The Social Consequences of Mild Head Injury and Executive Dysfunction

by

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Abstract

Mild head injury (MHI) is a serious cause of neurological impairment as is evident by the substantial percentage (15%) of individuals who remain symptomatic at least 1-year following "mild" head trauma. However, there is a paucity of research investigating the social consequences following a MHI. The first objective of this study was to examine whether measures of executive functioning were predictive of specific forms of antisocial behaviour, such as reactive aggression, impulsive antisocial behaviour, behavioural disinhibition, and deficits in social awareness after controlling for the variance accounted for by sex differences. The second objective was to investigate whether a history of MHI was predictive of these same social consequences after controlling for both sex differences and executive functioning. Ninety university students participated in neuropsychological testing and filled out self-report questionnaires. Fifty-two percent of the sample self-reported experiencing a MHI. As expected, men were more reactively aggressive and antisocial than women. Furthermore, executive dysfunction predicted reactive aggression and impulsive antisocial behaviour after controlling for sex differences. Finally, as expected, MHI status predicted reactive aggression, impulsive antisocial behaviour, and behavioural disinhibition after controlling for sex and executive functioning. MHI status and executive functioning did not predict social awareness or sensitivity to reward or punishment. These results suggest that incurring a MHI has serious social consequences that mirror the neurobehavioural profile following severe cases of brain injury. Therefore, the social sequelae after MHI imply a continuum of behavioural deficits between MHI and more severe forms of brain injury.

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List of Abbreviations of Tests and Questionnaires

1) BIS-11: Barratt Impulsiveness Scale-11
2) BIS/BAS Scale: Behavioural Inhibition/Behavioural Activation System Scale
3) BPAQ: Buss and Perry Aggression Questionnaire
4) CTONI: Comprehensive Test of Nonverbal Intelligence, Pictorial Analogies
5) Letter-Number: Letter-Number Sequencing
6) MC-8: Mental Control task (switching condition)
7) NEPSY-B: Auditory Response Set
8) SRP-III: Self-report Psychopathy Scale III R11
9) Stroop-3: Stroop Colour-Word Interference Task
10) ToM: Theory of Mind
11) Trails-4: Trail Making Test, Number-Letter Sequencing

The social consequences of mild head injury and executive dysfunction

The neurofunctional implications of closed head injury

Head trauma is a serious cause of neurological impairment across the lifespan. In particular, traumatic brain injury (TBI) refers to injury to the brain as a result of a sudden force or impact. TBI can be further differentiated into open-head (OHI) and closed-head injury (CHI); the former consists of injuries in which the cranium is penetrated by a projectile which damages the underlying brain tissue, whereas in the latter injury the brain is propelled against the bony projections of the intact cranium (Kolb & Whishaw, 2003). OHI, however, is observed in research settings less often due to high fatality rates (related to increased infection, blood loss, and diffuse brain damage; Thurman, Alverson, Dunn, Guerrero, & Sniezek, 1999), whereas research on CHI is more prominent as these injuries are predominantly centralized in the fronto-temporal cortices and has a range of severity levels.

The physical dynamics of CHI is referred to as the "coup-contre coup" injury, as an initial trauma propels the brain against the inner topography of the skull ("coup"), then in the opposite direction, once again making contact with the skull ("contre-coup"; Kolb & Whishaw, 2003). Furthermore, the orbital and the temporal bones form bony inner convolutions which make the fronto-temporal cortices particularly susceptible to injury. Also, rotational forces from the impact result in the shearing of white matter and diffuse axonal injury (DAI). DAI is prevalent across the entire spectrum of impact injuries, as twisting and shearing of axons can occur with minimal force to the head suggesting that mild head injury (MHI) may have potentially serious implications. Kushner (1998) found a positive relationship between force and amount of axonal injury, as greater amounts of force resulted in more axonal damage. Furthermore, Arfanakis et al. (2002) found that

patients with mild traumatic brain injury (mTBI) had a significant reduction of anisotropy using Diffusion Tensor Imaging, which indicated white matter abnormalities due to DAI in patients with relatively minor head trauma. Ultimately, the underlying neuropathology of CHI is a function of a continuum of severity levels. Therefore, the neurobehavioural correlates of MHI should be similar to those of TBI due to the susceptibility of the frontal lobes to the entire spectrum of CHI. Focal lesions, hematoma, and cortical edema are primarily a result of moderate and severe brain injuries which tend to be identifiable with traditional structural neuroimaging scans. However, MHI might not reveal gross intracranial abnormalities on neuroradiological scans (Giza & Hovda, 2001) which makes the assessment of MHI a complex enterprise.

Diagnosing traumatic brain injury: Clinical definitions, misnomers, and anomalies

TBI is acutely diagnosed based on the Glasgow Coma Scale (GCS; Teasdale & Jennett, 1976) at the time of injury and intracranial abnormalities are predominantly verified with a Computed Tomography (CT) scan. Results from these initial measures are potentially corroborated with length of coma (LOC), post-traumatic amnesia (PTA), neuropsychological assessment, and perhaps more sensitive neuroimaging techniques. The GCS, itself, is a behavioural measure of consciousness designed to objectively assess depth of coma after a TBI. The scale consists of 15 total points distributed across 3 subscales: eye-opening (ranging from 1-4), motor (ranging from 1-6), and verbal (ranging from 1-4). Thus, a score of 3 is indicative of complete unconsciousness, whereas a score of 15 indicates normal wakefulness. Head trauma resulting in a GCS score of eight or less is diagnosed as a severe TBI. Furthermore, PTA exceeding one day, LOC exceeding one day, and gross intra-cranial abnormalities on neuroimaging scans are also associated with

severe TBI (Lezak, Howieson, & Loring, 2004). A GCS score ranging from nine to twelve is diagnosed as a moderate TBI. PTA lasting from 1 to 24 hours, LOC from 1-24 hours, and evident intracranial abnormalities are also indicative of moderate TBI. Furthermore, CT scans can provide crucial corroborative evidence pertaining to the location and size of moderate and severe injuries (Besenski, 2002). Hence, CT scans are primarily used upon hospital admission after a TBI in order to provide neuroradiological evidence of intracranial damage.

Diagnosing mTBI, however, based on the current clinical criteria is a haphazard task. GCS scores ranging from 13-15, an "altered state of consciousness," and PTA of less than 60 minutes are all indicative of a MHI. However, a score of 15 also suggests appropriate functioning, an altered state of consciousness may also only indicate transitory disruption, and PTA of less than 60 minutes could go down to 'zero.' Therefore, all three indicators might also signal and be confusable with "normal" function. This poses a caveat in the assessment of mTBI, as patients may suffer from cognitive impairment due to their injuries in the absence of clinical GCS scores (Giza & Hovda, 2001; Witol & Webbe, 2002).

Furthermore, the GCS has been shown to be a poor predictor of long-term cognitive outcome in cases of mild and even moderate injury (Van der Naalt, Van Zomeran, Sluiter, & Minderhoud, 1999). There is also much debate pertaining to the validity of neuroimaging results in the diagnosis of mTBI. On the one hand, Kurca, Sivak, and Kucera (2006) have suggested that an organic basis for cognitive dysfunction after mTBI requires neuroradiological evidence. On the other hand, Bigler and Snyder (1995) have shown that neuropsychological deficits may persist after mTBI in the

absence of intracranial abnormalities on Magnetic Resonance Imaging (MRI). Furthermore, Gurgoff, Giza, and Hovda, (2006) revealed that mild brain trauma in rats resulted in decreased cognitive performance on the Morris Water Maze Task in the absence of significant neuronal death. These results ultimately support the theory that neuronal dysfunction (not necessarily cell death) is also related to impaired cognition and behaviour. As a consequence, GCS scores and structural neuroimaging scans can potentially perpetuate the misconception that mTBI is inconsequential despite serious cognitive and behavioural sequelae. Thus, neuropsychological assessment of cognitive performance is particularly important in the diagnosis of subtle neurofunctional deficits (Lezak et al., 2004)

As many as 50% of TBI emergency room admissions are within the "mild" range. Many of these patients go on to suffer significant cognitive disturbances (Kolb & Whishaw, 2003). Concussive injuries, however, are assumed to endogenously resolve after approximately two weeks, as prolonged behavioural, emotional, and cognitive deficits after this time-frame are generally categorized as Post Concussive Syndrome (PCS). PCS has many negative social connotations associated with it, not the least of which is the notion that the person is malingering for secondary gain in order to obtain monetary compensation or attention. In reality, however, individuals who have a history of MHI can suffer from prolonged consequences as a direct result of their injuries. For instance, Moser and Schatz (2002) found that incurring 2 or more concussions was related to persistent cognitive deficits on the Repeatable Battery for the Assessment of Neuropsychological Status, reinforcing the premise that MHI may have enduring effects. Furthermore, college football players with a history of MHI (2 or more concussions)

performed significantly lower on the Trail Making Test-B, indicating impaired executive functioning following mild head trauma (Collins et al., 1999). Therefore, individuals with a history of MHI may exhibit persistent frontal lobe symptomatology (such as executive dysfunction) which makes MHI a serious medical concern.

Also of particular concern is the fact that mTBI is globally one of the most common causes of neurological impairment across the lifespan, resulting in approximately 57 million hospitalized cases worldwide (Langlois, Rutland-Brown, & Wald, 2006). Epidemiologically, the incidence rate of persistent symptomatic patients has been estimated in the literature at 15% of the mTBI population (Alexander, 1995). Unfortunately, mTBI statistics are plagued by inaccuracies and underestimations due to inconsistencies in clinical diagnoses and because many individuals self-diagnose, thereby not seeking appropriate medical attention after incurring a head injury. Incidence rates in the literature range from 100 to 300 per 100, 000 hospitalized cases each year (Bernstein, 1999; Carroll et al., 2004), suggesting a serious socio-medical issue. Furthermore, Vanderploeg, Curtiss, Luis, and Salazar (2007) found that a history of self-reported mTBI was related to significantly more marital problems, more employment issues, lower income, and a greater incidence of major depression than their non-injured counterparts.

Impact injuries are prevalent across the entire lifespan, but they reveal specific demographical trends. For example, men are at least twice as likely to incur a TBI across the lifespan. Furthermore, young men (18-34 years) are at the highest risk of incurring a TBI (Langlois et al., 2006). Children and seniors are more likely to incur a TBI due to falling, whereas adolescents and young adults are more likely to incur a TBI due to motor vehicle accidents, sports injuries, assaults, and general risk-taking. MHI appears to have

an even more diverse and alarming demographic as Segalowitz and Lawson (1995) have shown that between 25-37% of undergraduate university students have incurred a MHI. This finding indicates a substantial proportion of university students have sustained a MHI even though they are not generally social delinquents or extreme risk takers. This finding mitigates the stereotypical notion that individuals who experience a MHI possess an overwhelming predisposition for high-risk behaviour which makes them inherently different from their non-injured counterparts.

Chan et al. (2005) investigated the relation between MHI (as defined as GCS score ranging from 13-15) and long-term outcome assessed with the Glasgow Outcome Scale Extended (GOSE) and found that 28.7% of MHI patients received a score of 6 (out eight point categories) indicating disability after injury. Furthermore, 11.3% of patients showed relativity good recovery; however, they continued to suffer from persistent symptoms such as headache and dizziness. Interestingly, evidence of intracranial abnormalities upon admission to hospital was not predictive of long term disability.

The pathophysiology of concussion

The seminal work of Giza and Hovda (2001) has revealed that a pathophysiological, neurometabolic cascade occurs following mechanical injuries to the head which is not necessarily accompanied by gross tissue damage. Immediately following a head injury, voltage-dependent potassium (K^+) channels open, resulting in an efflux of K^+. Excitatory neurotransmitters (predominantly glutamate) are then indiscriminately released which bind to N-methyl-D-aspartate (NMDA) receptors, exacerbating the ionic shift of K^+. Elevated K^+ levels trigger neuronal depolarization, which in turn results in increased glutamate release (along with the opening of excitatory

channels, such as NMDA) and a greater shift in ionic concentration. The sodium-potassium pump is then activated in an attempt to re-establish ionic homeostasis. This is an active process which requires adenosine triphosphate (ATP), implying further glucose necessity. Furthermore, cerebral blood flow (CBF) is tightly coupled to glucose metabolism under normal circumstances; however, CBF can be reduced to as low as fifty percent of normal capacity after a head injury. Therefore, a decrease in CBF and a concurrent increase in glucose metabolism further exacerbate the cellular energy crisis, as CBF is responsible for supplying glucose to neurons. This abrupt increase in required glucose is eventually satisfied by glycolysis, which is an endogenous mechanism that produces usable cellular energy. A potentially dangerous product of accelerated glycolysis, however, is increased lactate production. Elevated lactate levels can result in neuronal dysfunction through acidosis and edema. Furthermore, increased lactate levels render neurons increasingly more susceptible to neuronal death following a subsequent injury.

Following this surge of excitation is a refractory period of spreading depression, in which multiple neural regions are simultaneously rendered in a state of neuronal depression, in which cognitive deficits are most pronounced. During this pathophysiological cascade, individuals may score within the normal range on the GCS and neuroimaging scans might not reveal intracranial abnormalities.

The histological events following MHI comprise complex processes which result in an alteration of neuronal functioning without "observable" pathology by means of structural neuroimaging or GCS. Therefore, the GCS and neuroimaging data may not be sensitive enough to detect subtle neuropathology following mild impact injuries.

Ultimately, the above mentioned issues pertaining to the persistent symptomatology following head trauma are essentially consequences of neurological dysfunction, particularly to the frontal lobes. As mentioned before, the frontal lobes are particularly vulnerable to impact injuries of a range of severity levels due to their anatomical location behind the jagged projections of the orbits. Furthermore, the functional role of the frontal lobes in higher cognitive processing and social behaviour make the consequences of injury to this region particularly detrimental to human behaviour. Therefore, a sound understanding of the underlying functional neuroanatomy of the frontal lobes is crucial in order to formulate hypotheses of cognitive and behavioural deficits after MHI.

The adaptive significance of the frontal lobes in humans

The frontal lobes comprise the cortical tissue anterior to the central sulcus and superior to the lateral fissure and they are reciprocally connected to both the posterior cortex and the subcortex. The association areas of the frontal lobes are of particular importance to adaptive human behaviour, as the prefrontal region mediates the complex cognitive skills that have come to define humanity, referred to as executive functions (Kolb & Whishaw, 2003). Executive functions include a constellation of higher-ordered cognitive skills such as, impulse control, abstract reasoning, problem-solving, sustained attention, and emotional regulation, which are products of modulated cortical and subcortical input. Therefore, the prefrontal cortex (PFC) is crucial in the integration of goal-directed behaviour with cognitively prescribed social norms in order to select the most appropriate behaviour to satisfy both the internal milieu and adhering to social context (Stuss & Alexander, 2000).

Phylogenetically, the frontal lobes are the most recent cerebral structures to have developed and are structurally massive in the primate brain, composing approximately one third of the entire human cortex (36.7%; Semendeferi, Damasio, Frank, & Van Hoesen, 1997). The exponential evolutionary development of the PFC in humans is most probably due to the increased environmental pressure for complex social behaviour. Therefore, a larger and more complex frontal lobe was crucial for the reproductive fitness of humans in socially demanding environments. Furthermore, the crucial role of the frontal lobe in social behaviour is also evident from an ontogenetic perspective. Human development of the PFC mirrors that of phylogeny, as the PFC is the final region to develop in humans, reaching cortical maturity in young adulthood (i.e. myelination; Fuster, 2002). Ultimately, mature human behaviour is clearly dissociable from the behaviour of both children and non-humans primarily as function of higher executive functions which are mediated by the frontal lobe.

Functional neuroanatomy of the orbitofrontal cortex (OFC)

The orbitofrontal region of the PFC is of particular importance to human social behaviour as it is reciprocally connected to numerous subcortical and cortical structures. The OFC comprises the ventral tissue of the PFC located posterior to the orbits and is divided into 5 neuroanatomical subdivisions. The anterior pole of the OFC consists of area 10 followed by areas 11 and 13 posteriorly, area 47/12 laterally, and area 14 medially (Wallis, 2007).

Functionally, the location of the OFC at the ventral pole of the PFC is crucial for the integration of multimodal sensory information (from all sensory systems) with the reward and motivational systems of the subcortex (Price, Carmichael, & Drevets, 1996;

Rolls, 1999). Furthermore, the lateral region of the OFC contains the secondary gustatory projection zone, while the medial region contains the secondary olfactory projection zone (the anterior olfactory nucleus; Elliott, Dolan, & Frith, 2000; Eslinger, 1999). Visual information and auditory information are projected to the OFC from the inferior temporal sulcus and the superior temporal gyrus respectively (Rolls, 1999), while somatosensory input is provided by the secondary projection zone of the parietal lobe (Wallis, 2007). Furthermore, the superior temporal sulcus provides the OFC with complex multimodal information. The lateral OFC is also crucial in the regulation of emotional and autonomic impulses through reciprocal connections with the amygdala and hypothalamus, such that homeostatically motivated behaviour is either activated or inhibited contingent on the social context determined by cortical processes. Thus, the OFC is often referred to as the "executive" region of the limbic system as it is richly connected to numerous limbic structures. The medial OFC subserves the processing of reward expectation based on previously learned cause-effect relationships (Roberts, 2006). Furthermore, the OFC is connected to the nucleus accumbens, which has a crucial role in the adaptive processing of reward (Haber, Kunishio, Mizobuchi, & Lynd-Balta, 1995). The OFC is also intricately connected to the dorsolateral PFC and the anterior cingulate cortex in order to selectively attend to relevant stimuli and to further elaborate the processing of sensory information and goal-directed behaviour. Thus, the OFC is a supramodal region which integrates multimodal sensory information with subcortical motivation in order to generate the most adaptive behaviour. Therefore, dysfunction to the OFC due to head trauma can result in a constellation of cognitive and behavioural deficits which can lead to antisocial behaviour.

Decoding incentive value of reinforcers and punishers: A precursor to decision-making

The OFC has a crucial role in the representation of reward, which is integral to decision-making (Wallis, 2007). For instance, Roberts (2006) concluded that the OFC is crucial for mediating behaviour based on previously learned cause-effect relationships and responding to the value of reward. He found that primates with lesions to the OFC failed to choose a novel object in order to obtain a conditioned reinforcer that has been previously paired with a food reward, whereas controls selected the conditioned reinforcer based on appropriately interpreting the underlying value. Furthermore, monkeys with lesions to the OFC perseverated in the absence of the re-exposure of unconditioned stimuli, whereas extinction occurred significantly more quickly in primates without OFC damage (Roberts, 2006). Thus, primates with an intact OFC can spontaneously modify their behaviour based on contextual changes in the environment, whereas primates with damage lack cognitive flexibility. Furthermore, Fellows and Farah (2005) revealed that damage to the OFC in humans created a lack of foresight into the future consequences of their behaviour indicating that the OFC also has an integral role in the expectation of future consequences and in goal selection. For example, neurons in the OFC of rats have been shown to be active during both the presentation of a conditioned stimulus and following a conditioned response (Schoenbaum, Setlow, Saddoris, & Gallagher, 2003). Thus, the OFC has a crucial role in the anticipation of reward which invariably has a substantial impact on social behaviour. Ultimately, perseveration on a behavioural pattern that evokes no real reward or that is potentially punishing may lead to antisocial and maladaptive behaviour.

Interestingly, learning is differentially impaired after orbitofrontal damage as performance deficits are observed on conditioned tasks, such as the object discrimination reversal task, while initial learning remains unimpaired (Roberts, 2006). It has also been well established in humans that performance deficits on the visual discrimination reversal task is differentially affected by orbitofrontal damage (Hornak et al., 2006). Impaired performance may be due to the inability to inhibit initially rewarded responses, such that patients continue to respond with a particular response set in spite of the fact that they may be punished for this behaviour.

Overall, then, damage to the OFC may result in deficits in context-based learning, such as perseveration and failure to adapt to changes in reinforcement contingencies (Rolls, 2004). The social implications of these data suggest that patients with orbitofrontal damage are more prone to making disinhibited, inappropriate, and potentially risky decisions.

Clinical case studies highlighting the importance of the OFC in social behaviour

Numerous case studies have exemplified the crucial role of the OFC in social behaviour and decision-making in humans. The seminal work of Harlow (1848) was probably the most famous depiction of behavioural change following an OHI. This case study described the aberrant and abrupt personality changes of railroad foreman, Phineas Gage, after a steel tamping rod penetrated his skull, damaging the anterior pole of his brain. Preinjury, Gage was described by both family and friends as a well-mannered leader. Interestingly and tragically, Gage underwent a drastic change in personality after his brain injury. The once mild-mannered leader was transformed into a vulgar, socially inappropriate individual. Although Harlow was unable to corroborate his anecdotal

evidence with standardized neuropsychological measures or neuroimaging techniques, he provided one of the earliest and most famous descriptions of the effects of prefrontal injury. Over a century later Damasio, Grabowski, Frank, Galaburda, and Damasio (1994) used advanced computer techniques to estimate the location of Gage's injury. As expected, the OFC was estimated to be crucially damaged.

Since Harlow's famous account of Phineas Gage, a plethora of cases studies have demonstrated the importance of the OFC in social behaviour and highlighted the maladaptive effects of orbitofrontal injury, such as poor impulse control, lack of social awareness, reactive aggression, and antisocial behaviour (e.g. Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Gratton & Eslinger, 1992). For example, Eslinger and Damasio (1985) describe a patient (E.V.R.) who had an orbitofrontal meningioma resected in adulthood, resulting in gross tissue damage to the OFC and a marked change in personality. Pre-injury, E.V.R. was described by family members as a responsible husband and father with a normal childhood. He had fruitful employment as he was promoted from his job as an accountant to comptroller, he was an active member of his church, and he was described by his siblings as a natural leader. Post-injury, E.V.R. underwent an extreme change in his personality as he began to make blatantly unwise financial decisions (peculiar for an accountant), was forced to claim bankruptcy, and was unable to maintain any sort of employment because of his (newly acquired) tardiness and disorganization. Surprisingly, previous neurological examination and neuropsychological assessment were unremarkable as E.V.R. scored in the average to superior range on all behavioural measures. Thus, he was diagnosed with emotional and psychological adjustment issues and was recommended to undergo psychotherapy, as he was deemed

not to have any organic frontal lobe impairment. Interestingly, E.V.R suffered from bilateral anosmia (inability to smell), indicative of perhaps a damaged anterior olfactory nucleus (located in the OFC), which appeared to be overlooked in the initial sessions.

His aberrant social behaviour continued to result in maladaptive decision-making and his wife eventually filed for divorce. Furthermore, E.V.R. was never able to maintain a healthy romantic relationship after his injury as he was remarried two months after his initial divorce and was divorced for a second time two years later. In addition, he decided to pursue another relationship that was most probably destined for failure as he negatively described his new fiancé as spoiled. Neuroimaging techniques (Eslinger & Damasio, 1985) verified that the damage was indeed predominantly localized to the OFC as the entire right hemisphere of his OFC and part of his left hemisphere OFC were ablated. Furthermore, functional neuroimaging scans with positron emission tomography (PET) revealed low blood flow in both frontal lobes, particularly in the right hemisphere, corresponding with the structural evidence.

More recent empirical studies have also revealed the relation between changes in personality and orbitofrontal damage. For instance, Barrash, Tranel, and Anderson (2000) found that patients with bilateral ventromedial lesions (revealed through MRI and/or CT) were more antisocial and emotionally dysfunctional than their control counterparts on the Iowa Rating Scales of Personality Change. The ventromedial patients suffered from various antisocial consequences including: blunted emotional experience, inappropriate affect, emotional lability, poor judgment, and social inappropriateness. Therefore, both empirical and clinical case studies have been extremely useful in revealing the relation between orbitofrontal damage and aberrant changes in social behaviour.

