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Predictors of Postconcussion Symptomatology in a Mild Head Injury College Population.

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PREDICTORS OF POSTCONCUSSION SYMPTOMATOLOGY
IN A MILD HEAD INJURY COLLEGE POPULATION

A Dissertation

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

in

The Department of Psychology

by
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ABSTRACT

Mild head injury is the most common form of head injury and the majority of individuals who sustain such injuries are young adults. Following mild head injury, individuals often complain of a number of physical, cognitive, and behavioral symptoms referred to as postconcussion symptoms (PCS). The most commonly reported postconcussion symptoms are headache, dizziness, decreased concentration, memory problems, irritability, fatigue, visual disturbances, sensitivity to noise, judgement problems, and anxiety. These symptoms can persist from months to years following injury and may even be permanent and cause disability (Brown, Fann, & Grant, 1994; Gouvier, Cubic, Jones, Brantley, and Cutlip, 1992). Both organic and psychological etiologies have been suggested for persistent PCS and most investigators now believe that a combination of multiple organic and psychological factors contribute to the development and continuation of these symptoms (Bohnen & Jolles, 1992). A number of neurocognitive, psychosocial, premorbid, and injury-related variables have been implicated in the development of persistent PCS including reduced information processing, increased psychological distress, external locus of control, female gender, positive premorbid history of psychological disturbance, and previous history of head injury. However, the findings among the various research studies have been conflicting. Determining the variables that influence the development of persisting PCS is important for identifying those at risk for chronic PCS following mild head injury and subsequently for tailoring preventative and palliative intervention strategies to manage PCS. Given this information, the present study attempted to identify premorbid/injury-related,
neurocognitive, and psychosocial factors associated with persistent postconcussion symptomatology among mildly head-injured college students. The findings demonstrated that current psychological distress and female gender were the best predictors of PCS; high rates of PCS were associated with the presence of either of these factors. Decreased information processing and external locus of control were also related to PCS, but the relationships were weak. Prior head injury and premorbid history of psychological problems were not related to PCS. The results suggest that emotional status and gender are more important in predicting persistent PCS than neurocognitive status, psychological history, or history of previous mild head injury.
INTRODUCTION

Mild head injury is the most common form of head injury and the majority of individuals who sustain such injuries are young adults. Mild head injury was once thought to be trivial in terms of consequences, however, there is now substantial evidence that neuropathological, neurophysiological, and neurocognitive changes occur with such mild injuries (Dikmen & Levin, 1993). Diffuse axonal injury has been identified as a consistent feature of mild head injury in both animal and human studies (Povlishock, Erb, & Astrug, 1992). In healthy young adults, the neurocognitive effects of mild head injury tend to be selective and subtle. Specifically, a reduction in speed of information processing appears to be the primary deficit observed (Dikmen & Levin, 1993; Gronwall, 1989). Individuals sustaining mild head injuries also complain of a number of physical, cognitive, and behavioral symptoms referred to as postconcussion symptoms (PCS). The most commonly reported postconcussion symptoms are headache, dizziness, decreased concentration, memory problems, irritability, fatigue, visual disturbances, sensitivity to noise, judgement problems, and anxiety. These symptoms can persist from months to years following injury and may even be permanent and cause disability (Brown, Fann, & Grant, 1994; Gouvier, Cubic, Jones, Brantley, and Cutlip, 1992). Both organic and psychological etiologies have been suggested for persistent postconcussion symptoms which has caused much controversy and debate in the literature. Most investigators now believe that multiple organic and psychological factors contribute to the development and continuation of these symptoms in those sustaining mild head injuries (Bohnen & Jolles, 1992). While a number of factors have
been suggested as being related to and/or predictive of postconcussion symptoms, the results from different studies have been conflicting. Yet determining the variables that predict postconcussion symptomatology is important in terms of identifying those at risk following mild head injury and in developing preventative and palliative intervention strategies tailored specifically towards these subgroups. While brain injury may not be reversible, the impact of its effects may be reduced (Dikmen & Levin, 1993). Recently, Mittenberg, Tremont, Zielinski, Fichera, and Rayls (1996) demonstrated the effectiveness of brief, early psychoeducational intervention in reducing the incidence PCS following mild head injury.
EPIDEMIOLOGY OF MILD HEAD INJURY

Mild Head Injury Occurrence

Head injury is a frequent occurrence in the United States and other industrialized nations and is the leading cause of brain injury. Estimates suggest that annually about 200 out of every 100,000 people in the United States sustain head injuries with resulting brain damage (Frankowski, 1986). While the severity of head injuries vary, the majority, 70 to 90%, are considered mild (Rimel, Giordani, Barth, Boll, & Jane, 1981). Mild head injury has been described as any external trauma to the head, such as the head being struck, the head striking an object, or the head undergoing violent motion, that results in alteration of or brief loss of consciousness without neurological emergency and only brief or no hospitalization (Kay, Newman, Cavallo, Ezrachi, & Resnick, 1992). Despite the fact that the majority of head injuries are mild, epidemiological investigation of mild head injury has not been as extensive as that concerning more severe injuries. Kraus and Nourjah (1989) studied the occurrence of head injury among residents of San Diego County, California. These investigators found that 82% of those hospitalized for brain injury had sustained mild head injuries with an incidence rate of 131 per 100,000. Kay and colleagues (1992) reported data indicating that 72.5% of head injury patients admitted to Bellevue Hospital in New York had sustained mild injuries. The authors note that these statistics when extrapolated to the U.S. population yield an estimate of about 300,000 cases of mild head injury admitted to hospitals per year. However, these numbers may be underestimates of the actual occurrence of mild head injury. Most studies as those presented above have utilized only hospitalized cases and as a result
those individuals who incur mild head injuries but who are not hospitalized or who are not even seen by medical personnel are left out. Fife (1987) examined data of persons sustaining mild head injuries who had sought some sort of medical attention and found that 82% were not hospitalized. He estimated that 1,664,300 individuals seek medical help for mild head injuries each year of which 82% or 1,357,00 individuals are not hospitalized. However, once again patients who do not even see medical personnel are not included.

It has been estimated that 20 to 40% of individuals sustaining mild head injuries never seek medical attention (Gualtieri, 1995). Some researchers have utilized self-report as a means of including those do not receive medical attention for their injuries in their investigations of the occurrence of mild head injury. Crovitz, Horn, and Daniel (1983) examined head injury among college students and found that 24% of males and 16% of females reported experiencing a head injury with some loss of consciousness. Segalowitz, Lawson, and Berge (1993) examined occurrence of head injury among three samples (high school, university, general population) and found an overall prevalence rate of 30% with about half reporting concomitant unconsciousness. The majority of head injuries, particularly among the student samples, were reported as mild. Segalowitz and Lawson (1995) recently reported results from a large study of mild head injury in high school and college students. Similar to other findings, prevalence was found to be 25% for college students and 35% for high school students with the majority reporting only one injury. In addition, 81% of the college students and 74% of the high school students reported that they were not hospitalized for the injury. A very recent study
(Ryan, O'Jile, Gouvier, Parks-Levy, & Betz, 1996) found similar rates of head injury among a sample of college students. These investigators found an overall prevalence of self-reported head injury of 23%. The majority, 87%, had sustained mild head injuries with 84% reporting having received only brief or no medical attention. In addition, among those who had been injured, the majority, 81%, incurred only a single injury. The results of these studies indicate a fairly high prevalence of mild head injury even among presumably high functioning individuals attending college.

The occurrence of head injury is related to both age and gender as well as other sociodemographic variables. There appears to be three age-related peaks of head injury occurrence: ages 1 to 5, 15 to 24, over 65, and of these, the highest incidence rates are seen among people in the 15 to 24 age range (Frankowski, 1986; Naugle, 1990). Persons most at risk of sustaining head injuries, the majority of which will be mild, are those just entering young adulthood. In terms of gender, males have been found to consistently outnumber females in rates of head injury, with males making up from approximately 61 to 79% of head injury samples. This gender bias seems to be at its highest in the mid adolescence to early adulthood range. Males also have been found to sustain more severe injuries and have a higher mortality rate than females (Naugle, 1990). Alcohol consumption is well established as a correlate of head injury. One third or more of all head injuries have been estimated to occur after alcohol consumption with even higher rates for motor vehicle accidents and assaults (Kraus & Nourjah, 1989; Naugle, 1990). Higher premorbid rates of alcohol and drug use have been reported among those sustaining even mild head injuries as well (Robertson, Rath, Fournet,
Zelhart, & Estes, 1994; Ryan et al., 1995). In addition, alcohol appears to be more of a factor in head injuries sustained by adult males than females (Naugle, 1990). There has been some suggestion that psychological disturbance is related to head injury occurrence (Sims, 1985). However, recent studies of mild head injury patients have failed to find differences with controls in terms of premorbid psychological adjustment, physical or somatic disorders, or in emotional and behavioral factors (Dicker, 1992; Robertson, et al., 1994). Finally, history of a head injury itself has been found to be a risk factor for subsequent head injury. Individuals with a history of head injury have a greater likelihood of sustaining a head injury than those without such a history (Naugle, 1990).

Causes of Injury

In terms of etiology of head injuries in general, most studies report that motor vehicle accidents account for about 50% or more of all head injuries. Next are falls which overall account for approximately 28% and are more often found in the very young and older individuals. Falls are followed by assaults which account for about 16% and then sporting/recreational activities which comprise about 7% (Kraus & Nourjah, 1989; Naugle, 1990). However, this pattern has not been found when investigating the causes of mild head injury. Gronwall (1991) states that the majority of mild head injuries are caused by sporting accidents, falls, or hitting objects such as tree limbs because high velocity events like motor vehicle accidents, are more likely to cause greater injury. One study (Wrightson & Gronwall, 1981) of mild head injury among employed men aged 17 to 48 found that motor vehicle accidents were responsible for only 10.5% of the injuries while sporting accidents accounted for about 66%. Recently, Ryan, et al. (1996)
reported similar results with a group of college students, most of whom had sustained mild injuries. The most frequent cause of head injury was sporting accidents (37.1%), followed by falls (23.2%), then motor vehicle accidents (20%), and finally assaults (5.3%). Subjects in this sample may have been pre-selected for very mild injuries given their ability to attend college and so it is not surprising that sporting accidents and falls were more common than motor vehicle accidents which typically involve acceleration-deceleration injuries and as a result have the highest potential for more severe brain injury.

