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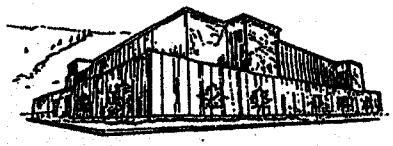

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DETECTING SIMULATED COGNITIVE IMPAIRMENT WITH MMPI-2 NEUROCORRECTION SCALES

by

Paul D. Dukarm

B.A. Texas A & M University - Corpus Christi

M.A. Texas A & M University - Corpus Christi

presented in partial fulfillment of the requirements

for the degree of

Doctor of Philosophy

The University of Montana

May 2006

Approved By:

Stuart Hall

Chairperson

Dean, Graduate School

5-31-06

Date

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Dukarm, Paul D., PhD, May 2006 Clinical Psychology

Detecting Simulated Cognitive Impairment with MMPI-2 Neurocorrection Scales.

Chairperson: Stuart Hall, PhD. SH

This study provides the first evidence that special neurocorrection scales of the second edition of the Minnesota Multiphasic Personality Inventory (MMPI-2; Butcher, Graham, Tellegan, & Kaemmer, 1989) are superior to the Fake Bad Scale (FBS; Lees-Haley, Dunn, & English, 1991) at differentiating traumatic brain injury (TBI) simulators compared to controls. Two groups of undergraduate psychology students were assigned to either a TBI simulator (n = 15) or control group (n = 17). Simulators were instructed to answer the MMPI-2 items within the context of simulating late effects of a mild-to-moderate head injury while pursuing financial compensation through litigation. A sample of community TBI patients (n = 22) was used as a comparison group. Results indicated that the Alfano et al. (1993) neurocorrection scale was clinically sensitive to TBI simulation (d = 3.3), and superior to the FBS (d = 1.8) compared to student controls. The Alfano scale achieved a sensitivity rate of 86.7% compared to 73.3% for the FBS. Specificity for the Alfano scale was 94.1% for an overall hit rate of 90.6%. The FBS achieved a specificity rate of 82.4% with a total hit rate of 78.1%. The neurocorrection scales were not effective at differentiating TBI simulators from community TBI patients. Limitations include small sample size and significant age differences between simulators and community TBI patients. The sample was skewed toward females (66%) and Caucasians (95%). Future studies should consider applying the neurocorrection scales in known-group studies investigating TBI simulation. The validity of MMPI-2 results in neuropsychological contexts is discussed.

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Preface

I would like to thank my committee at large and specifically, my research advisor Stuart Hall for his guidance and leadership in this process. I would like to thank him for making this process a truly great learning experience and increasing my level of understanding in the area of malingering and in neuropsychological assessment in general.

I would also like to thank my parents for their unwavering support during this journey. They instilled a respect and value for education and supported me in many ways. I would also like to thank my wife, Stephanie, for her support and tolerance during this process. She provided me with love, inspiration, and perseverance to continue pursuing my goal to become a clinical psychologist.

I would also like to thank the University of Montana Neuropsychology Laboratory for helping run participants for this project.

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Detecting Simulated Cognitive Impairment with MMPI-2 Neurocorrection Scales

Introduction

Traumatic brain injury (TBI) is responsible for more deaths and disability than any other neurological condition for individuals under age 50 (Center for Disease Control, 2004). The United States reports between 1.5 and 3 million new cases of TBI annually and up to 75% are classified as "mild" in severity. In spite of this fact, there are still 50,000 deaths each year, with 5.3 million Americans estimated to be living with a traumatic brain injury. This accounts for an annual figure of approximately 80,000 Americans experiencing long-term disability related to head trauma.

Clinical neuropsychologists are often called upon to evaluate the degree of cognitive impairment secondary to TBI. Many assessment devices, including self-report inventories, are often very useful in understanding a variety of psychosocial variables associated with cognitive complaints after a head injury. However, these instruments are not without certain vulnerabilities. For instance, self-report inventories of personality functioning are particularly susceptible to many types of deceptive responding. Attorneys have also been known to "coach" clients on how to respond to psychological inventories (Youngjohn, 1995).

It has been reported that complaints related to TBI are the most common neurological syndrome feigned (Haines & Norris, 1995). Consequently, base rates for malingering vary considerably in forensic contexts and have been reported to occur at rates up to 40% (Binder & Rohling, 1991; Fox, Lees-Haley, Earnest, & Dolezal-Wood,

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1995; Lees-Haley, Willis, & Brown, 1993; Mittenberg, Patton, Canyock, & Condit, 2002), and 10 times the base rate of bona fide cognitive impairment (Larrabee, 2000). These data have prompted clinicians in the field of forensic neuropsychology to incorporate measures of effort into their assessments as a standard of practice (Bender & Rogers, 2004; Bush et al., 2005; Iverson & Binder, 2000; Slick, Sherman, & Iverson, 1999).

Nichols and Greene (1997) describe "simulation" as one end of a continuum (i.e. ranging from simulation to dissimulation) where simulation refers to masking coping resources and simulating a greater degree of impairment than an individual may actually be experiencing and dissimulation is where individuals mask impairment in hopes of presenting a favorable impression (p. 255). The evaluation of malingering and symptom exaggeration poses numerous problems for clinicians. Many have resorted to examining constructs that are believed to be directly associated with malingering as well as discrepancies in assessment information. One of these constructs is the amount of effort one utilizes during the evaluation.

Effort has been defined as the "investment in performing at capacity levels" on a given effort based test (Bush, et al., 2005, p. 420). Inferences about performance are conceptualized along continuums of effort and honesty in which these two constructs may vary given the setting and circumstances of examination. Additional continua include intention, or volition, and incentive. The intention continuum ranges from conscious, volitional control over the decision to simulate impairment, as in malingering, to an unconscious, or unawareness that one is producing the symptoms, such as in

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conversion disorder (Slick, et al., 1999). Regarding incentives or rewards, the range is from internal or psychological incentive to an external reward such as financial compensation or avoidance of responsibility.

Slick et al. (1999) present criteria for diagnosing the malingering of neurocognitive dysfunction based on a variety of pieces of converging evidence. These include the presence of a substantial external incentive, negative response bias or failure on measures of effort, discrepancies between test data and behavior, inconsistencies between test data and reliable collateral reports, and inconsistencies between a person's historical information and test data. Additional considerations for diagnosing malingering include differences in cultural background, differential diagnoses, premorbid behavior, and the psychometric properties of tests used in the evaluation (Slick, et al., 1999).

Considering recent evidence that suggests that effort accounts for over half of the overall variance in neuropsychological test batteries (Green, Rohling, Lees-Haley, & Allen, 2001), it is prudent for psychologists to incorporate measures of effort in their examinations. The current study focuses on one approach that may improve the ability to make accurate judgments about score validity using the second edition of the Minnesota Multiphasic Personality Inventory (MMPI-2; Butcher, Dahlstrom, Graham, Tellegan, & Kaemmer, 1989).

The MMPI-2 and its predecessor, the MMPI (Hathaway & McKinley, 1943), have been the most common personality instruments used by clinical neuropsychologists in the United States for a number of years (Camara, Nathan, & Puente, 2000; Lees-Haley, 1992; Lubin, Larsen, & Matarazzo, 1984; Rabin, Barr, & Burton, 2005). The MMPI-2 is

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routinely utilized as a measure of psychopathology and personal adjustment in both clinical and neuropsychological evaluations and can provide useful information regarding the degree of distress and symptoms one is experiencing from a variety of neurological conditions (Lezak, Howieson, & Loring, 2004; Reitan & Wolfson, 1993).

The MMPI-2 is also often integrated into forensic and neuropsychological evaluations for its well established validity scales that measure exaggeration of psychiatric symptoms (Berry, Baer, & Harris, 1991; Graham, Watts, & Timbrook, 1991; Rogers, Sewell, & Salekin, 1994). Lees-Haley, English, and Green (1991) developed the Fake Bad Scale (FBS) and subsequently validated its use for detecting emotional and somatic exaggeration in personal injury evaluations (Larrabee, 1998). Recent research also claims that the FBS is a useful measure for detecting exaggeration of cognitive complaints associated with head injury (Larrabee, 2003; Martens, Donders, & Millis, 2001; Ross, Millis, Krukowski, Putnam, & Adams, 2004). This study hypothesizes that there may be a superior alternative to the FBS when the primary complaints are related to cognitive impairment.

There have been a series of studies that have developed special neurological scales from MMPI-2 items that have demonstrated sensitivity to detecting individuals with brain injury (Alfano, et al., 1993; Artzy, 1996; Gass, 1991; Gass & Russell, 1991; Van Balen, et al., 1997). We propose that these special neurocorrection scales contain item content that simulators of traumatic brain injury are more likely to endorse. In addition, it is believed that the neurocorrection scales are more sensitive to TBI simulation than the Fake Bad Scale.

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Neurological Scale Development with the MMPI

The first neurological scale for the MMPI was the Caudality (Ca) scale (Williams, 1952). The Ca scale contained 37 items and successfully differentiated patients with frontal lesions from patients with parietal lesions. Differences in response profiles indicated that individuals with frontal lesions were characterized by "inhibition, low aspiration, and peculiar thought processes," where as patients with parietal lesions exhibited "introversion, anxiety, and depression" (p. 296).

Creating and applying special neurological scales to identify personality changes associated with cognitive impairment became a major focus of research in the years following (Meier, 1969). Several researchers developed scales that were designed to distinguish between neurological and schizophrenic groups, with follow-up and validation studies demonstrating minimal clinical utility (Hovey, 1964; Horton, 1983; Horton & Wilson, 1981; Meier & French, 1964; Sand, 1973; Shaw & Mathews, 1973; Siskind, 1976; Upper & Seeman, 1968).