Acquired sociopathy and the developmental effects of orbitofrontal injury

Eslinger and Damasio (1985) also revealed in their case study that patient E.V.R. was capable of forming complex and sensible social decisions when given hypothetical situations and would discuss current events intelligently. Interestingly, however, was the disconnection between the factual knowledge that he possessed pertaining to social norms and the actual execution of socially adaptive behaviour in his own life. Thus, Eslinger and Damasio hypothesized that these learned social norms perhaps no longer evoked the corresponding somatic or visceral markers that tend to initiate normal social behaviour. They described this condition as an "acquired" sociopathy since E.V.R.'s aberrant social behaviour was "acquired" after his orbitofrontal injury and because his behaviour somewhat mimicked that of a sociopath (with the exception that sociopaths approach real-life and hypothetical situations in an identical manner). E.V.R.'s debilitating antisocial behaviour was overlooked by initial assessments due to his ability to formulate sensible decisions in a controlled environment which masked his inability to actually execute these socially appropriate behaviours under natural circumstances.

Anderson et al. (1999) found in their case study that time of injury was crucial for a specific antisocial behavioural presentation of orbitofrontal damage marked by instrumental antisocial behaviour. More specifically, prefrontal injury before two years of age produced a severely antisocial profile that mimicked psychopathic behaviour, such that individuals with brain injuries involving the frontal lobe behaved in a manipulative, predatory manner, and goal-directed manner. Similarly, Timonen et al. (2002) found that the risk for developing a mental disorder doubled after childhood TBI and was significantly related to criminality in men supporting the view that early injury to the

frontal lobes results in more severe antisocial behaviour and more severe social consequences later in life. Therefore, head trauma has been shown to result in antisocial behaviour due to dysfunction of the frontal lobes.

The orbitofrontal influence on decision-making, social awareness, and emotional control

Adaptive decision-making is a complex process that requires the integration of both cognition and affect. Damasio (1996) has hypothesized that adaptive human reasoning and decision-making require the interpretation of somatic markers. According to the somatic marker hypothesis, the OFC modulates the linkage between previously learned cause-effect relationships (such as the factual knowledge of consequences) and the somatic state or bioregulatory processes that are evoked by the learned contingency. For instance, individuals with orbitofrontal damage have been shown to perseverate on decks that resulted in a net loss of money on the Iowa Gambling Task, seemingly unable to modify their behaviour based on changes in reinforcement contingencies, whereas healthy controls have been shown to consistently select from decks that resulted in a net gain of money after about 40 or 50 selections. Furthermore, healthy individuals revealed a pattern of heightened autonomic arousal (measure with Galvanic Skin Response) when choosing from the maladaptive decks even before conscious recognition that these decks were disadvantageous (after approximately 10 trials), whereas patients with OFC damage failed to show this change in physiological arousal (Bechara, Damasio, Damasio, & Anderson, 1994; Damasio, Tranel, & Damasio, 1990). Therefore, visceral (or somatic) feedback can be crucial to the execution of socially appropriate behaviours, as injury to the OFC results in a disconnection between cause and effect, in which individuals may behave in an impulsive manner based solely on their current primal physiological

reactions, lacking the foresight to anticipate future consequences of the behaviour. Thus, the "acquired" sociopathic behaviour exhibited by E.V.R. was purportedly due to a failure to produce somatic markers in response to real-life situations, even though he was aware of the sensible solution under hypothetical circumstances. Ultimately, social behaviour is a complex construct that requires adaptive decision-making skills based on an intricate feedback system which integrates cognition and visceral activity, through the OFC.

Impaired social awareness after OFC injury can be extremely debilitating as human beings rely on an intricate set of adaptive social skills in order to survive in complex social networks. Social awareness has been shown to be related to prefrontal integrity, and orbitofrontal function in particular (Gallagher & Frith, 2003). For example, Stuss and Anderson (2004) have created a neurocognitive model of consciousness that is hierarchically organized and culminates in the prefrontal function with the most complex form of self-awareness, Theory of Mind (ToM). ToM is the ability to formulate mental state attributions of other people which involve not only an awareness of one's own experiences, but also the abstract ability to predict the intentions of others.

The neurocognitive basis of social awareness has been shown to range from simple facial processing to clinical syndromes of unawareness following cortical damage to the frontal lobes. Experimental studies have used functional neuroimaging techniques to study the relation between facial recognition and brain activity. For example, Kringelbach and Rolls (2003) used Functional Magnetic Resonance Imaging (fMRI) to investigate the brain regions that are activated during a facial expression reversal

paradigm, in which they found that the processing of changes in facial expression differentially activated the OFC and anterior cingulate cortex.

Clinical studies have focused on the onset of anosagnosia, following severe TBI, in which patients lack the self-awareness of their injuries (Hier, Mondlock, & Caplan, 1983). For instance, Lannoo et al. (1998) found that patients with moderate to severe TBI were impaired on tests of neuropsychological performance. However, no correlations were found between neuropsychological performance and subjective complaints regarding their injuries indicating that patients with deficient cognitive functioning lacked awareness of their deficits. Thus, both facial processing and anosagnosia have been shown to be useful in elucidating more simplistic aspects of social awareness.

Blair and Cipolotti (2000) addressed the more complex role of the OFC in social cognition, particularly the ability to modify behaviour in response to rapidly changing social cues, referred to as Social Response Reversal (SRR) theory. SSR addresses the human ability to modify behaviour based specifically on the perception of anger cues from fellow conspecifics. Interestingly, the OFC has been shown to be differentially activated when viewing angry, but not sad faces (Blair, Morris, Frith, Perret, & Dolan, 1999). Orbitofrontal involvement is integral to adaptive social functioning, as it is imperative to be able to detect physically or socially dangerous signals in the environment, such as angry faces. ToM is perhaps the most complex and definitively human social skill, as the ability to predict the emotions, cognitions, and behaviours of others requires self-awareness and perspective taking. Accordingly, the neural circuitry of ToM is highly complex and involves numerous structures. Ultimately, the PFC (particularly the OFC and the inferior frontal gyrus) is the primary integrating node of the

ToM network. Thus, numerous studies have revealed that ToM is profoundly impaired in patients with acquired brain injury (e.g. TBI, cerebrovascular accidents, anoxia, or cerebral infections; Valentine, Powell, Davidoff, Letson, & Greenwood, 2006) and severe TBI in both men and women (Bibby & McDonald, 2005; Rowe, Bullock, Polkey, & Morris, 2001). For instance, patients with TBI have been shown to be impaired on the "Reading the Mind in the Eyes" task (a facial recognition test of ToM) compared to controls (Havet-Thomassin, Allain, Etcharry- Bouyx, and Le Gall, 2006; Henry, Phillips, Crawford, Ietswaart, & Summers, 2006). Furthermore, ToM impairment as a result of TBI has been shown to be stable at least one year post-injury suggesting that deficits in ToM can be both profound and persistent (Milders, Ietswaart, Crawford, & Currie, 2006). Poor executive functioning (e.g. phonemic fluency) in TBI patients has also been shown to be related to ToM performance on the Reading the Mind in the Eyes task (Henry et al. 2006). This finding further reinforces the link between deficits in social cognition, TBI status, and frontal-lobe dysfunction, as individuals with frontal lobe damage due to TBI revealed significant impairments in social awareness. Ultimately, deficits in ToM are profoundly debilitating as people are constantly forced to engage in making mental state attributions during social interactions. Furthermore, deficits in interpreting the thoughts and emotions of others can potentially exacerbate antisocial behaviour caused by emotional/behavioural dyscontrol, as the inability to accurately interpret the mental states of others may lead the affected individual to misconstrue pertinent social information as harmful or threatening resulting in an inappropriate retaliatory response.

The neural circuit mediating the expression of emotion is composed of the OFC and several interconnected cortical and subcortical limbic structures including: the

amygdala, hypothalamus, anterior cingulate cortex, insular cortex, ventral striatum, and the hippocampus (Davidson, 2000). The OFC is specifically involved in the regulation of negative emotional expression through reciprocal connectivity with the amygdala. Furthermore, the OFC modulates the behavioural expression of autonomic arousal in response to stress and threat through connections with the hypothalamus-pituitary-adrenal axis (HPA). More specifically, the medial nucleus of the amygdala and the OFC are both connected to the hypothalamus, modulating the activation the HPA in response to environmental stressors. Increased activity of the HPA may result in increased activation of the dorsal periaqueductal gray in order to enable the production of defensive behaviour, such as: freezing, fleeing, and/or aggression depending on the intensity of the threat and the social context. Thus, focal lesions to the OFC result in dysfunctional behaviour in response to autonomic arousal, referred to as reactive or impulsive aggression (Blair, 2001). These patients form a homogeneous population that is impaired at inhibiting aggressive responses after provocation or threat (Blair 2001; Blair, 2004). This form of aggression is based on an inability to control primal aggressive reactions which are in contrast to goal-directed or instrumental aggression. Lack of inhibitory control after TBI predominantly results in antisocial levels of physical aggression and marked emotional lability. For example, TBI patients are more aggressive than their non-brain injured counterparts (Tateno, Jorge, & Robinson, 2003). Rosenbaum and Hoge (1989) found that head injuries were significantly related to antisocial levels of marital aggression (in men) reinforcing aggressive behaviour as a consequence of TBI. The OFC has also been shown to be differentially activated (as indicated by PET) when healthy individuals viewed angry faces of increasing intensity (Blair et al., 1999) and when anger

was induced in healthy individuals (Dougherty et al., 1999). Corroboratively, Blair and Cippolotti (2000) observed pathological levels of emotional dysfunction in patients with focal orbitofrontal lesions. Therefore, the OFC has been shown to be crucial for modulating the threat response, presumably through its connections to the limbic system.

Poor performance on neuropsychological measures of executive function has been shown to be related to aggressive behaviour (Giancola & Zeichner, 1994). For instance, the Stroop and Judgment of Line Orientation Tests have been shown to predict frequency of aggressive behaviour in a forensic sample (Foster, Hillbrand, & Silverstein, 1993). Furthermore, impulsive aggressive university students (ascertained through self-report) have been shown to perform significantly more poorly on the Wisconsin Card Sorting Task than their non-impulsive aggressive counterparts (Stanford, Greve, & Gerstle, 1997), suggesting an executive processing deficit in high functioning individuals with social difficulties, particularly in controlling aggression.

Current Study: The relation between MHI and social behaviour

Previous research has revealed the relation between TBI status and deficits in executive functioning, antisocial behaviour, and poor decision-making. These relations, however, have not been rigorously studied in high-functioning populations with MHI, such as university students. Undergraduates are a particularly intriguing population to study as their cognitive abilities are high functioning in general; nevertheless, they may still present with subtle, but deleterious behavioural and cognitive deficits in comparison to their non-injured counterparts. Furthermore, due to the alarming prevalence of mild head trauma across the lifespan (and especially during adolescence and young adulthood) it is of great importance to explore the social consequences of MHI in more detail.

Due to the vulnerability of the OFC in mechanisms of MHI and its associated role in behavioural control, individuals with a history of MHI are hypothesized to be more impulsive, reactively aggressive, antisocial, and less socially aware than their non-injured counterparts even after controlling for sex and executive functioning. Furthermore, poorer executive functioning is hypothesized to be related to higher levels of impulsivity, reactive aggression, and antisocial behaviour, as well as less social awareness after controlling for sex, as executive functioning is a putative measure of frontal lobe functioning. Finally, based on previous research examining sex differences in head trauma and social behaviour, men are hypothesized to be more likely to incur a MHI and to be more aggressive, antisocial, and impulsive than women. Ultimately, social deficits are expected to be present after MHI due to its impact on orbitofrontal function.

Hypothesis 1:

Consistent with earlier research, being male is expected to be positively correlated with physical aggression, verbal aggression, hostility, and anger. Poorer executive functioning is predicted to be related only to higher levels of physical aggression, but not hostility, anger, or verbal aggression, as only the items on the physical aggression specifically address issues of executive functioning, such as impulse control and reactive aggression, whereas the other factors more aptly describe mood state. However, beyond these variables, a history of MHI status is expected be additionally predictive of physical aggression after controlling for sex and executive functioning due to its relationship to OFC disruption as well as the particular relationship between the OFC and reactive impulsiveness. Because of this, MHI status is not expected to be a predictor of verbal

aggression, hostility, or anger, as these measures are more representative of mood states, as opposed to emotional dyscontrol.

Hypothesis 2:

TBI involving the frontal lobes has been shown to be related to maladaptive decision-making and antisocial behaviour; however, there is no known study to date that has investigated the relation between MHI and antisocial behaviour. Thus, the SRP-III R11 will be utilized in order to determine the presence of this relationship. Being male is hypothesized to be related to higher scores on the erratic lifestyle, antisocial behaviour, callous affect, and interpersonal manipulation subscales, as men tend to be more antisocial than women in general. Executive dysfunction is expected to be differentially related to higher erratic lifestyle and antisocial behaviour scores, but not the callous affect or interpersonal manipulation scales, as the former two factors contain items that best represent impulsivity and poor decision-making, whereas the latter two subscales require adequate executive functioning in order to perform instrumentally antisocial acts. However, over and above these variables a history of MHI is hypothesized to be predictive of the erratic lifestyle and the antisocial behaviour subscales due to its influence on OFC functioning, which has a crucial role in cognitive and affective processes, such as impulsive antisocial behaviour.

Hypothesis 3:

Previous research has shown that severe TBI is related to behavioural dyscontrol and impulsivity. Thus, it is predicted that being male, having executive dysfunction and a history of MHI will be related to increased levels of impulsivity. Specifically, it is hypothesized that MHI status will be a significant predictor of only BIS-motor (but not

BIS-attentional or BIS-nonplanning), after controlling for sex and executive functioning because only the items that of the BIS-motor subscale describe behavioural disinhibition which is a common consequence of TBI.

MHI status is also expected to be related to the Behavioural Inhibition System/Behavioural Activation System (BIS/BAS). Specifically, MHI is hypothesized to be predictive of lower BIS levels and higher BAS levels after controlling for sex and executive functioning, due to the relationship between MHI and disruption to the OFC.

Hypothesis 4:

Previous research has shown that ToM performance on the Reading the Mind in the Eyes task (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001) is detrimentally impacted by TBI. Research has also shown that impairments in the perception of angry faces are related to orbitofrontal injury. There is no such research to date that has been conducted on the relation between MHI and ToM; however, based on what has been found with regards to the relationship between social awareness and TBI involving the frontal lobes, it is hypothesized that subtle, yet similar deficits will be delineated between individuals with MHI and their non-injured counterparts. Therefore, it is predicted that MHI status be a significant predictor of ToM performance, after controlling for sex and executive functioning. Furthermore, poor executive functioning is expected to be related to lower ToM accuracy, as abstract decision-making is expected to be a crucial component in assessing the mental states of others.

Method

Participants

The participants in this study consisted of 90 undergraduate university students (27 men, 63 women) between the ages of 18 and 28 years ($M = 20.6$ years; $SD = 2.22$), recruited through the online undergraduate participant pool at Brock University in St. Catharines, Canada. Eighty-two participants were right-handed, 6 were left-handed, and 2 were ambidextrous, based on self-report. Self-report of a MHI was obtained based on answering "yes" to the following question on a demographic questionnaire (Appendix A), "Have you ever hit your head against a surface or object sufficient to alter your consciousness (e.g. loss of consciousness, vomiting, dizziness)?" Furthermore, a severity indicator was obtained by qualifying the statement, "Did it result in a concussion?" Over half of the sample (52%) reported incurring at least one MHI, while 31% of these individuals also reported acknowledged sustaining a concussion. Of those who reported sustaining a MHI, 32% reported requiring stitches and 81% incurred a single head trauma. Furthermore, 21% of individuals with a history of MHI sustained their injuries more than 10 years ago, 68 % incurred their injuries between 1 and 10 years ago, whereas only 11% incurred their injuries within the past year. Consistent with previous research investigating the lack of medical attention after incurring a MHI, 36% of the MHI sample did not seek hospitalized attention (n = 17).

Students received 2 participation hours in selected undergraduate courses in return for their participation. Furthermore, the study was strategically advertised as a "Cognitive Abilities" study in order to safeguard against a potential sampling bias in recruiting

individuals who have incurred a MHI due to any sort of overwhelming predisposing factor.

Two cases were deleted listwise from the sample due to a technical error with the tape player required for one of the executive functioning tasks. Furthermore, an additional case was deleted from the BIS-11 analyses due to the fact that the participant failed to answer several questions on that questionnaire. Therefore, the adjusted sample size for all analyses was 88, except for the BIS-11 which had 87 participants.

Materials

The materials consisted of a combination of neuropsychological assessment measures and self-report questionnaires. The neuropsychological measures were selected based on their specificity to frontal lobe/executive functioning (e.g. working memory, attention, behavioural inhibition, abstract reasoning, etc.). Thus, several subtests from larger standardized neuropsychological test batteries were selected in order to assess frontal-lobe functioning from a diverse array of neurocognitive measures. A stopwatch was used in order to record timed tasks. Writing materials were also provided for tasks that required the participant to record information. Furthermore, a tape player was required for the Auditory Attention and Response Set task.

Measures of executive functioning. The Pictorial Analogies subtest from the Comprehensive Test of Non-verbal Intelligence (CTONI; 1996) was administered in order to assess abstract reasoning skills, problem solving, and complex decision making. Participants were presented with four quadrants in which 2 of the 4 quadrants contained pictures that fulfilled an analogous relationship. Participants were then asked to match a

lone picture with another picture from a list of 5 options based on the analogous relationship formed by the initial pair within 30 seconds over 25 trials.

All 5 conditions of the Trail Making Test from the Delis-Kaplan Executive Function System (DKEFS; 2001) were administered; however, the Number-Letter Sequencing task was of crucial interest as it was designed to assess working memory, scanning, sequencing, cognitive flexibility and sustained attention. Participants were asked to find and connect numbers and letters in an alternating sequence (as quickly as possible) while maintaining both ascending numerical and alphabetical order (e.g. 1-A-2-B, etc).

The Auditory Attention and Response Set subtest from the NEPSY (Korkman, Kirk, & Kemp, 1998) was administered in order to assess sustained attention and impulse control. The testing materials for this task consisted of an audio tape and a box of coloured squares (e.g. blue, red, yellow and black). The Auditory Attention section required participants to listen to the audio tape (dictating random words at a rate of one per second) and to put a red square into the lid of the box immediately after hearing the word "red" while inhibiting responding to any other word. The Auditory Response Set subsection consisted of an audio recording (on the same audio tape) requiring participants to place a red square in the lid after hearing the word "yellow," a yellow square after hearing the word "red," a blue square after hearing "blue", and to inhibit responding to the word "black".

The Mental Control and Letter-Number Sequencing subtests from the Wechsler Memory Scale – III (WMS-III; 1998) were administered in order to assess working memory and online manipulation of information. Specifically, the Letter-Number

Sequencing task was administered to assess auditory working memory of verbal information, as it required participants to listen to a series of numbers and letters and then to report back the numbers first in ascending order followed by the letters in alphabetical order. Furthermore, the string of letters and numbers successively increased in size, increasing the working memory load. The Mental Control subtest was designed in order to test overly learned and prepotent information (e.g. the alphabet from A-Z) and to assess online manipulation of information, and was hence a working memory task. It required participants to perform a series of speeded mental manipulations (e.g. repeat the alphabet backwards) as quickly and accurately as possible.

The Stroop Colour-Word Interference subtest from DKEFS (2002) was administered in order to assess behavioural inhibition while being timed for speed of completion. All 4 conditions were administered, however; the classic inhibition task was of central interest to this study. In the inhibition task, participants were presented with names of colors written in different ink colors and were asked to report the ink color as quickly as possible, as opposed to the automatic or prepotent tendency to read the word, itself.

Self-report measures

The Reading the Mind in the Eyes test (Baron-Cohen et al., 2001) was administered in order to assess ToM accuracy. Participants were asked to look at photographs of eyes and to select the most appropriate descriptor of what the person in the photograph was thinking or feeling from 4 options in multiple-choice format over 36 trials.

The Barratt Impulsiveness Scale (BIS-1; Barratt, 1959) is a well known self-report measure of impulsivity as it has been revised 11 times (BIS-11; Patton, Stanford, & Barratt, 1995; Appendix B). It consists of 3 factors: motor, non-planning, and

attentional, in which participants were asked to indicate how much they agreed or disagreed with 30 statements on a 4-point Likert scale (never to almost always/always). The motor factor is particularly interesting as it is consistent with the neuropsychological definition of behavioural dyscontrol. For example, the item, "I act on impulse" is indicative of the inability to withhold a prepotent response, which may be related to clinical presentations, such as reactive aggression. The non-planning factor refers to a general myopia for the future. For example, the item, "I am more interested in the present than in the future" is indicative of the inability to plan for future events. The attentional factor refers to the inability to maintain concentration as exemplified in the item, "I don't pay attention."

Patton et al. (1995) calculated the internal consistency of the BIS-11 in several populations (using Cronbach's alpha), in which this questionnaire was shown to be a stable measure of impulsivity in undergraduate university students (.82), substance abuse patients (.79), general psychiatric patients (.83), and prison inmates (.83). They also found that the measure was predictive of impulsive tendencies in that they found undergraduates had significantly lower BIS-11 total scores than both patient groups ($p <$ .05). Male undergraduates and patient groups scored significantly lower than prison inmates, but no differences were found between male patients and undergraduates. Female undergraduates scored significantly lower than both female patient groups; however, no differences were found between female patient groups. Therefore, the BIS-11 is a valid and consistent measure of impulsive behaviour for a variety of populations, including undergraduate university students. Furthermore, the BIS-11 is sensitive to impulsive behaviour between populations that have marked impulse control and

antisocial behaviour tendencies, such as prison inmates and those that tend not to have issues with impulsivity, such as university students. The BIS-11 has also been shown to be related to neuropsychological measures of executive function. Keilp, Sackeim, and Mann (2005) revealed a negative correlation between scores on the BIS motor and attentional subscales and the Go/No Go, Verbal Fluency test, and Trail Making Test-B in a healthy sample. Furthermore, Barratt, Stanford, Kent, and Felthous (1997) found that self-reported impulsiveness on the BIS-11 was inversely correlated with frontal P300 amplitude and verbal skills. Thus, scores on the BIS-11 have been shown to be related to aberrant frontal-lobe activity as well as deficits in executive functioning. Ultimately, the BIS-11 (and the BIS-motor subscale in particular) appears to measure OFC dysfunction, such as behavioural disinhibition, which is also a sequel of head trauma.

Gray (1970) proposed that two complementary, motivational systems are responsible for the regulation of behaviour, The Behavioural Approach System (BAS) and the Behavioural Inhibition System (BIS). Thus, Carver and White (1994) devised the BIS/BAS scale (based on Gray's theory) in order to assess BIS/BAS levels in a variety of populations. The BAS is believed to be related to positive affect, appetitive behaviour, and is differentially activated in response to reward. In contrast to the BAS, higher scores on the BIS are related to greater sensitivity to punishment and are crucial for the inhibition of behaviour in response to aversive consequences. For example, elevated BIS levels have been shown to be related to the prevention of behavior that is expected to lead to punishment in the future (Knyazev & Slobodskoj-Plusnin, 2007). Ultimately, these systems are believed to work in synchrony with each other and imbalances in either system are related to different forms of psychopathology. For instance, an overactive BIS

is related to hypersensitivity to stressors, which are related to anxiety disorders (Arnett & Newman, 2000), whereas excessive activity of the BAS is related to impulsive, high-risk, and potentially antisocial behaviour (Carver & White, 1994). For example, Johnson, Turner, and Iwata (2003) examined the relation between BIS/BAS levels and diagnosis of psychiatric disorders, in which elevated BIS levels were related to anxiety disorders, whereas elevated BAS levels were related to increased drug abuse. Thus, in the former case individuals possess overactive perception of threat in the environment, whereas in the latter case individuals are overly driven by the subcortical drives of primal reward systems (e.g. nucleus accumbens). The OFC has a crucial role in both cases, as it is has a crucial role in regulation in processing of reward behaviour as well as in the adaptation of behaviour after changes in environmental contingencies. Furthermore, OFC dysfunction is implicated in the social consequences of head trauma. Therefore, the BIS/BAS is a valid measure of appetitive and aversive motivational systems which would be affected by MHI involving the frontal lobes. Therefore, the Behavioural Inhibition System/Behavioural Approach System Scale (Carver and White, 1994; Appendix C) was used to assess impulsivity and to determine responsiveness to reward and punishment. Participants were asked to indicate how much they agreed or disagreed with each statement on the 24-item self-report questionnaire (4-point Likert scale).