Classification of Injury Severity

Measurement of Severity

The severity of a head injury has been measured by a number of methods. The length of loss of consciousness (LOC) at the time of injury is the oldest means of assessing severity. However, it is now well established that brain injury can occur without complete loss of consciousness (Bigler, 1990). Alteration of consciousness or mental status, i.e., being dazed, disoriented, or confused, following head trauma is considered the minimal grade of concussion/cerebral injury (Ommaya & Gennarelli, 1974). Another method used to assess severity has been length of post-traumatic amnesia (PTA; Smith, 1961). As Bigler (1990, p. 16) states “PTA is not to be confused with coma and recovery from coma. PTA assumes that the patient is alert and functioning and has recovered from the comatose state but has persistent, severe deficits in retaining new information and processing new memories.” The Glasgow Coma Scale (GCS) developed by Teasdale & Jennett (1974) is the first empirically validated measure
of head injury severity. The GCS quantifies level of consciousness/coma. However, the GCS is typically administered on initial presentation to medical personnel and as a result applicable only to those individuals with head injury who receive immediate medical attention. Utilizing the GCS in research, scores of 13 to 15 (out of a maximum score of 15) are generally considered mild. Variability among studies is seen with the use of LOC and PTA where mild injury classifications range from LOC of one hour or less to 20 minutes or less and PTA of 24 hours or less to one hour or less. In addition, some studies only include those with brief but full loss of consciousness and others have included those with only a brief alteration of consciousness. As Esselman and Uomoto (1995) note there needs to be a single definition of mild head injury that is used consistently in research in order to gain an accurate understanding of the incidence, effects, and recovery following mild head injury.

Definition of Mild Head Injury

In order to alleviate the variability in the classification of mild head injury the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine recently developed a definition of mild head injury (Kay, et al., 1993). This definition states that the event of a mild traumatic brain injury or mild head injury consists of a traumatically induced physiological disruption of brain function that is manifested by at least one of these symptoms: loss of consciousness for 30 minutes or less; loss of memory for events immediately before (retrograde amnesia) or after the accident (PTA not greater than 24 hours); any alteration in mental state at the time of the injury, i.e., feeling dazed,
disoriented, or confused; and the presence of focal neurological deficits that may or may not be transient. In addition, if given, initial GCS scores between 13 to 15 but not lower. The committee notes that this definition includes the head being struck, the head striking an object, and/or the brain being subjected to acceleration/deceleration movement (i.e., whiplash) without direct external trauma to the head.
MECHANISMS OF MILD HEAD INJURY

Neuropathology

There is a large body of evidence now indicating that neuropathological changes occur with even mild head injury. Mild head injury represents the low end of the spectrum where pathological changes increase as the severity of injury increases (Dixon, Taft, & Hayes, 1993). In all head injuries, mechanical force to the head, either through direct impact or acceleration-deceleration motion, leads to a rapid displacement of the skull, which if severe enough can cause differential motion between the brain and skull. The severity of displacement is determined by the path of motion of the head, the anatomical surfaces surrounding the brain, and the violence of the motion. Deformation of brain tissue is a result of such displacement and is thought to be the primary factor in brain damage. Cerebral deformation can result in structural alterations of neurons, such as axonal and cytoskeletal injury, and vasculature, such as contusions and hemorrhage, generation of oxygen radicals, and/or excessive neural depolarization causing abnormal neurochemical agonist-receptor interactions related to excitotoxic processes (Dixon, et al., 1993). Specifically, there is evidence that activation of muscarinic cholinergic and/or NMDA glutamate receptors is involved (Hayes, Jenkins, & Lyeth, 1992). Such neurochemical alterations may play a role in the behavioral changes associated with head injury. Lyeth, et al. (1990) found prolonged spatial memory deficits in rats after mild to moderate head injury in the absence of cell death in the hippocampus or axonal injury in hippocampal pathways. These researchers suggested that widespread neuronal
excitation may cause prolonged pathological changes in neural function such as within the hippocampus which could disrupt memory.

Diffuse axonal injury has been demonstrated in clinical and laboratory studies of head injury and seems to be a consistent feature of all injuries regardless of severity with the distribution and number of axons involved increasing with injury severity (Povlishock, et al., 1992). Axonal injury may come from physical shearing or tearing at the time of injury and/or a delayed pathophysiological reaction that may occur over several hours. There is building evidence to suggest that delayed physiological processes may be more relevant to mild head injuries given that axonal damage has been found in the absence of gross structural damage (Dixon, et al., 1993). Povlishock, Becker, Cheng, and Vaughan (1983) found that axonal changes occurred without the presence of focal parenchymal or vascular damage in cats subjected to mild head injuries, which suggests that mild brain injury may disrupt axonal functioning without physical shearing or tearing. These investigators reported that such axonal alteration seems to result from focal or discrete changes within the axon which progresses in severity until it results in actual axonal separation. They further reported that axonal change can occur without clinical neurological abnormalities and suggested that it may be that damage occurs to a limited number of axons within a given fiber tract and as such may not significantly compromise the entire system. A comparable mechanism of action is likely in head-injured humans (Povlishock & Coburn, 1989). Often, humans with mild head injury do not show overt clinical deficits but may have multiple subtle subjective complaints which may represent alterations to a limited number of axons (Povlishock, et al., 1983). In
addition, axonal injury may contribute to neurotransmitter changes by tissue destruction and/or deaffernation (Dixon, et al., 1993).

**Neuroimaging Findings**

Structural neuroimaging, computed tomography (CT) and magnetic resonance imaging (MRI) have had limited utility in the evaluation of mild head injury. Abnormalities on CT scans are rarely found in patients with mild injuries. Abnormalities on MRI have been found more frequently but only in some patients. In a group of patients with mild to moderate injuries, Eisenberg and Levin (1989) found multifocal lesions primarily in frontotemporal regions. A recent study by Bigler and Synder (1995) involving a small group of patients with documented mild head injury who had prior neuroimaging evaluated pre and post-injury scans. Despite persistent mild neurocognitive deficits and emotional sequelae post-injury MRI did not show changes from pre-injury scans or differences from control subjects.

Functional neuroimaging, positron emission tomography (PET) appears to be more promising in identifying metabolic alterations in mild head injury although research has been limited. A study by Pogacnik (1989) demonstrated alterations in regional cerebral blood flow during a visual memory activation task in mild head injury patients compared to normal controls. The mild head injury patients showed impaired adaptability to metabolic needs. Blood flow alterations were primarily noted in frontal and temporal regions. A more recent study by Ruff and colleagues (1994) examined glucose metabolism in mild head injury patients with persistent neuropsychological deficits but little or no abnormalities on CT/MRI and controls. The mild head injury
patients included those with and without loss of consciousness (LOC). Abnormalities, particularly hypometabolism, was found in the mild head injury patients but not in controls. These abnormalities were seen primarily in frontal and anterior temporofrontal regions. No significant differences were found between patients with and without LOC.

The findings of frontal and temporal abnormalities on neuroimaging in mild head injury is not surprising given that structural damage is observed to occur first in these regions after head injury in general. Orbitofrontal and anterior temporal regions are particularly vulnerable to contusions, lacerations, abrasions, hematomas, and intercerebral hemorrhages due to forceful contact with the rough bony surface of the skull in these areas during head injury (Mattson & Levin, 1987; Varney & Menefee, 1993). In addition, diffuse axonal damage may disrupt frontal pathways to other cortical and subcortical regions, including the limbic system. Damage to these regions have been linked to deficits in complex neurocognitive functioning including attention and memory as well as to emotional changes (Mattson & Levin, 1987).
SEQUELAE OF MILD HEAD INJURY

Individuals with mild head injury have been found to exhibit a number of physical, cognitive, and behavioral symptoms as a result of the injury. The physical symptoms include nausea, dizziness, headache, problems with vision, etc. which are unrelated to peripheral injury or other causes. Cognitive symptoms following mild injury may include decreased attention/concentration, memory problems, etc. Behavioral changes or emotional symptoms may include irritability, disinhibition, emotional lability, etc. Such symptoms may be present relatively shortly following injury and may be persistent. Mild head injury can result in long-term and even permanent sequelae resulting in functional disability (Brown, et al., 1994; Kay, et al., 1993).

Neurocognitive Changes

A number of studies over the last two decades have examined neurocognitive functioning following mild head injury. Gross deficits in intelligence or memory have not been demonstrated but subtle dysfunction in attention/information processing have been found (Bohnen, Jolles, Twijnstra, Mellink, & Wijnen, 1995). Head injury typically results in diffuse damage which produces a reduction in information processing capacity which has been broadly described as the number of operations the brain can carry out at the same time. Individuals with mild head injury demonstrate problems when they are required to analyze or process more information than they can handle simultaneously (Gronwall, 1989). Decreased information processing is manifested primarily by problems with attention but has also been implicated in memory impairment (Kay, et al., 1992; Szymanski & Linn, 1992).
While the nature of the neurocognitive changes following mild head injury are well established and accepted there is some question as to how long these changes persist. Some studies have demonstrated persistent neurocognitive deficits while others have not. Such conflicting results may be due to methodological differences in the selection of subjects and tests used. Barth, Macciocchi, Giordani, Rimel, Jane, and Boll (1983) found that three months post-injury, mild head injury patients demonstrated mild neuropsychological test impairment which was secondary to reduced cognitive/information processing efficiency. Dikmen, McLean, and Temkin (1986), however, found that patients with uncomplicated mild head injuries, showed deficits in attention and learning initially but were generally recovered to within normal limits by 12 months post-injury.

As Gronwall (1989) notes, however, while scores on many measures of information processing return to normal within weeks/months in healthy young adults, there is now good evidence that impairment may persist longer on some measures of attention and reaction time. In addition, there is evidence that just because scores return to normal does not mean that full recovery has been reached. In an initial study Gentilini, et al. (1985) failed to find significant differences in overall cognitive performance between mild head injury patients one month post-injury and matched controls but did find a trend for an isolated deficit in selective/focused attention. To further evaluate this finding, mild head injury patients one and three months post-injury and controls were assessed with a battery designed to tap different aspects of attention including selective, sustained, divided, and distributed attention. Significant impairment
in selective attention was noted at one and three months post-injury but the deficit was not manifested in number of errors but rather in increased execution time for the task suggesting compensation for defective performance that was reflected as slowing. Impairment in sustained, divided, and distributed attention was also noted (Gentilini, Nichelli, & Schoenhuber, 1989). Recent studies have similarly found isolated deficits in sustained and focused attention initially and later post-injury (Batchelor, Harvey, & Bryant, 1995; Newcombe, Rabbitt, & Briggs, 1994).