Item Bias and Symptom Nonspecificity

In a study which would have substantial impact on how the MMPI was interpreted, Ayers, Templar, and Ruff (1975) demonstrated that a scale devised to differentiate schizophrenic men from those with brain damage faired no better than scale 8 – Schizophrenia in doing so. In a follow-up study, they validated their conclusions by reporting that the special neurological scales were related more to indices of psychopathology rather than indices of brain damage. In fact, they raised the contention that the special scales were biased toward psychopathology in general and schizophrenia

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specifically (Ruff, Ayers, & Templar, 1977). The study also demonstrated that items could be endorsed by diagnostically separate groups, including neurological and psychiatric groups with similar symptomatology. When this happens, clinicians interpreting the MMPI in the standard fashion can inadvertently equate elevated MMPI scales with psychopathology instead of neurological sequelae.

Previous studies have produced equivocal results linking MMPI variables to neuropsychological variables (Cripe, 1996). For instance, several studies have indicated that the MMPI is insufficient at determining degrees of psychopathology for lateralized brain injuries and language disturbances (Dikmen & Reitan, 1974a; Dikmen & Reitan, 1974b; Moehle & Fitzhugh-Bell, 1988). Similarly, another study found that the MMPI was unable to detect post-operative changes in temporalobectomy patients regardless of side of onset (Trenerry, et al., 1996). Highlighting this trend, another study demonstrated that measures of personality and measures of neuropsychological constructs may create an artificial distinction due to the method variance contained in the MMPI. In other words, the reason that the MMPI appears to have an orthogonal, or distinctive structure, was based on the method of taking the test and not because an actual, statistical difference exists between tests of cognition and tests of personality (Zillmer & Perry, 1996).

This began to illuminate the inherent problem that individuals may endorse items on the MMPI-2 representative of cognitive impairment instead of psychopathology (Reitan & Wolfson, 1997; Tate, 1999). Bornstein and Kozora (1990) demonstrated that the MMPI-2 items were nonspecific to psychiatric or neurological content. In their study,

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scale 8 was unable to differentiate patients diagnosed with epilepsy from patients diagnosed with schizophrenia. Their research amplified once again the inherent problem of item nonspecificity that Ayers and colleagues discussed. However, others have found evidence that the MMPI is useful in neuropsychological evaluations (Gass, 1991b; Larrabee, 2003).

Subsequent studies aimed at associating MMPI variables with neuropsychological test variables revealed that the MMPI does contain item content reflective of neuropsychological domains. In a series of studies research demonstrated that cognitive variables such as attention, memory, speech impairment, information processing speed, and cognitive efficiency are associated with certain MMPI elevations (Gass, 1991; Gass, 1996; Ross, Putnam, Gass, Bailey, & Adams, 2003). Some suggested that, if interpreted correctly, the MMPI could offer useful information in the neuropsychological context (Gass, 1991b). Researchers continued to pursue approaches that might improve the validity of the MMPI in neurological populations. Unlike prior research that focused on scale development or decision rules (Russell, 1975; Watson & Thomas, 1968), some researchers devised procedures for altering the actual profile by eliminating items from the Clinical scales that contain "neurologically related" content (Gass & Russell, 1991).

Correcting the MMPI-2 for TBI Symptoms

The understanding that items on the MMPI could reflect both neurological and psychiatric symptoms led to questioning the appropriateness of the MMPI / MMPI-2 in neuropsychological examinations (Cripe, 1996). One approach that attempted to address this issue is described as "neurocorrection." Neurocorrection refers to the process of

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identifying items that contain neurological content and subsequently deleting those items from their respective scales and then replotting the profile. The profile is then said to be neurocorrected. Essentially, this method corrects for scale elevations in which items could theoretically be endorsed due to neurological symptoms rather than symptoms of psychopathology.

Gass and Russell (1991) conducted the first study of neurocorrecting the MMPI with patients with head injury. MMPI prorated raw scores were applied to neurological items that assigned a new value to the items that were "equivalent to the individual's ratio of non-neurological item endorsement in each scale" (p. 255). By adjusting profiles with a weighted score, the raw score can be interpreted in proportion to the original profile.

In a study that attempted to address the limitations of the first, Gass (1991) added a control group and derived neurological items in an empirical manner. First, items were selected that could reasonably be indicative of head injury. The researchers then gave the 370 item version MMPI to 75 patients diagnosed with various degrees of brain injury. The researchers then derived through principal components analysis two factors. Factor one consisted of 14 items that accounted for the main source of variance (24%). The items reflect somatic and cognitive related content.

This correction of the scales attenuates or suppresses MMPI profiles in patients with head injury. Gass (1991) explains that this deflation in the profile more accurately reflects the individual's psychiatric status because the scales have been corrected for neurological complaints. Interpreting the basic profile becomes more meaningful than the attempts at using the instrument as a diagnostic tool. Other researchers have followed

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Gass's original study and constructed their own neurocorrection scales by using both rational and empirical methods. (Alfano, et al., 1993; Artzy, 1996; Van Balen, et al, 1997).

Brulot, Strauss, and Spellacy (1997) tested the validity of the empirical correction scales related to injury severity, neuropsychological test performance, and depression. The authors devised a hybrid scale in which at least two items overlapped from the Gass (1991), Alfano et al. (1993), or Artzy (1996) correction scales. There were no associations between the correction factors and injury severity as measured by loss of consciousness (LOC and post-traumatic amnesia (PTA), and neuropsychological test performance overall. In addition, the authors found that the correction factors did significantly relate to the MMPI-2 content scale of depression (DEP), suggesting that items on this scale may also contribute to content bias as observed in previous studies with scale 8.

This finding led the authors to questions the validity of the correction factors as reflecting physical and cognitive impairments rather than emotional disturbances. Thus, due to the moderate correlations between the DEP scale and the correction scales, the nonspecificity of mild head injury symptoms and depression lead to problems in differentiating the respective domains of impairment. As in the Edwards et al. (2003) study, acute symptoms of TBI reflect more of the physical attributes of impairment, and as symptoms persist, somatic and affective symptoms may dominate complaints. However, as cognitive symptoms remit, so should symptoms reflecting emotional and psychological maladjustment (Gualtiere, 1995).

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In an attempt to validate the neurobehavioral content of Gass's (1991) correction scale, Rayls, Mittenberg, Burns, and Theroux (2000) tested Gass's neurological correction scale in patients meeting criteria for mild head trauma. Their results indicated that Gass's items reflect acute mild head trauma sequelae, given that there were no significant differences in item endorsement rates for the chronic profiles compared to the normative group. As suggested by the authors, one possible explanation for their results could be that endorsement of neurologically related items is "related to depression rather than cognitive impairment" (p. 549). They go on to add that test-takers may endorse items by misattributing item content reflective of emotional maladjustment.

Edwards et al. (2003) administered the full MMPI-2 to a group of 35 patients with differing levels of head injury severity. In addition to the head injury sample, a group of 35 psychiatric patients with no reported history of organic brain syndrome or neurological disorder were selected as a comparison group. The psychiatric group consisted of patients diagnosed with a variety of affective disorders and substance abuse. The authors compared three different correction scales, one of which was developed on group of multiple sclerosis patients (Meyerink, Reitan, & Setz, 1988). Results indicated that that none of the scales produced significant differences in rates of item endorsements. In addition, profile changes led to clinically relevant information for interpretive means. Yet the authors acknowledge that correction of the clinical scales may lead to psychometrically new scales yet to be validated.

Glassmire et al. (2003) also tested the classification ability of the neurocorrection scales from Gass (1991), Alfano et al. (1993), and Gass & Russell (1991). Like other

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studies, results indicated that the neurocorrection scales demonstrated good ability to differentiate head injured patients from normals. However, they failed to demonstrate adequate ability to differentiate TBI patients from psychiatric patients.

Studies examining the use of the MMPI-2 in neuropsychological contexts have produced inconsistent results (Cripe, 1996). A large body of literature has identified additional variables that may influence MMPI-2 scale profiles. These variables may prolong cognitive impairment and maintain neuropsychological complaints.

Variables That Moderate MMPI-2 Profiles

Larrabee (1999) discusses several moderating variables that could influence the degree of symptom expression indicated on the MMPI-2 profile. Included are injury variables such as length PTA, duration of LOC, or the emergence of post-traumatic seizures. Persons with mild head injuries reflect a greater deficit or dysfunctional level on the MMPI-2 clinical scales than those with more severe head injuries. This is referred to as the "paradoxical severity effect" (Youngjohn, Burrows, & Erdal, 1995). The phenomenon is amplified even more when individuals are involved in litigation (Binder & Rohling, 1996; Hoffman, Scott, Emick, & Adams, 1999; Youngjohn, Davis, & Wolf, 1997). Other variables include subject variables such as age, adverse life events, chronic social difficulties, gender, and symptom expression like somatization (Larrabee, 1999).

TBI Simulation and the MMPI-2 Fake Bad Scale

Historically, clinicians have attributed late effects of mild head injury to psychogenic factors or external incentives (Gasquoine, 1998; Strauss & Savitsky, 1934; Tate, 1998, 2003). In a recent survey of the National Academy of Neuropsychology and

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Division 40 of the American Psychological Association, it was reported that forensic referrals make up the second highest reimbursement source in private practice clinical neuropsychology (Sweet, Peck, Abramowitz, & Etzweiller, 2003). In addition, referrals from attorneys represent the third highest referral source behind psychiatry and neurology, respectively (Sweet, Moberg, & Suchy, 2000). It has been demonstrated through meta-analyses that the MMPI-2 Validity and malingering scales are sensitive to certain forms of psychopathology (Berry, Baer, & Harris, 1991; Graham, et al., 1991; Rogers, 2003). However, the validity scales and special malingering scales are not specific to head injury or malingering of cognitive impairment (Greiffenstein, Gola, & Baker, 1995; Lamb, Berry, Wetter, & Baer, 1994).

