Social competence following mild head injury and moderate traumatic brain injury: Investigating the neuropsychological relationships between arousal, social decision-making and depression

By

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Abstract

Client-directed long-term rehabilitative goals and life satisfaction following head injury emphasize the importance of social inclusion, rather than cognitive or physical, outcomes. However, very little research has explored the socio-emotional factors that pose as barriers to social reintegration following injury. This study investigates social barriers following head injury (i.e., decision-making - Iowa Gambling Task [IGT] and mood – depression) and possible amelioration of those challenges (through treatment) in both highly functioning university students with and without mild head injury (MHI) and in individuals with moderate traumatic brain injury (TBI). An arousal manipulation using emotionally evocative stimuli was introduced to manipulate the subject's physiological arousal state. Seventy-five university students (37.6% reporting a MHI) and 11 patients with documented moderate TBI were recruited to participate in this quasi-experimental study. Those with head injury were found to be physiologically underaroused (on measures of electrodermal activation [EDA] and pulse) and were less sensitive to the negative effects of punishment (i.e., losses) in the gambling task than those without head injury, with greater impairment being observed for the moderate TBI group. The arousal manipulation, while effective, was not able to maintain a higher state of arousal in the injury groups across trials (i.e., their arousal state returned to pre-manipulation levels more quickly than their non-injured cohort), and, subsequently, a performance improvement was not observed on the IGT. Lastly, head injury was found to contribute to the relationship between IGT performance and depressive symptom acknowledgment and mood status in persons with head injury. This study indicates the possible important role of physiological arousal on socio-emotional behaviours (decision-making, mood) in persons with even mild, non-complicated head injuries and across the injury severity continuum.

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Table of Contents

List of Tables

List of Figures

List of Appendices

Background Literature

Approximately 57 million people worldwide have been hospitalized with one or more traumatic brain injuries (TBI; Langlois, Rutland-Brown, & Thomas, 2004), and within Canada between 2009 and 2010, 98,440 individuals or 2.4% of the population sustained a head injury, the majority of whom are adults (Statistics Canada, 2011). Moreover, it is estimated that TBI accounts for approximately nine percent of all trauma-related admissions into hospitals (Canadian Institute for Health Information, 2006) and much of the long-term consequences associated with TBI have considerable social and economical burden, placing considerable strain on both the medical and social resources (Thurman, 2001; Thurman et al., 1999). Injuries on the mild end of the TBI continuum (i.e., mild head injury [MHI]) account for approximately 80% to 90% of all head injuries (Iverson & Lange, 2009; Ruff, 2011; Schoenberg, 2011) and make up approximately 44% of the total economic costs associated with TBI in general (Thurman, 2001).

While much of the literature and applied clinical work has focused on the cognitive and physical sequelae following TBI (Schoenberg, 2011), comparatively less attention has been allocated to socio-emotional symptomatology that pose as a barrier to socially reintegrating into schools, work-places and communities. Moreover, client-directed long-term rehabilitative goals and life satisfaction following TBI concern primarily social, rather than cognitive or physical, goals (whereas cognitive and physical goals are the primary focus of clients, and rehabilitation professionals, immediately following the injury; Burleigh, Farber, & Grillard, 1998; Corrigan, Bogner, Mysiw, Clinchot, & Fugate, 2001; LoBello, Underhill, Valentine, Stroud, Bartolucci, & Fine, 2003). As a result, the variables that underlie the major barriers to reintegration following MHI and TBI are important considerations from a social, interpersonal, rehabilitative, and brain and behaviour perspective.

While many barriers to social reintegration have been identified (i.e., impairments in emotional processing, adherence to social standards, etc.; Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Ietswaart et al., 2007), two major influences on social re-integration include impaired decision-making as well as depressive symptomatology following TBI. As a result, this thesis investigates these two barriers following mild head injury and compares their sequelae to individuals who have sustained moderate TBIs. Lastly, this thesis investigates an arousal-based emotional manipulation (i.e., presentation of high arousal stimuli from the International Affective Picture System [IAPS]) to determine whether raising physiological arousal may serve as a therapeutic target.

Chapter I: General Overview of Traumatic Brain Injury

Mild Head Injury

Despite its considerable prevalence, the definition, diagnostic indictors, and respective neurocognitive and neuropsychiatric impairments following injury, as well as the chronicity and longevity of these impairments following MHI are hotly debated within the literature. Unfortunately, there is a pervasive ideology within the medical and legal communities that those who experience a MHI do not suffer persistent, but proportionally less, impairment even one year post injury (Buck, 2011; Dikman, McLean, & Temkin, 1986; Hartvigsen, Boyle, Cassidy, & Carrol, 2014; Kristman et al., 2014). While there is significant evidence that majority of individuals will recover from a MHI (i.e., approximately 85% - 90%; Iverson, Zasler, & Lange, 2007), there is a considerable number of individuals reporting long-term post-concussive symptomatology even years after their injury (Bigler, 2008; King & Kirwilliam, 2013; Ponson, Cameron, Fitzgerald, Grant, & Mikocka-Walus, 2011). Unfortunately, as a result, these

individuals frequently suffer from residual symptomatology that is left underaddressed as they are unable to gain access to appropriate medical and rehabilitative resources.

Definition of MHI

For the purposes of this study, the definition of MHI has been adapted from Kay et al. (1993) and is synonymous with the term concussion. It is characterized by a closed injury to the head caused by any biomechanical forces that results in sufficient neuronal disruption to precipitate any of the following symptomatology: an alteration to mental status, a loss of consciousness (must less than 30 minutes in duration), post-traumatic amnesia (must less than 24 hours in duration), and/or focal neurological deficits.

The pathophysiology and biochemistry of MHI

The mechanism of injury behind MHI is not unlike that of more severe TBIs (Matteer & D'Arcy, 2000), where linear and/or rotational biomechanical forces act in a acceleration-deceleration fashion to cause the brain to impact with bones that make up the skull (coupe). When these forces are sufficient, they also cause the brain to rebound in the opposite direction, causing the brain to impact bones that make up the other side of the skull (counter-coupe; Barth, Freeman, Broshek & Varney, 2001; Iverson & Lange, 2009; Lui, 1999). This initial impact can cause contusions (i.e., bruising) and edema, and can be accompanied by, albeit uncommonly, hemorrhages and hematomas (Iverson & Lange, 2009; Ono et al., 2007). The most common areas of the brain implicated in these injuries includes: the posterior portions of the occipital lobes, the inferior cerebellum, parietal opercular area, anterior portions of the temporal lobe, posterior temporal gyrus, and the orbitofrontal cortex/ventromedial prefrontal cortex (Iverson & Lange, 2009). While microstructural damage has been related to disrupted neuronal functioning, in MHI, identifiable structural changes are relatively uncommon (using convention imaging

technologies; Alexander, 1995; Bigler & Orrison, 2001). However, more subtle impacts such as axonal shearing (Alves, Macciocchi, Barth, 1993; Gennareli, 1996; Iverson & Lange, 2009), neurochemical (Hayes & Dixon, 1994) and metabolic changes have been implicated in the disrupting of cellular function within the brain following MHI (Giza & Hovda, 2001; Giza & Hovda, 2004; Lifshitz, Sullivan, Hovda, Weiloch, & McIntosh, 2004).

Giza and Hovda (2001; 2004) illustrated in animal models that following MHI, axonal shearing, stretching and twisting begins to occur and the resulting tension placed on these cells causes membranes to become disrupted and breakdown. This subsequently results in widespread non-specific depolarization through the efflux of potassium (K^+) into the axon and the indiscriminate and uncontrolled release of predominately excitatory neurotransmitters (i.e., glutamate and acetylcholine) at the synapse. This release of these neurotransmitters, particularly that of glutamate, further magnifies the efflux of K^+ efflux by causing widespread depolarization by activation of d-amino-3-hydroxy-5-methyl-4-isoxazole-propionic acid (AMPA) and N-methyl-D-aspartate (NMDA) receptors. With the cell reaching high states of depolarization, the sodium-potassium pump (Na^+K^+ ATPase) is up-regulated to meet these increased demands to repolarize the cell, resulting increased energy requirements (i.e., adenosine triphosphate [ATP]; Giza & Hovda, 2001; Giza & Hovda, 2004; Hayes & Dixon, 1994; Lifshitz et al., 2004).

Unfortunately, at the same time, reduced vascular capacity following the injury results in decreased cerebral blood flow, reducing the availability of oxygen and subsequently metabolism shifts from oxidative phosphorylation to being centralized and solely dependent on glycolysis. With glycolysis being the predominate source of ATP at this time, lactic acid beings to accumulate with little capacity to be placed into the electron transport chain and undergo oxidative phosphorylation. This build up of lactic acid subsequently further disrupts the

mitochondria's capacity to produce energy. The mitochondria, which is solely responsible for creating the proton gradient that drives the phosphorylation of adenosine diphosphate to ATP is placed under increased oxidative stress, due to disrupted antioxidant properties which results in increased generation of reactivity oxygen species (i.e., free radicals; Lifshitz et al., 2004). The increased production of free radicals have unpaired electrons which react with the cell membrane of the mitochondria, altering its structure and reducing its functional capacity to produce ATP. This is further compounded and exacerbated by the increased activation of glutaminergic NMDA receptors that results in a build up of calcium that is toxic to mitochondria (Giza & Hovda, 2001; Giza & Hovda, 2004; Hayes & Dixon, 1994; Lifshitz et al., 2004).

Together these altered pathways result in a decreased capacity for energy production during a time when surviving neurons actually require more energy to meet the demands of over-excitation and repolarization. The result of this energy crisis in neuronal communication disruptions (typically lasting days or weeks) and necrosis (Giza & Hovda, 2001; Giza & Hovda, 2004) which have been highly implicated in the pathophysiology of diffuse axonal injury (DAI; Kushner, 1998). DAI is of particular importance for MHI (already well accepted in the moderate and severe TBI literature), as it has been a consistent finding in the advanced neuroimaging literature (Kirov, Tal, Babb, Lui, Grossman, & Gonen, 2013; Messe et al., 2011; and has been linked to the resulting neuropsychological and cognitive sequelae that follow MHI (Scheid, Walther, Guthke, Preul & von Cramon, 2006). This provides considerable support for the notion that MHI are not trivial events and require careful clinical assessment and follow-up.

Diagnostic Indicators and Classifiers of Head Injury

Due to the heterogeneity of clinical characteristics and varying pathophysiological mechanisms implicated in MHI, there has been little consensus within the empirical literature

regarding which diagnostic indicators or series of indicators are most optimal in measuring injury severity and predicting long-term rehabilitative outcomes (Gomez, Lobato, Ortega, & De La Cruz, 1996). As a result, diagnostic indicators utilized within the medical community vary depending on the type of the institute providing the assessment (i.e., family doctors offices and urgent care clinics compared to the emergency department, etc.). The most commonly utilized tools to classify the severity of an individual's TBI include (1) Glasgow Coma Scale, (2) duration of loss of consciousness, and (3) duration of post-traumatic amnesia (Iverson & Lange, 2009). Less commonly used, neuropsychological testing and neuroimaging have also illustrated predictive validity for rehabilitative outcome (Iverson et al., 2007; Shenton et al., 2012).

Glasgow Coma Scale.

Firstly, the Glasgow Coma Scale (GSC) is a behavioural instrument that grossly measures an individual's level of consciousness (Teasdale & Jennett, 1974) on three independent domains of arousal, including eyes opening as a response to stimuli, verbal, and motor functions, all of which are rated on a scale of one to four, five and six respectively (Teasdale & Jennett, 1974). The minimal score is three and a maximum score of 15. Mild injuries are typically classified when GCS follows within scores of 13 to 15 measured approximately 30 minutes post-injury; however, many individuals who sustain a MHI meet the criteria for a maximum score of 15 (Gomez, et al., 1996; Thompson & Irby, 2003), not distinguishing them from uninjured controls. Moreover, retrospective examinations of MHI patient samples have illustrated considerable heterogeneity in neurological findings in GCSs of 13, 14 and 15. For example, Gomez et al. (1996) found in MHI sample of 2000 participants, out of the individuals with GCS of 15 upon initial examination, 26% had reported neurological symptoms (i.e., loss of consciousness and post-traumatic amnesia, etc.). Moreover, 41% of individuals with a GCS of 13

had abnormal computed tomography (CT) findings (i.e., epidural hematoma, subdural hematoma, brain contusions, subarachnoid hemorrhage, etc.; Gomez et al., 1996). Moreover, GCS has not been found to be predictive of neuropsychiatric or occupational outcome (i.e., psychological distress, psychiatric symptomatology, duration before returning to work, etc.; McCullagh, Ouchterlony, Protzner, Blair, & Feinstein, 2001) and even its predictive capacity of gross indices of 'recovery' as measured by the Glasgow Outcome Scale decrease with time since injury. Together, these findings suggest that while worsening symptomatology is associated with lower GCS scores, the sensitivity of this measure is low resulting in substantial variability between symptom presentation and acute and post-acute functional outcomes associated with each of the mild GCS ratings (Gomez, et al., 1996; McCullagh et al., 2001; Thompson & Irby, 2003).

Loss of Consciousness

The duration of post-injury loss of consciousness (LOC) is another commonly used indicator of severity (Iverson & Lange, 2009; Kay et al., 1993). From a behavioural standpoint, a patient is label as being unconscious if they appear to be in a sleep-like state and cannot be immediately aroused by command or physical contact (often referred to duration till responsive to commands; Whyte, Cifu, Dikmen, & Tempkin, 2001). In addition to this, during this time patients remain unaware of oneself and their respective surroundings (Iverson & Lange, 2009; Thompson & Irby, 2003). Within in the past, there has been debate as to whether a LOC of is required within the definition of MHI (American Psychiatric Association, 2000; Feinstein & Rapoport, 2000); however, considerable evidence has been collected to suggest that neuronal functioning can be disrupted even with injuries not involving a LOC but still result in post-injury functional impairment (i.e., cognitive, emotional, social and behavioural sequelae; Iverson,

Lovell, Smith, & Franzen, 2000; Kay et al., 1993, Kay & Teasdale, 2001). LOC is a positive predictor of intracranial abnormalities identified by CT scans with increasing odds-ratios associated with greater LOC, particularly in moderate and severe injuries. Individuals without an LOC were found to have a quarter of the risk of intracranial pathologies relative to their non-LOC cohort (Iverson et al., 2000; Ono, Wada, Takahara, & Shirotani, 2007). Smits et al., (2007) has suggested, however, that intracranial pathologies may be underrepresented within the literature with respect to individuals sustaining MHI and having no LOC, finding that in their sample of over two thousand patients almost equal portions of intracranial pathology regardless of LOC status. These findings illustrate the importance of including injuries that are without LOC in the mild end of the spectrum of injuries. As a result, for head injuries to be considered 'mild,' one must have an LOC shorter in duration than 30 minutes (Kay et al., 1993; Thompson & Irby, 2003), whereas 'moderate' and 'severe' classifications are typically considered when LOC is greater than 30 minutes and 24 hours respectively (Thompson & Irby, 2003).

Post-Traumatic Amnesia

Post-traumatic Amnesia (PTA) is the duration of memory disruption immediately following the traumatic incident to the time memories can be clear and consecutively recalled (Iverson & Lange, 2009). While this measure is not as straightforward as it might seem, given that 'islands' of memory which can be readily confused with continuous stream of memory may make clinical assessment difficult, it has been found to be a strong predictor of functional outcome one-year post-injury (van der Naalt, van Zomeren, & Minderhoud, 1999). With respect to injury severity, individuals with a PTA duration of less than 24 hours are typically classified as having a MHI, whereas PTA durations of one to seven days, and eight days or more, are

considered moderate and severe injuries, respectively (Levin, Benton, & Grossman, 1982; Iverson & Lange, 2009; Thompson & Irby, 2003).

Neuroimaging

Traditional neuroimaging techniques readily available to medical personnel have not shown the sensitivity and specificity required to measure the respective pathophysiological changes associated with TBIs on the mild end of the spectrum (Bigler, 1999; Bigler & Orrison, 2001). Computerized tomography (CT) is one of the most commonly used neuroimaging methodologies given their relative speed, low costs and patients not requiring medical stabilization. While CT imaging has been illustrated particularly effective in delineating bone abnormalities (i.e., skull fractures, etc.), hemorrhagic blood (subdural hemorrhage, subarachnoid hemorrhage, etc.), and edema (Bigler & Orrison, 2001) and subsequently identifying individuals who may require neurosurgical intervention following their head injury (Haydel, et al., 2000; Smits et al., 2007; Stiell et al., 2005), they have, however, shown minimal capacity to differentiate individuals who have sustained a MHI or provide predictive capacity for functional outcomes (Bigler & Orrison, 2001; Iverson et al., 2000; Livingston et al., 2000). Many studies have illustrated the limitations in sensitivity that CT imaging possesses, whereby in one study 16% of 912 patients who had sustained a MHI were found to have abnormalities on CT (Iverson et al., 2000), only 5% of over one thousand patients with a MHI had intracranial lesions visible (Livingston et al., 2000; Ono et al., 2007), and CT findings are minimally associated of long-term outcome (Jacobs et al., 2010).

Magnetic resonance imaging (MRI; T1 and T2-weighted imaging), unlike CT methods, have shown considerably greater anatomical resolution with the capacity to pick up subtle lesions presentations (Bigler & Orrison, 2001), has an increased sensitivity to lesions typically

seen in mild and moderate patients that are hospitalized; however, these lesions correlate poorly with neuropsychological profiles (Bigler & Orrison, 2001; Levin, Williams, Eisenber, High, & Guinto, 1992). Use of structural imaging techniques in MHI populations has been further complicated by the presence of neuropsychological dysfunction in the absence of abnormal structural findings (Leninger al., 1990; Voller et al., 1999; Yarnell & Rossie, 1988). Yarnell and Rossie (1988) found that individuals suffering from minor whiplash head trauma were impaired in cognitive flexibility, non-verbal reasoning, learning and memory, psychomotor agility and attention compared to their non-injured counterparts, despite unremarkable findings on the neurological examination, neuroimaging and electrophysiological studies. Estimates vary, but between 43% to 68% of individuals with MHI have unremarkable MRI findings (Belanger, Vanderpleog, Curtiss, & Warden, 2007; Hughes, Jackson, Mason, Berry, Hollis, & Yates, 2004). The pathophysiology of MHI, whereby alterations in synaptic, biochemical and metabolic processes occur (Alexander, 1995; Giza & Hovda, 2004; Ogden, 2005), may be an underlying contributor to the difficulty in identifying structural abnormalities through CT and MRI (Bigler & Orrison, 2001). Unfortunately this has been one of the major barriers in having medical personnel and third party insurance companies recognize the sequelae following mild injuries (Bigler, 1999; Bigler & Orrison, 2001).

Susceptibility weighted MR techniques utilize clinical MRI scanning units available conventionally to develop sharper and clearer images pertaining to hemorrhages. These techniques utilize the paramagnetic properties of deoxygemoglobin and methemoglobin to outline micro-bleeding that occurs within tissue at higher rates of sensitivity compared to regular MRI (Shenton et al., 2012). Within the brain injury literature, susceptibility weighted imaging has been a new avenue for examining diffuse axonal injury which has been elusive utilizing

more conventional neuroimaging techniques (Scheid, Ott, Roth, Schroeter, & Cramon, 2007; Shenton et al., 2012). Babikian et al. (2007) found that images collected within 10 days postinjury illustrated abnormalities indicative of diffuse axonal injury and volumetric indices of lesions were predictive of 32% of the variance in neuropsychological performance 1-4 years following the injury. This illustrates promising data that begins to link imaging findings to functional measures of cognition.

Diffusion tensor imaging (DTI) is a more novel form of structural MR technology which measures diffusion of water molecules to calculate diffusion in three dimensional space (i.e., tensor). In white myelinated fiber tracts, myelin surrounding axons is found to have lower solubility than the axon, this provides indices of diffusion direction and subsequently of structural integrity. Typically this measure is quantified in fractional anisotropy (FA; ranging from 0 to 1), an index of the amount of diffusion that occurs in a single direction within each three dimensional voxel (Belanger et al., 2007). Deviated FAs outside the norm have been associate increased inflammation (high FA values) and neuronal/white matter degradation (low FA values) and serves as a proxy for fast and reliable neuronal communication (Bigler & Bazarian, 2010).

Despite its novelty, DTI has already illustrated utility within the literature, whereby patients with mild injuries were found to have reduced FA in their corpus callosum, internal capsule, and external capsule just 24 hours after injury. Mayer et al. (2010) illustrated that DTI was sensitive to cytotoxic edema in semi-acute mild injuries (on average 12 days postinjury). Mayor, Mannell, Ling, Gasparovic and Yeo (2011) found reduced connectivity profiles in fronto-parietal networks as well as default-mode networks for individuals with a MHI relative to matched control subjects. A quantitative review of the DTI literature performed by Kulkower,

Poliak, Rosenbaum, Zimmerman, and Lipton (2013) suggests that based on over a 100 articles, DTI is has a strong sensitivity to white matter microstructural alterations that follow TBI and despite considerable variability in clinical presentations, this advanced imaging technique has the capacity to differentiate those with TBI from controls, regardless of injury severity characteristics. Moreover, there is increasing evidence that imaging findings collected by DTI can be predictive of long-term outcomes (Kulkower et al., 2013).

Functional neuroimaging (positron emission tomography [PET] and functional MRI [fMRI]) has also shown some promising results for the identification of the neuropathology implicated in MHI that relates to impaired neurocognitive functioning (Belanger et al., 2007; Bigler & Orrison, 2001; Jantzen, 2010). PET imaging techniques measures regional cellular metabolism and thus indirectly by detecting changes to regional cerebral blood flow (rCBF). rCBT changes as a function of cellular demand for glucose and oxygen (up-regulated based on cellular needs). Several studies have illustrated that metabolic abnormalities persist chronically in some individuals with MHI. For example, Humayun et al. (1989) illustrated that despite normative MRI and CT images of three individuals with MHI, they were found to have abnormal metabolic profiles in the temporal (anterior, medial and posterior portions) and the frontal lobes (anterior and posterior portions) relative to controls. Chen, Kareken, Fastenau, Trexler and Hutchins (2003) illustrated that PET has increased sensitivity to cortical dysfunction that is not typically identified through structural imaging techniques and this has been found to correlate with neuropsychological profiles (Belanger et al., 2007; Bigler & Orrison, 2001, Chen et al., 2003).

Instead of measuring indices of metabolism through glucose, fMRI utilizes measures of blood oxygen typically in the form of blood-oxygen-level-dependent (BOLD) techniques. This

provides an indirect measure of cellular activity by measuring differential magnetic field gradients due to changing deoxyhemoglobin concentrations that result from oxygen consumption (i.e., with increasing cellular activity, oxygen consumption increases and local blood flow increase reducing the concentration of deoxyhemoglobin; Belanger et al., 2007). fMRI has been found to have spatial resolution of several millimeters and a temporal resolution of between one and two seconds (Bigler & Orrison, 2001; Jantzen, 2010). While the relationship between vascularization and BOLD signal is thought to be nonlinear and is not yet fully understood, several authors have found blood flow abnormalities that differentiate those who have had a MHI from those with no history of head injury (McAllister et al., 1999; McAllister et al., 2001; Jantzen, Anderson, Steinberg, & Kelso, 2004).

Despite a proliferation of utilization of fMRI techniques in other areas of cognitive neuroscience, utilization of fMRI in MHI populations has been relatively minimal comparatively (Jantzen, 2010; McDonald, Saykin, & McAllister, 2012). This may be due to a number of reasons, but the heterogeneity of symptomatology presenting following head injury (e.g., diversity of cognitive impairments, neuropsychiatric symptoms, etc.), sample characteristics (e.g., time since injury, severity indictors, age, etc.), genetic variability (see McDonald et al., 2012) which may contribute to considerable variability that has made looking at group differences more challenging (Jantzen, 2010; McDonald et al., 2012). Jantzen (2010) review the literature and found that of the 10 studies review examining working memory deficits in those with MHI, three illustrated increased BOLD response and five illustrated decreased BOLD response. Various explains are given attenuated BOLD amplitude, such that individuals with MHI have reduced capacity to recruit or engage neural process for the task demands and heightened BOLD response is associated with less efficient processes (Jantzen, 2010). It is clear

that more research is required to disentangle the nature of differential BOLD responses to decreased performance on cognitive measures following injury. Despite these limitations, fMRI has shown considerable promise when combined with other neuroimaging methodologies (i.e., DTI, MR spectroscopy, etc.) and appears promising in the diagnosing and monitoring of treatment progress following injury (McDonald et al., 2012). Unfortunately, due to the current costs and limited availability of these methodologies, they are presently not readily accessible for clinical use, which continues to serve as a continued barrier to the acknowledgement of impairment following MHI (Jantzen, 2010).

Post-concussive Syndrome & Neuropsychological Sequelae following MHI

Following a MHI, the most common set of symptomatology that presents is typically referred to as post-concussive syndrome (PCS), which has been characterized one or more of the following: headache, fatigue, photophobia, sensitivity to noise, double vision, tinnitus, dizziness, nausea, vomiting, irritability, aggression, alterations in mood (i.e., anxiety, depression, etc.), as well as changes in cognition (American Psychological Association, 2000; Binder, 1986; World Health Organization, 1993). PCS is highly common in the weeks following an injury but many patients report these symptoms persisting even three months postinjury (Mittenberg & Stauman, 2000). Mittenberg and Stauman (2000) suggested that approximately 30% of untreated patients with a MHI will have symptomology sufficient to meet the criteria of PCS six months postinjury.

There remains an ongoing debate within the literature about the legitimacy of PCS lasting longer than one year in duration postinjury (Mathias, Beall, & Bigler, 2004; Rohling, Binder, Demakis, Larrabee, Ploetz, & Langhinrichsen-Rohling, 2011; Wang et al., 2006; Zakzanis & Yeung, 2011). Estimates vary from 10 to 50% of individuals report long-term PCS (Alexander, 1995; Kay et al., 1992; King & Kirwilliam, 2013; McCauley et al., 2008; Ryan, & Warden,

2003), but many argue that the etiological contribute of these symptoms may have more to do with premorbid personality (e.g., neuroticism), emotional/psychiatric or other neurological factors unrelated to the head injury (Clarke, Genat, & Anderson, 2012; Ruff & Grant, 1999; Ryan & Warden, 2003). For example, Clarke et al. (2012) found that emotional/affective symptoms and personality traits such as neuroticism best predicted symptoms over and above neuropsychological test performance for individuals with MHI, spinal cord injury patients and health neurological controls. They interpret these findings to suggest that PCS represent psychological symptoms and not damage to the nervous system itself (Clarke et al., 2012). Much of this debate stems from the fact that PCS symptoms are nonspecific and all can be accounted for by other medical and psychiatric disorders (Bigler, 2008; Zakzanis & Yeung, 2011). While it is important to understand the premorbid factors that contribute to PCS, it would be facetious to state that all of these symptoms are due to premorbid states alone and even if much of this symptomatology can be accounted for "psychological" changes post-injury. Neuropsychological investigation as means to better understand the respective neural functional changes is required (Bigler, 2008). All too often clinicians and scientists appear to be quick to blame these symptoms on the individual not "wanting" to get better and not investigating the etiology, perhaps reflecting a philosophical approach difference rather than a scientific one.

From a neuropsychological standpoint, the clinical presentation of symptoms is considerably variable, interacting with many preinjury variables, and is highly complex (i.e., varying depending on when testing takes place – e.g., immediately after relative to 3 months and 6 months postinjury; Bigler, 2008; Bohnen, Jolles, Twinjnstra, 1992; Mathias, Beall, & Bigler, 2004; Rohling, Binder, Demakis, Larrabee, Ploetz, & Langhinrichsen-Rohling, 2011; Wang et al., 2006). However, impairments in processing speed, attention allocation and regulation,

memory functions and executive function (including emotional dysregulation) are the most common cognitive findings following MHI (Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005; Bigler, 2008; Lezak, Howieson, Bigler & Tranel, 2012; Lundin, De Boussard, & Borg, 2006;). Belanger and colleagues (2005) conducted a meta-analysis summarizing 39 studies involving almost 1500 cases of MHI compared to 1200 control subjects, finding that the impact to neuropsychological function on average was of a moderate effect size (d = .54) with some domains of executive functioning and delayed memory processes having larger effect sizes (i.e., d = 1.03 and .89). Symptomatology postinjury has been correlated to disability and impairment in activities in daily living (Lundin et al., 2006). This suggests that there are distinct areas of the nervous system that may be more "vulnerable" to injury than others.

Even in highly functioning University students, incidents of MHI is relatively high (25 to 45%; DeBono & Good, 2008; Segalowitz & Lawson, 1995) individuals who have a history of MHI have been found to have similar, but proportionally less severe changes in neurocognitive functioning (Baker & Good, 2013; Segalowitz et al., 2001; van Noordt & Good, 2011). Segalowitz et al. (2001) found that those with a MHI demonstrated less optimal performance on measures of a relatively easy attention task, despite normal function in standard cognitive batteries. They also gathered altered electrophysiological evidence from ERPs, which was interpreted as changes in information processing.

Chapter II: The Present Study

The purpose of the present study is to replicate findings suggestive of decision-making changes following MHI and compare this to the presentation of individuals who have sustained moderate TBI. Moreover, we will investigate and attempt to replicate an arousal-based emotional

manipulation to observe if this can attenuate sequelae observed for individuals who previously sustained a head injury. Lastly, this study will explore whether MHI is a possible confound within psychopathology literature whereby the depressive symptomatology can be accounted for by neurocognitive sequelae.

Vulnerable Brain Regions in MHI: Prefrontal Cortex

While several areas of the brain are particularly vulnerable to the biomechanical forces applied during a head injury (see cone of vulnerability; Bigler, 2008; Lezark et al., 2012), the prefrontal cortex (PFC) is an area that is implicated in governing, regulating, executing and monitoring all of the nervous systems respective activities (Struss & Benson, 1984). Typically these processes are referred to as executive function, constituting attention, inhibition, planning and sequencing, self-monitoring, memory, and emotion regulation, and initiation of tasks (behavioural and cognitive). The PFC is frequently subdivided into four gross subdivisions, dorsolateral prefrontal cortex (DLPFC), medial prefrontal cortex (mPFC), ventromedial prefrontal cortex (VMPFC)/orbitofrontal cortex (OFC), and ventrolateral prefrontal cortex (VLPFC). These are subdivided based on different sets of behavioural disorders (Lezak et al., 2012).

While the functions of these structures are detailed extensively elsewhere, a brief summary of respective functions will be described here. The DLPFC is made up of Brodmann areas 8, 9, 46 and 10 and it has been implicated in attentional processes, spatial working memory tasks, verbal memory retrieval, conditioned learning, and planning among other functions (Cabeza & Nyberg, 2000; Kolb & Whishaw, 2009). It has been argued that the DLPFC is broadly involved in “on-line” processing of information (Cabeza & Nyberg, 2000). Conversely, the mPFC, which involves the anterior cingulate cortex, has been implicated in motivation, drive

modulating emotional activity and emotional regulation (Lezak et al., 2012), self-reference (Gusnard, Akbudak, Shulman, & Raichle, 2001), and the default mode network (Raichle, 2009). Moreover, there is some evidence for its role in the modulating some neuroendocrine functions (i.e., hypothalamic-pituitary adrenal axis), having extensive projections with both the hypothalamus and other subcortical systems (Diorio, Viau, Meaney, 1993; Kolb & Whishaw, 2009). The VLPFC has been particularly implicated in cognitive control, motor inhibition, and reflexive visual orientation and spatial attention (Badre & Wagner, 2007; Levy & Wagner, 2011). Lastly, the VMPFC, which is defined by Brodman areas 25, 32, ventral portions of 24, and medial portions of 10, 11, and 12 respectively (Bechara, 2004; Bechara, Tranel, & Damasio, 2000b), has been illustrated to play a role in regulation and monitoring behaviour (i.e., particularly social behaviour, as well as integration of salient feedback into decision-making processes, among other functions (Bechara, Damasio, Damasio, & Lee, 1999; Bechara et al., 2000a; Bechara et al., 2000b; Bechara, Damasio, Tranel, & Anderson, 1998; Clark, Cools, & Robbins, 2004; Fellows & Farah, 2005; Kolb & Whishaw, 2009; Rolls, 2004; Wheeler & Fellows, 2008).

The orbitofrontal cortex (OFC) makes up the most ventral and interior areas of the VMPFC, being situated immediately posterior to the orbital bones that protect the eyes (Rolls, 2004; Wallis, 2007). This area is thought to be highly vulnerable to biomechanical injuries (such as head injury) due to its close proximity to the boney protrusions of the skull in the surrounding area (i.e., cribiform plate and sphenoid bone; Bigler & Orrison, 2001; Wallis, 2007). In addition to this, given that the frontal lobe is one of the largest structures in the brain and is furthest from support of the brain stem, it is most highly sensitive to rotational forces. This, paired with the coup-contre-coup/acceleration-deacceleration forces that often cause portions of the OFC to

contact the skull bones, makes this area of the brain one of the most highly susceptible areas in TBI-related neural disruption (Alves et al., 1993; Bigler, 2001; Wallis, 2007). Given its implicated role in social sequelae following neural injury and its high vulnerability to injury, the OFC serves as a good candidate for better understanding symptomatology postinjury.

The Orbitofrontal Cortex

Individuals with lesions to the OFC often have a pleura of social difficulties, including impairments in decision-making (Bechara, 2004; Bechara et al., 2000b), inability to follow social and ethical conventions, antisocial behaviour (Blair, 2004), disinhibition (Berlin, Rolls, & Kischka, 2004; Damasio, 1994), impaired identification and appraisal of emotional stimuli (Hornak, Rolls, & Wade, 1996), and alterations in reward and punishment processing (Berlin et al., 2004; Bechara et al., 1994). Converging evidence suggests that the OFC is implicated in the development and production of visceral-based cues (i.e., states of physiological arousal), which can guide decision-making in times of uncertainty in the form of "gut-feelings" (Bechara et al., 2000). Literature from both human and animal neuroanatomy suggests that the OFC is part of a highly complex integrative network and has the necessary projections estimated to summate neural inputs that guide decision-making processes. The OFC receives dense projections from all sensory modalities, including the olfactory cortex, tertiary auditory information from the auditory cortex, somatosensory information from secondary somatosensory and parietal cortices respectively, and lastly processed visual information from the inferior temporal cortex (Carmichael & Price, 1995a; Rolls, 2004; Wallis, 2007). The OFC has bidirectional projections with areas from the limbic system and other associated areas, including (1) the amygdala, implicated among other areas in high intensity emotion (Barbas, Saha, Rempel-Clower, & Ghashghaei, 2003; Carmichael & Price, 1995b; Kolb & Whishaw, 2009); (2) the hypothalamus,

implicated, with the amygdala, in the modulation of modulate visceral and autonomic arousal by influencing brain stem structures, such as the periaqueductal gray area, reticular formation and raphe nucleus (Barbas et al., 2003; Carmichael & Price, 1995b; Kadda, Pribram, & Epstein, 1949); (3) the cingulate cortex, associated with modulation of attention, emotional and memory functions, and modulating behavioural responsiveness, among many functions (Crottaz-Herbette & Menon, 2006; Kalat, 2010; Kolb & Whishaw, 2009; Lezark et al., 2012); (4) the hippocampus, implicated in the consolidation of memory (Wallis, 2007); and (5) the nucleus accumbens, involved in motivational reward processing (Cardinal, Pennicott, Sugathapals, Robbins, & Everitt, 2001).

Damage to areas of the OFC disrupts these connections, and greatly impacts the modulation of visceral-arousal based on the accumulation of the various structural inputs, thereby compromising one's ability to be responsible in an optimal fashion to complex environmental stimuli (Barbas et al., 2003; Bechara et al., 2000a; Bechara, Tranel, Damasio, & Damasio, 1996). Evidence from both the neuroanatomy and neurophysiology literatures supports the notion that the OFC is highly implicated in the modulation of emotional and visceral arousal based on motivational and environmental inputs, thus providing an influence on behaviours and decision-making in the form of "gut-feelings" or "somatic markers"(Bechara et al., 1996; 2000a; 2000b; Damasio, 1994).

The 'Somatic Marker' hypothesis developed by Damasio, Bechara and colleagues, postulates that decision-making processes, particularly in times of uncertainty or ambiguity, are informed, and otherwise shaped, by bioregulatory affective or emotional states that are indexed by the beholder as changes in visceral states (i.e., galvanic skin response/electrodermal activation, blood pressure, respiration, gut motility, etc.). These states serve to provide contextual

information that facilitates more optimal learning and decision-making outcomes (Bechara et al., 1996; Damasio, 1994; Damasio, 1996). Optimal decision-making is hypothesized to be dependent on effective and adaptive modulation and regulation of these bioregulatory affective states. There is extensive literature on the Somatic Marker's hypothesis and is one of the major neurological theories of decision-making. A complete framework is provided by the most recent review provided by Reinmann and Bechara (2010).

Related to the Somatic Marker hypothesis, research conducted in our lab has repeatedly found that participants reporting a MHI (as mild as sustaining a hit to the head sufficient to alter one's state of consciousness) are physiologically underaroused compared to their non-injured cohort (Baker & Good, 2010; van Noordt & Good, 2011), in a manner similar to that found in persons with severe TBI. Expanding on this finding, we have found that decision-making processes during conditions of uncertainty, are similarly impacted and diminished (e.g., Robb & Good, 2012) using Damasio and colleague's Iowa Gambling Task (IGT), a task that is widely accepted as a measure of OFC functioning (Bechara et al., 1994), and are accompanied by impoverished measures of autonomic nervous system (ANS) arousal (i.e., electrodermal activation [EDA]). The IGT requires participants to make 100 individual selections from one of four unmarked decks of cards (referred to as Decks A through D). The task is designed to mimic real-life 'gambling' decision-making in a laboratory, whereby choices are made in the context of uncertainty since the consequences of decisions cannot be predicted. In this case, the uncertainty mimics a gambling situation such that the outcomes vary in both the frequency/schedule and magnitude of reward (point gains) and punishment (point losses). Since there is no explicit algorithm available to the subjects and, at least initially, subjects must 'guess', and otherwise (eventually) 'intuit' which selection will be more or less advantageous, the situation forces

participants to rely more so on their "gut feelings" for their selections (Bechara et al., 1994).

Typically, individuals without any OFC compromise (either normal controls or lesions elsewhere) initially select more from the "high risk" decks, due to their initial high appeal (i.e., greater initial gains, but similarly greater losses), but quickly shift to "low risk" decks (i.e., more meager gains, but similarly, less pronounced losses) upon learning of the more substantive long-term costs of the "riskier" decks. Conversely, patients with substantial OFC lesions illustrate a pattern of decision-making on this task that reflects a preference for the disadvantageous decks that continues throughout the 100 trials despite periodic sampling from all of the decks (Bechara et al., 1994; Bechara et al., 2000a, Bechara et al., 2000b), and having opportunity to experience the relative outcomes of high or low risks. Moreover, a pivotal study conducted by Bechara et al. (1996) illustrated that individuals with OFC lesions have reduced ANS arousal in anticipation of making a decision-selection, and despite experiencing significant losses, compared to non-injured controls, despite fully intact, and heightened, ANS arousal in response to feedback following a decision.

Similar to the research conducted with OFC lesion patients, we (Robb & Good, 2013) found that individuals reporting a MHI were found to illustrate a pattern of limited, non-optimal decision-making whereby they made a slower transition from high risk deck selections to low risk deck selections, ultimately leading to less overall gains. As well, MHI subjects were found to be less aversive to high-risk decks, as they would more quickly return to high risk deck selections following punishment compared to their non-injured cohort. Moreover, MHI participants were physiologically underaroused in anticipation of making a card selection compared to their non-MHI cohort. In contrast, non-MHI participants developed increasingly noticeable anticipatory arousal states across time. These findings may reflect a neurally-based

affective in-sensitivity to consequences of decision outcomes (i.e., prior losses) resulting in alterations in behaviour and decision-making as a function of OFC competence (i.e., less avoidance of disadvantageous/risky decisions following MHI).

Replication - Hypothesis I: Underarousal

Based on the literature presented and our previous findings, it is hypothesized that arousal will vary as a function of injury severity at initial testing, whereby individuals reporting increasing injury severity (measured by loss of consciousness, post-traumatic amnesia, post-concussive symptoms, etc.) will be found to be physiologically underaroused relative to their age-matched peers. More specifically, at initial testing, individuals who have a moderate TBI will present with the least arousal and those reporting a MHI will have lower arousal as compared to those without a history of MHI. This pattern of arousal is also expected for anticipatory arousal measures observed prior to participants making a selection on the IGT, but not in response to selection feedback. These findings would replicate our previous findings of underarousal for individuals with a history of MHI, and illustrate the continuum of injury severity with respect to physiological underarousal.

Replication - Hypothesis II: IGT Performance

IGT performance is expected to vary as a function of injury severity such that individuals who have sustained more significant injury to the head will also demonstrate the slowest learning transitions. Persons with moderate TBI will be slower at transitioning from disadvantageous selections to advantageous selections than individuals who report mild head injuries who, in turn, will be slower to transition than their non-injured healthy cohort. Moreover, the rate of return to a punishing (disadvantageous) selection will be faster (i.e., fewer trials in returning to a selection following a punishment by that same selection on a previous trial) as a function of injury severity

(i.e., individuals with moderate TBI, followed by those reporting a MHI and lastly non-injured healthy subjects). These findings would replicate our previous findings of decision-making for individuals with a history of MHI, and expand the findings to persons living with a moderate TBI, illustrating the continuum of injury severity with respect to decision-making during conditions of uncertainty.