Even individuals who appear well recovered following mild head injuries are susceptible to periodic impairments under conditions of physiological or psychological stress (Alexander, 1995). Ewing, McCarthy, Gronwall, and Wrightson (1980) examined cognitive processing performance in students under a mild hypoxia stressor who had fully recovered from a mild head injury one to three years before. These mildly head-injured students were significantly poorer than normal controls on a vigilance/attention and memory task when mildly hypoxic. The investigators state that the mild head injury resulted in residual damage that caused an impaired ability to withstand another central nervous system stressor. So while functional recovery may take place in healthy young adults, cerebral damage is not reversible. Persistent impairment is seen clinically as well when situational stressors arise (Gronwall, 1989).

Given that attentional deficits predominate following mild head injury, some studies have compared mild head injury subjects to those with diagnosed attention deficit disorders. One such study found that both mild head injury subjects (mean time since injury about one year) and attention deficit disorder subjects had significantly greater...
difficulty than controls on measures of sustained attention but the impairment in mild head injury subjects was characterized by generalized slowness in speed of information processing while the impairment in ADD subjects was characterized by problems with impulsivity and regulation of attention (Arcia & Gualtier, 1994).

**Postconcussion Symptoms.**

Postconcussion symptoms (PCS) are a cluster of symptoms that frequently occur following mild head injury. It should be noted, however, that such symptoms occur after more severe injuries as well. PCS consist of a number of self-reported physical, cognitive, and emotional/behavioral symptoms. The most commonly documented symptoms include headache, dizziness, irritability, difficulty concentrating, memory problems, fatigue, visual disturbances, sensitivity to noise, judgement problems, and anxiety (Gouvier, et al., 1992). This cluster of PCS when persistent in nature is frequently referred to as the postconcussion syndrome. These symptoms have also been called late symptoms because they are often reported a few days and weeks following the head injury. Early symptoms are those that individuals complain of immediately following their injury such as nausea, vomiting, and drowsiness and are short-lived (Bohnen & Jolles, 1992; Rutherford, 1989). Rutherford (1989) has suggested that while the underlying brain dysfunction is present from the moment of injury it takes time and the everyday stressors of life to elicit these PCS. However, given that there is good evidence that axonal injury may come from a delayed pathophysiological reaction occurring over several hours (Dixon, et al., 1993) it is also possible that such a mechanism is responsible for the delayed onset of PCS following mild head injury.
It was once believed that PCS resolved by one month in all but a very few individuals following mild head injury. Numerous studies have now demonstrated that PCS are reported for months and even years post-injury in a percentage of those sustaining mild head injuries. In fact, symptoms have been reported to persist for 15 years or more (Binder, 1986; Bohnen & Jolles, 1992; Rutherford, 1989). However, there is much variation in the reported prevalence of persistent PCS among different studies. Prevalence rates at three months post-injury have been found to range from 24% to 84% (Rutherford, 1989). Such variability may be related to methodological differences between studies but may also reflect a lack of homogeneity among the population of individuals with persistent PCS (Bohnen & Jolles, 1992). A recent study by Alves, Macciocchi, and Barth (1993) evaluated PCS in 587 patients admitted to the hospital with uncomplicated mild head injury. Two thirds of these patients were symptomatic at discharge and a clinically significant proportion were symptomatic at three (60%), six (45%) and 12 months (40%). Interestingly, while symptoms at discharge were not related to symptoms at follow-up, patients symptomatic at three or six months were more likely to be symptomatic at six or 12 months. Similarly, another study also found that the presence of PCS at six weeks was a strong predictor of chronicity at later follow-up (Fenton, McClelland, Montgomery, MacFlynn, & Rutherford, 1993).

The etiology of persistent postconcussion symptoms or the postconcussion syndrome has generated much controversy concerning whether such symptoms are due to alterations in neurophysiology and neuropathology secondary to the injury, or due to
psychological factors prior to/after the injury (Szymanski & Linn, 1992). The subjective and rather nonspecific nature of PCS has helped fuel this controversy. The frequency of postconcussion type symptoms or base rates among non-head-injured normal controls, non-head-injured personal injury claimants, medical patients, and psychiatric patients has been found to be high (Gouvier, Uddo-Crane, & Brown, 1988; Lees-Haley & Brown, 1993; Fox, Less-Haley, Earnest, & Dolezal-Wood, 1995a; Fox, Lees-Haley, Earnest, & Dolezal-Wood, 1995b). However, in the studies of medical and psychiatric patients, greater symptom endorsement occurred in patients who reported being knocked unconscious, particularly for some of the more traditional PCS (headache, memory and concentration problems, sensitivity to noise, etc.). Further regression analyses demonstrated that being knocked unconscious was a strong predictor of more PCS. The authors state that the results suggests that some PCS are related to head trauma while others may be related to situational factors or general psychological distress (Fox, et al, 1995a; 1995b).

Psychological and motivational factors such as involvement in litigation and premorbid/postmorbid psychological problems have been listed as causes of persistent PCS and the terms posttraumatic or compensation neurosis have been used. Compensation neurosis dates back to the 1960's with Miller being the most outspoken supporter that PCS directly resulted from involvement in litigation, i.e., that patients exaggerate/malinger these symptoms in order to gain monetary compensation (Gouvier, et al., 1992; Miller, 1961). However, in contrast to Miller, numerous other researchers documented the presence of PCS in patients not involved in litigation and have failed to
show a relationship between PCS and litigation (Bohnen & Jolles, 1992; Rimel, et al., 
1981; Wrightson & Gronwall, 1981). The influence of psychological disturbance is still 
an issue, however. Premorbid emotional problems and post-injury level of psychological 
distress have been implicated in persistent PCS but not consistently demonstrated.

As noted earlier, much evidence now exists that indicates that mild head injury 
does result in neuropathological and neurophysiological changes. Such neurological 
changes may be the cause of PCS following mild and more severe head injuries. There is 
evidence that PCS are related to neurocognitive impairment. However, as with the 
psychological factors the results are not consistent across studies. In addition, it is clear 
that PCS occur in a number of individuals following mild head injury but not in all. It 
appears that both purely psychogenic and purely physiogenic views are limited. Most 
researchers now believe persistent PCS to be the result of multiple premorbid, injury-
related, and postmorbid neuropathological and psychological factors (Alexander, 1995; 

PCS and Neurocognitive Functioning

There is evidence to indicate that mildly head-injured individuals with persistent 
PCS also demonstrate impairment in neurocognitive functioning relative to both controls 
and to mildly head-injured individuals without PCS although the results are not 
completely consistent. Rimel, et al. (1981) published one of the first studies to show a 
high rate of PCS and neuropsychological impairment in mild head injury patients. At 
three months post-injury 79% reported PCS with headache and memory problems being 
the most common symptoms. These patients also showed mild impairment in
neurocognitive functioning, primarily in the areas of attention/concentration, memory, and judgement/problem solving. A later study by Levin, et al. (1987) found conflicting results. These researchers examined neurobehavioral changes in mild head injury patients consecutively admitted to three medical centers who were screened for premorbid psychiatric disorders, at one week, one month, and three months post-injury. Neuropsychological test impairment was noted initially but generally resolved by the third month. Postconcussion symptoms did not appear related to neurocognitive recovery. Subjective complaints were frequently still present at one and three months post-injury even in patients whose cognitive functioning improved to within normal limits. Another study evaluated uncomplicated mild head injury patients with PCS, 6 to 18 months post-injury and did find deficits in attention/concentration and memory (Mariadas, Rao, Gangadhar, & Hegde, 1989). In addition, Leininger, Gramling, Farrell, Kreutzer, and Peck (1990) demonstrated that mild head injury patients with persistent PCS, 1 to 22 months post-injury, performed significantly poorer than controls on measures of information processing, reasoning, and verbal learning. There was no difference in performance between patients who experienced LOC and those who experienced disorientation/confusion but no LOC. Also there was no evidence that litigation status affected performance or time since injury. It should be noted, though that all patients were referred for testing because of neurocognitive complaints and nearly all of the patients were injured in motor vehicle accidents which the authors note may consist of greater acceleration-deceleration injury and thus greater possibility of brain damage. Also, in a study of mild head injury patients with posttraumatic headache...
secondary to motor vehicle accidents a high rate of PCS were noted and the patients
with more pronounced PCS showed significantly greater impairment on 6 of 13
neuropsychological measures assessing verbal fluency, memory, attention and
information processing (Gfeller, Chibnall, & Duckro, 1994).

Bohnen and colleagues attempted to further clarify the issue of PCS and
neurocognitive functioning by directly comparing subjects with and without persistent
PCS who had sustained uncomplicated mild head injuries. In one study, these
investigators evaluated patients with uncomplicated mild head with and without PCS six
months post-injury and normal controls. PCS patients showed deficits in selective and
divided attention compared to patients without PCS and controls (Bohnen, Jolles,
& Twijnstra, 1992). In a later study, these investigators examined frequency of
postconcussion symptoms as well as emotional symptoms and neuropsychological test
performance in patients with uncomplicated mild head injury who were seen but not
admitted to the hospital. Patients were tested initially after the injury and at five weeks,
three months, and six months post-injury. One fourth of the patients had multiple
symptoms (at least three) at three and six months post-injury. These patients with
persistent PCS also complained of more emotional symptoms than those with few or no
symptoms. In terms of neuropsychological performance, patients with persistent
symptoms demonstrated significantly poorer performance on a measure of selective
attention as well as a reduced tolerance to light and sound. Postconcussion-cognitive
symptoms were significantly related to decreased neuropsychological performance while
the emotional symptoms were not (Bohnen, Twijnstra, & Jolles, 1993). In a recent study
by these researchers, mild head injury patients were evaluated even longer post-injury, 12 to 34 months. No overall gross neurocognitive differences were found between patients with and without PCS or controls but an isolated deficit in sustained attention was noted for patients with PCS. Also among PCS patients, those with higher ratings on postconcussive-cognitive symptoms performed less well on a sustained attention task than PCS patients with lower scores (Bohnen, et al., 1995). As these authors note, it may be that persistent neurocognitive deficits may be more prevalent among the subgroup of individuals experiencing PCS.

**PCS and Psychosocial/Injury-related Factors**

A number of psychosocial and injury-related factors have been implicated in the development of persistent PCS including prior history of head injury, neurological signs, female gender, older age, and psychological problems pre and post-injury. As with the relationship between neurocognitive dysfunction and PCS the results from various studies have been conflicting.