In order to address malingering in personal injury contexts, Lees-Haley, English, and Glenn (1991) developed a scale specifically for the purpose of detecting exaggerated emotional distress. Inspired by Gough's Dissimulation Scale (Ds; Gough, 1947) and applied practically based on clinical experience, Lees-Haley and colleagues selected items that represented both simulation and dissimulation characteristics. These include not only the exaggeration of neurobehavioral symptoms, but also the concealment of deviant behavior and presenting oneself in a positive manner. The result of their effort was the creation of the Fake Bad Scale and studies have reported the clinical utility of detecting exaggeration of complaints related to TBI (Larrabee, 2003; Martens, Donders, & Millis, 2001; Ross, Millis, Krukowski, Putnam, & Adams, 2004).

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The FBS consists of 43 items obtained from MMPI responses from personal injury claimants who "appeared to be clearly malingering" (p. 205). The responses tended to fit a model that reflected:

> "1) appearing honest; 2) appearing psychologically normal except for the influence of the alleged cause of injury; 3) avoiding admitting preexisting psychopathology; 4) attempting to minimize the impact of previously disclosed preexisting complaints; 5) minimizing or hiding perjury, antisocial or illegal behavior; and 6) presenting a degree of injury or disability within perceived limits of plausibility" (Lees-Haley, et al., 1991, p. 204).

Utilizing a cutoff point of 20 on the FBS for all groups, the scale classified 96% of the malingering group, as opposed to the non-malingering claimants who were correctly classified at the 90% level. A total accuracy rate of 93% for both groups filing claims was obtained. For the medical outpatients simulating a motor vehicle accident, toxic exposure, and job stress, the scale correctly classified 88%, 53%, and 83% respectively. It was clear that the performance of the FBS initially looked promising for the purpose of detecting simulated impairment during personal injury evaluations (Lees-Haley, Iverson, Lange, Fox, & Allen, III, 2002). However, additional studies would call the construct validity of the FBS into question, sparking a current debate questioning the utility of the FBS in personal injury examinations (Butcher, Arbisi, Atlis, & McNulty, 2003; Rogers, 2003).

Concerned about the construct validity on the FBS, Butcher et al. (2003) examined MMPI-2 archival files from 108,791 subjects compiled between 1990 and 1996. Six settings were represented in the analysis, and, when a conservative cutoff of 26 was applied, between 2.4 - 30% of the individuals were classified as malingering. The authors concluded that the FBS over-predicts malingering despite adjusting cutoff scores

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for differences in setting base rates. Instead, Butcher et al. state that the FBS reflects a broad variety of somatic symptoms and is "associated more with the expression of psychopathology in which physical symptoms are experienced" (p.482). Butcher and colleagues subsequently called for the abandonment of the FBS for detecting somatic malingering (Arbisi & Butcher, 2004).

Dearth et al. (2005) used an analog design in which they tested the ability of the FBS to classify TBI simulators above and beyond that of the other validity scales contained in the MMPI-2. Their results showed that the FBS was superior to the traditional validity scales, suggesting a different "operating profile" than the standard F family of validity scales, particularly in settings with high base rates of malingering head injury.

However, there has only been one study that considered neurocorrection procedures as a useful technique in differentiating MMPI profiles between patients undergoing a forensic neuropsychological evaluation for head injuries and patients involved in a forensic psychological evaluation. Dunn and Lees-Haley (1995) tested the effectiveness of the Gass (1991) correction procedure in which 14 items were deleted from the standard profile. Results demonstrated that the correction procedure did not significantly differentiate persons litigating personal injury or primarily whiplash and head injured group profiles. In fact, they reported that only 5 items were significantly differentially endorsed by the two groups out of the total 14. They subsequently rescored the profiles by only eliminating the five items, again finding nonsignificant mean group

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differences. The authors stated that the Gass correction procedure should therefore not be used in forensic neuropsychological examinations.

Purpose of Current Study

To date, there has been no published research examining the effectiveness of MMPI-2 neurological correction scales as indicators of possible simulation of cognitive impairment. It is thought that the neurocorrection scales carry a unique "operating profile" given their neurologically specific content. Unlike the Dunn and Lees-Haley (1995) study, this study utilizes the neurocorrection scales as independent ordinal scales, and not a "correction" procedure by eliminating items from the standard profile. This study examines the ability of each of the published head injury neurocorrection scales to differentiate TBI simulators and student controls. It utilizes a comparison group of archival patients with documented mild to moderate head injury to test if the scales can differentiate between TBI simulators and community TBI patients.

However, the main purpose of the current study is to compare the most effective neurocorrection scale against the Fake Bad Scale in a simulated TBI malingering paradigm. If the hypotheses are correct, this study will provide grounds for further investigation on the clinical utility of the neurocorrection scales beyond profile analysis. It could also call into question the use of the MMPI-2 in forensic neuropsychological examinations. Specifically, it will draw attention to the practice of using self-report inventories developed on psychiatric patients for assessment with neuropsychological populations.

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Hypotheses

The first hypothesis concerns the ability of the neurocorrection scales to differentiate TBI simulators from controls effectively. Although the Gass (1991) scale has the most published research focused on its sensitivity to head injury, this study will instead predict that the Van Balen et al. (1997) scale will be able to predict simulators of cognitive impairment above the other scales. This hypothesis is based on the premise that the scale is derived from the complete MMPI-2 item set, compared to the 370-item version of the Gass (1991), and Gass and Russell (1991) scales. The scale also reflects item content that was endorsed by at least two professionals from the field of neurology, neuropsychology, psychiatry, and physiatry as pertaining to head injury sequelae. Thus, individuals who intend to express cognitive impairment might endorse items that, on the surface, reflect cognitive content consistent with bona fide mild head injury (Huskey, 2004) and, popular misconceptions of head injury symptoms (Guilmette & Paglia, 2004; Swift & Wilson, 2001).

The second hypothesis concerns the effectiveness of the correction scale(s) when compared to the Fake Bad Scale. It is predicted that the most effective neurocorrection scale will outperform the Fake Bad Scale in the ability to differentiate effectively TBI simulators from actual TBI patients. This prediction is based on the premise that the neurocorrection scale will be more likely to detect cognitive symptomatology, as opposed to the FBS, which was developed to detect emotional and somatic distress (Lees-Haley, et al., 1991).

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Method

Participants

University of Montana undergraduate psychology students were recruited during the spring and summer sessions of the year 2005. Sixty students were randomly assigned to either a TBI simulation or control group. Students were allowed to participate if they were 18 years of age or older and current psychology students at The University of Montana. The 60 students received 3 credits toward their respective courses for participation. Student information sheets were then screened and protocols excluded from the final analysis if participants endorsed a history of neurological or psychiatric treatment. Specifically, students were eliminated if they reported a history of any mood related disorder, anxiety condition, substance abuse, Attention Deficit Hyperactivity Disorder (ADHD) migraine headache, a seizure condition, or a mild to moderate head injury. After screening, the sample included 15 students in the TBI simulation group and 17 students in the control condition. Every other student was assigned to the TBI simulation group until 30 participants for each group completed the MMPI-2.

Thirty archival files from community TBI patients were also collected. The same inclusion criteria applied for this group, age 18 years or older and a completed full MMPI-2. Additionally, informed consent must have been documented in their records and have met the clinical criteria for a mild to moderate head injury. The sample was taken from records between the years 2002-2005. After screening for the prior neurological and psychiatric conditions mentioned above, the inclusive sample served as a clinical comparison group ($n = 22$).

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Determining the Severity of Head Injury

The severity of head injury was defined according to one of three possible injury variables (see appendix D). Measures of injury severity consisted of the length of time one experienced a loss of consciousness (LOC) due to a blow the head, length of time one experienced post-traumatic amnesia (PTA), or score on the Glascow Coma Scale (GCS; Williamson, Scott, & Adams, 1996). For mild head injury, the criteria consist of a) LOC for less than 20 minutes, b) PTA of less than 24 hours, and c) GCS of 13-15. For moderate head injury, the criteria consist of either a) LOC of 20 minutes - 36 hours, b) PTA of 1-7 days, and c) GCS of 9-12 (DeKruijk, Twijnstra, & Leffers, 2001). An individual was required to have met one of the criteria to be included in the current study.

Materials

The 567-item version of the MMPI-2 was administered or collected from each participant. Validity and Clinical scale raw scores were transformed into uniform T - scores for validity and K-corrected Clinical scales. Raw scores from five neurocorrection scales and the Fake Bad Scale were also computed.

MMPI-2 Validity Scales

The validity scales included the family of F scales typically used to identify over-reporting of psychiatric symptoms. The scales included the F (Infrequency) scale, which measures the frequency of items that are symptoms rarely experienced by psychiatric patients; F(b) - Back Infrequency, which consists of items of infrequent psychiatric symptoms for the last half of the MMPI-2; and F(p) - Infrequency Psychopathology (Arbisi & Ben-Porath, 1995), which is a measure of infrequent symptoms endorsed by no

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more than 20% of a sample of psychiatric inpatients. Two other validity scales that measure under-reporting of psychopathology were included. The Lie scale (L) is a measure of defensiveness defined as a refusal to admit even minor faults, and the correction scale (K) which is a measure of subtle or sophisticated attempts to present oneself favorably (Meehl & Hathaway, 1946).

Finally, consistency of item endorsement was measured by including the Variable Response Inconsistency (VRIN) scale and the True Response Inconsistency (TRIN) scale (Butcher, et al., 1989). The VRIN scale consists of 67 pairs of items that have similar or opposite item content. The TRIN scale measures whether the respondent answered the item pairs as either "true" or "false" (Greene, 2000).

