group, where 75.6% of subjects were male compared with 58.9% in the control group, $X^2(1) = 14.55, p < .001$. The total sample was predominantly Caucasian (75.4%), with African-Americans (22.8%) comprising the next largest ethnic group. There were a higher proportion of African-Americans in the violence group (36.7%) compared with the control group (15.8%), $X^2(4) = 34.79, p < .001$.

Table 1
Age range frequencies for subjects in total sample, violence and control groups

Age Group	Group		Total
	Control	Violence	
15-24	101(28.1%)	52 (28.9%)	153 (28.3%)
25-34	40 (11.1%)	37 (20.6%)	77 (14.3%)
35-44	61 (16.9%)	42 (23.3%)	103 (19.1%)
45-54	38 (10.6%)	27 (15.0%)	65 (12.0%)
55-64	27 (7.5%)	10 (5.6%)	37 (6.9%)
65-74	32 (8.9%)	6 (3.3%)	38 (7.0%)
75+	61 (16.9%)	6 (3.3%)	67 (12.4%)

As expected due to the matching procedure employed, the violence and control groups did not differ significantly in income before injury, $X^2(7) = 10.90, p = .143$. The most prominent type of insurance in both the violence and control groups was commercial insurance, 46.1% and

49.7%, respectively. However, there was a significant difference among those without commercial insurance, with a higher proportion of uninsured (22.8%) and Medicaid patients (25.6%) in the violence group and more Medicare patients (26.4%) in the control group, $X^2(3) = 49.50, p < .001$. There was no difference between the violence and control groups in the type of residence, urban versus rural, $X^2(1) = 1.55, p = .213$.

Table 2
Frequency of demographic variables in the total sample, violence and control groups

		Group		Total
		Control	Violence	
Gender	female	148 (41.1%)	44 (24.4%)	192 (35.6%)
	male	212 (58.9%)	136 (75.6%)	348 (64.4%)
Race	white	298 (82.8%)	109 (60.6%)	407 (75.4%)
	black	57 (15.8%)	66 (36.7%)	123 (22.8%)
	hispanic	5 (1.4%)	3 (1.7%)	8 (1.5%)
	oriental	0 (0.0%)	1 (0.6%)	1 (0.2%)
	other	0 (0.0%)	1 (0.6%)	1 (0.2%)
Income Before Injury	<10k	100 (29.8%)	48 (28.1%)	148 (29.2%)
	10k-15k	52 (15.5%)	28 (16.4%)	80 (15.8%)
	15k-20k	38 (11.3%)	24 (14.0%)	62 (12.2%)
	20k-25k	37 (11.0%)	27 (15.8%)	64 (12.6%)
	25k-35k	50 (14.9%)	21 (12.3%)	71 (14.0%)
	35k-50k	28 (8.3%)	13 (7.6%)	41 (8.1%)
	50k-75k	10 (3.0%)	8 (4.7%)	18 (3.6%)
	75k+	21 (6.3%)	2 (1.2%)	23 (4.5%)
Insurance Group	Uninsured	41 (11.4%)	41 (22.8%)	82 (15.2%)
	Medicaid	45 (12.5%)	46 (25.6%)	91 (16.9%)
	Medicare	95 (26.4%)	10 (5.6%)	105 (19.4%)
	Commercial	179 (49.7%)	83 (46.1%)	262 (48.5%)
Type Residence	Rural	212 (58.9%)	116 (64.4%)	328 (60.7%)
	Urban	148 (41.1%)	64 (35.6%)	212 (39.3%)

For the total sample, the mean AIS score was 3.21 ($SD = .91$), with 161 individuals (29.8%) falling in the mild injury group and 379 (70.2%) in the moderate to severe injury group. Comparing the etiology groups, there were significantly higher AIS scores in the violence group (mean = 3.33, $SD = .859$) compared with the control group (mean = 3.14, $SD = .924$), $t(538) = 2.29, p = .02$. Placing AIS scores into categories of mild and moderate to severe injuries

yielded a similar result, with a higher proportion of moderate to severe injuries in the violence group ($n = 138$, 76.7%) than the control group ($n = 241$, 66.9%), $X^2(1) = 5.42$, $p = .02$. These results should be interpreted in the context of the sampling technique used to recruit subjects for the original study, where 67% of subjects were pre-selected for moderate to severe injuries and 33% for mild injuries.

Factor Analysis Results

Maximum likelihood extraction with varimax rotation was performed through SPSS FACTOR on 20 items thought to measure symptoms of depression and anxiety for a sample of 540 individuals. Both a three-factor and four-factor solution were looked at; however, the three-factor solution provided a better theoretical fit to the DSM-IV criteria shown in Appendices A and B. The three factors extracted were: (1) Depression/Anxiety Symptoms, (2) Cognitive Symptoms, and (3) Fatigue/Loss of Energy. Loadings of variables on factors, communalities, and percents of variance and covariance are shown in Table 3. Variables are ordered and grouped by size of loading to facilitate interpretation. Loadings on factors under .45 (20% of variance) are not shown. The variables comprising each factor were summed to derive a total score for each factor, then the means and standard deviations of these summed scores were used to calculate standardized z-scores for each factor, with higher z-scores reflecting greater endorsement of symptoms. The descriptive values of the z-scores for these factors are given in Table 4.

Regression Results

Sequential regression was employed for two purposes; (1) to assess differences between the violence and control groups on the dependent variables, and (2) to determine if addition of injury severity, gender, race, and age, and then etiological group improved prediction of each of

the dependent variable factors. Alpha was set at .017 for each of the analyses, which were performed using SPSS REGRESSION.

Table 3
Factor loadings, communalities (h^2), and percents of variance for maximum likelihood extraction and varimax rotation on depression and anxiety items

Item	F ₁ ^a	F ₂	F ₃	h^2
Downhearted and blue	.76	---	---	.76
Down in the dumps	.75	---	---	.73
Happy person ^b	.65	---	---	.59
Calm and peaceful ^b	.56	---	---	.46
Very nervous person	.54	---	---	.52
Tense or keyed up	.49	---	---	.30
Irritable and grouchy	.49	---	---	.31
Trouble controlling your temper	.49	---	---	.30
Trouble getting along with others	.46	---	---	.28
Difficulty solving problems	---	.72	---	.63
Confused or disoriented	---	.70	---	.64
Difficulty concentrating and thinking	---	.70	---	.62
React slowly	---	.66	---	.50
Forget a lot	---	.61	---	.47
Do not keep attention	---	.55	---	.42
Confused/several actions	---	.54	---	.48
Tired	---	---	.79	.72
Worn out	---	---	.73	.64
Have energy ^b	---	---	.54	.49
Full of pep ^b	---	---	.46	.31
Percent of variance	40.90	5.72	4.22	
^a Factor labels:				
F ₁ Depression/Anxiety Symptoms				
F ₂ Cognitive Symptoms				
F ₃ Fatigue/Loss of Energy				
^b Reverse-scored item				

Table 4
Descriptive values of z-scores for dependent variables yielded by the factor analysis; higher z-scores indicate greater endorsement of symptoms

Factor	Minimum	Maximum	Mean	Std. Deviation
Depression/Anxiety Symptoms	-1.37	2.56	0	1
Cognitive Symptoms	-1.07	1.62	0	1
Fatigue/Loss of Energy	-2.14	1.91	0	1

The Depression/Anxiety Symptoms factor was examined first. There was not a significant difference in endorsement of this factor between individuals in the violence and control groups, $t(521) = 1.40, p = .16$. Table 5 displays the correlations between the variables, the unstandardized regression coefficients (B) and intercept, the standardized regression coefficients (β), the semipartial correlations (sr^2), and R, R^2 , and adjusted R^2 after entry of all the IVs. R was not significantly different than zero at the end of either step. After step 2, with all the IVs in the equation, $R = .13, F(5, 517) = 1.83, p = .11$. After step 1, with gender, race, age, and AIS group in the equation, $R^2 = .014, F_{inc}(4, 518) = 1.79, p = .13$. After step 2, with etiology added to the prediction of the Depression/Anxiety Symptoms factor by gender, race, age, and AIS group, $R^2 = .017, F_{inc}(1, 517) = 1.96, p = .16$. Addition of etiology to the equation did not reliably improve R^2 .