The Aggression Questionnaire (BPAQ; Buss & Perry, 1992; Appendix D) was designed to assess the multifactorial nature of aggressive behaviour. It was devised based on a factor analysis of the Hostility Inventory (Buss & Durkee, 1957), the rewriting of certain items from the Inventory, and the addition of new items. Four factors were generated: verbal aggression, physical aggression, anger, and hostility. Participants were

asked to indicate how much they agreed or disagreed with each statement on the 29-item self-report questionnaire (5-point Likert Scale).

Buss and Perry (1992) originally administered the BPAQ to a large group of undergraduate introductory psychology students predominantly ranging from 18 to 20 years of age. The sample was composed of 1,253 participants (612 men and 641 women). The internal consistency of the questionnaire was evaluated using Cronbach's alpha, such that physical aggression (.85), verbal aggression (.72), anger (.83), hostility (.77), and total score (.89), all revealed considerable internal consistency. Furthermore, test-retest correlations were conducted on 372 participants, such that, physical aggression (.80), verbal aggression (.76), anger (.72), hostility (.72), and total score (.80), suggested moderate to good stability over time. Finally, men were significantly more aggressive on physical aggression, verbal aggression, hostility, and the total score ($p < .01$), but not on the anger factor ($p > .20$).

Smits and Kuppens (2005) examined the relation between the BPAQ and the BIS/BAS in a university sample. Intuitively, BIS levels were negatively correlated with physical aggression, verbal aggression, and anger, whereas BAS levels were positively correlated with these factors. These results indicated a relation between impulsivity and the expression of aggression, reinforcing the psychometric basis for reactive aggression.

The physical aggression factor is particularly interesting as it is consistent with the neuropsychological construct, reactive aggression. For example, "Once in a while, I can't control the urge to strike another person" and "Given enough provocation, I may hit another person" are definitively measuring emotional dyscontrol and more specifically, reactive aggression. Therefore, the BPAQ is an ideal measure for studying aggression in

undergraduate students. Furthermore, high scores on the physical aggression subscale of the BPAQ suggest an inability to control emotional outbursts, which is a consequence of head trauma impacting the OFC. Therefore, due to the possible involvement of the OFC in MHI, the physical aggression factor of the BPAQ is a measure of reactive aggression.

The Self-Report Psychopathy Scale (SRP-III R11; Paulhus, Hemphill, & Hare, in press; Appendix E) has been shown to be a valid measure of sub-clinical forms of antisocial behaviour and psychopathy. It is a 64-item self-report questionnaire that uses a 5-point Likert scale. The scale has four factors: interpersonal manipulation, erratic lifestyle, callous affect, and antisocial behaviour. Cronbach's alpha was derived from 222 undergraduate university students, in which interpersonal manipulation (.81), callous affect (.81), erratic lifestyle (.79), antisocial behaviour (.85), and the overall psychopathy score (.88), all revealed high levels of internal consistency. These results indicate that the SRP-III R11 is a valid measure of antisocial behaviour in high-functioning populations, such as university students. Specifically, the erratic lifestyle and antisocial behaviour factors measure impulsive forms of antisocial behaviour which tend to be consequences of TBI involving the frontal lobes, whereas the interpersonal manipulation and callous affect subscales require frontal lobe integrity in order to successfully execute acts of instrumental antisocial behaviour. Interestingly, this scale has yet to be used in any investigation of the behavioural sequelae of MHI; however, given the purported involvement of the OFC in CHI and impulse control, the SRP-III R11 is an appropriate measure of the consequences of more "subtle" forms of head trauma due to its sensitivity to less severe impulsive, antisocial tendencies.

Procedure

All participants were individually tested in single, 2-hour sessions. Each session commenced with the researcher reading the informed consent form, answering initial questions from the participant, and having him/her sign 2 copies of the form (one for both the researcher and the participant; Appendix F). Next, neuropsychological testing was conducted according to the standard guidelines provided by the authors, in the same order to all participants. The order of the tests was: Stroop, Letter-Number Sequencing, CTONI Pictorial Analogies, Mental Control, Trails, and the Auditory and Attention task. Upon completion of the neuropsychological measures, the Reading the Mind in the Eyes task was administered. The demographic questionnaire as well as the self-report measures was then given to the participant to complete in the following order (Demographic, BIS-11, SRP-III, BIS/BAS, and BPAQ). Finally, the participant received a full debriefing (Appendix G) explaining the rationale for the research, as well as the purpose of the study.

Results

Approach to Data Analysis

The alpha level for all statistical tests was set at .05. Hierarchical multiple regression analyses were used in order to obtain the variance accounted for by MHI status after controlling for sex and executive functioning. Therefore, sex was entered on the first step, executive functioning on the second step, and MHI status on the third step of every regression analysis.

Analysis of Residuals

An analysis of standardized residuals was conducted to test for multivariate outliers for each solution, in which no value was beyond +/- 3 standard deviations. Independence of residuals was determined by the Durbin-Watson test, which was within the range of independence for each regression model. Furthermore, the residuals did not appear to violate homoscedasticity, as scatterplots of standardized residuals versus predicted criterion measures revealed relatively random and equal scattering. Furthermore, the normality of the residuals was assessed by examining normal P-P plots and the histograms of standardized residuals, in which only minor deviations from normality were determined. Therefore, after a thorough analysis it was determined that the residuals successfully met the assumptions.

Examining Differences and Similarities between individuals with and without a MHI

The means and standard deviations of executive functions between individuals with and without a history of MHI were presented in Table 1. Although individuals without a history of MHI appeared to have faster mean response times and higher accuracy scores than the MHI group on the executive functioning measures, these differences were not statistically significant (Stroop, t (88) = .03, p = .98; Letter Number Sequencing, t (88) = -.73 p = .47; CTONI, t (88) = -.27, p = .79; Mental Control, t (88) = .87, p = .39; Trail Making Test, t (88) = .66, p = .51; Auditory Response Set: NEPSY-B, t (86) = -.64, p = .53). Furthermore, the mean, mean, and mode for all of the executive functioning measures were equivalent between the MHI and non-MHI group, indicating that both groups were extremely competent, regardless of whether or not they incurred a MHI (see Appendix H).

On average, there was no difference in age between the MHI group ($M = 20.47$, $SD = 2.04$) and the non-MHI group ($M = 20.81$, $SD = 2.36$; $t\ (88) = -.74$, $p = .46$). Furthermore, the proportion of individuals in the MHI group (high school = 54.5%, 1st year = 48.5%, 2nd year = 58.8%, 3rd year = 50%, 4th year = 33.3%, greater than 4th year = 100%) did not differ from the proportion of individuals in the non-MHI group (high

Table 1

Means and standard deviations of executive functioning measures for individuals with and without a history of MHI

		M	*SD*	N
Non-MHI	Stroop-3 Response Time (s)	43.40	8.99	43
	Letter-Number (# correct)	10.91	2.06	43
	CTONI (# correct)	15.86	3.43	43
	MC-8 Response Time (s)	17.53	6.32	43
	Trails-4 Response Time (s)	59.06	16.96	43
	NEPSY-B (# correct)	36.40	4.07	42
MHI	Stroop-3 Response Time (s)	43.45	7.60	47
	Letter-Number (# correct)	10.62	1.69	47
	CTONI (# correct)	15.68	2.82	47
	MC-8 Response Time (s)	18.88	8.11	47
	Trails-4 Response Time (s)	61.54	18.47	47
	NEPSY-B (# correct)	35.85	4.13	46

school = 45.5%, 1st year = 51.5%, 2nd year = 41.2%, 3rd year = 50%, 4th year = 66.7%, greater than 4th year = 0%) with respect to their highest level of education completed (χ (5) = 1.90, $p > .05$). The proportion of people in the MHI group (yes =50.0, no = 53.8%) also did not differ from the non-MHI group (yes =50.0%, no = 46.2%) in the amount of extra assistance they received throughout their educational history (χ (1) = .13, $p > .05$), or in the number of hospitalizations for fractures, illness, surgery and neurological complications (MHI group: hospitalized yes = 30, no = 17; Non-MHI group hospitalized yes = 19, no = 24; χ (1) = 3.49, $p > .05$). Furthermore, the proportion of men (yes = 38%, no = 21%) to women (yes = 62%, no = 79) who reported incurring a MHI did not differ (χ (1) = 3.23, $p > .05$). Men and women also did not differ in their executive functioning performance on any of the neuropsychological measures (see Table 2).

Intercorrelations between MHI status and executive functioning measures

Intercorrelations were conducted between executive functioning measures and MHI status (see Table 3) in order to determine whether incurring a MHI was related to poorer executive functioning. Interestingly, MHI status was not related to any of the executive functioning measures. Nevertheless, consistent relationships were found amongst the executive functioning measures themselves. Slower ability to inhibit interfering information on the Stroop Colour-Word Interference task (Stroop3) was related to poorer performance remembering alphanumeric strings on the Letter Number Sequencing subtest (Letter-Number), slower processing ability on the Mental Control task (MC-8), poorer inhibitory control on the Auditory Response Set subtest (NEPSY-B), and poorer cognitive flexibility on the Trail Making Test (Trails-4). Poorer performance remembering alphanumeric strings on the Letter Number Sequencing task was related to

slower processing ability on the Mental Control task, and poorer cognitive flexibility on the Trail Making Test. Furthermore, poorer abstract reasoning ability as measured by the CTONI was related to slower processing ability on the Mental Control task and poorer cognitive flexibility on the Trail Making Test, while slower processing speed on Mental Control task was related to poorer cognitive flexibility on the Trail Making Test and

Table 2

Means and standard deviations of executive functioning measures for men and women

		M	*SD*	N
Men	Stroop-3 Response Time (s)	44.55	6.38	27
	Letter-Number (# correct)	10.30	1.44	27
	CTONI (# correct)	15.11	2.82	27
	MC-8 Response Time (s)	17.92	6.60	27
	Trails-4 Response Time (s)	64.61	17.96	27
	NEPSY-B (# correct)	35.85	4.43	26
Women	Stroop-3 Response Time (s)	42.95	8.93	63
	Letter-Number (# correct)	10.95	2.00	63
	CTONI (# correct)	16.05	3.21	63
	MC-8 Response Time (s)	18.37	7.63	63
	Trails-4 Response Time (s)	58.53	17.42	63
	NEPSY-B (# correct)	36.23	3.97	62

Table 3

Intercorrelations between sex, executive functioning measures, and MHI status

	1	2	3	4	5	6	7
1. Sex	-						
2. Stroop-3 ResponseTime(s)	-.09	-					
3. Letter-Number (# correct)	.16	-.34**	-				
4. CTONI (# correct)	.14	-.07	.06	-			
5. MC-8 Response Time (s)	.03	.44**	-.40**	-.21	-		
6. Trails-4 Response Time (s)	-.16	.39**	-.23*	-.21*	.52**	-	
7. NEPSY-B (# correct)	.04	-.22*	.16	.24*	-.26**	-.18*	-
8. MHI Status	-.19	.00	-.08	.03	.09	.07	-.07

*$p < .05$, **$p < .01$

poorer inhibitory control on the Auditory Response Set subtest. Finally, poorer inhibitory control on the Auditory Response Set subtest was related to poorer cognitive flexibility on the Trail Making Test. Therefore, although there was no relationship between MHI status and executive functioning, the correlations between the executive functioning measures themselves were in the expected directions. Executive functions that are reportedly mediated by a common neural region (the PFC) were found to be related with each other, reinforcing their shared neuroanatomical bases.

Hypothesis 1

Being male was expected to be positively correlated with physical aggression, verbal aggression, hostility, and anger. Poorer executive functioning was predicted to be related only to higher levels of physical aggression, as only the items on this scale

specifically address issues of executive functioning, such as impulse control. Above and beyond these variables, a history of MHI status was expected to be additionally predictive of physical aggression after controlling for sex and executive functioning.

The means and standard deviations for each aggression subscale are described in Table 4. Men appeared to have higher mean scores than women on physical aggression, verbal aggression, and anger, whereas women had higher means on the hostility scale. As expected, men scored significantly higher than women on the physical aggression subscale (F (1, 88) = 25.70, $p < .001$); however men did not differ from women on verbal aggression (F (1, 88) = 3.25 $p = .08$), hostility (F (1, 88) = .33 $p = .57$), or anger (F (1, 88) = .25 $p = .62$). Given the types of items endorsed, these results indicate that men are more physically reactive to provocation than women, but not more argumentative, hostile, or angry. However, the means for both men and women on the aggression measures were within the mid-range (non-clinical) on each scale, as they did not approach the ceiling for physical aggression (45 points), verbal aggression (25 points), anger (35 points), or hostility (40 points).

Intercorrelations were conducted between the predictors (each measure of executive functioning and MHI status) and each aggression subscale in order to examine the relationship between MHI, executive functioning, and aggression (see Table 5). As expected, sex (being male) was related to higher physical aggression and verbal aggression scores. Sex was not related to anger or hostility. Further, executive functioning as measured by poorer abstract reasoning (lower CTONI scores) and cognitive flexibility (longer Trails-4 times) was related to higher levels of reactive

aggression (physical aggression) and angry mood (anger); however, it was not related to verbal aggression or hostility. Most strikingly, MHI status was related to higher verbal aggression and physical aggression scores, suggesting that a history of concussive injury is differentially associated with increased argumentative and reactively aggressive behaviour.

Table 4

Means and standard deviations for aggression variables

		M	*SD*	N
Men	Physical aggression*	25.74	7.89	27
	Verbal aggression	16.81	3.54	27
	Hostility	21.85	4.62	27
	Anger	16.44	6.22	27
Women	Physical aggression*	17.92	6.14	63
	Verbal aggression	15.19	4.06	63
	Hostility	22.59	5.91	63
	Anger	15.86	4.58	63

* $p < .001$

Table 5

Intercorrelations between predictor variables (sex, executive functioning, MHI status) and aggression subscales (N = 88)

	PA	VA	Hostility	Anger
Sex	-.48**	-.21*	.06	-.07
Stroop-3 Response Time (s)	-.01	-.11	-.06	-.06
Letter-Number (# correct)	-.13	.07	-.02	.02
CTONI Score (# correct)	-.29**	-.12	-.11	-.21*
MC-8 Response Time (s)	-.05	-.09	.13	.08
Trails-4 Response Time (s)	.21*	.16	.12	.25*
NEPSY-B (# corect)	.14	.12	.14	.14
MHI Status	.29**	.20*	-.14	.11

*$p < .05$, **$p < .01$

Each aggression measure was regressed on sex, executive functioning, and MHI status as described above. The overall multiple regression model predicting physical aggression accounted for a significant amount of variance, ($R^2 = .42$; $F(8, 79) = 7.26$, $p < .001$; see Appendix I). Furthermore, sex, executive functioning (particularly the CTONI), and MHI status individually accounted for a significant amount of variance on their respective steps of entry (see Table 6). Being male accounted for significant amount of variance (23.04%), as did poorer abstract reasoning (6.76%), as well as self-reported MHI (5.29%) when predicting retaliatory forms of aggression (i.e. physical aggression). Notably, a history of MHI significantly predicted higher levels of reactive aggression, after controlling for executive functioning and sex differences.

Table 6

Summary of hierarchical multiple regression analysis in which physical aggression was regressed on sex, executive functioning, and MHI status (N = 88)

Variable	*B*	*SE B*	*B*	*sr*
Step 1				
Sex	-7.95	1.56	-.48**	-.48
Step 2				
Sex	-6.71	1.53	-.41**	-.39
Stroop-3 Response Time (s)	-.04	.10	-.04	-.03
Letter-Number (# correct)	-.44	.41	-.11	-.10
CTONI Score (# correct)	-.68	.23	-.28**	-.26
MC-8 Response Time (s)	-.16	.12	-.16	-.12
Trails-4 Response Time (s)	.09	.05	.20	.16
NEPSY-B (# correct)	.42	.18	.23*	.21
Step 3				
Sex	-6.02	1.50	-.37**	-.34
Stroop-3 Response Time (s)	-.01	.09	-.01	-.01
Letter-Number (# correct)	-.41	.39	-.10	-.10
CTONI Score (# correct)	-.71	.22	-.29**	-.27
MC-8 Response Time (s)	-.19	.12	-.18	-.14
Trails-4 Response Time (s)	.08	.04	.20	.16
NEPSY-B (# correct)	.45	.17	.24**	.22
MHI Status	3.57	1.32	.24**	.23

Note. $R^2 = .23$ for Step 1; $\Delta R^2 = .14$ for Step 2; $\Delta R^2 = .05$ for Step 3.
*$p < .05$, **$p \leq .01$

The overall multiple regression models examining sex, executive functioning, and MHI status predicting verbal aggression ($R^2 = .16$; $F\,(8, 79) = 1.92$, $p = .07$; see Appendix J and K), hostility ($R^2 = .10$; $F\,(8, 79) = 1.14$, $p = .34$; see Appendix L and M), and anger ($R^2 = .16$; $F\,(8, 79) = 1.94$, $p = .07$; see Appendix N and O) were not significant. Therefore, a history of MHI did not predict any of the other forms of aggression: verbal, hostility, or anger, after controlling for sex and executive functioning. These findings further reinforce the differential effect of frontal lobe injury on reactively aggressive behaviour, as opposed to argumentativeness, infuriated mood, or hostile interpersonal style.

Hypothesis 2

Being male was hypothesized to be related to higher scores on the erratic lifestyle, antisocial behaviour, callous affect, and interpersonal manipulation subscales. Executive dysfunction was expected to be differentially related to higher erratic lifestyle and antisocial behaviour scores, but not the callous affect or interpersonal manipulation scales. However, over and above these variables a history of MHI was hypothesized to be predictive of the erratic lifestyle and the antisocial behaviour. The means and standard deviations for all of the antisocial subscales of the SRP-III are described in Table 7, in which men appeared to have higher mean scores than women on erratic lifestyle, antisocial behaviour, callous affect, and interpersonal manipulation.

Upon further investigation, men were significantly higher than women on all of the subscales: erratic lifestyle ($F\,(1, 88) = 8.20$, $p < .01$), antisocial behaviour ($F\,(1, 88) = 11.09$, $p = .001$), callous affect ($F\,(1, 88) = 34.03$, $p < .001$), and interpersonal manipulation ($F\,(1, 88) = 26.42$, $p < .001$). These results indicate that men present with

Table 7

Means and standard deviations for antisocial variables

		M	*SD*	N
Men	Erratic*	45.78	9.76	27
	Antisocial*	27.81	7.82	27
	Callous**	42.44	7.79	27
	Interpersonal**	47.00	10.67	27
Women	Erratic*	39.71	8.97	63
	Antisocial*	22.94	5.65	63
	Callous**	33.63	5.98	63
	Interpersonal**	36.37	8.47	63

*$p < .01$, **$p < .001$

more instrumental antisocial behaviour on the callous affect and interpersonal manipulation scales, as well as more impulsive antisocial behaviour assessed by the erratic lifestyle and antisocial behaviour subscales.

Correlations between the predictors and each antisocial subscale are presented in Table 8. Poorer abstract reasoning (measured on the CTONI) was related to more risky decision-making, as measured by erratic lifestyle. Interestingly, better inhibitory control (on the NEPSY-B) was positively correlated with interpersonal manipulation, suggesting that superior executive functioning is crucial for calculated, instrumental antisocial behaviour. Furthermore, being male and a history of MHI was positively correlated with all 4 antisocial subscales.

Table 8

Intercorrelations between predictor variables (sex, executive functioning, MHI status) and antisocial subscales (N = 88)

	Erratic	Antisocial	Callous	Interpersonal
Sex	-.29**	-.33**	-.52**	-.47**
Stroop-3 Response Time (s)	-.10	.13	-.08	-.06
Letter-Number (# correct)	-.07	-.17	.08	-.05
CTONI Score (# correct)	-.20*	-.17	-.10	-.05
MC-8 Response Time (s)	-.05	.02	-.08	-.10
Trails-4 Response Time (s)	-.06	.11	.02	-.02
NEPSY-B (# correct)	-.16	.05	-.03	.25**
MHI Status	.30**	.33**	.22*	.19*

*$p < .05$, **$p \leq .01$

The overall multiple regression models predicting erratic lifestyle ($R^2 = .26$; F (8, 79) = 3.41, $p < .01$; see Table 9 and Appendix P), antisocial behaviour ($R^2 = .25$; F (8, 79) = 3.33, $p < .01$; see Table 10 and Appendix Q), callous affect ($R^2 = .33$; F (8, 79) = 4.84, $p < .001$; see Table 11 and Appendix R), and interpersonal manipulation ($R^2 = .32$; F (8, 79) = 4.55, $p < .001$; see Table 12 and Appendix S) each accounted for a significant amount of variance. Sex significantly accounted for variance when predicting erratic lifestyle (8.41%), antisocial behaviour (10.89%), callous affect (27.04%), and interpersonal manipulation (22.09%). Furthermore, executive dysfunction (abstract reasoning as measured by the CTONI) was a significant predictor of erratic lifestyle after controlling for sex, contributing 5.76% of variance, whereas better executive functioning (specifically inhibitory control as measured by the NEPSY-B) predicted 6.76% of

Table 9

Summary of hierarchical multiple regression analysis in which SRP-erratic lifestyle was regressed on sex, executive functioning, and MHI status (N = 88)

Variable	*B*	*SE B*	*β*	*sr*
Step 1				
Sex	-6.11	2.16	-.29**	-.29
Step 2				
Sex	-5.94	2.22	-28**	-.27
Stroop-3 Response Time (s)	-.09	.14	-.08	-.07
Letter-Number (# correct)	-.42	.59	-.08	-.07
CTONI Score (# correct)	-.74	.33	-.24*	-.22
MC-8 Response Time (s)	.04	.17	.03	.02
Trails-4 Response Time (s)	-.07	.09	-.12	-.10
NEPSY-B (# correct)	.49	.25	.21	.20
Step 3				
Sex	-4.92	2.16	-.24*	-.22
Stroop-3 Response Time (s)	-.05	.13	-.05	-.04
Letter-Number (# correct)	-.37	.56	-.07	-.06
CTONI Score (# correct)	-.78	.32	-.25*	-.24
MC-8 Response Time (s)	.00	.17	.00	.00
Trails-4 Response Time (s)	-.07	.06	-.13	-.11
NEPSY-B (# correct)	.53	.24	.23*	.21
MHI Status	5.32	1.90	.28**	.27

Note. $R^2 = .09$ for Step 1; $\Delta R^2 = .10$ for Step 2; $\Delta R^2 = .07$ for Step 3.
*$p < .05$, **$p \leq .01$

Table 10

Summary of hierarchical multiple regression analysis in which SRP-antisocial behaviour was regressed on sex, executive functioning, and MHI status (N = 88)

Variable	*B*	*SE B*	*B*	*sr*
Step 1				
Sex	-4.81	1.49	-.33**	-.33
Step 2				
Sex	-4.14	1.56	-.28**	-.27
Stroop-3 Response Time (s)	.11	.10	.13	.11
Letter-Number (# correct)	-.42	.41	-.12	-.10
CTONI Score (# correct)	-.36	.23	-.17	-.16
MC-8 Response Time (s)	-.07	.12	-.08	-.06
Trails-4 Response Time (s)	.01	.05	.02	.01
NEPSY-B (# correct)	.22	.18	.14	.13
Step 3				
Sex	-3.36	1.1	-.23*	-.22
Stroop-3 Response Time (s)	.13	.10	.16	.14
Letter-Number (# correct)	-.38	.39	-.11	-.10
CTONI Score (# correct)	-.39	.22	-.18	-.17
MC-8 Response Time (s)	-.10	.18	-.11	-.09
Trails-4 Response Time (s)	.01	.05	.01	.01
NEPSY-B (# correct)	.25	.17	.15	.14
MHI Status	4.01	1.33	.30**	.29

