Abstract

This investigation was designed to provide preliminary support for cognitive reserve theory in closed head injury (CHI), and demonstrate the effectiveness of using the Oklahoma premorbid intelligence estimate (OPIE) in research and clinical activities. Out of a possible 124 consecutive referrals, 26 patients ($N = 26$) who underwent neuropsychological assessment following brain injury met study inclusion/exclusion criteria. Participants were included if they had exited post-traumatic amnesia (PTA), demonstrated uncompromised upper extremity use, displayed adequate verbal communication, and were judged capable of completing a full neuropsychological evaluation. Participants were divided into a closed head injury—negative premorbid history (CHI$^-$) or closed head injury—positive premorbid history (CHI$^+$) group based upon premorbid variables (e.g., history of alcoholism). Groups did not differ in terms of demographic variables or premorbid IQ. Despite having less severe head injuries, the CHI$^+$ group had a greater pre–post difference for PIQ, and a significantly larger VIQ/PIQ discrepancy than the CHI$^-$ group. In conclusion, these findings suggest that the CHI$^+$ group had diminished cognitive reserve secondary to the aggregate effects of premorbid insult, which resulted in greater cognitive decline following an additional stressor (i.e., CHI) than what might otherwise be expected from the head injury alone.

Keywords: Closed head injury; Cognitive reserve; Oklahoma premorbid intelligence estimate; Post-traumatic amnesia; Severity of head injury; Premorbid history
In 1993, Paul Satz wrote a seminal paper on cognitive reserve, reviewing all the literature up to 1993 relevant to threshold theory for acquired brain injury. This article addressed the concept of brain reserve capacity (a.k.a., cognitive reserve) as a factor that may explain threshold differences in the expression of clinical symptoms or poorer neurocognitive performance following an acquired brain injury among individuals with similar biological markers of disease. In theory, greater cognitive reserve serves as a protective factor for the development and/or expression of future neurologic conditions, whereas lower cognitive reserve is a vulnerability factor that lowers the threshold for the clinical presentation of symptoms of conditions such as dementing illnesses.

Cognitive reserve can be measured directly through head circumference or brain size, or indirectly via general intelligence, educational level, and occupational attainment. It has been suggested that greater cognitive reserve is achieved with higher education and occupational attainment, as well as higher intelligence as calculated by standardized IQ measures or IQ estimates. Studies have demonstrated that individuals with more education have higher intelligence, greater brain weight, larger neurons, and increased arborization of neurons, as compared to less educated individuals (Katzman et al., 1988; Mortimer, 1997). Furthermore, higher occupational attainment generally requires greater skills and abilities, whereas lower occupational attainment places less demands on individuals’ thinking abilities and can predispose them to specific environmental conditions (e.g., chemicals) known to adversely affect cognitive functioning.