Table 5
Sequential regression of demographics, injury severity, and etiological group on Anxiety/Depression Symptoms factor

Variables	Dep./Anx. (DV)	Gender	Race	Age	AIS	Etiology	B	β	sr^2 (incremental)
Gender	-.06						-1.391	-.09	.014
Race	-.03	-.07					-.214	-.01	
Age	-.07	-.17	.09				-.026	-.08	
AIS	.03	.08	-.07	.04			.602	.04	
Etiology	.07	.16	-.24	-.19	.10		1.014	.06	.004
Intercept = 10.901									
Means	10.10	.65	.75	42.01	1.70	.33			
SD	7.39	.48	.43	21.52	.46	.47			$R^2 = .017$
									Adjusted $R^2 = .008$
									$R = .132$

The Cognitive Symptoms factor was examined next. Members of the violence group did not differ from the control group on this factor, $t(516) = .35, p = .73$. Table 6 displays the correlations between the variables, the unstandardized regression coefficients (B) and intercept, the standardized regression coefficients (β), the semipartial correlations (sr^2), and R, R^2 , and adjusted R^2 after entry of all the IVs. R was significantly different from zero ($p < .017$) at the end of the first step, but not after the second step. After step 2, with all IVs in the equation, $R = .162, F(5, 512) = 2.76, p = .018$. After step 1, with gender, race, age, and AIS group in the equation, $R^2 = .026, F_{inc}(4, 513) = 3.43, p < .01$. After step 2, with etiology added to the prediction of the Cognitive Symptoms factor by gender, race, age, and AIS group, $R^2 = .026, F_{inc}(1, 512) = .123, p = .73$. Addition of etiology to the equation did not reliably improve R^2 .

Table 6
Sequential regression of demographics, injury severity, and etiological group on Cognitive Symptoms factor

Variables	Cognitive (DV)	Gender	Race	Age	AIS	Etiology	B	β	sr^2 (incremental)
Gender	-.04						-.184	-.03	.026*
Race	-.06	-.07					-.360	-.06	
Age	.12	-.18	.09				.015*	.12	
AIS	.09	.09	-.08	.04			.443	.08	
Etiology	.01	.18	-.25	-.18	.11		.089	.02	.000
Intercept = 1.774									
Means	2.78	.64	.75	41.57	1.70	.34			
SD	2.60	.48	.43	21.23	.46	.47			$R^2 = .026$
									Adjusted $R^2 = .017$
									$R = .162$

* $p < .01$

Finally the Fatigue/Loss of Energy factor was examined. Again, there was no difference between the violence on control groups on this factor, $t(531) = .58, p = .56$. Table 7 displays the correlations between the variables, the unstandardized regression coefficients (B) and intercept, the standardized regression coefficients (β), the semipartial correlations (sr^2), and R, R^2 , and adjusted R^2 after entry of all the IVs. R was significantly different from zero ($p < .017$) at the end of each step. After step 2, with all IVs in the equation, $R = .235, F(5, 527) = 6.17, p < .001$. After step 1, with gender, race, age, and AIS group in the equation, $R^2 = .055, F_{inc}(4, 528) = 7.63, p < .001$. After step 2, with etiology added to the prediction of the Fatigue/Loss of Energy factor by gender, race, age, and AIS group, $R^2 = .055, F_{inc}(1, 527) = .334, p = .56$. Addition of etiology to the equation did not reliably improve R^2 .

Table 7
Sequential regression of demographics, injury severity, and etiological group on Fatigue/Loss of Energy factor

Variables	Fatigue (DV)	Gender	Race	Age	AIS	Etiology	B	β	sr^2 (incremental)
Gender	-.11						-.856	-.08	.055*
Race	.09	-.06					.829	.07	
Age	.21	-.17	.09				.044*	.19	
AIS	.03	.07	-.07	.05			.299	.03	
Etiology	-.04	.16	-.23	-.18	.10		.271	.03	.001
Intercept = 8.049									
Means	10.57	.64	.76	42.30	1.70	.33			
SD	4.94	.48	.43	21.60	.46	.47			
									$R^2 = .055$
									Adjusted $R^2 = .046$
									$R = .235$

* $p < .01$

MANOVA Results

A two-by-two between-subjects multivariate analysis of variance was performed on three dependent variables: Depression/Anxiety Symptoms, Cognitive Symptoms, and Fatigue/Loss of Energy. Independent variables were etiology group (violence and controls) and injury severity group (mild and moderate to severe). SPSS MANOVA was used for the analysis with the sequential adjustment for nonorthogonality. Order of entry of IVs was etiology, then injury severity.

With the use of Wilk's criterion, the combined DVs were significantly affected by etiology, $F(3, 490) = 3.03, p < .05$, but not by injury severity, $F(3, 490) = 2.05, p > .05$, or their interaction, $F(3, 490) = 1.28, p > .05$. While the main effect for etiology was significant, the association was not very substantial, partial $\eta^2 = .02$. To investigate the impact of this effect for etiological group on the individual DVs, the univariate F s were examined; however, none of these contributions were significant.

Etiology Group Results by Injury Severity

The total sample was divided based on injury severity groups and separate one-way Analysis of Variance (ANOVA) were used to examine differences in z-scores on each of the dependent variables for the etiological groups. Scores are shown in Figures 1, 2, and 3. For those with mild injuries, there were no significant differences between the violence and control groups on any of the dependent variables, all $p > .05$. For those with moderate to severe injuries, z-scores for the Depression/Anxiety Symptoms factor were significantly higher for the violence group (mean = .17, $SD = 1.02$) than the control group (mean = -.06, $SD = .98$), $F(1, 364) = 4.36, p = .04$. However, there were no significant differences between the violence and control groups for the Cognitive Symptoms and Fatigue/Loss of Energy factors, all $p > .05$.