Note. $R^2 = .11$ for Step 1; $\Delta R^2 = .06$ for Step 2; $\Delta R^2 = .09$ for Step 3.
*$p < .05$, **$p \leq .01$

Table 11

Summary of hierarchical multiple regression analysis in which SRP-callous affect was regressed on sex, executive functioning, and MHI status (N = 88)

Variable	*B*	*SE B*	*β*	*sr*
Step 1				
Sex	-8.82	1.55	-.52**	-.52
Step 2				
Sex	-9.35	1.64	-.56**	-.53
Stroop-3 Response Time (s)	-.06	.10	-.07	-.06
Letter-Number (# correct)	.63	.43	.15	1.4
CTONI Score (# correct)	-.13	.25	-.05	-.05
MC-8 Response Time (s)	.06	.13	.05	.04
Trails-4 Response Time (s)	-.01	.05	-.03	-.03
NEPSY-B (# correct)	.07	.19	.04	.04
Step 3				
Sex	-8.92	1.65	-.53**	-.50
Stroop-3 Response Time (s)	-.05	.10	-.05	-04
Letter-Number (# correct)	.65	.43	.16	.14
CTONI Score (# correct)	-.15	.24	-.06	-.06
MC-8 Response Time (s)	.04	.13	.04	.03
Trails-4 Response Time (s)	-.01	.05	-.03	-.03
NEPSY-B (# correct)	.09	.19	.05	.04
MHI Status	2.20	1.45	.14	.14

Note. $R^2 = .27$ for Step 1; $\Delta R^2 = .04$ for Step 2; $\Delta R^2 = .02$ for Step 3.
*$p < .05$, **$p \leq .01$

Table 12

Summary of hierarchical multiple regression analysis in which SRP-interpersonal manipulation was regressed on sex, executive functioning, and MHI status (N = 88)

Variable	*B*	*SE B*	*β*	*sr*
Step 1				
Sex	-10.45	2.13	-.47**	-.47
Step 2				
Sex	-10.65	2.19	-.48**	-.46
Stroop-3 Response Time (s)	-.01	.14	-.01	-.01
Letter-Number (# correct)	-.18	.58	-.03	-.03
CTONI Score (# correct)	-.24	.33	-.07	-.07
MC-8 Response Time (s)	.00	.17	.00	.00
Trails-4 Response Time (s)	-.04	.07	-.07	-.05
NEPSY-B (# correct)	.70	.25	.28**	.26
Step 3				
Sex	-10.12	2.21	-.45**	-.43
Stroop-3 Response Time (s)	.01	.14	.00	.00
Letter-Number (# correct)	-.15	.58	-.03	-.03
CTONI Score (# correct)	-.27	.33	-.08	-.08
MC-8 Response Time (s)	-.02	.17	-.01	-.01
Trails-4 Response Time (s)	-.04	.07	-.07	-.06
NEPSY-B (# correct)	.72	.25	.29**	.27
MHI Status	2.69	1.95	.13	.13

Note. $R^2 = .22$ for Step 1; $\Delta R^2 = .08$ for Step 2; $\Delta R^2 = .02$ for Step 3.
*$p < .05$, **$p < .01$

variance in interpersonal manipulation. However, these measures did not predict callous affect or antisocial behaviour. The final predictor, MHI status, accounted for a significant amount of variance above and beyond sex and executive functioning for erratic lifestyle (7.29%; impulsive behaviour) and antisocial behaviour (8.41%; reckless behaviour).

MHI status did not predict callous affect or interpersonal manipulation, nor was it expected to, since there is no research indicating that injury to the OFC would cause cold,

calculated lack of empathy (although right-sided injury is associated with flattened/neutral affect) and, if anything, frontal lobe injury would contraindicate competent manipulation (since perspective taking, prediction and anticipation of outcomes and reactions of others would be impaired cognitive skills in frontal injury). Therefore, consistent with research examining focal injury to the OFC, a history of MHI differentially predicted impulsive antisocial behaviour.

Hypothesis 3

Being male, executive dysfunction, and a history of MHI were expected to be related to higher levels of impulsivity. Specifically, it was hypothesized that MHI status would be a significant predictor of only BIS-motor (but not BIS-attentional or BIS-nonplanning), after controlling for sex and executive functioning. MHI status was also expected to be related to the Behavioural Inhibition System/Behavioural Activation System (BIS/BAS). Specifically, MHI was hypothesized to be predictive of lower BIS levels and higher BAS levels after controlling for sex and executive functioning.

The means and standard deviations for all of the BIS-11 subscales are described in Table 13. Men appeared to have higher means on BIS-motor and BIS-attentional, whereas BIS-nonplanning appeared virtually identical. However, men did not differ from women on the BIS-motor (F (1, 87) = .09 p = .77), BIS-attentional (F (1, 87) = .74 p = .39), or BIS-nonplanning (F (1, 87) = .00 p = 1.00) subscales. These results indicate that men did not exhibit more symptoms of behavioural disinhibition, racing thoughts, or deficits in prospective thinking. Furthermore, both the men and women in this sample had mean scores that were within the mid-range on each BIS-11 measure which did not

Table 13

Means and standard deviations for impulsivity variables

		M	*SD*	N
Men	BIS-motor	23.48	4.54	27
	BIS-attentional	17.81	3.43	27
	BIS-nonplanning	23.33	3.52	27
Women	BIS-motor	23.16	4.76	62
	BIS-attentional	17.05	4.05	62
	BIS-nonplanning	23.34	4.48	62

approach the ceiling score of 44 points on either the BIS-motor, BIS-nonplanning, or BIS- attentional scales.

Significant correlations revealed that working memory ability was negatively correlated with the BIS-attentional subscale, but was not related to the other subscales (see Table 14). Furthermore, a history of MHI was positively correlated with the BIS-motor and BIS-attentional subscales, but not BIS-nonplanning. Therefore, whereas executive functioning (working memory in particular) was positively correlated with sustained attentional capacity; a history of MHI was positively correlated with both increased symptoms of behavioural disinhibition and difficulties sustaining attention.

Each impulsivity measure was regressed on sex, executive functioning, and MHI status as described above. The overall multiple regression model predicting BIS-motor accounted for a significant amount of variance, ($R^2 = .22$; $F\,(8, 78) = 2.72$, $p = .01$; see

Table 14

Intercorrelations between predictor variables (sex, executive functioning, MHI status) and impulsivity subscales of the BIS-11 (N = 87)

	BIS-motor	BIS-attention	BIS-nonplan
Sex	.00	-.08	.04
Stroop-3 Response Time (s)	-.09	.05	.01
Letter-Number (# correct)	-.12	-.14	-.16
CTONI Score (# correct)	-.12	-.11	-.18
MC-8 Response Time (s)	.06	.22*	.12
Trails-4 Response Time (s)	-.16	.11	-.15
NEPSY-B (# correct)	.09	-.07	-.05
MHI Status	.34**	.19*	.05

*$p < .05$, **$p < .01$

Appendix T). Furthermore, MHI status was a significant predictor of behavioural disinhibition, after controlling for sex and executive functioning, as it accounted for 11.56% of variance (see Table 15). Therefore, as hypothesized, a history of MHI status predicted behavioural disinhibition above and beyond executive functioning, and sex differences in terms of impulse control.

The overall multiple regression models predicting BIS-attentional ($R^2 = .09$; F (8, 78) = .95, $p = .48$; see Appendix U and V) and BIS-nonplanning ($R^2 = .13$; F (8, 78) = 1.41, $p = .21$; see Appendix W and X) were not successful in accounting for a significant amount of variance.

Table 15

Summary of hierarchical multiple regression analysis in which BIS-motor was regressed on sex, executive functioning, and MHI status (N = 87)

Variable	*B*	*SE B*	*β*	*sr*
Step 1				
Sex	-.03	1.10	.00	.00
Step 2				
Sex	-.18	1.14	-.02	-.02
Stroop-3 Response Time (s)	-.06	.07	-.10	-.09
Letter-Number (# correct)	-.37	.30	-.15	-.13
CTONI Score (# correct)	-.23	.18	-.15	-.14
MC-8 Response Time (s)	.11	.09	.18	.14
Trails-4 Response Time (s)	-.07	.04	-.25	-.20
NEPSY-B (# correct)	.14	.13	.13	.12
Step 3				
Sex	.38	1.09	.04	.04
Stroop-3 Response Time (s)	-.04	.07	-.07	-.06
Letter-Number (# correct)	-.33	.28	-.13	-.12
CTONI Score (# correct)	-.25	.17	-.16	-.15
MC-8 Response Time (s)	.09	.08	.15	.11
Trails-4 Response Time (s)	-.07	.03	-.26*	-21
NEPSY-B (# correct)	.16	.12	.14	.13
MHI Status	3.19	.95	.34**	.34

Note. $R^2 = .00$ for Step 1; $\Delta R^2 = .11$ for Step 2; $\Delta R^2 = .11$ for Step 3.
*$p < .05$, **$p \leq .01$

In summary, the results from the regression analyses of each subscale from the BIS-11 revealed a dissociable relationship between MHI status and specific forms of impulsivity. Therefore, whereas the intercorrelations identified additional significant relationships, when other factors, such as sex and executive functioning are controlled, only BIS-motor, a measure of disinhibition and impulsivity is predicted by having a history of MHI. These results reinforce the differential impact of OFC injury on behavioural disinhibition.

The means and standard deviations for all of the BIS/BAS subscales are described in Table 16, in which women appeared to have higher BIS, BAS, and BIS/BAS scores. Women scored significantly higher than men on the BIS (F (1, 88) = 37.73, $p < .001$) and BIS/BAS ratio than men (F (1, 88) = 15.46, $p < .001$); however, there was no difference between men and women on the BAS (F (1, 88) = 1.72 $p = .19$). Therefore, women were shown to be more sensitive to punishment (i.e. BIS measure) than men. Significant correlations between measures of executive functioning and sensitivity to reward (i.e. BAS levels) revealed that better inhibitory control (higher scores on the NEPSY-B) was positively correlated with BAS levels (see Table 17); however, a history of MHI was not related to BIS or BAS levels.

Hierarchical regressions predicting BIS and BAS levels were conducted in order to determine the variance accounted for by sex, executive functioning, and MHI status. The overall multiple regression model predicting BIS ($R^2 = .31$; F (8, 79) = 4.39, $p < .001$; see Appendix Y and Z) and BIS/BAS ($R^2 = .21$; F (8, 79) = 2.60, $p = .01$; see Appendix AA and BB) accounted for a significant amount of variance. With respect to both the BIS and BIS/BAS regression models, sex (being a woman) was the only significant predictor of sensitivity to aversive consequences, accounting for 28.1% and 13.7% of unique variance respectively. Therefore, executive functioning and a history of MHI were not predictors of increased sensitivity to punishment.

With respect to BAS, the overall multiple regression model was not successful in accounting for a significant amount of variance, ($R^2 = .11$; F (8, 79) = 1.21, $p = .30$; see Appendix CC and DD). Inconsistent with the original hypotheses, executive functioning and MHI were not significant predictors of sensitivity to reward.

Table 16

Means and standard deviations for BIS/BAS variables

		M	*SD*	N
Men	BIS*	18.30	4.44	27
	BAS	39.26	7.01	27
	BIS/BAS Ratio*	.47	.11	27
Women	BIS*	23.11	2.86	63
	BAS	40.97	5.00	63
	BIS/BAS Ratio*	.57	.11	63

*$p < .001$

Table 17

Intercorrelations between predictor variables (sex, executive functioning, MHI status) and BIS/BAS subscales (N = 88)

	BIS	BAS	BIS/BAS Ratio
Sex	.53**	.13	.37**
Stroop-3 Response Time (s)	.00	-.10	.06
Letter-Number (# correct)	.08	-.04	.10
CTONI Score (# correct)	.10	.07	.04
MC-8 Response Time (s)	.14	-.06	.13
Trails-4 Response Time (s)	-.03	-.14	.04
NEPSY-B (# correct)	-.07	.24*	-.19*
MHI Status	-.06	.08	-.11

*$p < .05$, **$p < .01$

In summary, the results from the regression analyses of each subscale from the BIS/BAS scales revealed that a history of MHI and executive dysfunction did not predict sensitivity to punishment or reward. Interestingly, however, is the finding that sex (being female) differentially predicted BIS levels.

Hypothesis 4

It was predicted that MHI status would be a significant predictor of ToM performance, after controlling for sex and executive functioning. Furthermore, poorer executive functioning was expected to be related to lower ToM accuracy.

ToM performance was compared between men and women and demonstrated a trend towards significance for women to be more accurate than men at interpreting/labeling eye expressions (F (1, 88) = 3.40 p = .07; Table 18). Correlations between MHI, executive functioning, and social awareness are reported in Table 19. Abstract reasoning ability (as measured with the CTONI) was positively correlated with better affect recognition abilities. A history of MHI, however, was not related to performance on the ToM task.

Table 18

Means and standard deviations for Theory of Mind accuracy

	M	*SD*	N
Men	26.11	4.15	27
Women	27.57	3.10	63

Table 19

Intercorrelations between predictor variables (sex, executive functioning, MHI status) and Theory of Mind accuracy (N = 88)

	ToM Total
Sex	.17
Stroop-3 Response Time (s)	.00
Letter-Number (# correct)	.13
CTONI Score (# correct)	.21*
MC-8 Response Time (s)	.00
Trails-4 Response Time (s)	-.08
NEPSY-B (# correct)	.02
MHI Status	-.09

*$p < .05$

Further, the overall multiple regression model predicting ToM accuracy was not successful in accounting for a significant amount of variance, ($R^2 = .09$; $F(8, 79) = .96$, $p = .47$; see Appendix EE). The individual contributions of each predictor are presented in the appendices (see Appendix FF). Hence, although better abstract reasoning ability was positively correlated with more accurate affect recognition, sex, executive functioning, and a history of MHI were not significant predictors of social awareness.

Additional Analyses

Since sex differences were pronounced throughout these measures, and expected given the literature on sex and aggression (Buss & Perry, 1992), it is possible that MHI would interact with sex differences in terms of demonstrating sex-specific differences in social behaviour (such as on the subscales of aggression and impulsivity). Therefore,

each regression model was also conducted separately for both men and women. In men, none of the regression models were significant; however, with women, while the majority of regression models were nonsignificant, significant findings were observed with the aggression subscale of anger and the psychopathic subscale of interpersonal manipulation. Ultimately, due to the large disparity in sample size between men and women (27 men; 63 women), insufficient power was a serious statistical limitation when attempting to analyze 7 predictors (especially in men).

When predicting anger in women, the overall regression model accounted for a significant amount of variance ($R^2 = .26$; $F\,(7, 54) = 2.65$, $p = .02$), however, MHI did not add any additional information. Interestingly, poorer working memory performance (Trails-4; $sr = .36$, $p = .003$) was the only predictor of increased angry mood in women, suggesting that the ability to hold and manipulate information in memory is important for prosocial behaviour.

The overall regression model predicting interpersonal manipulation in women accounted for a significant amount of variance ($R^2 = .22$; $F\,(7, 54) = 2.21$, $p = .05$). Similar to the initially tested model, better inhibitory control (NEPSY-B; $sr = .39$, $p = .01$) predicted more manipulative behaviour, but MHI did not add any additional variance.

Taken together, three out of four hypotheses were supported and showed that a history of MHI predicted higher levels of reactive aggression, impulsive antisocial behaviour, and behavioural disinhibition, while affect recognition was unimpaired. Furthermore, executive functioning differentially predicted antisocial behaviour, as poorer abstract reasoning predicted higher levels of social deviance and reactive

aggression, whereas better inhibitory control was predictive of more manipulative behaviour.

Discussion

The central purpose of this study was to elucidate the behavioural and social consequences of incurring a MHI. Furthermore, the relationship between executive functioning and social behaviour was explored as a putative measure of frontal lobe integrity. As predicted, the results convincingly indicated that a history of MHI is predictive of specific forms of antisocial behaviour which mirror the sequelae of more severe forms of brain injury, after controlling for sex differences and executive functioning. More specifically, the presence of a MHI differentially predicted reactive forms of aggression, impulsive antisocial behaviour, and poor impulse control.

With respect to the relationship between MHI, executive functioning, and aggression, poorer abstract reasoning skills predicted higher levels of reactive aggression. The former finding suggests that the ability to cognitively control and manipulate information in order to make abstract decisions is a crucial component of emotional control, as individuals with better reasoning abilities on the CTONI Analogies task were significantly less reactively/physically aggressive than those that performed more poorly. Furthermore, after accounting for sex differences and executive functioning, individuals with a history of MHI were less capable of inhibiting emotional impulses and had poorer frustration tolerance. Therefore, emotional dyscontrol in individuals with a MHI is most probably due to subtle OFC dysfunction as a result of the head trauma. Furthermore, MHI did not result in increased argumentativeness, suspiciousness, or changes in overall mood state which reinforces the dissociation in aggressive behaviour (increased reactivity while

other forms of aggression are generally unaffected) due the underlying disruption of the OFC in both MHI and more severe forms of TBI. Although the relationship between sustaining a brain injury and reactive aggression has been well established in the literature (e.g., Blair, 2001; Blair & Cipolotti, 2000), extending this finding to asymptomatic university students with a history of MHI suggests a serious social issue that has been neglected in both research and clinical practice, as deficits in emotional control can result in increased rates of physical injury to oneself and others, as well as severe legal ramifications due to deficits with respect to withholding physically reactive outbursts. Ultimately, MHI disrupts OFC integrity which results in a dissociable pattern of aggressive behaviour characterized by poor emotional control and retaliation to provocation even in high functioning, asymptomatic university students.

The relationship between MHI, executive functioning, and antisocial behaviour was further examined, as impulsive forms of antisocial behaviour were expected in a sample of individuals with subtle head trauma due the fact that poor decision-making and impulsive behaviour has been shown to be a common consequence of frontal lobe injury (Anderson et al., 1999; Blair, 2001; Eslinger & Damasio, 1985, etc.). The dissociation between impulsive and instrumental antisocial behaviour was particularly noteworthy in this study. Specifically, poorer abstract reasoning was a significant predictor of risky decision-making (impulsive antisocial behaviour), whereas better inhibitory control predicted instrumental, manipulative behaviour. These findings suggest that abstract reasoning ability is a crucial component of adaptive decision-making, as a plethora of (sometimes conflicting) information must be integrated to guide adaptive, future behaviour. Furthermore, individuals who were more manipulative have been shown to be

better able to control their impulses, such that they modulate their behaviour contingent on environmental cues in order to obtain specific gains. Surprisingly, executive functioning, such as abstract reasoning or inhibitory control did not predict more general forms of antisocial behaviour (of the antisocial behaviour scale). This finding may have been due to the fact that these specific measures of executive functioning might not have been able to differentiate between the subtle differences in this type of antisocial behaviour due to the relatively low endorsement rate by both the MHI and non-MHI group. More specifically, university students generally have not committed serious legal transgressions that were described by this subscale.

Most strikingly, MHI differentially predicted impulsive antisocial behaviour, as individuals with a history of MHI endorsed engaging in more risky decision-making and impulsive antisocial behaviour than non-injured controls. Moreover, individuals with a history of MHI were not more manipulative or emotionally callous than healthy controls. These results further suggest a link between frontal lobe disruption (caused by a MHI) and impulsive forms of antisocial behaviour, as the antisocial consequences of MHI are reactive in nature as opposed to predatory or instrumental which has been supported by research examining focal injury to the frontal lobes (Blair, 2001, Blair, 2004). Ultimately, incurring a MHI has been shown to be predictive of the same antisocial trajectory (albeit to a lesser degree) as focal orbitofrontal injury, as the inability to modulate impulses and make adaptive decisions has been shown to have negative social consequences even in high-functioning populations, such as university students.

Behavioural disinhibition is a common consequence of TBI (McAllister, 1992), but has not been rigorously examined in populations with MHI. Results from this study

revealed that MHI was a significant predictor (after controlling for executive functioning and sex) of only the BIS-motor subscale, but not the BIS-attentional or the BIS-nonplanning subscales. This dissociation was consistent with the literature and other findings in this study, as the BIS-motor component measured behavioural disinhibition and lack of impulse control which was hypothesized to be affected by a MHI, whereas distractibility and failure to anticipate were not. Therefore, individuals with a history of MHI exhibit subtle deficits in impulse control, mirroring individuals with focal injury to the OFC.

Counter intuitively, a history of MHI was not predictive of BAS (designed to assess appetitive motivation/sensitivity to reward) or BIS (designed to measure aversive motivation/sensitivity to punishment). However, based on Carver and White (1994), individuals high on BAS are driven predominantly by reward-based cues, regardless of the future consequences of their behaviour. This lack of prospective thinking ability with respect to the cause-effect relationships of one's actions is similar to the effects of orbitofrontal injury on decision-making (Wallis, 2007). Furthermore, individuals high on BIS are more inhibited and more vigilant to the consequences of their behaviour. Hence, it was predicted that incurring a MHI would increase BAS levels and decrease BIS levels. This was not the case, as individuals with a history of MHI did not differ from non-injured controls on the BAS, the BIS, or the BIS/BAS ratio. Furthermore, none of the measures of executive functioning predicted BIS, BAS, or BIS/BAS ratio. The failure to find an effect is most probably due to the subtly of the neurological disruption, as this measure (BIS/BAS) might not have been sensitive enough to detect changes in appetitive and aversive behaviour in asymptomatic university students.

Consistent with previous research by Jorm et al. (1999), women had higher BIS levels (and hence higher BIS/BAS ratio levels). Furthermore, being a woman significantly predicted BIS levels. Jorm et al. (1999) also found that BIS was correlated with neuroticism, and internalizing disorders, such as anxiety and depression. Furthermore, women are more likely than men to suffer from internalizing disorders, such as depression (*DSM-IV-TR*; American Psychiatric Association, 2000). Hence, the sex differences in BIS levels found in this study were consistent with previous research.

The relationship between MHI, executive functioning, and ToM was also examined in order to determine whether a history of MHI resulted in deficits with social awareness. In contrast to pervious research examining injury involving the frontal lobes, MHI was not predictive of deficits in social awareness. Interestingly, however, executive dysfunction (specifically deficits in abstract reasoning) was related to poorer social awareness on the Reading the Mind in the Eyes task. This finding appears to be somewhat in contrast to Rowe et al. (2001) who found that executive functioning was not related to deficits on measures of ToM and that TBI was related to impairments in social awareness. However, several substantial differences exist between Rowe et al. and the current study. The current study consisted of an executive functioning battery which included an abstract reasoning task (the CTONI), whereas the research by Rowe et al. only used tasks assessing cognitive initiation (e.g. Controlled Oral Word Association Test), inhibition, (e.g. Stroop), mental flexibility (e.g. Trails), and monitoring. Thus, the inconsistency in findings may be due to the fact that the significant executive functioning measure (e.g. CTONI) used in the current study was not administered by Rowe et al.

Furthermore, there are numerous measures of ToM performance in research which tap slightly different neurocognitive processes within the overall construct of ToM. Rowe et al. (2001) used a primarily cognitive attribution measure of ToM, such that participants were required to read a series of stories and answer questions regarding "false beliefs," whereas the ToM task in the current study assessed affect recognition from a series of photographs of human eyes. Hence, the measure in the current study more precisely examined orbitofrontal functioning, as Blair et al. (1999) found that the processing of facial cues (particularly anger) differentially activated the OFC (using PET). Furthermore, Kringelbach and Rolls (2003) found that the OFC and the anterior cingulate cortex were differentially activated (using fMRI) while viewing changing facial expressions. Therefore, the results from the current study revealed a significant relationship between abstract reasoning skills and affect recognition. As for MHI not predicting social awareness deficits, university students with a history of MHI in the present study were extremely high functioning, such that the measure of ToM might not have been sensitive enough to detect any discernible functional impairment. Ultimately, the CTONI has not been used to date in studies investigating ToM and thus represents an exciting and promising neurocognitive task which needs to be further used in social awareness research in order to continue to elucidate the relationship between abstract reasoning and social awareness.