MMPI-2 Clinical Scales

The ten clinical scales, in order, are contemporarily referred to by their numerical codes. These are Scale 1 – Hypochondriasis, 2 - Depression, 3 - Hysteria, 4 - Psychopathic Deviate, 5 - Masculinity/Femininity, 6 - Paranoia, 7 - Psychasthenia, 8 - Schizophrenia, 9 - Hypomania, and 0 - Social Introversion.

Scale 1 reflects a wide variety of nonspecific somatic concerns. Somatic areas include complaints regarding the abdomen and back, and persist despite negative medical evidence. It must be noted that individuals who are actually ill will obtain moderate elevations. High scorers on this scale, if physically ill, will also tend to have hypochondriacal features associated with their illness (Greene, 2000). Personality traits often include self-centeredness, demandingness, cynicism, and stubbornness (deMendonca, et al., 1984).

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Scale 2 is characterized as a diverse scale that includes features of poor morale, worry, self-punishing, hopelessness about the future, and dissatisfaction with one's life (deMendonca, et al., 1984; Graham, 2000). There are also items that reflect physical symptoms, such as sleeplessness and gastrointestinal problems. The scale also includes content related to apathy, psychological sensitivity, and social withdrawal. Greene (2000) states that scale 2 is a difficult scale to interpret in isolation given its many factors.

Scale 3 was developed on patients either diagnosed with Hysteria with what currently be called Histrionic Personality Disorder. Item content reflects somatic symptoms in the head, arms, and legs, as well as a perception of social adjustment. Persons with high scores on this scale are friendly, self-centered, demanding, immature, and suggestible (deMondonca, et al., 1984) and only avoid responsibility and develop conversion symptoms when under overwhelming stress (Greene, 2000).

Scale 4 was developed on young adults who were diagnosed with a "psychopathic personality" (McKinley & Hathaway, 1946). The scale typically represents social maladjustment and a lack of pleasant experiences. Poor impulse control, emotional shallowness, a disregard for social conventions, and hostility towards authority figures are features that describe high scorers.

Scale 5 measures gender stereotypes and was developed to detect potential homosexual trends when homosexuality was once considered a psychiatric disorder by the American Psychiatric Association (APA) as indicated in the second edition of the . Diagnostic and Statistical Manual of Mental Disorders (DSM-II, see APA, 1968). This scale is not considered a "clinical" scale and will not be included in the analysis.

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Scale 6 represents item content reflective of interpersonal sensitivity, self-righteousness, and suspiciousness (deMondonca, et al., 1984). The scale does not represent pure cases of paranoid individuals, but rather individuals who were deemed to be in a paranoid state, including persons diagnosed with "paranoid schizophrenia" or with a "paranoid condition" (Greene, 2000, p. 155).

Scale 7 is generally considered a measure of psychological discomfort and turmoil, with high scorers often experiencing obsessive thinking and serious levels of insecurity (Graham, 2000). The condition of "psychasthenia" is contemporarily conceptualized as obsessive-compulsive disorder. But rather than tapping specific behaviors or rituals, the scale reflects personality characteristics such as interpersonal sensitivity, neuroticism, anxiety, and social withdrawal, as well as poor physical health (deMondonca, et al., 1984; Graham, 2000).

Scale 8 reflects the most diverse Clinical scale given its seven factors. The scale taps a wide variety of symptoms that include "paranoia, concern about sex, sensitivity to rejection, psychotic tendencies, poor concentration, poor health, and social withdrawal" (Graham, 2000). Additional characteristics include confusion, worry, imaginativeness, unconventional attitudes, and impulsiveness (deMondonca, et al, 1984). Not only can high scoring protocols be related to the above variables, elevations will also be produced by individuals with neurological impairment such as epilepsy (Bornstein & Kozora, 1995).

Scale 9 was derived from psychiatric patients who demonstrated characteristics of hypomania. Hypomania is characterized on the MMPI-2 as disturbances in "activity

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level, excitability, irritability, and grandiosity" (Graham, 2000, p. 82). Specifically, high scorers endorse items that describe accelerated psychomotor behavior and speech, irritable and depressed mood, and flight of ideas. The scale is sensitive to the effects of age, with elderly populations often producing scores below average, and ethnicity, with American Indian/Alaska Native, Hispanic, and African-American groups scoring on average higher than Caucasians (p. 82). High scorers on this scale are typically viewed as having excessive energy and a tendency to manifest an unrealistic self-appraisal.

The study will also include scale 0 although this scale was not developed on a particular patient group. Rather, it was developed to represent the continuum of personality traits of social introversion – extroversion. The scale should not be interpreted in isolation due to its development on university students, but it can provide useful information when additional scale elevations are observed (Graham, 2000).

Five MMPI-2 Neurocorrection Scales

Five neurocorrection scales were used in this study. Three of the scales were derived through empirical methods and two were developed from a rational approach. The Gass (1991) 14-item scale comprises items that were derived through Principle Components Analysis. The oblique rotation yielded a two factor solution for the 370-item version of the inventory. The first factor included 28.4% of the overall variance and was labeled "neurological complaints" (p. 29). The items represented neurological symptoms of cognitive inefficiency, muscle weakness, tremor, and speech related content.

The Alfano et al. (1993) 13-item scale was also derived through Principle Components Analysis but the analysis was conducted on the full 566-item MMPI. This

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scale accounted for 25% of the overall variance and was labeled a "neurobehavioral" factor. The items contained neurological symptom complaints related to poor attention, sensory and motor dysfunction, or problems with psychosocial adjustment such as vocation or with sexual satisfaction.

The Artzy (1996) 17-item TBI scale was another scale in which Principle Components Analysis was used to derive a head injury factor. Artzy compared patients with head injuries to patients with chronic pain. The subsequent TBI factor accounted for 28.5% of the overall variance and represented item content such as dizziness, attention and concentration problems, and poor psychosocial adjustment.

The rationally derived Gass and Russell (1991) 42-item scale predated the aforementioned Gass (1991) study. An innovative approach was used instead of the empirical method. The authors had professionals in the field of neurology select items that could possibly represent common "physical, not emotional" sequelae for brain damage (p. 255). This approach of using professionals to select items based on their content and clinical representation of symptoms is referred to as a rational method. The neurological items in this scale are represented in the MMPI-2 Harris-Lingoes subscales (Gass & Russell, 1991).

Instead of adopting the usual procedure of deleting the items from the profile, Gass and Russell assigned a weighted score to neurological related items. The weighted scores were based on the ratio of non-neurologically related items that were endorsed in each Clinical scale. The weighted scores were entered into the overall scoring of each

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scale. The Clinical scales were then K-corrected. This procedure allowed for scale adjustment without the potential psychometric pitfalls associated with item deletion.

Finally, the Van Balen et al. (1997) 22-item scale was also derived through rational methods. Forty experts from four professions that commonly treat patients with central nervous system (CNS) related impairments were asked to imagine patients with CNS dysfunction in general and identify items that could possibly reflect common symptoms. Then, the judges were asked to do the same with 3 major patient groups, namely "TBI, stroke, and whiplash" (p. 358). The items that obtained inter-rater agreement levels of 70% or higher and were endorsed by at least two different professions were selected as indicative of a particular patient population.

Procedure

Each student was given instructions in a private room by a trained research assistant. Participants assigned to the control group received standard instructions as indicated in the MMPI-2 clinical manual (Butcher, et al., 1989). Participants assigned to the TBI simulator group were provided with instructions in which participants were instructed to simulate a mild to moderate head injury (see appendix F). Participants in the TBI simulator group were also told to imagine that they had experienced a head injury in a motor vehicle accident. Additionally, TBI simulators were to take the MMPI-2 as part of the legal process for obtaining maximum financial compensation for their injuries. Finally, even though their injuries were to have diminished, they were to answer the questions from the MMPI-2 in a manner that suggested they were continuing to experience the effects of a head injury. As a measure of response set, student TBI

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simulators were asked if they put forth maximum effort to simulate symptoms of a head injury. All student participants received the allotted credit regardless of their ability to simulate a head injury. Completed MMPI-2 protocols were obtained on the community TBI group where informed consent to participate in research had been documented in their clinical record.

Analysis of the Data

In order to examine the utility of the neurocorrection scales as possible indicators of TBI simulation, a series of data analyses were conducted to answer the following research questions:

1) Can MMPI-2 neurocorrection scales detect simulated cognitive impairment in a compensation seeking paradigm?
2) Can MMPI-2 neurocorrection scales differentiate between groups of analog TBI malingerers, actual TBI patients, and controls?
3) Can MMPI-2 neurocorrection scales classify group membership equal to or better than the Fake Bad Scale?

Data procedures involved generating descriptive statistics for demographic variables age, years of education, gender, and ethnicity. Means, standard deviations, and results from one-way analysis of variance (ANOVA) and the Tukey honestly significant difference (HSD) test were computed to detect group differences on MMPI-2 Validity and Clinical scales (see appendix H-I). ANOVA, Tukey's HSD, and overall measure of association expressed as eta-squared (η^2) for neurocorrection scales were also conducted to ascertain group mean differences.

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Effect Size Contrasts of Neurocorrection Scales

In order to answer the question of whether the neurocorrection scales could differentiate between the three groups, a series of pairwise effect size contrasts were conducted. The first contrast determined what neurocorrection scale was clinically sensitive to TBI simulation, as well as meeting the selection criteria for entry into the classification analysis. The next two contrasts answer the research questions regarding whether the neurocorrection scales could differentiate community TBI patients and controls, and TBI simulators and community TBI patients. In order to measure the magnitude of separation, a converted Cohen's *d* effect size statistic was calculated with pooled standard deviations based on unequal group sizes (Zakzanis, 2001).