In his article, Satz (1993) proposed that, at that time, no study to date had determined whether the concept of brain reserve capacity might account for individual differences in vulnerability for the onset of neurodegenerative processes. Since 1993, support for cognitive reserve theory has been found in various conditions including Parkinson’s disease (PD), Alzheimer’s disease (AD), schizophrenia, and HIV research. Glatt et al. (1996) compared cognitively intact Parkinson’s patients to Parkinson’s patients with dementia. They found that the most significant predictor of dementia onset in PD was education, and that educational attainment may modify the risk of cognitive decline in PD. These authors concluded, “Educational attainment, independent of dementia etiology, may be due to greater functional brain reserve” (p. 20). Stern and colleagues (Stern, Alexander, Prohovnik, & Mayeux, 1992; Stern et al., 1994; Stern et al., 1995; Stern, Albert, Tang, & Tsai, 1999) have extensively studied cognitive reserve and AD. These authors have consistently found that low educational and occupational attainment are related to increased risk for and prevalence of AD, and that these variables are also related to the rate of memory decline in AD. Alexander and colleagues (1997) investigated the relationship between metabolism in the prefrontal, pre-motor, and left superior parietal association areas and premorbid intellectual abilities, and found an inverse relationship between premorbid IQ and cerebral metabolism in these areas. They concluded, “These findings provide support for a cognitive reserve that can alter the clinical expression of dementia and influence the neurophysiological heterogeneity observed in AD” (p. 165). Dwork et al. (1998) examined the brains of deceased elderly institutionalized psychiatric patients, most of whom had schizophrenia, and compared them to the brains of deceased individuals without neurologic or psychiatric disease for the presence of neuritic senile plaques and neurofibrillary tangles. They also examined these patients’ hospital records for psychometric test results, mental status examination performance, and a diagnosis of dementia. Their findings suggest
that schizophrenia and senile degeneration are “synergistic” and result in cognitive decline, consistent with cognitive reserve theory. Stern, Silva, Chaisson, and Evans (1996) used years of education, occupational attainment, and performance on a measure of premorbid intelligence as indirect measures of cognitive reserve with HIV-1 seropositive and HIV-1 seronegative participants. They found that HIV-1 seropositive participants with low cognitive reserve exhibited greater neuropsychological impairments (i.e., impairment in attention, information processing speed, verbal learning and memory, executive functioning, and visuospatial abilities) as compared to the high cognitive reserve HIV-1 seropositive group. These authors concluded, “As has been found in other neurologic disorders, such as AD, individuals with greater cognitive reserve may be less sensitive to the initial clinical effects of the underlying neuropathologic process” (p. 148). Basso and Bornstein (2000) conducted a 12-month study examining cognitive reserve in homosexual males who were HIV+ asymptomatic, HIV+ symptomatic, or healthy participants using indirect measures of cognitive reserve including education, occupational attainment, and an estimate of premorbid intelligence based on demographic variables. These authors found, regardless of further cerebral insult resultant from HIV, that greater cognitive dysfunction occurred in those with lower levels of cognitive reserve, and that greater cognitive reserve helped mediate neurobehavioral declines across time. Overall, to date, a literature review has revealed support for cognitive reserve theory in these various conditions. Though there has also been some theoretical support and case studies on cognitive reserve theory with head injury patients (e.g., Lye & Shores, 2000; Rudelli, Strom, Welch, & Ambler, 1982), no empirical research was found that directly examined cognitive reserve theory and closed head injury (CHI).

The purposes of the present investigation were to empirically examine the aggregate effects of premorbid history variables and head injury on cognitive reserve, and demonstrate the effectiveness of using the Oklahoma premorbid intelligence estimate (OPIE) in cognitive reserve theory research. The OPIE formulas are standardized, validated, reliable linear prediction algorithms developed from the WAIS-R standardization sample with demonstrated utility with normal and neurologically impaired populations (Krull, Scott, & Sherer, 1995; Ropacki & Elias, 1999; Scott, Krull, Williamson, Adams, & Iverson, 1997; Spreen & Strauss, 1998). Krull et al. (1995) took the original WAIS-R standardization sample, divided it randomly—half being used to derive the OPIE equations and the other half used to validate these equations—and found that the OPIE formulas produced distributions of predicted scores that closely approximated the means and standard deviations of the original WAIS-R standardization sample. The OPIE formulas combine an individual’s demographic information with their performance on WAIS-R subtests (i.e., Vocabulary and Picture Completion) that have been empirically demonstrated to be unaffected by central nervous system changes. Basso, Bornstein, Roper, and McCoy (2000) have spoken against the use of premorbid IQ estimates because of their high standard error of the estimate. However, these authors found that “the OPIE generally yielded the smallest SEEs” (p. 338) and they acknowledge that previous research “has generally shown that each (premorbid IQ estimation) method is reasonably accurate in estimating mean WAIS-R IQ scores for groups of individuals” (p. 336). Given these considerations, and the fact that Basso and Bornstein (2000) themselves have used a premorbid intelligence estimate when looking at cognitive reserve and HIV, the use of the OPIE was deemed appropriate for use as a measure of premorbid intelligence in the present study.
This investigation is unique because it separated groups based upon premorbid history variables such as alcoholism, drug abuse, psychiatric history and/or previous neurologic insult (e.g., cerebrovascular accident, head injury, etc.) in order to differentiate the effects of head injury from the aggregate effects of head injury and other insults on individuals’ cognitive reserve. To accomplish this, two groups were created. One group, a closed head injury—positive premorbid history group (CHI+), was comprised of individuals with a history positive for any or all the aforementioned premorbid history variables. The other group, a closed head injury—negative premorbid history group (CHI−), contained individuals free from any of these premorbid conditions. These groups should not differ statistically on variables that serve as indirect measures of cognitive reserve such as education, occupational attainment, and/or premorbid IQ. Furthermore, they should not differ statistically on other variables known to detrimentally affect cognitive performance (i.e., age). If age is found to be significantly different by group due to chance, then any age-effect could be well controlled through the use of age-corrected norms or statistical analyses (e.g., analysis of covariance). With demographic variables and indirect measures of cognitive reserve relatively equal, it is hypothesized that the CHI− group will have greater cognitive reserve that serves as a protective factor against loss of cognitive abilities from pre-to-post-head injury, whereas the CHI+ group will have diminished cognitive reserve and thus greater vulnerability to the effects of the head injury, as reflected in greater loss from pre-to-post-injury on standard measures of neurocognitive functioning.