Iowa Gambling Task, Executive Functions, Reversal Learning & Explicit Knowledge

Of the considerable research conducted using the IGT, there are three major critiques of the task's validity that are particularly pertinent to the investigation of barriers to social reintegration. These include, firstly, whether the IGT measures a unique and dissociable constructive of emotion/cognition that is separable from 'typical' executive functions, thereby providing a unique contribution of understanding barriers to social reintegration. It has already been well documented that executive functions are fundamental for independent functioning, activities of daily living, and social and community reintegration (Lezak, Howieson, Bigler & Tranel, 2012). Secondly, Fellows and Farah (2004) have illustrated that reversal learning, at least in part, is required for successful performance in the IGT. They modified the IGT to eliminate the need to overcome immediate gains from the risky decks (i.e., cards one through eight were placed at the bottom of the decks for all four decks of cards). VMPFC/OFC patients illustrated impairments on the original IGT but not in the modified task. Fellows has suggested this finding illustrates that VMPFC/OFC's impairment in IGT performance is a reversal learning problem (i.e., impaired updating of stimulus reward/punishment associative learning) and not reflect of impairments in decision-making, somatic markers, or gut feeling (Fellows, 2004; Fellows & Farah, 2004). Lastly, whether the IGT is able to measure implicit emotional learning in the form of "gut-feeling" or intuition and is not confounded by the development of explicit knowledge

prior to the development of implicit markers or behavioural changes (i.e., using implicit learning that some things lead to negative outcomes and should be avoided). If explicit knowledge occurs prior to the development of implicit markers, then IGT performance may not be tapping the construct of gut-feelings in conditions of uncertainty and may not be informative, or indicative, of decision-making in social situations.

Although no neuropsychological measure is expected to measure a single cognitive construct, the IGT has been suggested to tap into emotional implicit learning, sometimes referred to as intuition or "gut-feelings" and is suggested to do so independently of other executive functions typically associated with other regions of the PFC (Bechara et al., 1994; Bechara et al., 2000a, Bechara et al., 2000b). Bechara et al. (1998) illustrated a dissociation between VMPFC/OFC patients and right dorsolateral PFC (DLPFC) patients on measures of decision-making and working memory. VMPFC/OFC patients were found to have deficits in decision-making on the IGT and not tasks of working memory whereas DLPFC patients illustrated deficits in working memory, but not decision-making. Moreover, IGT performance was not found to change with increasing working memory load in healthy control subjects (Turnbull, Evans, Bunce, Carzolio, & O'Conner, 2005). Toplak et al. (2010) performed a systematic review of the literature examining the associations of several common executive functions hypothesized to be important for performing well on the IGT, including (1) inhibition, (2) working memory, and (3) set-shifting. Of the 43 studies examined, the majority of studies reported no statistically significant relationships between indices of executive function and IGT performance. Of the small number of studies that did find a relationship, effect sizes were reported to be small to moderate. Collectively, these findings suggest that intuition or "gut-feeling"/implicit emotional learning is minimally related to 'typical' executive functions and

reflects a distinct and separable form of cognition that provides a unique contribution to understanding the barriers to social/community reintegration.

The second criticism of the IGT, where IGT performance has been proposed to be highly dependent on the ability to reverse a learned association that initial rewarding of risky decks (A and B) and does not lead to the most advantageous outcomes, and thus is not indicative of decision-making per se, but simply a learning task requires careful examination. Bechara and colleagues (2005) have responded to this critique arguing that reversal learning within the IGT is likely to be accounted for by somatic markers theory, whereby gut feeling/somatic states serve as the underlying mechanism by which these learned associations can change (i.e., stop signal). In support of this, it is important to distinguish reversal learning from cognitive flexibility or set shifting. The latter reflects the ability to move back and forward between several mental sets or approaches in response to changing goals (Lezak et al., 2012). It is clear that while the IGT may evaluate this skill type to some degree, VMPFC/OFC patients are able to shift away from risky decks for some of their trials, they are just more quickly to return to them relative to control participants, illustrated by Busemeyer & Stout (2002) utilizing the expectancy valance model and we have mimicked in MHI samples utilizing a more simple methods by averaging the number of trials it takes to return to a trial that was previously punishing (Robb & Good, 2013). These findings collectively suggest that participants with alterations in IGT performance, whether they are VMPFC/OFC patients or individuals reporting a MHI, can switch away from risky decks, albeit for shorter periods of time. Moreover, these behavioural changes on the IGT appear to occur separable from explicit knowledge of strategy regardless of injury status and point to a more, or at least predominate, implicit mechanism for behavioural changes. This critique, however, does point out the interesting debate about distinguishing where learning ends

and decision-making begins, if they can, in fact, be separable.

Further, there has been considerable controversy within the literature regarding the role explicit knowledge plays throughout and during decision-making in the IGT (Bechara, Damasio, Tranel & Damasio, 1997; Maia & McClelland, 2004), and that explicit knowledge about decision-making outcomes may occur prior to implicit markers (i.e., SCR) and subsequent behavioural change (Maia & McClelland, 2004). Bechara et al. (1997) originally examined whether decision-making on the IGT is initially driven by explicit knowledge based on overt reasoning or whether overt reasoning is preceded by implicit biasing cues. They found that individuals developed SCRs in 'anticipation' of risky potentially-punishing decisions and transitioned to making selections from the less risky/advantageous decks well before they could explicitly acknowledge which decks were risky and which were not. However, when Maia and McClelland (2004) attempted to re-examine these findings by directly and deliberately asking questions about one's explicit knowledge about the task in a more probing and elaborate fashion (i.e., asking subjects to provide a general rating of how 'good' each of the decks were, an explanation for the conclusions made about deck preference, as well as estimations of average punishment values, reward values and net totals for each of the four decks respectively), they found support that indeed the subject's reportable and aware knowledge of the task preceded any changes in his/her behavioural selections. That said, an important limitation to this study is that the type of questioning that took place throughout the task (i.e., sampled at 10 trial intervals) occurred in such a fashion as to promote and induce the active and intentional use of, and meta-review of, potential solving strategies. In other words, by making participants more explicitly aware of tracking, assessing and attending to reward and punishment outcomes, they may have

elicited an explicit learning strategy in their subjects that then, indeed, guided behavioural performance and response selections.

Nonetheless, in line with the finding that explicit knowledge may influence IGT task performance, Gutbrod et al. (2006) found that individuals who live with an amnesic syndrome and, thereby, have deficits in making use of, if not retain, explicit knowledge, had impaired IGT performance compared to their non-amnesic counterparts. Further, more recent literature (e.g., Guillaume et al., 2009) has found no association between SCRs and explicit knowledge measured during the IGT; however, performance on the task and SCRs were positively related, and performance differed as a function of one's conscious awareness of the reward and punishment contingencies.

Collectively, these findings suggest that advantageous decision-making on the IGT may be associated with two independent systems, both implicit somatic signals and explicit knowledge of strategies. As a result, we sought to investigate explicit knowledge about the IGT contingencies at the end of the task as a means to reduce the confound of eliciting, rather than witnessing the emergence of explicit strategies and its influence on performance. We found that individuals reporting a MHI illustrated a slower transition from high risk/disadvantageous decks to low risk/advantageous decks producing fewer overall gains; and this occurred, despite both MHI and non-MHI participants reporting a preference for the riskier decks at the end of the task. Thus, both groups of subjects illustrated a dissociation between what they thought was happening in the task (explicit knowledge) and how they made selections and choices (implicit behaviour). This implies that implicit learning, perhaps 'gut-feelings' can drive decision-making performance independently of explicit knowledge of strategy.

Replication - Hypothesis III: Explicit Knowledge & IGT

Based on the above literature, it is hypothesized that individuals, regardless of head injury status, will report both a preference and increased propensity for winning (i.e., will report that they prefer the high risk/disadvantageous decks) despite making selection choices during the IGT indicative of 'learning' that these high-risk decks are disadvantageous (i.e., transition from disadvantageous to advantageous selections). Moreover, a dissociation will observed whereby it is anticipated there will be no predictive relationship between the participants' knowledge (i.e., reported strategy of selection, understanding of the value of each deck) and their behavioural selections. This will replicate previous findings demonstrating the dissociation of "knowing" versus "doing".

Manipulating Physiological Arousal

To examine the role of physiological underarousal contributing to decision-making, arousal has been manipulated through the presentation of emotionally salient stimuli (e.g., classical music). It has been well established within the literature that arousal and cognition are related in a curvilinear fashion (i.e., Yerkes-Dodson law), whereby cognitive performance increases with increasing arousal, until it reaches an optimal level of performance at which the point an individual becomes too aroused and cognitive performance proportionally decreases (Yerkes & Dodson, 1908). Classical music has been shown to increase physiological arousal, improve learning rates on measures of decision-making and attenuate differences in the rate of return to risky selections following punishment feedback for individuals reporting a MHI, but not for those who did not report a history of MHI. More specifically, negatively-valenced music (e.g., The Planets: Mars (Holdst), Piano Trio #5 – Ghost (Beethoven), etc.) was more effective in improving the decision-making performance for MHI participants, whereas positively-valanced music (e.g., Scherzo (Dvorak), Largo ma ton tanto (Bach), etc.) was more effective for non-MHI

participants. This provided preliminary evidence that the act of elevating arousal may be a potential therapeutic option for individuals who have previously sustained a TBI.

Subsequently, our lab has examined the influence of emotionally-evocative visual stimuli from the International Affective Picture System (IAPS), finding that this too can benefit cognitive performance in persons with MHI (Baker & Good, 2012). We expect to replicate and expand these findings for individuals with mild and moderate TBI by experimentally manipulating physiological arousal with visual stimuli and examining whether similar improvements in decision-making are observed.

Replication - Hypothesis IV: Manipulating Arousal

Based on the above findings, it is hypothesized that emotionally-evocative imagery used for the arousal manipulation will produce a corresponding increase in physiological arousal (as reflected by increases in EDA, pulse, etc.). Moreover, increases in arousal will benefit the decision-making performance on the IGT for those reporting a head injury (across the injury severity continuum) in two ways: by decreasing the number of trials required for transitioning from high risk to low risk choices; and by increasing the number of trials before returning to high risk decision following punishment feedback. Similarly, persons who do not report a history of MHI may be disadvantaged by increased arousal, should their levels of arousal introduce distress.

TBI & Depression

In addition to altered decision-making, depressive mood has been found to be another considerable barrier to social reintegration (Jorge et al., 1993). Depression has been associated with saddened mood or irritability, anhedonia, cognitive (i.e., changes to concentration/attention, cognitive slowing, etc.) and physical symptoms (i.e., psychomotor retardation or agitation, etc.)

as well as a number of cognitive biases (American Psychiatric Association, 2000; 2013, Beck, 1964). This constellation of symptomatology greatly hinders social reintegration (Brown, Gordon, & Spielman, 2003; Gomez-Hernandez, Max, Kosier, Paradiso, & Robinson, 1997). This is particularly concerning, as the literature indicates that between 18 and 49% of individuals with TBI experience comorbid psychiatric illness (Fann et al., 2004), with a particular emphasis on affect disorders (van Reekum et al., 2000). Moreover, between 40 and 90% of individuals who have sustained a TBI report some form of depressive symptomatology (Busch & Alpern, 1998; Jorge et al., 1993; Mooney & Speed, 2001; Seel et al., 2003). Despite the prevalence of depressive symptomatology, the mechanisms by which neural disruption from TBI results in greater affective dysregulation have not been well studied, nor is it clear that the pathophysiology of major depressive disorder (MDD) following TBI parallels that of typical MDD.

While the literature has addressed these questions in a limited manner, Maller et al. (2010) conducted a meta-analysis of the diffuse tensor imaging (DTI) literature that independently examined TBI and MDD. They identified an overlap in the number of brain structures involved in the pathophysiology of each of these conditions, including the corpus callosum, basal ganglia and frontotemporal structures. Moreover, the connectivity profile of individuals with TBI or MDD, which provides an indication of how many axonal connections lead to, and from, a particular brain region, indicated that prefrontal structures, and specifically that of the medial and lateral OFC were found to be considerably below that of a normative cohort (Maller et al., 2010). In addition to this, individuals diagnosed with MDD were found to have significantly reduced OFC volume compared to healthy participants (Bremner et al., 2002); and functional neuroimaging data has supported the role of the OFC in affective dysregulation

that may account for depressive symptomatology (Bremner et al., 1999; Drevets, 2007).

In addition to structural and functional imaging techniques implicating the OFC in MDD, Must et al. (2006) illustrated that individuals with MDD displayed a pattern of decision-making indicative of altered reward and punishment processing on the IGT, whereby they did not transition from high risk to low risk decks. MDD participants performed similarly to controls on a modified version of the IGT which seeks to determine if decision-making problems are due to failure of high reward to outweigh immediate punishment (Must et al., 2006). Individuals with MDD, like individuals with TBI, may possess a decision-making impairment indicative of a hypersensitivity to reward due to a neurally-based affective insensitivity to consequences of decision outcomes. This finding was further corroborated by Jollant et al. (2010) who reported that previous suicide attempters were found to be impaired on IGT. Further, functional neuroimaging indicated that less activation in lateral OFC regions during risky decisions was associated with poorer decision-making outcomes which could not be explained by executive function capacity (Must et al., 2006).

These two studies, the only two found to investigate IGT performance in individuals with unipolar MDD, both obtained decision-making profiles counter to expectations set much of the rest of the depression literature. It has been well established that individuals with MDD often have maladaptive responses to punishment, often referred to as being a "catastrophic response to perceived failure" (Eshel & Roiser, 2010; Roiser & Sakakian, 2013). For example, Elliott et al., (1996) in their landmark study illustrated that using the CANTAB battery of neuropsychological tests with individuals with unipolar depression illustrated a pattern whereby failure on one neuropsychological problem dramatically increased the likelihood of failure on the following problem/item. They postulated that this oversensitivity to negative feedback reflects differential

motivational and emotional feedback processing (Elliott et al., 1996). Moreover, considerable evidence demonstrates that individuals with MDD illustrate blunted emotional reactivity and anticipatory response to reward (albeit much of this data is self-report and very little has examined recordings of physiological arousal [GSR, EDA, etc.]; Bylsma, Morris & Rottenberg, 2008; Eshel & Roiser, 2010; Roiser & Sahakian, 2013). For example, McFarland and Klein (2009) had individuals with and without subclinical depression complete a puzzle-based neuropsychological measure while being provided positive (rewarded with money) and negative (cold pressor task) feedback. Participants higher in depressive symptomatology produced diminished self-reported positive emotion in anticipation of , or response to, reward despite no differences in self-reported anxiety in anticipation of, or response to, punishment. These findings coincide with much of the depression and anhedonia research, whereby one of the core features of MDD is the loss of pleasurable feelings associated with tasks or activity that were once found pleasurable (American Psychiatric Association, 2012; Bylsma et al., 2008).

The studies conducted by Must et al. (2006) and Jollant et al., (2010) demonstrating that individuals with MDD had impairments on the IGT, whereby immediate gains outweighed larger punishment overtime, is a difficult finding to reconcile. It would have been expected (and was expected by the authors) that individuals would perform similarly if not in a superior fashion to control subjects. However, neither study controlled for, or excluded, participants who had a history of MHI. Given the high prevalence of MHI (35-45%; Baker & Good, 2014; Segalowitz & Lawson, 1995; van Noordt & Good, 2011) and the association found between MHI and impairments in decision-making processes as measured by the IGT (Robb & Good, unpublished data; van Noordt & Good, 2011), it is suggested that the results pertaining to MDD and decision-making on the IGT are possibly confounded by MHI. As a result, this study will serve to provide

clarification to these findings.

To our knowledge, there is no research that has examined decision-making processes as measured by the IGT in individuals with and without head injury and examined this in the context of associated depressive symptomatology. It is expected that those with head injury will illustrate patterns of physiological underarousal and subsequently have reduced capacity to regulate, and otherwise modulate, anticipatory bioregulatory somatic/visceral cues which help to inform decision-making processes. These changes are thought to reflect functional and structural alterations in the OFC which contribute to and influence the way emotionally-salient feedback is processed (Berlin et al., 2004), potentially contributing to the development of maladaptive cognitive biases typically observed in persons with major depression (Beck, 1963). As a result, this study will examine the influence alterations in OFC function (e.g., emotional dysregulation) and feedback have on the neuropsychological sequelae of TBI and ongoing affective states.

Exploratory - Hypothesis V: IGT and Depression

Based on the above literature, it is possible that previous literature investigating decision-making processes utilizing the IGT in depressive patients may be confounded by a lack of control for a history of MHI. As a result it is hypothesized that learning performance on the IGT, and the rate of return to punishing selections, will predict self-reported depressive symptomatology in individuals who report previously sustaining an injury (lower performance will be associated with higher depression) but not for their non-injured cohort.

Summary

Collectively this research will be the first study, to our knowledge, that examines 'social' decision-making and its relationship to depression as a function of TBI injury severity. As well, it will provide further insight regarding the role of physiological (under)arousal in persons who

have experienced injuries to the head and the consequent potential for arousal-based therapeutic techniques for TBI populations. Such findings will have practical and theoretical implications regarding the relationship between cognition and arousal. Lastly, this study will highlight the potential role of functional and structural alterations of the OFC and its impact on processing of emotionally-salient feedback and depressive symptomatology

Methods

Participants

75 participants (39 females, 36 males) were recruited using Brock University's Psychology Department research website (SONA), in addition to posters placed around the university campus[1]. Of these, 32 participants or 37.6% reported a history of MHI (17 females, 15 males). An additional 11 persons who have documented traumatic injuries to the brain (4 females, 7 males) were recruited from various clinical offices within the Niagara community. Participants were not recruited on the basis of head injury, but were informed that they would be participating in a study examining emotion and cognition. This was done to reduce *diagnosis threat* (Suhr & Gunstad, 2002; 2005) and its affect on expectations and performance. Ages for the participants ranged between 18 and 34 years (M = 20.78, SD = 2.68) for University students and between 15 and 33 for patients (M = 22.91, SD = 6.07). Table 1 describes means and standard deviations for participant's age and education as well as maternal and paternal education as a function of head injury status. As observed, the groups do not differ considerably in any of these variables; most participants have one or two years of post-secondary education and parents who achieved at least some post-secondary education. With respect to parental income, participants do not vary considerably with respect to head injury status and are

[1] Originally 76 university students participated in the study but unfortunately a single participant was not included in any of the statistical analyses as their data (i.e., IGT performance) was lost in a computer malfunction.

predominately upper middle class. Lastly, the majority of participants, regardless of head injury status, identified themselves as being Caucasian.

Table 1:

Descriptive statistics of age, and proportions of completion the years of education, parental income, and parental education.

	Control (n = 43)		MHI (n = 32)		Moderate TBI (n = 11)	
Variable:	*M*	*SD*	*M*	*SD*	*M*	*SD*
Age	20.77	2.98	20.78	2.38	22.91	6.07
Years of Education completed	13.86	1.39	13.91	1.53	14.36	2.11
Years of Education Mother completed	15.00	1.76	14.28	1.97	15.36	1.91
Years of Education Father completed	14.35	2.06	14.56	2.15	15.20	1.69
Parental Income			Percentage (n)			
Under $25,000	2.3 (1)		3.1 (1)		0.0 (0)	
$25,000 to $49,999	25.6 (11)		18.8 (6)		18.2 (2)	
$50,000 to $74,999	16.3 (7)		12.5 (4)		9.1 (1)	
$75,000 to $99,999	11.6 (5)		21.9 (7)		18.2 (2)	
$100,000 to $124,000	25.6 (11)		18.8 (6)		54.5 (6)	
$125,000 to $149,999	2.3 (1)		3.1 (1)		0.0 (0)	
$150,000 or more	16.3 (7)		21.9 (7)		0.0 (0)	
Ethnicity			Percentage (n)			
Hispanic	4.7 (2)		0.0 (0)		0.0 (0)	
Caucasian	62.8 (27)		75.0 (24)		81.8 (9)	
European-born	7.0 (3)		6.3 (2)		0.0 (0)	

African	2.3 (1)	3.1 (1)	9.1 (1)
East Indian	4.7 (2)	6.3 (2)	0.0 (0)
West Indian	2.3 (1)	0.0 (0)	0.0 (0)
Chinese	0.0 (0)	3.1 (1)	0.0 (0)
Other	16.3 (7)	6.3 (2)	9.1 (1)

Table 2 describes descriptive statistics with respect to sex, handedness, diagnoses of neurological and psychiatric conditions, learning disabilities, use of psychotropic medications, and injury characteristics as a function of head injury status. As observed, the majority of the participants are right-handed and there were a minimal number of reported psychiatric or neurological conditions (n=6; evenly distributed among the groups). More participants from the moderate TBI group were taking psychotropic medications than other groups, and these medications were reported to be of the selective serotonin re-uptake inhibitor (SSRI) class (i.e., Cipralex [escitalopram]) and the serotonin norepinephrine re-uptake inhibitor (SNRI) class (i.e., Effexor [venlafaxine]) respectively[2].

For students who reported a history of MHI, the majority indicated that these resulted from sports-related injury, followed by falls. Further, 15.5% of these subjects reported sustaining more than one head injury. It is interesting to note that a considerable portion of those who sustained MHI sought medical attention (40%), which is much higher than previous samples from our lab (van Noordt & Good, 2011; Baker & Good, 2014). For patients, the majority of individuals identified motor-vehicle collisions, followed by sports-related injury, as being

[2] Analyses were conducted with and without these participants; no differences in the results were obtained as a function of psychotropic medication.

responsible for their head injuries. Almost half of these patients required admissions to a hospital and 36% of this group sustained multiple head injuries.

Table 2:

Descriptive statistics of sex, handedness, diagnoses of interest, and injury characteristics as a function of head injury status

Variable: Percentage (n)	Control (n = 43)	MHI (n = 32)	Moderate TBI (n = 11)
Male	48.8 (21)	46.9 (15)	63.6 (7)
Right Handed	86.0 (37)	93.8 (30)	90.9 (10)
Psychiatric Diagnosis	4.7 (2)	6.3 (2)	63.6 (2)
Taking psychotropic medications	2.3 (1)	3.1 (1)	27.3 (3)
Neurological condition	7.0 (4)	9.4 (3)	36.4 (4)
Diagnosed with a learning disability	0.0 (0)	6.3 (2)	0.0 (0)
Injury Characteristics			
Symptoms > 20 minutes	-	40.6 (13)	100 (11)
Loss of consciousness (LOC)	-	53.1 (17)	81.8 (9)
Duration of LOC			
Less than 5 minutes	-	46.9 (15)	27.3 (3)
Less than 30 minutes	-	6.3 (2)	27.3 (3)
Less than one week	-	0.0 (0)	27.3 (3)
Unknown	-	46.9 (15)	18.2 (2)
Where head was struck:			
Front	-	37.5 (12)	36.4 (4)
Right	-	15.6 (5)	9.1 (1)

Left	-	6.3 (2)	9.1 (1)
Other	-	6.3 (2)	27.3 (3)
Cannot recall	-	33.4 (11)	18.2 (2)
Cause of Injury			
MVC		3.1 (1)	54.5 (6)
Sports-related Injury		56.3 (18)	36.4 (4)
Falls		28.1 (9)	0.0 (0)
Other		12.5 (4)	9.1 (1)
Result in a diagnosed concussion		56.3 (18)	100(11)
Required stitches		9.4 (3)	2 (18.2)
Receive medical treatment		40.6 (13)	90.9 (10)
Stayed overnight in hospital		3.1 (1)	45.5 (5)
More than one injury		15.6 (5)	36.4 (4)

Neuropsychological Measures

Iowa Gambling Task (IGT). The IGT is a measure of decision-making performed during conditions of uncertainty and simulates a 'gambling' situation. It assesses one's ability to adapt one's selections or choices based on longer-term prospects of overall gain. This paradigm is made up of a highly complex reward and punishment schedule, previously described (see Bechara et al., 1994 for full details) and, as a result, taps into the construct of associative reverse learning. The task consists of four decks of cards - Decks A and B are considered disadvantageous (i.e., overall, selections from these decks result in losses that outweigh the gains); while Decks C and D are considered advantageous (i.e., overall, selections from these

decks result in gains that outweigh the losses). Participants complete 100 trials. The reward and punishment contingencies vary as a function of both deck and ten-trial blocks. Bechara et al. (1997) typically classified the trials into four stages: (1) Trials 0 – 20 represent the 'learning' phase, whereby participants often sample from all of the decks; (2) Trials 21 – 50 represent the 'pre-hunch' phase, during which individuals often begin developing reactions (as measured by skin conductance response increases) in response to selections from the punishing or disadvantageous decks; (3) Trials 51 – 80 represents the 'hunch' phase, during which participants commonly make the transition from selecting cards from the disadvantageous (A and B) decks to preferring to select cards from the advantageous (C and D) decks; and lastly, (4) Trials 80 – 100, referred to as the 'conceptual' phase, participants can often report which decks are preferred, if not advantageous, and which are not preferred, if not disadvantageous.

While there is an extensive body of literature examining the validity of the IGT and its neuropsychological correlates, as well as converging evidence from clinical case studies, behavioural reports, and neuroimaging studies indicating that the IGT can provide a measure of OFC functionality (Bechara et al., 1994; 2000a; 2000b; Buelow & Suhr, 2009; Domasio, 1994; Li et al., 2010), its use in this study was solely to provide a measure of decision-making under conditions of uncertainty. It may also reflect 'group' differences that may be attributable to OFC functionally between persons with and without a history of MHI, but certainly no inference regarding an individual's performance or OFC competency is being confirmed in this study. For this study, the computerized version of the IGT was used, in addition to the use of a point system of gains and losses (as a facsimile of money), both of which have been illustrated to be comparable to the manual/original version of the task (Bechara et al., 2001; Bowman & Turnbull, 2003; Buelow & Suhr, 2009).

Matrix Reasoning (Wechsler Adult Intelligence Scale-IV [WAIS-IV]). Matrix Reasoning is a subtest Wechsler Scales and has been viewed predominately as a measure of fluid intelligence, logical reasoning, abstract thought, visuospatial ability, broad visual intellect, and perceptual organization (PsychCorp, 2008). Participants are asked to solve a series of 26 increasingly difficult visual pattern completions. While this has not shown to be highly sensitive to the effects of neural injury, it is commonly held as a good measure of premorbid intellectual functioning (Tranel, Manzel & Anderson, 2008).

Social Perception (Faces [Affect Naming], Advanced Clinical Solutions Supplement to WAIS-IV and WMS-IV). Affect naming requires that participants look at 18 different faces and identify the emotional expression displayed in each (i.e., either happy, sad, angry, fear, disgust, surprise, and no feeling/neutral). This test assesses one's capacity to identify affective emotions being expressed by others (Wechler, 2009), a difficulty for individuals who have experienced TBI (e.g., Radice-Neumann, Zupan, Babbage, & Willer, 2007). As a result, this test is considered to be sensitive to impairments associated with traumatic head injuries.

Word Reading (Wide Range Achievement Test [WRAT]). Participants are asked to read a list of words that increase in vocabulary difficulty across trials. Participants are given a maximum of 10 seconds per word and the task is discontinued after 10 incorrect trials. This test has been associated with providing a good approximation of reading ability and academic achievement (Lezak et al., 2012; Wilkinson & Roberson, 2006) and is included in this study as an overall intellectual competency measure.

Psychophysiological Measures

All electrophysiological measures were collected using Polygraph Professional Suite Datapac USB 16-bit Data Acquisition equipment and respective version 2.6.0.0 software.

Measures of electrodermal activation and pulse oximetry were taken from the non-dominant hand, allowing for the dominant hand to be used to manipulate the mouse for the computer-based tasks, permitting minimized participant movement on the recording side, thereby reducing the prevalence of artifacts.

Electrodermal Activation (EDA). Measures of EDA are similar to that of SCR and galvanic skin response (GSR), providing a continuous index of autonomic arousal through the measurement of skin perspiration. EDA has been illustrated to be highly sensitive to alterations in sympathetic nervous system arousal, which in turn affect production of perspiration. Measures of skin perspiration are highly common within the literature for examining arousal during cognitive, neuropsychological and behavioural tasks (Lykken & Venables, 1971). Like many forms of electrophysiology, EDA has excellent temporal resolution.

For this study, pure metal alloy electrodes were used and placed on the distal end of the index and fourth fingers. Averages were derived for epochs based on amplitude of electrical change, measured in microsiemens. EDA was measured in real time throughout the IGT, whereby researchers demarcated eight-second anticipatory epochs, prior to each selection a participant made, and four-second feedback epochs, after the selection had been made (time durations were based on previous literature) (Bechara et al., 1996; van Noordt & Good, 2011).

Pulse Oximetry. A pulse oximeter was used to measure pulse rate, in beats per minute, throughout the IGT by detecting rates of blood perfusion through the determination of light absorption. It was sampled continuously throughout the task whereby the device was placed on the distal portion of the middle finger, and epochs were demarcated in the same fashion that was described for EDA.

Endocrine Measures:

Salivary Cortisol – Enzyme-linked Immunosorbent Assays (ELISA). Participants provided six saliva samples in total, including three as part of another study outside of the testing session and three samples during the testing session as part of the present study as an index of emotion and stress responsivity. Participants were asked to passively drool into plastic polystyrene culture tubes and these samples were then placed in freezer storage until analyses could be conducted. Cortisol (ng/mL) concentrations were obtained using ELISA commercial kit and preparation/analysis occurred over two separate days. Samples were defrosted and duplicate 100 μL of saliva were assayed and optimal densities were determined using a Biotek Synergy Plate reader at 450 nm.

Questionnaire-based Measures

Explicit Knowledge Questionnaire. The explicit knowledge questionnaire was originally developed by Maia and McClelland (2004) and was used to assess the extent of knowledge a participant was aware of, and could report, regarding the reward-punishment contingencies of each of the IGT decks. It is administered after completing 100 trials of the IGT. Items on the questionnaire include a rating of each deck (-10 = worst deck, +10 = best deck), as well as an estimate regarding the average net outcome, average winnings, frequency of losses, and average losses for each of the four decks based on 10 trial blocks. Lastly, subjects are asked to rate how confident they are in their estimates, and which deck they would prefer to choose if they could play again (Maia & McClelland, 2004; See Appendix B).

Demographics Questionnaire. A demographics questionnaire was used to gather information about a person's educational, medical and social histories, with a particular interest in ascertaining a participant's history of sustaining a MHI (i.e., have you ever hit your head with a force sufficient to alter your consciousness [e.g., loss of consciousness, vomiting, dizziness]?).

Questions are asked about each subject's age, education, mother and father's level of education and income, in addition to measures of head injury severity (i.e., LOC, PTA, hospitalization, etc.). Variables that may confound or alter test performance, such as the presence of learning disabilities, psychiatric or neurological conditions were also measured (See Appendix B).

Life Stressors Scale. The life stressors scale was adapted from Holme and Rahe (1967)'s social readjustment rating scale, as a measure of self-reported life stressors on overall health. This modified version provides participants with a list of 18 highly stressful events and they are asked to endorse and rate any of those that have been experienced in past six months. Each stressor has an individual rating based on potential life impact (based on norms), providing scores for both a weighted total score and a frequency measure of life stressors, indexing the amount of readjustment recently experienced by participants (See Appendix B).

Post-Concussive Syndrome Checklist (PCSC). This modified PCSC consists of a list of 10 post-concussive symptoms that are commonly experienced by individuals after a head injury (i.e., headache, dizziness, irritability, memory problems, difficulty concentrating, fatigue, visual disturbance, aggravated by noise, judgment problems, and anxiety). Participants were asked to rate the frequency (*1 = not at all*, *5 = all of the time*), intensity (*1 = not at all*, *5 = crippling*) and duration (*1 = not at all*, *5 = constant*) of experiencing each of these symptoms for the last two months (Gouvier, Uddo-Crane, & Brown, 1988; See Appendix B).

Beck's Depression Inventory (BDI; Beck, Ward, Mendelson, Mock & Erbaugh, 1961). The BDI is a self-report measure consisting of depressive criteria frequently reported by psychiatric patients but infrequently reported by non-depressed individuals. It is made up of 21 items, including mood, pessimism, sense of failure, lack of satisfaction, guilt feelings, sense of punishment, self-dislike, self-accusation, suicidal thoughts, crying, irritability, social withdrawal,

indecisiveness, distorted body image, work inhibition, sleep disturbances, fatigability, loss of appetite, weight loss, somatization, procuration, and loss of libido. Items are reported on a scale of 0 to 3 in terms of intensity experienced by the participant and their total score is rated as indicating either no, mild, moderate or severe depression (Beck, Steer, & Garbin, 1988).

Symptom Assessment-45 (SA-45). The SA-45 is a 45 item brief psychological symptom checklist that provides an overall rating of psychiatric symptomatology as well as domain specific ratings of symptomatology, including anxiety, hostility, obsessive-compulsive, phobia anxiety, somatization, depression, interpersonal sensitivity, paranoid ideation, and psychoticism. Each of the items are rated on a Likert scale of 1 (not at all) to 5 (extremely). The psychometric properties of this measure have been provided elsewhere (e.g., Davison, Bershadsky, Silversmith, Maruish, & Kane, 1997) but has been supported in several clinical and research environments. Norms for this measure are derived from a database of 18,000 subjects based on both nonpatient community samples and inpatient psychiatric samples (Davison et al., 1997; Maruish, 1999).

Emotional Arousal Induction Manipulation:

Emotional arousal was manipulated by having participants view and rate pictures derived from the International Affective Picture System (IAPS; Lang et al., 2008). The pictures are approximately 10” x 8” in size and were presented on a 17” computer screen with a viewing distance of approximately 60 cm. 45 pictures were displayed for five seconds and participants were asked to rate each in terms of intensity, pleasantness, arousal, and empathy elicitation on a scale from 1 (minimal) to 9 (significant). The scenes in the pictures involved either 15 emotionally-neutral, 15 unpleasant, or 15 pleasant stimuli (as ranked by IAPS ratings based on

normative data, Lang et al., 2005; Libkuman et al., 2007; Mikels et al., 2005; and pilot data in our lab, Baker & Good, 2014) including pictures of persons, animals, and inanimate objects.

Procedure

Participant testing was completed individually by one of three researchers (one female, two male). Each had direct training in the administration of both the neuropsychological measures and the electrophysiological measures. This study was part of a collaborative project examining emotion and cognition, whereby sessions lasted approximately 150 minutes in duration. Only the procedure pertinent to this study will be described below. Brock University students and individuals from the community (recruited from clinical practices in Niagara) were recruited to participate in this study. With the exception of those individuals who have sustained a moderate TBI, subjects were not explicitly recruited as a function of having a history of head injury as a means to prevent against *diagnosis threat* (Suhr & Gunstad, 2002; 2005)[3]. Participants were interviewed via telephone and answered screening questions regarding their history of medication use, sleep patterns, age, level of education and gender. Participants were excluded if they were shift workers or taking steroid-based inhalers, each of which have been shown to have an impact on measures of salivary cortisol. If deemed appropriate for the study, participants were provided with a salivary sampling kit and instructions on when and how to conduct their samples prior to arriving at the test session.

On the test day, and after completing a written informed consent, participants were asked for their home-based salivary-samples and then instructed (and assisted as needed) on how to administer the physiological activity recording equipment in order to collect heart rate, electrodermal activation (EDA) and respiratory rates. The researcher then evaluated the

[3] Diagnosis threat is defined as the impact that negative expectations about performance given being labeled with a particular diagnosis may have on cognitive performance (Suhr & Gundstad 2002).

equipment to ensure it was working correctly, and completed a three-minute initial testing measure, delineating time epochs for later comparison. Subjects were then asked to produce a salivary sample.

Participants were then asked to complete a series of neuropsychological tasks including word reading (WRAT-4) and Matrix Reasoning (WAIS-IV), after which measures of physiological arousal (i.e., EDA, respiration, and Pulse) and a salivary sample were taken again. Participants were randomly assigned to either the pre-exposure (i.e., completion of the imagery ratings, followed by the IGT) or the post-exposure (i.e., completion of the IGT, followed by completion of imagery ratings) arousal manipulation condition. Participants completed the emotional arousal manipulation by viewing, and subsequently rating, 45 images taken from the IAPS. The images were displayed for 5 seconds followed by a request to rate each on its intensity, pleasantness, arousal, and empathy elicitation on a scale from 1 (minimal) to 9 (significant). Physiological recordings of arousal were taken throughout. Following the manipulation, another three minute measure of physiological arousal and a salivary sample was taken.

Participants were provided with a set of paper and pencil questionnaires (i.e., BDI, SA-45, and Everyday Living Demographics) to complete and, then, debriefed about the nature of the study. All participants received either research participation credits or money as an honorarium for their time.

Statistical Analyses:

Hypothesis I (Underarousal): A one-way analysis of variance (ANOVA) was conducted on each of the dependent variables (EDA, salivary cortisol) to test whether initial testing arousal varies as a function of injury severity (moderate TBI, MHI, no MHI). Post-hoc

analyses of least significant difference (LSD; equal-variances assumed) and Dunnett's C (equal-variances not assumed) were also conducted in order to investigate group differences**.** *It was expected that individuals with no history of head injury would have the highest physiological arousal, followed by those reporting a MHI and, less so, those with moderate TBI.* In addition, a severity index variable was developed to examine the spectrum of injury severity based on symptom indicators (i.e., loss of consciousness, post-traumatic amnesia, post-concussive symptoms, number of previous injuries, etc.) and a Pearson-r correlation conducted. It was expected that an inverse linear relationship would exist, whereby as injury severity increases, physiological arousal will decrease.

Hypothesis II (IGT performance): To test whether IGT performance varied as a function of injury severity, a ratio of disadvantageous to advantageous selections was calculated (Disadvantageous decks [Deck A + Deck B] – Advantageous decks [Deck C + Deck D]) for each of the last 50 trials in 10 trial-blocks, and was examined across time (i.e., the last 50 trials were examined in order to permit sufficient selection from, and familiarity with, each of the four decks). Thus, a 3 × 5 (Head Injury Status [non-MHI, MHI, moderate TBI] × Trials [trials 51-60, trials 61-70, trials 71-80, trials 81-90, trials 91-100] mixed analysis of variance (ANOVA) was conducted on the ratio of the decks. Lastly, a 3 × 2 (Head Injury Status [non-MHI, MHI, moderate TBI] × Decision Type [Advantageous, Disadvantageous] mixed ANOVA was conducted on the number of trials between selecting from a deck that was punished and returning to that very same deck. Post-hoc analyses of least significant difference (LSD; equal-variances assumed) and Dunnett's C (equal-variances not assumed) were conducted on the main effect of injury severity in order to locate group differences. *It was expected that those with no history of injury would have the highest ratio of advantageous to disadvantageous selections and the*

longest return rate (i.e., increased number of trials) between being punished by a selection and returning to this same deck, followed by those with MHI and those with a moderate TBI.

Hypothesis III (Explicit Knowledge & IGT): Hypothesis three is that participants, regardless of head injury status, would endorse a preference for disadvantageous selections/risky decks, a 3 × 4 (Head injury Status [non-MHI, MHI, moderate TBI] × Deck Type [deck A, deck B, deck C, deck D]) mixed ANOVA was conducted. In addition to this, Pearson-r correlations were conducted between a participant's rating of preference for each of the four decks and the frequency of selections made. *It was expected that there would be an effect of deck type, whereby participants endorse a greater preference for disadvantageous decks (A & B) and, further, that there would not be any relationship (linear or otherwise) between preference and behaviour.*

Hypothesis IV (Manipulating Arousal): To test whether the emotionally-evocative imagery acts as an arousal manipulation, each of the physiological measures (EDA, pulse, salivary cortisol) and the IGT performance was compared across groups (i.e., those subjects who completed the imagery analysis prior to IGT testing and those who completed the imagery analysis post-IGT testing – a between-subject variable) and across head injury status (also a between-subject variable). Thus, a 3 (Head Injury Status [non-MHI, MHI, moderate TBI]) x 2 (Arousal Manipulation [pre-exposure, post-exposure]) mixed ANOVA for each of the physiological measures was completed, as well as a 3 × 5 × 2 (Head Injury Status [non-MHI, MHI, moderate TBI] × IGT Performance on the last set of 50 Trials [trials 51-60, trials 61-70, trials 71-80, trials 81-90, trials 91-100] × Arousal Manipulation status [pre-exposure, post-exposure]) mixed ANOVA. Further, a 3 × 2 × 2 (Head Injury Status [non-MHI, MHI, moderate TBI] × Deck Type [Advantageous, Disadvantageous] × [pre-arousal measures, post-arousal measures]) mixed ANOVA was completed on the number of trials between a selection from a

deck that was punished and returning to that very same deck as a function of head injury status and arousal manipulation. Post-hoc analyses of least significant difference (LSD; equal-variances assumed) and Dunnett's C (equal-variances not assumed) were conducted on the main effects to investigate group differences. *It was anticipated that a main effect of the emotional arousal manipulation would be observed in all three tasks, with physiological arousal increasing, and IGT performance improving (e.g., greater selection from advantageous decks) following the arousal induction and that performance for the head injury groups would improvement particularly, whereas for the non-injured group, performance will decrease.*

Hypothesis V (IGT and Depression). *IGT performance for those who have sustained an injury will predict self-reported depressive symptomatology (as measured on the BDI and SA-45).* To test this, a simultaneous linear regression was conducted entering the ratio of disadvantageous-to-advantageous and head injury status on the first step and then the above variables as well as the interaction variables on the second step. *It is expected that the second step of the model will account for variability over and above that of just IGT performance alone, suggesting a moderation effect of head injury status.* In addition, Pearson-r correlations were conducted between the rate of return to a deck selection following punishment and a participant's depression scores. It was anticipated that decision-making performance would only predict depression scores for those with a history of head injury. Lastly, a simultaneous linear regression was conducted entering the rate of return to a deck selection following punishment and head injury status on the first step and then the above variables as well as the interaction variables on the second step. *It is expected that the second step of the model will account for variability over and above that of just rate of return alone, suggesting a moderation effect of head injury status.*

Results

Neuropsychological measures were taken to ensure participants did not differ on estimates of (1) academic achievement and general intellectual functioning, as measured by Word Reading Subtest (WRAT-4; Figure 1), (2) reasoning capacity and abstract thought, as measured by Matrix Reasoning (WAIS-IV; Figure 2), and (3) capacity to correctly identify emotional expressions, as measured by affect recognition (Advanced Clinical Solutions Supplement to WAIS-IV and WMS-IV; Figure 3). No differences were observed for word reading (F (2, 83) = 2.25, p = .11, ns), matrix reasoning (F (2, 82) = 1.58, p = .212, ns), or affect recognition (F (2, 83) = 1.35, p = .266, ns), indicating no group differences in reasoning capacity, abstract thought or affect recognition as a function of head injury status. In summary, there were no differences in academic achievement and general intellectual functioning.