Cohen's *d* is the estimated difference between the target and non-target group means calibrated in pooled standard deviation units (Cohen, 1988). This statistic has been deemed the most appropriate measure of effect size because it does not assume homogeneity of variances among groups, and is calculated independently of sample size. Zakzanis' (2001) approach converts standard Cohen's *d* statistics to display the degree of test score overlap between the two groups being compared by subtracting the amount of test score nonoverlap from 100. Percentages of test score overlap between the groups can be contrasted with effect sizes, and it ranges from 0-4. A converted effect size of 0 would be interpreted as "no effect" and an effect size of 4 would represent "absolute discriminability" (Zakzanis, 2001, p.658).

This study used the clinical significance marker of 3.0, which converts to 7% overlap between groups on a particular test measure. This implies that 93% of the target

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group participants actually produced test scores that were not produced by non-target group members (Zakzanis, 2001). Calculation of effect sizes using pooled standard deviations for unequal group sizes were used to answer research questions one and two.

Classification Analysis

Binary logistic regression analyses was conducted to answer question three regarding the ability of the best neurocorrection scale(s) to classify TBI simulators compared to the FBS. Logistic regression does not assume homogeneity of variance among groups and is based on the odds that a random score on a given measure will be classified as in the target category (Grim & Yarnold, 2001).

In this analysis, the best neurocorrection scale and the FBS were the independent variables, while group membership was the dependent variable. The analysis generated odds ratios that represented the likelihood a random participant's score would be classified as coming from the target group. Unstandardized regression coefficients (B) represented the nonlinear transformation of the predictor for a binary or dichotomous dependent variable (Cohen et al., 2004). Since this study was aimed at determining the effectiveness of the neurocorrection scales to detect TBI simulation, the target group for the analysis was the TBI simulator group.

Operating Statistics

Sensitivity, specificity, and overall correct classification percentages were conducted for the best neurocorrection scale and the FBS. Sensitivity refers to the percentage correctly classified target group members. Specificity is the percentage of

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correctly classified non-target members. Hit rate refers to the overall correct classification percentages, a combination of sensitivity and specificity rates.

Results

Results of Demographic Variables

Table 1 displays group means for age and years of education. Significant age differences were observed among groups, [F (1, 54) = 31.7, $p \leq .001$, $\eta^2 = .55$]. Tukey's HSD test revealed that the TBI simulator and control groups were significantly younger than the community TBI group. However, there were no significant differences found between the TBI simulator and control groups. There were no significant differences in years of education across groups, [F (2, 54) = .03, $p = .97$, $\square^2 = 01$]. Out of the 15 TBI simulators, 100% stated that they put forth maximum effort while simulating a head injury.

Table 2 presents gender and ethnic composition of each group. Gender composition did not differ significantly across groups, χ^2 (2) = .04, $p = .98$. Essentially each group had identical female-to-male percentages. All groups were approximately two-thirds female with just over one-third male. The total sample composition was 65% female and 35% males. Additionally, there were no significant differences in ethnic composition across groups, χ^2 (6) = 5.9, $p = .43$. The TBI simulator group was 100% Caucasian, controls 91% Caucasian, and the community TBI group approximately 94% Caucasian.

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Analysis of Variance Comparing Groups on MMPI-2 Neurocorrection Scales

Analysis of variance procedures were computed for the neurocorrection scales across groups. All scales produced significant mean differences and are displayed in Table 5.

Significant mean differences were observed on the Gass (1991) scale, [F (2, 54) = 34.1, $p < .05$, $\eta^2 = .57$]. Tukey's HSD revealed significant differences between both TBI simulators and community TBI patients compared to controls, $p < .05$. No significant differences were observed between the TBI simulators and community TBI patients.

Significant differences among the 3 groups were observed on the Alfano scale, [F (2, 54) = 28.1, $p < .05$, $\eta^2 = .53$]. Both TBI simulators and community patients differed from controls, $p < .05$, but not between the simulator and TBI patient groups.

The Artzy scale revealed significant group mean differences, [F (2, 54) = 13.7, $p < .05$, $\eta^2 = .35$]. TBI simulators differed significantly from controls and community TBI patients, $p < .05$, but did not differ between community TBI patients and controls.

The Gass and Russell (1991) scale revealed significant differences among groups, [F (2, 54) = 27.3, $p < .05$, $\eta^2 = .52$]. Both TBI simulators and community patients differed from controls, $p < .05$, but did not differ between each other.

The Van Balen scale also revealed significant mean differences among the 3 groups, [F (2, 54) = 33.3, $p < .05$, $\eta^2 = .57$]. Tukey's HSD revealed the TBI simulators differed significantly from control subjects, $p < .05$, but not from community TBI patients. Similarly, community TBI patients differed from controls, $p < .05$, but not TBI simulators.

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The FBS was also included in a one-way ANOVA procedure. The FBS produced significant differences among groups, [F (2, 54) = 20.3, $p < .05$, $\eta^2 = .44$]. It was the only validity scale that did not produce significant differences among scores for the TBI simulators and the community TBI group. However, both TBI simulators and community TBI patients did differ from controls, $p < .05$.

A description of group differences on the Basic scales can be in the appendix H. The Validity scales demonstrated significant group differences on the family of Infrequency scales (see appendix I). The typical profile on the MMPI-2 Clinical scales for patients with mild head injury, and involved in compensation litigation, was replicated in this study (see appendix J).

Differential Ability of the Neurocorrection Scales

In order to address the question regarding using the neurocorrection scales to differentiate between TBI simulators and controls, a pairwise effect size contrast utilizing a converted Cohen's *d* effect size estimate based on unequal group sizes was calculated for each scale (see Table 6). The results indicate that Alfano's scale produced the only effect size to reach the clinical significance marker of 3.0 ($d = 3.3$). The large effect size indicated a 4% overlap between neurocorrection scores between the two groups.

All of the remaining neurocorrection scales produced moderate effects. The Gass scale produced a moderate effect size of $d = 2.9$, indicating a 7.0 % test score overlap. Artzy's scale produced a moderate effect size of $d = 2.0$, indicating 18.9% overlap among scores between the two groups. The Gass and Russell scale also yielded a moderate effect size ($d = 2.7$) that indicates 9.5% test score overlap. The Van Balen scale produced an

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effect size in the moderate range ($d = 2.8$) that translates into an 8.8% overlap in group scores.

In order to examine whether the neurocorrection scales are able to differentiate between community TBI patients and controls, a second series of effect size contrasts were conducted (see Table 6). Results indicated that there were no clinically significant effect size estimates produced by the scores. Gass and Russell's scale obtained a moderate effect size ($d = 2.4$) with 13% scale score overlap. Van Balen's scale also produced a moderate effect size ($d = 2.3$) that translates into 17.1% test score overlap. The Gass scale also produced a moderate effect size, ($d = 2.4$), indicating 15.7% score overlap. The Alfano scale and the Artzy scale produced small and no effects ($d's = 1.5$ and .50, respectively).

A final contrast was conducted in order to examine the ability of the neurocorrection scales to differentiate TBI simulators from community TBI patients. Results revealed that most neurocorrection scales produced trivial effect sizes between group scores. The exception was the small effect size produced by Artzy's scale ($d = 1.3$), which represents 34.7% of overlap between group scores on this scale (see Table 6).

In order to be included in the analysis comparing the classification ability against the FBS, neurocorrection scales must have met or exceeded the clinical significance marker for effect sizes (3.0; Zakzanis, 2001). The Alfano scale was the only scale to reach clinical significance, $d = 3.3$. Therefore, the Alfano scale was selected as the only neurocorrection scale to be compared to the FBS in the classification analyses.

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Classification Results of Logistic Regression Analyses

Table 7 displays two separate binary logistic regression procedures examining the classification ability of the Alfano neurocorrection scale and FBS. Results from logistic regression revealed TBI simulators were .21 times more likely to classified as simulators than controls using the Alfano scale, Wald χ^2 (1, 32) = 4.3, p = .04. The second logistic regression procedure examining the FBS revealed that TBI simulators were .56 times more likely to be classified as a simulator than a controls, Wald χ^2 (1, 32) = 8.8, p = .003.

Table 8 displays frequency counts for observed and predicted group membership. The Alfano scale correctly classified 13 of 15 TBI simulators and 16 of 17 control subjects. The FBS correctly classified 11 of 15 TBI simulators and 14 out of 17 controls.

Effect Size Comparison Between Alfano and the FBS

Table 9 presents Cohen's *d* effect size contrast for the FBS examining the degree of test score overlap compared to the Alfano scale. The FBS produced a small effect size of 1.8 which translates into a 22.6% test score overlap. In other words, 22.6% of scores on the FBS were shared among both groups, compared to approximately 5% overlap on the Alfano scale. All of the neurocorrection scales produced effect sizes larger than the FBS when differentiating TBI simulators from controls.

Sensitivity, Specificity, and Overall Hit Rate

Table 10 displays the sensitivity, specificity, and overall correct classification rates of both scales while using cutoff scores based on their harmonic means. The Alfano scale classified 86.7% of the TBI simulators correctly while classifying 94.1% of the control subjects correctly for an overall hit rate of 90.6%. Comparatively, the FBS

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classified 78.3% of the TBI simulators while classifying 82.4% of the control subjects for an overall hit rate of 78.1%. Both scales were able to classify control participants at a higher rate than TBI simulators.

Discussion

Performance of Neurocorrection Scales Compared to the FBS

Previous research has focused on the FBS as a measure of exaggeration on the MMPI-2 in neuropsychological evaluations. This study provides the first evidence suggesting that the MMPI-2 neurocorrection scales are superior to the FBS as measures of TBI simulation. In fact, all of the neurocorrection scales produced effect sizes larger than the FBS. This substantiates the research hypothesis that the neurocorrection scales would be more sensitive to head injury simulation than the FBS. A major impetus for testing this hypothesis is due to the scales design for identification of cognitive complaints. This study hypothesized that the neurocorrection scales would perform as good as or better than the FBS, which is designed to detect somatic and emotional exaggeration.