Bohnen, Twijnstra, and Jolles (1992) attempted to examine the influence of premorbid problems on PCS following mild head injury. Patients with complicated mild head injury, that is those with multiple head injuries or premorbid emotional problems, and those with uncomplicated mild head injury completed a questionnaire including both traditional postconcussion symptoms and nonspecific emotional/functional symptoms. Factor analysis yielded two distinct groups of symptoms: postconcussive-cognitive symptoms which consisted of typical PCS symptoms such as headache, dizziness, sensitivity to noise, decreased concentration, etc., as well as a second set of problems
including decreased work capacity/efficiency, fatigue, etc., and emotional-vegetative symptoms such as heart palpitations, gastrointestinal problems, depression, emotional lability, etc. Patients were tested initially one to two weeks after injury and then at four to six weeks post-injury. There was no significant relationship between time since injury and the scores on the two scales. Patients with uncomplicated mild head injuries had significantly higher scores on the postconcussive-cognitive scale than controls but not on the emotional-vegetative scale. Patients with complicated mild head injuries scored significantly higher on both scales than patients with uncomplicated mild head injury. These authors suggest that emotional/vegetative complaints appear to be secondary symptoms that may reflect a reduced ability to cope with environmental stressors in those with preexisting complications.

The concept of PCS as reflecting a reduction in the ability to compensate for stressful conditions secondary to residual damage was examined by Gouvier and colleagues (1992). PCS and the influence of stress was evaluated in mildly head-injured and normal control college students. Their results failed to support this concept. The number of PCS were similar for both groups and were correlated with levels of daily reported stress for both head-injured and control subjects. Stress was related to higher symptom rates. In a conflicting report, investigators examined the relationship between PCS and stress after hospital discharge and found that at follow-up (6 to 19 days post-injury), 71% of the mild head injury subjects reported still experiencing PCS and 29% experienced a worsening of symptoms, but stress was not found to have a significant effect (Moss, Crawford, & Wade, 1994).
Age and gender have been identified as factors related to the development of persistent PCS. Older age (over age 40) and female gender have been associated with higher rates of PCS in mildly head-injured individuals. Early studies by Rutherford and colleagues found that at six months post-mild head injury, 51% reported PCS, and at one year post-injury, 14.5% still reported symptoms. Symptoms were more common in females, older patients and those with positive neurological signs at 24 hours following injury (Rutherford, Merrett, & McDonald, 1977; 1979). More recently, a study by Packard, Weaver and Ham (1993) overall found a high rate of PCS but noted that a higher percentage of females reported symptoms than did males. Fenton, et al. (1993) similarly found that older age and female gender were significantly related to PCS but in addition found that mild head injury patients with persistent PCS had greater frequency of premorbid social difficulties as well as a higher frequency of problems with anxiety or depression at six weeks post-injury. Alves, et al. (1993) found that female gender and Glasgow Coma Scale scores was significantly related to PCS at 12 months post-mild head injury but failed to find a relationship with age or psychiatric/substance abuse history. A large study by Bohnen, et al. (1994) investigated the intensity of vague everyday and postconcussion-type symptom complaints including dysthymic complaints (depression, anxiety, tearfulness, etc.), vegetative/bodily complaints (headache, vertigo, lightheadedness, etc.), and performance complaints (decreased work performance, forgetfulness, etc.) in mild head injured patients one to five years post-injury and matched controls via a questionnaire mailing. Overall these complaints were more prevalent and more severe in mild head injured subjects although the pattern of complaints was similar.
for both patients and controls. They also found that a few factors increased the likelihood of these symptoms in patients: older age, female gender, premorbid emotional problems, comorbid medical problems, neurological complication at time of injury, orthopedic fracture, hospitalization, lower education, and intoxication at the time of injury. Other factors were not related such as time since injury and insurance claim. The authors state that the results suggest that mild head injury can result in neurobehavioral sequelae in terms of suboptimal physical and mental health that may not be completely reversible. They further note that the results indicate that both physiological and psychological factors contribute to the persistence of symptoms.

Other recent studies have failed to find a relationship between PCS and psychosocial factors, however. Bohnen, et al (1993) found no significant relationship between incidence of PCS and gender, age, education, or duration of posttraumatic amnesia. Karzmark, Hall, and Englander (1995) investigated the nature of the subjective impact of PCS in mild head injury patients seeking treatment. They found that level of psychological distress was strongly correlated with PCS symptom impact but there was a notable lack of association with other premorbid, injury-related, demographic, and post-injury factors including litigation status, prior mild head injury, cause of injury, physical pain, prior psychological treatment, age, education, gender, time since injury, duration of PTA, and neuropsychological test scores. However, this study is notably different from others in that it assessed the subjective impact of PCS rather than the presence of PCS.

External locus of control has been indirectly suggested as a factor in the study of persistent PCS but has not received much attention. The study by Rutherford and
colleagues (1977) found that among mild head injury patients six months post-injury those who blamed their employer or some large impersonal body for their injury were more likely to have symptoms than those who blamed themselves. While not specifically examining PCS, Moore, Stambrook, and Wilson (1991) attempted to identify factors associated with good psychosocial recovery regardless of head injury severity. These authors suggested that cognitive factors such as locus of control may be important in recovery from head injury. They noted that multiple studies have shown that locus of control is important in adjustment to a variety of chronic illness and that there is much evidence indicating that poorer outcomes are related to external locus of control while better outcomes are related to internal locus of control in a number of diseases. These researchers evaluated locus of control and outcome in moderate to severe head injury patients approximately six years post-injury. These authors used a locus of control scale that scored onto one internal and two external locus of control factors; the external factors consisted of powerful others (outside authority such as God, the government, etc.) and chance (luck, fate). There were no significant difference between moderate and severely injured patients in locus of control beliefs. Locus of control beliefs were significantly related to outcome, however, even when injury severity and education were controlled for. Specifically, higher chance external locus of control and lower internal locus of control were associated poorer outcome, i.e., greater overall mood disturbance and greater physical difficulties. They suggest that these results indicate that locus of control beliefs may be associated with quality of life independent of factors related to injury severity and educational level. Further research by Moore and Stambrook (1992)
examined coping strategies and locus of control as related to long-term outcome in mild to severe head injury patients. The results suggested that patients with high external locus of control (powerful others and/or chance) tended to use significantly less self-controlling and positive reappraisal coping strategies and had poorer outcome than patients with high internal locus of control. Finally, a recent study by this group (Lubrosko, Moore, Stambrook, & Gill, 1994) examining locus of control beliefs and employment status following severe head injury found that low internal and high powerful others external locus of control beliefs were associated with a reduced level of employment following injury.

In summary, a number of neurocognitive, psychosocial, and premorbid/injury-related variables have been implicated in the development of persistent PCS but the findings among the various research studies have not been consistent. Methodological differences may be partly responsible for this lack of consistency.
PURPOSE OF THE STUDY

From the research reviewed it is clear that persistent PCS do occur in a large number of individuals following mild head injury. The accurate delineation of which variables are predictive of the development of chronic PCS has not yet occurred. A number of variables have been suggested and it is likely that multiple factors come into play. Determining variables that influence the development of persisting PCS is important for identifying those at risk for chronic PCS following mild head injury and subsequently for tailoring intervention strategies towards these groups. Given this information, the present study attempted to identify premorbid, injury-related, neurocognitive, and psychosocial factors associated with persistent postconcussion symptomatology among mildly head-injured college students. The variables chosen for study were those implicated in previous research. The premorbid and injury-related variables of gender, history of previous head injury, and history of premorbid psychological problems were examined. A number of studies have suggested that these variables are associated with PCS. Age has also been implicated in chronic PCS in some studies. However, age is not relevant in the present study as the population evaluated was college students and the range of ages was restricted. Neurocognitive deficits have also been suggested to be related to continued PCS. Specifically, decreased information processing, as manifested by deficits on measures of focused and selective attention, have been demonstrated following mild head injury and implicated in the maintenance of PCS. Information processing capacity was therefore studied. Finally, current level of
psychological distress and locus of control were included. Both of these factors have been suggested as possibly important in PCS but studies have been limited.

The present study sought to address the following research questions. First are premorbid/injury-related (gender, history of previous head injury, and previous history of psychological problems), neurocognitive (information processing, specifically focused and selective attention), and psychosocial (psychological distress and locus of control) factors associated with or predictive of persistent postconcussion symptomatology? It was hypothesized that female gender, previous history of mild head injury (MHI), previous history of psychological disturbance, decreased information processing, psychological distress, and high external locus of control, specifically high powerful others/chance external locus of control, would account for a significant proportion of the variance in postconcussion symptomatology. Because of the inconsistency noted in the literature regarding PCS-related factors, no hypothesis was made concerning which variables would be the most powerful predictors.

It has been suggested that some PCS may be more related to mild head injury and its effects while others may be more related to situational or psychological factors. Specifically, the more physical/cognitive PCS such as dizziness, memory problems, etc., have been suggested to relate to the neurocognitive effects of mild head injury while the more nonspecific emotional/behavioral PCS such as anxiety, etc., have been suggested to relate to psychological functioning (Fox, et al., 1995a, 1995b). In addition, the recent findings by this investigator and colleagues (Ryan, et al., 1996) on the intensification of PCS in head-injured and control subjects suggests that some PCS may be more
situational-emotional/stress-related while others are not. Specifically, the symptoms of irritability, fatigue, noise sensitivity, difficulty concentrating, and anxiety may be more psychologically based while the symptoms of dizziness, headache, memory problems, visual disturbances, and judgement problems may be more neurologically/physiologically based. To test this hypothesis PCS were divided into a physical/cognitive factor and emotional/behavioral factor. These two PCS factors were rationally developed based on previous findings in the literature. The physical/cognitive factor consists of the symptoms of dizziness, headache, memory problems, visual disturbances, and judgement problems while the emotional/behavioral factor consists of the symptoms of irritability, fatigue, noise sensitivity, difficulty concentrating, and anxiety. It was hypothesized that neurocognitive and injury-related variables (decreased information processing, prior history of head injury) and female gender but not premorbid psychological disturbance or psychosocial variables (current psychological distress, external locus of control) would account for a significant proportion of the variance in PCS physical/cognitive scores while premorbid psychological, psychosocial variables, and gender but not neuropsychological and injury related variables would account for a significant proportion of the variance in PCS emotional/behavioral scores.