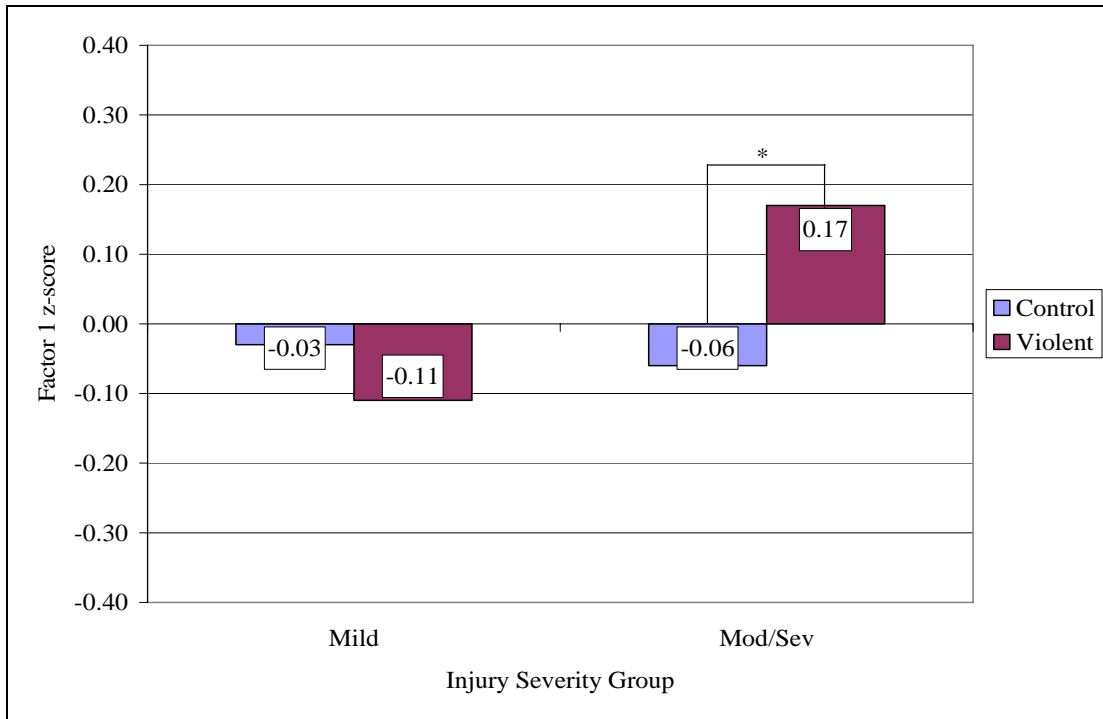


Figure 1. Z-scores on the Depression/Anxiety Symptoms factor arranged by etiological group and injury severity group. Higher scores indicate more reported symptoms.

* Significant at $p < .05$

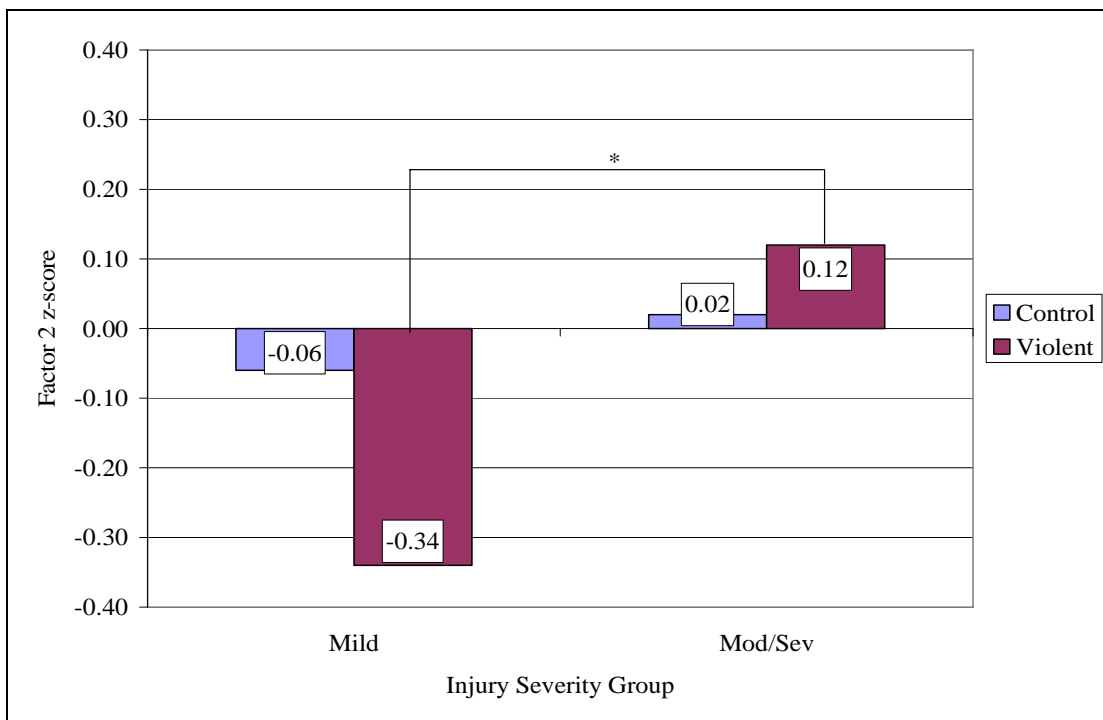


Figure 2. Z-scores on the Cognitive Symptoms factor arranged by etiological group and injury severity group. Higher scores indicate more reported symptoms.

* Significant at $p < .05$

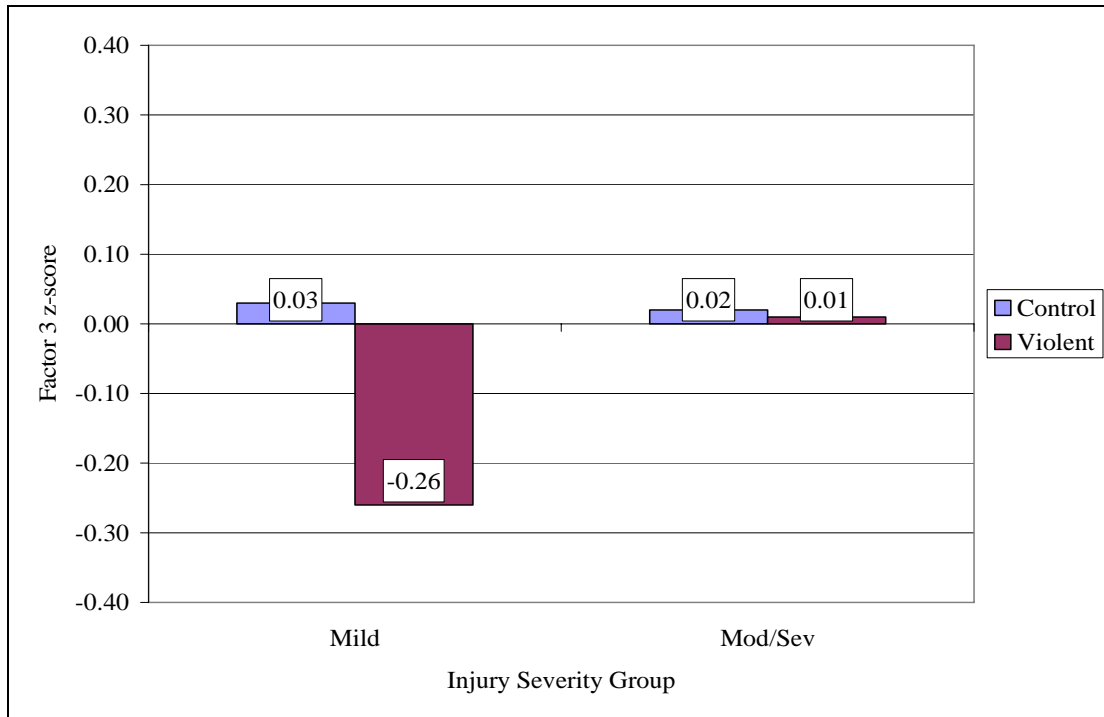


Figure 3. Z-scores on the Etiology/Loss of Energy factor arranged by etiological group and injury severity group. Higher scores indicate more reported symptoms.

Injury Severity Group Results by Etiological Group

The total sample was also divided based on etiological groups and one-way ANOVAs were used to examine differences in z-scores on the dependent variables for the injury severity groups. Scores are shown in Figures 1, 2, and 3. Within the control group, there were no significant differences on any of the dependent variables between those with mild and moderate to severe injuries, all $p > .05$. Within the violence group, there were significantly higher z-scores on the Cognitive Symptoms factor for the moderate to severe injury group (mean = .12, $SD = 1.01$) than for the mild injury group (mean = -.34, $SD = .93$), $F(1, 173) = 6.81, p = .01$. However, there were not significant differences between the two injury severity groups on the Depression/Anxiety Symptoms and Fatigue/Loss of Energy factors, all $p > .05$.