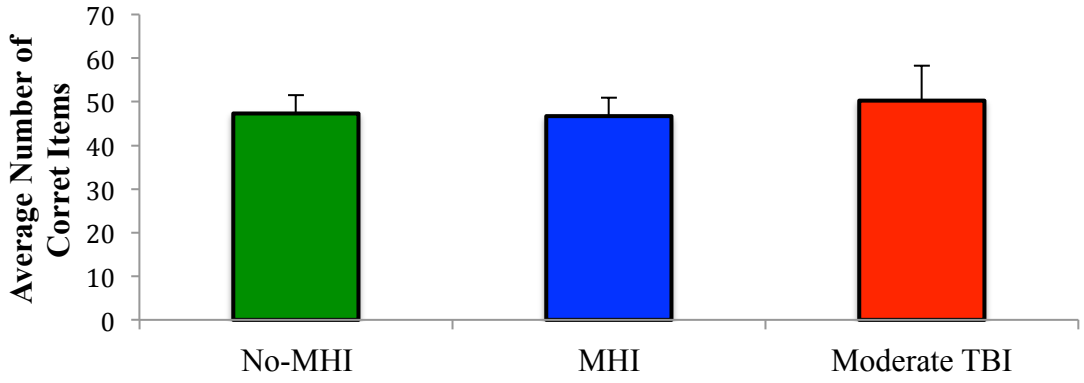

Figure 1: **Performance on Word Reading subtest from the Wide Range Achievement Test-4th Ed (WRAT-4) as a function of head injury status**.

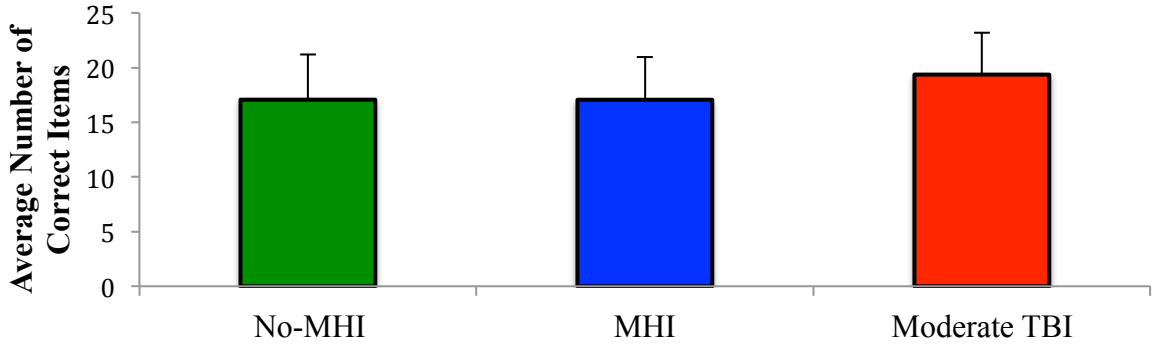

Figure 2: **Performance on Matrix Reasoning from the Wechsler Adult Intelligence Scale-IV [WAIS-IV] as a function of head injury status**

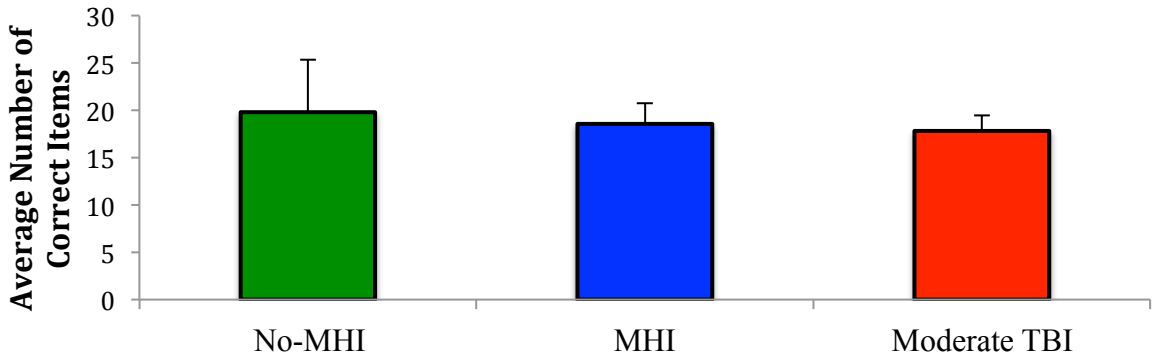

Figure 3: **Performance on Social Perception, subtest Faces (Affect Naming) from the Advanced Clinical Solutions Supplement to WAIS-IV and WMS-IV as a function of head injury status**

To measure self-reported psychiatric symptomatology (i.e., anxiety, depression, obsessive-compulsive features, phobias, thought disorders, etc.), participants completed the SA-45. Table 3 depicts the means and standard deviations for the nine different subscales as well as the global severity index. There were no differences observed as a function head injury status (Table 3). Neither were there any differences in the Beck's Depression score, or the number, or severity, of life stressors (Table 4) demonstrating that the groups did not differ on psychiatric symptomatology or amount of life stress. Post-concussive symptomatology was also assessed using the Post-Concussive Syndrome Checklist (Table 5). Participants only differed in the number of headaches (trend) reported and their experience of judgment difficulties. Post-hoc analyses indicated that those with a moderate TBI reported greater severity for both of these symptoms compared to MHI and no-MHI, but the latter two groups did not differ from one another. This suggests that with respect to post-concussive symptomatology, these groups only differ on two of the ten most commonly reported symptoms following injury.

Table 3:

Descriptive statistics (means [standard deviations]) of the subscales of the SA-45 as a function of head injury status with inferential statistics (one way ANOVAs for each subscale)

SA-45	Control	MHI	Moderate TBI	*p* value
Anxiety	8.12 (3.17)	8.71 (3.87)	8.54 (3.29)	.75
Depression	10.30 (4.34)	10.19 (4.65)	10.45 (4.57)	.98
Hostility	6.65 (3.28)	7.06 (2.12)	6.27 (1.90)	.67
Interpersonal Sensitivity	10.18 (4.80)	10.65 (4.96)	9.36 (4.43)	.74
Obsessive Compulsive	11.37 (4.29)	11.78 (3.25)	11.18 (3.92)	.87
Paranoid ideations	9.02 (3.97)	9.22 (4.63)	8.09 (3.36)	.74
Phobic Anxiety	6.60 (2.44)	6.625 (2.62)	7.36 (3.93)	.70
Psychoticism	6.48 (2.21)	6.68 (2.68)	6.36 (3.01)	.91
Somatization	8.90 (3.51)	8.22 (2.52)	7.18 (2.56)	.23
Global Severity Index	77.65 (26.06)	79.15 (25.38)	74.81 (25.37)	.89

Note: One-way ANOVAs were conducted to test whether symptoms differed as a function of head injury status.

Table 4:

Descriptive statistics (means [standard deviations]) of symptoms from the Life Stressors Scale and Beck's Depression Score as a function of head injury status with inferential statistics (one way ANOVAs)

Life Stressors Scale	Control	MHI	Moderate TBI	*p* value
Frequency	2.84 (2.02)	3.06 (1.88)	3.00 (1.32)	.88
Total Score	101.49 (80.33)	108.25 (74.13)	118.89 (49.12)	.80
Beck's Depression Inventory Total Score	10.49 (8.71)	8.62 (6.99)	8.90 (6.86)	.57

Table 5:

Descriptive statistics (means [standard deviations]) of symptoms (frequency, intensity, duration [5 point likert scales] averaged) from the Post-Concussive Syndrome Checklist as a function of head injury status with inferential statistics (one way ANOVAs for each symptom)

Post Concussive Symptom:	Control	MHI	Moderate TBI	*p* value
Headache	2.72 (0.92)	2.52 (1.02)	3.37 (0.56)	.06
Dizziness	1.57 (0.78)	1.71 (0.78)	1.81 (1.04)	.62
Irritability	2.69 (0.84)	2.47 (1.05)	2.66 (1.06)	.61
Memory	2.03 (1.03)	2.02 (0.95)	2.66 (1.11)	.21
Concentration Difficulties	2.71 (0.85)	2.82 (0.97)	2.18 (1.16)	.20
Fatigue	2.93 (1.05)	2.85 (1.10)	3.22 (0.8165)	.65
Visual Disturbances	1.35 (0.66)	1.38 (0.86)	1.85 (1.15604)	.24
Sensitivity to Noises	2.15 (0.88)	1.97 (1.09)	2.18 (1.00154)	.71

Judgment Difficulties	1.65 (0.85)	1.39 (0.60)	2.11 (0.84984)	.04
Anxiety	2.26 (0.99)	2.58 (1.17)	2.37 (0.87312)	.44

Replication - Hypothesis I: Underarousal

Initial testing measures of physiological arousal were measured by EDA, pulse rate and salivary cortisol. Figure 4 illustrates the pattern of arousal for EDA amplitude across injury status. A main effect of injury status was obtained (F (2, 83) = 67.82, $p < .001$, $\eta_p^2 = .62$). Follow-up post-hoc LSD comparisons illustrated that the no-MHI participants had significantly higher EDA amplitude compared to the MHI and moderate TBI groups ($p < .001$). Further, while the means were in the expected direction (i.e., moderate TBI participants having the lowest average EDA amplitude), arousal did not differ between the two injury groups ($p = .35$, ns)[4].

The same pattern of results was obtained for pulse rate (bpm), illustrated in Figure 5 (i.e., main effect of injury severity - F (2, 82) = 5.56, $p = .005$, $\eta_p^2 = .12$). The no-MHI group produced significantly higher pulse rates compared to the MHI and moderate TBI groups (LSD post-hoc: $p < .05$), but MHI and moderate TBI were not found to differ ($p = .36$). Interestingly, the effect size for EDA amplitude as a function of injury status is considerably larger than for pulse rate.

[4] The head injury groups were not found to differ as a function of reported sleep schedule, duration of sleep or effectiveness of their sleep.

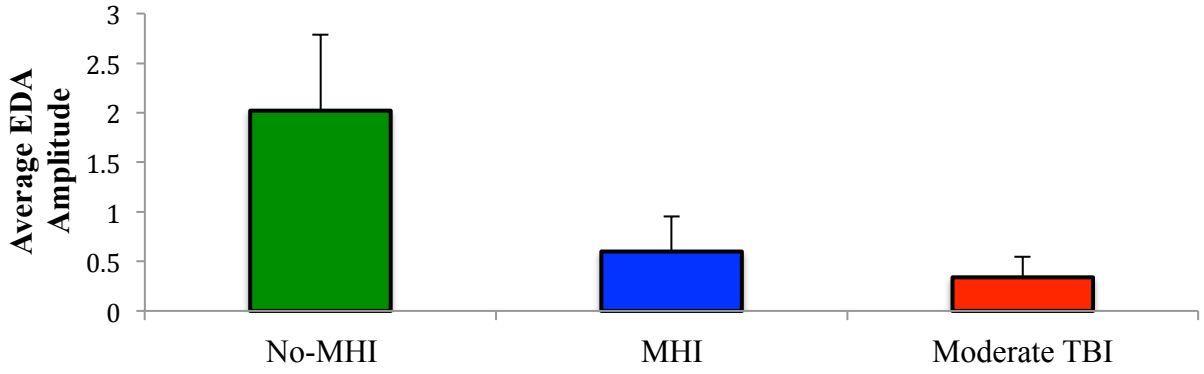

Figure 4. **Initial testing physiological arousal (as measured by electrodermal activation [EDA]) depicted as a function of head injury status.**

Measures of salivary cortisol taken within the testing session did not produce a significant difference across the groups (see Figure 6). A trend was observed for the main effect of head injury status on salivary cortisol for both the overall effect (ANOVA - F (2, 80) = 2.33, p = .10, η_p^2 = .06)[5] and the post-hoc analyses, whereby those with a moderate TBI illustrated the highest salivary cortisol levels, followed by MHI, and non-MHI (non-MHI differed from MHI and moderate TBI $p < .10$, but the injured groups did not differ from one another $p = .56$).

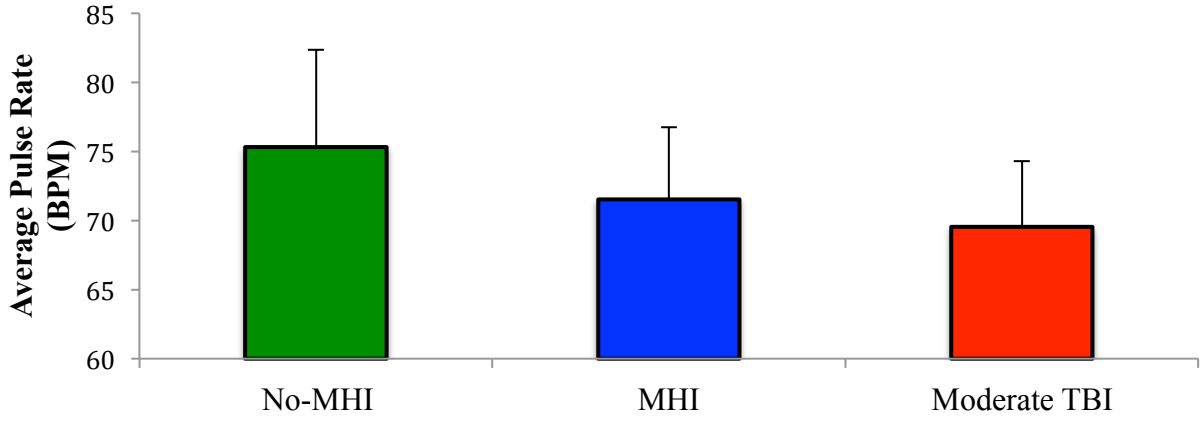

Figure 5. **Initial testing physiological arousal (as measured by pulse rate) depicted as a function of head injury status.**

[5] Degrees of freedom are lower in this analysis as two participants with moderate TBI were tested after salivary cortisol assays were performed; one further participant's results were not usable due to contamination.

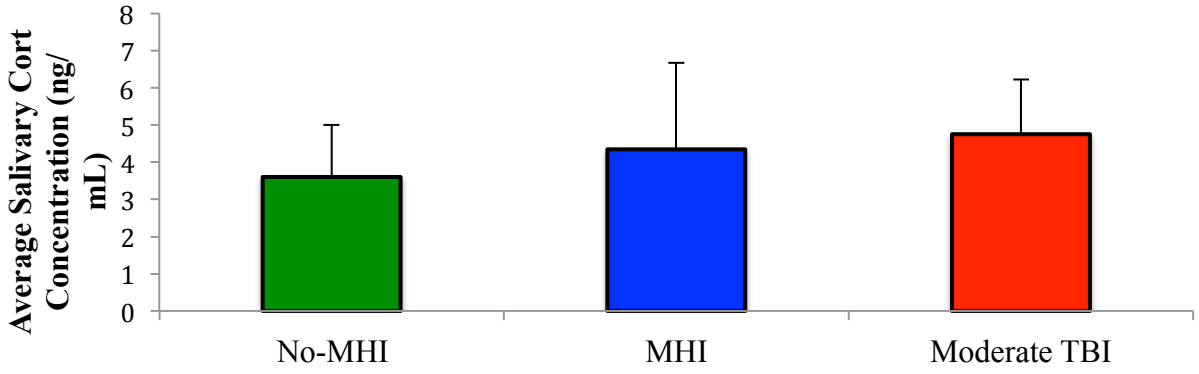

Figure 6. **Initial testing physiological arousal (as measured by salivary cortisol concentrations) depicted as a function of head injury status.**

A composite variable was calculated only for participants who reported previous injury by adding each factor related to injury severity (i.e., symptoms lasting more than 20 minutes [no = 0, yes = 1], loss of consciousness [no = 0, yes = 1], duration of LOC [less than 5 minutes = 1, less than 30 minutes = 2, less than 24 hours = 3, less than 1 week = 4, less than 1 month = 5, greater than 1 month = 6], whether a concussion was diagnosed [no = 0, yes = 1], stitches were required [no = 0, yes = 1], medical treatment was sought [no = 0, yes = 1], whether admission to the hospital occurred [no = 0, yes = 1], and whether there were multiple injuries [no = 0, yes = 1]. Scores could range from 0 to 25 (multiple injuries and endorsement of all items). Figure 7 illustrates a scatter plot of EDA amplitude depicted as a function of injury severity.

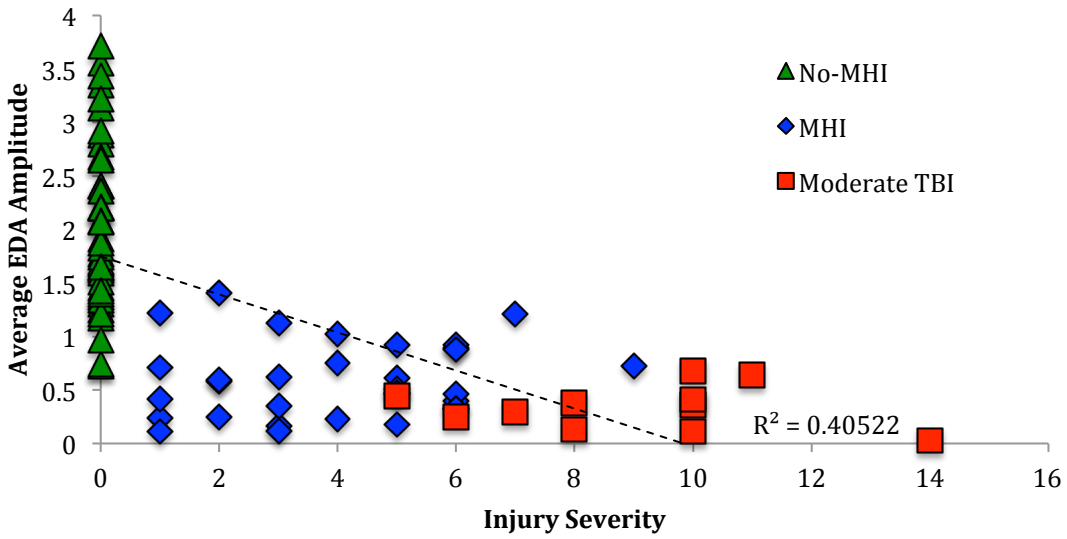

Figure 7. **Initial testing physiological arousal (as measured by electrodermal activation [EDA]) depicted as a function of injury severity for each of the head injury groups**

Table 6 outlines all of the correlations between injury severity and initial testing physiological measures. As observed in Figure 7, significant linear relationships were observed between injury severity and initial testing physiological arousal[6] as measured by both EDA amplitude and pulse rate and demonstrating graduated decreases in arousal across head injury groups (from no-MHI to MHI followed by moderate TBI). EDA and pulse rate were also correlated, both showing similar arousal activation. EDA and pulse rate collectively accounted for 41.4% of the variability of injury severity (F (2, 83) = 29.30, $p < .001$). Moreover, a similar significant linear relationship was obtained for injury severity and PCS. As injury severity increases, so does the reporting of symptoms related to concussion. This is the case, despite the fact that **none** of the MHI subjects are 'complicated' (i.e., they do not report or complain of experiencing any persisting symptoms spontaneously). Interestingly, PCS was not significantly correlated with the physiological arousal measures of EDA and pulse rate (Figure 8).

[6] Linear relationships account for 41%, 10% and 12% of the variance for initial testing physiological arousal EDA, pulse and PCS, respectively.

Table 6:

Correlation matrix depicting the relationships between measures of injury severity (i.e., composite injury severity) and initial testing physiological arousal (EDA, Pulse, Salivary Cortisol)

Variables	Injury Severity	PCS	EDA	Pulse	Cortisol
Injury Severity	-	**.34***	**-.64***	**-.32***	.08
PCS Severity	-	-	-.15	.02	.05
Initial testing EDA Amplitude	-	-	-	**.37***	-.17
Initial testing Pulse Frequency	-	-	-	-	.03

Note: PCS and physiological correlations is completed with both injured and non-injured samples (results are the same when analyzed apart); **bold** and * reflects statistical significance ($p < .05$)

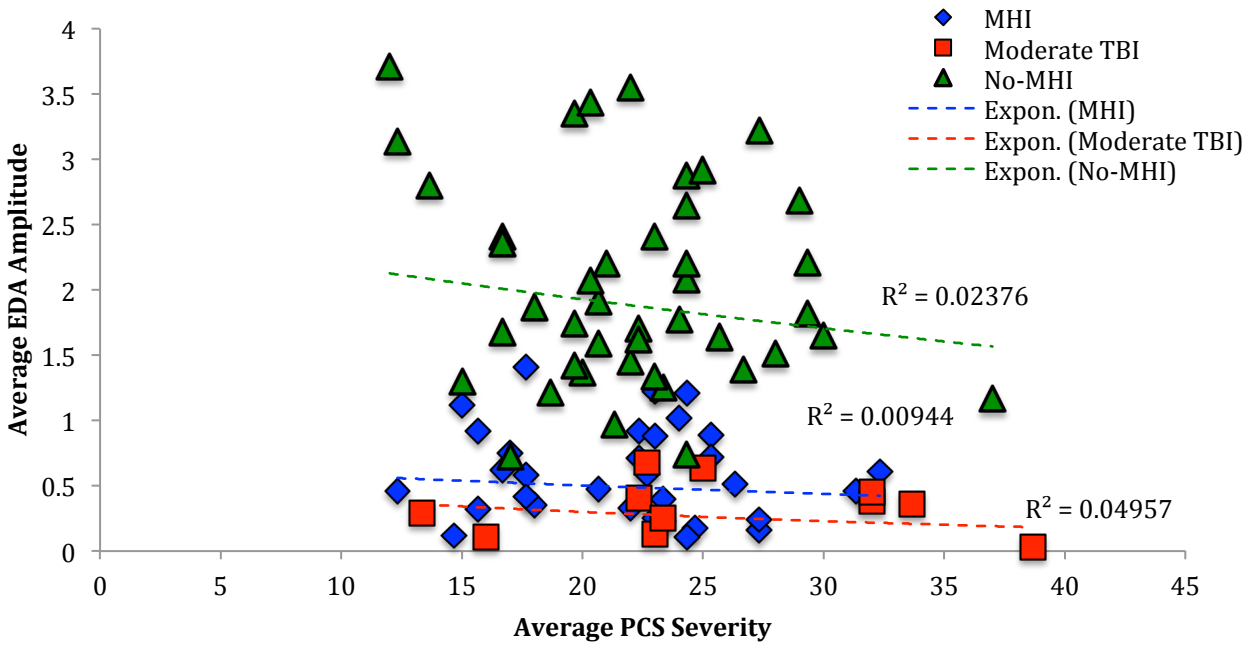

Figure 8. **Initial testing physiological arousal (as measured by electrodermal activation [EDA]) depicted as a function of post-concussive symptoms (PCS) severity and head injury status.**

Anticipatory arousal prior to making selections on the IGT and during feedback of selections was also assessed. Figure 9 depicts a summary of recordings taken at initial testing, just prior to task introduction, as well as in anticipation of making a card selection (averaged over the 8 seconds preceding each trial), and in response to reward and punishment feedback of a selection (averaged over the 4 seconds following a selection) across injury groups. A mixed model 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 4 (Arousal Measure [deck A, deck B, deck C, deck D]) ANOVA revealed a significant main effect of arousal condition ($F^{G\text{-}G}$ (1.81, 74.22) = 38.33, $p < .001$, $\eta_p^2 = .48$) and head injury status (F (2, 41) = 37.94, $p < .001$, $\eta_p^2 = .65$), as well as a significant interaction ($F^{G\text{-}G}$ (3.62, 74.22) = 22.05, $p < .001$, $\eta_p^2 = .52$)[7].

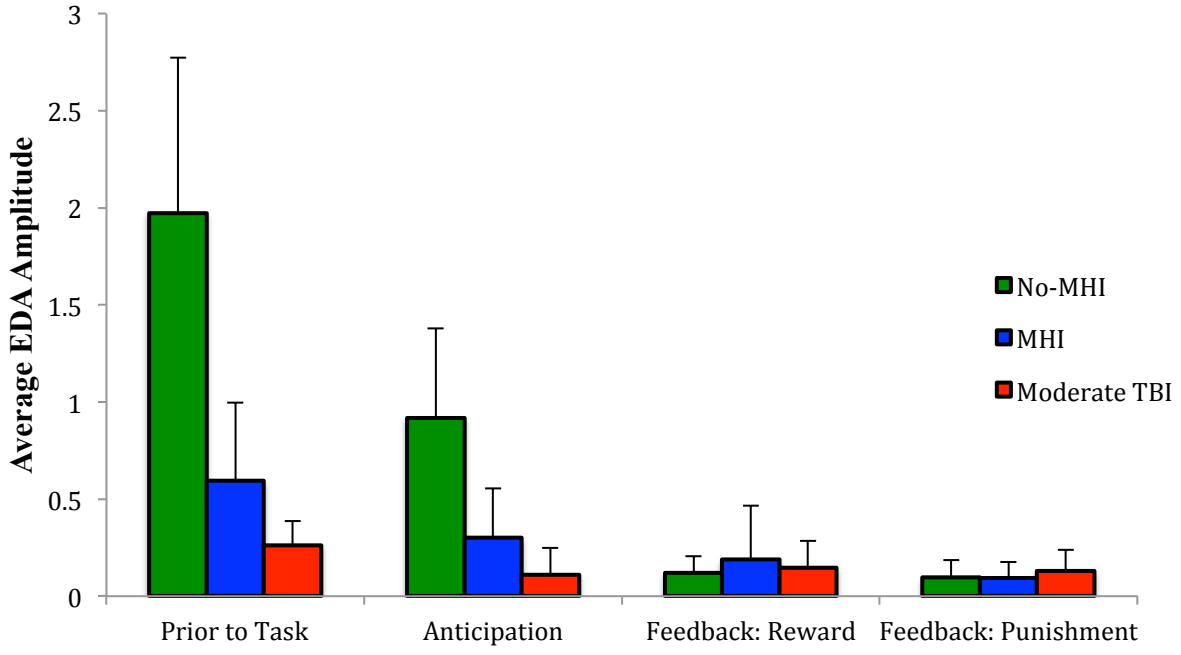

Figure 9: **Physiological arousal measured at initial testing/prior to task and in anticipation of, and in response (reward and punishment) to, selections made on the Iowa Gambling Task.**

[7] Sphericity could not be assumed, as Mauchly's W was significant. As a result, a Greenhouse-Geisser correction of degrees of freedom was made.

Follow-up one-way ANOVAs were conducted to explore the effects of the interaction. For the pre-task recording, an effect of injury status was observed (F (2, 41) = 32.31, $p < .001$, $\eta_p^2 = .61$). Post-hoc analyses illustrate that no-MHI differs from MHI and the moderate TBI groups respectively ($p < .001$), but the two injured groups do not differ ($p = .25$). Similarly, a main effect of injury status was found for anticipatory arousal (F (2, 41) = 19.66, $p < .001$, $\eta_p^2 = .49$), whereby, again, no-MHI differs from MHI and the moderate TBI groups ($p < .001$), but the injury groups did not differ from one another ($p = .27$). Conversely, there were no main effects for reward (F (2, 41) = .57, $p = .57$, ns) or punishment feedback (F (2, 41) = .41, $p = .67$, ns). These results support the prediction that differences in initial physiological arousal would persist into anticipatory physiological arousal prior to making selections on the IGT, but no differences in response to feedback would be observed.

Lastly, a 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 5 (Trial Blocks [trials 51-60, trials 61-70, trials 71-80, trials 81-90, trials 91-100]) × 2 (Arousal Manipulation status [pre-exposure, post-exposure]) mixed model ANOVA was conducted for each of the four decks of cards[8]. Starting with Deck A (Disadvantageous deck; Figure 10; Table 7 – see appendix A), a main effect of head injury status, trial blocks, and an interaction between head injury and trial blocks was observed. Follow-up analyses revealed that head injury status was not statistically significant, albeit a trend, for trials 51 to 60 (F (2, 40) = 2.57, $p = .09$), but was significantly different for trials 61 to 70 (F (2, 39) = 4.24, $p = .02$), 71 to 80 (F (2, 39) = 8.65, $p = .001$), 81 to

[8] This ANOVA includes the arousal manipulation pertinent to hypothesis 4, but also encompasses information for hypothesis one (anticipatory arousal for the group of participants who did not experience manipulation). This was done to conserve the number of statistical analyses done and keep type I error as low as possible.

90 (F (2, 40) = 5.86, p = .006), and 91 to 100 (F (2, 40) = 12.13, p < .001)[9]. Post-hoc analyses for these ANOVAs illustrated the same pattern for all five blocks of 10 trials, whereby no-MHI differed significantly from MHI and moderate TBI (p < .05), but these injured groups did not differ from one another (p > .05). To determine if anticipatory arousal varied across trials, three repeated measures ANOVAs were conducted on each of the three groups (no-MHI, MHI and moderate TBI), illustrating that arousal increased significantly as the trials progressed only for the no-MHI group ($F^{G\text{-}G}$ (2.29, 38.99) = 4.67, p = .01, η_p^2 = .22), but not for the MHI ($F^{G\text{-}G}$ (2.29, 38.09) = 1.36, p = .27, ns) or moderate groups ($F^{G\text{-}G}$ (1.30, 5.21) = 1.10, p = .37, ns)[10]. There was no effect of the Arousal Manipulation. In summary, the no-MHI illustrate a pattern of anticipatory physiological arousal measured by EDA that differs significantly (i.e., larger) from injured participants and this anticipatory arousal increases as the number of trials experienced increases for Deck A items.

[9] Varying degrees of freedom occurs for these analyses as not all participants selected from a particular deck during a 10-trial block.

[10] Paired samples t-tests revealed that trials 51-60 differed from the rest of the last 40 trials and trials 61-70 differed (albeit as a trend) in the same pattern.

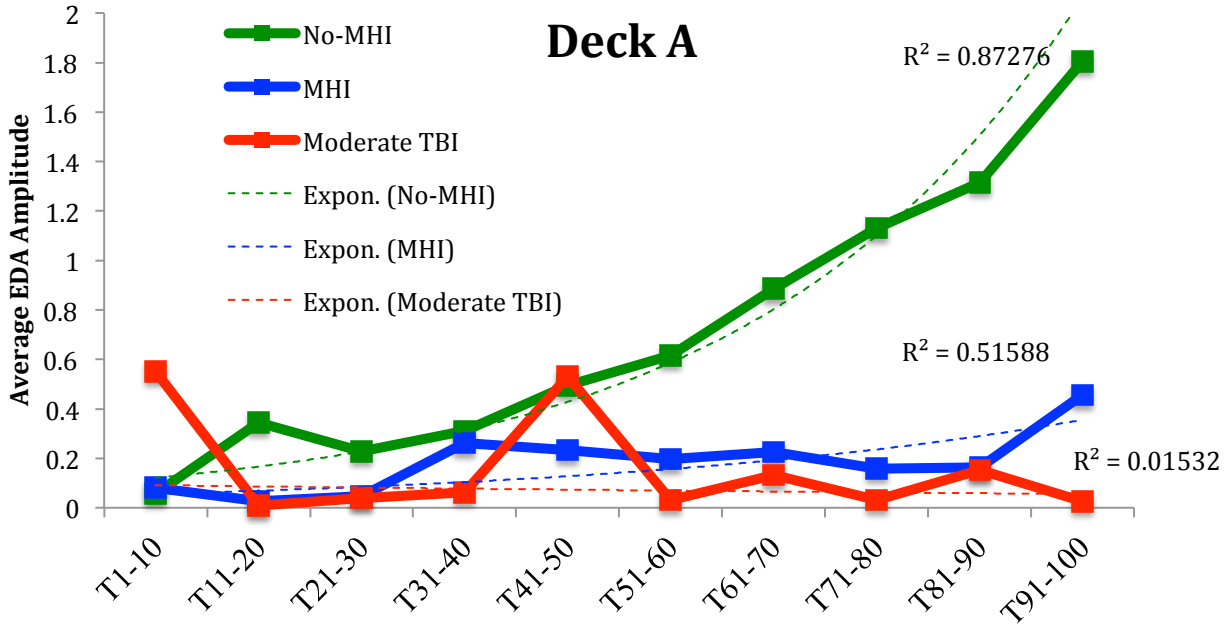

Figure 10: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck A on the Iowa Gambling Task across 10 trial blocks as a function of head injury status.**

Similar to Deck A, a significant main effect for head injury status and for trial blocks was found for Deck B (disadvantageous deck; Figure 11; Table 8 – see appendix A), but there was no significant interaction. Follow-up analyses revealed that head injury status was statistically significant for trial blocks 61 to 70 (F (2, 40) = 3.34, p = .045) and 81 to 90 (F (2, 40) = 5.46, p = .008), but not the remaining three blocks (i.e., 51 to 60 (F (2, 41) = 1.26, p = .29, ns), 71 to 80 (F (2, 39) = .76, p = .47, ns), 91 to 100 (F (2, 39) = .84, p = .44, ns))[11]. Post-hoc analyses for trial block 61 to 70 illustrated that no-MHI significantly differed from the moderate TBI group (p < .05), but the MHI group did not differ from either of the other two (p < .05). For trial block 81 to 90, however, the no-MHI group differed significantly from both the MHI and moderate TBI groups (p < .05), but the injured groups did not differ from one another (p > .05).

[11] Varying degrees of freedom occurs for these analyses as not all participants selected from a particular deck during a 10-trial block.

To explore the interaction of head injury status and trial blocks, three repeated measures ANOVAs were conducted, finding that, as a trend, arousal increased as the trials progressed for the no-MHI ($F^{G\text{-}G}$ (2.45, 41.70) = 2.00, p = .10, η_p^2 = .11; trend), and MHI ($F^{G\text{-}G}$ (1.81, 30.84) = 3.14, p = .06, η_p^2 = .16; trend) groups, but not for the moderate group (F (4, 5.12) = .43, p = .79, ns). In summary, the no-MHI and MHI groups illustrate a pattern of anticipatory physiological arousal measured by EDA that is greater than that of the moderate TBI group and this anticipatory arousal increases as the number of trials increases for Deck B.

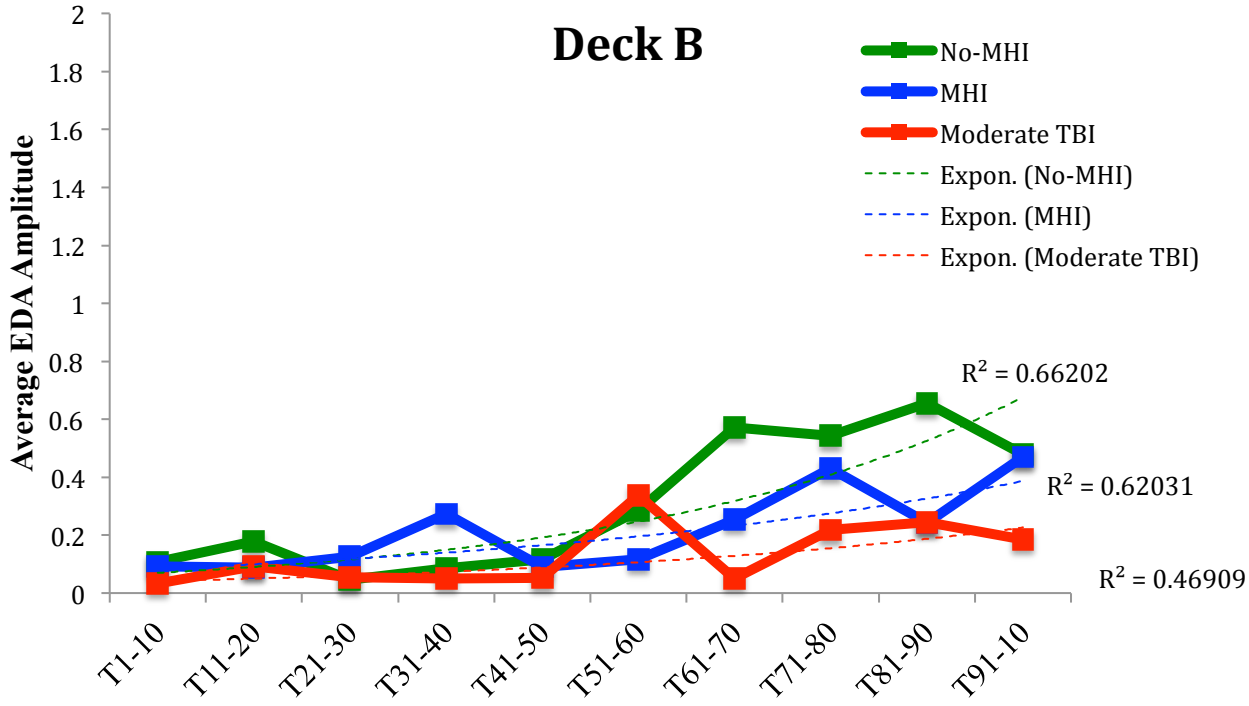

Figure 11: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck B on the Iowa Gambling Task across 10 trial blocks as a function of head injury status.**

For Deck C (advantageous deck; Figure 12), a significant main effect of head injury status and an interaction between head injury and trial blocks was found for anticipatory arousal (Table 9 – see appendix A). Follow-up analyses revealed that head injury status was statistically significant for trial blocks 51 to 60 (F (2, 41) = 3.76, p = .032), 61 to 70 (F (2, 40) = 4.06, p =

.025), 71 to 80 (F (2, 41) = 2.64, p = .083, trend), and 91 to 100 (F (2, 41) = 44.11, p < .001) [12]. Trial blocks 81 to 90 were not found to be significant (F (2, 41) = 1.49, p = .24, ns)[13]. Post-hoc analyses for all of these trial blocks illustrated the same pattern, no-MHI significantly differed from the MHI and moderate TBI group (p < .05), but the two injury groups were not found to differ (p > .05).

To explore the main effect of trial blocks, three repeated measures ANOVAs were conducted and, in contrast to the results for the previous two Decks (A and B), none of the groups differed as the trials progressed (no-MHI [$F^{G\text{-}G}$ (1.19, 22.56) = 1.64, p = .22, ns), MHI ($F^{G\text{-}G}$ (1.80, 28.82) = 0.30, p = .72, ns) and moderate groups ($F^{G\text{-}G}$ (1, 6.10) = 2.00, p = .21, ns) respectively. In summary, the no-MHI group illustrates a pattern of anticipatory physiological arousal that is greater than that of MHI and the moderate TBI groups, and this relationship remains relatively constant across the trials for Deck C.

[12] Varying degrees of freedom occurs for these analyses as not all participants selected from a particular deck during a 10-trial block.

[13] Block trials 81 to 90 have a large standard deviation of 2.15, so despite having the highest mean this is being influenced by a select number of participants and as a result is not statistically significant.