The finding that MHI status was not related to any of the executive functioning measures was unexpected; however, there are several possible explanations for this outcome. First, the individuals in this study with a history of MHI were extremely high functioning and were expected to have the most subtle deficits. Therefore, the head

trauma was sufficiently subtle; such that it did not permit variability that was distinct from the non-MHI group. More specifically, both groups clearly demonstrated competency (they were all university students) which might have overlapped far too greatly to be discriminated with regression or grouping analyses. Furthermore, the executive functioning measures that were used might have assessed more synergistic involvement of the DLPFC and more posterior regions of the frontal lobe which would not have been harmed by the mild nature of the coup-contre coup in the MHI group, as the central point of impact would have been the OFC due to its location directly behind the jagged orbits of the eyes. A future study using the IGT and/or visual discrimination reversal task might help to further investigate the relationship between orbitofrontal compromise and MHI.

Ultimately, the findings from this study have convincingly supported the hypotheses that MHI (similar to severe TBI) was a significant predictor of many forms of antisocial behaviour consequences. Most striking was the fact that MHI status predicted emotional dyscontrol, impulsive antisocial behaviour, and behavioural disinhibition – all of which affect social encounters, relationships and community reintegration. Therefore, many of the social consequences observed with persons who have experienced TBI involving the OFC (e.g. loneliness, social rejection, inability to manage one's emotional expression, inability to withhold comments or reactions) are also differentially impacted by MHI, albeit to a lesser degree. Thus, the social ramifications of MHI can be much more serious than assumed in the literature.

Furthermore, the effects of MHI may not necessarily be transient, or inconsequential. Instead, the presence of antisocial behaviour after head injury of varying

severity (mild to severe) should be interpreted based on the shared underlying disruption of the frontal lobe and the OFC in particular. Therefore, the common medical dichotomy between TBI (organic) and MHI (perhaps malingering and/or psychosocial) is a dangerous fallacy, especially when the individuals with a history of MHI in this sample were asymptomatic and extremely high-functioning. Hence, MHI should be treated as a serious neurological concern, such that appropriate and compassionate treatment is provided to individuals whom have incurred milder head trauma. Furthermore, individuals with MHI in this sample endorsed specific antisocial behaviour associated with orbitofrontal dysfunction, as they only differed on crucial aspects of social behaviour pertaining to orbitofrontal functioning.

Furthermore, the fact that these students are currently enrolled in university and have performed comparably to non-injured controls on measures of executive functioning mitigates the argument that these individuals possess a predisposition to be more antisocial behaviour which has resulted in them incurring a MHI; otherwise they most probably would not have successfully been able to enter and remain in a university program. Clearly, while individuals with MHI were significantly more antisocial than their non-injured counterparts, they were not near the ceiling of any of the social measures, indicating that they were still in a non-clinical or lower risk range (which was expected due to the subtly of the injury). Nevertheless, due to the cross-sectional and correlational nature of this research, the argument that individuals with a history of MHI were premorbidly more antisocial which increased the probability of incurring a MHI cannot be completely ruled out.

Therefore, although the results from this study have elucidated several crucial relationships between MHI and social behaviour, there are some limitations that must be addressed in order to continue to make substantial contributions in the area of MHI research, as well as to develop innovative research designs to further elaborate on the findings from the current study.

Although this study targeted the inclusion criterion of the subjects acknowledging a history of experiencing an impact injury to the head sufficient to produce an altered state of consciousness, it did not include a specific measure of injury severity, and as such, it is unclear as to the extent of injury individuals may have experienced. The study emphasizes the "mild" aspect of the injury in the subject population, but it is entirely possible that some individuals had experienced a more extensive injury (e.g. loss of consciousness for a prolonged period of time) and therefore include individuals with moderate head injury, and possibly moderate brain injury, as there were no exclusion criteria. Therefore, the qualification of MHI in the current study might not be accurate since the population of subject injuries might not be homogenous. However, it is also likely that the MHI subject population's injuries are "mostly mild" since it is clear that the MHI and non-MHI groups in this study were selected for cognitive and functional competence (i.e. only university students were examined) and they did not differ on various indices that would implicate experiencing more severe forms of head injury. For instance, they did not differ in the amount of extra assistance required throughout their educational history, such as from a learning resource teacher, tutor, educational assistant, speech language pathologist, occupational therapist, or physical therapist, all of which are sensitive markers of more severe cognitive, behavioural, or orthopedic sequelae. Both

groups were nominally equivalent in the amount of education they had completed and in their overall satisfaction of their academic experience and their lives more generally, which are both predominantly affected by more severe forms of head injury. Furthermore, neither group differed on a global measure pertaining to the number of hospitalizations, operations or neurological complications the subject had reported, which is particularly sensitive to moderate as well as catastrophic forms of TBI, as individuals who incur these more severe injuries require more hospitalized attention and can suffer from various neurological complications due to their injuries. Most convincingly, the MHI group did not perform significantly more poorly than the non-MHI group on neuropsychological measures of executive performance which are sensitive to frontal lobe integrity, exemplifying the subtle and "mild" nature of the head injuries in the MHI group. Taken together, although there was no specific exclusion criterion for more severe head trauma in the current study, post hoc analyses revealed ample evidence demonstrating the high competency of both groups, as well as the subtle nature of the MHI, and so at the very least the subject population injuries are arguably "mostly" mild in nature. Future studies, however, would benefit from including questions of injury severity, for example regarding whether or not the head trauma resulted in a loss of consciousness (LOC) and if so, the approximate length of LOC in order to more specifically exclude, and/or identify, individuals with more severe forms of head injury.

The cross-sectional and correlational nature of the present study requires some consideration when addressing the issue of direction or causality. Logistically, however, a randomized design with human participants would be impossible and extremely unethical, as MHI could never be randomly assigned to human participants. An

alternative, future study could use a longitudinal design to attempt to directly determine pre-injury social behaviour. For instance, university athletes could be tested at the beginning of their first season with social measures and then after any subsequent head injuries throughout the year in order to obtain a rough estimate of premorbid and post-injury social behaviour. This prospective design; however, is also limited by a number of constraints. A crucial methodological flaw is the lack of a true "baseline," as university-caliber athletes most probably have incurred a MHI over the course of their earlier training. Furthermore, the results of social behaviour measures (such as aggression) in athletes may not be particularly generalizable to the average university student, especially if the preferred sport is highly violent in nature (e.g. football or wrestling). Therefore, designing a future longitudinal study using athletes is a possible alternative design; however, it is also limited by a number of inherent constraints.

Future studies should also include corroborative information from family and/or close friends regarding the extent of the individual's change in personality and social behaviour after incurring a MHI. Therefore, a questionnaire could be developed that requires the observer to estimate the individual's behaviour both before and after his/her injury. This additional information might help to further elucidate changes in social behaviour after a MHI, as opposed to relying solely on self-report information from the injured individual.

With respect to creating potential experimental designs, animal studies can be used to investigate causal relationships in MHI research. The promising research of Gurgoff, Giza, and Hovda (2006) has revealed a causal relationship between mild trauma to the brain (that does not result in significant neuronal loss and, rather, simulates

concussion) and cognitive impairment in rats. Unfortunately, the generalizability of these results to humans is limited as there is a substantial difference in the neural and/or cognitive complexity between the frontal lobe of humans and rats, although this research remains highly pertinent and crucial to the field. Even more promising would be a study using non-human primates due to the large overlap in cytoarchitecture and social behaviour (Wallis, 2007). There have been numerous studies utilizing non-human primates investigating the effects of brain injury on social behaviour, but there is a paucity of research examining the relationship between MHI and social behaviour in non-human primates. Therefore, primate research could be used to investigate many of the causal impossibilities that plague research design using human participants; however, imposing neural trauma on primates is a serious ethical issue which would have to be approved by the most stringent animal care guidelines.

Future studies would also benefit by asking specific information regarding the etiology of the MHI in order to further investigate pre-morbid differences in social behaviour. For instance, a MHI could have been the result of a predisposing characteristic (e.g. a boxer receiving a head injury during a match) or could have been incurred by an incident unrelated to pre-morbid social behaviour (e.g. due to a random assault). This information is relatively easy to obtain and may be useful in order to help further clarify the directionality of the correlational data.

Conclusion and Implications

Taken together, the results of this study have impressively demonstrated the constellation of social deficits that can afflict the lives of individuals who have incurred a MHI among a university sample. Furthermore, these social consequences are identical

(albeit to a lesser degree) to the sequelae of OFC damage which includes reactive aggression, impulsive antisocial behaviour, and behavioural disinhibition. Thus, these findings further implicate the damaging effects of MHI on orbitofrontal functioning, as the proximity of the bony orbits of the eyes facilitates injury to the OFC at all severity levels of trauma. Furthermore, the findings of this study imply a continuum of behavioural deficits between MHI and more severe forms of brain injury, as opposed to the literature that dichotomizes brain injury and head injury as distinct neurological disorders.

Unfortunately, this dichotomy stigmatizes individuals with a history of MHI as "malingerers," whereas the results of this study have convincingly revealed that "mild" head trauma is far from inconsequential, as the effects of these injuries disrupt the social competencies that human beings rely on the most for adaptive social interaction. Therefore, the effects of MHI can also have a devastating and widespread impact on the lives of family and friends. These issues must be further addressed in the research domain through multidisciplinary studies involving researchers in the areas of social psychology and neuropsychology, in order better understand both the neurological changes that occur after MHI, as well as the effects that these injuries have on social interactions more generally. These issues also require more clinical insight into more sensitive diagnostic measures, as well as empirically supported treatment modalities that address the social deficits that may accompany individuals with a MHI.

Ultimately, much like individuals with more severe forms of TBI, the social consequences of MHI can have devastating ramifications impacting interpersonal relationships, vocational/educational success, and quality of life more generally. The

need for societal acknowledgement is crucial, since impact injuries are a leading cause of neurological impairment across the lifespan and the untreated effects of head trauma can have serious social consequences. Therefore, it is crucial to the lives of many individuals in need of help to continue to elucidate the consequences of MHI through innovative research in order to catalyze change in clinical practice and legislation.

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Appendix A - Demogarphics

1. How old are you? ____

2. Gender? M___ F____

3. What is the highest level of education you have completed?
   High School/Gr.12 Univ. 1 2 3 4 Univ. 4+

4. Handedness? Right Left Both

5. Have you ever hit your head against a surface or object sufficient to alter your consciousness (e.g. loss of consciousness, vomiting, dizziness)? Y N

6. Did it result in a concussion? Y N

7. Did it require stitches? Y N

8. How old were you at the time(s) 1-3 4-6 7-9 10-12 13-15 16-18 19-21 22+

9. Have you ever been hospitalized for fractures, illness, surgery or neurological complications? Y N

10. Do you have sensitivity to perfumes or scents? Y N

    If yes, please rate your sensitivity:

Not at all Very
1 2 3 4 5 6 7 8 9

11. Do you wear glasses or contacts? Y N

12. Do you have a valid driver's license? Y N

13. How long have you had a driver's license? 1-3 years 4-6 years 7+ years

14. Do you smoke cigarettes? Y N
    If yes, approximately how many a day? _____

15. Do you live: on your own with roommates with parents/guardians

16. Have you ever received any extra assistance during your educational history (e.g. learning resource teacher, tutor, educational assistant, speech language pathologist, occupational therapist, physical therapist)? Y N

17. How many university credits (courses) are you currently taking?

0.5 1 1.5 2 2.5 3 3.5 4 4.5 5 5.5 6

18. How many academic assignments or exams have you completed in the past month?

1 2 3 4 5 6 7 8 9 10

11 12 13 14 15 16 17 18 19 20

19. On a scale of 1 to 9 rate your enjoyment of academics:

Not at all Very
1 2 3 4 5 6 7 8 9

20. On a scale of 1 to 9 rate your enjoyment of your life situation:

Not at all Very
1 2 3 4 5 6 7 8 9

21. Circle any of the following that apply to your experience over the past 6 months:

Moved
New Job
Loss of Job
Loss of Relationship
New Relationship
Reconciliation with partner
Reconciliation with Family
Divorce (of self or parents)
Entered 1st year at university
Death of a family member
Death of a close friend
Financial Difficulties
Illness of someone close to you
Personal Illness/Injury
New Baby
Wedding/ Engagement (self)
Vacation
Sleep less than 8 hours per night

Appendix B – Barratt Impulsiveness Scale-11

Please indicate to what extent you agree with the following statements using the following scale:

1	2	3	4
Rarely/Never	Occasionally	Often	Almost Always/Always

1	I plan tasks carefully.	1	2	3	4
2	I do things without thinking	1	2	3	4
3	I make up my mind very quickly	1	2	3	4
4	I am happy-go-lucky.	1	2	3	4
5	I don't 'pay attention'	1	2	3	4
6	I have 'racing' thoughts.	1	2	3	4
7	I plan trips well ahead of time.	1	2	3	4
8	I am self-controlled.	1	2	3	4
9	I concentrate easily	1	2	3	4
10	I save regularly.	1	2	3	4
11	I "squirm" at plays or lectures	1	2	3	4
12	I am a careful thinker	1	2	3	4
13	I plan for job security.	1	2	3	4
14	I say things without thinking.	1	2	3	4
15	I like to think about complex problems.	1	2	3	4
16	I change jobs.	1	2	3	4
17	I act 'on impulse'	1	2	3	4
18	I get easily bored when solving thought problems.	1	2	3	4
19	I act on the spur of the moment	1	2	3	4
20	I am a steady thinker	1	2	3	4
21	I change residences.	1	2	3	4
22	I buy things on impulse	1	2	3	4
23	I can only think about one problem at a time.	1	2	3	4
24	I change hobbies.	1	2	3	4
25	I spend or charge more than I can earn.	1	2	3	4
26	I often have extraneous thoughts when thinking.	1	2	3	4
27	I am more interested in present than in future.	1	2	3	4
28	I am restless at the theater or lectures	1	2	3	4
29	I like puzzles.	1	2	3	4
30	I am future oriented.	1	2	3	4

Appendix C – BIS/BAS Scale

Each item of this questionnaire is a statement that a person may either agree with or disagree with. For each item, indicate how much you agree or disagree with what the item says. Please respond to all the items; do not leave any blank. Choose only one response to each statement. Please be as accurate and honest as you can be. Respond to each item as if it were the only item. That is, don't worry about being "consistent" in your responses. Choose from the following four response options:

1 = very true for me
2 = somewhat true for me
3 = somewhat false for me
4 = very false for me

1. A person's family is the most important thing in life.
2. Even if something bad is about to happen to me, I rarely experience fear or nervousness.
3. I go out of my way to get things I want.
4. When I'm doing well at something I love to keep at it.
5. I'm always willing to try something new if I think it will be fun.
6. How I dress is important to me.
7. When I get something I want, I feel excited and energized.
8. Criticism or scolding hurts me quite a bit.
9. When I want something I usually go all-out to get it.
10. I will often do things for no other reason than that they might be fun.

11. It's hard for me to find the time to do things such as get a haircut.
12. If I see a chance to get something I want I move on it right away.
13. I feel pretty worried or upset when I think or know somebody is angry at me.
14. When I see an opportunity for something I like I get excited right away.
15. I often act on the spur of the moment.
16. If I think something unpleasant is going to happen I usually get pretty "worked up."
17. I often wonder why people act the way they do.
18. When good things happen to me, it affects me strongly.
19. I feel worried when I think I have done poorly at something important.
20. I crave excitement and new sensations.

21. When I go after something I use a "no holds barred" approach.2

22. I have very few fears compared to my friends.
23. It would excite me to win a contest.

24. I worry about making mistakes.

Appendix D – Buss and Perry Aggression Questionnaire

Instructions:
Using the 5 point scale shown below, indicate how uncharacteristic or characteristic each of the following statements is in describing you. Place your rating to the right of the statement.
1 = extremely uncharacteristic of me
2 = somewhat uncharacteristic of me
3 = neither uncharacteristic nor characteristic of me
4 = somewhat characteristic of me
5 = extremely characteristic of me

1. Some of my friends think I am a hothead
2. If I have to resort to violence to protect my rights, I will.
3. When people are especially nice to me, I wonder what they want.
4. I tell my friends openly when I disagree with them.
5. I have become so mad that I have broken things.
6. I can't help getting into arguments when people disagree with me.
7. I wonder why sometimes I feel so bitter about things.
8. Once in a while, I can't control the urge to strike another person.
9.*I am an even-tempered person.
10. I am suspicious of overly friendly strangers.
11. I have threatened people I know.
12. I flare up quickly but get over it quickly.
13. Given enough provocation, I may hit another person.
14. When people annoy me, I may tell them what I think of them.
15. I am sometimes eaten up with jealousy.
16.*I can think of no good reason for ever hitting a person.
17. At times I feel I have gotten a raw deal out of life.
18. I have trouble controlling my temper.
19. When frustrated, I let my irritation show.
20. I sometimes feel that people are laughing at me behind my back.
21. I often find myself disagreeing with people.
22. If somebody hits me, I hit back.
23. I sometimes feel like a powder keg ready to explode.
24. Other people always seem to get the breaks.
25. There are people who pushed me so far that we came to blows.
26. I know that "friends" talk about me behind my back.
27. My friends say that I'm somewhat argumentative.
28. Sometimes I fly off the handle for no good reason.
29. I get into fights a little more than the average person.

Appendix E – Self-Report Psychopathy Scale - III (R11)

Please rate the degree to which you agree with the following statements about you. You can be honest because your name will be detached from the answers as soon as they are submitted.

1	2	3	4	5
Disagree Strongly	Disagree	Neutral	Agree	Agree Strongly

1. I'm a rebellious person.
2. I'm more tough-minded than other people.
3. I think I could "beat" a lie detector.
4. I have taken illegal drugs (e.g., marijuana, ectasy).
5. I have never been involved in delinquent gang activity.
6. I have never stolen a truck, car or motorcycle.
7. Most people are wimps.
8. I purposely flatter people to get them on my side.
9. I've often done something dangerous just for the thrill of it.
10. I have tricked someone into giving me money.
11. It tortures me to see an injured animal.
12. I have assaulted a law enforcement official or social worker.
13. I have pretended to be someone else in order to get something.
14. I always plan out my weekly activities.
15. I like to see fist-fights.
16. I'm not tricky or sly.
17. I'd be good at a dangerous job because I make fast decisions.
18. I have never tried to force someone to have sex.
19. My friends would say that I am a warm person.
20. I would get a kick out of 'scamming' someone.
21. I have never attacked someone with the idea of injuring them.
22. I never miss appointments.
23. I avoid horror movies.
24. I trust other people to be honest.
25. I hate high speed driving.
26. I feel so sorry when I see a homeless person.
27. It's fun to see how far you can push people before they get upset.
28. I enjoy doing wild things.
29. I have broken into a building or vehicle in order to steal something or vandalize.
30. I don't bother to keep in touch with my family any more.
31. I find it difficult to manipulate people.
32. I rarely follow the rules.
33. I never cry at movies.
34. I have never been arrested.
35. You should take advantage of other people before they do it to you.

36. I don't enjoy gambling for real money.
37. People sometimes say that I'm cold-hearted.
38. People can usually tell if I am lying.
39. I like to have sex with people I barely know.
40. I love violent sports and movies.
41. Sometimes you have to pretend you like people to get something out of them.
42. I am an impulsive person.
43. I have taken hard drugs (e.g., heroin, cocaine).
44. I'm a soft-hearted person.
45. I can talk people into anything.
46. I never shoplifted from a store.
47. I don't enjoy taking risks.
48. People are too sensitive when I tell them the truth about themselves.
49. I was convicted of a serious crime.
50. Most people tell lies everyday.
51. I keep getting in trouble for the same things over and over.
52. Every now and then I carry a weapon (knife or gun) for protection.
53. People cry way too much at funerals.
54. You can get what you want by telling people what they want to hear.
55. I easily get bored.
56. I never feel guilty over hurting others.
57. I have threatened people into giving me money, clothes, or makeup.
58. A lot of people are "suckers" and can easily be fooled.
59. I admit that I often "mouth off" without thinking.
60. I sometimes dump friends that I don't need any more.
61. I would never step on others to get what I want.
62. I have close friends who served time in prison.
63. I purposely tried to hit someone with the vehicle I was driving.
64. I have violated my probation from prison.

Appendix F – Informed Consent

Title of Study: Cognitive abilities and their relationship with decision-making and personality

Principal Investigator: Tony DeBono (Masters candidate, Dept. of Psychology)

Co-investigator: Dr. Dawn Good, Associate Professor, Department of Psychology, Brock University

Thank you for participating in this research study. The principal investigator of this study Tony DeBono, Masters Candidate, and co-investigator Dr. Dawn Good, Assoc. Prof., Psychology Dept., Brock, invite you to participate in a research project.

The purpose of this research project is to investigate the relationship between performance on cognitive tasks and measures of decision making in university students, as well as investigating whether personality change can be predicted by cognitive abilities.

The expected duration of this study is two hours. Although there are no foreseeable risks for participating in this study it is possible that you may feel challenged or anxious by completing tests and wondering how you did. Know that you are not being evaluated and that the tests will increase in difficulty so that we can measure optimum performance

As a participant you will be able to gain first-hand experience of scientific approach that neuroscientists take while studying mind-brain relationships. Additionally, you will get a chance to be exposed to standardized and protected neuropsychological measures that are not readily available to the public.

This research is being conducted through the Psychology Department and is a single-site project that will only take place at Brock University.

Name of Participant (please print): _____

- I understand that this study involves research, and that I am being invited to participate
- I understand that the purpose of this study is to evaluate the relationship between cognitive measures and decision making.
- I understand that the expected duration of my participation in this study is two hours
- I understand the procedures to be followed, which include
  - Reading the invitation letter and signing the informed consent form
  - Completing self-report questionnaires asking to rate a number of statements on a given scale or choose one of the provided options
  - Completing cognitive tasks that assess attention, problem-solving and memory
  - At the conclusion of testing, reading the debriefing statement

- I understand the risks & benefits associated with this research, which include receiving credit and/or bonus marks for participation in research for the PSYC1F90 class and other upper year PSYC courses
- I understand that I may experience mild symptoms of discomfort (physical, or cognitive) simulating stress not unlike that which I could experience during a test or assignment in a university environment
- I understand that all data will be numerically coded to ensure confidentiality.
- I understand that only the Principal & Co - Investigators & the Research Assistants will have access to my data, and that all information will be stored securely until the completion of this study and all data will be kept for five years
- I understand that participation is voluntary; refusal to participate will involve no penalty or loss of benefits to which I am otherwise entitled and I may discontinue participation at any time without penalty or loss of benefits, to which I am otherwise entitled
- I understand that the data that is collected is anonymous and, therefore, cannot be withdrawn once submitted
- I understand that I will be able to speak with the Principal Investigator and also with the academic and personal counselling services provided at Brock University regarding any difficulties/responses to this study
- I understand that I may contact by the Principal/Co-Investigators if I am interested in finding out the results of this study
- I understand that the results of this study will be published (thesis report and research articles) and presented (thesis defence presentation and graduate conference presentation).
- I understand that this research is being conducted through the Psychology/Neuroscience Department
- I understand that if I have any pertinent questions about my rights as a research participant, I can contact the Brock University Research Ethics Officer (905 688-5550 ext. 3035, reb@brocku.ca)

I _____ *(Please print)*

1. *Have read and understood the relevant information regarding this research project*
2. *Understand that I may ask questions in the future*
3. *Understand that I am participating in this experiment for up to a maximum of two research participation hours in a psychology course and will not receive monetary payment for this experiment*
4. *Indicate free consent to research participation by signing this research consent form*
5. *Have received a copy of the consent form*

Course Participation (Please circle only one course)
PSYC 1F90 2F12 2F20 2F23 2F36 3P39
Other:_____
Participant's Signature: _____ Date:
_____

I have explained this study to the participant
Researcher's Signature: _____ Date: _____

Dr. Dawn Good
Department of Psychology
(905) 688-5550 x3869
dawn.good@brocku.ca

Tony DeBono
Masters candidate
(905) 688-5550 x3034
td06al@brocku.ca

This study has been reviewed and received ethics clearance through Brock University's Research Ethics Board (file #06-192)

Appendix G – Debriefing Form

Dear Participant:
Thank you for your participation in this research study. As you are aware, this research study was conducted by Tony DeBono and Dr. Dawn Good in the Psychology Department at Brock University. The purpose of this study was to investigate whether personality change could be predicted by having incurred a mild head injury and by performance on neuropsychological measures.