1. Method

1.1. Participants

One hundred and twenty-four participants were referred for a neuropsychological assessment from various hospitals (i.e., Methodist Hospital, St. Mary’s Hospital, and University Medical Center) in the Lubbock, Texas area following brain insult and evaluated by the first author as part of his dissertation project. Although the first author completed all of the neuropsychological evaluations, by the dissertation committee’s design and to avoid potential confounding of the findings, the first author had no a priori knowledge of who would ultimately meet study inclusion/exclusion criteria at the time of examination. The first author was also not allowed to examine, code, or enter any of the data into a dataset, until a sufficiently large enough number of participants was collected. Upon examination of the archived data, 26 consecutive patients (N = 26), 17 males and 9 females (see Table 1), sustained a closed head injury, completed a full battery assessment, and met strict inclusion/exclusion criteria. Participants were included if they had uncompromised use of their upper extremities as determined during clinical interview (i.e., a brief motor screen was completed), were able to verbally communicate, had exited post-traumatic amnesia (PTA), and were judged capable of completing a full length neuropsychological evaluation. All participants were tested after their transition to inpatient rehabilitation or as soon as possible after their discharge from the hospital before beginning outpatient rehabilitation. At the time of the evaluations, no patients were involved in litigation related to their head injury or pursuing either a worker’s compensation
or social security disability claim, and all evaluations were completed solely for rehabilitation purposes.

Participants in the CHI− group (n = 17) were excluded for a history of alcoholism, drug abuse, head trauma, psychiatric problems, or neurologic disorder. Moreover, all participants’ charts were examined (e.g., history and physical, lab results, physician notes) to ensure that there was no confounding of assessment findings due to complications resulting from alcohol or drug use at the time of their head injury. The CHI− group had a mean age of 42.94 years (S.D. = 23.57; range 18–86), with an average of 12.06 years of education (S.D. = 3.01; range 5–16). As illustrated in Table 2, participants in the CHI+ group (n = 9) had a history of one or more of these variables. The CHI+ group’s mean age was 62.11 (S.D. = 25.03; range 19–90), with an average of 12.56 years of education (S.D. = 3.47; range 7–16). Despite the difference in mean age by group, a comparison failed to reveal a statistically significant group difference in age F(1, 25) = 3.73, P = .065 (d = .375; power = .469). It should also be kept in mind that all analyses on measures from this investigation were completed using age, education, and, where appropriate, gender corrected standard scores. Furthermore,
Table 2
Breakdown of premorbid history variables for the CHI+ group

<table>
<thead>
<tr>
<th>Subject #</th>
<th>Mental illness</th>
<th>Alcoholism</th>
<th>Neurologic</th>
<th>Drug abuse</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>+</td>
<td>–</td>
<td>+</td>
<td>–</td>
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<tr>
<td>2</td>
<td>–</td>
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<td>–</td>
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<td>+</td>
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<tr>
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<td>+</td>
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<tr>
<td>5</td>
<td>–</td>
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<td>8</td>
<td>–</td>
<td>–</td>
<td>+</td>
<td>–</td>
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<tr>
<td>9</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>–</td>
</tr>
</tbody>
</table>

a Depression.
b Obsessive-compulsive disorder.
c Generalized anxiety disorder.

correlational analyses were completed to ensure there were no age-effects on the standardized variables of interest that significantly differed by group. All correlations between age and these age-corrected standardized variables were not significant (all \( P \)'s > .10). Therefore, this circumvented the need to statistically control for age (i.e., analysis of covariance) in any subsequent analyses. Finally, the also groups did not differ on level of occupational attainment \( \chi^2(5, N = 26) = 3.59, P = .609 \).