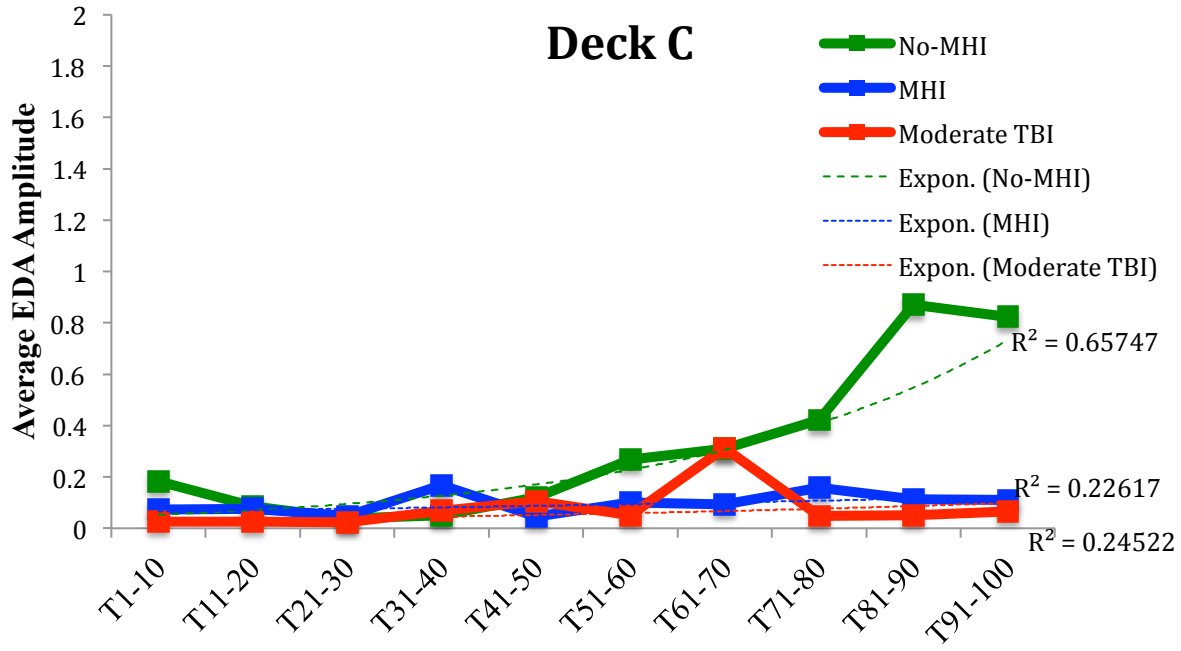

Figure 12: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck C on the Iowa Gambling Task across 10 trial blocks as a function of head injury status.**

For Deck D (advantageous deck; Figure 13), a significant main effect of head injury status and an interaction between head injury and trial blocks was found for anticipatory arousal (Table 10 – see appendix A). Follow-up ANOVAs revealed that head injury status differed for trial blocks 51 to 60 (F (2, 41) = 2.28, p = .12; trend), 81 to 90 (F (2, 40) = 3.93, p = .03), and 91 to 100 (F (2, 40) = 3.63, p = .04)[14], but not trial blocks 61 to 70 (F (2, 41) = .68, p = .51, ns), and 71 to 80 (F (2, 41) = 1.77, p = .18, ns). Post-hoc analyses for trial blocks 51 to 60 and 91 to 100 revealed that no-MHI differed from MHI (p < .05), but not the moderate TBI group. For trial blocks 81 to 90, the previously reported pattern emerged; no-MHI was found to have greater anticipatory physiological arousal than the MHI and moderate TBI groups (p < .05), but the two injury groups did not differ from one another (p > .05).

[14] Varying degrees of freedom occurs for these analyses as not all participants selected from a particular deck during a 10-trial block.

To explore the interaction between head injury status and trial blocks on anticipatory arousal, three repeated measures ANOVAs were conducted and, similar to the results found for Decks A and B, it was found that no-MHI had a pattern of steadily increasing arousal as the trials progressed ($F^{G\text{-}G}$ (2.52, 47.67) = 8.42, $p < .001$, $\eta_p^2 = .31$). No changes across block trials were observed for the MHI ($F^{G\text{-}G}$ (1.51, 25.70) = 1.42, $p = .24$, ns) or the moderate TBI groups ($F^{G\text{-}G}$ (4, 16) = 1.03, $p = .42$, ns). In summary, the no-MHI illustrate a pattern of anticipatory physiological arousal that is higher than that of MHI and the moderate TBI groups, but this relationship increases with increasing trial blocks.

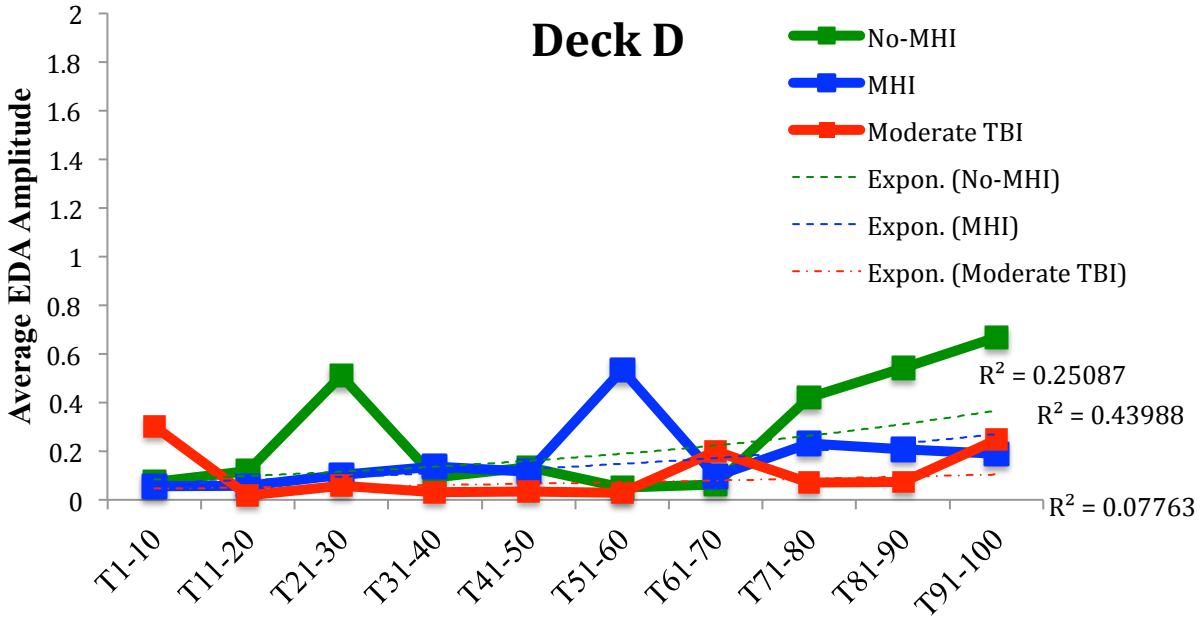

Figure 13: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck D on the Iowa Gambling Task across 10 trial blocks as a function of head injury status.**

Thus, it was hypothesized that physiological arousal at initial testing (measured by EDA, pulse and cortisol) and in anticipation of making a selection on the IGT (measured by EDA) would vary as a function of injury status, with individuals who have a moderate/ TBI presenting with the least arousal; and those reporting a MHI will have lower arousal as compared to those

without a history of MHI. Conversely, no difference would be observed in physiological arousal in response to feedback on the IGT. Lastly, it was predicted that initial testing arousal would vary as a function of injury severity indictors. It was found that initial testing arousal (as measured by EDA and pulse) did differ as a function of injury status, whereby no-MHI had greater initial arousal than those with injuries, but the injury groups did not differ significantly, (albeit, descriptively, the average performance was in the predicted direction - MHI had greater arousal than the moderate TBI group). Measures of salivary cortisol taken during testing were found to produce a significant trend in the opposite direction, with the moderate TBI group having the greatest levels, followed by the MHI and non-MHI, respectively.

The hypothesis was further supported by a significant linear relationship between initial testing arousal (as measured by EDA and pulse) and injury severity. Lastly, a pattern of underarousal was observed for the MHI and the moderate TBI group relative to the no-MHI group in anticipation of making a selection, although the two injury groups did not differ. This pattern was maintained across the four decks of cards, as illustrated for the last 50 trials of testing. For three out of the four decks of cards (A, B and D), a pattern was obtained showing that the no-MHI group's heightened anticipatory arousal increased as trials progressed, whereas the other two injury groups produced minimal changes in arousal (with the exception that the MHI group did have increasing arousal as trials progressed for Deck B). For Deck C, the main effect of injury status was observed as well, but this relationship did not increase with the progression of trials. Lastly, no differences in the subjects' physiological response to feedback was observed across the groups.

Replication - Hypothesis II: IGT Performance

It was predicted that IGT performance would vary as a function of injury status, whereby

persons with moderate TBI would be slower at transitioning from disadvantageous selections to advantageous selections, than individuals who report mild head injuries who, in turn, will be slower to transition than non-injured participants. Figure 14 depicts the ratio of advantageous (C and D) to disadvantageous (A and B) selections across 10 trial blocks. A 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 5 (Trial Blocks [trials 51-60, trials 61-70, trials 71-80, trials 81-90, trials 91-100]) × 2 (Arousal Manipulation status [pre-exposure, post-exposure]) mixed model ANOVA was conducted to examine the whether learning rates on the IGT varied on the last 50 trials as a function of head injury status (Table 11 – see appendix A)[15]. A significant main effect of head injury status and an interaction between head injury status and trial blocks was observed (Table 11).

To explore the main effect of head injury status and interaction between head injury status and trial blocks, follow-up ANOVAs were conducted on each of the five trial blocks, finding that trials 51 to 60 (F (2, 41) = 5.99, p = .005) and 91 to 100 (F (2, 41) = 5.02, p = .011) differed significantly. A pattern similar to that found for anticipatory physiological arousal was observed, whereby no-MHI illustrated a higher rate of advantageous-to-disadvantageous selections relative to those with a MHI and a moderate TBI ($p < .05$). The injured groups were not found to differ from one another ($p > .05$). Moreover, three repeated measures ANOVAs were conducted to evaluate the interaction between head injury status and trials blocks. A trend for the main effect was observed for the no-MHI group only as the trials progressed (F (4, 76) = 2.01, p = .10, η_p^2 = .10; trend); no differences were observed for the two other groups (MHI: F (4, 68) = .33, p = .86, ns; moderate TBI: F (4, 20) = .21, p = .93, ns)). For the no-MHI group, the

[15] This ANOVA includes the arousal manipulation pertinent to hypothesis 4, but also encompasses information for hypothesis two (IGT performance for those who did not experience the arousal manipulation). This was done to conserve the number of statistical analyses done and keep type I error as low as possible

ratio of advantageous-to-disadvantageous selections during trials 51 to 60 was higher than that found for trials 61 to 70; and the ratios for trials 61 to 70 and 71 to 80 were significantly lower than that of trials 91 to 100 ($p < .05$). Thus, as predicted, no-MHI participants selected more advantageous selections (relative to disadvantageous) as compared to the MHI and moderate TBI groups, but this occurs only at two peaks (trials 51 to 60 and 91 to 100). The prediction that the two injury groups would differ was not supported by the results[16]. Lastly, as predicted, no-MHI participants illustrated a greater transition from disadvantageous selections to advantageous selections, however it was not observed to be consistent (i.e., there was no difference among groups from trials 61 to 90) and it was non-linear in nature[17].

Figure 15 depicts the relationship between overall T-scores[18] on the IGT at the completion of all 100 trials and measures of injury severity for each of the groups with head injury. A linear relationship was observed ($r = -.22$, $p = .03$) illustrating that IGT performance decreases as injury severity increases, demonstrating that one is less likely to do well on measures of decision-making when injuries are more severe[19].

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[16] Repeated measures ANOVAS were conducted across all 10 trial blocks for each of the groups (i.e., no-MHI, MHI, moderate TBI), finding that main effects of increased selections on advantageous (relative to disadvantageous) decisions were observed for no-MHI (F (9, 171) = 5.08, $p < .001$, $\eta_p^2 = .21$) and MHI (F (9, 153) = 1.93, $p = .05$, $\eta_p^2 = .10$), but not for the moderate TBI group (F (9, 45) =.24, $p = .99$, ns)

[17] Trials 41 to 50 were tested to see if they differed significantly as a function of head injury status; a trend was observed (F (4, 41) = 2.37, $p = .10$, trend), with no-MHI differing from the moderate TBI group only ($p < .05$) in post-hoc tests.

[18] T-scores (i.e., converted normative values such that they have a mean of 50 and a standard deviation of 10) reflect overall performance across all 100 trials on the IGT and are based on norms derived by Bechara (2007).

[19] The same pattern of results is observed for post-concussive symptoms (PCS).

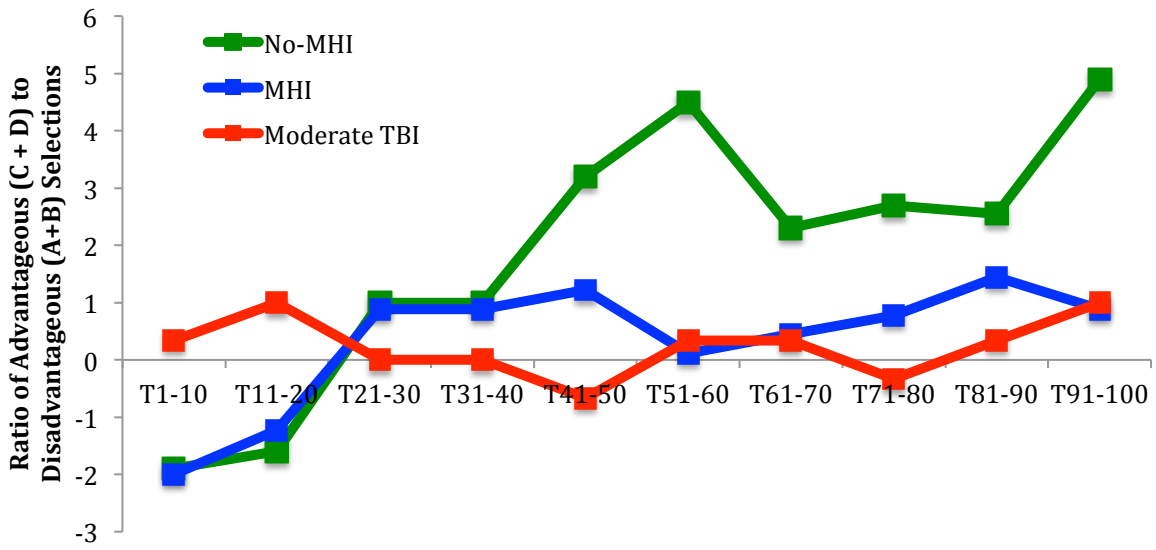

Figure 14: **Ratio of advantageous (C + D) to disadvantageous (A + B) selections on the Iowa Gambling Task (IGT) as a function of head injury status across 10 trial blocks**

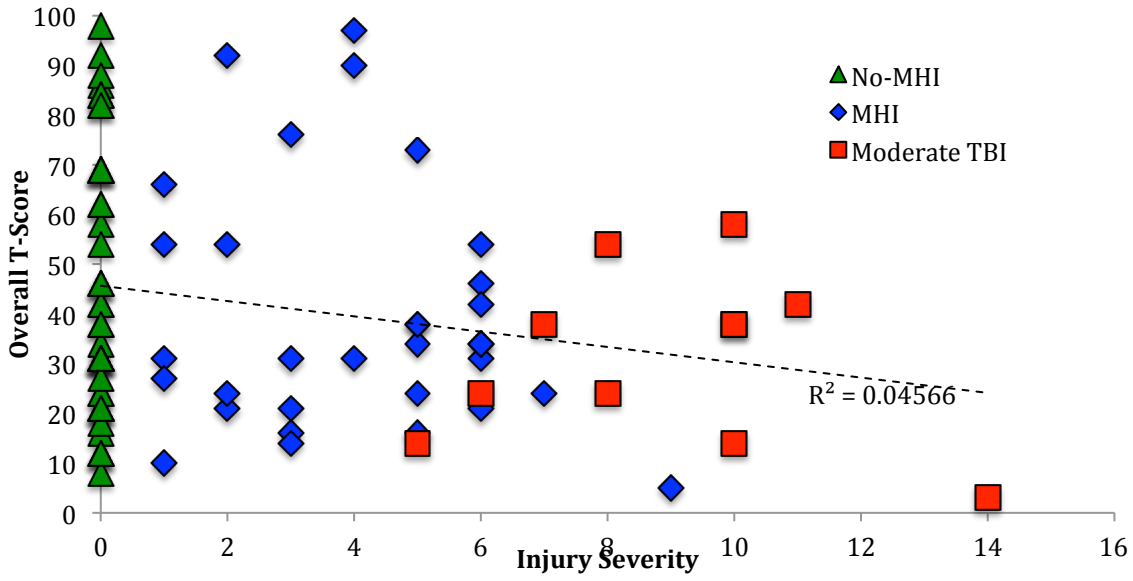

Figure 15. **Overall T-Score on Iowa Gambling Task at task completion depicted as a function of injury severity and head injury status**

Lastly, it was predicted that the rate of return to a punishing (disadvantageous) selection would be faster (i.e., fewer trials in between returning to a selection following being punished by that selection previously) as a function of injury severity (i.e., individuals with moderate TBI, followed by those reporting a MHI and lastly non-injured healthy subjects). Figure 16 depicts the

number of selections taken following a punishment before returning to that same deck (averaged as a function of disadvantageous [A and B] and advantageous [C and D] selections). A 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 2 (deck type [disadvantageous, advantageous]) × 2 (Arousal Manipulation status [pre-exposure, post-exposure]) mixed model ANOVA for rate of return produced a significant main effect of head injury status, deck type, and an interaction between head injury status and deck type (Table 12 – see appendix A)[20]. Follow-up ANOVAs indicated that the return rate for disadvantageous selections (F (2, 40) = 5.82, p = .006), but not advantageous selections, differed (F (2, 41) = .52, p = .52, ns) as a function of head injury status. As observed relatively consistently for physiological arousal and IGT performance, post-hoc analyses illustrate that for the disadvantageous rate of return, no-MHI was found to significantly differ from the MHI and moderate TBI groups ($p < .05$), but the two injury groups were not found to differ from one another ($p > .05$).

To investigate the interaction between deck type and head injury status, paired samples t-tests were conducted for all three injury groups, revealing that the rate of return was significantly slower for disadvantageous selections (i.e., more intervening trials before selecting from the punishing deck on a subsequent trial) for the no-MHI (t (18) = 3.22, $p < .005$ CI [1.75, 8.31]), but not the MHI (t (18) = 3.22, p = .005 CI [1.75, 8.31]) or moderate TBI (t (17) = .406, p = .69 CI [1.21, 1.78]) groups. Both the MHI and moderate TBI groups illustrate a faster rate of return to a deck that was previously punishing compared to the no-MHI group, whereas no differences were obtained for advantageous selections. Lastly, figure 17 depicts a linear relationship between rate of return to a selection following a punishing trial on the IGT and measures of injury

[20] Two non-MHI participants were removed from this analysis as their return rates following punishment were extreme outliers (5+ standard deviations away from the mean; scores of 72 and 74). The results of this ANOVA did not change as a result of removal of these outliers.

severity for each of the groups with head injury (r = -.29, p = .007). More specifically, as participant's injury severity increases, how quickly (i.e., number of trials) they return to a previous selection following being punished by that selection in the past decreases.

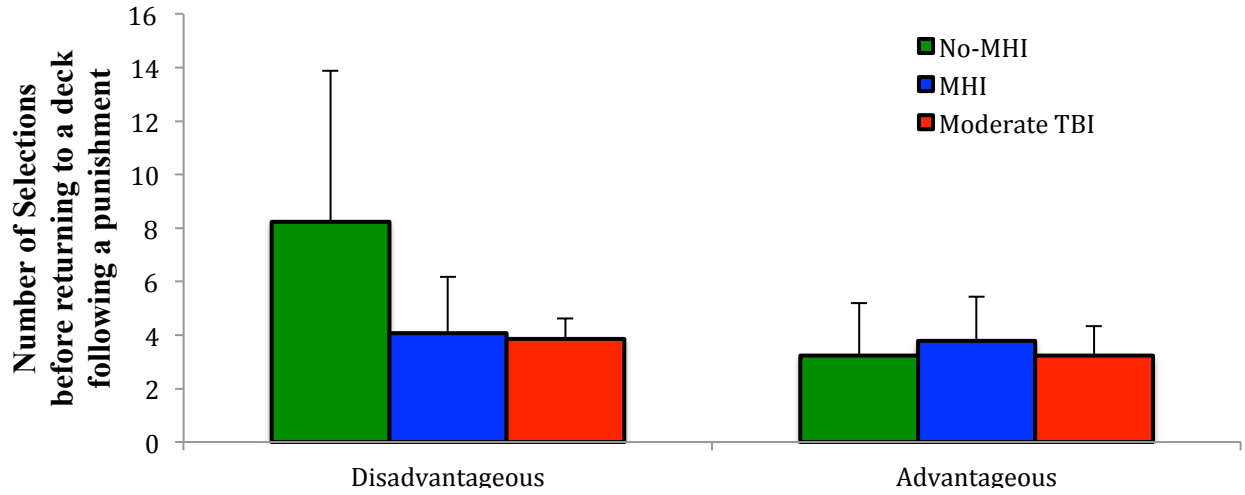

Figure 16. **Number of 'other deck' selections made following a punishment before returning to the punishing deck (grouped by disadvantageous [A and B] and advantageous [C and D] decks) as a function of head injury status**

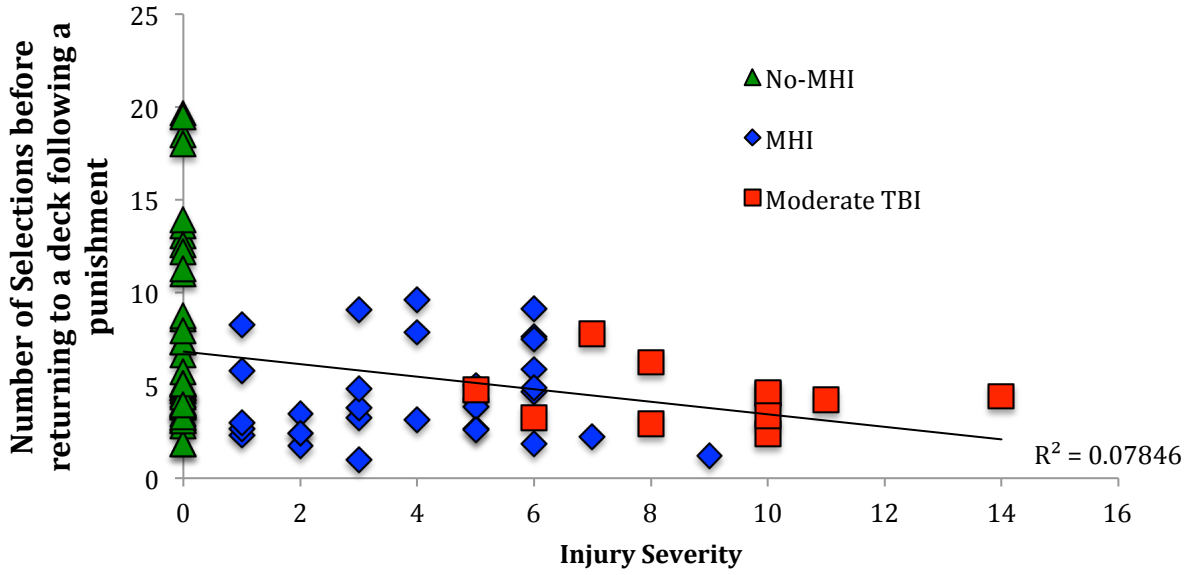

Figure 17. **The relationship between the number of 'other deck' selections made following a punishment before returning to the punishing deck (grouped by disadvantageous [A and B] and advantageous [C and D] decks) as a function of injury severity, grouped by head injury status**

In summary, as predicted, moderate TBI and MHI were found to have slower rate of transitioning from disadvantageous selections to advantageous selections and faster rates of returning following punishment from a disadvantageous selection (relative to an advantageous selection). However, for both of these findings, there were no observed differences between the injured groups. An unexpected finding for the moderate TBI group was that that they did not display any variations in their decision-making pattern (i.e., the ratio of advantageous to disadvantageous selections did not change across the 100 trials). Lastly, indices of decision-making changed as a function of injury severity, whereby increasing injury severity was associated with decreasing performance.

Injury Severity and Linking Physiological Arousal and Decision-making

As cited above, injury severity was found to be predictive of physiological arousal and decision-making performance. It was also found that the relationships between physiological arousal measured by EDA and decision-making performance were found to be significant (Figures 18 and 19), specifically for IGT T-scores ($r = .24$, $p = 0.03$) and the rate of return to a deck that was previously punishing ($r = .33$, $p = .002$). Both of these findings illustrate that as participants' physiological arousal increases, their decision-making performance improves and their rate of return to a previously punishing decision decreases (i.e., increased time spent selecting from other decks following punishment), indicating a greater response/sensitivity to the consequences of previous selections. Furthermore, in both figures, a graduated change to good decisions is observed across injury groups, illustrating the continuum of head injury severity.

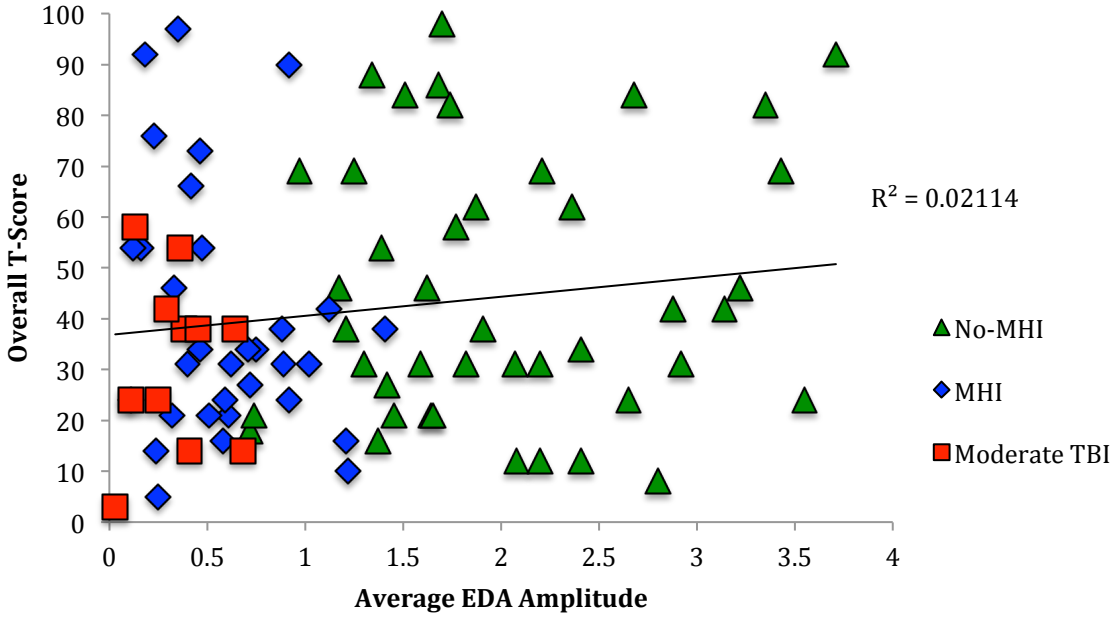

Figure 18. **The relationship between overall T-Score on Iowa Gambling Task at task completion depicted as a function of physiological arousal (as measured by EDA) and head injury status**

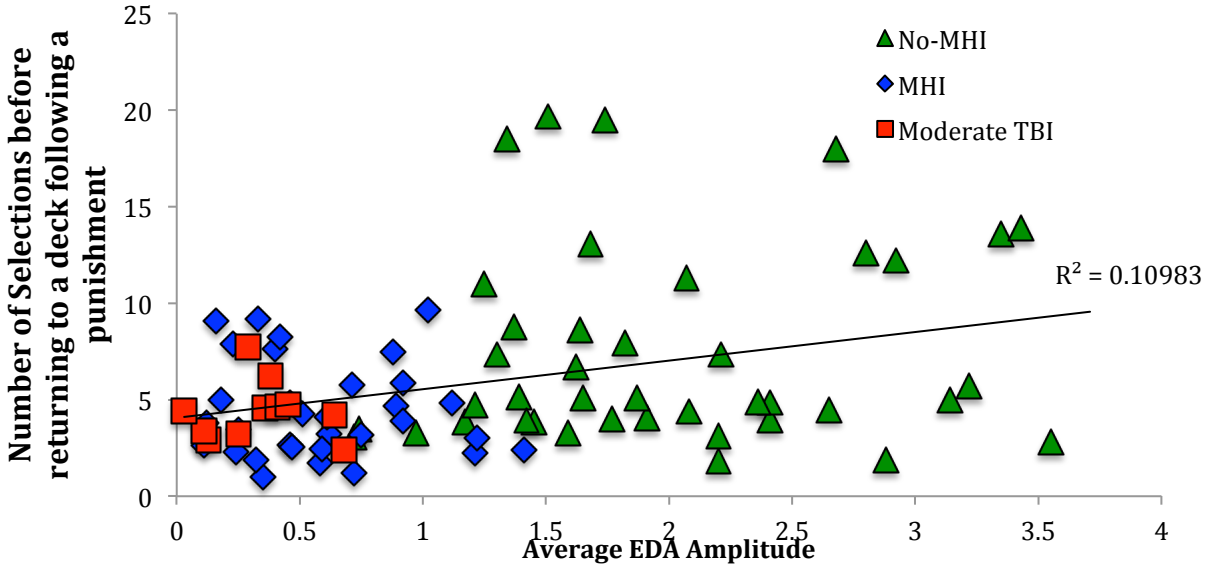

Figure 19. **The relationship between the number of 'other deck' selections made following a punishment before returning to the punishing deck (grouped by disadvantageous [A and B] and advantageous [C and D] decks) as a function of physiological arousal (measured by EDA), grouped by head injury status**

To further investigate this further, a post-hoc meditational analysis was conducted to determine whether physiological arousal mediated the relationship between injury severity and decision-making performance. Two mediation analyses were conducted to explore the nature of the relationship between injury severity with IGT overall T-score and rate of return to a previously punishing decision. A mediation relationship was not found for physiological arousal between injury severity and overall IGT T-score. However, complete mediation was found for injury severity and rate of return to a previously punishing decision by physiological arousal (Figure 20). When injury severity and physiological arousal measured by EDA were regressed on rate of return, physiological arousal continued to be predictive of decision-making (accounting for 3.7% of variability; pathway b in Figure 20), whereas injury severity ceased to be predictive (pathway c' in Figure 20). This implies that the mechanism by which injury severity predicts decision-making performance occurs through changes to physiological arousal.

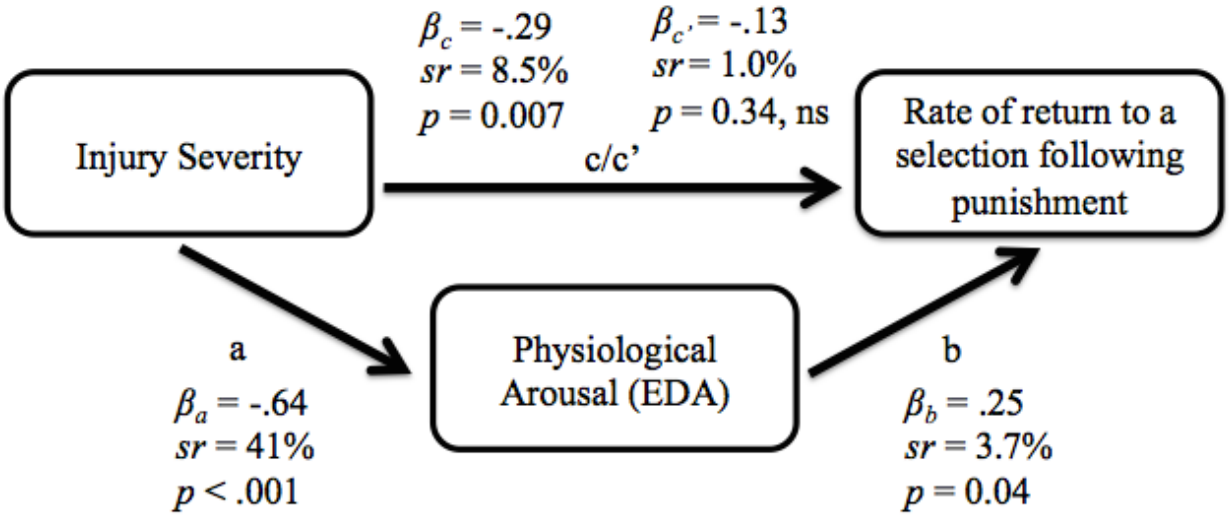

Figure 20: **A mediation relationship between injury severity and the number of 'other deck' selections made following a punishment before returning to the punishing deck for disadvantageous selections by physiological arousal as measured by EDA**

Replication - Hypothesis III: Explicit Knowledge & IGT

The overall number of selections made from each of the four decks of cards for each of the three groups is depicted in Figure 21[21]. This figure can be compared to the overall ratings of preference for each of the Decks in Figure 21. A 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 4 (deck [deck A, deck B, deck C, deck D]) × 2 (Arousal Manipulation status [pre-exposure, post-exposure]) mixed model ANOVA assessing overall explicit deck preference (following completion of the IGT) revealed a significant main effect of deck, head injury status (trend), and an interaction between deck and head injury status (Table 13 – see appendix A). Follow-up analyses demonstrated that only Decks C (F (2, 41) = 5.29, p = .009) and D (F (2, 41) = 3.05, p = .06, trend) varied as a function of injury status (i.e., Decks A - F (2, 41) = .73, p = .49, ns; and B - F (2, 41) = 1.70, p = .20, ns, did not). Post-hoc analysis revealed that the moderate TBI group preferred Deck C (advantageous deck) less than the MHI group and no-MHI groups and preferred Deck D (advantageous deck) less than the MHI group.

To explore the interaction between head injury status and type of deck with respect to participants' ratings of preference for each of the four decks, four repeated measures ANOVAs were conducted (Figure 22). The no-MHI (F (2.17, 41.27) = 9.86, p < .001, η_p^2 = .34) and MHI (F (2.23, 76) = 6.61, p = .003, η_p^2 = .28) groups produced significantly different ratings for their preferences of the decks, whereas the moderate TBI group did not (F (3, 15) = 1.93, p = .21, ns). Post-hoc analyses illustrated that advantageous decks (A and B) differed from disadvantageous decks (C and D; p < .05), but they did not differ from one another (i.e., A and B did not differ from each other, nor did C and D). Thus, for all three groups, their preferences of the decks did match their behaviour. No-MHI and MHI groups illustrated a transition from disadvantageous to

[21] No main effect of head injury status or interactions. The only differences observed was that Deck A was selected significantly less than the other decks.

advantageous decks and reported a preference for advantageous selections, whereas moderate TBI did not differentiate between decks according to their trial selections which was also reflected by their ratings of explicit preference. These findings were not supportive of the hypothesis.

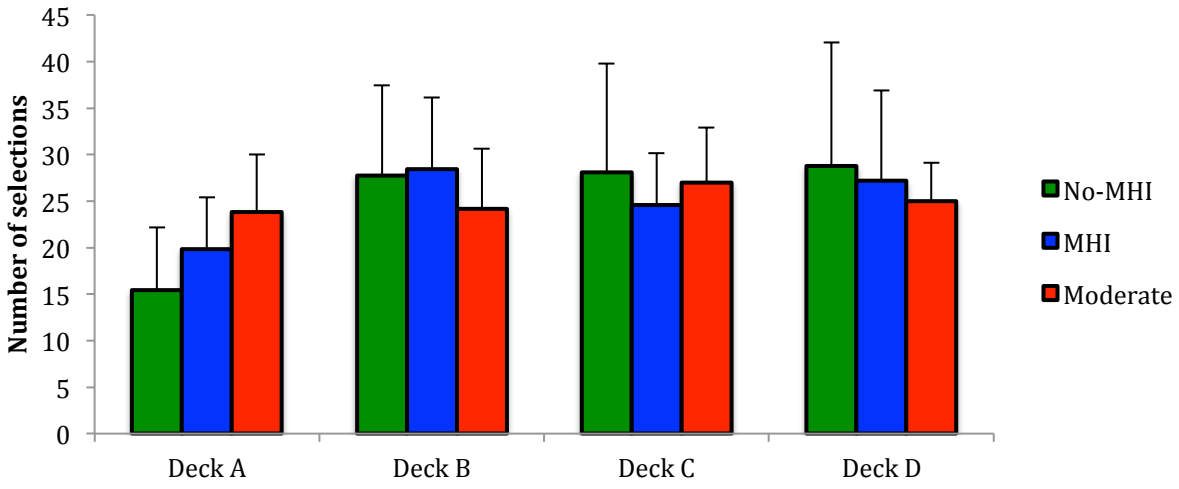

Figure 21: **Number of selections as a function of each of the four decks of cards on the Iowa Gambling task and head injury status**

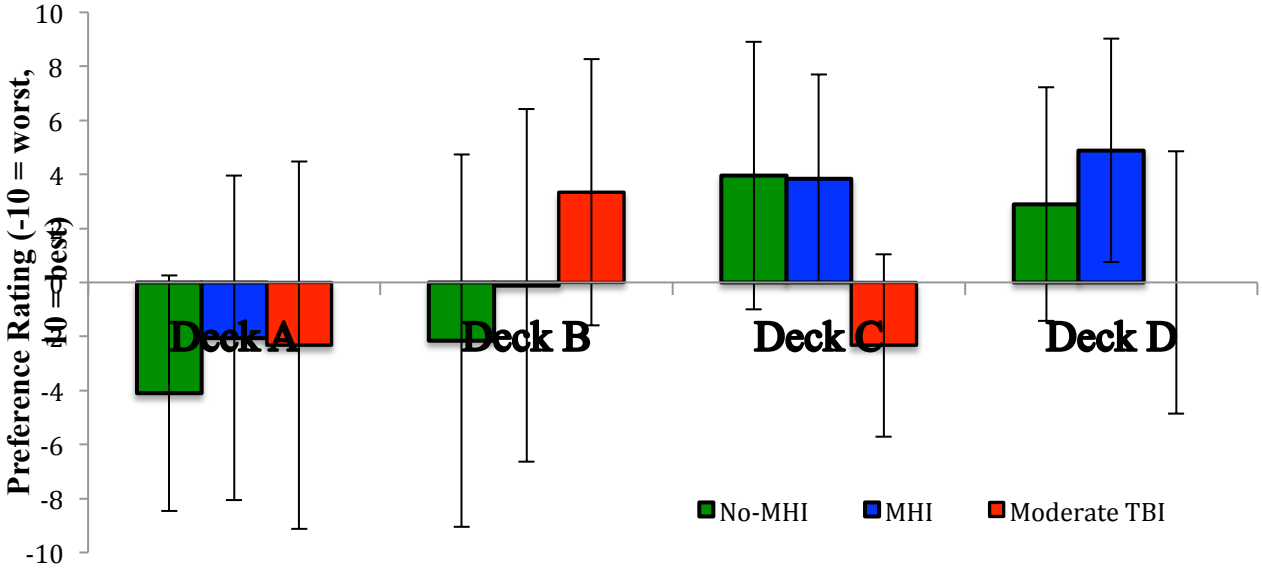

Figure 22: **Explicit knowledge measured by preference rating (-10 = worst, 10 = best) for each deck on the Iowa Gambling Task as a function of head injury status**

Whereas the ratings of preference of IGT decks at the end of the task mimicked the participant's behavioural data, explicit predictions of expected earnings for each deck (as calculated by Maia & McClelland, 2004) illustrated a different pattern of results (Figure 23; Table 14 – see appendix A). A 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 4 (deck [deck A, deck B, deck C, deck D]) × 2 (Arousal Manipulation status [pre-exposure, post-exposure]) mixed model ANOVA was conducted produced a significant main effect of type of deck, but no effect of head injury status or interactions. As follow up, paired sampled t-tests were conducted to examine if the decks differed from one another. It was found that Decks A and B differed from C and D (A relative to C: t (19) = 2.47, p = .02 CI [44.31, 532.99]; A relative D: t (19) = 2.27, p = .04 CI [35.22, 874.28]; B relative to C: t (19) = 1.72, p = .10 CI [-68.52, 697.42], trend; B relative to D: t (18) = 2.28, p = .04 CI [38.60, 922.50]). Participants estimated they would win more money for disadvantageous decks (A and B), relative to advantageous decks (C and D), despite both no-MHI and MHI groups producing a transition from disadvantageous to advantageous when actually making trial-to-trial selections. This contrasting behaviour (explicit monetary predictions in favour of Decks A and B post-IGT, but performance selections during the IGT showing a bias moving away from Decks A and B and towards behavioural choices in favour of Decks C and D – in the case of no-MHI and MHI, and no differential trial-to-trial bias at all in the case of the moderate TBI group) supports the prediction that there would be a dissociation between what participants know about the decks of cards at the end of the task and how they behave.