Finally, it has been suggested that high PCS reporters demonstrate greater cognitive impairment and more psychological symptoms than low PCS reporters (Bohnen et al., 1992, 1995; Gfeller, et al., 1994) and this hypothesis was also tested. Subjects were divided into high and low PCS reporters and it was hypothesized that high PCS subjects would demonstrate poorer information processing performance, higher
psychological distress, and greater external (powerful others and chance) locus of control than low PCS subjects.
METHOD

Participants

Participants were 173 undergraduate student volunteers who reported having sustained one or more mild head injuries. A total of 214 volunteers were recruited from the Baton Rouge campus of Louisiana State University from psychology courses and via sign-up sheets. Volunteers received psychology course credit for their participation. Forty-one participants (19 females/22 males) were later excluded from analysis which left a total sample of 173. All subjects were required to meet the criteria for mild head injury established by the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (Kay, et al., 1993). Based on this criteria, 9 subjects were excluded because they did not sustain any head injury and 10 subjects were excluded because they had sustained a moderate or severe head injury. Given that symptom complaints acutely following injury have not been found to be related to persisting symptom complaints at six weeks or more following injury (Alves, et al., 1993; Fenton, et al., 1993) only subjects who sustained head injuries more than six weeks prior to testing were included. Two subjects were excluded because they had sustained head injuries less than six weeks prior to testing. The other 20 subjects were excluded because they failed to complete necessary information. Eleven subjects failed to complete the PCSC and nine subjects failed to provide information about the severity of head injury or time of injury.

The sample of 173 undergraduate participants consisted of 102 females and 71 males. The sample was predominantly Caucasian (149 Caucasian, 18 African-American,
6 other) which is generally consistent with total university population. Mean age was 20.25 (SD = 2.19) with a range of 17 to 34. Mean education was 14.65 (SD = .97) with a range of 13 (freshmen) to 16 (seniors). Most subjects reported having sustained only one mild head injury (n = 106) and most reported lengths of loss of consciousness of under ten minutes (n = 160). In addition, the majority of subjects were not hospitalized for their head injuries (n = 125). The most frequently reported cause of injury was from sporting accidents (n = 67), then falls (n = 53), followed by motor vehicle accidents (n = 40), assaults (n = 7) and other (n = 6). These results are consistent with our previous findings in a college sample (Ryan, et al., 1996). Also, most subjects denied a history of premorbid psychological problems (n = 158).

**Materials**

**Postconcussion Symptoms Measure**

Postconcussion symptoms were measured with the Postconcussion Syndrome Checklist (PCSC) developed by Gouvier, et al. (1992), which is presented in Appendix A. The PCSC consists of self-ratings for frequency, intensity, and duration of the nine most commonly reported PCS: headaches, dizziness, irritability, memory problems, decreased concentration, visual disturbance, sensitivity to noise, judgement problems, and anxiety. Symptom frequency, intensity, and duration are rated on a five point Likert-type scale from 1 “being not at all” to 5 being “all the time,” “crippling,” or “constant.” Scores for frequency, intensity, duration, and a total score are obtained by summing the ratings for each item. This scale has been demonstrated by the authors to be a valid measure of postconcussion symptoms. For the total score significant correlations with a
measure of common head injury sequelae (Postconcussion Checklist; Oddy, Humphrey, & Uttley, 1978) ranged from .73 to .79 indicating good construct validity. In addition, Gouvier, et al. (1992) demonstrated that the total score reliably differentiated head-injured subjects complaining of postconcussion symptoms from non-head-injured controls; overall 64% of subjects were correctly classified into the appropriate groups by PCSC score. In addition to the total PCSC score, a cognitive/physical factor score consisting of the sum of the frequency, intensity, and duration ratings for the dizziness, headache, memory problems, visual disturbances, and judgement problems items was calculated, as well as a emotional/behavioral factor score consisting of the sum of the frequency, intensity, and duration ratings for the of irritability, fatigue, noise sensitivity, difficulty concentrating, and anxiety items were computed.

Premorbid/Injury-related Information

A neurological screening questionnaire (presented in Appendix B) developed by Ryan, et al. (1996) was used to obtain information on basic demographics, head injury variables, and premorbid head injury and psychological disturbance history. Premorbid history of psychological disturbance was operationally defined as preexisting psychological problems for which professional treatment was obtained.

Neurocognitive Measures

The Symbol Digit Modalities Test (SDMT; Smith, 1982) was one of the measures used to assess information processing. The SDMT is a symbol substitution task which requires subjects to substitute numbers (one through nine) for geometric symbols within 90 seconds. The test can be administered in a written format or oral
format. A total correct score is calculated by summing the number of correct substitutions made. This task has been found to involve a number of components of information processing particularly focused attention (Laux & Lane, 1985; Lezak, 1995). Test-retest reliability for both formats has been good with .80 for the written and .76 for the oral version. In addition, the test has been found to be sensitive to the reduced processing speed commonly seen following head injury (Smith, 1982).

The Ruff 2 and 7 Selective Attention Test (Ruff, Evans, & Light, 1986; Ruff, Niemann, Allen, Farrow, & Wylie, 1992), presented in Appendix C, is a cancellation task involving visual selective attention. Subjects are required to cross out all the two’s and seven’s from among either a group of numbers or letter. There are a total of 20 trials (ten number and ten letter) with 15 seconds allotted per trial. The total number of correctly canceled two’s and seven’s and the total number of errors (misses and incorrect cancellations) is calculated and the final score consists of the total number of correct minus the total number of errors. Test-retest reliability was found to be in the .84 to .97 range (Ruff, et al., 1986). This test has been shown to be sensitive to differential cerebral dysfunction including frontal lobe lesions, right hemisphere lesions, AIDS, and diffuse head injury (Lezak, 1995; Ruff, et al., 1992).

Psychosocial Measures

The Profile of Mood States (POMS) developed by McNair, Lorr, and Droppleman (1992) was used to assess current level of psychological distress. The POMS consists of 65 adjective items on which subjects rate themselves on a five point Likert-type scale from 0 “not at all,” to 4 “extremely.” This tests yields a global distress
score (Total Mood Disturbance) as well as six subscale scores. The Total Mood Disturbance score was used as the measure of overall psychological distress. Internal consistency estimates have been found to be high, ranging from .84 to .95. Test-retest reliabilities have been found to be moderately high, ranging from .65 to .74.

Locus of control beliefs were measured utilizing the Revised Internal-External Scale (RIES) developed by Levenson (1974) which is presented in Appendix D. The RIES consists of 24 items making up three subscales tapping internal control (I), powerful others external control (P), and chance external control (C). Subjects rate the degree of agreement with each item statement on a six point Likert-type scale from 1 “strongly agree” to 6 “strongly disagree.” Internal-consistency was moderately high ranging from .64 for the I scale to .77 for the P scale and .78 for the C scale. Test-retest reliabilities similarly ranged from .64, .74, and .78 respectively (Levenson, 1974). Greater powerful others and chance external locus of control beliefs have been found to be associated with poorer outcome following head injury (Moore, et al., 1991; Stambrook & Moore, 1992).

Procedures

Subjects were administered the questionnaires, SDMT, and Ruff 2 and 7 Selective Attention Test in a group format. At the time of testing all subjects completed an informed consent form which is presented in Appendix E.

Data Analysis

A power analysis was conducted to estimate of the number of subjects required for sufficient power in the proposed analyses utilizing nine variables (Cohen & Cohen,
The power analysis indicated that a minimum of 115 subjects would be needed for a medium effect size at a power of .80 and a significance level of .05. Statistical analyses were performed utilizing Systat for Windows, Version 5 computer software (Systat, Inc., 1992). To test the main research hypothesis, i.e., that female gender, previous history of mild head injury (MHI), previous history of psychological disturbance, decreased information processing, psychological distress, and high external locus of control, specifically high powerful others/chance external locus of control, would account for a significant proportion of the variance in postconcussion symptomatology, a forward selection stepwise multiple regression analysis was conducted in which premorbid and injury-related variables, neurocognitive variables, and psychosocial variables were entered.

For the next hypothesis that neurocognitive and injury-related variables (decreased information processing, prior history of head injury) and female gender but not premorbid psychological disturbance or psychosocial variables (current psychological distress, external locus of control) would account for a significant proportion of the variance in PCS physical/cognitive scores while premorbid psychological, psychosocial variables, and gender but not neuropsychological and injury related variables would account for a significant proportion of the variance in PCS emotional/behavioral scores, two hierarchical multiple regression analyses were performed (Cohen & Cohen, 1983). In the first analysis, physical/cognitive PCS total was the dependent variable and the neurocognitive and injury-related variables were entered, followed by gender, and then followed by psychosocial, and premorbid psychological variables. In the next analysis,
the PCS emotional/behavioral total was the dependent variable and the psychosocial and premorbid psychological variables were entered followed by gender and then the neurocognitive and injury-related variables.

Finally, to test the third hypothesis that high PCS subjects would demonstrate poorer information processing performance, higher psychological distress, and greater external (powerful others and chance) locus of control than low PCS subjects, subjects were divided into low and high PCS reporter groups by taking the top and bottom third of scores on the PCSC. A MANOVA and chi-square analyses comparing high and low PCS reporters on the neurocognitive, premorbid, and psychosocial variables were conducted.
RESULTS

Hypothesis One

It was hypothesized that female gender, previous history of mild head injury (MHI), previous history of psychological disturbance, decreased information processing, psychological distress, and high external locus of control would be predictive of PCS. The forward selection stepwise analysis revealed that a combination of psychological distress (POMS total score), female gender, powerful others external locus of control (RIES-P), and decreased information processing, specifically selective attention (Ruff 2 and 7 score) accounted for a significant proportion of the variance (37%) in postconcussive symptomatology (PCSC total score) therefore partially supporting the first hypothesis. Examination of squared semipartial correlations indicated that the POMS total score accounted for the largest proportion of variance (25%), followed by gender (9%), then powerful others external locus of control (2%), and then selective attention (1%). All of the variables were significantly related to the PCSC total score except for selective attention which showed only a trend towards significance. The first table displays the results of the stepwise regression including the multiple correlation coefficients ($R$), the correlations of multiple determination ($R^2$), the standardized regression coefficients ($\beta$) or beta weights, the squared semipartial correlations ($r_{sp}^2$), and significance levels.