Severity level, based on the Glasgow Coma Scale (GCS), was retrieved from hospital records. Duration of PTA was determined through the course of clinical interview questioning (i.e., last memory prior to incident, first memory following the incident, and assessment for return of continuous day-to-day memory), an accepted, reliable, and valid method for assessing PTA (McMillan, Jongen, & Greenwood, 1996). Furthermore, a check to ensure that the patients’ PTA had remitted and that they were suitable for and able to withstand a neuropsychological evaluation was completed by the first author. Participants were judged to have exited PTA from the results of their mental status examination, memory surrounding the incident, combined with an assessment to ensure continuous memory for day-to-day events had returned. All participants were judged to have cleared PTA before neuropsychological evaluation.

Due to heterogeneity of variance, a Kruskal–Wallis test was used to compare PTA duration and GCS scores by group. Statistically, the CHI– and CHI+ groups were not significantly different by GCS score (\( P = .068 \)) or duration of PTA (\( P = .316 \)). The CHI– group had a mean GCS score of 9.41 (S.D. = 5.34) and mean PTA duration of 365.82 hours (S.D. = 506.22). The CHI+ group mean GCS score was 13.56 (S.D. = 2.79) and mean PTA duration 105.67 hours (S.D. = 185.09). According to the GCS severity classification system in Lezak (1995), the GCS severity levels by group were as follows: the CHI– group had 7 mild, 2 moderate, and 8 severe participants; the CHI+ group had 7 mild, 1 moderate, and 1 severe participant. Severity levels by group according to the PTA severity classification system (Lezak, 1995) were as follows: the CHI– group had 5 mild, 2 moderate, and 10 severe participants; the CHI+ group had 3 mild, 3 moderate, and 3 severe participants. Although not reflected statistically, the
CHI− group had more severely impaired individuals according to the GCS and duration of PTA severity classification systems. An artifact of increasingly severe injuries is the length of time between injury and assessment; more severe injuries take longer to clear the acute phase of injury before a reliable assessment can be completed. Nevertheless, despite the CHI− group’s greater number of severe injuries according to severity classification systems, the average time from injury to assessment was not significantly different by group $F(1, 25) = .737, P = .400$.

1.2. Procedure

As noted above, participants’ severity level GCS scores were retrieved from hospital records and PTA duration was ascertained according to the guidelines proposed by McMillan et al. (1996). All participants were administered the Wechsler Adult Intelligence Scale-Revised (WAIS-R), Wechsler Memory Scale/Wechsler Memory Scale-Revised (WMS/WMS-R), Stroop Color–Word Test, and Trail Making Test (A & B) as part of a comprehensive neuropsychological evaluation. Standardized administration and scoring practices were used, and all data analyzed in this investigation was standardized using age, education, and, when available, gender corrected norms. Premorbid intelligence was estimated using standardized procedures for obtaining the Best Full Scale IQ, Verbal IQ, and Performance IQ estimates with the OPIE formulas, as described in Scott et al. (1997).

2. Results

The calculation of OPIE premorbid IQ scores permitted a unique analysis for the IQ measures. One-way ANOVA comparisons of the various OPIE premorbid indices (i.e., OPIE-FSIQ, OPIE-VIQ, and OPIE-PIQ) by group (i.e., CHI− and CHI+) were not statistically different, as expected. Comparison of OPIE VIQ/PIQ discrepancy by group was also not significant $F(1, 24) = .027, P = .871$, as expected. This lack of difference in the pre-injury OPIE scores is why the main effect for group, which is a combination of pre- and post-injury scores, is underestimated in this design and not interpretable (Huck & McLean, 1975). Therefore, a group (i.e., CHI−, CHI+) by time (i.e., pre-, post-test) analysis was conducted with the OPIE scores as premorbid IQ scores and the FSIQ, VIQ, and PIQ scores as the post-measures. In this analysis, the group by pre–post interaction is the same as the main effect for a change

| Table 3 |
|------------------|------------------|
Table 4
Mean difference between obtained and predicted IQ’s using the OPIE best