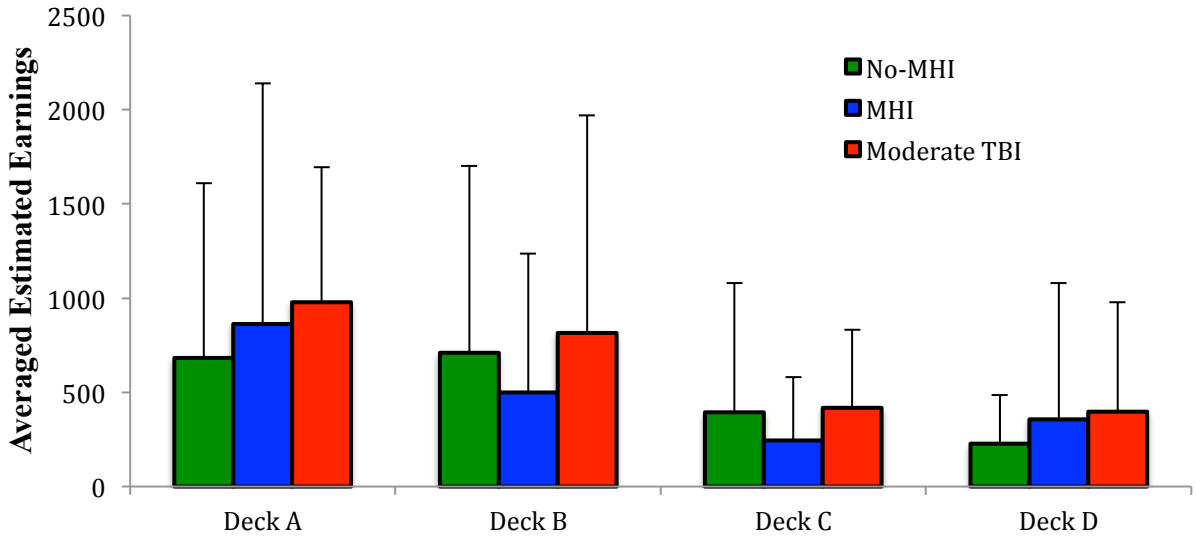

Figure 23: **Explicit knowledge as reflected by extrapolated total earnings (based on 100 trials per deck) calculated using Maia & McClelland's (2004) question set (i.e., estimates amount and frequency of wins and losses over 10 trials) as a function of head injury status**

Lastly, it was predicted that there would be no linear relationship between participant's knowledge (i.e., preference and expected earnings) and their behaviour. Table 15 illustrates the relationships between preference and expected earnings, and total selections of each of the four decks of cards on the IGT, as a function of head injury status. For the no-MHI group, a significant positive linear relationship was found between preference ratings for Decks C and D and their respective total number of trial-to-trial selections, whereby higher ratings of preference was associated with a higher number of selections. For the MHI group, positive linear relationships were observed between preferences for Decks B and D and their respective number of selections. No relationships were observed for moderate TBI preference. There were no relationships found between the predicted expected earnings for each deck and the frequency at which they selected that deck (Table 15).

Table 15

Correlation matrix depicting the relationships between measures of explicit knowledge (i.e., preference of decks and estimated earnings had they chosen a deck for all 100 trials) as a function of head injury status

Explicit Knowledge Measure	Total Selections of Deck A	Total Selections of Deck B	Total Selections of Deck C	Total Selections of Deck D
Preference	Deck A	Deck B	Deck C	Deck D
No-MHI	.06	.22	**.38***	**.37***
MHI	.03	**.43***	-.12	**.31***
Moderate TBI	-.47	.14	-.18	-.30
Estimated Winnings of each Deck (per 100 trials)	Deck A	Deck B	Deck C	Deck D
No-MHI	-.01	-.05	.00	-.14
MHI	-.09	.12	-.25	-.12
Moderate TBI	.28	.08	-.02	.01

Note: **Bolded** and * reflects statistical significance ($p < .05$)

In summary, it was found that, regardless of their head injury status, participants' preferences of the decks did match their behaviour and some linear relationships were observed between preference and frequency of deck selection (predominately for advantageous decks). As a result, the prediction regarding preference as not supported. However, it was also found, as expected, that participants overestimated their earnings for the disadvantageous decks (A and B) relative to the advantageous decks (C and D), despite both no-MHI and MHI groups illustrating a transition from disadvantageous to advantageous deck selections. Lastly, there was no observed relationship between expected earnings and frequency of selections on the IGT. This supports the

hypothesis that there may be a dissociation between the participant's explicit knowledge regarding preference and expected earnings and the participant's implicit behaviour for both no-MHI, and injured, groups.

Replication - Hypothesis IV: Manipulating Arousal

It was predicted that emotionally-evocative imagery (i.e., arousal manipulation) will produce a corresponding increase in physiological arousal (i.e., as reflected by increases in EDA and pulse). Moreover, it was predicted that increases in arousal will benefit the decision-making performance on the IGT for those reporting a head injury (across the injury severity continuum) in two ways: by decreasing the number of trials required for transitioning from high risk to low risk choices; and by increasing the number of trials before returning to high risk decision following punishment feedback. Lastly, it was predicted that persons who do not report a history of MHI may be disadvantaged by increased arousal, should their levels of arousal introduce distress.

Figure 24 represents physiological arousal as measured by electrodermal activation measured at six times during the testing session, including before and after the arousal manipulation. A 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 6 (Time [initial testing, recording 2, pre-manipulation, post-manipulation, recording 5, end of testing session]) mixed model ANOVA for EDA as a function of head injury status was conducted, and produced a significant main effect for head injury status, time and an interaction between head injury status and time (trend; Table 16 – see appendix A). Post-hoc analyses reveal that, similar to previously discussed arousal data, the no-MHI group had significantly greater arousal than the MHI and moderate TBI groups ($p < .001$), but the two injury groups did not differ from one another.

Follow-up repeated measures ANOVAs examined the interaction between head injury

status and time, for each of the head injury groups, and demonstrated that arousal changed as a function of time for each (no-MHI: $F^{G\text{-}G}$ (1.57, 64.22) = 3932.18, $p < .001$, $\eta_p^2 = .99$; MHI: $F^{G\text{-}G}$ (2.02, 62.49) = 4597.72, $p < .001$, $\eta_p^2 = .99$; moderate TBI: $F^{G\text{-}G}$ (1.69, 16.88) = 1112.02, $p < .001$, $\eta_p^2 = .99$). Post-hoc analyses of this illustrates that post-manipulation measures of arousal (the recording that takes place immediately following the arousal manipulation) are significantly higher than initial testing measures, recording 2, pre-manipulation and the end of the testing session for all injury groups ($p < .05$). However, for the no-MHI group, no differences were found between pre-manipulation and post-manipulation ($p = .51$), whereas for the injury groups, the subjects returned to initial testing measures by recording 5 ($p < .05$). The manipulation of arousal by emotionally evocative stimuli lasted longer for the no-MHI participants relative to the injury groups (thus the injury groups returned to initial testing levels [i.e., underarousal] more quickly). As a result, an increase in physiological arousal was observed, as measured by EDA, for all groups in response to the emotionally evocative stimuli, but these changes to arousal were short-lived for injury groups, relative to the no-MHI group. Furthermore, the no-MHI group differed from injured groups, having greater EDA and this was maintained at all time epochs. The two injury groups did not differ (but the descriptive means continued to be in the expected direction, i.e., MHI > moderate TBI).

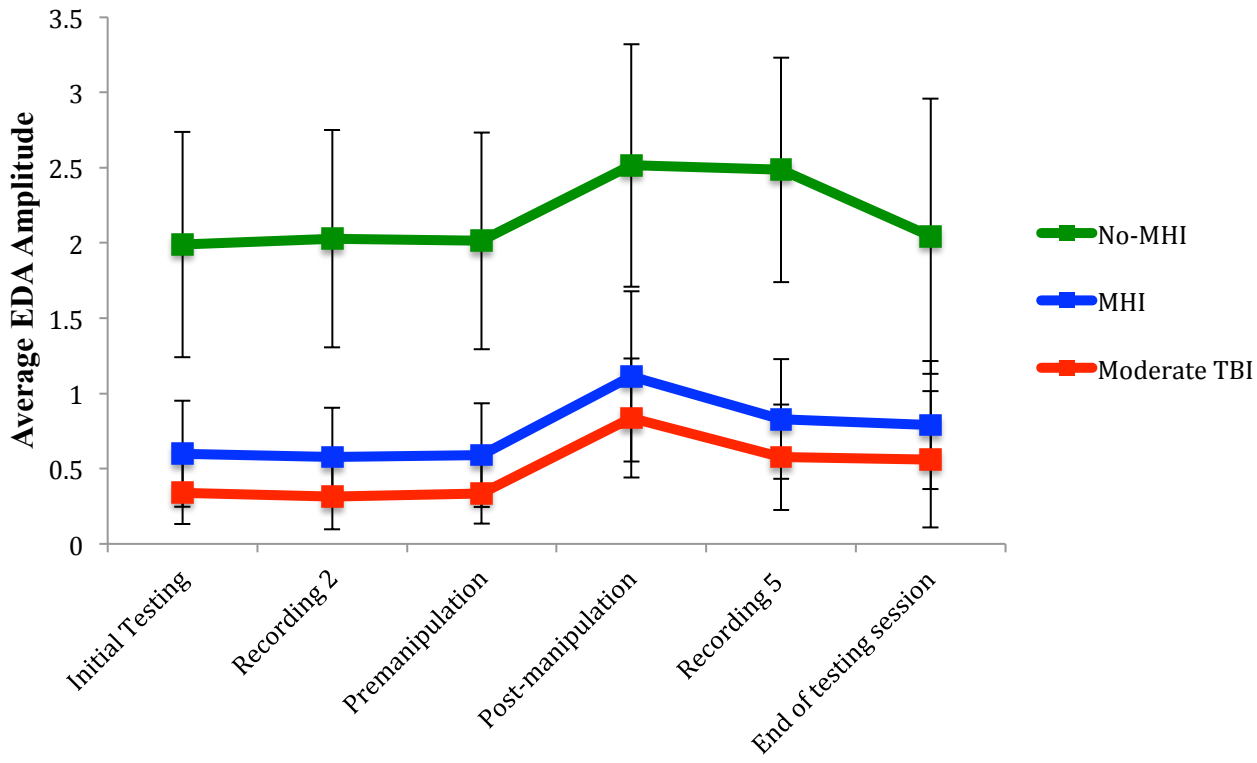

Figure 24: **Measures of physiological arousal (electrodermal activation [EDA]) across six measurements taken throughout the testing session as a function head injury status.**

A similar pattern of results is observed for pulse rate (Figure 25) across three collection points (i.e., initial testing, just after the arousal manipulation, and at the end of the testing session). A 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 3 (Time [initial testing, post-manipulation, end of testing session]) mixed model ANOVA for measures of pulse rate (BPM) was conducted, producing significant main effects of head injury status and time, with no interaction (Table 17 – see appendix A). Similar to EDA, the no-MHI group was found to have higher pulse rates relative to the MHI and moderate TBI groups ($p < .05$), whereas the injury groups were not different from one another ($p > .05$). Follow-up analyses revealed that pulse differed at initial testing (F (2, 83) = 5.56, p = .005) and at the end of the session (F (2, 82) = 3.42, p = .037, but was only a trend immediately following the arousal manipulation (F (, 2.07) =

3932.18, $p = .13$, trend). Post-hoc analyses illustrate for both the initial testing and the end of the testing session measures, the no-MHI group had higher arousal than both MHI and moderate TBI groups ($p < .05$), who were not different. No differences were observed in post-hoc analysis for the arousal manipulation.

Follow-up analyses of the main effect for time revealed that each time period differed from one another. More specifically, initial testing pulse rate was found to be higher than post-manipulation measures (t (84) = 12.13, $p < .001$ CI [5.12, 7.13]) and end of session measures (t (84) = 3.03, $p = .003$ CI [.52, 2.52]); post-arousal manipulation was significantly higher than the end of session measure (t (84) = 8.92, $p < .001$ CI [3.58, 5.64]). In summary, the results parallel the results obtained with measures of EDA, demonstrating that the injury groups are underaroused relative to the no-MHI group, but all are responsive to the arousal manipulation, supporting the predicted results.

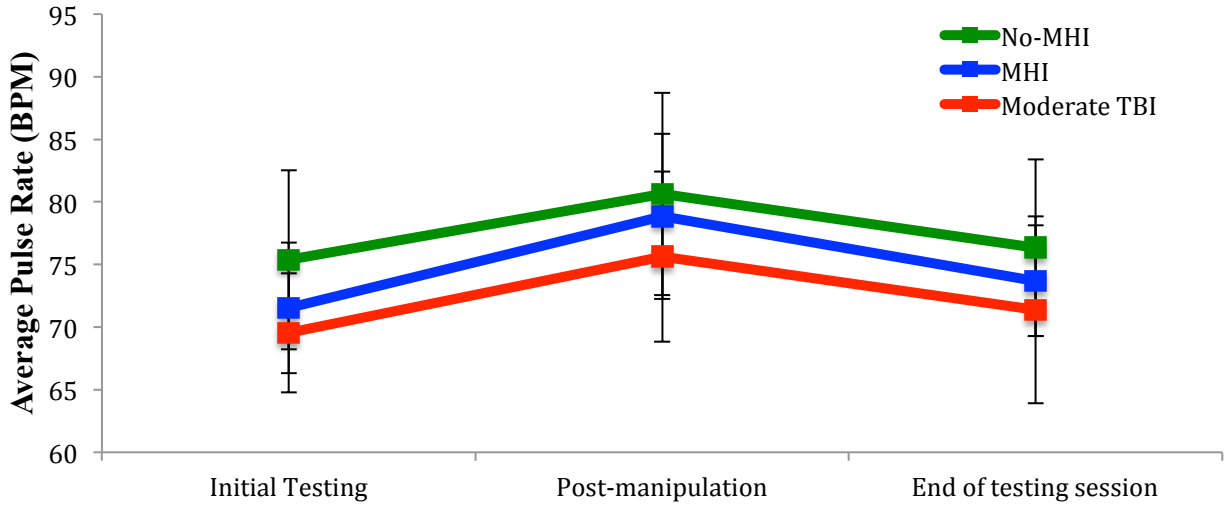

Figure 25: **Measures of physiological arousal (Pulse) across three measurements taken throughout the testing session as a function head injury status.**

Relative to EDA and pulse rate, a very different pattern of results was observed for measures of salivary cortisol across time periods and as a function of injury status (Figure 26). A

3 (head injury status [no-MHI, MHI, Moderate TBI]) × 3 (Time [initial testing, post-manipulation, end of testing session]) mixed model ANOVA for measures of salivary cortisol was conducted and revealed a significant main effect of head injury status (trend), time, and interaction between head injury status and time (trend; Table 18 – see appendix A). Follow-up one-way ANOVAs for each of the measurement times illustrated that the salivary cortisol measures differed as a function of head injury status for initial testing (F (2, 80) = 2.33, p = .10, trend) and post-manipulation (F (2, 81) = 2.03, p = .14, trend), but not at the end of session (F (2, 80) = .15, p = .86). Post-hoc analyses illustrated that the no-MHI group had lower salivary cortisol than the MHI group and moderate TBI group ($p < .05$), but the two head injury groups did not differ. Moreover, it was found that all groups, regardless of head injury status, had significantly lowering salivary cortisol as time progressed (no-MHI: $F^{G\text{-}G}$ (1.58, 64.85) = 17.77, $p < .001$, $\eta_p^2 = .30$; MHI: $F^{G\text{-}G}$ (1.14, 34.11) = 13.10, $p = .001$, $\eta_p^2 = .30$; moderate TBI: F (2, 16) = 13.71, $p < .001$, $\eta_p^2 = .63$) with each time session differing from one another significantly ($p < .05$). This more rapid decline in cortisol may reflect the quickened return-to-initial testing arousal levels and reduced stress/arousal pattern post stress-manipulation observed in other studies (e.g., Baker & Good, 2014) for persons with head injuries relative to a matched control cohort.

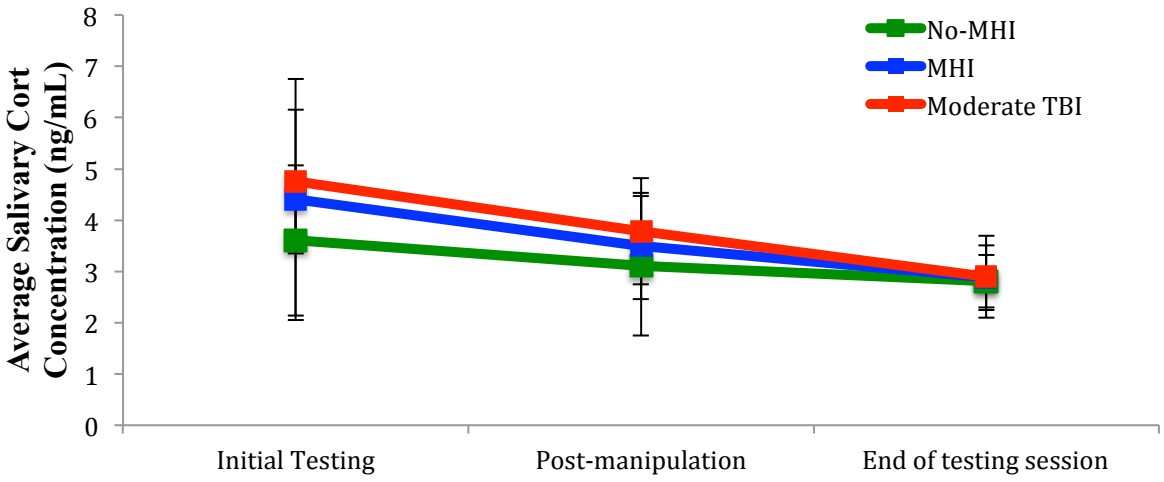

Figure 26: **Measures of physiological arousal (salivary cortisol) across three measurements taken throughout the testing session as a function head injury status.**

It was predicted that measures of physiological arousal (i.e., EDA) would increase during the IGT task for those exposed to the arousal manipulation relative to those who had not been exposed to the manipulation prior to completing the IGT. Figures 27, 28 and 29 depict measures of EDA just prior to the task, in anticipation of making selections on the IGT and in response to feedback (positive and negative) on the IGT as function of the arousal manipulation for the no-MHI, MHI and moderate TBI groups. As observed in these figures, a 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 4 (Arousal Type [prior to task, anticipation, positive feedback, negative feedback]) × 2 (Arousal Manipulation status [pre-exposure, post-exposure]) mixed model ANOVA for measures of EDA during the Iowa Gambling Task (IGT) as a function of head injury status and arousal manipulation illustrated no main effects or interactions for the arousal manipulation (Table 19 – see appendix A)[22]. The arousal manipulation did not have an effect on measures of EDA.

[22] Other effects in Table 14 are discussed in hypothesis 2

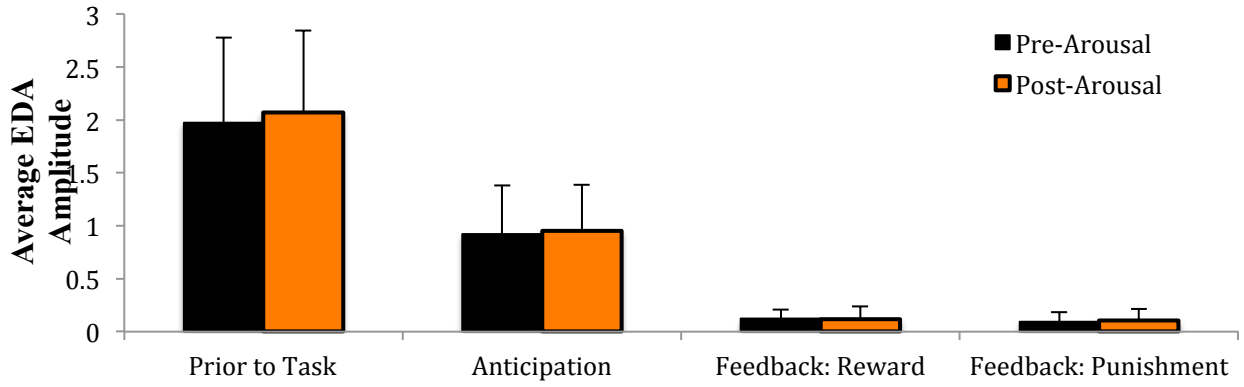

Figure 27: **Physiological arousal measured prior to the task and in anticipation of, and in response (reward and punishment) to, selections made on the Iowa Gambling Task as a function of the arousal manipulation for no-MHI participants**

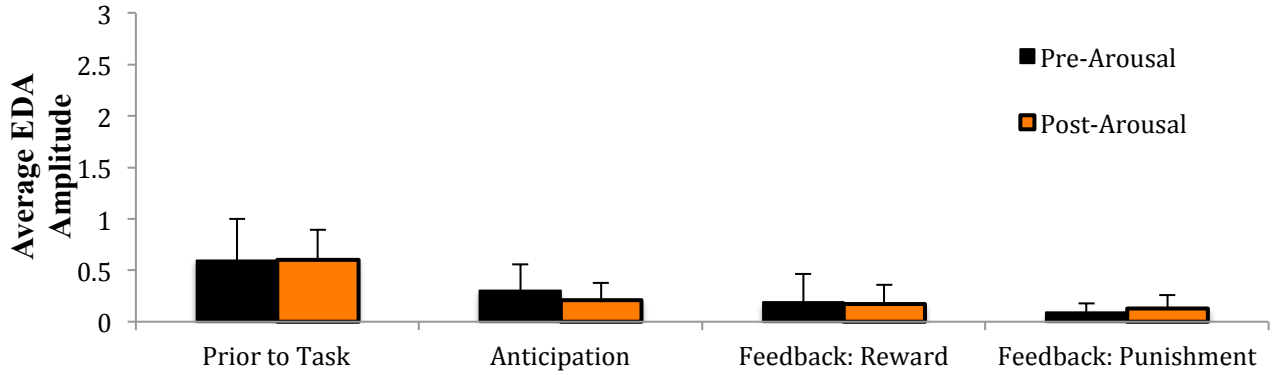

Figure 28: **Physiological arousal measured prior to the task and in anticipation of, and in response (reward and punishment) to, selections made on the Iowa Gambling Task as a function of the arousal manipulation for MHI participants**

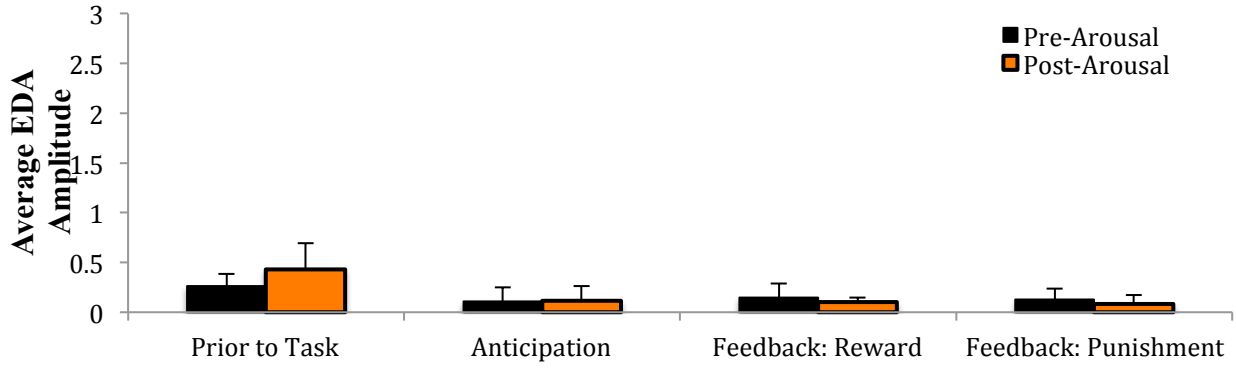

Figure 29: **Physiological arousal measured prior to the task and in anticipation of, and in response (reward and punishment) to, selections made on the Iowa Gambling Task as a function of the arousal manipulation for moderate TBI participants**

Similar to gross measures of overall anticipation presented in Figures 27 through 29, no significant differences were observed when examining anticipatory arousal prior to making selections on the IGT for Decks A (no-MHI: Figure 30, MHI: Figure 31, moderate TBI: Figure 32; Table 7 – see appendix A), B (no-MHI: Figure 33, MHI: Figure 34, moderate TBI: Figure 35; Table 8 – see appendix A), C (no-MHI: Figure 36, MHI: Figure 37, moderate TBI: Figure 38; Table 9 – see appendix A) and D (no-MHI: Figure 39, MHI: Figure 40, moderate TBI: Figure 41; Table 10 – see appendix A) as a function of the arousal manipulation. Again, this shows that while anticipatory arousal differs as a function of head injury status, it was not found to increase with the arousal manipulation and, thus, did not support the prediction that emotionally-evocative stimuli would increase anticipatory physiological arousal.

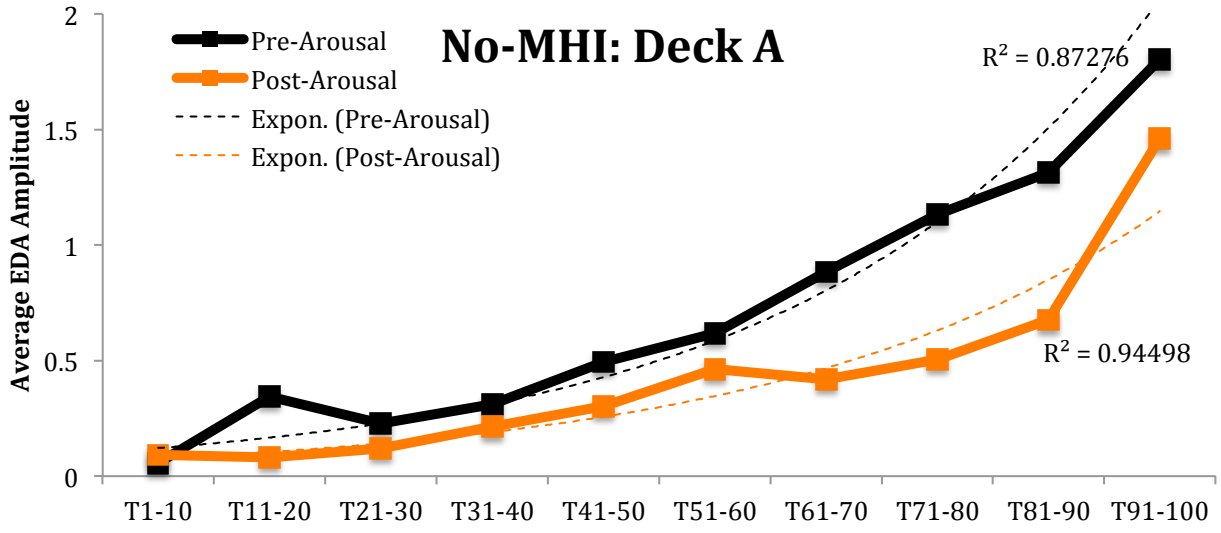

Figure 30: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck A on the Iowa Gambling Task across 10 trial blocks as a function of the arousal manipulation for no-MHI participants.**

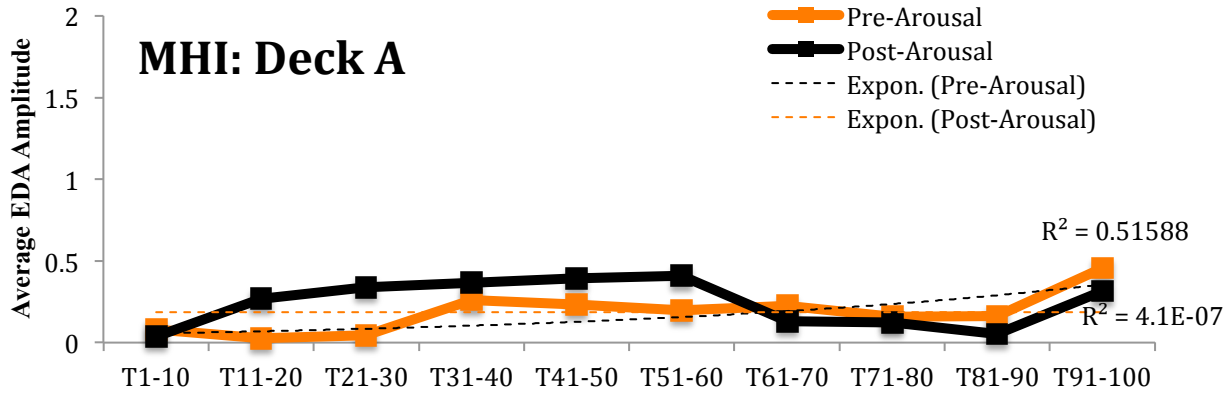

Figure 31: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck A on the Iowa Gambling Task across 10 trial blocks as a function of the arousal manipulation for MHI participants.**

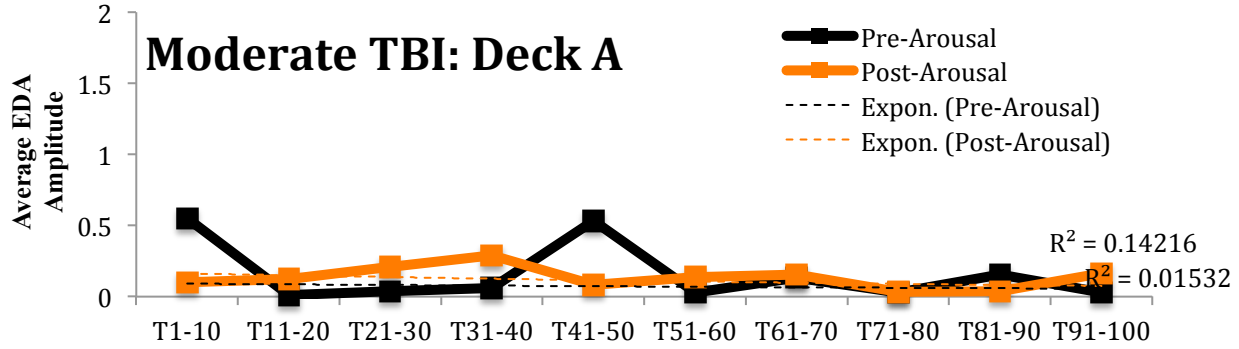

Figure 32: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck A on the Iowa Gambling Task across 10 trial blocks as a function of the arousal manipulation for moderate TBI participants.**

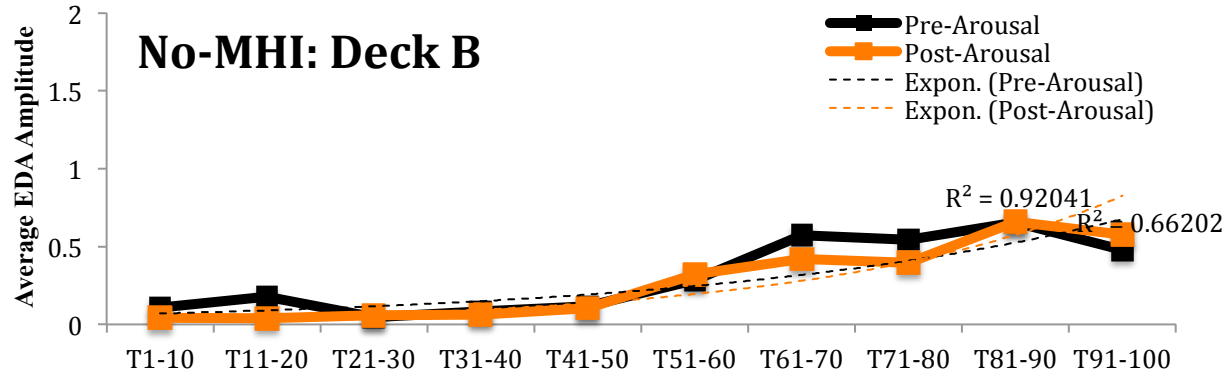

Figure 33: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck B on the Iowa Gambling Task across 10 trial blocks as a function of the arousal manipulation for no-MHI participants.**

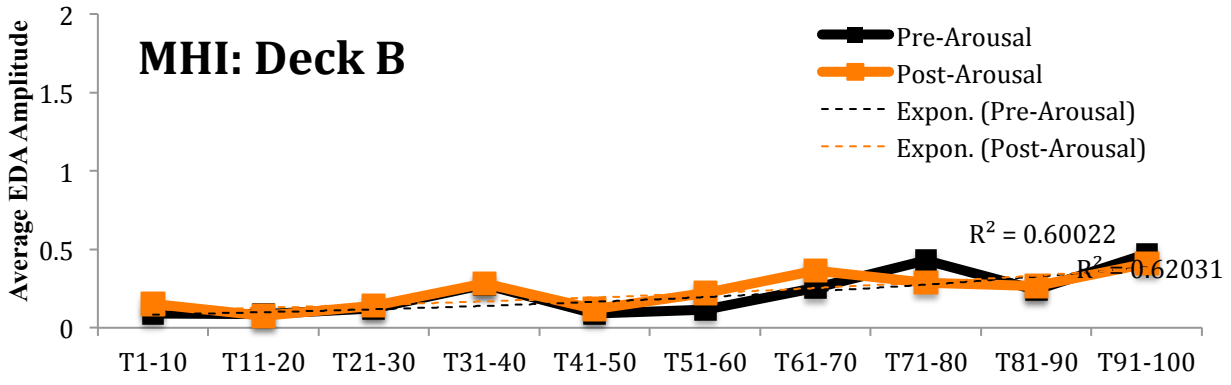

Figure 34: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck B on the Iowa Gambling Task across 10 trial blocks as a function of the arousal manipulation for MHI participants.**

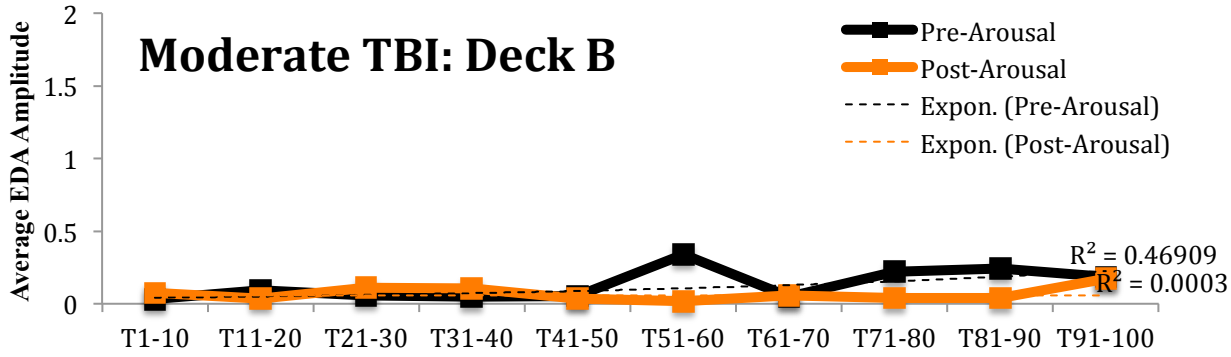

Figure 35: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck B on the Iowa Gambling Task across 10 trial blocks as a function of the arousal manipulation for moderate TBI participants.**

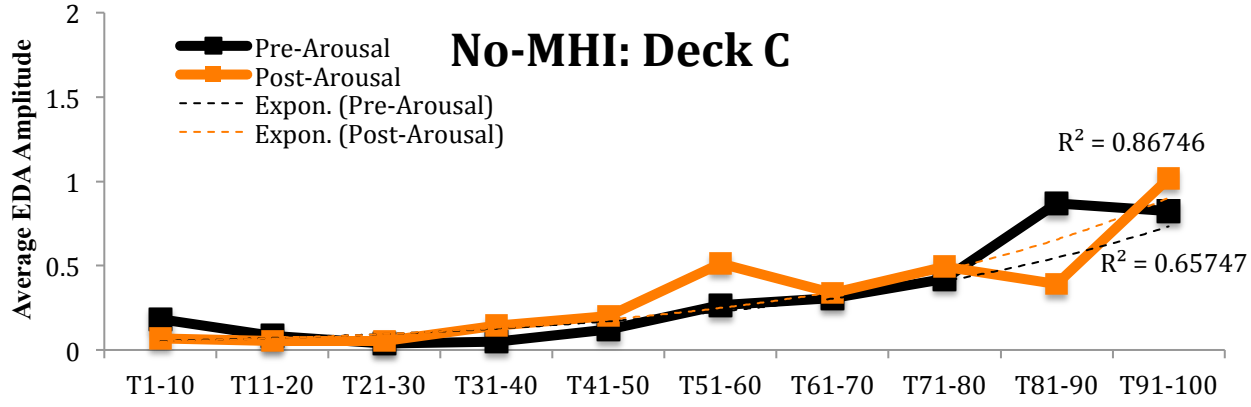

Figure 36: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck C on the Iowa Gambling Task across 10 trial blocks as a function of the arousal manipulation for no-MHI participants.**

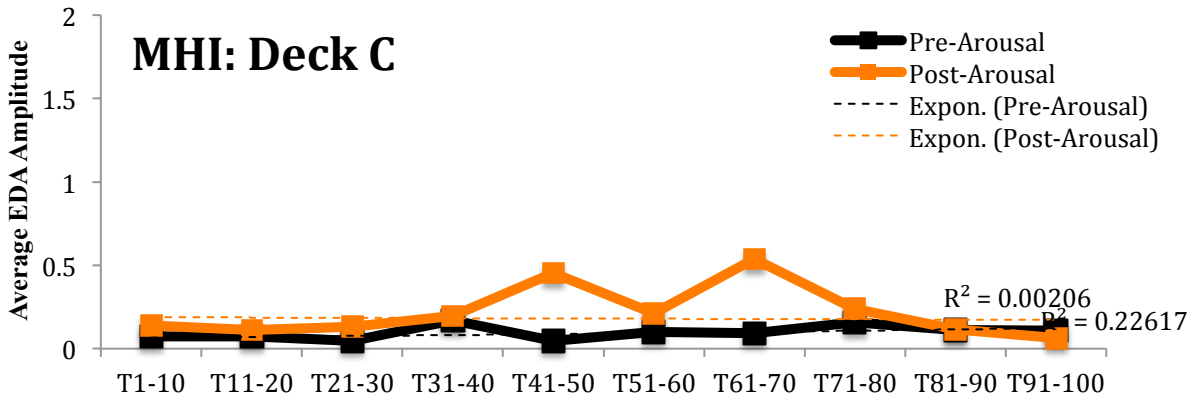

Figure 37: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck C on the Iowa Gambling Task across 10 trial blocks as a function of the arousal manipulation for MHI participants.**

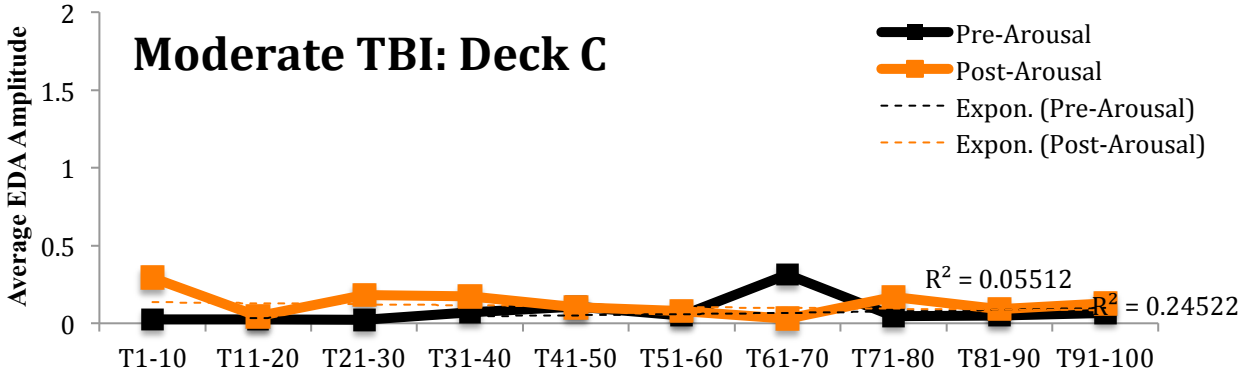

Figure 38: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck C on the Iowa Gambling Task across 10 trial blocks as a function of the arousal manipulation for moderate TBI participants.**

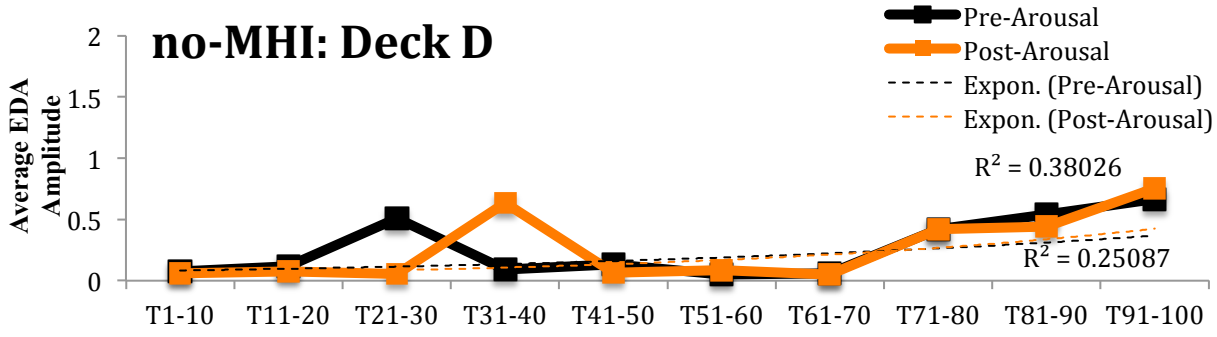

Figure 39: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck D on the Iowa Gambling Task across 10 trial blocks as a function of the arousal manipulation for no-MHI participants.**

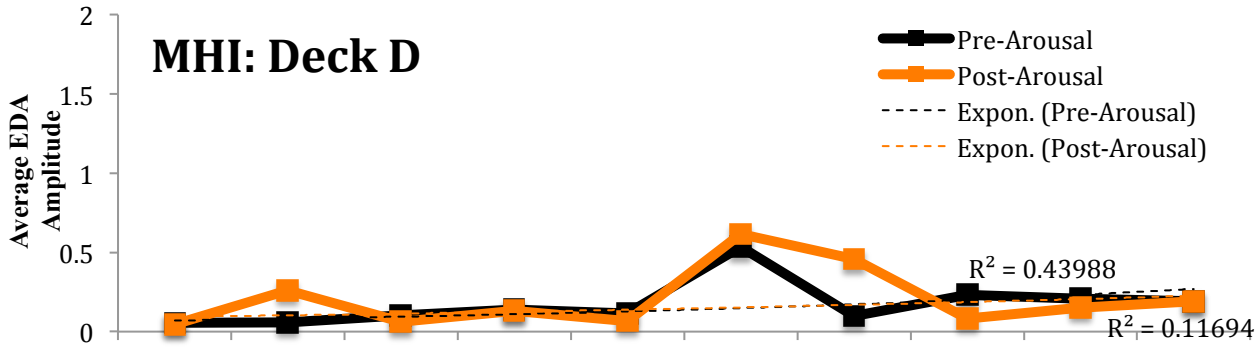

Figure 40: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck D on the Iowa Gambling Task across 10 trial blocks as a function of the arousal manipulation for MHI participants.**

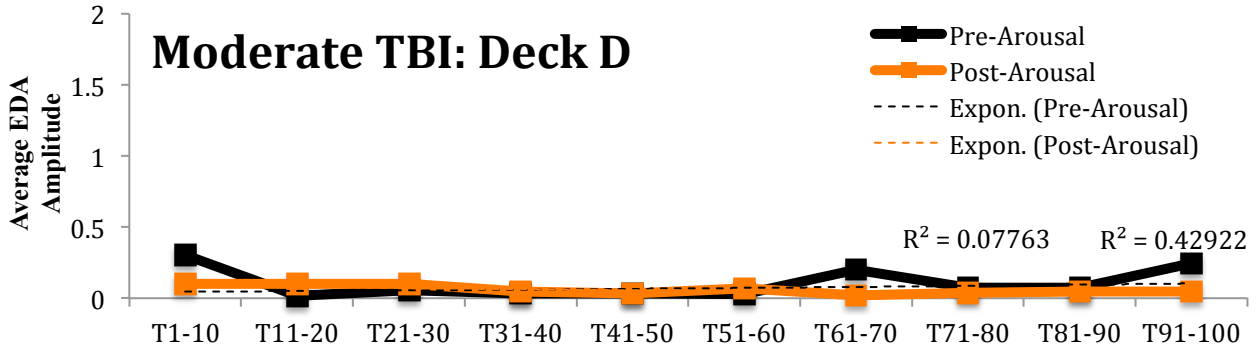

Figure 41: **Average anticipatory physiological arousal (as measured by electrodermal activation [EDA]) prior to making selections for Deck D on the Iowa Gambling Task across 10 trial blocks as a function of the arousal manipulation for moderate TBI participants.**

Lastly, it was predicted that the presentation of emotionally-evocative stimuli would boost arousal and subsequently this would improve decision-making in two ways for those who had sustained injuries: by decreasing the number of trials required for transitioning from high risk (disadvantageous decks) to low risk (advantageous) choices; and by increasing the number of trials before returning to high risk selections following punishment feedback. Furthermore, it was predicted that persons who do not report a history of MHI may be disadvantaged by increased arousal, should their levels of arousal introduce distress. Figures 42, 43 and 44 depict the ratio of advantageous selections across 10-trial blocks as a function of the arousal

manipulation for no-MHI, MHI and moderate TBI respectively. It was found that the arousal manipulation did not affect the ratio of selections on the IGT (Table 11 – see appendix A). In summary, there were no observable improvements in decision-making as measured by a ratio of advantageous to disadvantageous selections for the injury groups as a result of the arousal manipulation.