None of the other variables, prior history of head injury, SDMT total score, prior history of psychological disturbance, chance external locus of control (RIES-C), or internal locus of control (RIES-I), entered into the regression equation.
Table 1

Stepwise Multiple Regression of PCSC Predictors

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\beta$</th>
<th>$R$</th>
<th>$R^2$</th>
<th>$r^2_{np}$</th>
<th>$F$</th>
<th>$p$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>POMS Total</td>
<td>.496</td>
<td>.496</td>
<td>.246</td>
<td>.246</td>
<td>55.88</td>
<td>.0001</td>
</tr>
<tr>
<td>Gender</td>
<td>-.306</td>
<td>.582</td>
<td>.339</td>
<td>.093</td>
<td>23.92</td>
<td>.0001</td>
</tr>
<tr>
<td>RIES-P</td>
<td>.128</td>
<td>.596</td>
<td>.355</td>
<td>.016</td>
<td>4.149</td>
<td>.043</td>
</tr>
<tr>
<td>Ruff 2&amp;7</td>
<td>-.105</td>
<td>.605</td>
<td>.366</td>
<td>.011</td>
<td>2.86</td>
<td>.093</td>
</tr>
</tbody>
</table>

Note: Variables are listed in order of entrance into the stepwise regression analysis.

Given the influence of gender noted in the regression analysis, MANOVA and chi-square analyses were conducted comparing females and males on PCSC total, POMS total, RIES-P, I, C, Ruff 2 and 7, SDMT, number of head injuries, and premorbid history of psychological disturbance. The MANOVA revealed a significant group difference, Wilks' $\Lambda = .866$, $F(4, 168) = 6.507$, $p<.0001$. Follow-up univariate ANOVA's revealed a significant difference only for PCSC total score, $F(1, 171) = 22.247$, $p<.0001$, in which females ($M = 71.66$, $SD = 16.28$) had significantly higher scores than males ($M = 60.03$, $SD = 15.46$). Females and males were not significantly different on any other
measures including the POMS total score (females $M = 47.51$, $SD = 37.04$; males $M = 42.41$, $SD = 33.32$).

**Hypothesis Two**

It was hypothesized that neurocognitive and injury-related variables and female gender but not premorbid psychological disturbance or psychosocial variables would be predictive of cognitive/physical PCS while premorbid psychological, psychosocial variables, and gender but not neurocognitive and injury related variables would be predictive of emotional/behavioral PCS. In the first analysis, a hierarchial multiple regression of variables for PCSC physical/cognitive total was performed in which the neurocognitive (Ruff 2 and 7, SDMT) and injury-related (previous history of mild head injury) variables were entered first, then gender, and then psychosocial (POMS total, RIES-P, I, C) variables and premorbid history of psychological disturbance. The analysis indicated that a combination of female gender, psychological distress (POMS total), and decreased selective attention (Ruff 2 and 7) accounted for a significant proportion of the variance (39%) in PCSC physical/cognitive scores. Examination of the squared semipartial correlations revealed that the largest proportion of variance was accounted for by gender (23%) followed by POMS total score (15%), and Ruff 2 and 7 score (1%). Gender and POMS total score were significantly related to PCSC physical/cognitive scores but the Ruff 2 and 7 score was not (see Table 2).
Table 2

Hierarchical Multiple Regression of PCSC Physical/Cognitive Predictors

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\beta$</th>
<th>R</th>
<th>$R^2$</th>
<th>$R^2_{np}$</th>
<th>F</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ruff 2&amp;7</td>
<td>-.102</td>
<td>.104</td>
<td>.011</td>
<td>.011</td>
<td>4.40</td>
<td>.230</td>
</tr>
<tr>
<td>SDMT</td>
<td>-.072</td>
<td>.137</td>
<td>.019</td>
<td>.008</td>
<td>0.71</td>
<td>.402</td>
</tr>
<tr>
<td>Prior MHI</td>
<td>-.026</td>
<td>.139</td>
<td>.019</td>
<td>.000</td>
<td>0.11</td>
<td>.736</td>
</tr>
<tr>
<td>Gender</td>
<td>-.404</td>
<td>.502</td>
<td>.252</td>
<td>.233</td>
<td>25.51</td>
<td>.0001</td>
</tr>
<tr>
<td>POMS Total</td>
<td>.382</td>
<td>.635</td>
<td>.403</td>
<td>.151</td>
<td>19.38</td>
<td>.0001</td>
</tr>
<tr>
<td>RIES-P</td>
<td>.072</td>
<td>.641</td>
<td>.411</td>
<td>.008</td>
<td>0.54</td>
<td>.463</td>
</tr>
<tr>
<td>RIES-I</td>
<td>-.084</td>
<td>.646</td>
<td>.418</td>
<td>.000</td>
<td>1.15</td>
<td>.285</td>
</tr>
<tr>
<td>RIES-C</td>
<td>.008</td>
<td>.646</td>
<td>.418</td>
<td>.000</td>
<td>0.01</td>
<td>.941</td>
</tr>
<tr>
<td>Psych. Hx.</td>
<td>.068</td>
<td>.650</td>
<td>.422</td>
<td>.004</td>
<td>0.80</td>
<td>.374</td>
</tr>
</tbody>
</table>

Note. Variables are listed in order of entrance into the hierarchical multiple regression analysis. Prior MHI = prior history of mild head injury; Psych. Hx. = premorbid history of psychological disturbance.

In the second analysis, a hierarchical regression of variables for PCSC emotional/behavioral total was performed in which the psychosocial variables (POMS total, RIES-P, I, C) and premorbid history of psychological disturbance were entered, followed by gender, then the neurocognitive variables (Ruff 2 and 7, SDMT) and history of previous mild head injury. The results of the analysis showed that a combination of
psychological distress (POMS total) and female gender accounted for a significant proportion of variance (41%) in PCSC emotional/behavioral scores. Examination of the squared semipartial correlations revealed that the POMS total score accounted for 35% of the variance and gender accounted for 6% of the variance. Both variables were significantly related to PCSC emotional/behavioral total (see Table 3).

Table 3
Hierarchical Multiple Regression of PCSC Emotional/Behavioral Predictors

<table>
<thead>
<tr>
<th>Variable</th>
<th>β</th>
<th>R</th>
<th>$R^2$</th>
<th>$r^2_{rp}$</th>
<th>F</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>POMS Total</td>
<td>.546</td>
<td>.593</td>
<td>.352</td>
<td>.352</td>
<td>39.27</td>
<td>.0001</td>
</tr>
<tr>
<td>RIES-P</td>
<td>.038</td>
<td>.595</td>
<td>.354</td>
<td>.002</td>
<td>0.15</td>
<td>.696</td>
</tr>
<tr>
<td>RIES-I</td>
<td>.052</td>
<td>.595</td>
<td>.354</td>
<td>.000</td>
<td>0.44</td>
<td>.507</td>
</tr>
<tr>
<td>RIES-C</td>
<td>.030</td>
<td>.596</td>
<td>.355</td>
<td>.001</td>
<td>0.08</td>
<td>.780</td>
</tr>
<tr>
<td>Psych. Hx.</td>
<td>.027</td>
<td>.596</td>
<td>.355</td>
<td>.000</td>
<td>0.13</td>
<td>.722</td>
</tr>
<tr>
<td>Gender</td>
<td>-.233</td>
<td>.642</td>
<td>.412</td>
<td>.057</td>
<td>8.40</td>
<td>.005</td>
</tr>
<tr>
<td>Ruff 2 &amp; 7</td>
<td>-.027</td>
<td>.643</td>
<td>.414</td>
<td>.002</td>
<td>0.73</td>
<td>.396</td>
</tr>
<tr>
<td>SDMT</td>
<td>-.064</td>
<td>.645</td>
<td>.416</td>
<td>.002</td>
<td>0.55</td>
<td>.461</td>
</tr>
<tr>
<td>Prior MHI</td>
<td>.050</td>
<td>.647</td>
<td>.418</td>
<td>.002</td>
<td>0.40</td>
<td>.527</td>
</tr>
</tbody>
</table>

Note. Variables are listed in order of entrance into the hierarchical multiple regression analysis. Prior MHI = prior history of mild head injury; Psych. Hx. = premorbid history of psychological disturbance.
The hypothesis that the physical/cognitive symptoms and the emotional/behavioral symptoms would have distinctly different predictors was not supported. While there were some differences in the combination and strength of predictor variables for the two factors, psychological distress and female gender were the best predictors for both.

**Hypothesis Three**

It was hypothesized that high PCS subjects would demonstrate poorer information processing, higher psychological distress, and greater external locus of control than low PCS subjects. In order to examine the difference between high and low postconcussion symptom reporters, the top and bottom third of scores on the PCSC were used. The top one third was the high PCS group (n = 59) and the bottom one third was the low PCS group (n = 57). The mean PCSC total score for the high PCS group was 85.34 (SD = 10.7) while the mean PCSC total score for the low PCS group was 48.84 (SD = 7.0); this difference was statistically significant, F (1, 114) = 464.25, \( p < .0001 \). There were no differences between the groups for age, education, race, number of mild head injuries, or premorbid history of psychological disturbance. There was, however a significant difference for gender, \( \chi^2 (1, N = 116) = 11.80, p < .008 \). There was a greater proportion of females in the high PCS group (46 females/13 males) compared to the low PCS group (22 females/35 males).

The MANOVA indicated a significant group difference, Wilks’ \( \Lambda = 0.189 \), F (9, 106) = 50.44, \( p < .0001 \). Follow-up univariate ANOVA’s revealed that the high PCS subjects had significantly higher POMS total scores, F (1, 114) = 51.25, \( p < .0001 \),
RIES-P scores, $F(1, 114) = 7.37, p < .008$, and RIES-C scores, $F(1, 114) = 14.82, p < .001$, than low PCS subjects (see Table 4).

Table 4
High and Low PCS Reporter Mean Scores

<table>
<thead>
<tr>
<th></th>
<th>High PCS</th>
<th>Low PCS</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>POMS Total</td>
<td>67.07 (SD = 38.98)</td>
<td>24.74 (SD = 22.09)</td>
<td>.0001</td>
</tr>
<tr>
<td>RIES-P</td>
<td>24.51 (SD = 8.08)</td>
<td>20.96 (SD = 5.73)</td>
<td>.008</td>
</tr>
<tr>
<td>RIES-I</td>
<td>35.07 (SD = 4.67)</td>
<td>36.56 (SD = 5.80)</td>
<td>.129</td>
</tr>
<tr>
<td>RIES-C</td>
<td>24.81 (SD = 6.73)</td>
<td>20.39 (SD = 5.58)</td>
<td>.0001</td>
</tr>
<tr>
<td>Ruff 2 &amp; 7</td>
<td>298.72 (SD = 47.22)</td>
<td>297.21 (SD = 47.17)</td>
<td>.863</td>
</tr>
<tr>
<td>SDMT</td>
<td>60.44 (SD = 11.24)</td>
<td>61.81 (SD = 10.49)</td>
<td>.500</td>
</tr>
<tr>
<td>Number of MHI’s</td>
<td>1.51 (SD = 0.60)</td>
<td>1.67 (SD = 0.81)</td>
<td>.233</td>
</tr>
</tbody>
</table>

Note. MHI’s = mild head injuries.