Measures taken to improve both internal and external validity included using homogenous groups in which prior histories of neurological and psychiatric conditions were excluded, as well as adding a comparison group of community patients with documented mild to moderate head injury as a comparison group. The instructions for the simulator group were purposely left symptomatically ambiguous in order to limit the degree of "coaching" provided to TBI simulators. In addition, effect size contrasts were

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employed in order to compensate for high degrees of score variability between the simulator, TBI, and control groups, as well as unequal group sizes (Zakzanis, 2001).

Artzy's scale produced a small effect size differentiating TBI simulators from community TBI patients. However, the 35% overlap of scores is unacceptable in clinical practice. On a side note, the FBS produced an effect size that translated into approximately 66% score overlap between these two groups. The latter result is consistent with previous studies in that the FBS largely over-estimates malingering of neurological symptoms (Butcher et al., 2003).

The Influence of Prior Probabilities and Base Rates

One important feature of classification studies is the prior probability rates for identifying a certain condition. Prior probabilities are based on base rates, or the likelihood that any given individual will be classified with a specified condition in a particular setting. It has been demonstrated that differences in base rate percentages across settings can influence the sensitivity and specificity rates of classification.

The current study had a base rate of malingering of 28%. This is well within the reported range of 15% - 40% base rates reported in previous studies (Binder & Willis, 1991; Fox, et al., 1995; Lees-Haley & Brown, 1993; Mittenberg, et al., 2002). Overall correct classification of 78% for the FBS in the current study was slightly higher than the Dearth et al. (2005) study in which the FBS achieved an overall hit rate of 73%. It was also similar to the Butcher et al. (2003) study that found up to 30% over-prediction of malingering by the FBS.

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Previous studies on the FBS have also reported classification results considering alternative cutoff scores and base rates. The contrast in this study between simulators and controls utilized a cutoff of 7 for the Alfano and 18 for the FBS. When the cutoff scores were adjusted for the Alfano scale to 8 and 10, the result was a higher rate of classifying control subjects; however, the sensitivity to malingering was lowered. This result is attributable to the fact that 3 of the simulators produced scores of 7 or below on this scale. Therefore, the elevation of the cutoff score did nothing to improve the sensitivity to TBI, only improving the identification of non-brain injured subjects.

Similarly, previous studies have suggested cutoff scores between 21 and 26 for the FBS. In the current study, a cutoff of 18 was used in the analog malingerers and control contrast that produced the most effective classification levels. In fact, the mean of 18 for the simulator group was actually lower than the mean of 21 for the TBI group. This finding is consistent with previous studies in that the FBS appears to be capturing item content related to somatic and emotional maladjustment rather than cognitive impairment. Consequently, when used to differentiate groups of malingerers from actual TBI patients, the FBS will tend to classify those individuals with bona fide TBI as malingerers.

This finding is somewhat expected, given the instructions of the current scenario referred to being "knocked out and dazed" rather than suggesting specific emotional and physical symptoms. Research on common misconceptions of brain injury symptoms by the general public and neurological patients consistently finds that that people tend to overestimate the degree of injury related to mild head injuries as well as overestimate

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premorbid psychological health and cognitive ability (Coolidge, et al., 1998; Mittenberg, et al., 1991; Williams, et al., 1998). In addition, it remains a diagnostic dilemma that post-concussive symptoms are not specific to mild head injuries and have been reported to occur in the general population at rates as high as 75% within a two week period (Iverson & Lange, 2003).

Performance of MMPI-2 Validity Scales

Considering the performance of the MMPI-2 Basic scales, the family of F scales surprisingly, differentiated TBI simulators from controls and community TBI patients. Like the Dearth et al. (2005) study, the F scale may indeed show promise in detecting simulated TBI. Perhaps it would have outperformed the FBS in this study given that the mean score of community TBI patients exceeded the TBI simulators on the FBS, but were not significantly different.

Performance of MMPI-2 Clinical Scales

The pattern of elevation on the Clinical scales was generally consistent with previous research in the area. In addition to the well documented elevations on scales 1, 2, 3, 6, 7, and 8. Additional differences were observed on scale 0. MMPI-2 Clinical scales 6 and 8 obtained significant differences among all three groups. These results are generally consistent with previous research examining MMPI scores with groups of analog cognitive malingers and persons involved in financially compensable litigation for head injuries (Berry, et al., 1995; Heaton, et al., 1978; Larrabee, 2003; Ross, et al., 2004).

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Applications of Neurocorrection Scales for Clinical Practice

The clinical practice of evaluating individuals who might be suspected of malingering in neuropsychological contexts involves a multidimensional procedure that utilizes tests that have been validated for this purpose (Iverson & Binder, 2000). The MMPI-2 is most often used in these circumstances to enhance clinician sensitivity for individuals who may exaggerate their cognitive complaints. The neurocorrection scales may offer an improved method of detecting cognitive exaggeration.

Considering the neurocorrection scales and their ability to differentiate between TBI simulators and community TBI patients, the Artzy scale produced a clinically small effect. Approximately 35% of the scores on this scale were produced by both TBI patients and the simulators. Although this figure is unacceptable in clinical practice, it is approximately half the percentage than the overlap generated by the FBS (66%). However, more research needs to be directed at validating the neurocorrection scales as measures of cognitive exaggeration.

Limitations of the Current Study

There are a number of limitations associated with the current study. Due to the small sample size, it is possible that if there were additional participants, results would have produced coefficients that were more robust and not subject to variability due to low power. This may have proven that more than just one neurocorrection scale would have demonstrated clinically significant effect sizes for comparison against the FBS.

This study also employed the use of research credits as an incentive that were part of the course requirements in which the students were recruited. A more realistic

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monetary incentive such as those used by Dearth et al. (2005) may have produced better over-reporting response sets by the simulator group. Using archived records dating 2-3 years prior to the study may have also influenced test scores. Using a randomized design for the TBI group at the time of evaluation, additional screening measures, and temporally consistent data would decrease threats to internal validity.

There are also inherent weaknesses with using analog designs compared to known- group designs. The primary feature limiting the generalizability of analog studies is the inability to simulate real-world forensic contexts. On the other hand, analog studies can, by randomization, exert a certain degree of control over experimental variables (Rogers, 1997).

Implications for Future Research

This study was limited by a number of factors; however, some significant and interesting findings did emerge and are worthy of further examination. The neurocorrection scales outperformed the FBS in the TBI simulator versus control contrast. This is the first study to look at the effectiveness of these scales for this particular purpose and, as a result, preliminary evidence suggests that the scales may improve the state of affairs when attempting to identify TBI simulation rather than the FBS. It also appears that the neurocorrection scales contain a different operating profile compared to the FBS, which captures a different set of symptoms and deceptive strategies rather than just simulation of cognitive impairment.

This study suggests it may be more prudent to employ neurocorrection scales in lieu of the FBS for patients who complain of late effects of mild head injury. Results

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from the current study suggest that at least one, if not a few of the neurocorrection scales, may offer clinical utility in differentiating TBI simulators in forensic contexts. Future studies might focus on adding comparison groups in addition to TBI, as well as employ scenarios involving different levels of information provided to participants about TBI sequelae.

For example, depression and emotional dysregulation become more salient as TBI symptoms persist (Reitan & Wolfson, 1997). This information would likely influence the sensitivity of the neurocorrection scales given their strict cognitive content. In addition, using known-groups of probable malingerers would also provide interesting evidence of the head-to-head comparability of the scales under real-world forensic contexts.

Lees-Haley, Iverson, Lange, Fox, and Allen (2002) explicitly state that the MMPI-2 does not meet the criteria for legal admissibility of psychological tests regarding neurological patients because it does not measure brain injury. This study perhaps provides further evidence that the use of the MMPI-2 as an indicator of malingering in forensic neuropsychological evaluation must be further explored. As Arbisi and Ben-Porath (2003) suggest, accurate normative studies aimed at cross-validating the neurocorrection scales with the content and component scales of the MMPI-2 rather than the clinical scales are needed. With the introduction of the MMPI-2 Restructured Clinical (RC) scales and the common factor of "Demoralization" represented in scale D (Tellegan, et al., 2003), another opportunity exists to examine profiles of patients with various types of neurological compromise.

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Perhaps the most important question not yet investigated is, if one is using the MMPI-2 in a forensic neuropsychological evaluation, how much weight should be placed on it as a measure of TBI simulation? Perhaps it is in the best interest of the field of psychological assessment to continue to pursue the answer to this question. It may also be prudent to continue to examine novel and innovative strategies to detect cognitive malingering. Perhaps the self-report format is too susceptible to deceptive responding, particularly if the measure has items that are not specific to neurological conditions. It is in this spirit that future studies evaluating the MMPI-2 in forensic neuropsychological contexts consider using the various neurocorrection scales as indicators of TBI simulation.

The current study suggests that there may be an alternative to the FBS in forensic neuropsychological evaluations that employ the MMPI-2. It also demonstrated that the neurocorrection scales do not meet standards that would consider them as valid measures of cognitive malingering. In fact, the problem of symptom nonspecificity still remains, even with the narrow neurocorrection scales. The item bias problem remains and calls into question the clinical utility of the MMPI-2 as a measure of neuropsychological exaggeration in forensic evaluations.

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Tables

Table 1

Age and years of education for three groups.

	TBI Simulators		Student controls		TBI	
Variable	Mean	SD	Mean	SD	Mean	SD
Age	21.6_a	5.3	20.7_a	3.3	40.9_b	13.1
Education	12.8	3.1	13	2.8	13.23	1.96

Note: Means that do not share subscripts differ significantly at p = .05 level. Education represented in years.

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Table 2

Gender and ethnic composition for three groups.