Effect of Race

One-way Analysis of Variance was used to examine differences in z-scores on each of the dependent variables among racial groups. Minority groups were combined for the purpose of this analysis and compared with Caucasians. For the total sample, there was no difference on the Depression/Anxiety Symptoms factor between the Caucasian (mean = $-.02$, $SD = 1.02$) and non-Caucasian (mean = $.05$, $SD = .95$) groups, $F(1, 521) = .50$, $p = .48$. There was also no difference on the Cognitive Symptoms factor between the Caucasian (mean = $-.03$, $SD = 1.00$) and non-Caucasian (mean = $.10$, $SD = 1.00$) groups, $F(1, 516) = 1.67$, $p = .20$. However, there was a significant difference on the Fatigue/Loss of Energy factor, $F(1, 531) = 3.97$, $p = .047$, with higher z-scores in the Caucasian group (mean = $.05$, $SD = 1.02$) than the non-Caucasian group (mean = $-.15$, $SD = .93$). When the total sample was split according to etiology, there were no significant differences on any of the dependent variables between Caucasians and non-Caucasians within the violence and control groups, all $p > .05$.

Impairment and Treatment Findings

Examining the total sample, 230 individuals (43.5%) reported functional impairment in at least one area. Of those with impairment, almost half ($n = 108$, 48.4%) received some form of treatment for a psychiatric disorder. In contrast, only 16.5% of those who did not endorse functional impairment reported receiving treatment. The rates of functional impairment and reception of treatment in each of the etiology groups was also examined. A greater proportion of those in the violence group (51.5%) compared with the control group (39.3%) reported functional impairment, $X^2(1) = 7.44$, $p < .01$. Looking at all respondents, 27.8% of those in the violence group received some form of treatment and 31.6% of those in the control group received treatment. There was not a significant difference between these groups in the

percentage that received treatment, $X^2(1) = .80, p = .37$. An examination of only those subjects reporting impairment evidenced no difference in the percentage of individuals in the violence (42.4%) and control groups (52.6%) who reported receiving treatment, $X^2(1) = 2.33, p = .13$.

DISCUSSION

Demographics for the total sample were similar to those previously reported for TBI in general (Langlois et al., 2006; Sosin et al., 1996), with young males comprising most of the sample. The overall sample was predominantly Caucasian (75.4%), which contradicts some past research showing nonwhites to be at higher risk for TBI (Bruns & Hauser, 2003). However, this finding is consistent with Bushnik and colleagues (2003) report of individuals injured secondary to MVA or fall being primarily white. Similar to past studies on TBI due to violence, the violence group had a demographic pattern of predominantly younger males (Bogner et al., 2001; Hanks et al., 2003; Harrison-Felix et al., 1998; Schopp et al., 2006; Sosin et al., 1996; Wagner, Sasser, et al., 2000). However, racial demographics were somewhat inconsistent with these past studies, which found African-Americans to be at predominant risk for TBI from violence. In the present study, there were mostly Caucasians (60.6%) in the violence group, though there were proportionally more African-Americans in the violence group (36.7%) compared with the control group (15.8%). Compared with the overall rate of African-Americans residing in the state of South Carolina (29.2%; US Census Bureau, 2007), the violence group was overrepresented by members of this demographic while the control group was underrepresented.

While income didn't differ between the violence and control groups due to the matching procedure used in the sampling, there were still some signs of lower socioeconomic status in the violence group. Those in the violence group were more likely to be uninsured or have Medicaid. This could place the violence group at greater risk for problems following their injury as their access to medical and psychiatric care may not be equivalent to the access available to the control group, which is more likely to be insured via commercial insurance or Medicare.

The percentage of the total epidemiological sample with TBI due to violence (8%) was slightly lower than in previous studies on TBI due to violence coming out of the TBI Model Systems (e.g., 29%, Harrison-Felix et al., 1998; 26%, Hanks et al., 2003). However, the percentage found in the present study is close to recent CDC reports of an 11% prevalence for TBI due to violence (Langlois et al., 2006). Several factors have been proposed which could potentially explain these discrepancies. In examining the nonrandom loss of individuals to follow-up, Corrigan et al. (2003) found that these individuals tend to be young, single men, injured in a violent act. Machamer and colleagues (2003) also found a significantly higher dropout rate for individuals with TBI due to violence, but showed that demographic factors such as age, race, and gender played a larger role in predicting who would drop out than circumstances of injury.

Regarding injury severity, the ability to interpret group differences is limited due to the sampling methodology used. The original epidemiological study from which this data was obtained was designed to contain two-thirds moderate to severe injuries and one-third mild injuries, which would restrict the variance in injury severity. However, taking this factor into account, a significant difference was observed in injury severity between groups, with a greater proportion of moderate to severe injuries in the violence group (76.7%) compared with the control group (66.9%). From this data, a conservative conclusion could be made that in this sample the violence group had more severe injuries than the control group. However, caution should be used in extending this finding to the general population of individuals with TBI. Still, if this difference is in fact real, then it could reflect a greater risk for poorer cognitive and psychological outcomes in individuals with TBI due to violence.

The results of the factor analysis were in line with the theoretical model, laid out in Appendices A and B, of how the survey questions fit DSM-IV criteria for depression and anxiety. The first factor (Depression/Anxiety Symptoms) was comprised of a mixture of items thought to assess general symptoms of depression and anxiety, with the items believed to reflect depressed mood (MDE Criterion A-1) contributing most to the variance on this factor, followed by the items believed to measure anxiety/restlessness (GAD Criterion A and C-1) and finally those thought to examine irritability (GAD Criterion C-4). The second factor (Cognitive Symptoms) was comprised entirely of items reflective of difficulties with thinking and concentration (MDE Criterion A-8, GAD Criterion C-3) and psychomotor retardation (MDE Criterion A-5). The third factor (Fatigue/Loss of Energy) reflected all the items originally theorized to reflect symptoms of fatigue or loss of energy (MDE Criterion A-6, GAD Criterion C-2).

The overlap between depressive and anxious symptoms in these factors is not surprising given the similar diagnostic criteria between many symptoms of depression and anxiety and the frequent comorbidity of these disorders. However, even with the good theoretical fit of these factors with DSM-IV criteria, only about 50% of the variance in the original survey questions was accounted for by the combination of the three factors. One possible reason for this finding could be overlap between the questions, with many of them asking basically the same question in a different manner. Also, only select DSM-IV criteria for MDE and GAD were assessed due to limits in the data available for study. This could be improved in future studies by expanding the questionnaire to include items assessing a broader, more inclusive range of MDE and GAD criteria. Alternatively, a measure with already established validity for assessing these criteria could be used.

The results of the regression analysis were not as expected. The violence group and the control group did not differ significantly on any of the dependent variables. Also, etiological group membership had no significant value in prediction of the dependent variables. However, the other independent variables were not very powerful predictors either. The combination of demographics and injury severity accounted for a small, but significant, portion of variance in Cognitive Symptoms (2.6%) and Fatigue/Loss of Energy (5.5%), but failed to contribute to prediction of the Depression/Anxiety Symptoms factor. For the Cognitive Symptoms and Fatigue/Loss of Energy factors, age appears to be the primary contributing variable, as it was the only variable with a significant beta coefficient (see Tables 9 and 10). A potential explanation for the low effect sizes observed is the controlling of SES prior to analysis. As mentioned previously, SES has been shown to influence rates of depression and anxiety in the general population and would likely account for much of the unexplained variance in the dependent variables, though other unknown factors could also play a role.