Many questions pertaining to the effects of mild head injury remain relatively unanswered due to an overall lack of research in the field. Thus, the majority of research in the area has only focused on moderate and severe brain injuries. For instance, Rowe et al. (2001) have found that the ability to infer the mental states of others (Theory of Mind) is impaired in patients with frontal lobe lesions. Furthermore, Blair (2001) found marked changes in behaviour after severe damage to the orbitofrontal cortex (a region of the prefrontal cortex). These individuals display antisocial levels of reactive aggression, which means that they retaliate (usually violently) whenever they feel that they have been provoked primarily due to a lack of inhibitory control. Presently, there is no known study that addresses the impact of mild head injury on personality change, particularly in high performing populations, such as university students. It is hypothesized that frontal lobe performance, presence of mild head injury, and impaired Theory of Mind will predict discernible results on self-report assessments of impulsivity, antisocial behaviour, and aggression.

The standardized neuropsychological measures used in this study were subtasks from the Wechsler Adult Intelligence Scale – III (1997), the Delis Kaplan Executive Function System (2002), the NEPSY (1997) and the Comprehensive Test of Nonverbal Intelligence (1996). These tasks were used to assess problem solving skills, abstract reasoning, motor sequencing, cognitive flexibility, visual scanning, working memory and attention. The Stroop (an experimental measure) was used to measure behavioural inhibition. The Reading the Mind in the Eyes task (2001) was used to measure the ability to accurately describe photographs of human eyes.

The questionnaires in this study asked you to rate your level of agreement with real life scenarios. The Barratt Impulsiveness Scale (BIS 11; 1995) and Carver's BIS/BAS scale (1994) assessed levels of impulsiveness, whereas the Buss and Perry aggression Questionnaire (1992) and the Self Report Psychopathy Checklist III (R 11; in press) asked questions about antisocial and aggressive behaviour.

Your participation has been essential to the advancement of mild head injury research in exceptional populations, such as university students. You are invited to view the results of this study upon its completion (August 31, 2008) and to attend the thesis defence, which will be scheduled closer to the completion date.

If you experienced any negative emotions as a result of participating in this research study and wish to speak with a counsellor please contact: Brock University Counselling Services, ST 400, (905) 688-5550 extension 3240. If you feel you have not been treated according to the descriptions in this form, or your rights as a participant in research have been violated during the course of this project, you may contact the Research Ethics Officer at (905) 688-5550, extension 3035, please cite REB file #: 06-192

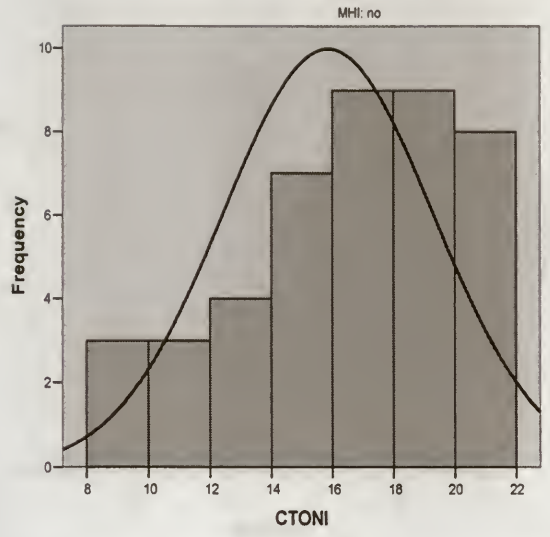
Thank you again for your time and support in participating in this study!!!

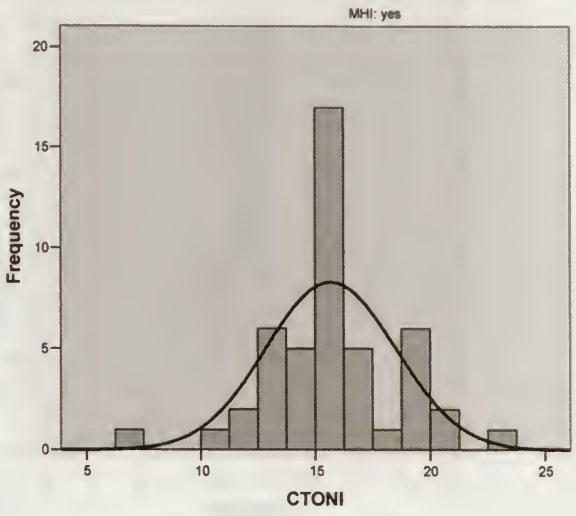
If you have any questions or concerns please feel free to contact us:

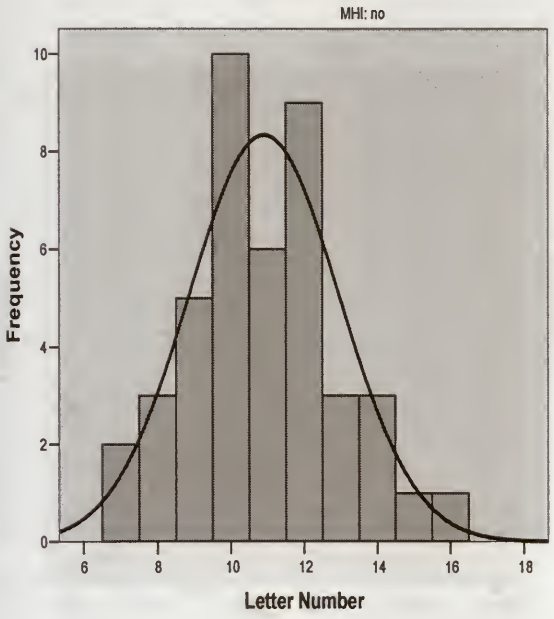
Tony DeBono
(905) 688-5550 ext: 3034
td06al@brocku.ca

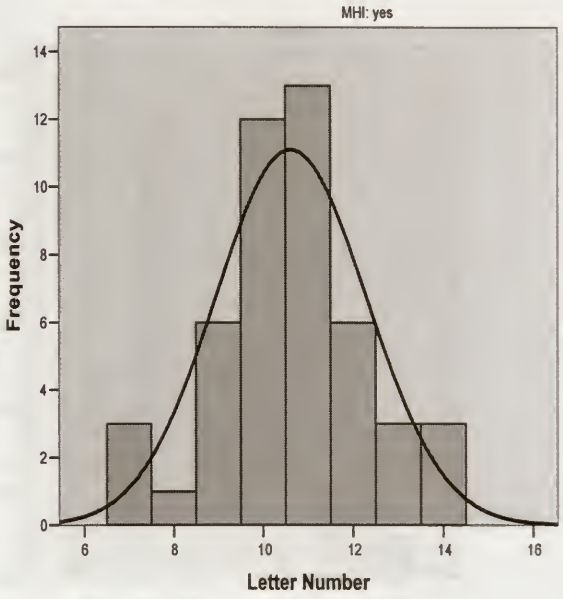
Dr. Dawn Good
(905) 688-5550 extension 3869
dawn.good@brocku.ca

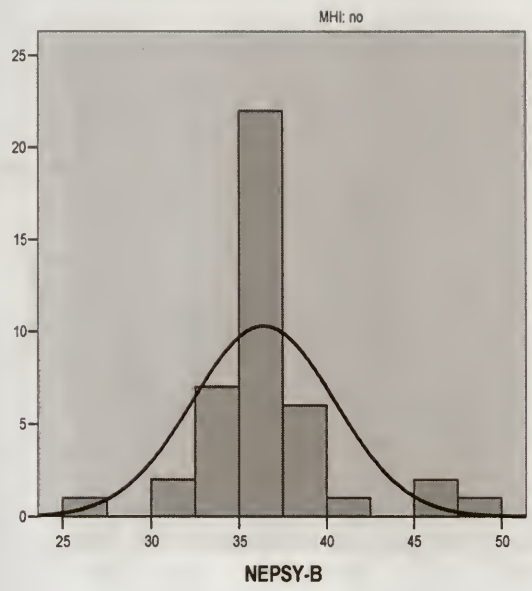
Appendix H – Frequency Distributions for Executive Functioning Measures







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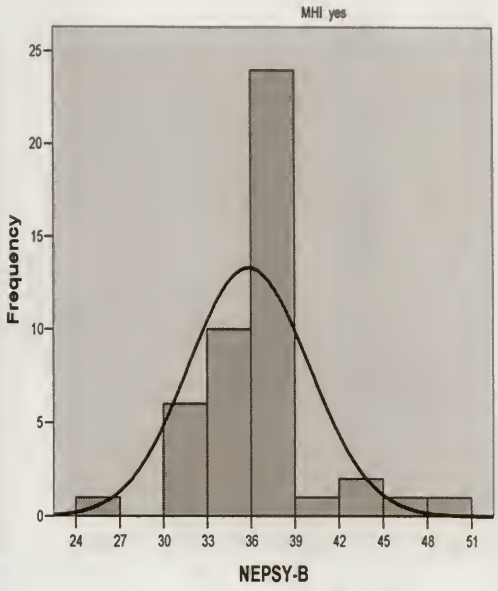
NEPSY-B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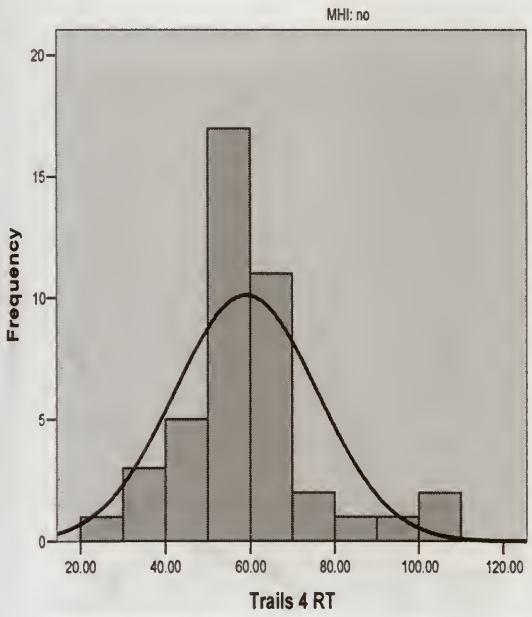
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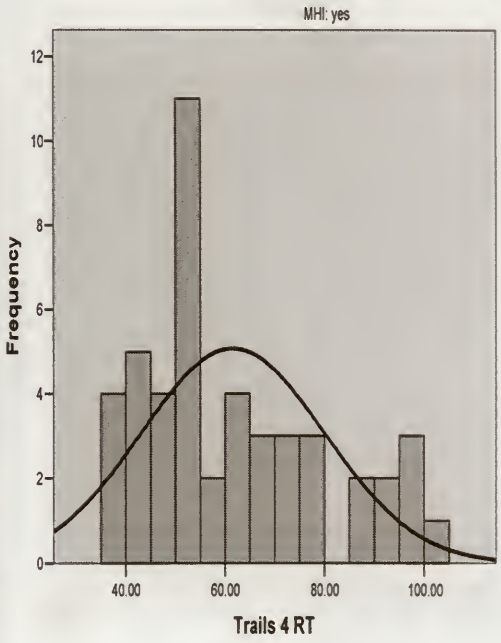
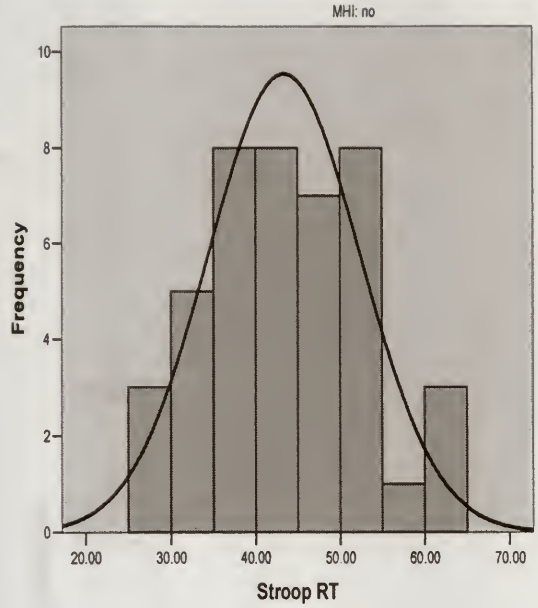
Frequency
Trails 4 RT


MHI: yes
Frequency
Trails 4 RT


MHI: no
Frequency
Stroop RT
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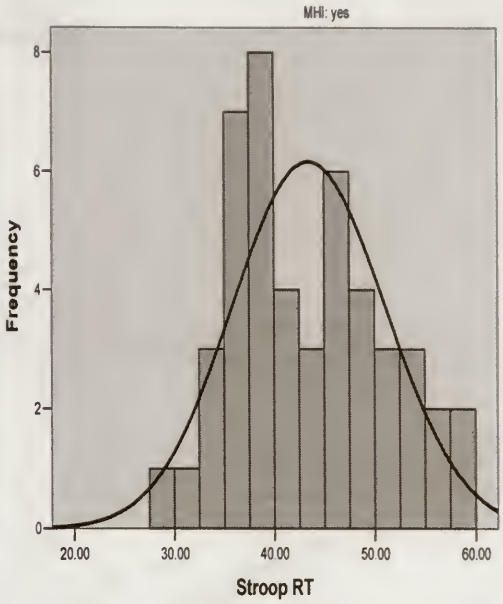
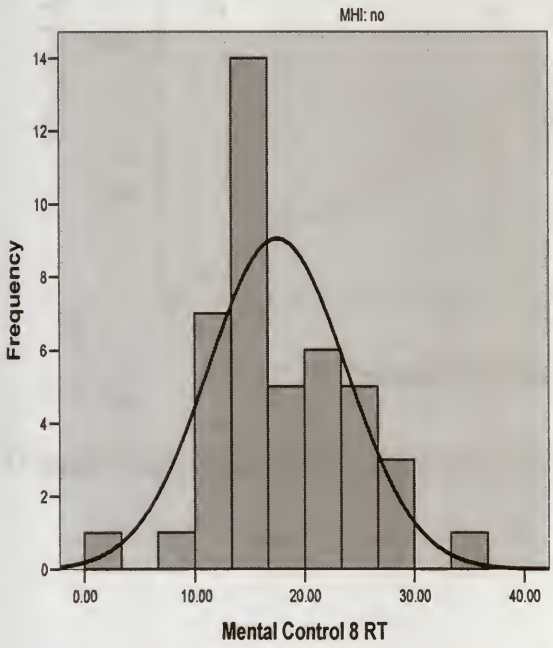
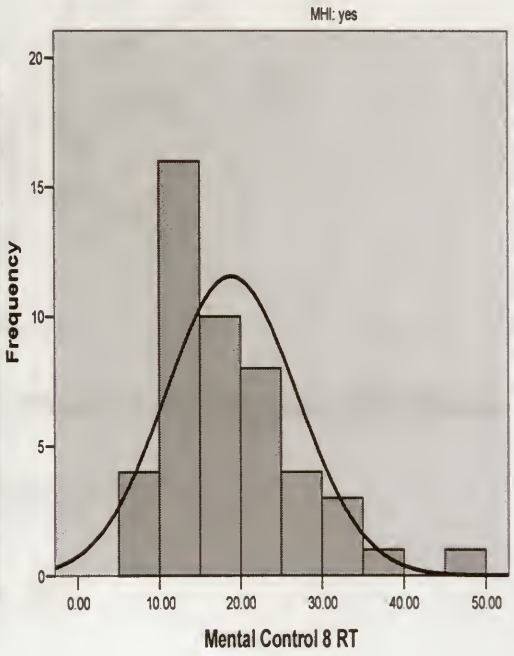
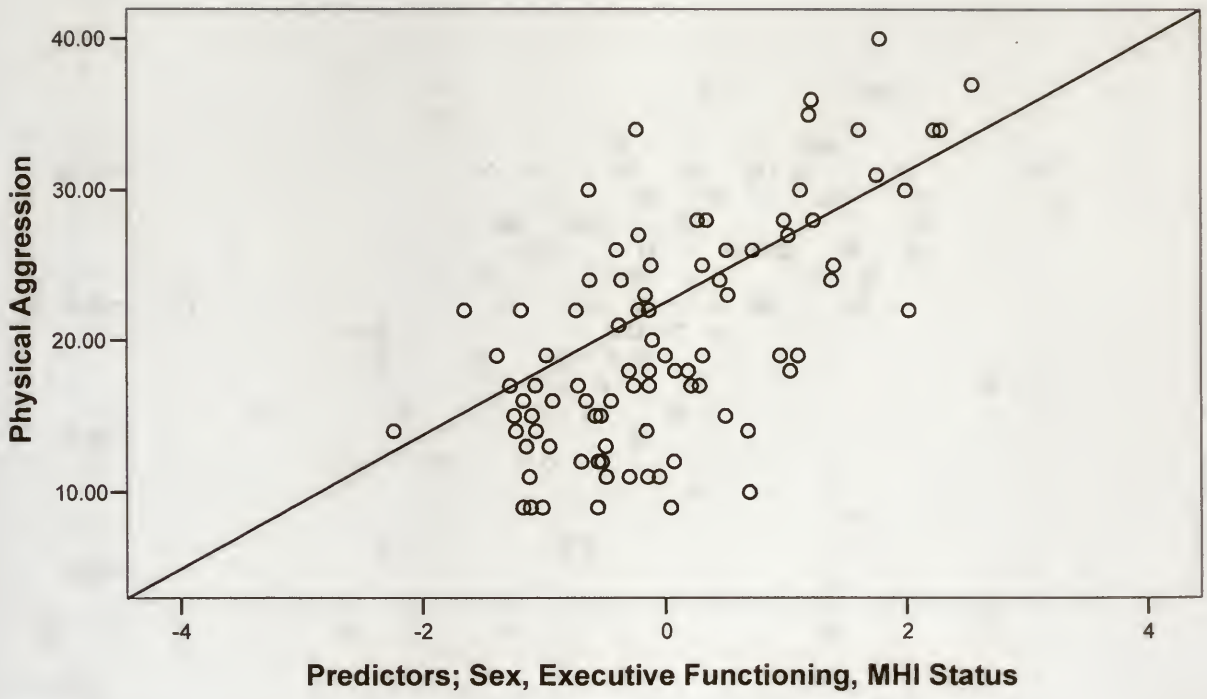
30.00
40.00
50.00
60.00
70.00


MHI: yes
Frequency
Stroop RT
20.00
30.00
40.00
50.00
60.00


MHI: no
Frequency
Mental Control 8 RT
0.00
10.00
20.00
30.00
40.00

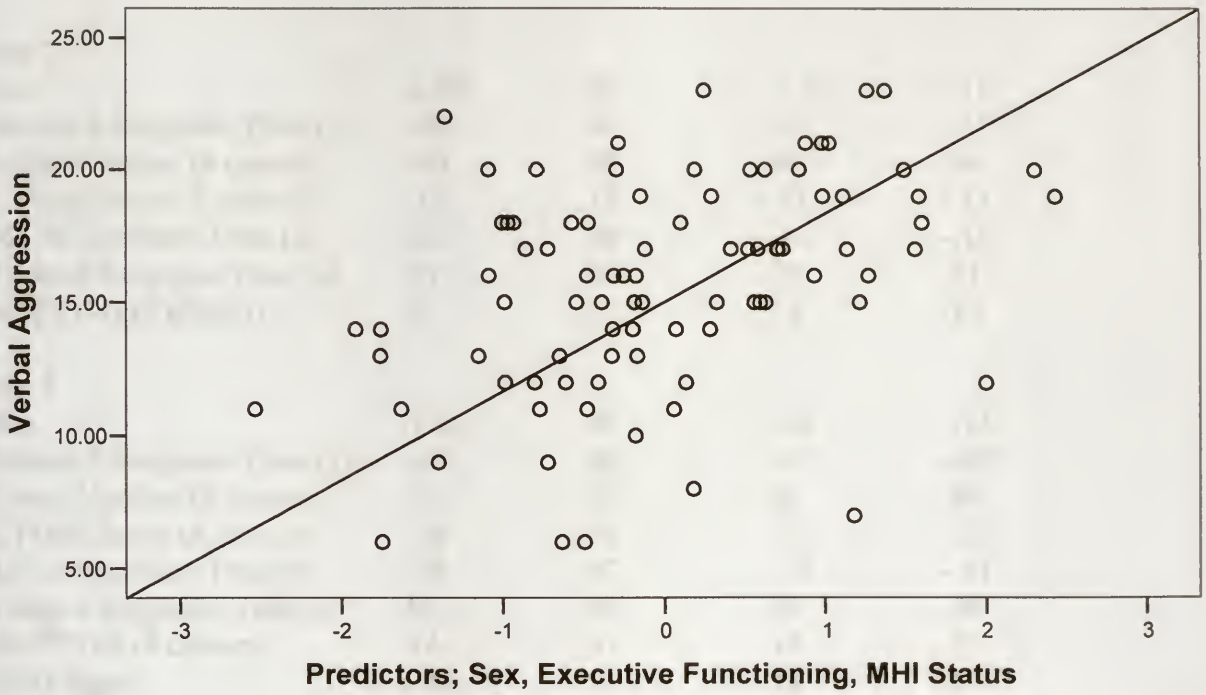

MHI: yes
Frequency
Mental Control 8 RT
0.00
10.00
20.00
30.00
40.00
50.00

Appendix I



Overall Regression Model of Physical Aggression

Appendix J



Overall Regression Model of Verbal Aggression

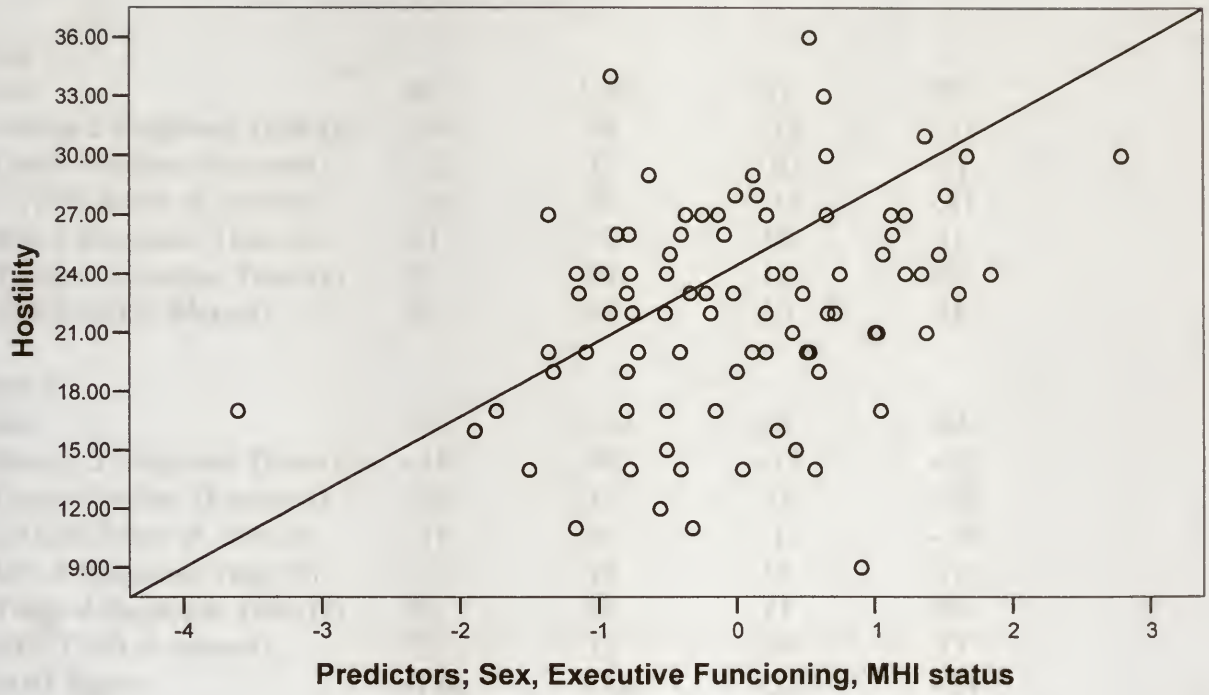
Appendix K

Summary of hierarchical multiple regression analysis in which verbal aggression was regressed on sex, executive functioning, and MHI status (N = 88)

Variable	*B*	*SE B*	*β*	*sr*
Step 1				
Sex	-1.80	.91	-.21	-.21
Step 2				
Sex	-1.48	.95	-.17	-.16
Stroop-3 Response Time (s)	-.06	.06	-.12	-.11
Letter-Number (# correct)	-.09	.25	-04	.04
CTONI Score (# correct)	-.15	.14	-.11	-.11
MC-8 Response Time (s)	-.07	.08	-.13	-.10
Trails-4 Response Time (s)	.06	.03	.25	.21
NEPSY-B (# correct)	.13	.11	.13	.12
Step 3				
Sex	-1.20	.95	-.14	-.13
Stroop-3 Response Time (s)	-.05	.06	-.11	-.09
Letter-Number (# correct)	.10	.25	.05	.04
CTONI Score (# correct)	-.16	.14	-.12	-.12
MC-8 Response Time (s)	-.08	.07	-.15	-.11
Trails-4 Response Time (s)	.06	.03	.25	.20
NEPSY-B (# correct)	.14	.11	.14	.13
MHI Status	1.42	.84	.18	.17

Note. R^2 = .04 for Step 1; ΔR^2 = .09 for Step 2; ΔR^2 = .03 for Step 3.