	TBI Simulators		Student controls		TBI	
	Frequency	%	Frequency	%	Frequency	%
Gender						
Male	5	33.3	6	35.3	8	36.4
Female	10	66.7	11	64.7	14	63.6
Ethnicity						
Caucasian	15	100	14	82.4	20	91
American Indian	0	0	1	5.9	2	9.1
African American	0	0	1	5.9	0	0
Hispanic	0	0	1	5.9	0	0

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Table 3

Analysis of variance comparing groups on MMPI-2 neurocorrection scales and the FBS.

		TBI simulators (n=15)		Student controls (n=17)		TBI (n=22)		ANOVA	
Scale	n	Mean	SD	Mean	SD	Mean	SD	*F (2, 54)*	η^2
Gass	14	10.3_a	3.7	2.1_b	1.8	7.5_a	3.0	34.1*	.57
Alfano	13	9.7_a	1.9	3.4_b	1.9	7.1_a	3.0	28.1*	.53
Artzy	17	11.1_a	2.2	6.4_b	2.5	7.7_b	3.0	13.7*	.35
Gass & Russell	42	23.5_a	4.9	12.4_b	3.4	21.5_a	5.2	27.3*	.52
Van Balen	22	12.9_a	3.5	3.9_b	3.1	13.5_a	4.7	33.3*	.57
FBS	43	18.4_a	3.7	11.7_b	3.6	21.0_a	6.0	20.3*	.44

Note: n - number of items contained within each scale. η^2- eta-squared measure of effect size. Means and standard deviations derived from raw scores. Means in the same row that do not share subscripts differ significantly in the Tukey HSD comparison, * $p < .05$.

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Table 4

Pairwise comparison of groups using converted Cohen's d effect size estimates for MMPI-2 neurocorrection scales and the FBS.

Scale	TBI Simulators v. Controls	TBI v. Controls	TBI Simulators v. TBI
Gass	2.9	2.2	.86
Alfano	3.3*	1.5	.59
Artzy	2.0	.50	1.3
Gass & Russell	2.7	2.4	.40
Van Balen	2.8	2.3	.14
Fake Bad Scale	1.8	1.8	.51

Note: Pooled standard deviations based on unequal group size. Interpretation of effect sizes; 0-1: minimal effect; 1-2: small effect; 2-3: moderate effect; and 3-4: large effect.
*Meets clinically significant criteria (Zakzanis, 2001).

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Table 5

Logistic regression analysis for Alfano and the FBS predicting group membership.

Scale	B	SE	Wald χ^2	Odds ratio	p
Alfano	-1.6	.78	4.3	.21	.04
FBS	-.52	.18	8.8	.56	.003

Note: B – unstandardized regression coefficient. SE – standard error.

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Table 6

Classification frequencies for Alfano and the FBS

		Predicted	
Scale	Observed	TBI simulator	Control
Alfano	Simulator	13	2
	Control	1	16
FBS	Simulator	11	4
	Control	3	14

Note: Hit rate – overall percentage correctly classified.

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Table 7

Sensitivity, specificity, and hit rate for Alfano and the FBS.

Scale & cutoff	Sensitivity	Specificity	Hit Rate
Alfano (≥ 7)	86.7	94.1	90.6
FBS (≥ 18)	73.3	82.4	78.1

Note: Percentages for TBI simulators and student controls.

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Appendix A.

Participant Information and Consent Form

Title: The Effectiveness of Special Correction Scales of the MMPI-2

Investigators

Paul Dukarm, Dept. of Psychology, The University of Montana, Missoula MT. 59812

243-6347

Dr. Stuart Hall, Dept. of Psychology, The University of Montana, Missoula MT. 59812

243-5667

Special Instructions to the potential subject

Thank you for considering participating in this study. This consent form may contain words that are unfamiliar to you. If the contents of this form are unclear, please as the person who gave you this form to explain it to you.

Purpose

The purpose of the current study is to examine how effective certain scales contained within a common measure of personality are at distinguishing between different types of people . By signing the form below, you are giving your voluntary consent to participate in this research study.

Procedures

As a participant in the current study, you will asked to provide "true" or "false" answers to a variety of questions about personality features. Your answers to these questions will be kept completely confidential. The personality form can take between 35 minutes to approximately 1-1/2 hours to complete. Completion of the form will take place in the Skaggs building, Room #246, located at the top of the stairs on the second floor, directly above the psychology department.

Risks / Discomforts

As a research participant, it is expected that the amount of discomfort you will experience as a result of this study will be minimal. It is possible that some of the questions on the inventory may cause you to feel uncomfortable due to their personal nature. If you

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become uncomfortable, please feel free to discuss your concerns with the research examiner or contact the principal investigator or faculty supervisor at the numbers provided above. Additionally, you may feel fatigued due to the length of the inventory. If you feel as if you need to take a break from answering the questions, you will be allowed to take a brief break when it is needed.

Benefits

Participating in the current study may benefit you by assisting you in obtaining three (3) experimental research credits and providing you with exposure to scientific research in psychology. Your participation will also provide very beneficial information to researchers and professionals in the field of psychology.

Confidentiality

The information you provide will be held strictly confidential by the research examiners (*see limitations below). Your name will not be marked on the answer sheet or questionnaire. However, if you agree to participate in this study, you will need to sign this form, which will be kept in a separate and locked filing cabinet from all testing materials. We will have you note your age, gender, ethnicity, and years of education for demographic purposes, but this personal information will not be attached to this form that contains your name. All demographic information will be kept separate from your response form, and will be used for data analysis purposes only. You will be assigned an identification number that will be used to help the research team keep your data forms organized. The information you provide will be read only by the principal investigator (Paul Dukarm), the faculty supervisor (Dr. Stuart Hall), and individual research assistants. Your answers to the inventory will be kept a minimum of 5-years after the study has ended, however the demographic form will be destroyed at the conclusion of the current study. The data from this study will be used for research publication purposes only, as well as presentations for research at academic conferences.

**There are certain situations in which confidentiality may be breached. If you indicate that you have a desire to harm yourself or someone else during the course of this experiment. If this situation occurs, you will be provided with information on where you may obtain mental health services. Because of this, we also require that you provide your name and your telephone number below. If you do not have a telephone number, please indicate a phone number where you can be reached.*

_____ _____

Print Name Telephone Number

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Although there is minimal risk associated with your participation in this study, The University of Montana requires that the following paragraph be included in all consent forms. "In the event that you are injured as a result of this research you should individually seek appropriate medical treatment. If the injury is caused by the negligence of the University or any of its employees, you may be entitled to reimbursement or compensation pursuant to the Comprehensive State Insurance Plan established by the Department of Administration under the authority of M.C.A., Title 2, Chapter 9. If the event of a claim for such injury, further information may be obtained from the University's Claims representative or University Legal Counsel. (Reviewed by University Legal Counsel, July 6, 1993."

Voluntary Participation / Withdrawal

Your participation in this study is entirely voluntary, and you may withdraw without penalty or any negative consequences. If you choose to withdraw, all your records will be destroyed, ad the data you provided will not be used in this study. If you decide to withdraw from this study, you will still receive your experimental credits.

Questions

If you have questions about this study now or at any time during the examination, please feel free to ask the examiner. Additionally, you may contact the principal investigator (Paul Dukarm, 243-4521) if you have any further questions about the study. We will not be able to give you feedback regarding your responses, however, you will be provided with additional information at the conclusion of the study. This information will be presented in the form of a debriefing sheet. If you have any questions regarding your rights as a research participant, you may contact the Institutional review Board Chair at 243-6670.

Subjects Statement of Consent

I have read the above description of this study and have been informed of the benefits and risks involved. All of my questions have been answered to my satisfaction, and I have been provided with the principal investigators contact information and the faculty supervisor in the event that I have concerns or questions in the future. By signing below, I voluntarily agree to participate in this study and give my consent to the examiners to use the information I provide for the purposes of this experiment.

_____

Participant's name - Printed

_____

Participant's signature

_____

Examiner's signature

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Appendix B.

UM Participant Information Form

Code _____

Age_____

Ethnicity: (Please Check One)

American Indian _____
Asian _____
African American _____
Hispanic _____
Caucasian _____

Years of Education _____

Please answer the questions below

Have you ever experienced a head injury or other neurological condition (e.g. concussion, seizures, migraines)? Yes____ No _____. Please explain. LOC____ PTA_____

_____

_____

Have you ever seen a psychiatrist, psychologist, or other mental health specialist for psychological conditions? Y __ N__
Please explain.

_____

_____

MMPI-2 Basic Scales T-Score

L ____
F ____
K ____
1 ____
2 ____
3 ____
4 ____
5 ____
6 ____
7 ____
8 ____
9 ____
0 ____

Special Correction Scales - Raw

Gass _____

Gass & Russell _____

Van Balen et al. _____

Alfano et al. _____

Artzy _____

(Research staff only) Manipulation check: YES____ NO_____

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Appendix C.

MPM Participant Information Form

Code _____

Age _____

Ethnicity: (Please Check One)

American Indian _____
Asian _____
African American _____
Hispanic _____
Caucasian _____

Years of Education _____

Neurological Data

LOC _____

PTA _____

GCS _____

PTS yes___ no ____

MMPI-2 Basic Scales T-Score

L ____
F ____
K ____

1 ____
2 ____
3 ____
4 ____
5 ____
6 ____
7 ____
8 ____
9 ____
0 ____

Neurocorrection Scales - Raw

Gass _____

Gass & Russell _____

Van Balen _____

Alfano _____

Artzy _____

FBS _____

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Appendix D.

Head Injury Severity Classification Criteria

	Traumatic Brain Injury	
Measure	Mild	Moderate
Loss of Consciousness	$\leq$ 20 minutes	20 minutes - 36 hours
Post-traumatic Amnesia	$\leq$ 24 hours	1 - 7 days
Glascow Coma Scale	13 - 15	9 - 12

Adapted from Williamson, Scott, and Adams (1996).