While these results are initially discouraging, further examination of the data revealed some differences between the etiological groups once severity of injury was controlled for. Multivariate analysis of variance revealed a significant main effect for group, though the resulting effect size was small. To further explore this finding, the sample was divided according to injury severity. Looking at only those individuals with mild injuries, no difference in symptomology between the violence and control groups was detected. However, among those with moderate to severe injuries, a significantly higher number of depressive and anxious symptoms were detected in the violence group (see Figure 1). Taking into consideration the greater proportion of African-Americans in the violence group, potential racial differences in reported symptoms were examined and no differences were found for report of depressive and

anxious symptoms between Caucasians and non-Caucasians. Thus, it can be conservatively concluded based on this data that those with TBI secondary to violence are not only at greater risk for more severe injuries, but when those injuries are more severe, they are at greater risk for depression and anxiety compared with individuals injured by other means.

Another difference between individuals in the violence and control groups was observed in the area of reported cognitive symptoms. Dividing the sample by etiological groups, those in the violence group with moderate to severe injuries reported more cognitive problems than those with mild injuries (see Figure 2), which could be expected based on the more severe nature of their injuries. However, this difference was not present in the control group. Thus, individuals with injuries caused by means other than violence had a similar number of cognitive complaints whether their injuries were mild or moderate to severe. This is not meant to imply that there was a difference in the amount of actual cognitive impairment experienced by those with mild and moderate to severe injuries in the violence group but not in the control group, but just that there was a difference in the tendency to make cognitive complaints between these groups. The influence of depression and anxiety on reporting of cognitive complaints could be responsible for this difference.

Support for the view that psychological symptoms can influence cognitive complaints comes from research on symptoms of postconcussion syndrome (PCS), which refers to the cluster of symptoms often observed following mild TBI. These symptoms, as summarized by Busch and Alpern (1998), can be physical (headache, dizziness, fatigue, decreases libido, insomnia and sleep disturbance, hypersensitivity to noise, photophobia), cognitive (attention, concentration, information processing, memory, problems solving, and thinking deficits), and psychological (irritability, anxiety, hypochondriacal concern, lack of confidence, depression,

mood swings). As can be seen from looking at this list, many of these symptoms overlap with those for depression and anxiety, as well as posttraumatic stress disorder (PTSD). This can make it extremely difficult to determine if a person's self-reported symptoms following a TBI are due to depression, a persistent PCS, or both (Iverson, 2006).

Research on patients with TBI has shown that depression correlates with both subjective complaints and objective test findings of cognitive difficulties, and as such is a major risk factor for cognitive and functional outcome in these individuals (McCauley, Boake, Levin, Contant, & Song, 2001). Symptoms of depression as well as other forms of psychopathology can have a large influence on severity and duration of PCS. Patients with mild TBI and depression have been found to report more PCS symptoms (Busch & Alpern, 1998) and those with PCS have been found to report more depression, anxiety, and stress (Meares et al., 2006). This may result in a vicious cycle which perpetuates both the PCS and psychological symptoms. Chronic PCS symptoms are more likely to persist because of psychological factors (Mittenberg & Strauman, 2000). In fact, Iverson (2005) argues a person who has depression, chronic pain, PTSD, or a combination of problems is virtually guaranteed to endorse many 'postconcussion-like' symptoms so it is very easy to misdiagnose these individuals as having a persistent PCS.

Reports of symptoms consistent with PCS have also been found in individuals without any history of TBI. Gunstad and Suhr (2004) found elevated rates of current PCS symptoms in non-neurological persons who were depressed or suffering headaches requiring treatment. Other groups that may report symptoms consistent with PCS include individuals seeking outpatient therapy (Fox, Lees-Haley, Earnest, & Dolezal-Wood, 1995), outpatients with minor medical problems (Lees-Haley & Brown, 1993), patients with PTSD (Foa, Cashman, Jaycox, & Perry, 1997), and personal injury claimants (Lees-Haley, Fox, & Courtney, 2001), among others

(Iverson, 2006). Demographic variables such as gender have also been shown to influence PCS symptom report (Santa Maria, Pinkston, Miller, & Gouvier, 2001).

Looking at the respondents' reports of functional impairment secondary to mood or anxiety symptoms, a greater percentage of individuals in the violence group reported functional impairment in at least one area. However, they are not receiving clinical treatment at a different level than those in the control group. Thus, while those with TBI due to violence are experiencing more functional impairment from mood and anxiety symptoms, they are not receiving clinical services at a similarly elevated rate.

Based on these findings, individuals who obtain a TBI secondary to violence appear to be at greater risk for poor psychological outcomes in the form of increased depressive and anxious symptomology. These same individuals may also be less likely to receive treatment for their symptoms. Special care should be taken to monitor those with TBI due to violence for these symptoms and to provide early treatment when appropriate. Measures could also be taken during rehabilitation to educate individuals with TBI, especially those with TBI due to violence, on the risk of psychiatric symptomology following their injury and how they can pursue treatment if they experience such problems.

There are several limitations to the present study. First, with the original study being epidemiological in nature, it was not designed to specifically examine psychological functioning. As a result, no standardized measures for assessing depression and anxiety were used in the study. For the purpose of analyzing the hypotheses in this study, novel dependent variables were designed using the available data source. Consequently, these variables have not been validated. Second, the sampling procedure used to pre-select subjects based on injury severity (i.e., two-thirds moderate to severe) may have resulted in an atypical distribution of injury

severity, possibly excluding many potential subjects, particularly those with mild TBI. However, this may have also contributed to the present findings of more depressive and anxious symptoms in those with moderate to severe injuries secondary to violence, a difference that would have been missed if all subjects were analyzed together regardless of injury severity. Third, the potential influence of comorbid PTSD symptoms was not examined in this study due to limitations in the data available from the epidemiological survey. Differences in the prevalence of PTSD between the violence and control groups could impact the rate of symptoms of depression and generalized anxiety due to the high comorbidity among these disorders. Finally, as the sample used in this study was comprised solely of individuals hospitalized in South Carolina, these results may not be representative of the United States population as a whole.

Future research should seek to further examine this topic using standardized, well-validated measures for assessing depression and anxiety. While the present findings were not very significant, some evidence of a relationship between etiology and symptoms of depression and anxiety was found, suggesting that TBI due to violence may put individuals at greater risk for the development of psychological sequelae, particularly in those who sustain moderate to severe injuries. As a result, care should be taken to consider injury severity when examining differences in psychopathology among those with TBI due to violence and other causes.

Also remaining to be explored is the topic of PTSD following TBI due to violence. The idea of PTSD following TBI is a controversial one, with some researchers arguing against the presence of PTSD in those with brain injury and amnesia for the traumatic event (e.g., Sbordone, 1999; Warden et al., 1997) and others arguing that PTSD symptoms are common following brain injury, despite loss of memory for all or part of the traumatic event (e.g., Bryant, 2001; Harvey,

Brewin, Jones, & Kopelman, 2003). Still others acknowledge that PTSD can occur following TBI, but that it is often misdiagnosed (e.g., Sumpter & McMillan, 2006).

Klein, Caspi, and Gil (2003) conclude that the overall balance of the findings seems to support the possibility that, in individuals with TBI, impaired memory of the traumatic event is associated with reduced prevalence of PTSD. Thus, amnesia for the event plays a protective role in reducing both the risk of developing PTSD and in the severity of symptoms if PTSD does develop, particularly symptoms involving intrusive memories. Other findings have suggested that it is not the severity of the injury but the nature of the injury that may be a major factor for reporting of PTSD symptoms in TBI (Williams, Evans, Needham, & Wilson, 2002). Based on that interpretation, it stands to reason that individuals who sustain a TBI secondary to violence may be at greater risk for developing PTSD symptoms than individuals injured through other means.