Appendix L



Overall Regression Model of Hostility

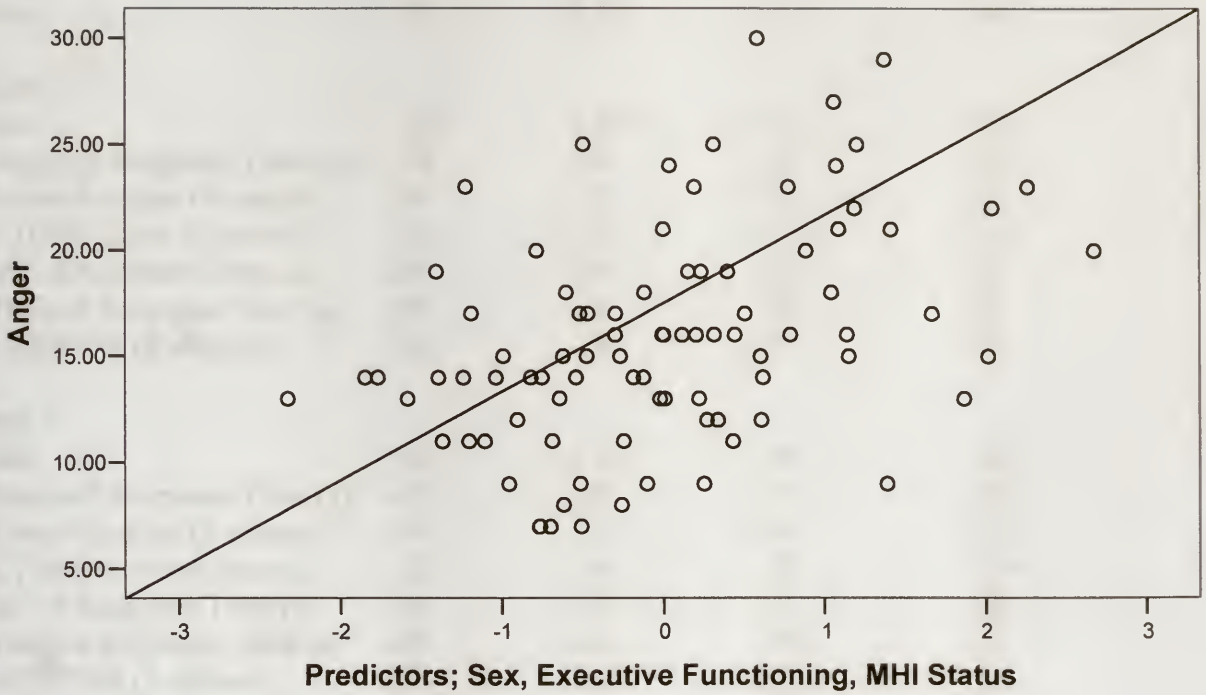
Appendix M

Summary of hierarchical multiple regression analysis in which hostility was regressed on sex, executive functioning, and MHI status (N = 88)

Variable	*B*	*SE B*	*β*	*sr*
Step 1				
Sex	.71	1.28	.06	.06
Step 2				
Sex	.84	1.33	.07	.07
Stroop-3 Response Time (s)	-.09	.08	-.13	-.11
Letter-Number (# correct)	-.05	.35	-.02	-01
CTONI Score (# correct)	-.20	.20	-.12	-.11
MC-8 Response Time (s)	.11	.11	.15	.11
Trails-4 Response Time (s)	.03	.04	.11	.09
NEPSY-B (# correct)	.26	.15	.20	.18
Step 3				
Sex	.53	1.34	.05	.04
Stroop-3 Response Time (s)	-.10	.08	-.15	-.12
Letter-Number (# correct)	-.06	.35	-.02	-.02
CTONI Score (# correct)	-.19	.20	-.11	-.10
MC-8 Response Time (s)	.12	.10	.16	.12
Trails-4 Response Time (s)	.03	.04	.11	.09
NEPSY-B (# correct)	.25	.15	.19	.17
MHI Status	-1.60	1.18	-.15	-.14

Note. R^2 = .00 for Step 1; ΔR^2 = .08 for Step 2; ΔR^2 = .02 for Step 3.

Appendix N



Overall Regression Model of Anger

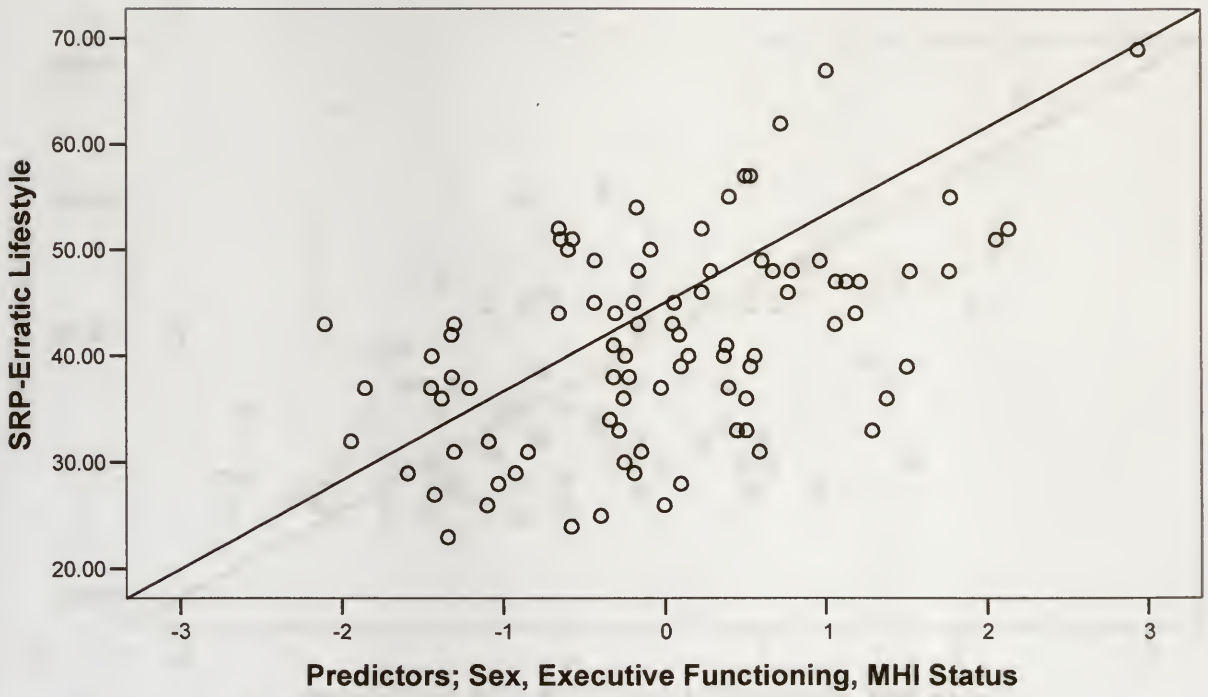
Appendix O

Summary of hierarchical multiple regression analysis in which anger was regressed on sex, executive functioning, and MHI status (N = 88)

Variable	*B*	*SE B*	*B*	*sr*
Step 1				
Sex	-.77	1.19	-.07	-.07
Step 2				
Sex	-.28	1.20	-.03	-.02
Stroop-3 Response Time (s)	-.08	.08	-.12	-.10
Letter-Number (# correct)	.09	.32	.03	.03
CTONI Score (# correct)	-.34	.18	-.21	-.19
MC-8 Response Time (s)	.01	.09	.01	.01
Trails-4 Response Time (s)	.08	.04	.28*	.23
NEPSY-B (# correct)	.26	.14	.21	.20
Step 3				
Sex	-.06	1.21	-.01	-.01
Stroop-3 Response Time (s)	-.07	.08	-.11	-.09
Letter-Number (# correct)	.10	.32	.04	.03
CTONI Score (# correct)	-.35	.18	-.21	-.20
MC-8 Response Time (s)	.00	.09	.00	.00
Trails-4 Response Time (s)	.08	.04	.29*	.23
NEPSY-B (# correct)	.27	.14	.22	.20
MHI Status	1.09	1.07	.11	.11

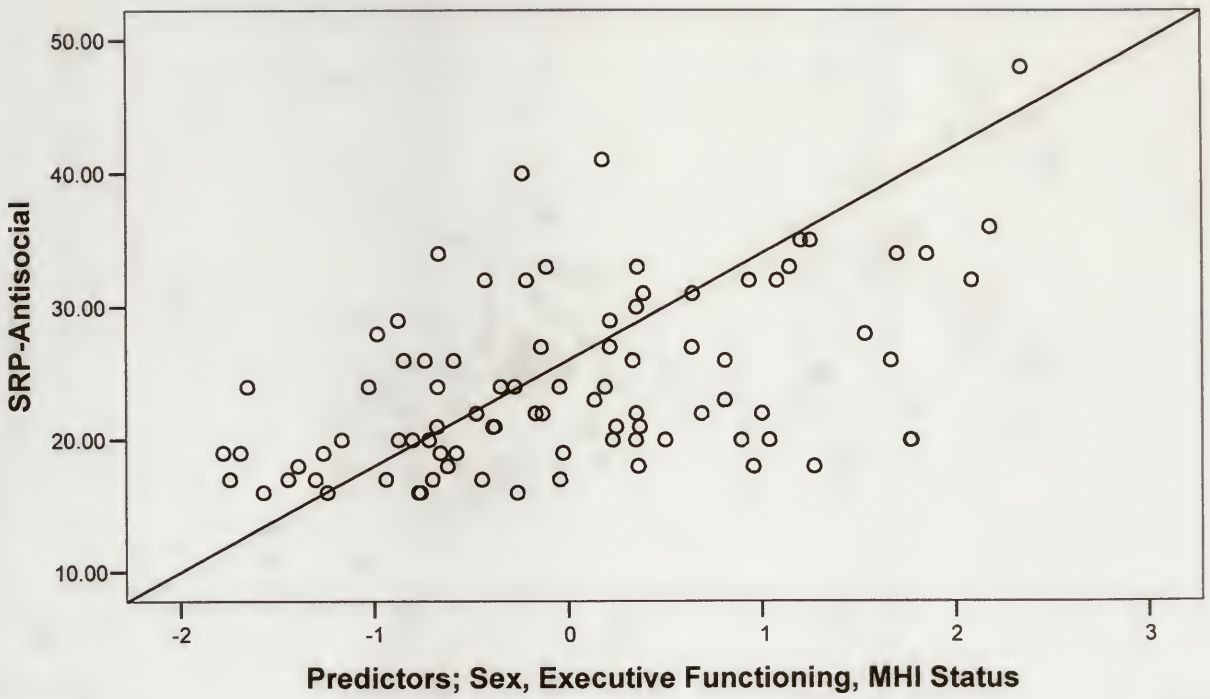
Note. $R^2 = .01$ for Step 1; $\Delta R^2 = .15$ for Step 2; $\Delta R^2 = .01$ for Step 3.
*$p < .05$

Appendix P



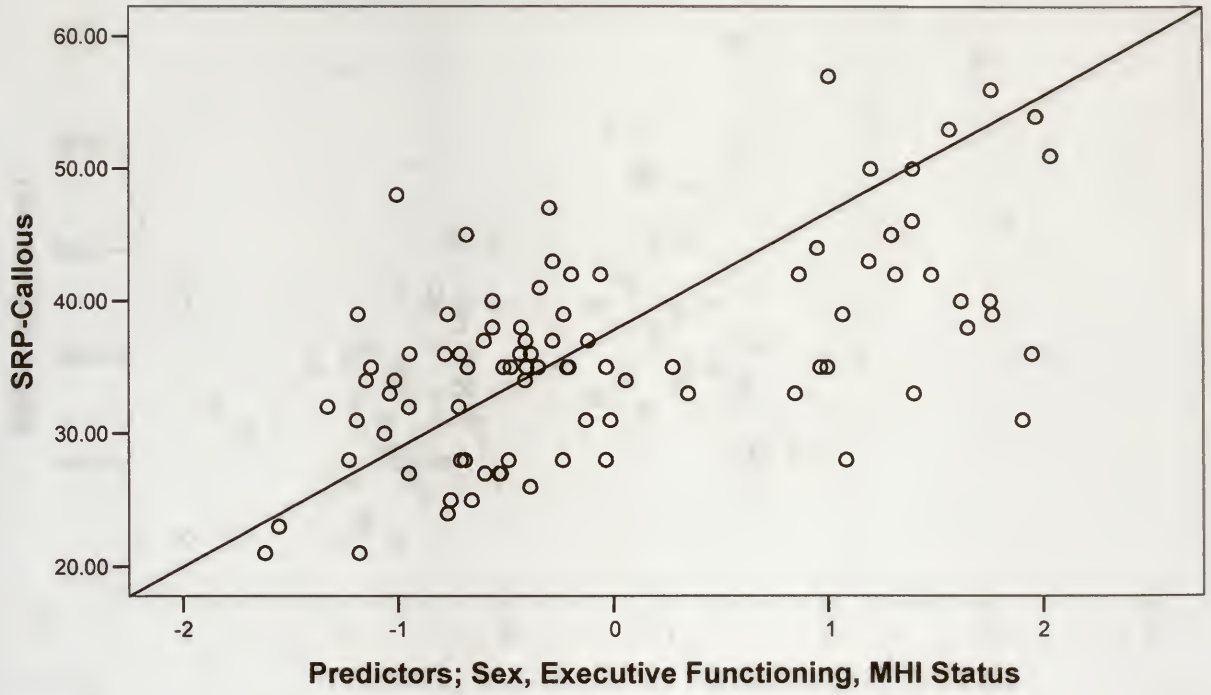
Overall Regression Model SRP-Erratic Lifestyle

Appendix Q



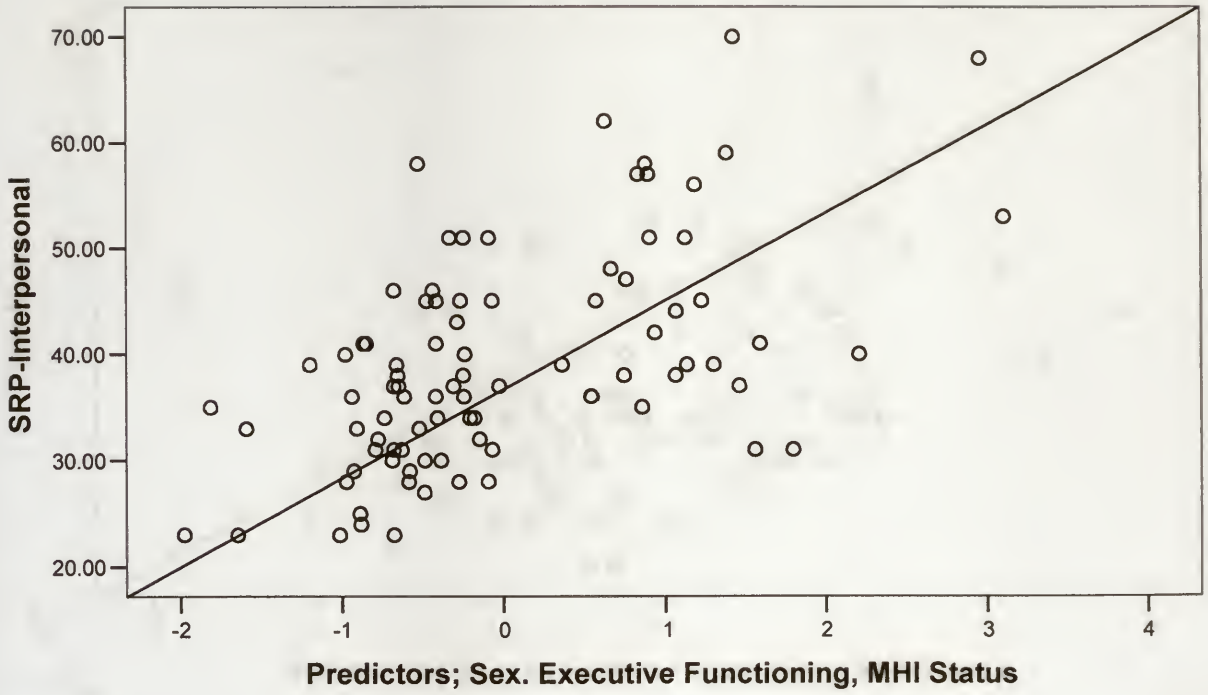
Overall Regression Model for SRP-Antisocial Behaviour

Appendix R



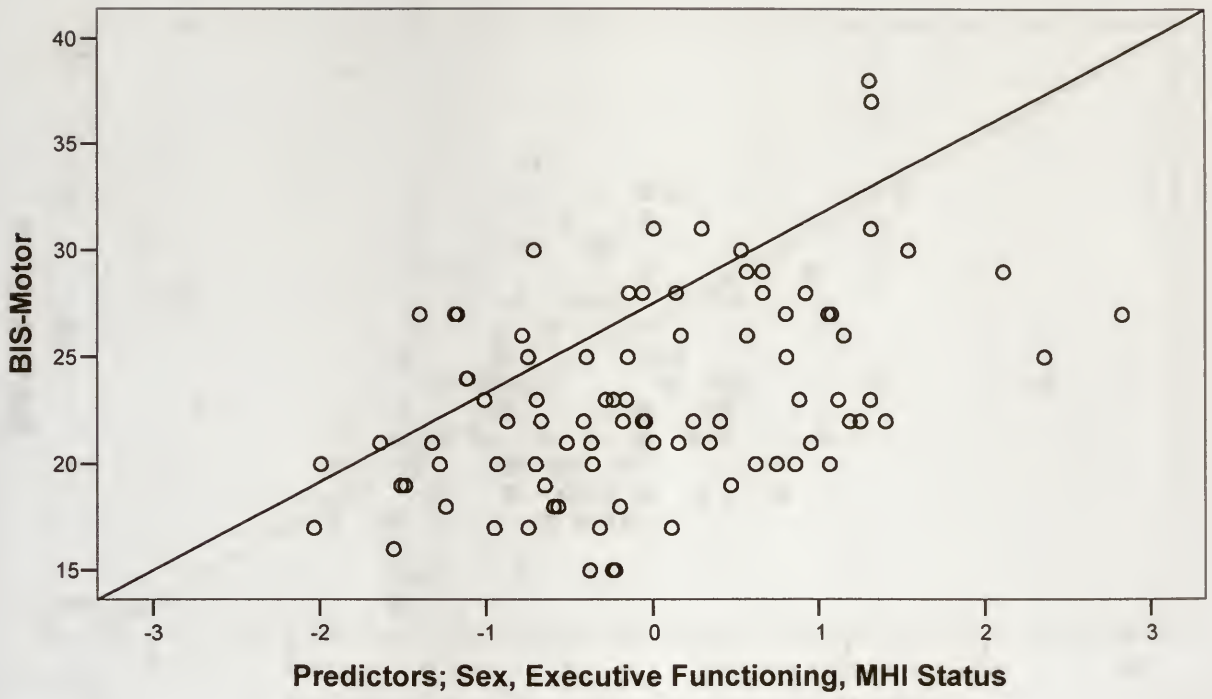
Overall Regression Model SRP-Callous Affect

Appendix S



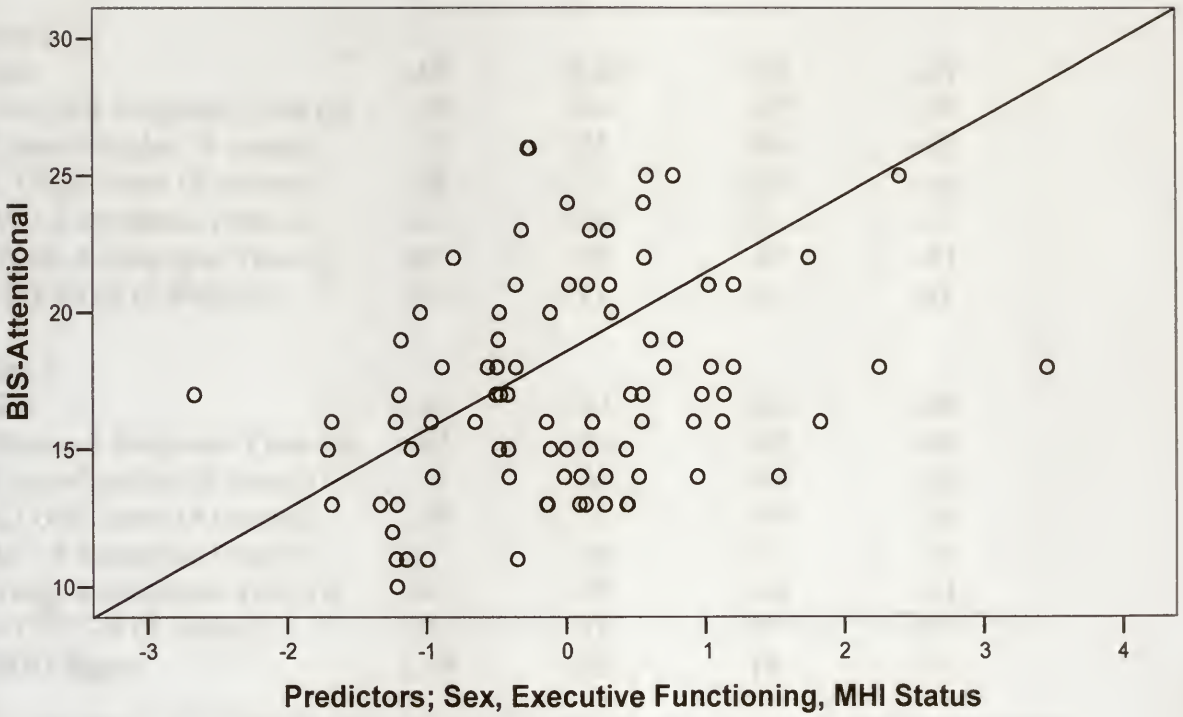
Overall Regression Model SRP-Interpersonal Manipulation