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>FSIQ Obtained</th>
<th>FSIQ Predicted</th>
<th>Difference</th>
<th>F</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHI−</td>
<td>17</td>
<td>87.38 (14.85)</td>
<td>97.11 (14.88)</td>
<td>9.73</td>
<td>19.45</td>
<td>.001</td>
</tr>
<tr>
<td>CHI+</td>
<td>9</td>
<td>85.00 (15.58)</td>
<td>99.58 (16.66)</td>
<td>14.58</td>
<td>20.52</td>
<td>.004</td>
</tr>
</tbody>
</table>

Note. CHI−: closed head injury—negative premorbid history; CHI+: closed head injury—positive premorbid history. Means with standard deviations in parentheses.

score analysis given that the correlation between pre- and post-scores is approximately $r = .90$ (Huck & McLean, 1975). In this investigation, the correlation between pre- and post-FSIQ was $r = .91$.

Table 3 shows the means and standard deviations for the premorbid OPIE scores and the obtained FSIQ, VIQ, and PIQ scores. The group by pre–post interaction was not significant for FSIQ, $F(1, 21) = 1.01, P = .32$, or VIQ $F(1, 24) = .01, P = .90$. However, the pre–post interaction for PIQ was significant, $F(1, 21) = 4.56, P = .04$, indicating that the estimated

![WAIS-R Subtests: CHI- vs. CHI+](image)

**WAIS-R Subtests**

Fig. 1. Mean age-scaled WAIS-R subtest scores for the CHI− and CHI+ groups.
18.60 point reduction from pre- to post-injury in the CHI+ group was statistically greater than the estimated 8.68 point loss for the CHI− group. An analysis of covariance with the premorbid scores as the covariate replicated this finding $F(1, 20) = 4.55, P = .04 (d = .178; power = .531)$.

As expected, significant differences were found within each group between obtained and predicted mean FSIQ scores, using the OPIE best as the overall estimate of premorbid abilities (see Table 4), indicating, not surprisingly, that head injury had a negative influence on cognitive performance. Comparison of the CHI− and CHI+ groups on age-corrected post-injury IQ scores failed to reveal any significant differences between group on $F(1, 21) = .121, P = .731$, VIQ $F(1, 21) = 1.802, P = .742$, or PIQ $F(1, 24) = 111, P = .194$.

Although the OPIE VIQ/PIQ discrepancy by group was not significant as expected, the post-injury VIQ/PIQ discrepancy was significantly different $F(1, 22) = 4.90, P = .033 (d = .420; power = .503)$, with the CHI− group demonstrating less discrepancy. The average VIQ/PIQ discrepancy for the CHI− group was 6.13 (S.D. = 10.22), while the CHI+ VIQ/PIQ discrepancy was 18.14 (S.D. = 15.54). As Figure 1 illustrates, the majority of this discrepancy resulted from group differences on Block Design, Object Assembly, and Digit Symbol, all of which are reported measures of “fluid” intelligence.

Analyses of the other standardized scores from measures in this investigation revealed significant group differences on the Trail Making Test and Stroop Color–Word Test. Specifically, the groups differed on time to completion for Trails A $F(1, 23) = 5.72, P = .025 (d = .449; power = .613)$, and the color–word condition of the Stroop Color–Word Test $F(1, 19) = 6.38, P = .022 (d = .685; power = .817)$. No other significant differences were noted on Trails A number of errors, Trails B time to completion, Trails B number of errors, the Stroop color or word trials, or other measures from the battery.

3. Discussion

Satz (1993) completed a comprehensive literature review on cognitive reserve that is helpful when attempting to understand the CHI− and CHI+ group differences. Review of all support for cognitive reserve theory is beyond the scope of this investigation, but is well reviewed by Satz (1993). In the current investigation, the head injured groups were essentially equal in regards to education, occupational attainment, and premorbid IQ, which serve as indirect measures of cognitive reserve. Groups were also not statistically different in terms of age, a factor with known effects on neurocognitive performance. Although this was the case, all group comparisons were completed on age-corrected standard scores to ensure that age-effects did not influence group differences. Therefore, with indirect measures of cognitive reserve essentially equal, age-effects controlled, and non-significant group differences on other demographic variables, any significant differences by group can be attributed with greater certainty to diminished cognitive reserve secondary to the aggregate effects of injury in the CHI+ group.