REFERENCES

- Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review*, *96*, 358-372.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.
- Aneshensel, C. S. (1992). Social stress: Theory and research. *Annual Review of Sociology*, *18*, 15-38.
- Association for the Advancement of Automotive Medicine. (1990). *The Abbreviated Injury Scale, 1990 revision*. Des Plaines, IL: Author.
- Benzel, E. C., Day, W. T., Kesterson, L., Willis, B. K., Kessler, C. W., Modling, D., & Hadden, T. A. (1991). Civilian craniocerebral gunshot wounds. *Neurosurgery*, *29*, 67-71.
- Bergner, M., Bobbit, R. A., Carter, W. B., & Gilson, B. S. (1981). The Sickness Impact Profile: Development and final revision of a health status measure. *Medical Care*, *19*, 787-805.
- Black, K. I., Hanks, R. A., Wood, D. L., Zafonte, R. D., Cullen, N., Cifu, D. X., Englander, J., & Francisco, G. E. (2002). Blunt versus penetrating violent traumatic brain injury: Frequency and factors associated with secondary conditions and complications. *Journal of Head Trauma Rehabilitation*, *17*, 489-496.
- Bogner, J. A., Corrigan, J. D., Mysiw, W. J., Clinchot, & Fugate, D. (2001). A comparison of substance abuse and violence in the prediction of long-term rehabilitation outcomes after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, *82*, 571-577.
- Bruce, M. L., Takeuchi, D. T., & Leaf, P. J. (1991). Poverty and psychiatric status. *Archives of General Psychiatry*, *48*, 470-474.
- Bruns, J., Jr., & Houser, W. A. (2003). The epidemiology of traumatic brain injury: A review. *Epilepsia*, *44*(Suppl. 10), 2-10.
- Bryant, R. A. (2001). Posttraumatic stress disorder and traumatic brain injury: Can they co-exist? *Clinical Psychology Review*, *21*, 931-948.
- Busch, C. R., & Alpern, H. P. (1998). Depression after mild traumatic brain injury: A review of current research. *Neuropsychology Review*, *8*, 95-108.
- Bushnik, T., Hanks, R. A., Kreutzer, J., & Rosenthal, M. (2003). Etiology of traumatic brain injury: Characterization of differential outcomes up to 1 year postinjury. *Archives of Physical Medicine and Rehabilitation*, *84*, 255-262.

- Center for Injury Research Policy of the Johns Hopkins University School of Public Health. (1997). ICMAP-90 Software. Baltimore, MD: The Johns Hopkins University and Tri-Analytics, Inc.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences (2nd ed.)*. Hillsdale, NJ: Erlbaum.
- Cooper, K., Tabaddor, K., Hauser, W. A., et al. (1983). The epidemiology of head injury in the Bronx. *Neuroepidemiology*, 2, 70-88.
- Corrigan, J. D., Harrison-Felix, C., Bogner, J., Dijkers, M., Terrill, M. S., & Whiteneck, G. (2003). Systematic bias in traumatic brain injury outcome studies because of loss to follow-up. *Archives of physical Medicine and Rehabilitation*, 84, 153-160.
- Derogatis, L. R., Lipman, R. S., Rickels, K., Uhlenhuth, E. H., & Covi, L. (1974). The Hopkins Symptom Checklist (HSCL): A self-report symptom inventory. *Behavioral Science*, 19, 1-15.
- Derogatis, L. R., & Melisaratos, N. (1983). The Brief Symptom Inventory: An introductory report. *Psychological Medicine*, 13, 595-605.
- Dohrenwend, B. P., & Dohrenwend, B. S. (1969). *Social status and psychological disorder: A causal inquiry*. New York: Wiley-Intersci.
- Dohrenwend, B. S. (1973). Social status and stressful life events. *Journal of Personality and Social Psychology*, 28, 225-235.
- Fann, J. R., Leonetti, A., Jaffe, K., Katon, W. J., Cummings, P., & Thompson, R. S. (2002). Psychiatric illness and subsequent traumatic brain injury: A case control study. *Journal of Neurology, Neurosurgery & Psychiatry*, 72, 615-620.
- Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, 39, 175-191.
- Foa, E. B., Cashman, L., Jaycox, L., & Perry, K. (1997). The validation of a self-report measure of posttraumatic stress disorder: The Posttraumatic Diagnostic Scale. *Psychological Assessment*, 9, 445-451.
- Fox, D., Lees-Haley, P., Earnest, K., & Dolezal-Wood, S. (1995). Postconcussive symptoms: Base rates and etiology in psychiatric patients. *The Clinical Neuropsychologist*, 9, 89-92.
- Gallo, L. C., & Matthews, K. A. (1999). Do negative emotions mediate the association between socioeconomic status and health? In N. E. Adler, M. Marmot, B. S. McEwen, & J. Stewart (Eds.), *Socioeconomic status and health in industrial nations: Social*,

- psychological, and biological pathways* (pp. 226-245). New York: New York Academy of Sciences.
- Gibb, B. E., Beevers, C. G., Andover, M. S., & Holleran, K. (2006). The hopelessness theory of depression: A prospective multi-wave test of the vulnerability-stress hypothesis. *Cognitive Therapy and Research, 30*, 763-772.
- Gunstad, J., & Suhr, J. A. (2004). Cognitive factors in postconcussion syndrome symptom report. *Archives of Clinical Neuropsychology, 19*, 391-405.
- Hanks, R. A., Wood, D. L., Millis, S., Harrison-Felix, C., Pierce, C. A., Rosenthal, M., Bushnik, T., High, W. M., Jr., & Kreutzer, J. (2003). Violent traumatic brain injury: Occurrence, patient characteristics, and risk factors from the traumatic brain injury model systems project. *Archives of Physical Medicine and Rehabilitation, 84*, 249-254.
- Hanlon, R. E., Demery, J. A., Martinovich, Z., & Kelly, J. P. (1999). Effects of acute injury characteristics on neuropsychological status and vocational outcome following mild traumatic brain injury. *Brain Injury, 13*, 873-887.
- Harrison-Felix, C., Zafonte, R., Mann, N., Dijkers, M., Englander, J., & Kreutzer, J. (1998). *Archives of Physical Medicine and Rehabilitation, 79*, 730-737.
- Hart, T., Bogner, J. A., Whyte, J., & Polansky, M. (2003). Attribution of blame in accidental and violence-related traumatic brain injury. *Rehabilitation Psychology, 48*, 86-92.
- Harvey, A. G., Brewin, C. R., Jones, C., & Kopelman, M. D. (2003). Coexistence of posttraumatic stress disorder and traumatic brain injury: Toward a resolution of the paradox. *Journal of the International Neuropsychological Society, 9*, 663-676.
- Holzer, C. E., Shea, B. M., Swanson, J. W., Leaf, P. J., Myers, J. K., George, L., Weissman, M. M., & Bednarski, P. (1986). The increased risk for specific psychiatric disorders among persons of low socioeconomic status: Evidence from the Epidemiological Catchment Area Surveys. *The American Journal of Social Psychiatry, 6*, 259-271.
- Horner, M. D., Ferguson, P. L., Selassie, A. W., Labbate, L. A., Kniele, K., & Corrigan, J. D. (2005). Patterns of alcohol use 1 year after traumatic brain injury: A population-based, epidemiological study. *Journal of the International Neuropsychological Society, 11*, 322-330.
- Ip, R., Hesch, P., Brandys, C., Dornan, J., & Schentag, C. (2000). Traumatic brain injury: Causes, severity, and outcomes. *Brain and Cognition, 44*, 42-44.
- Iverson, G. L. (2005). Outcome from mild traumatic brain injury. *Current Opinion in Psychiatry, 18*, 301-317.