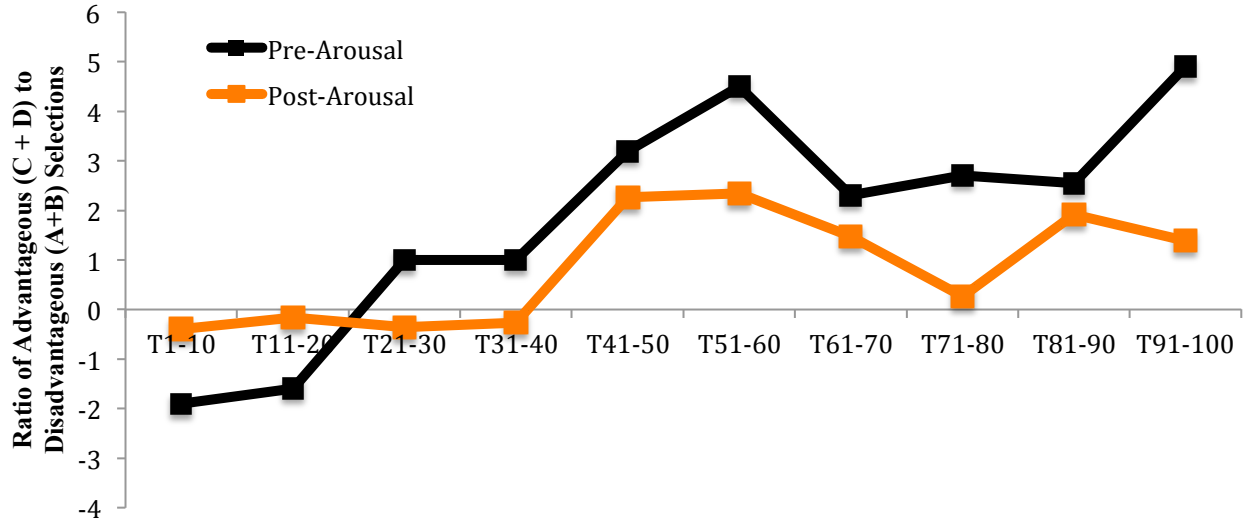

Figure 42: **Ratio of advantageous (C + D) to disadvantageous (A + B) selections on the Iowa Gambling Task (IGT) as a function of the arousal manipulation status across 10 trial blocks for no-MHI participants**

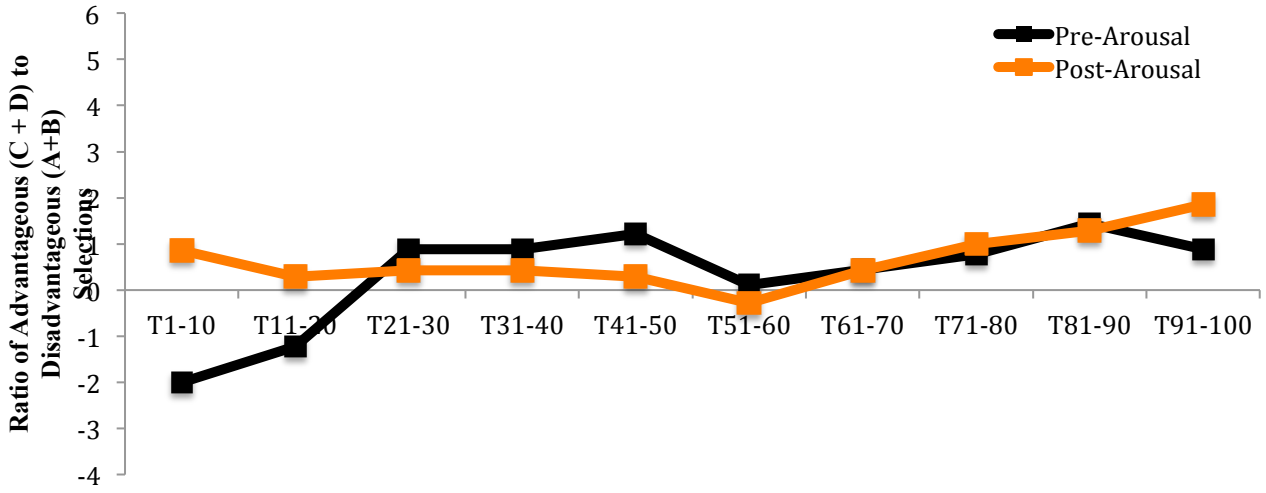

Figure 43: **Ratio of advantageous (C + D) to disadvantageous (A + B) selections on the Iowa Gambling Task (IGT) as a function of the arousal manipulation status across 10 trial blocks for MHI participants**

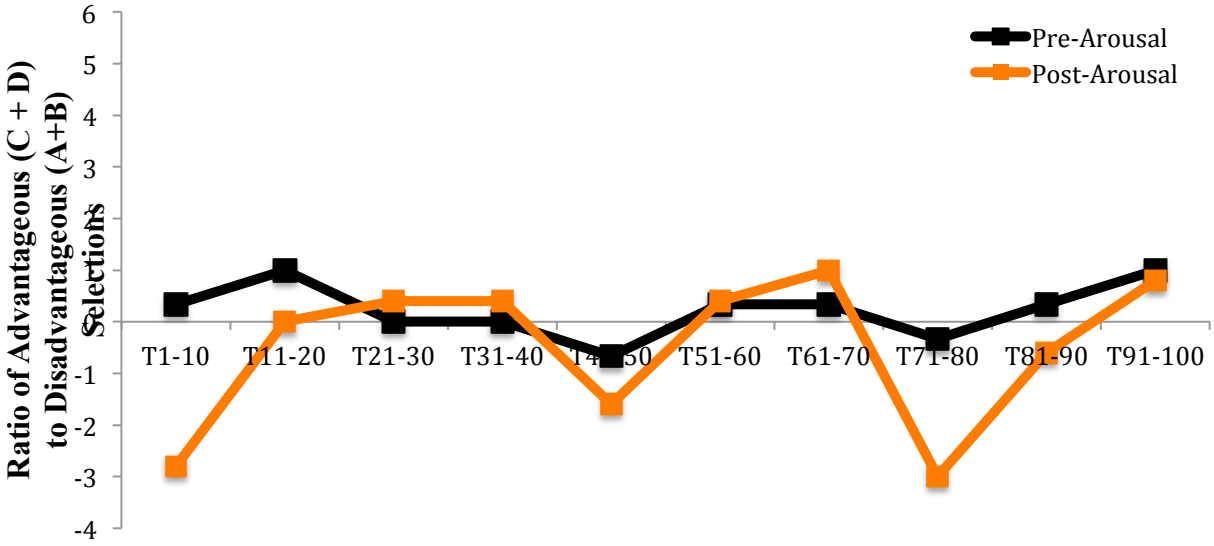

Figure 44: **Ratio of advantageous (C + D) to disadvantageous (A + B) selections on the Iowa Gambling Task (IGT) as a function of the arousal manipulation status across 10 trial blocks for moderate TBI participants**

Finally, as observed in Figures 45, 46 and 47, and in Table 12, there was no main effect or interaction with the arousal manipulation. Emotionally evocative stimuli did not have an improvement on decision-making as measured by number of trials before returning to high-risk selections following punishment feedback.

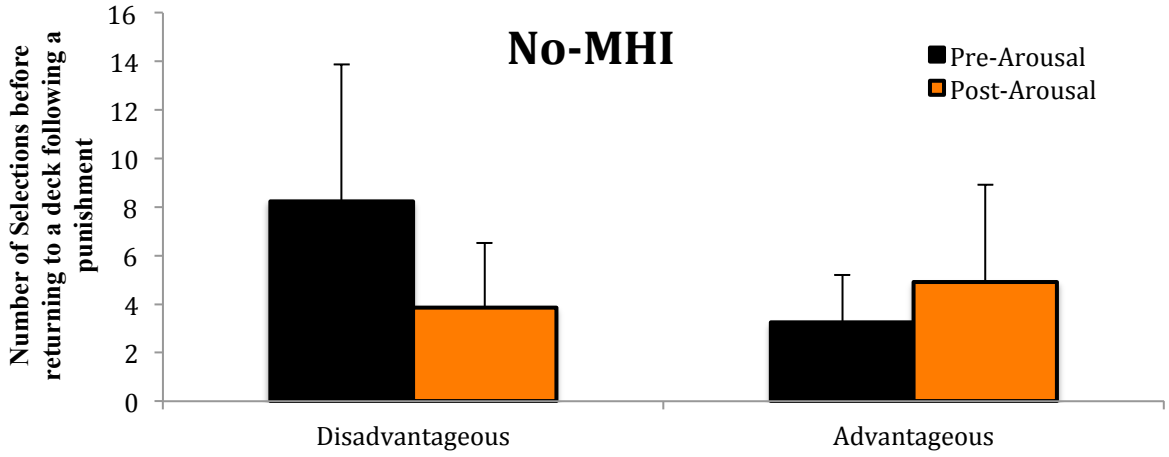

Figure 45. **Number of selections taken following a punishment before returning to that deck (averaged into disadvantageous [A and B] and advantageous [C and D] selections) as a function of the arousal manipulation for no-MHI participants**

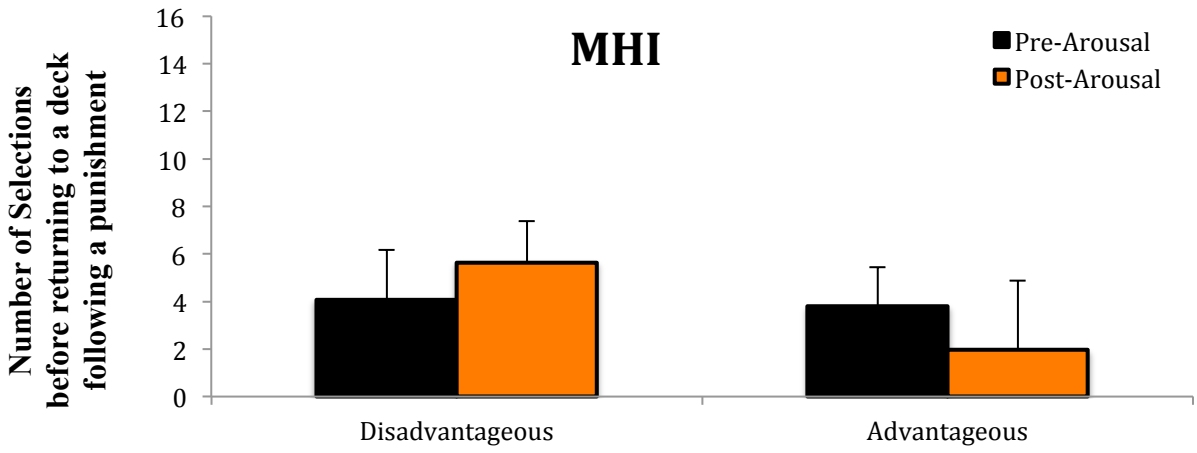

Figure 46. **Number of selections taken following a punishment before returning to that deck (averaged into disadvantageous [A and B] and advantageous [C and D] selections) as a function of the arousal manipulation for MHI participants**

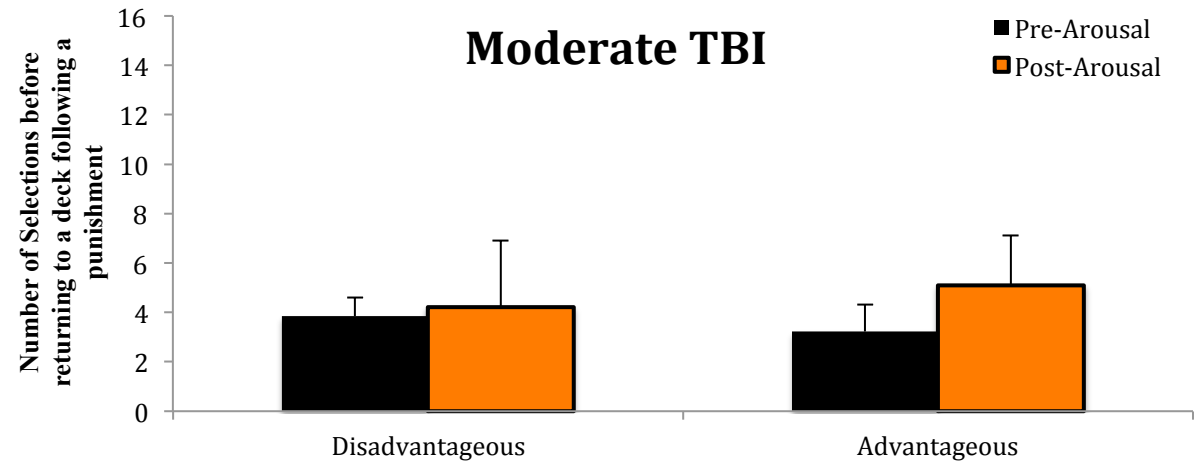

Figure 47. **Number of selections taken following a punishment before returning to that deck (averaged into disadvantageous [A and B] and advantageous [C and D] selections) as a function of the arousal manipulation for moderate TBI participants**

In summary, it was found that the arousal manipulation did increase measures of physiological arousal (i.e., pulse and EDA) immediately following the manipulation, but this did not persist for injury groups, whereas it did for the no-MHI group. However, there were no other improvements in decision-making performance as a result of the arousal manipulation. Overall, this hypothesis was not supported.

Exploratory - Hypothesis V: IGT and Depression

Figures 48 and 49 illustrate self-reported depressive symptomatology as a function of head injury status using the SA-45 and Beck's Depression Inventory. One-way ANOVAs were conducted and no group differences for head injury status were observed on the SA-45 (F (3, 83) = .02 p = .98) or the BDI (F (2, 83) = .56 p = .58). As a result, depression symptoms do not vary with head injury status.

A linear regression was conducted to examine whether head injury status and the ratio of advantageous-to-disadvantageous selections on the last five 10-trial blocks would predict self-reported measures of depression as measured by the SA-45 and whether this effect was moderated by head injury status. Hierarchal regression demonstrated that head injury status and the last five 10-trial blocks accounted for 7.6% of the variance in self-reported depressive symptomatology on the SA-45, however the model was not significant (F (6, 79) = 1.08 p = .38). When the interaction variables were included on the second step (with the previous variables), the model accounted for 20.3% of the variance. This accounted for 12.7% more variable in the first model (F (5, 84) = 2.25, p = .03). Table 20 depicts β values, tests of significance and semi-partial correlations. Simple slopes analysis revealed that this relationship was only present for the MHI group, whereby decreasing performance was associated with increasing depression scores. This relationship was not observed for the no-MHI or the moderate TBI group.

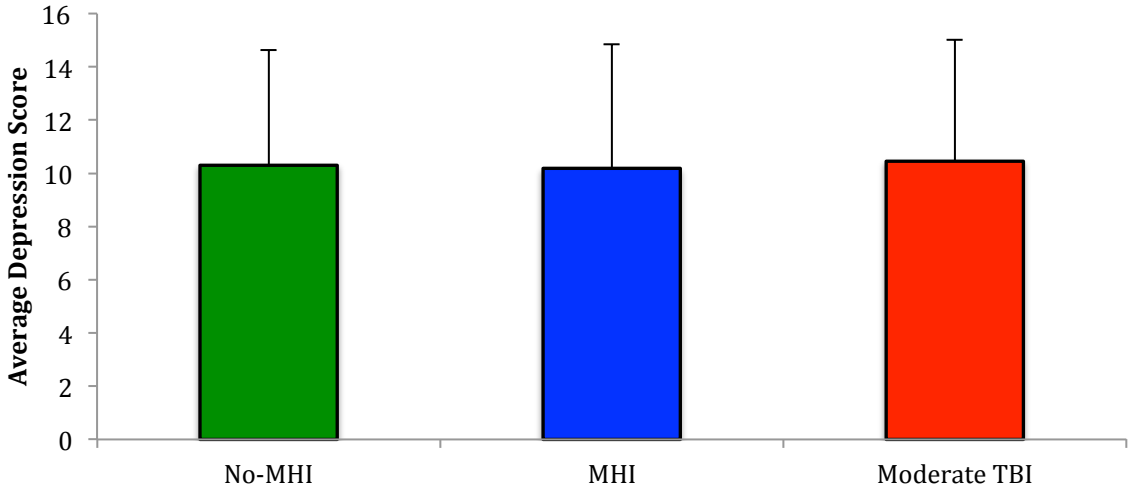

Figure 48. **Average self-reported depressive symptomatology ratings as measured by the SA-45 as a function of head injury status**

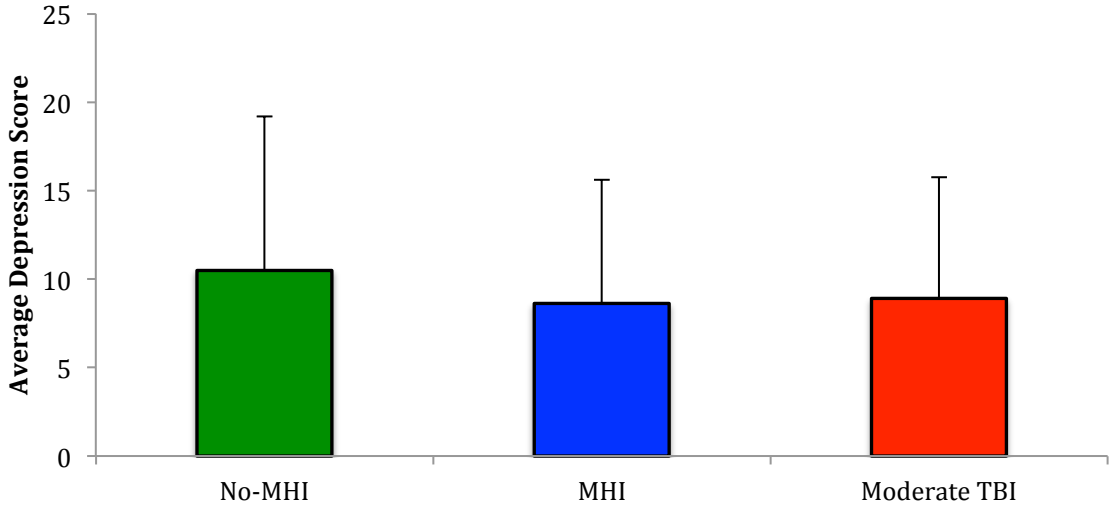

Figure 49. **Average self-reported depressive symptomatology ratings as measured by the Beck's Depression Inventory as a function of head injury status**

Table 20:

Results from hierarchical linear regression model predicting self-reported depressive symptomatology as measured on the SA-45

	Step 1				Step 2			
Predictors	β	t	p	part	β	t	p	part
Head injury (centered)	-.09	-.81	.42	-.09	-0.18	-1.45	0.15	-0.15
Net Ratio – Trials 51 to 60 (Centered)	.04	.36	.72	.04	0.18	1.40	0.16	0.15
Net Ratio – Trials 61 to 70 (Centered)	-.05	.39	.70	.04	0.05	0.44	0.66	0.05
Net Ratio – Trials 71 to 80 (Centered)	-.13	-.99	.33	.11	-0.23	-1.62	0.11	-0.17
Net Ratio – Trials 81 to 90 (Centered)	.24	1.83	.07	.20	0.31	2.35	0.02	0.25
Net Ratio – Trials 91 to 100 (Centered)	-.26	-2.15	.03	-.23	-0.36	-2.81	0.01	-0.30
Head Injury × Net Ratio – Trials 51 to 60					0.29	2.39	0.02	0.25
Head Injury × Net Ratio – Trials 61 to 70					0.15	0.94	0.24	0.12
Head Injury × Net Ratio – Trials 71 to 80					-0.27	-1.20	0.12	-0.16
Head Injury × Net Ratio – Trials 81 to 90					-0.01	-0.08	0.94	-0.01
Head Injury × Net Ratio – Trials 91 to 100					-0.20	-1.32	0.19	-0.14

Pearson-r correlations were conducted, indicating that the number of selections participants made prior to returning to disadvantageous decks after punishment was predictive of their self-reported depressive symptomatology only for the MHI group (r = -.38, p = .035), but not for either the moderate TBI group (r = -.33, p = .33) or no-MHI (r = .15, p = .36) groups (Figures 50 and 51). No relationships were observed between the number of selections participants made prior to returning to advantageous decks following punishment and self-reported depressive symptomatology (No-MHI: r = -.04, p = .81, MHI: r = -.18, p = .32, moderate TBI: r = .24, p = .48; Figures 52 and 53). A linear regression was conducted to examine whether head injury status and the rate of return following a punishing selection would predict depression scores and whether this effect was moderated by head injury status. Hierarchal regression demonstrated that head injury status and rate of return following a punishing selection accounted for 0.2% of the variance in self-reported depression, however the model was not significant (F (2, 81) = 0.83 p = .92). When the interaction variables were included on the second step (with the above variables), the model accounted for 4.4% of the variance. This accounted for 4.2% more variable in the first model (F (1, 80) = 3.84, p = .05). As observed, with IGT performance, simple slopes analysis revealed that this relationship was only present for the MHI group, whereby decreasing rate of return following punishment (i.e., less trials in between a punishment and returning to that same deck) was associated with increasing depression scores. This relationship was not observed for the no-MHI or the moderate TBI group.

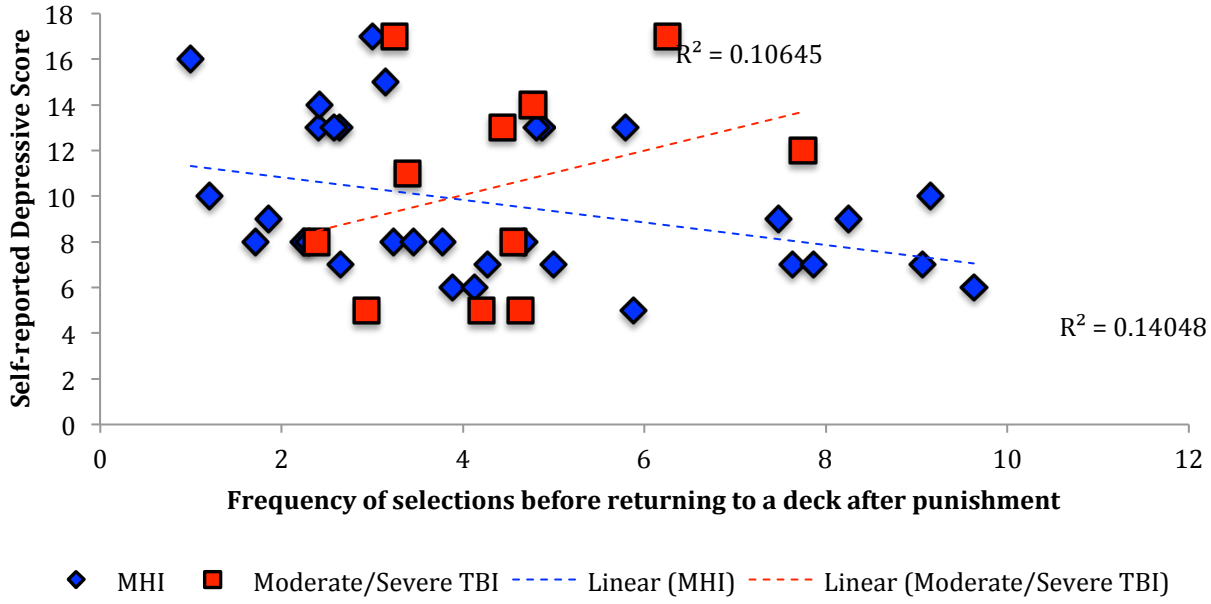

Figure 50. **Scatter plot depicting the relationships between self-reported depressive symptomatology on the SA-45 with frequency selecting from disadvantageous decks before returning to a deck after being punished for MHI and Moderate TBI groups**

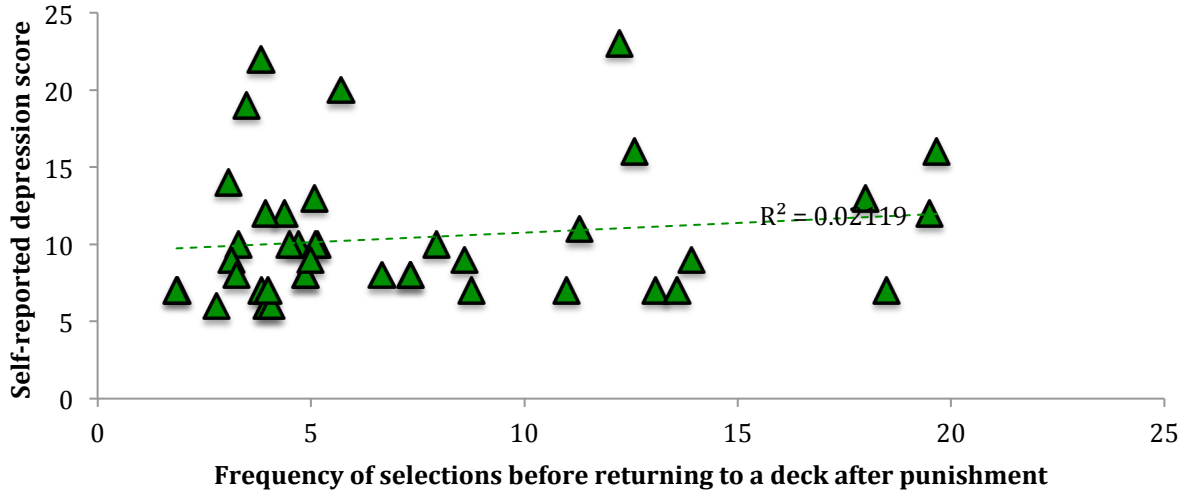

Figure 51. **Scatter plot depicting the relationships between self-reported depressive symptomatology on the SA-45 with frequency selecting from disadvantageous decks before returning to a deck after being punished for the no-MHI group**

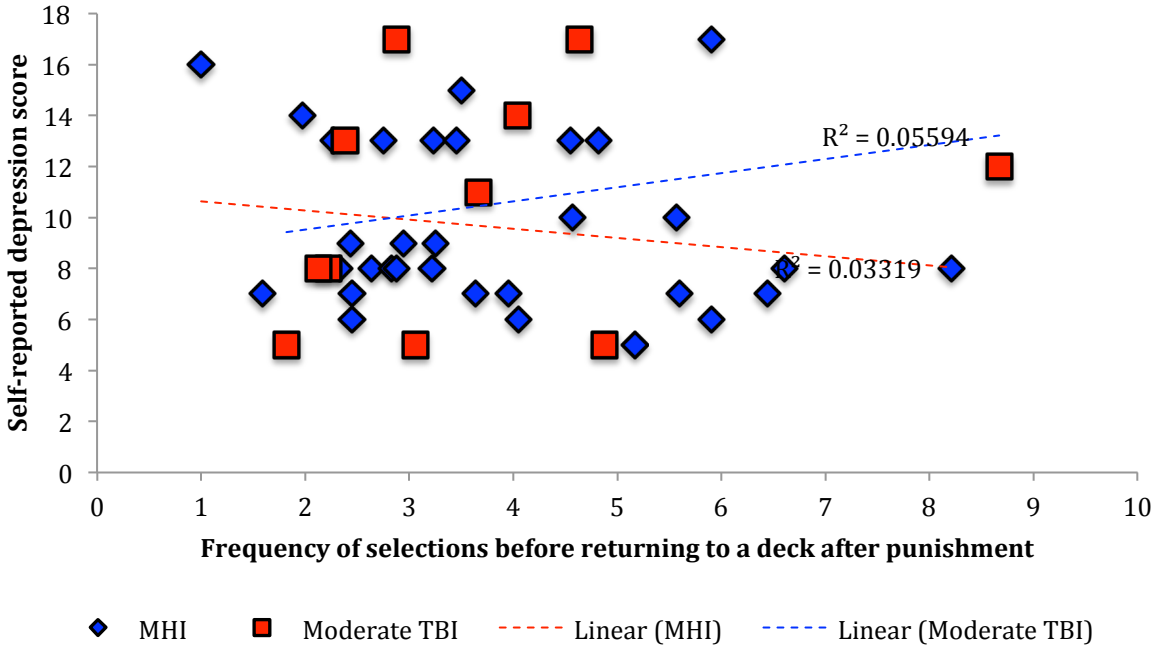

Figure 52. **Scatter plot depicting the relationships between self-reported depressive symptomatology on the SA-45 with frequency selecting from advantageous decks before returning to a deck after being punished for MHI and Moderate TBI groups**

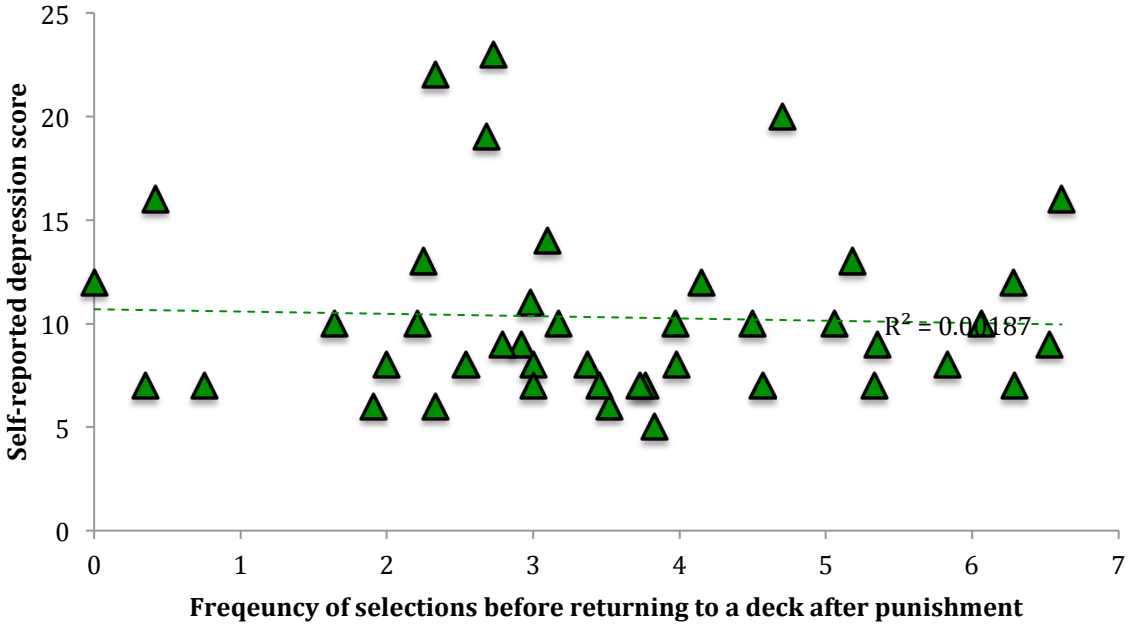

Figure 53. **Scatter plot depicting the relationships between self-reported depressive symptomatology on the SA-45 with frequency selecting from advantageous decks before returning to a deck after being punished for the no-MHI group**

In summary, the results illustrate that IGT performance on the last five blocks of 10-trials was predictive of self-reported depressive symptomatology and MHI status was found to be moderator of this relationship. Secondly, rate of return to a disadvantageous selection following punishment was predictive of depressive symptomatology for the MHI group only.

Supplementary Analyses:

To further explore the relationship between IGT performance and depressive symptomatology, participants were assigned to one of two groups based on self-reported depressive symptomatology intensity (i.e., lower versus higher depressive symptom severity). A median split procedure was conducted (no-MHI: Median = 9.00; MHI: Median = 8.50; Moderate TBI: Median = 11.00) and IGT performance was plotted as a function of depressive symptoms severity and head injury status (Figures 54, 55, and 56). A 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 5 (Trial Blocks [trials 51-60, trials 61-70, trials 71-80, trials 81-90, trials 91-100]) × 2 (Degree of Depression [low, high]) mixed model ANOVA for measures of the ratio of advantageous (C + D) to disadvantageous (A + B) selections on the IGT was conducted (Table 21 – see appendix A). A significant main effect of head injury was observed, in addition to an interaction between trial blocks and depressive symptoms severity.

Post-hoc analyses were completed. As noted previously, the no-MHI group illustrated a greater ratio of advantageous-to-disadvantageous selections relative to MHI and moderate TBI, but no differences were observed between injury groups. To explore the interaction between trial blocks and depressive symptoms severity, two repeated measures ANOVAs were conducted, and showed that those with lower depression improved their performance as trials progressed (F (4, 164) = 3.56, p = .008, η_p^2 = .08; Figure 57), whereas for individuals reporting higher depression (F (4, 164) = .77, p = .55, ns; Figure 58) no such relationship was obtained. Interestingly, no

differences were observed between those reporting lower and higher depression for the no-MHI group.