The results of these analyses support the hypothesis that high PCS subjects would demonstrate higher levels of psychological distress and greater external (powerful others and chance) locus of control than low PCS subjects but, these results do not support the prediction that high PCS subjects would demonstrate poorer information processing.
SUMMARY AND CONCLUSIONS

The findings of the present study indicate that a combination of psychosocial, premorbid, and neurocognitive factors are predictive of persistent postconcussion symptomatology. Specifically, the combination of psychological distress, female gender, powerful others external locus of control, and decreased selective attention were predictive of postconcussion symptom scores. However, the most predictive variables were level of psychological distress and female gender, accounting for most of the variance in PCS scores. The other factors, including focused attention, prior history of mild head injury, chance external locus of control, and premorbid history of psychological disturbance were not predictive of persistent PCS.

These results are consistent with other studies showing a positive relationship between post-injury level of psychological distress and report of PCS (Gouvier, et al., 1992; Karzmark, et al., 1995). The greater the self-report of psychological distress the greater the report of PCS. A very recent study by King (1996) examined predictors of persistent PCS among mild to moderately head-injured patients at seven to ten days and three months post-injury and found similar results. The findings demonstrated that a combination of psychological and neurocognitive (e.g., information processing, memory) measures given early on following injury were predictive of later PCS. However, the best predictors of persistent PCS were level of anxiety/depression and stress. Unlike some previous studies (Bohnen, et al., 1995; Fenton, et al., 1993) premorbid history of psychological disturbance was not related to PCS in the present study. One possible reason for this finding may be the fact that the majority of subjects, 91% (n=158), denied
any premorbid psychological problems requiring treatment and therefore this sample was primarily reflective of individuals without such preexisting conditions.

One possible explanation for the strong positive relationship between PCS and level of psychological distress is that PCS are exacerbated by such subjective distress. The results of the study by Gouvier, et al. (1992) is consistent with this idea. They found that the number of PCS reported by both head-injured and normal control subjects covaried with level of subjective stress. Another possible explanation is that subjects experiencing PCS following mild head injury are more vulnerable to psychological stressors. In this sample, higher PCS and thus higher levels of psychological distress cannot simply be explained by a premorbid vulnerability for psychological disturbance as premorbid history of psychological problems was not predictive of PCS and in fact the vast majority of mild head injury subjects denied any such psychological history.

Female gender has also been found to be positively related to persistent PCS in many studies (Alves, et al., 1993; Bohnen, et al., 1994; Fenton, et al., 1993; Packard, et al, 1993; Rutherford, et al., 1977; 1979) as in the present study. While a number of investigators have found this relationship, possible explanations for the relationship have been very limited. Rutherford, et al. (1979) suggested that this female gender bias is due to sex differences in psychological reactions but did not elaborate on this explanation. A consistent finding in the literature is the greater prevalence of affective disorders as well as higher rates of minor physical illness among women. It has been suggested that these difference in symptom rates are the result of a general tendency for women to admit to and/or report more symptoms than men or to have lower thresholds for perceiving
symptoms (MacIntyre, 1993). However, research has failed to support this hypothesis. A study of gender differences in depression varied the number of criterion variables required for the diagnosis of major depressive disorder and demonstrated that women had a greater number of symptoms only at higher symptom levels thus suggesting a true sex difference in the rate of depression rather than a general trend for women to report more symptoms (Young, Fogg, Scheftner, Keller, & Fawcett, 1990).

A large British study examined the differing rates of minor physical morbidity and affective disorders and among males and females and possible hormonal, social, and psychosomatic explanations for this difference (Popay, Bartley, & Owen, 1993). As expected, females did report greater levels of minor physical morbidity and affective disorders than males. These higher rates were not related to problems associated with menstruation or menopause. In addition, while higher symptom rates were related to lower social position, there was no interaction between social status and gender. Also, while higher physical symptom rates were related to higher rates of affective disorders for men and women, the correlation was actually stronger among men. The higher symptom rates for females were not explained by hormonal fluctuations, social status, or affective disorders. The authors suggest that the higher rates of minor physical illness may actually lead to higher rates of affective disorders among women. Another study by MacIntyre (1993) examining gender differences in the perceptions of cold symptoms found that while female subjects were significantly more likely to have cold symptoms as rated by an observer they were no more likely than males to assess themselves as having a cold. In addition, male subjects were significantly more likely to over-rate the severity
of their cold symptoms. The results failed to support the hypothesis that women have a lower threshold for perceiving and reporting minor symptoms.

Further, the incidence of whiplash injuries resulting from motor vehicle accidents has been found to be significantly higher among women with rates 4.8 times higher than men in metropolitan areas and 1.7 times higher in nonmetropolitan areas reported (Schutt & Dohan, 1970). Women also have been found to have higher rates of persistent symptoms following whiplash injury including neck and back pain, headache, dizziness, and tinnitus (Gargan & Bannister, 1990). Recently, Squires, Gargan, and Bannister (1996) reported that 14 to 17 years following whiplash injury, 80% of their female patients continued to have symptoms compared with 50% of their male patients.

The results of the above mentioned studies suggest that there are actual gender differences in the rates of affective and minor physical illnesses rather than just a female reporting bias. In the present study, female gender was predictive of PCS. Examination of gender effects revealed that while women generally had significantly higher PCSC scores than men they did not have higher levels of psychological distress as measured by the POMS. Thus suggesting that higher rates of PCS among women were not simply the result of a tendency to report more symptoms in general which is consistent with literature on affective and minor physical illness. It appears that women may be more susceptible to PCS following mild head injury than men but why this may be so is still unclear.

In the present study, decreased information processing, specifically decreased selective attention, in combination with psychological distress, gender, and powerful
others external locus of control was predictive of PCS. However, selective attention alone was only insignificantly and weakly related to the PCSC scores, accounting for just 1% of the variance in those scores. In addition, high PCS reporters were not significantly different than low PCS reporters on either of the measures of information processing. These results suggest that information processing is not an important factor in the prediction of persistent PCS in this sample of mildly head-injured college students.

A recent study by Karzmark, et al. (1995) similarly failed to find a significant relationship between subjective impact of PCS and several neurocognitive measures, including information processing and memory, among mild head injury patients with persistent symptoms who were seeking evaluation.

Powerful others external locus of control was also predictive of PCS scores when combined with the other variables noted above. Alone, powerful others external locus of control was significantly but only weakly related to PCSC total scores. Also, high PCS reporters had significantly greater powerful others and chance external locus of control scores compared to low PCS reports. Although, while the difference between high and low PCS reporters was statistically significant the size of the difference in means was relatively small. It appears that while external locus of control is related to persistent PCS reporting the relationship is relatively weak.

When PCS were divided into physical/cognitive and emotional/behavioral symptom clusters the results of the analyses were similar to those found utilizing the total PCSC scores with level of psychological distress and female gender being the best predictors for both symptom clusters. The hypothesis that there would be different
predictors for the physical/cognitive cluster relative to the emotional/behavioral cluster was not supported. The two PCS factors were rationally developed based on inferences from the literature. It has been suggested but not empirically validated that the physical/cognitive PCS such as dizziness and memory problems are more related to the neurocognitive effects of mild head injury while the emotional/behavioral PCS such as anxiety are more related to psychological functioning (Fox, et al., 1995a, 1995b; Ryan, et al., 1996). The results of the present study do not support such inference. There were similar predictor variables for both clusters. It appears that dividing traditional PCS into two separate symptom clusters is not useful or even warranted. It is not likely that these findings are just an artifact of the measure of PCS utilized. The PCSC (Gouvier, et al. 1992) assesses the frequency, intensity, and duration of the nine most commonly reported PCS which are the symptoms examined in most other studies regardless of the specific PCS measure used. A study by Bohnen, et al. (1992) examining PCS and premorbid factors utilized a questionnaire made up of two factors, a PCS-cognitive and emotional-vegetative factor. The first factor consisted of the traditional PCS symptoms and symptoms related to decreased work capacity while the second factor consisted of vague, nonspecific complaints such as heart palpations, restlessness, tearfulness, etc. In this study, the traditional PCS clustered together onto one factor thus supporting the idea that PCS should not be divided into cognitive/physical and emotional/behavioral symptom clusters.

Comparison of high and low PCS reporting subjects further supported the results of the regression analyses. The high PCS group had a significantly greater proportion of
females and as predicted, had significantly higher levels of psychological distress and external locus of control (powerful others and chance) than the low PCS group. However, there were no differences between the groups for information processing (selective and focused attention) as had been hypothesized. The similar performance of high and low PCS reporters on measures of information processing conflicts with the results of studies by Bohnen, et al. (1995) and Gfeller, et al. (1994) who found that mild head injury subjects with higher ratings of PCS performed less well on a measures attention/information processing. Such conflicting results may be due to the differing neurocognitive measures used, as well as in subject selection. In the studies just mentioned, subjects selected were mild head injury patients who had been hospitalized following their injuries, who typically sustained injuries in motor vehicle accidents, and who were generally older than college age which is in contrast to the present study of undergraduate students, the majority of whom were not hospitalized and who sustained injuries from sporting events or falls. Thus the subjects in the present study may have sustained more mild injuries than those in the other studies.

In conclusion, the present findings indicate that level of psychological distress and female gender are the best predictors of persistent PCS among mildly head-injured college students. A limitation of the present study was the use of a homogeneous population, i.e., college students. As a result, the findings may not be generalizable to other mild head injury populations. Future research needs to replicate these results as well as cross validate the results in other head-injured populations. In addition, the nature of the relationship between psychological distress, gender, and persistent
postconcussion symptoms needs to be explored further. Given the high base rate of PCS among nonhead-injured individuals and the link with level of psychological distress, further studies need to evaluate the predictors of PCS in nonhead-injured subjects.
REFERENCES


Neuropsychology, 2, 147-155.