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Appendix E.

Instructions for Psychology Student Control Group.

Instructions will be read as follows:

" This inventory is comprised of 567 items that require you to answer either "true" or "false" to a variety of questions regarding personal interests and behavior. Please read each item carefully and completely fill in the appropriate circle on your answer form. It is important for the purposes of this study that you answer the items as honestly as possible, and if you come to a statement that you are unsure, answer the item whether it is "mostly true" or "mostly false". If you have any questions, please ask the examiner. When you are finished, please put your answer booklet in the envelope and turn it in to the examiner.

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Appendix F

Instructions for Student Head Injury Simulation Group

" This inventory is comprised of 567 items that require you to answer either "true" or "false" to a variety of questions regarding personal interests and behavior. Please read each item carefully and completely fill in the appropriate circle on your answer form.

While responding to the inventory, please pretend that you have experienced brain damage from a head-on motor vehicle accident. You hit your head against the windshield and were knocked out for about 15-minutes. Afterwards, you felt "dazed" so you were hospitalized overnight for observation. Because the driver of the car is at fault, you have decided to go to court and obtain compensation from the person responsible. During the next few months following the accident, the negative effects begin to disappear. Your lawsuit has not yet been settled, and your lawyer has told you that you may get more money if you look like you are still suffering from brain damage.

As you pretend to be this accident victim, try to respond to each item as a patient who is trying to appear brain damaged in order to get money from the lawsuit. Your performance on the inventory should convince the examiner as well as the people involved in deciding the outcome of your lawsuit that you are still suffering from brain damage. If you are able to convince the examiner of your condition, you will be given an extra (3) research credits for your participation.

Note: Instructions adapted from Thombaugh (1997), Rose et al. (1995), and Huskey & Hall (2003).

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Appendix G.

Debriefing and Referral

Instructions to Examiner for Debriefing:

"*Thank you for your participation in this experiment. You have just provided us with valuable information that will help us ascertain whether the inventory you just completed is helpful in allowing clinicians to detect persons who deliberately fake brain damage on tests of personality. Although the study was a simulation of neuropsychological impairment and litigation, the information you provided will nonetheless provide us with preliminary information regarding continued research in this are.*

For control group:
As you were told prior to completing the MMPI-2, you will be given (3) research credits for your participation.

For experimental group:
You will receive six (3) credits for your participation.

In the event that a participant has questions regarding the answers they provided or requests psychological services, they will be provided with the following information.

Contact and Referral for Psychological Intervention

Project Coordinator – Paul Dukarm, M.A. 243-4521

Project Supervisor – Stuart Hall, Ph.D. 243 – 5667

CAPS - Counseling and Psychological Services - Curry Health Center
University of Montana, 243- 4711.

CPC – Clinical Psychology Center – 1444 Mansfield Ave., University of Montana, 243-2367.

WMMHC – Western Montana Mental Health Center – 1315 Wyoming, Missoula MT., 532-9700

Providence Center - St. Patrick's Hospital, Missoula, MT.

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Appendix H.

Results of Analysis of Variance Comparing Groups on MMPI-2 Validity and Clinical Scales

Analysis of variance procedures for the Validity and Clinical scales were conducted to examine the current results in relation to previous research. Specifically, inspection of within and between groups differences on each scale would provide evidence of the consistency of scores related to previous studies examining TBI simulators and controls.

Appendix I presents means, standard deviations, and results of one-way ANOVA for MMPI-2 validity scales. Strength of association between each validity scale and group mean score is displayed as the eta-squared (η^2) effect size statistic. Tukey's HSD test post hoc alpha levels were conducted in order to evaluate significant differences for each group pair. No significant differences between groups were observed on the VRIN, TRIN, L, and K validity scales.

One-way ANOVA revealed significant differences among groups on the F scale, [$F(2, 54) = 38.9, p < .05, \eta^2 = .60$]. Tukey's HSD revealed that the TBI simulators differed significantly on the F scale from both controls and the community TBI patients, $p < .05$. There were no significant differences observed between community TBI patients and student controls.

Significant differences were also observed among groups for the F(b) scale,[$F(2, 54) = 18.7, p < .05, \eta^2 = .42$]. Tukey's HSD revealed significant differences between the TBI simulators and both community TBI patients and controls, $p < .05$. No significant differences were observed between the community TBI patients and controls.

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The F(p) scale demonstrated significant differences among the 3 groups, [F (2, 54) = 9.1, $p < .05$, scale $\eta^2 = .26$]. Tukey's HSD indicated that the TBI simulators produced mean scores that were significantly different from both the community TBI patients and controls, $p < .05$. No significant differences were observed between the community TBI patients and controls.

Analysis of variance comparing groups on MMPI-2 Clinical scales

The one-way ANOVA results revealed significant differences on scales 1, 2, 3, 6, 7, 8, and 0 across groups and are displayed in Appendix J. Significant mean differences were observed among groups on scale 1, [F (2, 54) = 47.3, $p < .05$]. Scale 1 generated a large overall effect size across groups, $\eta^2 = .65$. Tukey's HSD revealed that TBI simulators and community TBI patients differed significantly from controls, $p < .05$, but not from each other.

A one-way ANOVA revealed significant mean differences on scale 2, [F (2, 54) = 23.9, $p < .05$, $\eta^2 = .48$]. Tukey's HSD revealed that TBI simulators and community TBI patients also differed significantly from controls, $p < .05$. The TBI simulators did not significantly differ from community TBI patients.

Significant differences were also observed across groups on scale 3, F (2, 54) = 21.2, $p < .05$, $\eta^2 = .45$. As with the two previous scales, Tukey's HSD revealed that the TBI simulators and community TBI patients differed from controls, $p < .05$, but not each other.

No differences were observed between groups on scale 4. The ANOVA revealed nonsignificant between group differences in mean scaled scores, [F (2, 54) = 3.9, $p = .05$, $\eta^2 = .13$].

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Significant mean differences among groups were produced for scale 6, [F (2, 54) = 17.6, $p < .05$, $\eta^2 = .41$]. Tukey's HSD revealed that TBI simulators differed significantly from control and community TBI patients, $p < .001$. Community TBI patients significantly differed from controls, $p < .05$.

Scale 7 also produced significant differences between groups, [F (2, 54) = 9.8, $p < .05$, $\eta^2 = .28$]. Tukey's HSD revealed TBI simulators and community TBI patients differed significantly than controls, $p < .05$. TBI simulators did not significantly differ from the community TBI patient group.

One-way ANOVA revealed significant differences on scale 8 across groups, [F (2, 54) = 17.2, $p < .05$, $\eta^2 = .40$]. Tukey's HSD revealed that the TBI simulators and community TBI patients significantly differed from controls, $p < .05$. Community TBI patients also differed from controls and TBI simulators, $p < .05$.

No significant differences were observed between groups on scale 9. The ANOVA revealed nonsignificant between group differences in mean scaled scores, [F (2, 54) = 3.8, $p = .05$, $\eta^2 = .13$].

Scale 0 produced significant mean differences among groups, [F (2, 54) = 7.1, $p < .05$, $\eta^2 = .22$]. Tukey's HSD test revealed that the TBI simulators and community TBI patients significantly differed from controls, $p < .05$. No significant differences were found between TBI simulators and community TBI patients.

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Appendix I.

Analysis of variance comparing groups on MMPI-2 Validity scales.

	TBI Simulators (n=15)		Student controls (n=17)		TBI (n=22)		ANOVA	
Scale	Mean	SD	Mean	SD	Mean	SD	*F(2, 54)*	η^2
VRIN	60.1	15.3	53.8	10.6	55.5	11.1	1.1	.04
TRIN	61.8	13.9	54.1	5.0	57.6	7.2	.9	.10
F	97.1_a	20.9	57.1_b	12.1	59.5_b	10.2	38.9*	.60
F(b)	91.1_a	23.4	55.0_b	21.1	56.1_b	10.9	18.7*	.42
F(p)	83.1_a	26.8	55.8_b	21.3	53.7_b	18.6	9.1*	.26
L	53.1	12.8	45.5	12.6	55.0	10.2	3.7	.13
K	42.4	7.6	49.5	9.5	47.7	7.5	3.2	.11

Note: η^2- eta-squared measure of effect size. Means in the same row that do not share subscripts differ significantly in the Tukey HSD comparison,* $p < .05$.

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Appendix J.

Analysis of variance comparing groups on MMPI-2 K-corrected Clinical scales

	TBI Simulators (n =15)		Student controls (n =17)		TBI (n =22)		ANOVA	
Scale	Mean	SD	Mean	SD	Mean	SD	*F (2, 54)*	η^2
1K	76.3_a	13.0	40.2_b	10.5	74.1_a	13.0	47.3*	.65
2	77.0_a	16.4	48.0	8.8	70.1_a	12.3	23.9*	.48
3	77.6_a	16.0	50.2_b	9.1	79.4_a	17.6	21.2*	.45
4K	62.5	14.4	50.5	11.6	60.2	12.5	3.9	.13
6	80.3_a	19.9	51.0_b	8.9	62.2_c	12.5	17.6*	.41
7K	62.9_a	18.3	44.7_a	14.5	66.1_b	14.8	9.8*	.28
8K	81.1_a	21.3	46.4_b	16.3	66.6_c	13.7	17.2*	.40
9K	64.9	11.8	56.4	13.9	52.5	14.2	3.8	.13
0	59.7_a	15.3	44.5_b	7.5	52.0_a	12.7	7.1*	.22

Note: η^2- eta-squared measure of effect size. Means in the same row that do not share subscripts differ significantly in the Tukey HSD comparison, *p < .05.

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