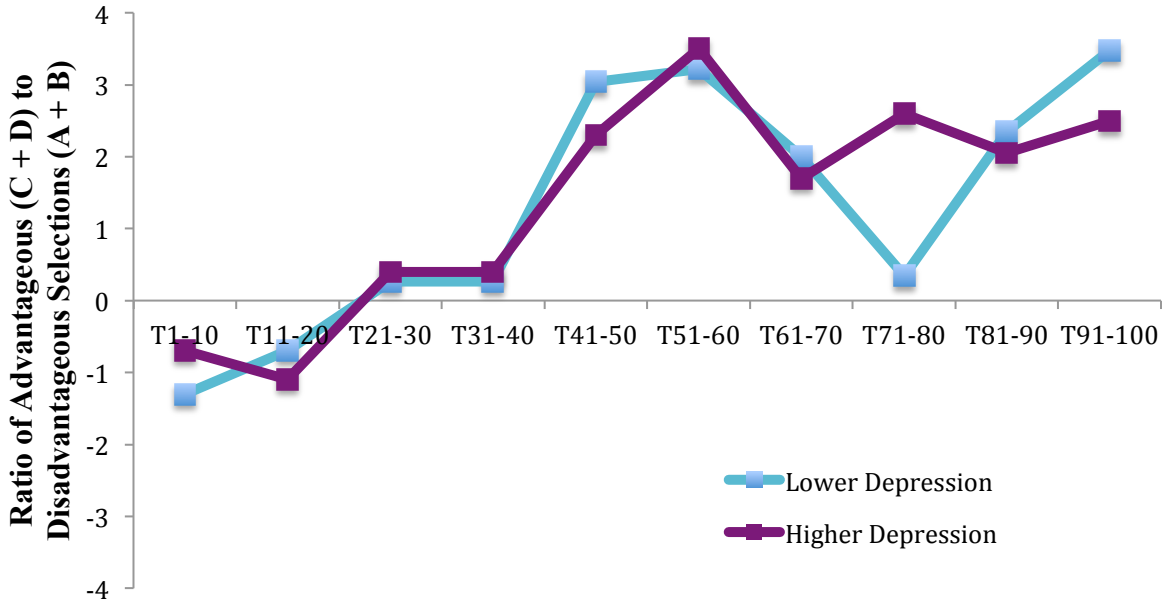

Figure 54: **Ratio of advantageous (C + D) to disadvantageous (A + B) selections on the Iowa Gambling Task (IGT) across 10 trial blocks as a function of higher and lower depression symptomatology for no-MHI participants**

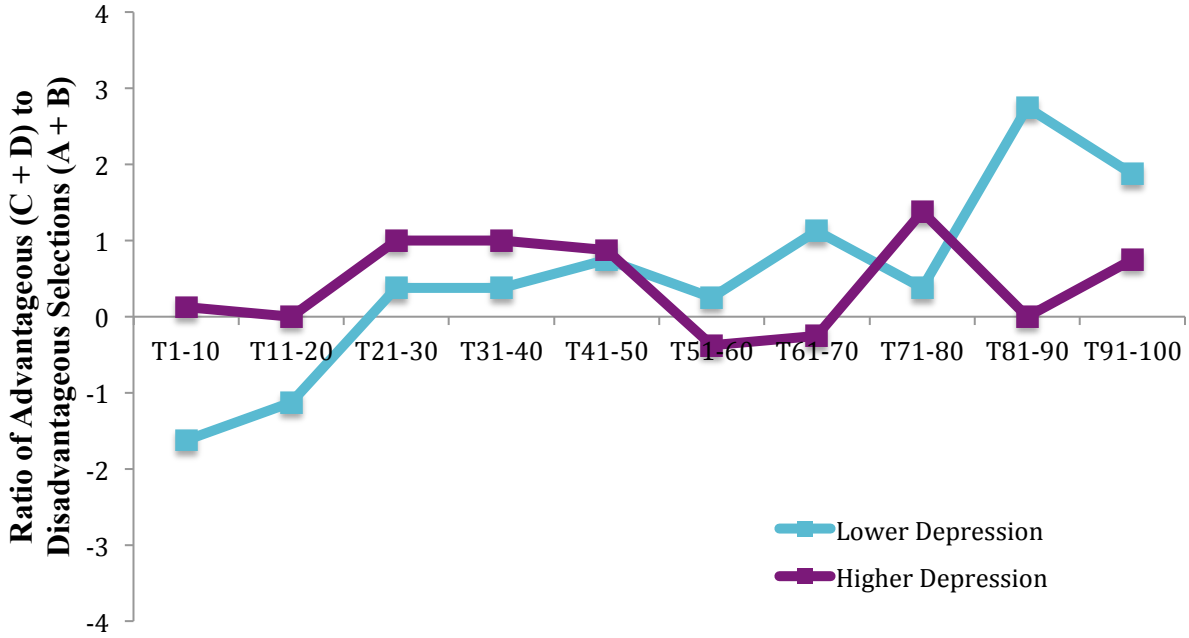

Figure 55: **Ratio of advantageous (C + D) to disadvantageous (A + B) selections on the Iowa Gambling Task (IGT) across 10 trial blocks as a function of higher and lower depression symptomatology for MHI participants**

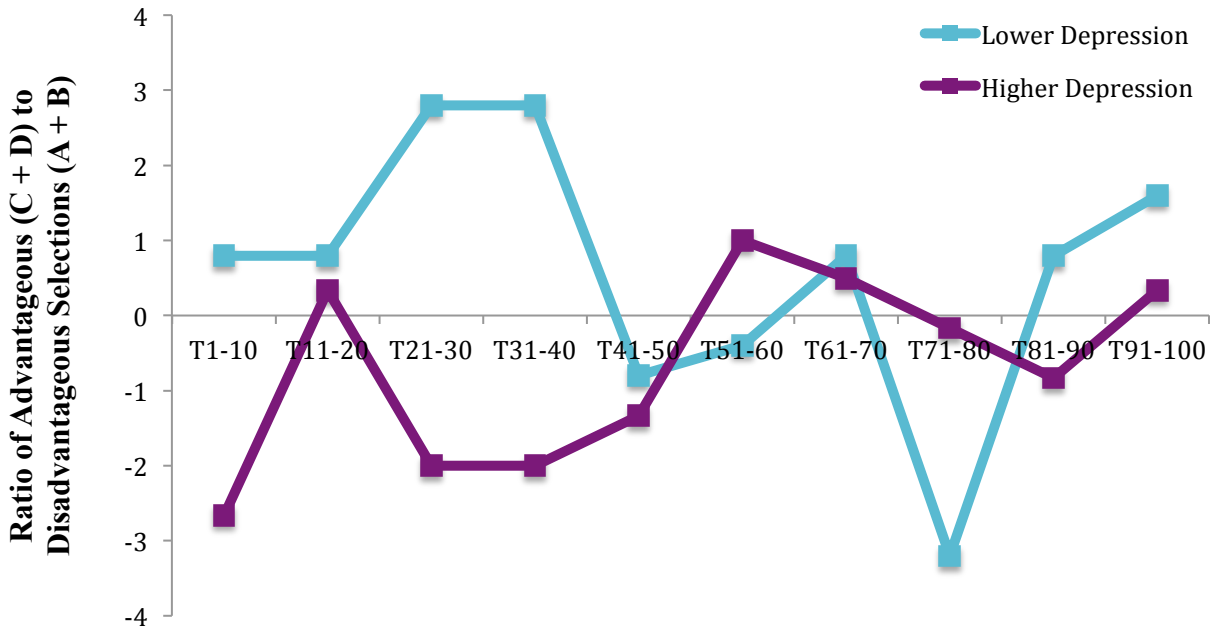

Figure 56: **Ratio of advantageous (C + D) to disadvantageous (A + B) selections on the Iowa Gambling Task (IGT) across 10 trial blocks as a function of higher and lower depression symptomatology for moderate TBI participants**

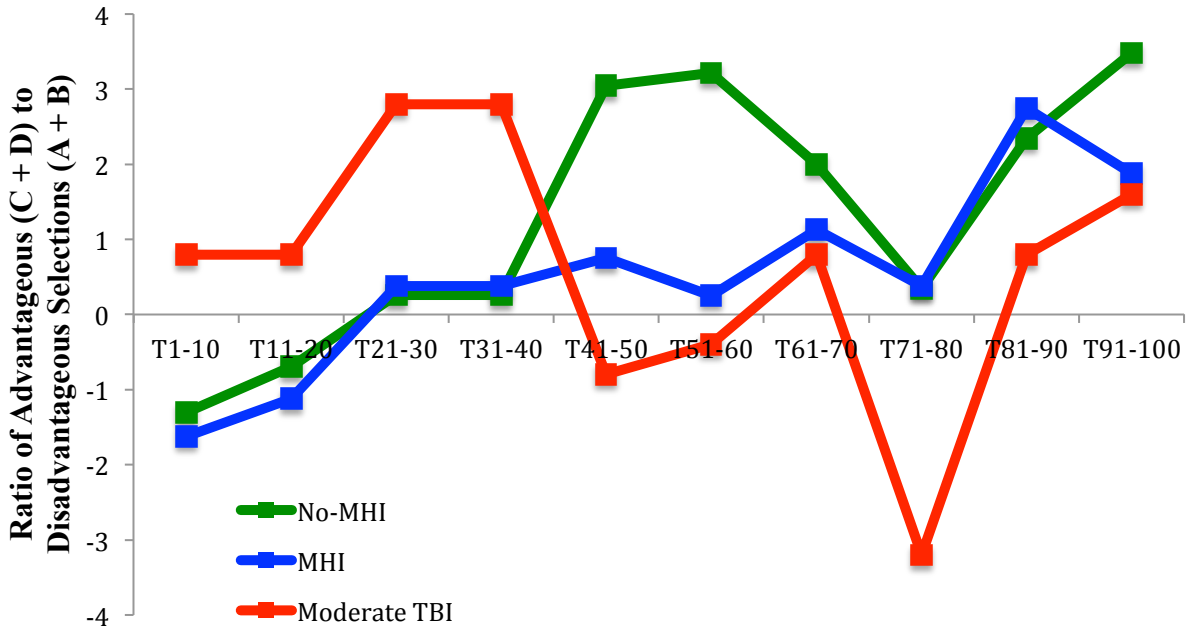

Figure 57: **Ratio of advantageous (C + D) to disadvantageous (A + B) selections on the Iowa Gambling Task (IGT) across 10 trial blocks as a function of head injury status for participants reporting lower depressive symptomatology**

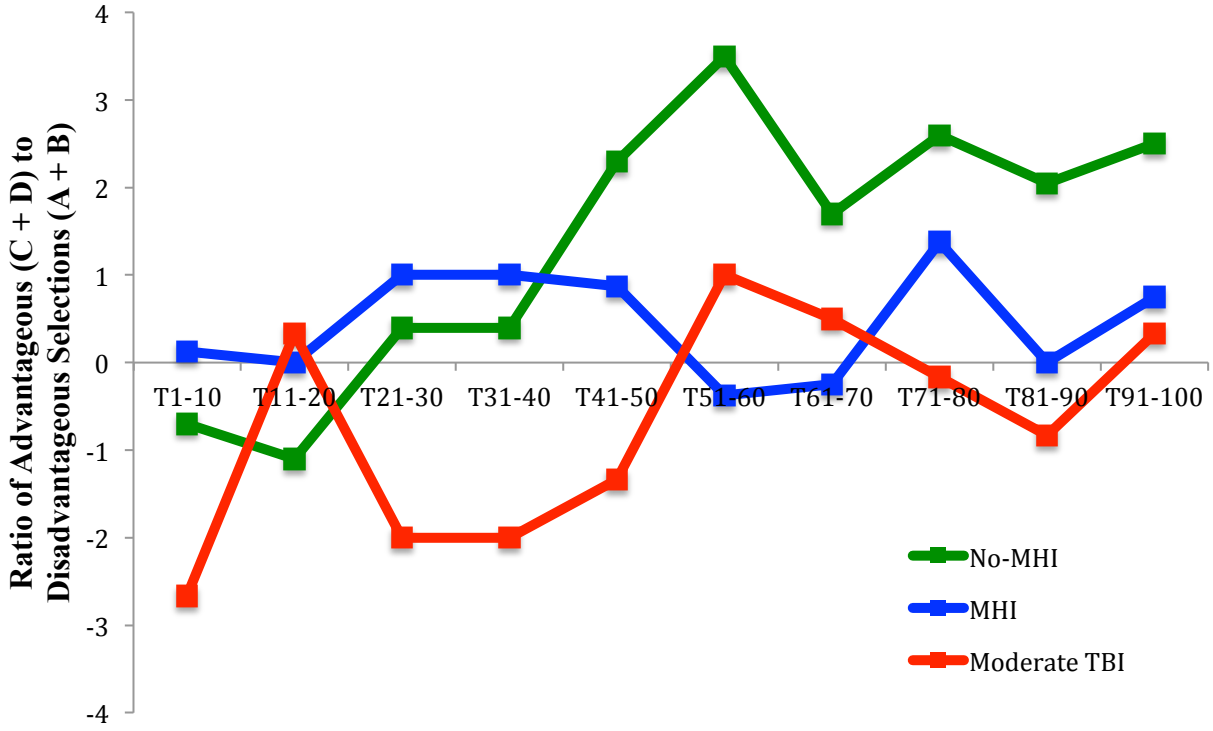

Figure 58: **Ratio of advantageous (C + D) to disadvantageous (A + B) selections on the Iowa Gambling Task (IGT) across 10 trial blocks as a function of head injury status for participants reporting higher depressive symptomatology**

Lastly, initial testing physiological arousal was found to correlate with self-reported affective (measured by SA-45) and somatic (measured by BDI) depression scores for the MHI group (Figure 59; affective depression: $r = .32$, $p = .045$; somatic depression: $r = -.36$, $p = .04$), but not the no-MHI group and moderate TBI groups respectively (Figure 60 and 61; $p > .05$)[23]. This illustrates, that for the MHI group, low EDA is associated with higher rates of somatic depression but lower rates of affective depression.

[23] Affective and somatic depression scores were derived from summing items 1, 2, 3, 27 and 42, and 11, 15, 16, 17, 18, 19, 20 and 21 from the SA-45 and BDI respectively. These sums were then multiplied by 3 and 5 respectively to equate their scales.

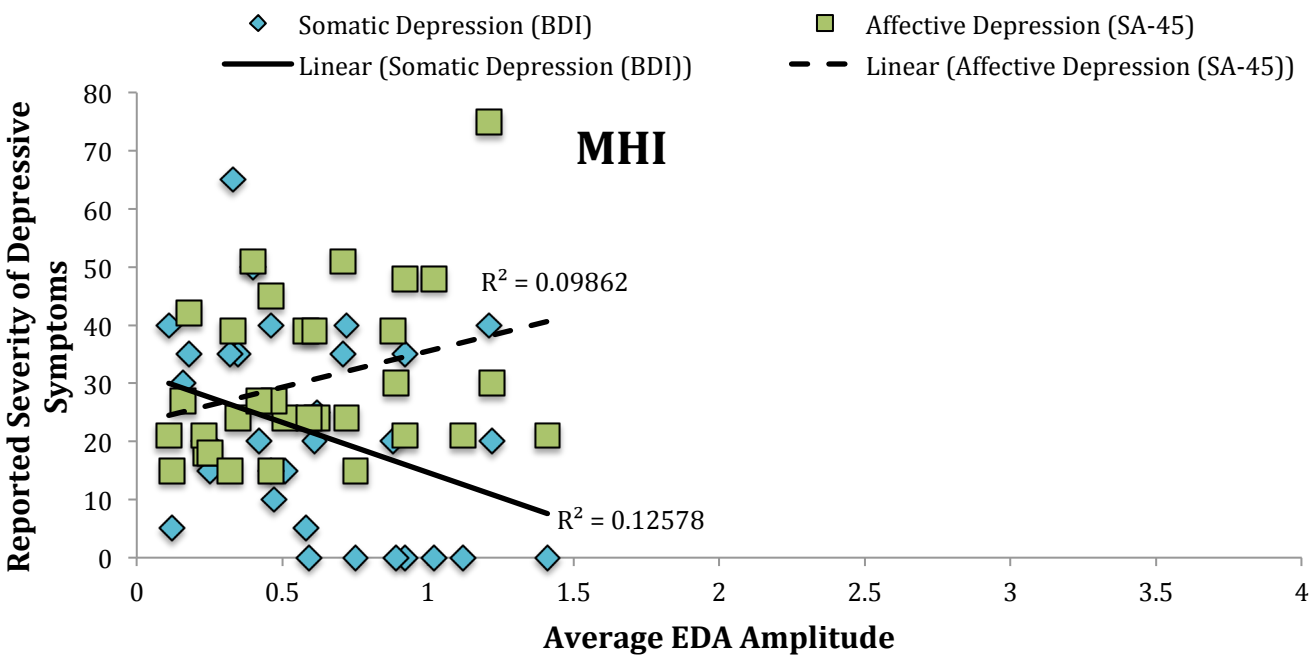

Figure 59. **Scatter plot depicting the relationships between self-reported affective (SA-45) and somatic (BDI) depressive symptomatology with initial testing physiological arousal as measured by EDA for the MHI group**

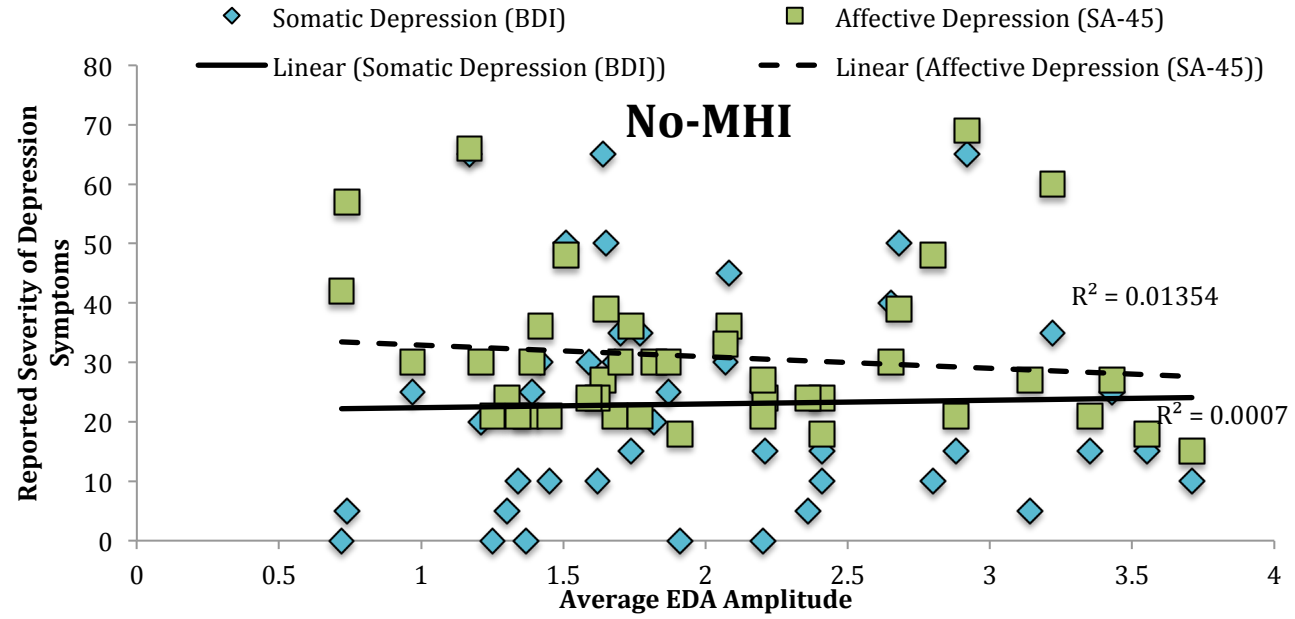

Figure 60. **Scatter plot depicting the relationships between self-reported affective (SA-45) and somatic (BDI) depressive symptomatology with initial testing physiological arousal as measured by EDA for the no-MHI group**

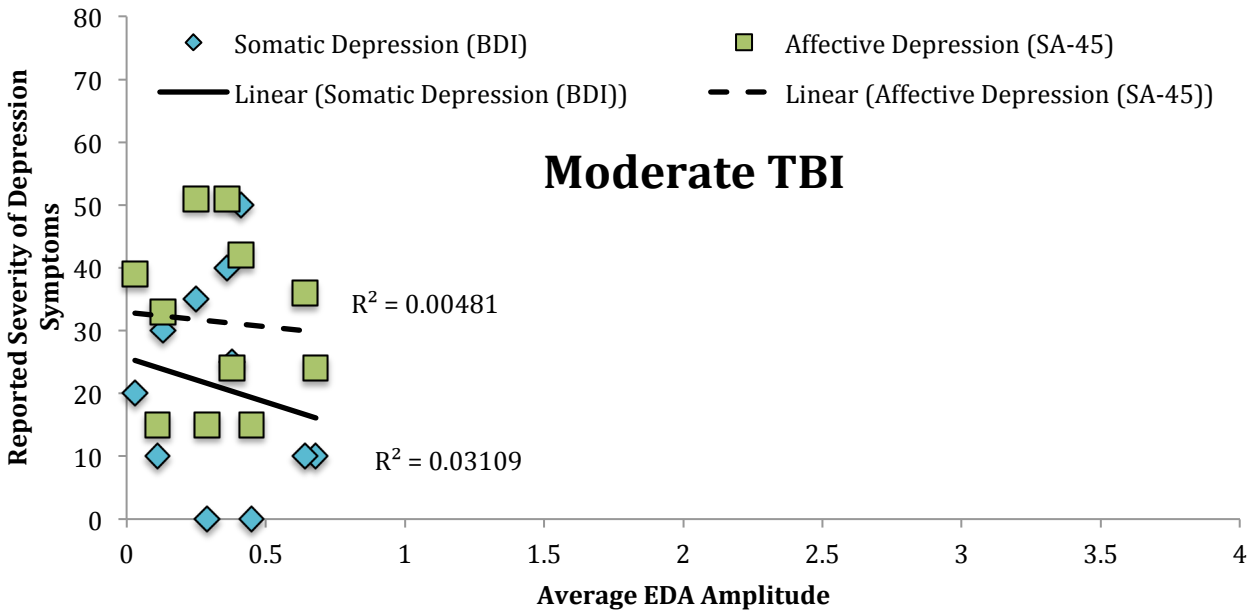

Figure 61. **Scatter plot depicting the relationships between self-reported affective (SA-45) and somatic (BDI) depressive symptomatology with initial testing physiological arousal as measured by EDA for the moderate TBI group**

Discussion

Overall, this study examined two major barriers to social competency following head injury - decision-making and depressive symptomatology in individuals with MHI and moderate TBI as a means to provide greater insight into the continuum of TBI. Research pertaining to long-term life satisfaction following TBI and client-centered rehabilitation goals primarily emphasize socioemotional symptomatology that pose barriers for achieving social reintegration. TBI survivors frequently report that the immediate goals addressed postinjury are focused on physical and cognitive rehabilitation, while long-term goals pertain to social rehabilitation (Burleigh et al., 1998; Corrigan et al., 2001; LoBello et al., 2003), and it is these social goals that are more meaningful in terms of community integration. Given that the research is limited in examining community integration, this study investigated variables that impact these longer-term social and life satisfaction outcomes. More specifically, this study investigated the relationship between decision-making processes under conditions of uncertainty (not unlike social situations)

as a function of injury severity and provide greater insight regarding the role of physiological (under)arousal in persons who have experienced injuries to the head. Moreover, this study explored whether arousal-based therapeutic techniques for MHI/TBI populations may serve as a potential future therapeutic strategy. Lastly, this study attempts to highlight the potential role of functional and structural alterations to the OFC using indirect neuropsychological measures of decision-making and processing of emotionally-salient feedback, and to examine its impact on depressive symptomatology in individuals who have reported neural injuries.

Minimal research has been published investigating individuals who have sustained very mild injuries, using a liberal definition of MHI such that a loss of consciousness is not required. This study serves to map decision-making processes and physiological arousal across the injury severity continuum and illustrate that injury severity continues to be a major underlying factor contributing to persistence of neurocognitive and emotional sequelae following head injuries (Green & Iverson, 2001; Iverson, 2005; Ruff et al., 2009). Much of the literature has examined mild TBI by obtaining participants from head injury clinics, hospitals, and physician/psychologist's private practices (Belanger et al., 2005), and the fact that these individuals are commonly involved in litigation or other legal precedings (that ultimately pertain to financial gains) may confound and/or exacerbate their performance is unfortunately cited as a rational to discount the persistence of their postinjury symptomatology (i.e., individuals are malingering for greater compensation; e.g., Green & Iverson, 2001; Iverson, Green & Rogers, 1999; Slick, Iverson, & Green, 2000). An advantage of the present study is that the recruited subjects in this study had no knowledge that the study was investigating head injury per se. The University sample which makes up the non-injured and MHI cohorts were recruited from a post-secondary education setting and to our knowledge, were not complaining of any

neuropsychological sequelae or involved in legal or compensation procedures. If persistent, albeit clinically insignificant, neurocognitive changes can be illustrated in the most mild forms of MHI, these finds can provide credence to the findings that those with more substantive injuries report persistence in post-concussive symptoms.

Summary of Findings

Underarousal.

It was expected that individuals who have sustained head injuries would illustrate a pattern of physiological underarousal as a function of head injury severity (i.e., participants with moderate TBIs would illustrate a pattern of lowest arousal, followed by those with a MHI and lastly those without an injury). Physiological arousal was measured using several different indices, including EDA, pulse and salivary cortisol, measures of sympathetic activation and HPA chronic stress activity respectively (Lykken & Venables, 1971).

As hypothesized, MHI and moderate TBI participants produced lower arousal prior to task EDA and pulse rates relative to non-injured participants, but the two injury groups were not shown to be statistically different from one another, despite mean values being in the expected direction, such that participants with moderate TBI had less physiological arousal than MHI subjects. It is possible that the moderate TBI participants reached a floor effect with respect to their EDA, and could not produce a significant difference relative to the MHI group, and the small number of subjects considerably limits the amount of power in this analysis. In general, the results support the hypothesis that individuals who sustain neural injury experience a pattern of dampened physiological arousal, a finding which is consistent with the literature implicating the OFC in sustaining subtle functional disruption following head injuries (Bigler & Orrison, 2001; Rolls, 2004; Wallis, 2007). The OFC receives extensive projections from all sensory modalities

(Carmichael & Price, 1995a; Rolls, 2004; Wallis, 2007) and has bidirectional projections with limbic structures (i.e., amygdala and hypothalamus) which serve to modulate autonomic arousal through afferent connections to the periaqueductal gray area, reticular formation and raphe nucleus, respectively (Barbas et al., 2003; Carmichael & Price, 1995b; Kadda et al., 1949). As a result, disruption to the OFC is likely to impair one's capacity to regulate and modulate autonomic arousal in an optimal fashion based on an informed summation of environmental and cognitive inputs (Barbas et al., 2003; Bechara et al., 2000a; Bechara et al., 1996). This attenuated physiological arousal represents a possible dampening of "physiological preparedness" for one's environment, leaving individuals who have sustained neural injury to be less vigilant with respect to their environmental situation and subsequently less capable in responding in optimal or proportional fashions (Bechara et al., 1996; 2000a; 2000b; Damasio, 1994). This is a finding that we have been able to replicate a number of times in our lab (Baker & Good, 2014; van Noordt & Good, 2011; van Noordt, Chiapettia, & Good, in press)[24].

In contrast, the measures of salivary cortisol produced a tendency for hypercortisolemia in the moderate TBI and MHI groups relative to their non-injury cohort. While this prediction of hypercortisolemia for this sample was exploratory in nature, the heightened result was not expected. Instead, it was hypothesized that the cortisol results would mimic other forms of underarousal as measured by EDA and pulse. Very little research has examined the impact head injury has on subtle pituitary functionality changes. There is, however, evidence that upwards of 40% of individuals with moderate TBI injuries develop clinical syndromes indicative of pituitary challenges (i.e., diabetes incipidus, hypoprolactinemia, growth hormone deficiency, etc.; Bondanelli, Ambrosio, Zatelli, Marinis, Uberti, 2005). One study, conducted by Bay and

[24] This finding has been replicated in a number of unpublished data sets, including various projects within the Neuropsychology Cognitive Research Lab.

Sikorskii (2009), reported an increase of hypercortisolemia across a 12-hour period postinjury. However, this finding of hypercortisolemia is not entirely inconsistent with the underarousal hypothesis proposed for other measures of physiological arousal, given that it was not a baseline measure, but rather was collected during the testing session. In line with evidence demonstrating that individuals who have sustained neural injury and are underaroused (e.g., Bechara & Naqvi, 2009; Ciaramelli, Muccioli, Ladavas, & di Pelligrino, 2007) their lack of "physiological preparedness" may frequently result in less anticipation or expectations (particularly for long-term consequences), and thereby, greater surprise, and/or distress associated with new environments or situations (which is likely to happen more often for this group relative to non-injured groups; e.g., Baker & Good, 2014). This greater perceived stress could then result in greater activation of the hypothalamus-pituitary-adrenal (HPA) axis, resulting in greater release of cortisol (e.g., Baker & Good, 2014). This is also potentially confounded with a greater HPA axis dysregulation or adrenial hypersensitivity following neural injury, with less capacity to "put the brakes" on stress responses and may not in fact reflect increases in subjective stress. These possibilities would be the basis for future studies (e.g., baseline cortisol would be expected to be low, then rise in reaction to introduced events, and given sufficient time to adjust to the environment, should drop again, and at a rate faster and lower than observed for non-injury participants)[25]. Further research is required to disentangle these possible explanations.

As hypothesized, injury severity measured by a composite variable that puts injury severity on a continuum did predict physiological arousal (i.e., EDA and pulse); and a graduated impact on arousal was observed across injury groups; however, this was not observed for PCS.

[25] Unfortunately this cannot be tested in this dataset, as all participants eventually underwent an arousal manipulation. Despite this, the pattern of results observed here is all participants had decreasing salivary cortisol concentrations with time while the pattern of head injury status remained (moderate TBI > MHI > No-MHI).

Together, EDA and pulse predicted 41.4% of injury severity. This builds on growing body of literature illustrating that severity of injury is predictive of neurocognitive outcome (Dunning et al., 2004; Goldstein & Levin, 2001; van der Naalt, van Zomeren, Sluiter, & Minderhoud, 1999; Whyte et al., 2001), but now adds to this literature by extending the finding across the spectrum of TBI to mild uncomplicated head injury. This implies that indices of injury severity such as duration of altered state of consciousness and LOC, among other variables, are more informative and predictive of outcome than PCS. Interesting, it was found that injury severity was predictive of the amount of PCS reported by participants, with those having greater injuries also reporting more PCS. There is an emerging body of literature showing that PCS is not highly predictive of outcome (Hukkelhoeven et al., 2005; Ponsford, Draper, & Schoneberger, 2008; Posford et al., 2011), as the symptomatology is highly common among the non-head injured population (Dean, O'Neill, & Sterr, 2012; van Reekum, 2013; Zakzanis, & Yeung, 2011), but is a useful proxy of recovery postinjury (King & Kirwilliam, 2011; Silver, 2014). Interestingly, there is accumulating evidence that the relationship between injury severity and measures of emotional, behavioural, social, and cognitive capacity is more strongly predicted immediately postinjury, but as time progress, more variables, such as access to rehabilitative resources, degree of inclusion into social/occupational programs, comorbid psychiatric disorders and many other variables, are likely to become increasingly influential and moderate this relationship (e.g., Draper, Ponsford, & Schonberger, 2007; Ezrachi, Ben-Yisbay, Kay, Diller, & Rattok, 1991; Novack et al., 2001; Walker et al., 2010; Wood & Rutterford, 2006). As a result, injury severity becomes less predictive of functional outcome with time as other variables become influential. Given the average 'time since injury' for our subjects was 7.25 years, it is very likely that had participants undergone testing closer to their injury, more variability in outcome measures would

have been accounted for by injury severity.

During the decision-making task (IGT), participants' physiological arousal was measured three times - at initial testing, in anticipation of making selections (i.e., recording of 8-seconds prior to a selection) and in response to positive and negative feedback (i.e., recording 4-seconds after selection). It was predicted that the underaousal would persist during the anticipation of making a selection on the IGT, but no physiological differences would be found during the feedback (outcome result as a function of selection made – i.e., win/loss), such that participants with moderate TBIs would illustrate a pattern of lowest arousal, followed by those with a MHI and lastly those without an injury. This prediction was partially supported. The MHI and moderate TBI groups produced lower anticipatory arousal relative to control participants, however the injury groups did not differ from one another. The groups did not differ during feedback, regardless of whether it was following a reward or punishment trial. These results replicate the findings that were observed in our previous studies examining MHI and non-MHI university students (Robb & Good, 2011; 2012) and results obtained by van Noordt & Good (2011; van Noordt, Chiapettia, & Good, in press) in a similar university population. The present study extends these findings to the moderate TBI population.

Studies in our lab have reproduced findings similar to what was found in the pivotal study conducted by Bechara and colleagues (1996) who originally illustrated patients with VMPFC lesions had reduced skin conductance responses (SCR) compared to controls in anticipation of making selections on the IGT. Bechara, Damasio and colleagues suggest that reduced indices of autonomic arousal in anticipation of making a decision reflect a neutrally-based attenuation of physiological preparedness, which may, in turn, reflect the dampening of bioregulatory somatic markers that typically aid in the guiding behaviour in advantageous

fashions (Bechara et al., 1996; 2000a; 2000b; Damasio, 1994; 1996).

To expand on Bechara's original work, anticipatory physiological arousal was mapped across 10-trial blocks and as a function of each deck of cards on the IGT (Decks A through D) and head injury status. As predicted, those who had sustained injuries illustrated a consistent pattern of underarousal across the last 50 trials, with no differences observed between injury groups, whereas no-MHI illustrated increasing anticipatory arousal as trials progressed for all but one of the decks of cards (Deck C). For Deck B, the MHI group also demonstrated increasing arousal across trials, but remained proportionally less than that of the no-MHI group; the moderate TBI group continued to illustrate flattened EDA response. For Deck C, no group differences were observed, except at approximately the 40 to 50 trial mark, in which case the no-MHI produced increasing anticipatory arousal and injured groups remaining consistently underaroused. Again, these findings generally replicate our previous findings examining MHI in university students (Robb & Good, 2011; 2012). To our knowledge, these two studies that are the only ones that have examined anticipatory arousal in 10 trial blocks on the IGT in head injury populations.

Collectively, these findings support the suggestion that head injury groups illustrate a pattern of underarousal relative to their non-injured cohort as measured by indices of sympathetic nervous system. However, hypercortisolemia, as measured by salivary cortisol, was observed for injured groups relative to the non-injury, and may be an indication of 'reactivity' to unexpected events. Further, individuals who report a history of head injury illustrate a pattern of persistent underarousal in anticipation of selections on the IGT across the latter 50 trials of decision-making. Thus, this may reflect a reduced capacity for "physiological preparedness" at initial testing in participants who reported injuries, leaving them proportionally less vigilant, and

having less capacity to produce, or otherwise regulate, bioregulatory states or "somatic markers" which help to optimally inform participants about their environmental situation (Bechara et al., 1996; 2000a; 2000b; Damasio, 1994). Interestingly, statistical differences were not observed between the MHI and moderate TBI group, but the TBI group did illustrate less arousal at initial testing and less development of anticipatory arousal status relative to their MHI counterparts. Lastly, injury severity was partially predictive of measures of arousal at initial testing, which was partially consistent with our hypothesis, and, in turn, supports a growing body of literature suggesting that outcome, regardless of nature (i.e., physiological, behavioural, emotive, social, etc.), is moderated by a great number of factors outside of injury severity.

IGT Performance

It was predicted that performance on the IGT would vary as a function of injury status and severity whereby persons with moderate TBI will be slower at transitioning from disadvantageous selections to advantageous selections, than individuals who report mild head injuries who, in turn, will be slower to transition than non-injured participants. Moreover, the rate of return to a punishing (disadvantageous) selection will be faster (i.e., fewer trials in returning to a selection after being punished by that selection previously) as a function of injury severity.

A ratio of decision-making performance was computed using Bechara et al. (1996)'s calculation, by subtracting disadvantageous selections (decks A + B) from advantageous selections (deck C + deck D). As hypothesized, participants with head injuries demonstrated a slower rate of learning to adjust their selections towards advantageous decks relative to their non-injured cohort. Despite exposure to punishment, those who report a history of MHI illustrated an increased propensity for continuing to make a 'risky' decision which has been

described by Bechara and his colleagues to reflect a less sensitivity to future consequences, similar to that found with VMPFC lesioned patients (Bechara et al., 1994; 1996; 1999; 2000a; 2000b). These results also parallel a number of studies that have found impaired IGT performance in catastrophic TBI individuals (Bonatti et al., 2008; Cotrena et al., 2014; Levine et al., 2005; Wiederkehr et al., 2005) whose injuries have been related to the structural integrity of the VMPFC (Levine et al., 2005). MHI subjects' impairments to decision-making are subtle relative to these groups. They do make the transition from disadvantageous to advantageous selections, suggesting that they are learning from decision-making outcomes; however, they just do so a slower rate and never reach the same ratio of advantageous to disadvantageous selections. It is important to note that these are high functioning, cognitively capable University students who have been successful academically suggesting that while these findings are statistically significant, they are unlikely to be clinically meaningful.

While the two injury groups did not differ, there is little variation in selection patterns across the trials for the moderate TBI group in particular. Relative to the MHI group, the moderate TBI group presented very little variability in their choices and did not appear to learn from punishment trials. Their pattern of results most closely resembles that found with VMPFC/OFC-lesioned patients (i.e., continued sampling from the disadvantageous decks and receiving greater punishment overall – e.g., Bechara et al., 1994; 1996; 1999). Individuals with no lesions to this structure or lesions elsewhere do not display these impairments, and instead illustrate a pattern of decision-making indicative of "learning" such that selections gradually transition from disadvantageous to advantageous selections. Moreover, functional neuroimaging studies, using PET and fMRI, have implicated the OFC and various other structures involvement in the "somatic marker network" (i.e., ACC, DLPFC, inferior parietal cortex, etc.) in healthy

participants (Ernst et al., 2002; Fukui, Murai, Fukuyama, Hayashi, & Hanakawa, 2005; Lawrence, Jollant, O'Daly, Zelaya, & Phillips, 2009; Northoff et al., 2006; Windmann et al., 2006) and have been contrasted with abnormalities in clinical populations with known decision-making abnormalities under conditions of uncertainty (i.e., substance abusers, mood disorders, etc.; e.g., Frangou, Kington, Raymont, & Shergill, 2008; Tanabe et al., 2007; Tucker, Potenza, Beauvais, Browndyke, Gottschalk, & Kosten. 2004). Lastly, the extensive neuroanatomical literature, as previously reviewed, outline the OFC's neural connections with neural structures presumed necessary for optimal decision-making performance, including sensory information, limbic structures/effector structures (implicated in producing somatic states), higher cognitive structures (i.e., DLPFC, ACC, etc.; Barbas et al., 2003; Cardinal et al., 2001; Carmichael & Price, 1995a; Carmichael & Price, 1995b; Crottaz-Herbette & Menon, 2006; Kalat, 2010; Kolb & Whishaw, 2009; Lezark et al., 2012; Rolls, 2004; Wallis, 2007).

Based on this literature, altered IGT performance in the context of the OFC being particularly vulnerable to the biomechanical forces that are applied to the brain during head injury suggests that this area of the brain (and possibly related neural circuitry) is implicated in the sequelae following head injury, both in the MHI group, albeit more subtly, and the moderate TBI group. This coincides with the underarousal observed in head injury groups. Injury groups illustrated impaired elicitation and/or maintenance of anticipatory physiological arousal and may reflect a neurally-based compromised affective insensitivity, with respect to "somatic markers", to consequences of decision outcomes (i.e., prior losses), resulting in lessened avoidance of disadvantageous decisions.

The underlying bases for these decision-making alterations in injury groups, have been speculated based on VMPFC/OFC lesion patients to reflect a lack of sensitivity to future

consequences, regardless if they are positive or negative. Bechara et al. (2000b) suggest that decision-making by persons with VMPFC/OFC lesions is driven by the immediate context, possessing what is commonly referred to as a "myopia for the future" (Bechara et al., 2000b). VMPFC-lesioned patients illustrate a hypersensitivity to reward and an insensitivity to punishment regardless of the magnitude or frequency of outcome (e.g., Yechiam, Busemeyer, Stout, and Bechara, 2005) as described by the expectancy-valence cognitive model with the original IGT.

In line with Bechara's findings, we found that head injury groups returned to a selection equally fast (i.e., frequency of trials) following a punishment trial for both advantageous and disadvantageous decks whereas the no-MHI group returned much more slowly (i.e., taking a greater number of trials)[26]. This suggests that head injury participants, similar to that observed with VMPFC/OFC-lesioned patients (Bechara et al, 2000b; Yechiam et al., 2005), may have increased difficulty in differentiating the relative risk of each of the decks. This finding replicates and expands upon a finding that we collected in individuals with and without MHI in a university sample (Robb & Good, 2012) and supports the hypothesis that the rate of return to a previously punishing selection for disadvantageous decks will be faster as a function of reporting a history of traumatic injury to the head, but not of severity per se.

Further, the relationships observed between injury severity and IGT performance was found to be linear, and, as expected, illustrated a negative relationship between learning from outcomes in the context of uncertainty as a function of injury severity, even in uncomplicated MHI. These results parallel the findings observed with injury severity and measures of physiological arousal. These findings extend the existing research, demonstrating that

[26] This was calculated on the last 80 trials to ensure that participant had an opportunity to sample from all of the decks.

neurocognitive outcome can be partially accounted for by injury severity, by replicating this in the uncomplicated mild high functioning population. Specifically, it was found that a decrease in IGT performance is observed as injury severity increases; as injuries become more severe, decision-making performance becomes less variable and less advantageous or successful in outcome and reflect poor performance. Individuals with reportedly less severe injuries demonstrate more variability, and more advantageous outcomes in decision-making. A possible interpretation of this is that the relationship is influenced by other variables, such as a more optimal reintegration into cognitive and social rewarding activities, which may modulate recovery from injury, and performance (Draper et al., 2007; Ezrachi, et al., 1991; Novack et al., 2001). As previously stated, most individuals who sustain a MHI will recover from their injury, but it remains illusive as to which factors best predict which individuals will experience a remission of their symptoms and which will continue to experience residual sequelae (Iverson & Lange, 2009; Ruff, 2011; Schoenberg, 2011). Further research is required to provide insight into these recovery variables.

Injury Severity, Physiological Arousal, and IGT Performance

Given the observed relationships between participants' retrospective reported symptomatology at the time of injury (injury severity) and physiological arousal in terms of (1) injury severity being related to both participants' physiological arousal, as measured by EDA, and their performance on measures of IGT decision-making, and (2) physiological arousal being predictive of decision-making performance, a meditational analysis was conducted. Physiological arousal was found to be a significant mediator between injury severity and decision-making performance, posing the possibility that one mechanism by which injury severity relates to decision-making is through changes in arousal. This provides indirect

additional support for the role of neural disruption in the VMPFC and associated neural connections responsible for optimal regulation and maintenance of bioregulatory states in changes to decision-making as a function of injury severity (Bechara et al., 1994; 1996; 1999; 2000a; 2000b; 2005; Reinmann & Bechara, 2010). Of particular interest is that this occurs even for very mild, uncomplicated injuries in individuals who are highly functioning university students, and remains true across the injury severity spectrum to documented moderate TBI.

Explicit Knowledge in the IGT

One of the major criticisms levied by Maia and McClelland (2004) against the Somatic Markers Hypothesis is that explicit knowledge about decision-making outcomes on the IGT may occur prior to implicit somatic markers reflected in physiological arousal. Limitations to this controversial study included the criticism that physiological arousal was not recorded and the nature of the questions they asked during the task may have changed the implicit characteristics. To clarify these findings, we assessed our subjects' explicit knowledge about the IGT contingencies at the end of the task so as to reduce the confound of eliciting, rather than witnessing, the emergence of explicit strategies and its influence on performance. We found that both individuals who had sustained a MHI and healthy controls produced a dissociation between what they reported was happening in the task (i.e., metacognition, explicit knowledge) and how they behaved (i.e., the selections and choices they made; implicit behaviour). This implies that implicit learning, perhaps guided by gut-feelings and physiological arousal, can drive decision-making performance independently of one's explicit knowledge of strategy (Robb & Good, 2012).

In the present study, we attempted to replicate these findings and predicted that regardless of head injury status, subjects' preferences would be independent of their making selection

choices during the IGT indicative of learning that these high risk decks are disadvantageous Interestingly, a dissociation was only partially observed - participants' preferences reported at the end of the task was found to match their performance/behavioural data on the IGT. On the other hand, participants' reported propensity for winnings (i.e., expected earnings as calculated for a deck had they selected a deck for all 100 trials calculated using Maia and McClelland's (2004) equation did not predict or reflect their behavioural choices. All participants reported that they expected to earn more on disadvantageous decks, despite no-MHI and MHI groups illustrating a transition from disadvantageous to advantageous, replicating our previous findings.

One possible explanation for dissociation between preference and participants' propensity for winnings is that preference taps into the construct of "gut-feeling" that is elicited by somatic markers, whereas questions pertaining to propensity for winnings were based on objective knowledge (i.e., average amount won and lost, frequency of losses, etc.) and were not "informed" by gut feeling. There is evidence that one's preference frequently diverges from cognitive-cold rational reasoning (Connolly & Ordonez, 2003). Another possible explanation is that both implicit and explicit knowledge of strategies is required for advantageous decision-making. This latter explanation has been particularly well supported in the literature, illustrating that those with amnestic syndromes perform poorly on the IGT (Guillaume et al., 2009; Gutbrod et al., 2006). Overall, a dissociation is observed between participant's explicit objective knowledge of the reward and punishment contingencies of the IGT and their respective behavioural performance. This finding provides support for the notion that the IGT is tapping into "gut-feeling" processes and thus participants are reliant on bioregulatary cues to guide decision-making processes.