In this investigation the CHI− group was more severely impaired according to the GCS and duration of PTA severity classification systems, although this was not reflected statistically. Hence, with all other variables (e.g., age, education, occupational attainment) relatively equal,
it would be expected that they should show greater pre–post differences in their neurocognitive performance. However, results suggested that the less severely impaired CHI+ group had greater pre–post differences for PIQ and a significantly larger VIQ/PIQ discrepancy. Examination of Figure 1 reveals that these significant discrepancies largely result from the divergence of age-corrected scaled-score performances across the WAIS-R Performance subtests by group, especially the two subtests that comprise the Perceptual Organization Index (POI; Block Design and Object Assembly), in particular. The subtests that make up the POI and PIQ are thought to measure “fluid” intelligence, which is believed to be more sensitive to the effects of brain insult. In comparison, there was little variability between groups on the subtests that comprise VIQ and reportedly reflect “crystallized” intelligence, which is thought to be relatively stable following brain injury (Kaufman, 1990). What strengthens these findings is that there were less severe head injuries in the CHI+ group, yet there was more loss from pre-to-post injury on measures of neurocognitive functioning. It should also be noted that all data were examined for outliers that may have erroneously skewed analyses and these findings. Overall, it appeared that the CHI+ group had diminished cognitive reserve to protect them from the effects of their head injury, as reflected in their greater decline from estimated premorbid intellectual functioning following their head injury, a finding which supports cognitive reserve theory.

While the results of the present investigation suggest that the CHI+ group was more sensitive to the effects of head injury than the CHI− group, future research is needed to expand and replicate these findings while focusing on some of this investigation’s shortcomings. First, a prospective study with a larger sample and more closely matched groups on demographic variables would be optimal. Although a larger sample would result in a greater likelihood of obtaining significant results, it should be kept in mind that the findings of this investigation were respectable, as reflected in the power analyses, for the limited sample size per group.

As noted earlier, all participants were referred for neuropsychological evaluations prior to entry in a rehabilitation program. Future investigations may wish to include a greater number of individuals not referred to rehabilitation to avoid a selection bias and increase external generalizability. Many individuals are often not referred following injury because they either do not seek initial medical attention and/or because they lack the resources (i.e., insurance). Individuals without insurance are typically only treated during the acute phase of their injury, then discharged. Conversely, individuals with insurance and continuing problems following head injury have a higher probability of referral for rehabilitation. For these reasons, future studies may also consider including multiple comparison groups (e.g., a referred rehabilitation group, non-rehabilitation group, rehabilitation referred litigating group, and a non-rehabilitation referred litigating group), and recruitment from sites other than rehabilitation units (e.g., emergency rooms).

Although participants were screened for and not involved in litigation or pursuing either a worker’s compensation or social security disability claim at the time of their examinations (which is admittedly not often the case with head injury) and the evaluations were for rehabilitation purposes, it is impossible to ensure that, at some point following the examination, a legal complaint or claim would not be filed. Therefore, the possibility that the current results were influenced by concerns about future litigation cannot be entirely ruled out. However, examination of both groups’ pattern of performance across the examination battery was inconsistent
with what is typically seen with malingered performance. Specifically, examination of the CHI+ and CHI− groups’ performance on the WAIS-R and with methods with recognized utility for detecting inconsistent and incomplete effort (i.e., Digit Span, Digit Span—Vocabulary difference scores, and discriminant function analysis of WAIS-R subtest performances), were consistent with good effort and motivation (Mittenberg, Theroux-Fichera, Zielinski, & Heilbroner, 1995). Nevertheless, future studies could strengthen the findings of this investigation by including a measure of motivation and effort, as well as looking at the pattern of performance across the evaluation battery for telltale signs of poor motivation and effort.

In conclusion, based upon the present preliminary results, it appears that individuals with conditions or illnesses that are known to detrimentally affect neurocognitive functioning, such as a history of alcoholism, drug abuse, psychiatric illness, and/or previous neurologic insult, may have diminished cognitive reserve, and hence suffer greater cognitive decline following an additional stressor (i.e., head injury) that is in excess of that expected from the head injury alone.

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References