APPENDIX A
POSTCONCUSSION SYNDROME CHECKLIST (PCSC)

Subject Number____________________ Date:____________________

Please rate the frequency, intensity, and duration of each of the following symptoms based on how they have affected you during the past week including today according to the following scale:

<table>
<thead>
<tr>
<th>FREQUENCY</th>
<th>INTENSITY</th>
<th>DURATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>1=Not at all</td>
<td>1=Not at all</td>
<td>1=Not at all</td>
</tr>
<tr>
<td>2=Seldom</td>
<td>2=Vaguely present</td>
<td>2=A few seconds</td>
</tr>
<tr>
<td>3=Often</td>
<td>3=Clearly present</td>
<td>3=A few minutes</td>
</tr>
<tr>
<td>4=Very often</td>
<td>4=Interfering</td>
<td>4=A few hours</td>
</tr>
<tr>
<td>5=All the time</td>
<td>5=Crippling</td>
<td>5=Constant</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>FREQUENCY</th>
<th>INTENSITY</th>
<th>DURATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dizziness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Irritability</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Memory Problems</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Difficulty Concentrating</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fatigue</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visual Disturbances</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aggravated by Noise</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Judgement Problems</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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APPENDIX B

NEUROLOGICAL SCREENING

Please answer all questions to the best of your ability. Remember all information is confidential. If you have any questions regarding your answers, please ask the examiner.

Subject Number_________________ Sex: Male___ Female____
Age:__________________________ Race: White____ Black___ Other_______
DOB:_________________________ Handedness: Right___ Left___ Ambidextrous___
Year in College:_________________

What is your current major and GPA? Please specify:_____________________________________
If you are a freshman in your first semester and do not have a current college GPA, please give your high school cumulative GPA here:________________________

Have you ever experienced a head injury. That is have you ever been hit in the head and/or hit your head, and been knocked out (unconscious) or been dazed (seeing stars, disoriented, confused) for several minutes thereafter? Yes___ No___

How many head injuries have you had:_____________________________________
When did each occur? Please specify your approximate age/year for each starting with the most recent:
1)____________________________________ (only/most recent), 2)________________________, 3)________________________, 4)________________________ additional________________________

Were you hospitalized for your head injury(s): Yes___ No___

Use the following scale for the next questions:
0 = None  4 = 1 hour or less (31 minutes to 1 hour)
1 = less than 1 minute (1 to 59 seconds)  5 = 12 hours or less (1 to 12 hours)
2 = less than 10 minutes (1 to 9 minutes)  6 = 24 hours or less (13 to 24 hours)
3 = 30 minutes or less (10 to 30 minutes)  7 = greater than 24 hours

1. Length of hospital stay: 1)__________________ (only/most recent), 2)__________________ 3)__________________ 4)__________________ additional_____________________
2. Duration of unconsciousness: 1)__________________ (only/most recent), 2)__________________ 3)__________________ 4)__________________ additional_____________________
3. After a head injury many people have trouble remembering things/events for a period of time just before and/or just after the injury, rate the duration of memory loss:
Before injury: 1)__________________ (only/most recent), 2)__________________ 3)__________________ 4)__________________ additional_____________________
After injury: 1)__________________ (only/most recent), 2)__________________ 3)__________________
4) Additional

What was the cause(s) of your head injury(s)? Please check one for each injury:

- Motor Vehicle Accident: __________________________________________________________
- Fall: __________________________________________________________
- Sporting Injury: __________________________________________________________
- Assault/Fight: __________________________________________________________
- Other (please explain): __________________________________________________________

Are you experiencing/have you experienced any of the following physical problems/limitations (please check):

- Current
- Past

- Clumsiness:___________________
- Weakness on one side:___________________
- Other (please explain):___________________

Have you ever experienced a seizure of any kind? Yes No

- Age at first seizure:___________
- Age at seizure disorder diagnosis:___________
- Seizure frequency (# per wk or yr):___________

What type of seizure(s) have you had (list if known):

If you have recurring seizures, describe a typical seizure:

Do you take any medication for seizures? If yes please list:

Have you ever experienced a central nervous system (CNS) disease? Yes No

1. When:___________________

2. What type(s) of CNS disease (if known):________________________________________

3. What type of treatment did you receive? Please list if known:

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Have you ever had a stroke?  

Yes____ No____

1. When: ____________________

2. Type of stroke (if known) ____________________________

Have you ever experienced any psychological/emotional problems such as anxiety, depression, etc. for which you received treatment/professional help (therapy/counseling/medication)?  Yes____ No____

Please describe the problem(s) you were treated for, when did it occur, what type of treatment you received:

__________________________________________________________________________________
__________________________________________________________________________________
__________________________________________________________________________________
__________________________________________________________________________________
__________________________________________________________________________________
__________________________________________________________________________________

List any medications you have taken for the above problem(s):

__________________________________________________________________________________
__________________________________________________________________________________
__________________________________________________________________________________
__________________________________________________________________________________
__________________________________________________________________________________

Have you ever received electro-convulsive shock treatment (ECT) for the above problem(s):  Yes____ No____

Have you ever used alcohol?  Yes____ No____

Have you ever used any other drugs?  Yes____ No____

Check all that apply below and list the amount used (# drinks per week or if less than # per month/year, please be sure to specify):

<table>
<thead>
<tr>
<th>Substance</th>
<th>Current/Amount</th>
<th>Past/Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>alcohol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>marijuana/hash</td>
<td></td>
<td></td>
</tr>
<tr>
<td>cocaine/crack</td>
<td></td>
<td></td>
</tr>
<tr>
<td>speed/amphetamines</td>
<td></td>
<td></td>
</tr>
<tr>
<td>barbiturates</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LSD/hallucinogens</td>
<td></td>
<td></td>
</tr>
<tr>
<td>heroine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>other (please specify)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
APPENDIX C

RUFF 2 AND 7 SELECTIVE ATTENTION TEST

<table>
<thead>
<tr>
<th>Name</th>
<th>Age</th>
<th>Sex</th>
<th>Education</th>
<th>Occupation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

![Image of the test page](image-url)

DATE:_____

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APPENDIX D

REVISED INTERNAL-EXTERNAL SCALE (RIES)

Subject Number:_____________________ Date:____________________

Please rate how strongly you agree with the following statements according to the following scale:

1=Strongly Disagree
2=Moderately Disagree
3=Slightly Disagree
4=Slightly Agree
5=Moderately Agree
6=Strongly Agree

_____ 1. Whether or not I get to be a leader depends mostly on my ability.

_____ 2. To a great extent my life is controlled by accidental happenings.

_____ 3. I feel like what happens in my life is mostly determined by powerful others.

_____ 4. Whether or not I get into a car accident depends mostly on how good a driver I am.

_____ 5. When I make plans, I am almost certain to make them work.

_____ 6. Often there is no chance of protecting my personal interest from bad luck happenings.

_____ 7. When I get what I want, it's usually because I'm lucky.

_____ 8. Although I might have good ability, I will not be given leadership responsibility without appealing to those in positions of power.

_____ 9. How many friends I have depends on how nice a person I am.

_____ 10. I have often found that what is going to happen will happen.

_____ 11. My life is chiefly controlled by powerful others.

_____ 12. Whether or not I get into a car accident is mostly a matter of luck.
13. People like myself have very little chance of protecting our personal interests when they conflict with those of strong pressure groups.

14. It's not always wise for me to plan too far ahead because many things turn out to be a matter of good or bad fortune.

15. Getting what I want requires pleasing those people above me.

16. Whether or not I get to be a leader depends on whether I'm lucky enough to be in the right place at the right time.

17. If important people were to decide they didn't like me, I probably wouldn't make many friends.

18. I can pretty much determine what will happen in my life.

19. I am usually able to protect my personal interests.

20. Whether or not I get into a car accident depends mostly on the other driver.

21. When I get what I want, it's usually because I worked hard for it.

22. In order to have my plans work, I make sure that they fit in with the desires of people who have power over me.

23. My life is determined by my own actions.

24. It's chiefly a matter of fate whether or not I have a few friends or many friends.
APPENDIX E

LOUISIANA STATE UNIVERSITY- BATON ROUGE CAMPUS
CONSENT FORM

1. Study Title: Predictors of Postconcussion Symptomatology
2. Study Site: Louisiana State University
3. Investigators: The following investigators are available for questions at the Psychology Department, 388-8745:
   Ms. Laurie M. Ryan
   Dr. Wm. Drew Gouvier
4. Purpose of the Study: By completing the study questionnaires and tasks volunteers will help provide information about the nature of postconcussion symptoms.
5. Subject Inclusion: The study includes undergraduate students who by self-report have sustained a mild head injury more than 6 weeks from the study date.
6. Subject Exclusion: Undergraduate students who have not sustained a mild head injury.
7. Description of Study: Subjects will complete a series of questionnaires and information processing tasks.
8. Benefits: Subjects will not benefit directly but will provide empirical information about the nature of postconcussion symptoms.
9. Risks: There are no risks to the subjects.
10. Alternatives: Not applicable.
11. Removal: Subjects who complete the questionnaire and tasks have fulfilled all the study requirements.
12. Right to Refuse: Subjects may choose NOT to participate or withdraw from the study at any time with no penalty.
13. Privacy: The results of this study may be published. The privacy of participating subjects will be protected and the identity of participants will not be revealed. Subject data will be assigned numbers and subjects will remain anonymous.

14. Release of Information: No information outside of subject data from the study will be reviewed by the investigators.

15. Financial Information: Not applicable.

16. Signatures:

The study has been discussed with me and all my questions have been answered. I understand that additional questions regarding the study should be directed to the investigators listed above. I understand that if I have questions about subject rights, or other concerns, I can contact the Vice Chancellor of the LSU Office of Research and Economic Development at 388-5833. I agree with the terms above and acknowledge that I have been given a copy of the consent form.

_________________________________________  
Signature of Subject Volunteer              Date

_________________________________________  
Witness                                      Date
VITA

Laurie M. Ryan was born in Washington, D.C., on January 28, 1964. She received her bachelor of arts degree in Human Development from St. Mary's College of Maryland in 1986 and her master of arts degree in Clinical Psychology from Loyola College in Maryland in 1991. She completed her internship in clinical psychology at the Medical University of South Carolina/Department of Veteran's Affairs Consortium in August 1997. She received her doctor of philosophy degree in Psychology from Louisiana State University in August 1997.
DOCTORAL EXAMINATION AND DISSERTATION REPORT

Candidate: Laurie M. Ryan

Major Field: Psychology

Title of Dissertation: Predictors of Postconcussion Symptomatology in a Mild Head Injury College Population

Approved:

[Signatures]

Major Professor and Chairman
Dean of the Graduate School

EXAMINING COMMITTEE:

[Signatures]

Date of Examination:

6/24/97