- Iverson, G. L. (2006). Misdiagnosis of the persistent postconcussion syndrome in patients with depression. *Archives of Clinical Neuropsychology*, *21*, 303-310.
- Jorge, R., & Robinson, R. G. (2003). Mood disorder following traumatic brain injury. *International Review of Psychiatry*, *15*, 317-327.
- Jorge, R. E., Robinson, R. G., Starkstein, S. E., & Arndt, S. V. (1993). Depression and anxiety following traumatic brain injury. *Journal of Neuropsychiatric & Clinical Neuroscience*, *5*, 369-374.
- Jorge, R. E., Robinson, R. G., Starkstein, S. E., & Arndt, S. V. (1994). Influence of major depression on one-year outcome in patients with traumatic brain injury. *Journal of Neurosurgery*, *81*, 726-733.
- Kaufman, H. (1993). Civilian gunshot wounds to the head. *Neurosurgery*, *32*, 962-964.
- Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., Wittchen, H. U., & Kendler, K. S. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States: Results from the National Comorbidity Survey. *Archives of General Psychiatry*, *51*, 8-19.
- Klein, E., Caspi, Y., & Gil, S. (2003). The relation between memory of the traumatic event and PTSD: Evidence from studies of traumatic brain injury. *Canadian Journal of Psychiatry*, *48*, 28-33.
- Kraus, J. F. (1993). Epidemiology of head injury. In P. R. Cooper (Ed.), *Head injury* (3rd ed., pp. 1-25). Baltimore, MD: Williams & Wilkins.
- Langlois, J. A., Rutland-Brown, W., & Thomas, K. E. (2006). *Traumatic brain injury in the United States: Emergency department visits, hospitalizations, and deaths*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.
- Lees-Haley, P. R., & Brown, R. S. (1993). Neuropsychological compliant base rates of 170 personal injury claimants. *Archives of Clinical Neuropsychology*, *8*, 203-209.
- Lees-Haley, P. R., Fox, D. D., & Courtney, J. C. (2001). A comparison of complaints by mild brain injury claimants and other claimants describing subjective experiences immediately following their injury. *Archives of Clinical Neuropsychology*, *16*, 689-695.
- Levin, H. S., Goldstein, F. C., & Mackenzie, E. J. (1997). Depression as a secondary condition following mild and moderate traumatic brain injury. *Seminars in Clinical Neuropsychiatry*, *2*, 207-215.
- Lezak, M. D. (1995). *Neuropsychological assessment* (3rd ed.). New York: Oxford.

- Lucas, J. A. (1998). Traumatic brain injury and postconcussive syndrome. In P. J. Snyder & P. D. Nussbaum (Eds.), *Clinical neuropsychology: A pocket handbook for assessment* (pp. 243-265). Washington, DC: American Psychological Association.
- Machamer, J. E., Temkin, N. R., & Dikmen, S. S. (2003). Neurobehavioral outcome in persons with violent or nonviolent traumatic brain injury. *Journal of Head Trauma Rehabilitation, 18*, 387-397.
- Massagli, T. L., Michaud, L. J., & Rivara, F. P. (1996). Association between injury indices and outcome after severe traumatic brain injury in children. *Archives of Physical Medicine and Rehabilitation, 77*, 25-132.
- McCarthy, M. L., Dikmen, S. S., Langlois, J. A., Selassie, A. W., Gu, J. K., & Horner, M. D. (2006). Self-reported psychosocial health among adults with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 87*, 953-961.
- McCauley, S. R., Boake, C., Levin, H. S., Contant, C. F., & Song, J. X. (2001). Postconcussional disorder following mild to moderate traumatic brain injury: Anxiety, depression, and social support as risk factors and comorbidities. *Journal of Clinical and Experimental Neuropsychology, 6*, 792-808.
- McLeod, J. D., & Kessler, R. C. (1990). Socioeconomic status differences in vulnerability to undesirable life events. *Journal of Health and Social Behavior, 31*, 162-172.
- Meares, S., Shores, E. A., Batchelor, J., Baguley, I. J., Chapman, J., Gurka, J., et al. (2006). The relationship of psychological and cognitive factors and opioids in the development of the postconcussion syndrome in general trauma patients with mild traumatic brain injury. *Journal of the International Neuropsychological Society, 12*, 792-801.
- Miller, S. M., & Seligman, M. E. P. (1982). The reformulated model of helplessness and depression: Evidence and theory. In R. W. J. Neufield (Ed.), *Psychological stress and psychopathology* (pp. 149-178). New York: McGraw-Hill.
- Mittenberg, W., & Strauman, S. (2000). Diagnosis of mild head injury and the postconcussion syndrome. *The Journal of Head Trauma Rehabilitation, 15*, 783-791.
- Moore, A. D., & Stambrook, M. (1994). Coping following traumatic brain injury: Derivation and validation of TBI sample Ways of Coping-Revised subscales. *Canadian Journal of Rehabilitation, 7*, 193-200.
- Ostler, K., Thompson, C., Kinmonth, A.-L. K., Peveler, R. C., Stevens, L., & Stevens, A. (2001). Influence of socio-economic deprivation on the prevalence and outcome of depression in primary care: The Hampshire Depression Project. *British Journal of Psychiatry, 178*, 12-17.

- Riley, G. A., Brennan, A. J., & Powell, T. (2004). Threat appraisal and avoidance after traumatic brain injury: Why and how often are activities avoided? *Brain Injury, 18*, 871-888.
- Rosenberg, M. L., & Fenley, M. A. (1991). *Violence in America: A public health approach*. New York: Oxford.
- Ross, C. E. (2000). Neighborhood disadvantage and adult depression. *Journal of Health and Social Behavior, 41*, 177-187.
- Santa Maria, M. P., Pinkston, J. B., Miller, S. R., & Gouvier, W. D. (2001). Stability of postconcussion symptomology differs between high and low responders and by gender but not by mild head injury status. *Archives of Clinical Neuropsychology, 16*, 133-140.
- Sbordone, R. J. (1999). Post-traumatic stress disorder: An overview and its relationship to closed head injuries. *NeuroRehabilitation, 13*, 69-78.
- Schopp, L. H., Shigaki, C. L., Bounds, T. A., Johnstone, B., Stucky, R. C., & Conway, D. L. (2006). Outcomes in TBI with violent versus nonviolent etiology in a predominantly rural setting. *Journal of Head Trauma Rehabilitation, 21*, 213-225.
- Selassie, A. W., Pickelsimer, E. E., Gravelle, W., Gu, J., Ferguson, P. L., & Lineberry, L. A. (2006). *South Carolina Traumatic Brain Injury Follow-up Registry and Surveillance System: Data codebook, data dictionary, descriptive data*. Charleston, SC: Medical University of South Carolina.
- Silver, J. M., Kramer, R., Greenwald, S., & Weissman, M. (2001). The association between head injuries and psychiatric disorders: Findings from the New Haven NIMH Epidemiological Catchment Area Study. *Brain Injury, 15*, 935-945.
- Slavik, S., & Croake, J. (2006). The individual psychology conception of depression as a stress-diathesis model. *The Journal of Individual Psychology, 62*, 417-428.
- Sosin, D. M., Sniezek, J. E., & Thurman, D. J. (1996). Incidence of mild and moderate brain injury in the United States, 1991. *Brain Injury, 10*, 47-54.
- Sumpter, R. E., & McMillan, T. M. (2006). Errors in self-report of post-traumatic stress disorder after severe traumatic brain injury. *Brain Injury, 20*, 93-99.
- Teasdale, G., & Jennet, B. (1976). Assessment and prognosis of coma after head injury. *Acta Neurochirurgica, 34*, 45-55.
- Thurman, D. J., Alverson, C., Dunn, K. A., Guerro, J., & Sniezek, J. E. (1999). Traumatic brain injury in the United States: A public health perspective. *Journal of Head Trauma Rehabilitation, 14*, 602-615.