Appendix T



Overall Regression Model for BIS-motor

Appendix U



Overall Regression Model of BIS-attentional

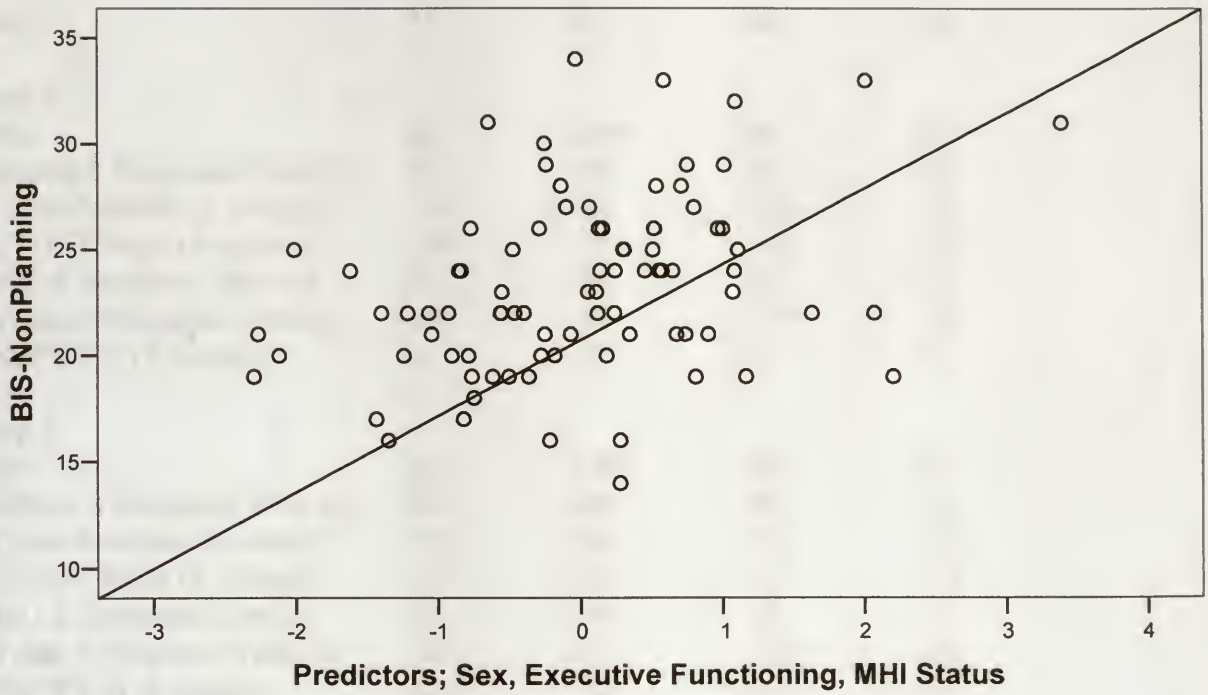
Appendix V

Summary of hierarchical multiple regression analysis in which BIS-attentional was regressed on sex, executive functioning, and MHI status (N = 87)

Variable	*B*	*SE B*	*β*	*sr*
Step 1				
Sex	-.67	.91	-.08	-.08
Step 2				
Sex	-.63	6.28	-.08	-.07
Stroop-3 Response Time (s)	-.03	.06	-.07	-.06
Letter-Number (# correct)	-.12	.25	-.06	-.05
CTONI Score (# correct)	-.08	.15	-.06	-.06
MC-8 Response Time (s)	.12	.08	.23	.17
Trails-4 Response Time (s)	.00	.03	-.01	-.01
NEPSY-B (# correct)	.00	.11	.00	.00
Step 3				
Sex	-.42	.97	-.05	-.05
Stroop-3 Response Time (s)	-.02	.06	-.05	-.04
Letter-Number (# correct)	-.11	.25	-.05	-.05
CTONI Score (# correct)	-.08	.15	-.06	-.06
MC-8 Response Time (s)	.11	.08	.21	.16
Trails-4 Response Time (s)	.00	.03	-.02	-.01
NEPSY-B (# correct)	.01	.11	.01	.01
MHI Status	1.19	.85	.16	.15

Note. $R^2 = .01$ for Step 1; $\Delta R^2 = .06$ for Step 2; $\Delta R^2 = .02$ for Step 3.
*$p < .05$

Appendix W



Overall Regression Model of BIS-nonplanning

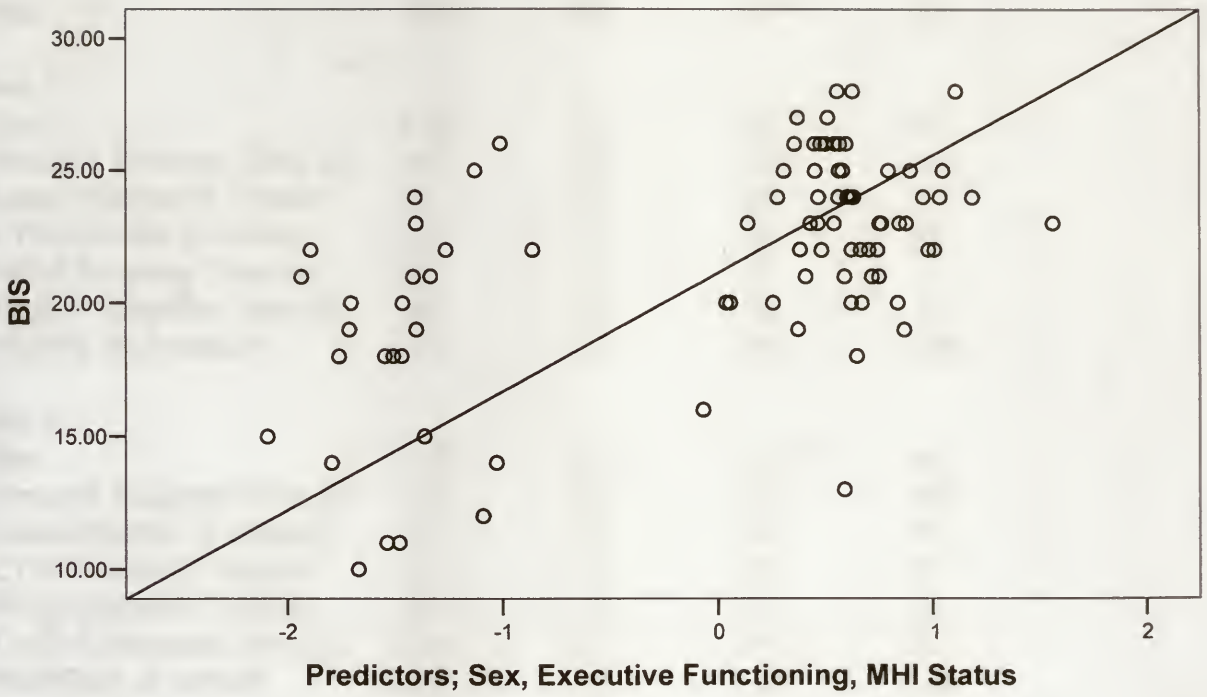
Appendix X

Summary of hierarchical multiple regression analysis in which BIS-nonplanning was regressed on sex, executive functioning, and MHI status (N = 87)

Variable	*B*	*SE B*	*β*	*sr*
Step 1				
Sex	.35	.97	.04	.04
Step 2				
Sex	.29	1.00	.03	.03
Stroop-3 Response Time (s)	.00	.06	.00	.00
Letter-Number (# correct)	-.33	.26	-.15	-.13
CTONI Score (# correct)	-.26	.15	-.19	-.18
MC-8 Response Time (s)	.10	.08	.18	.14
Trails-4 Response Time (s)	-.07	.03	-.30*	-.24
NEPSY-B (# correct)	.01	.12	.01	.01
Step 3				
Sex	.36	1.02	.04	.04
Stroop-3 Response Time (s)	.00	.06	.00	.00
Letter-Number (# correct)	-.33	.26	-.15	-.13
CTONI Score (# correct)	-.27	.16	-.19	-.18
MC-8 Response Time (s)	.10	.08	.18	.13
Trails-4 Response Time (s)	-.07	.03	-.30*	-.24
NEPSY-B (# correct)	.02	.12	.02	.01
MHI Status	.37	.89	.05	.04

Note. $R^2 = .00$ for Step 1; $\Delta R^2 = .12$ for Step 2; $\Delta R^2 = .00$ for Step 3.
*$p < .05$

Appendix Y



Overall Regression Model of BIS

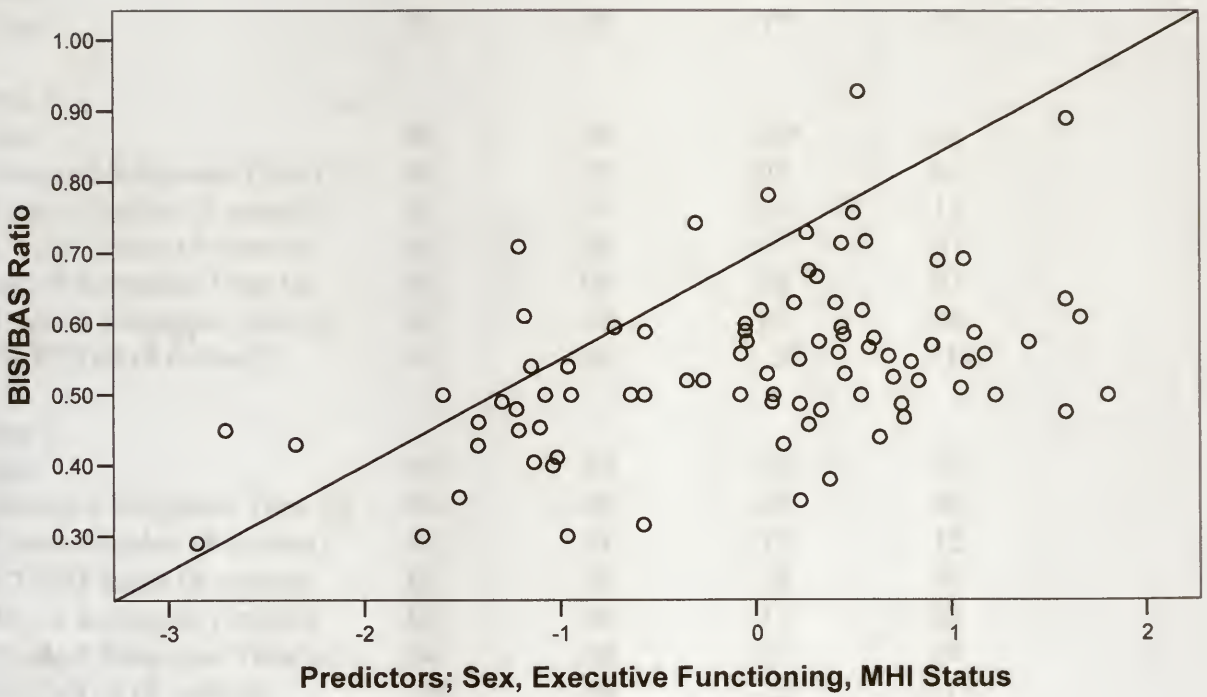
Appendix Z

Summary of hierarchical multiple regression analysis in which BIS was regressed on sex, executive functioning, and MHI status (N = 88)

Variable	*B*	*SE B*	*β*	*sr*
Step 1				
Sex	-4.63	.80	.53**	.53
Step 2				
Sex	4.43	.85	.51**	.49
Stroop-3 Response Time (s)	-.02	.05	-.03	-.03
Letter-Number (# correct)	.11	.22	.05	.04
CTONI Score (# correct)	.10	.13	.08	.08
MC-8 Response Time (s)	.08	.07	.14	.11
Trails-4 Response Time (s)	.00	.03	.00	.00
NEPSY-B (# correct)	-.08	.10	-.09	-.08
Step 3				
Sex	4.45	.87	.51**	.48
Stroop-3 Response Time (s)	-.02	.05	-.03	-.03
Letter-Number (# correct)	.11	.23	.05	.05
CTONI Score (# correct)	.10	.13	.08	.07
MC-8 Response Time (s)	.08	.07	.14	.11
Trails-4 Response Time (s)	.00	.03	.00	.00
NEPSY-B (# correct)	-.08	.10	-.09	-.08
MHI Status	.11	.76	.01	.01

Note. $R^2 = .28$ for Step 1; $\Delta R^2 = .03$ for Step 2; $\Delta R^2 = .00$ for Step 3.
*$p < .05$, **$p \leq .01$

Appendix AA



Overall Regression Model BIS/BAS Ratio

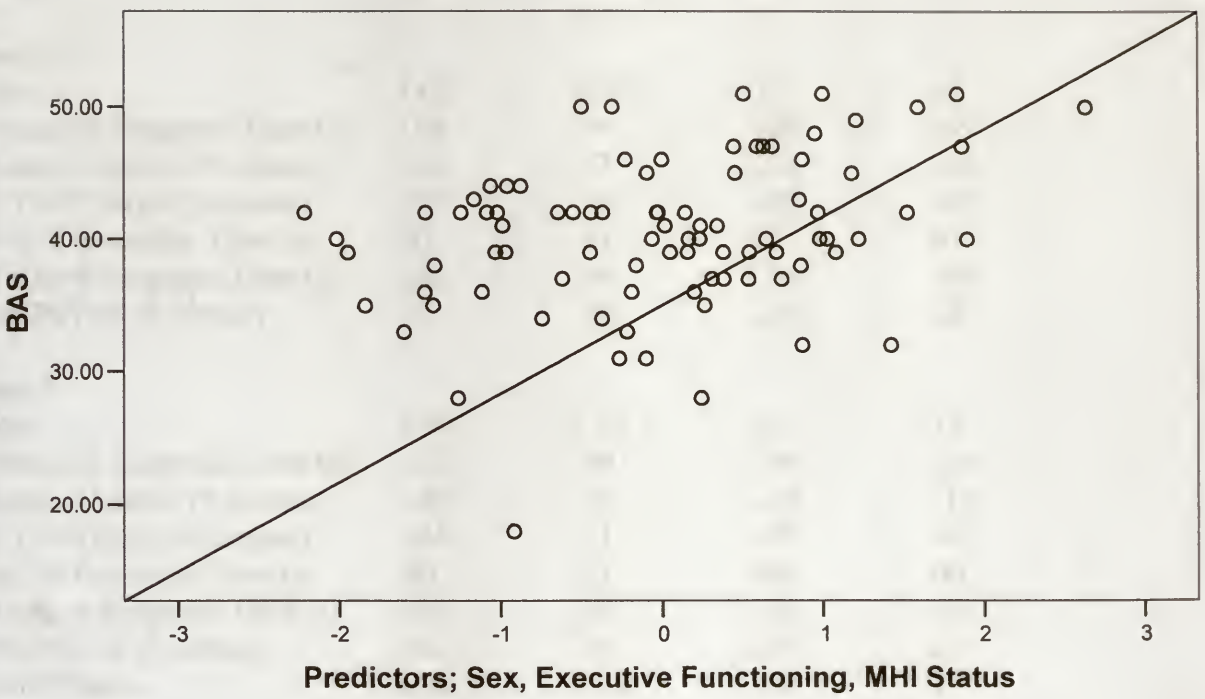
Appendix BB

Summary of hierarchical multiple regression analysis in which BIS/BAS Ratio was regressed on sex, executive functioning, and MHI status (N = 88)

Variable	*B*	*SE B*	*β*	*sr*
Step 1				
Sex	.10	.03	.37*	.37
Step 2				
Sex	.09	.03	.36*	.34
Stroop-3 Response Time (s)	.00	.00	.03	.02
Letter-Number (# correct)	.01	.01	.13	.12
CTONI Score (# correct)	.00	.00	.07	.07
MC-8 Response Time (s)	.00	.00	.10	.07
Trails-4 Response Time (s)	.00	.00	.05	.04
NEPSY-B (# correct)	-.01	.00	-.20	-.19
Step 3				
Sex	.09	.03	.34*	.32
Stroop-3 Response Time (s)	.00	.00	.02	.02
Letter-Number (# correct)	.01	.01	.13	.12
CTONI Score (# correct)	.00	.00	.08	.07
MC-8 Response Time (s)	.00	.00	.11	.08
Trails-4 Response Time (s)	.00	.00	.05	.04
NEPSY-B (# correct)	-.01	.00	-.21	-.19
MHI Status	-.02	.02	-.06	-.06

Note. $R^2 = .14$ for Step 1; $\Delta R^2 = .07$ for Step 2; $\Delta R^2 = .00$ for Step 3.
*$p < .01$

Appendix CC



Overall Regression Model of BAS

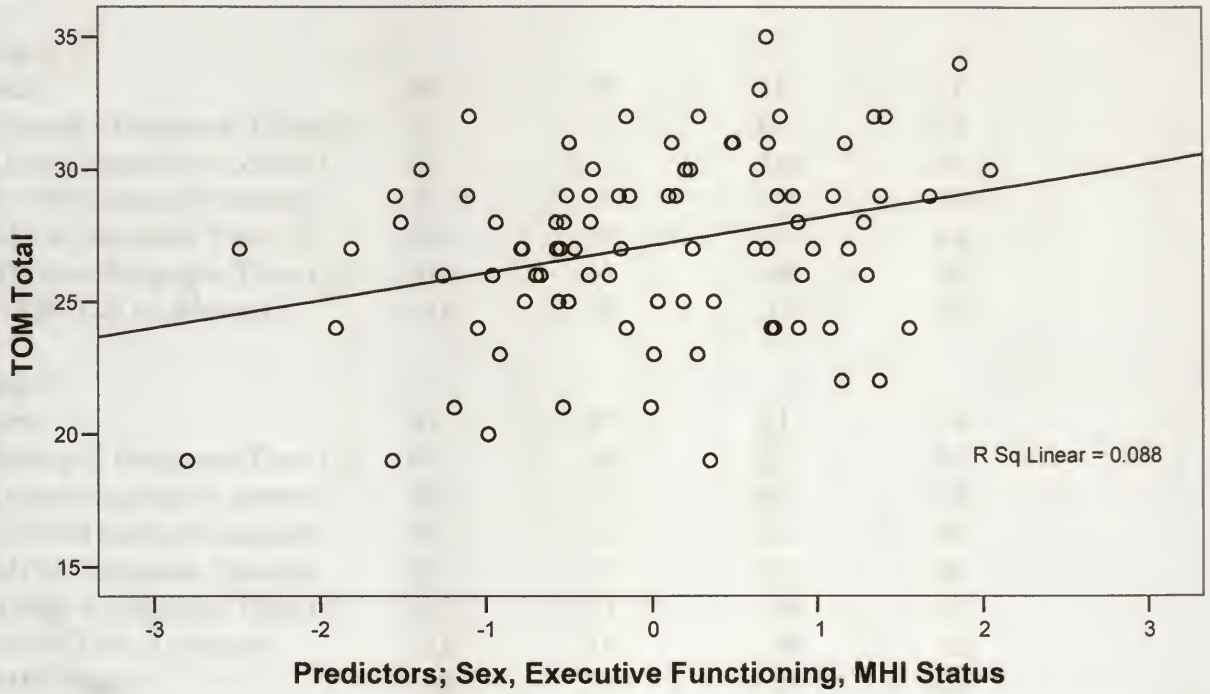
Appendix DD

Summary of hierarchical multiple regression analysis in which BAS was regressed on sex, executive functioning, and MHI status (N = 88)

Variable	*B*	*SE B*	*β*	*sr*
Step 1				
Sex	1.68	1.34	.13	.13
Step 2				
Sex	1.62	1.40	.13	.12
Stroop-3 Response Time (s)	-.04	.09	-.06	-.05
Letter-Number (# correct)	-.41	.37	-.13	-.12
CTONI Score (# correct)	-.03	.21	-.02	-.02
MC-8 Response Time (s)	.01	.11	.02	.01
Trails-4 Response Time (s)	-.03	.04	-.10	-.08
NEPSY-B (# correct)	.33	.16	.24*	.22
Step 3				
Sex	1.89	1.42	.15	.14
Stroop-3 Response Time (s)	-.03	.09	-.04	-.04
Letter-Number (# correct)	-.40	.37	-.13	-.11
CTONI Score (# correct)	-.04	.21	-.02	-.02
MC-8 Response Time (s)	.00	.11	.00	.00
Trails-4 Response Time (s)	-.03	.04	-.10	-.08
NEPSY-B (# correct)	.34	.16	.24*	.23
MHI Status	1.38	1.25	.12	.12

Note. $R^2 = .02$ for Step 1; $\Delta R^2 = .08$ for Step 2; $\Delta R^2 = .01$ for Step 3.
*$p < .05$, **$p \leq .01$

Appendix EE



Overall Regression Model for ToM Total

Appendix FF

Summary of hierarchical multiple regression analysis in which ToM was regressed on sex, executive functioning, and MHI status (N = 88)

Variable	*B*	*SE B*	*β*	*sr*
Step 1				
Sex	1.32	.81	.17	.17
Step 2				
Sex	.90	.85	.12	.11
Stroop-3 Response Time (s)	.02	.05	.04	.03
Letter-Number (# correct)	.26	.23	.14	.13
CTONI Score (# correct)	.22	.13	.20	.19
MC-8 Response Time (s)	.05	.07	.10	.07
Trails-4 Response Time (s)	-.01	.03	-.06	-.05
NEPSY-B (# correct)	-.03	.10	-.03	-.03
Step 3				
Sex	.81	.87	.11	.10
Stroop-3 Response Time (s)	.01	.05	.03	.03
Letter-Number (# correct)	.26	.23	.14	.12
CTONI Score (# correct)	.23	.13	.20	.19
MC-8 Response Time (s)	.05	.07	.10	.08
Trails-4 Response Time (s)	-.01	.03	-.06	-.05
NEPSY-B (# correct)	-.03	.10	-.04	-.03
MHI Status	-.46	.77	-.07	-.07

Note. $R^2 = .03$ for Step 1; $\Delta R^2 = .05$ for Step 2; $\Delta R^2 = .00$ for Step 3.

Appendix GG – Ethics Clearance Form

The Brock University Research Ethics Board has reviewed the above research proposal.

DECISION: Accepted as clarified.

This project has received ethics clearance for the period of February 9, 2007 to August 30, 2008 subject to full REB ratification at the Research Ethics Board's next scheduled meeting. The clearance period may be extended upon request. *The study may now proceed.*

Please note that the Research Ethics Board (REB) requires that you adhere to the protocol as last reviewed and cleared by the REB. During the course of research no deviations from, or changes to, the protocol, recruitment, or consent form may be initiated without prior written clearance from the REB. The Board must provide clearance for any modifications before they can be implemented. If you wish to modify your research project, please refer to http://www.brocku.ca/researchservices/forms to complete the appropriate form Revision or Modification to an Ongoing Application.

Adverse or unexpected events must be reported to the REB as soon as possible with an indication of how these events affect, in the view of the Principal Investigator, the safety of the participants and the continuation of the protocol.

If research participants are in the care of a health facility, at a school, or other institution or community organization, it is the responsibility of the Principal Investigator to ensure that the ethical guidelines and clearance of those facilities or institutions are obtained and filed with the REB prior to the initiation of any research protocols.

The Tri-Council Policy Statement requires that ongoing research be monitored. A Final Report is required for all projects upon completion of the project. Researchers with projects lasting more than one year are required to submit a Continuing Review Report annually. The Office of Research Services will contact you when this form *Continuing Review/Final Report* is required.

Please quote your REB file number on all future correspondence.

Brenda Brewster, Research Ethics Assistant
Office of Research Ethics, MC D250A
Brock University
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email: reb@brocku.ca
http://www.brocku.ca/researchservices/ethics/humanethics