- Thurman, D. J., Sniezek, J. E., Johnson, D., Greenspan, A., & Smith, S. M. (1995). *Guidelines for surveillance of central nervous system injury*. Atlanta, GA: Centers for Disease Control and Prevention.
- Turner, R. J., & Lloyd, D. A. (2004). Stress burden and the lifetime incidence of psychiatric disorder in young adults: Racial and ethnic contrasts. *Archives of General Psychiatry*, *61*, 481-488.
- United States Census Bureau. (2007, May 7). *State and county quickfacts: South Carolina*. Retrieved July 17, 2007, from <http://quickfacts.census.gov/qfd/states/45000.html>.
- van Zomeren, A. H., & van den Burg, W. (1985). Residual complaints of patients two years after severe head injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, *48*, 21-28.
- Wagner, A. K., Hammond, F. M., Grigsby, J., & Norton, H. J. (2000). The value of trauma scores: Predicting discharge after traumatic brain injury. *American Journal of Physical Medicine and Rehabilitation*, *79*, 235-242.
- Wagner, A. K., Hammond, F. M., Sasser, H., & Norton, H. J. (2000). Use of injury severity variables determining disability and community integration after traumatic brain injury. *Journal of Trauma-Injury Infection and Critical Care*, *49*, 411-419.
- Wagner, A. K., Sasser, H. C., Hammond, F. M., Wiercisiewski, D., & Alexander, J. (2000). Intentional traumatic brain injury: Epidemiology, risk factors, and associations with injury severity and mortality. *Journal of Trauma-Injury Infection and Critical Care*, *49*, 404-410.
- Warden, D. L., Labbate, L. A., Salazar, A. M., Nelson, R., Sheley, E., Staudenmeier, J., et al. (1997). Posttraumatic stress disorder in patients with traumatic brain injury and amnesia for the event? *Journal of Neuropsychiatry and Clinical Neurosciences*, *9*, 18-22.
- Ware, J. E., & Sherbourne, D. (1992). The MOS 36-Item Short-Form Health Survey (SF-36): I. Conceptual framework and item selection. *Medical Care*, *30*, 473-483.
- Weich, S., & Lewis, G. (1998). Poverty, unemployment, and common mental disorders: Population based cohort study. *British Medical Journal*, *317*, 115-119.
- Wenden, F. J., Crawford, S., Wade, D. T., King, N. S., & Moss, N. E. G. (1998). Assault, post-traumatic amnesia and other variables related to outcome following head injury. *Clinical Rehabilitation*, *12*, 53-63.
- Whitman, S., Coonley-Hoganson, R., & Desai, B. (1984). Comparative head trauma experiences in two socioeconomically different Chicago-area communities. *American Journal of Epidemiology*, *119*, 570-580.

Williams, W. H., Evans, J. J., Needham, P., & Wilson, B. A. (2002). Neurological, cognitive and attributional predictors of posttraumatic stress symptoms after traumatic brain injury. *Journal of Traumatic Stress, 15*, 397-400.

Zafonte, R. D., Mann, N. R., Millis, S. R., Wood, D. L., Lee, C. Y., & Black, K. L. (1997). Functional outcome after violence related traumatic brain injury. *Brain Injury, 11*, 403-407.

APPENDIX A

QUESTIONS USED TO ASSESS CRITERIA FOR DEPRESSION

Criteria A-1 – depressed mood most of the day, nearly every day...

D1. How much of the time during the past 4 weeks...have you felt so down in the dumps that nothing could cheer you up? ¹

D2. How much of the time during the past 4 weeks...have you felt downhearted and blue? ¹

D3. How much of the time during the past 4 weeks...have you been a happy person? ^{1*}

Criteria A-5 – psychomotor agitation or retardation nearly every day

D4. I react slowly to things that are said or done. ²

Criteria A-6 – fatigue or loss of energy nearly every day

D5. How much of the time during the past 4 weeks...did you feel full of pep? ^{1*}

D6. How much of the time during the past 4 weeks...did you have a lot of energy? ^{1*}

D7. How much of the time during the past 4 weeks...did you feel worn out? ¹

D8. How much of the time during the past 4 weeks...did you feel tired? ¹

Criteria A-8 – diminished ability to think or concentrate, or indecisiveness, nearly every day

D9. I am confused and start several actions at a time. ²

D10. I have difficulty reasoning and solving problems, for example, making plans, making decisions, or learning new things. ²

D11. I sometimes behave as if I were confused or disoriented in place or time, for example, where I am, who is around, directions, what day it is. ²

D12. I do not keep my attention on any activity for long. ²

D13. I have difficulty doing activities involving concentration and thinking. ²

1 = Question from the Short Form-36 (SF-36; Ware and Sherbourne, 1992)

2 = Question from the Sickness Impact Profile (Bergner, Bobbit, Carter, and Gilson, 1981)

* = Reverse-scored item

APPENDIX B

QUESTIONS USED TO ASSESS CRITERIA FOR ANXIETY

GAD Criteria A – excessive anxiety or worry

A1. How much of the time during the past 4 weeks...have you been a very nervous person? ¹

GAD Criteria C-1 – restlessness or feeling keyed up or on edge

A2. During the past 4 weeks, have you been tense or keyed up? ³

A3. How much of the time during the past 4 weeks...have you felt calm and peaceful? ^{1*}

GAD Criteria C-2 – easily fatigued

A4. How much of the time during the past 4 weeks...did you feel full of pep? ^{1*}

A5. How much of the time during the past 4 weeks...did you have a lot of energy? ^{1*}

A6. How much of the time during the past 4 weeks...did you feel worn out? ¹

A7. How much of the time during the past 4 weeks...did you feel tired? ¹

GAD Criteria C-3 – difficulty concentrating or mind going blank

A8. I forget a lot, for example, things that happened recently, where I put things, appointments. ²

A9. I do not keep my attention on any activity for long. ²

A10. I have difficulty doing activities involving concentration and thinking. ²

GAD Criteria C-4 – irritability (or outbursts of anger)

A11. During the past 4 weeks, have you been irritable or grouchy? ⁴

A12. During the past 4 weeks, have you had trouble controlling your temper? ⁵

A13. During the past 4 weeks, have you had trouble getting along with others? ⁵

1 = Question from the Short Form-36 (SF-36; Ware and Sherbourne, 1992)

2 = Question from the Sickness Impact Profile (Bergner, Bobbit, Carter, and Gilson, 1981)

3 = Question from the Modified Hopkins Symptom Checklist (Derogatis et al., 1974)

4 = Question from the Adapted Trauma Complaints List (van Zomeren and van den Burg, 1985)

5 = Non-standard question used as a part of the larger epidemiological study

* = Reverse-scored item

APPENDIX C

QUESTIONS USED TO ASSESS DISTRESS OR IMPAIRMENT IN FUNCTIONING

- F1. During the past 4 weeks, have you cut down the amount of time you spent on work or regular daily activities as a result of any emotional problems, such as feeling depressed or anxious?
 - F2. During the past 4 weeks, have you accomplished less than you would like as a result of any emotional problems, such as feeling depressed or anxious?
 - F3. During the past 4 weeks, did you not do work or other regular daily activities as carefully as usual as a result of any emotional problems, such as feeling depressed or anxious?
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VITA

The author is a native of Atlanta, Georgia, and completed his secondary education at Tucker High School, graduating in 1994. He received a Bachelor of Science in psychology from Birmingham-Southern College in 1998. He was elected to numerous honorary societies while at Birmingham-Southern College, including Psi Chi and Phi Beta Kappa. He received a Master of Arts in psychology from Louisiana State University in 2002. He is currently a candidate for the degree of Doctor of Philosophy in the Department of Psychology at Louisiana State University, and that degree will be conferred on him in December 2007.