A study by Persaud et al. (2007) also has since called into question the Maia &

McClelland (2004) findings. Using three different approaches, Persaud and his colleagues assessed explicit knowledge of IGT performance - a "no interruption" condition, an open question condition, and finally an interrupted condition using the Maia & McClelland questions. They found no differences between the task with no interruptions and the task with open questions which both served to replicate the results obtained by Bechara et al., (1997) supporting the indication of somatic markers occur prior to explicit knowledge (Persaud et al., 2007) whereas, they supported Maia & McClelland's results when they used their technique.

In summary, explicit knowledge reported by participants regarding propensity for winnings did not match their behaviour on the IGT, as predicted. Further, participants' reported preference for decks was related to their IGT behaviour, showing that both preference, and card selection, as implicit measures of performance, may tap into the construct of "gut feelings". In line with much of the recent literature, our results indicate that advantageous decision-making on the IGT is associated with two independent systems, both implicit somatic signals and explicit knowledge of strategies.

Arousal Manipulation on physiological arousal and IGT performance

Extensive research has examined the relationship between physiological arousal and cognition, finding that these are related in a curvilinear fashion (i.e., Yerkes-Dodson law; Hanoch & Vitouch, 2004; Lupien, Maheu, Tu, Fiocco, & Schramek, 2007; McEwen & Sapolsky, 1995; Salehi, Cordero, & Sandi, 2010; Yerkes & Doson, 1908). While the Yerkes-Dodson law generally describes the relationship between arousal and cognitive behaviour, it is likely that a complex system of neural processes jointly contribute to the cognitive changes in attentional, executive, decision-making, and memory processes, including catecholamine forebrain activation (Mair, Onos, & Hembrook, 2011), sympathetic nervous system transmitters (i.e.,

epinephrine, etc.) and HPA axis hormones (i.e., glucocorticoids, etc., for full review see: Mendl, 1999). To investigate a sample of this system and examine evidence that physiological underarousal results in sub-optimal decision-making in persons with a history of head injury, we manipulated arousal by exposing subjects to emotionally-evocative stimuli. Previous studies in our lab used classical music as a means to evoke arousal, as it has been shown to elevate mesolimbic activation and thalamic arousal in a number of fMRI studies in healthy subjects (Chanda & Levitin, 2013). In those studies, classical music did increase physiological arousal and accompanied improved learning rates on measures of decision-making and attenuated differences in the rate of return to risky selections following punishment feedback for individuals reporting a MHI, but not for those who did not report a history of MHI. Negatively-valenced music was more effective in improving the decision-making performance for MHI participants, whereas positively-valanced music was more effective for non-MHI participants.

In the present study, emotionally-evocative visual stimuli from the International Affective Picture System (IAPS) were used, as these too were found to benefit cognitive performance in persons with MHI (Baker & Good, 2012). It was predicted that emotionally-evocative imagery (i.e., arousal manipulation) would produce a corresponding increase in physiological arousal and this enhancement would inform, or otherwise influence, decision-making performance on the IGT.

The arousal manipulation did increase measures of physiological arousal (i.e., pulse and EDA) immediately following the arousal manipulation, but mirroring other studies (e.g., Baker & Good, 2014), the effect did not persist for the head injury groups. Heightened arousal does persist for the no-MHI group. These results collectively indicate that following an injury, individuals may not maintain the “priming effects” of emotional information for the same length

of duration as healthy controls. These findings may reflect the altered connections between the OFC and structures implicated in regulation, modulation and maintenance of autonomic arousal state, such as the amygdala and hypothalamus (Wallis, 2007).

Contrary to our results using classical music as the emotionally-evocative stimuli (Robb & Good, 2012), there were no differences observed in anticipatory arousal during the IGT as a function of the increasing arousal, nor were there observed improvements in decision-making performance on the IGT with either the number of trials required for transitioning from high risk to low risk choices or by the number of intervening trials before returning to high-risk decision following punishment feedback. The limited findings with respect to the arousal manipulation and IGT performance could be accounted for by the differences in stimuli exposure. For example, there are important differences between the types of stimuli used to evoke arousal in this study as compared to our previous ones. The IAPS pictures presented both positive and negative high arousal images *within subjects,* potentially leading to an 'averaging' of the arousal manipulation effect, whereas in the music studies, this manipulation (of necessity) was presented *between subjects* (Robb & Good, 2012). This could potentially account for the null finding observed here. Secondly, whereas in our earlier study the classical musical was played throughout the testing session, in the current study the IAPs pictures were displayed only prior to the task and only for 15 minutes in duration. As observed in other studies in which we have isolated the evocative stimuli from the testing (e.g., Baker & Good, 2014), longevity of emotional activation for persons with more resistant capability of physiological arousal may require persistent re-priming. Lastly, this challenge to the duration of evocative stimuli exposure was further aggravated by the fact that the IGT was assessed as the last test in a neuropsychological battery used to test participants' cognitive capacity across a variety of

domains. It is possible that had the IGT been placed in closer temporal proximity to the arousal manipulation, the emotional influence may have been more pronounced.

In summary, no effect of the arousal manipulation using IAPS was found for either the head injury groups or health control groups. Further research is required to investigate arousal-based therapeutic interventions in attempt to find one that will have more long-standing impacts for injured populations and are practically amenable for rehabilitation protocols and day-to-day living. Research has illustrated cognitive improvements as a function of increased physiological arousal in other populations, including cardiovascular exercise in elderly populations (Gates, Fiatarone Singh Kelly et al., 2014; Vaughan, Wallis, Polit, Steele, Shum, & Morris, 2014) and psychostimulants (i.e., methylphenidate, amphetamine salts, etc.) in ADHD (Berridge & Devilbiss, 2011; Rapport & Kelly, 1991; Spencer, Klein, & Berridge, 2012) and TBI populations (Nikles, et al., 2014; Tramontana, Cowan, Zald, Prokop, & Guillamondegui, 2014; Whyte, Vaccaro, Grieb-Neff, & Hart, 2002). Perhaps these would serve to improvement decision-making processes in head injury populations as well.

Decision-making Performance and Depression

Depressed mood has been identified as a considerable barrier to social reintegration (Brown et al., 2003; Gomez-Hernandez et al., 1997) and there appears to be an increasing link between TBI, particularly on the mild end of the injury severity spectrum, and depressive symptomatology (Jorge et al., 1993; Mooney & Speed, 2001; Seel et al., 2003). Moreover, overlapping neural structures between those vulnerable in traumatic head injury and in depression are being reported in the literature (Bremner et al., 1999; Drevets, 2007; Maller et al., 2010). Unfortunately, very little research has investigated the role that the pathophysiology of MHI may play in contributing to depressive symptomatology and whether the respective

socioemotional/cognitive sequelae following MHI may be accounting for some of the depressive symptomatology.

Two studies have investigated IGT performance in individuals with unipolar MDD and have found decision-making profiles similar to that observed in TBI and VMPFC lesion populations, characterized by a lack of transition from disadvantageous to advantageous deck selection. Specifically, these studies demonstrate a pattern of decision-making made by MDD participants such that immediate gains outweighed the negative impact of larger punishments they received overtime and, thus, they continued to make selections from disadvantageous decks (and did not transition to advantageous decks; Must et al., 2006; Jollant et al., 2010). This result is difficult to reconcile with the MDD literature as there is considerably research showing that individuals with MDD are highly sensitive to punishment (Eshel & Roiser, 2010; Roiser & Sakakian, 2013) and frequently experience anhedonia, whereby positive valence is less rewarding to them (American Psychiatric Association, 2012; Bylsma et al., 2008). Neither of these studies examined whether the samples had subjects with a history of MHI, and given that IGT performance differences have been observed as a function of MHI (van Noordt, & Good, 2011; Robb & Good, 2012), it is possible that the results pertaining to MDD and decision-making are confounded, or otherwise exacerbated, by MHI. As a result, this research served to provide clarification to these findings.

It was hypothesized that learning performance on the IGT, and the rate of return to punishing selections, would predict self-reported depressive symptomatology in individuals who report previously sustaining an injury but not for their non-injured cohort. MHI status was found to be a moderator of IGT performance and self-reported depressive symptomatology, whereby this relationship was found only for those who had sustained a MHI, but not for those without a

MHI or moderate TBI, accounting for 12.7% more of the variability. This relationship should be interpreted with caution, however, as the largest unique variability accounted for by any single trial block was 6.25%. Moreover, the rate of return to a disadvantageous selection following punishment was predictive of depressive symptomatology for the MHI group only. While these findings do not provide conclusive evidence that neuropsychological indices of OFC functionality are related to depressive symptomatology for those with a MHI, it does provide justification for further exploration as to the role that MHI may be playing in the presentation of depression. This finding does relate to the Kweon & Rho (2005) research suggesting that depressive symptoms are not uniform across the TBI population (Bahraini et al., 2013). They observed that individuals with MHI report more depressive symptomatology, particularly suicidal ideation, relative to moderate TBI subjects.

Supplementary analyses using a median-split procedure was performed to separate individuals into two groups based on their respective depression symptoms (based on each injury group): lower and higher self-reported depression symptoms. This procedure reflects the analyses conducted in the Must et al. (2006) and Jollant et al. (2010) studies. As observed in Figure 54, and consistent with the correlation analyses, there were no differences in IGT performance for the healthy control participants; IGT performance does not differ as a function of depression score. This further supports the notion that the findings reported by Must et al. (2006; Jollant et al., 2010) were confounded by a third variable, such as MHI.

If these findings are found to be reliable in acute psychiatric populations, , they could pose significant ramifications for the treatment literature regarding MDD. It is well acknowledged that there is considerable heterogeneity in the MDD population with respect to symptomatology and their etiological contributions (Albert & Benkelfat, 2013; Barch, 2013; Mill

& Petronis, 2007; Rush, 2007; Villanueva, 2013). Additionally, treatment responses are incredibly variable and there is little insight into the mechanisms that can account for this variability (Garthlehner et al., 2011; Hansen, Gartiehner, Lohr, Gaynes, & Carey, 2005; Kupfer, Frank & Phillips, 2012: Leucht, Hierl, Kissling, Dold, & Davis, 2012; Nemeroff et al., 2003). Our findings imply that head injury status may be an important potential contributor to variability, that is, MHI history of persons with MDD accounts for, or enhances, the challenged decision-making and additionally may reflect "hot" cognitive components associated with the depressive symptoms (i.e., decision-making impairments) following head injury. Another possibility is that the "underarousal" sequelae following head injury may present like, and otherwise appear like, the depressive symptomatology of MDD from a clinical presentation standpoint. That is, the emotional blunting (i.e., loss of interest and pleasure) and motivational/initiation difficulties that are observed in persons with traumatic head injury, as well as the evidence of emotion dysregulation (i.e., irritability, agitation, etc.), may initially appear as, and be described by and confusable with, depressive symptoms, but only mimics MDD. Interestingly, decreasing physiological arousal was found to relate to increasing self-reported of somatic but not affective depressive symptoms, suggesting that while individuals with MHI may appear "depressed", it is actually the somatic aspects of depression which are pronounced and may just be the behavioural representation of underarousal.

Regardless of the exact etiology, whether it reflects a possible "hot" cognitive correlate of depression symptoms in head injury populations or is a mischaracterization of underarousal sequelae, these findings with replication could have important implications for treatment paradigms. These socioemotional cognitive barriers (i.e., hot cognition) may impede treatment responses in a number of different ways, including additional etiological contributions (e.g., OFC

functionality differences), compliance, engagement in treatment, decision-making outside of treatment potentially leading to adverse outcomes, and response to psychotropic medications. Considering MHI when investigating MDD (and every psychopathology) may be fundamental to better characterizing the contributions to clinical heterogeneity, an area that requires considerably more investigation.

Chapter III: Future Research Implications, Study Limitations and Conclusions

Future Research Implications

The observe findings of underarousal and alterations in decision-making and learning on the IGT are consistent with the Somatic Marker's hypothesis proposed by Damasio and colleagues (1994, 1996), as this population is likely to have sustained alterations to the OFC and related neural processes given its high susceptibility in TBI (Bigler & Orrison, 2001; Wallis, 2007). This influence on decision-making, especially under conditions of uncertainty, has implications for social reintegration for individuals who sustain head injury (Body, 2007; Matussal, 2013). The ability to develop and maintain close interpersonal relationships with peers, friends and family members is dependent on one's social competence, or the capacity to maintain appropriate and positive social relations and cognitions with others utilizing effective social skills in the absence of maladaptive behaviours, regulate one's emotions and behaviours to meet social expectations, and utilize socio-emotional information to interact and respond appropriately (Feldman Barrett & Salovey, 2002; Wong, 1998). These social skills require highly complex processes and continuous monitoring and adjustment based on contextual feedback, and involve a vast number of neural systems (Lezak et al., 2012). VMPFC-lesioned patients (i.e., Phineas Gage, E.V.R, etc.) have revealed particular deficits in social competency, decision-making, and awareness, despite intact intellectual functioning (Damasio, 1994; Damasio, 1996; Eslinger &

Damasio, 1985). Persons with less precise traumatically-induced head injuries also struggle with social competency, particularly moderate TBI individuals who have been found to frequently lose lateral relationships and challenges with social reintegration (Milders, Fuchs, & Crawford, 2003; Yeates et al., 2004). Individuals with milder head injuries, on the other hand, are likely to experience social difficulties that reflect riskier choices which may lead to less favourable, more adverse, outcomes, likely to be attributed to, and confused with, reflecting individual differences, "character" flaws (i.e., "bad", "annoying", "poorly-mannered" individuals) or personality disturbances.

Study Limitations

This study has a number of limitations. Firstly, a potential limitation pertaining to the MHI sample in this study is a lack of formalized medical evidence to substantiate self-reported head injury status provided by participants. However, note that typically medical evidence does not exist for these types of injuries since participants do not seek out medical attention (i.e., only 40% of the MHI sample reported seeking medical attention and this was considerably higher than previous studies conducted in our laboratory; Baker & Good, 2014; van Noordt & Good, 2011) viewing the mild nature of the injury as not requiring medical intervention. When participants did seek medical attention, it is a rarity that diagnostic procedures (i.e., neuroimaging, neuropsychological assessment, etc.) were employed outside of clinical interview strategies since only half of participants reporting a MHI also experience an LOC and Canadian guidelines notes that Computerized Tomography (CT) scans are to ordered only if a subject's Glasgow Coma Scale (GCS) remains less than 15 two hours postinjury (Stiell et al., 2002). Furthermore, even if neuroimaging has been undertaken, it would have been unlikely to provide any confirmatory results, given that the neuroimaging methodologies used in the majority of

medical centers (i.e., CT, MRI, etc.) do not have the specificity to detect neural changes typically observed in these mild forms of injury (Bigler, 2010; Bigler, 2013; Bigler, 2014; Bigler, & Bazarian, 2010; Bigler & Maxwell, 2012; Leninger al., 1990; Livingston et al., 2000; Ono et al., 2007; Voller et al., 1999; Yarnell & Rossie, 1988).

Furthermore, the cross-sectional nature of this study limits the degree to which causality can be attributed to findings of underarousal and decision-making performance following head injury. It is possible that physiological underarousal and greater risk-taking (as measured by the IGT) may reflect the influence of underlying personality trait(s) in certain individuals that make them more prone to sustaining a MHI, and thereby is not reflective of MHI per se (i.e., impulsivity is associated with lowered physiological arousal and, as a trait, leads to risk-taking behaviour including increased possibility of head injuries). However, one piece of evidence that speaks to this is the degree of decision-making changes and underarousal that is observed across the injury spectrum from mild to moderate injuries; that is, decreases in optimal decision-making and physiological arousal is associated with evidence of increases in severity of head injury (Baker & Good, 2014; van Noordt & Good, 2011). Similarly, an interaction between preinjury personality traits and the neural disruption to produce the clinical presentation following head injury may be contributing to our findings (e.g., Silver, 2014; Silver, McAllister, & Arciniegas, 2009). Further research is required to clarify this causal relationship and the degree to which each of these variables contributes to clinical outcome postinjury and the nature of their respective interactions.[27]

An additional limitation is the generalizability of these findings since our sample was recruited exclusively from the university setting. While having cognitively capable subjects (i.e., attained higher education), and those of milder injuries, was intentional, it is also self-selecting

[27] A current study in our lab is undertaking this project.

highly functioning participants who had more minor injuries, or otherwise were resilient and able to overcome and/or manage any deleterious effects of traumatic injuries. These subjects may not be representative of individuals with MHI who did not, or could not, pursue further formal education. Furthermore, our sample reports living in upper middle class families, which may also reduce the generalizability of our findings in two ways. Firstly, individuals of low socioeconomic status are more likely to sustain head injuries (Bruns & Hauser, 2003; Roozenbeek, Maas, & Menon, 2013), and secondly, greater social resources have been well established as a protective factor for functional recovery, particularly for pediatric TBI (Anderson et al., 2006; Taylor et al., 1995). This does imply that the observed neurocognitive sequelae observed in this sample may be a conservative estimate relative to low socioeconomic populations. Future research should consider recruiting samples from the broader community to increase representativeness.

Conclusions

Despite these limitations, this cross-sectional study illustrates that observable, and predictable, differences in physiological arousal (i.e., EDA, pulse, salivary cortisol) and decision-making occur as a function of reporting a previous head injury. Alterations in decision-making processes can accompany even mild forms of head injury and can be mapped along a continuum of injury severity. Those with head injury were found to demonstrate reduced anticipatory arousal and decreased learning (i.e., slower transition from disadvantageous to advantageous elections) on the IGT, with greater impairment observed for persons with more serious injury, as in the moderate TBI group. Moreover, both groups demonstrated a propensity for continuing to make riskier, less advantageous, decisions despite exposure to punishment as compared to their non-injured cohort. Injury severity was found to be highly predictive of

outcome, both of physiological arousal and of decision-making performance. Physiological arousal was found to mediate the relationship between injury severity and decision-making performance. This provides additional support for the role of physiological arousal, and thus indirectly the VMPFC and respective neural correlates related to this area, in affecting decision-making as a function of head injury, akin and consistent with the Somatic Marker Hypothesis (Bechara et al., 1996; 2000a; 2000b; Damasio, 1994). Furthermore, this research implies that an injury to the head that is sufficient to produce a state of altered consciousness should not be considered as a trivial event in one's medical history and has significant implications for social outcomes and choices decision-making and these will serve as significant barriers to social rehabilitation and integration.

In addition, further support for Damasio and colleague's Somatic Markers Hypothesis was observed. Participants' explicit knowledge of strategy on the IGT could not account for their decision-making performance; whereas, similar findings to VMPFC patients and in contrast to controls or a no-MHI cohort, injury groups illustrated anticipatory underarousal and poor decision-making performance. Unfortunately, the arousal manipulation using emotionally-evocative picture stimuli was not associated with a performance improvement; however, this may be due to the limited persistence of the manipulation. Improving the duration of arousal enhancement may in fact alter and optimize performance in decision-making, consistent with the Yerkes-Dodson relationship, for this population. Future studies aimed at the endurance of arousal manipulations will be pursued. Lastly, evidence that head injury may contribute to the relationship between IGT performance and depressive symptomatology was obtained. Further research is required to determine whether head injury is a confounding factor in much of the depression literature, since while many studies control for significant neural trauma, they do not

reportedly control for MHI per se. Given that risky decision-making and depression can be significant barriers to social interactions and interpersonal relationships, this study has implications for the possible mechanisms influencing these outcomes and, therefore, how to address, or possibly improve, successful social reintegration following head injury.

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Appendix A: Statistical Analyses

Table 7:

Summary of results for a mixed model 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 5 (Trial Blocks [trials 51-60, trials 61-70, trials 71-80, trials 81-90, trials 91-100]) × 2 (Arousal Manipulation status [pre-exposure, post-exposure]) ANOVA for anticipatory EDA amplitude prior to selecting Deck A on the IGT across the last 50 trials as a function of arousal manipulation condition.

Source	*Df*	*F*	*p*	η_p^2
	Between Subjects Effects			
Head Injury Status	2	15.64	< .001	.31
Arousal Manipulation	1	.88	.35	-
Head Injury Status × Arousal Manipulation	2	1.31	.28	-
Error	69			
	Within Subjects Effects			
Trial Blocks	2.43	3.15	.02	.04
Trial Blocks × Head Injury Status	4.86	3.34	.007	.09
Trial Blocks × Arousal Manipulation	2.43	.49	.75	-
Trial Blocks × Head Injury Status × Arousal Manipulation	4.86	.13	.99	-
Error	167.76			

Note: Sphericity could not be assumed, as Mauchly's W was significant. As a result, a Greenhouse-Geisser correction of degrees of freedom was made.

Table 8:

Summary of results for a mixed model 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 5 (Trial Blocks [trials 51-60, trials 61-70, trials 71-80, trials 81-90, trials 91-100]) × 2 (Arousal Manipulation status [pre-exposure, post-exposure]) ANOVA for anticipatory EDA amplitude prior to selecting Deck B on the IGT across the last 50 trials as a function of arousal manipulation condition.

Source	*Df*	*F*	*p*	η_p^2
	Between Subjects Effects			
Head Injury Status	2	9.19	< .001	.20
Arousal Manipulation	1	.63	.43	-
Head Injury Status × Arousal Manipulation	2	.29	.75	-
Error	73			
	Within Subjects Effects			
Trial Blocks	3.55	1.74	.02	-
Trial Blocks × Head Injury Status	7.11	1.55	.15	-
Trial Blocks × Arousal Manipulation	3.55	.44	.65	-
Trial Blocks × Head Injury Status × Arousal Manipulation	7.11	.62	.65	-
Error	259.46			

Note: Sphericity could not be assumed, as Mauchly's W was significant. As a result, a Greenhouse-Geisser correction of degrees of freedom was made.

Table 9:

Summary of results for a mixed model 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 5 (Trial Blocks [trials 51-60, trials 61-70, trials 71-80, trials 81-90, trials 91-100]) × 2 (Arousal Manipulation status [pre-exposure, post-exposure]) ANOVA for anticipatory EDA amplitude prior to selecting Deck C on the IGT across the last 50 trials as a function of arousal manipulation condition.

Source	*Df*	*F*	*p*	η_p^2
	Between Subjects Effects			
Head Injury Status	2	14.57	< .001	.28
Arousal Manipulation	1	.15	.70	-
Head Injury Status × Arousal Manipulation	2	.17	.85	-
Error	75			
	Within Subjects Effects			
Trial Blocks	1.79	.58	.54	-
Trial Blocks × Head Injury Status	3.57	2.49	.05	.06
Trial Blocks × Arousal Manipulation	1.79	.44	.62	-
Trial Blocks × Head Injury Status × Arousal Manipulation	3.57	.30	.49	-
Error	134.00			

Note: Sphericity could not be assumed, as Mauchly's W was significant. As a result, a Greenhouse-Geisser correction of degrees of freedom was made.

Table 10:

Summary of results for a mixed model 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 5 (Trial Blocks [trials 51-60, trials 61-70, trials 71-80, trials 81-90, trials 91-100]) × 2 (Arousal Manipulation status [pre-exposure, post-exposure]) ANOVA for anticipatory EDA amplitude prior to selecting Deck D on the IGT across the last 50 trials as a function of arousal manipulation condition.

Source	*Df*	*F*	*p*	η_p^2
	Between Subjects Effects			
Head Injury Status	2	3.23	.05	.08
Arousal Manipulation	1	.02	.90	-
Head Injury Status × Arousal Manipulation	2	.18	.84	-
Error	76			
	Within Subjects Effects			
Trial Blocks	3.06	1.21	.31	-
Trial Blocks × Head Injury Status	6.12	6.90	< .001	.15
Trial Blocks × Arousal Manipulation	3.06	.21	.94	-
Trial Blocks × Head Injury Status × Arousal Manipulation	6.12	.50	.86	-
Error	232.62			

Note: Sphericity could not be assumed, as Mauchly's W was significant. As a result, a Greenhouse-Geisser correction of degrees of freedom was made.

Table 11:

Summary of results for a mixed model 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 5 (Trial Blocks [trials 51-60, trials 61-70, trials 71-80, trials 81-90, trials 91-100]) × 2 (Arousal Manipulation status [pre-exposure, post-exposure]) ANOVA for ratio of advantageous (C + D) to disadvantageous (A + B) selections on the Iowa Gambling Task (IGT) as a function of head injury status across 10 trial blocks and manipulation condition.

Source	*Df*	*F*	*p*	η_p^2
	Between Subjects Effects			
Head Injury Status	2	3.66	.03	.08
Arousal Manipulation	1	.94	.34	-
Head Injury Status × Arousal Manipulation	2	.92	.40	-
Error	80			
	Within Subjects Effects			
Trial Blocks	4	1.60	.31	-
Trial Blocks × Head Injury Status	8	6.90	< .001	.15
Trial Blocks × Arousal Manipulation	4	.21	.94	-
Trial Blocks × Head Injury Status × Arousal Manipulation	8	.50	.86	-
Error	320			

Table 12:

Summary of results for a mixed model 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 2 (deck type [disadvantageous, advantageous]) × 2 (Arousal Manipulation status [pre-exposure, post-exposure]) ANOVA for rate of return (duration of trials) following punishment on a selection before returning to that same deck on the Iowa Gambling Task (IGT) as a function of head injury status.

Source	*Df*	*F*	*p*	η_p^2
	Between Subjects Effects			
Head Injury Status	2	4.68	.01	.11
Arousal Manipulation	1	.49	.49	-
Head Injury Status × Arousal Manipulation	2	.89	.42	-
Error	78			
	Within Subjects Effects			
Deck Type	1	9.61	.003	.11
Deck Type × Head Injury Status	2	5.58	.005	.13
Deck Type × Arousal Manipulation	1	.07	.79	-
Deck Type × Head Injury Status × Arousal Manipulation	2	.85	.43	-
Error	78			

Table 13:

Summary of results for a mixed model 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 4 (deck [deck A, deck B, deck C, deck D]) × 2 (Arousal Manipulation status [pre-exposure, post-exposure]) ANOVA for preference following completion of the Iowa Gambling Task (IGT) as a function of head injury status and arousal manipulation.

Source	*Df*	*F*	*p*	η_p^2
	Between Subjects Effects			
Head Injury Status	2	2.3	.11	.05
Arousal Manipulation	1	1.41	.24	-
Head Injury Status × Arousal Manipulation	2	.40	.67	-
Error	80			
	Within Subjects Effects			
Deck	2.69	5.25	.002	.06
Deck × Head Injury Status	5.38	4.36	.001	.10
Deck × Arousal Manipulation	2.69	1.87	.18	-
Deck × Head Injury Status × Arousal Manipulation	5.38	.53	.77	-
Error	215.36			

Note: Sphericity could not be assumed, as Mauchly's W was significant. As a result, a Greenhouse-Geisser correction of degrees of freedom was made.

Table 14

Summary of results for a mixed model 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 4 (deck [deck A, deck B, deck C, deck D]) × 2 (Arousal Manipulation status [pre-exposure, post-exposure]) ANOVA for estimated earnings had participants chosen a deck for all 100 trials on the Iowa Gambling Task (IGT) as a function of head injury status and arousal manipulation.

Source	*Df*	*F*	*p*	η_p^2
	Between Subjects Effects			
Head Injury Status	2	.29	.75	-
Arousal Manipulation	1	.07	.79	-
Head Injury Status × Arousal Manipulation	2	1.22	.30	-
Error	79			
	Within Subjects Effects			
Deck	2.40	4.81	.006	.06
Deck × Head Injury Status	4.80	.21	.95	-
Deck × Arousal Manipulation	2.40	.45	.45	-
Deck × Head Injury Status × Arousal Manipulation	4.80	.53	.77	-
Error	189.44			

Note: Sphericity could not be assumed, as Mauchly's W was significant. As a result, a Greenhouse-Geisser correction of degrees of freedom was made.

Table 16

Summary of results for a 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 6 (Time [initial testing, recording 2, pre-manipulation, post-manipulation, recording 5, end of testing session]) mixed model ANOVA for measures of electrodermal activation (EDA) throughout the testing session as a function of head injury status.

Source	*Df*	*F*	*p*	η_p^2
	Between Subjects Effects			
Head Injury Status	2	78.89	< .001	.66
Error	82			
	Within Subjects Effects			
Time	2.29	30.87	< .001	.27
Deck × Head Injury Status	4.58	2.06	.08	.05
Error	187.62			

Note: Sphericity could not be assumed, as Mauchly's W was significant. As a result, a Greenhouse-Geisser correction of degrees of freedom was made.

Table 17

Summary of results for a 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 3 (Time [initial testing, post-manipulation, end of testing session]) mixed model ANOVA for measures of pulse rate (BPM) throughout the testing session as a function of head injury status.

Source	*Df*	*F*	*p*	η_p^2
	Between Subjects Effects			
Head Injury Status	2	4.03	.02	.09
Error	82			
	Within Subjects Effects			
Time	2	58.16	< .001	.42
Deck × Head Injury Status	4	.92	.46	-
Error	164			

Table 18

Summary of results for a 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 3 (Time [initial testing, post-manipulation, end of testing session]) mixed model ANOVA for measures of salivary cortisol throughout the testing session as a function of head injury status.

Source	*Df*	*F*	*p*	η_p^2
	Between Subjects Effects			
Head Injury Status	2	2.09	.13	.05
Error	79			
	Within Subjects Effects			
Time	1.31	33.57	< .001	.30
Deck × Head Injury Status	2.62	2.25	.10	.05
Error	103.46			

Note: Sphericity could not be assumed, as Mauchly's W was significant. As a result, a Greenhouse-Geisser correction of degrees of freedom was made.

Table 19:

Summary of results for a mixed model 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 4 (Arousal Type [initial testing, anticipation, positive feedback, negative feedback]) × 2 (Arousal Manipulation status [pre-exposure, post-exposure]) ANOVA for measures of physiological arousal (EDA) during the Iowa Gambling Task (IGT) as a function of head injury status and arousal manipulation

Source	*Df*	*F*	*p*	η_p^2
	Between Subjects Effects			
Head Injury Status	1.	91.71	<.001	.70
Arousal Manipulation	1	.08	.78	-
Head Injury Status × Arousal Manipulation	2	.16	.85	-
Error	80			
	Within Subjects Effects			
Arousal Type	1.76	77.86	< .001	.49
Arousal Type × Head Injury Status	3.53	42.93	< .001	.52
Arousal Type × Arousal Manipulation	1.76	.34	.68	-
Arousal Type × Head Injury Status × Arousal Manipulation	3.53	.14	.96	-
Error	141.12			

Note: Sphericity could not be assumed, as Mauchly's W was significant. As a result, a Greenhouse-Geisser correction of degrees of freedom was made.

Table 21:

Summary of results for a mixed model 3 (head injury status [no-MHI, MHI, Moderate TBI]) × 5 (Trial Blocks [trials 51-60, trials 61-70, trials 71-80, trials 81-90, trials 91-100]) × 2 (Degree of Depression [low, high]) ANOVA for measures of ratio of advantageous (C + D) to disadvantageous (A + B) selections on the Iowa Gambling Task (IGT) as a function of head injury status and degree of depressive symptomatology

Source	*Df*	*F*	*p*	η_p^2
	Between Subjects Effects			
Head Injury Status	2	3.31	.04	.08
Degree of Depression	1	.05	.83	-
Head Injury Status × Degree of Depression	2	.16	.85	-
Error	80			
	Within Subjects Effects			
Trial Blocks	3.58	1.57	.19	-
Trial Blocks × Head Injury Status	7.16	1.23	.29	-
Trial Blocks × Degree of Depression	3.58	2.71	.03	.03
Trial Blocks × Head Injury Status × Degree of Depression	7.16	.20	.99	-
Error	286.44			

Note: Sphericity could not be assumed, as Mauchly's W was significant. As a result, a Greenhouse-Geisser correction of degrees of freedom was made

Appendix B: Research Ethics Approval & Data Collection Materials

Reviewer Disposition

(For REB Use Only) ► File # :_____ Reviewers:_____ Due Date:_____

Decision: Accepted as is ☐ Approval Pending Revision ☐ Clarification Required ☐

Resubmission ☐ Full Review ☐ Withhold Approval ☐

Brock University Research Ethics Board (REB)

Application for Ethical Review of Research Involving Human Participants

If you have questions about or require assistance with the completion of this form, please contact the Research Ethics Office at (905) 688-5550 ext. 3035, or reb@brocku.ca.

Selecting a Research Ethics Board

Files will be allocated to one of two REB panels based upon the type of research to be undertaken.

If your research involves any of the following, submit to the Bioscience Research Ethics Board (BREB):

physiological measures such as EEGs, heart rate, GSR, temperature, blood pressure, respiration, vagal tone, x-rays, MRIs, CT or PET scans;
ingestion or other use of food, beverages, food additives, or drugs, including alcohol and tobacco;
medical techniques or therapies, including experimental medical devices;
physical exertion beyond normal walking;
physical movement in participants who have medical vulnerabilities (e.g., spinal cord injury, osteoporosis);
human biological materials (e.g., tissues, organs, blood, plasma, skin, serum, DNA, RNA, proteins, cells, hair, nail clippings, urine, saliva, bodily fluids);
interventions with the potential for physiological effects (e.g., diet, exercise, sleep restriction); and/or
use of medical or official health records (e.g., hospital records).

If none of the above points are characteristic of your research, submit to the Social Science Research Ethics Board (SREB)

Indicate which REB panel is appropriate for this application:

☒ **Bioscience (BREB)** **OR** ☐ **Social Science (SREB)**

Return your completed application and all accompanying material **in triplicate** to the
Research Ethics Office in MacKenzie Chown D250A.
Handwritten Applications will ***not*** be accepted

Please ensure all necessary items are attached prior to submission,
otherwise your application will not be processed (see checklist below).

No research with human participants shall commence prior to receiving approval from the REB.

DOCUMENT CHECKLIST **3 complete sets of the following documents (one original + 2 copies)**	**if applicable**
Recruitment Materials	
• Letter of invitation	☐
• Verbal script	☒
• Telephone script	☒
• Advertisements (newspapers, posters, SONA)	☒
• Electronic correspondence guide	☐
Consent Materials	
• Consent form	☒
• Assent form for minors	☐
• Parental/3rd party consent	☐
• Transcriber confidentiality agreement	☐
Data Gathering Instruments	
• Questionnaires	☒
• Interview guides	☐
• Tests	☐
Feedback Letter	☒
Letter of Approval for research from cooperating organizations, school board(s), or other institutions	☐
Any previously approved protocol to which you refer	☐
Request for use of human tissue sample in research Please Note: this form is required for all research projects involving human tissue, bodily fluids, etc.	☒
Signed Application Form	☒

SIGNATURES

PLEASE NOTE: The title "principal investigator" designates the person who is "in charge" of the research. In this position, the principal investigator is assumed to have the abilities to supervise other researchers, be responsible for the financial administration of the project, have the authority to ensure that appropriate guidelines and regulations are followed, and be competent to conduct the research in the absence of faculty supervision. The restriction of the term "principal investigator" to faculty or post-doctoral fellows does not have implications for ownership of intellectual property or publication authorship.

Given the above consideration, **a student cannot be identified as a "principal investigator"**. However, for the purpose of recognizing a student's leadership role in the research, a faculty member may designate a "principal *student* investigator" below.

INVESTIGATORS:

Please indicate that you have read and fully understand all ethics obligations by checking the box beside each statement and signing below.

☒ I have read Section III: 8 of Brock University's Faculty Handbook pertaining to Research Ethics and agree to comply with the policies and procedures outlined therein.
☒ I will report any serious adverse events (SAE) to the Research Ethics Board (REB).
☒ Any additions/changes to research procedures after approval has been granted will be submitted to the REB.
☒ I agree to request a renewal of approval for any project continuing beyond the expected date of completion or for more than one year.
☒ I will submit a final report to the Office of Research Services once the research has been completed.
☒ I take full responsibility for ensuring that all other investigators involved in this research follow the protocol as outlined in this application.

Principal Investigator

Signature _____ Date: _____

Principal Student Investigator (optional)

Signature _____ Date: _____

Co-Investigators:

Signature _____ Date: _____

Signature _____ Date: _____

FACULTY SUPERVISOR:

Please indicate that you have read and fully understand the obligations as faculty supervisor listed below by checking the box beside each statement.

☒ I agree to provide the proper supervision of this study to ensure that the rights and welfare of all human participants are protected.
☒ I will ensure a request for renewal of a proposal is submitted if the study continues beyond the expected date of completion or for more than one year.
☒ I will ensure that a final report is submitted to the Office of Research Services. I
☒ have read and approved this application and proposal.

Signature _____ Date: _____

SECTION A - GENERAL INFORMATION

1. **Title of the Research Project:** Emotion & Cognition Study

2. **Investigator Information:**

	Name	Position (e.g., faculty, student, visiting professor)	Dept./Address	Phone No.	E-Mail
Principal Investigator	Dr. Dawn Good	Associate Professor, Chair Centre for Neuroscience	Department of Psychology, Centre for Neuroscience, Brock University, 500 Glenridge Ave. St. Catharines, ON L2S 3A1	(905) 688-5550 x 3869, 3556, 5523	Dawn.Good@brocku.ca
Principal Student Investigator	Julie Baker	Ph.D. Candidate	Department of Psychology, Brock University, 500 Glenridge Ave. St. Catharines, ON L2S 3A1	(905) 688-5550 x 3034	js01cb@brocku.ca
Co-Investigator(s)	Sean Robb	M.A. Candidate	Department of Psychology, Brock University, 500 Glenridge Ave. St. Catharines, ON L2S 3A1	(905) 688-5550 x 3556	sr07by@brocku.ca
Co-Investigator(s)	Amanda George	Honours Thesis Candidate	Department of Psychology, Brock University, 500 Glenridge Ave. St. Catharines, ON L2S 3A1	(905) 688-5550 x 3556	ag09xy@brocku.ca
Faculty Supervisor(s)					

3. **Proposed Date of commencement:** upon approval, OR other. Please provide date **(dd/mm/yyyy)**

Proposed Date of completion (dd/mm/yyyy): 01/09/2013

4. **Indicate the location**(s) where the research will be conducted:

Brock University

Community Site ☐ Specify _____
School Board ☐ Specify _____
Hospital ☐ Specify Other
☐ Specify _____

5. **Other Ethics Clearance/Permission:**

(a) Is this a multi-centered study? ☐ **Yes** ☒ **No**
(b) Has any other University Research Ethics Board approved this research? ☐ **Yes** ☒ **No**

If **YES**, there is no need to provide further details about the protocol **at this time,** provided that **all** of the following information is provided:

Title of the project approved elsewhere: _____
Name of the Other Institution: _____
Name of the Other Board: _____
Date of the Decision: _____
A contact name and phone number for the other Board: _____

Please provide a copy of the application to the other institution together with all accompanying materials, as well as a copy of the clearance certificate / approval.

If **NO**, will any other University Research Ethics Board be asked for approval? ☐ **Yes** ☒ **No**
Specify University/College _____

(c) Has any other person(s) or institutions granted permission to conduct this research? ☐ **Yes** ☒ **No**
If yes, specify (e.g., hospital, school board, community organization, proprietor) **provide details and attach any relevant documentation.** _____

If **NO**, will any other person(s) or institutions be asked for approval? ☐ **Yes** ☒ **No**
Specify (e.g., hospital, school board, community organization, proprietor) _____

6. **Level of the Research:**

☐ Undergraduate Thesis
☐ Post Doctorate
☐ Undergraduate Course Assignment (specify course) _____
☒ Masters Thesis/Project
☒ Faculty Research
☐ Graduate Course Assignment (specify) _____
☒ Ph.D
☐ Administration
☐ Other (specify course) _____

7. **Funding of the Project:**

(a) Is this project currently being funded ☒ **Yes** ☐ **No**
(b) If **No**, is funding being sought ☒ **Yes** ☐ **No - from SSHRC special projects grant (November 2012)**

If Applicable:
(c) Period of Funding (dd/mm/yyyy): From: ongoing To: _____
(d) Agency or Sponsor (funded or applied for)

☐ CIHR ☐ NSERC ☐ SSHRC ☒ Other (specify): Faculty funds

(e) Funding / Agency File # (**not** your Tri-Council PIN) N/A

8. **Conflict of Interest:**

(a) Will the researcher(s), members of the research team, and/or their partners or immediate family members receive any personal benefits related to this study - Examples include financial remuneration, patent and ownership, employment, consultancies, board membership, share ownership, stock options. Do not include conference and travel expense coverage, possible academic promotion, or other benefits which are integral to the general conduct of research.

☐ **Yes** ☒ **No**

If **Yes**, please describe the benefits below.

(b) Describe any restrictions regarding access to or disclosure of information (during or at the end of the study) that the sponsor has placed on the investigator(s).

SECTION B - SUMMARY OF THE PROPOSED RESEARCH

9. **Rationale:**

Briefly describe the purpose and background rationale for the proposed project, as well as the hypothesis(es)/research question(s) to be examined.

Our research focuses on examining the cognitive sequelae and emotional regulation following head trauma, especially with high-functioning individuals such as university students. Previous research from our lab (Brock University Neuropsychology Cognitive Research Lab) has shown that individuals with self-reported mild head injury (MHI; e.g., concussion) are relatively underaroused and less responsive to stressors in their environment (both physiologically [e.g., electrodermal skin response] and self-reports) as compared to non-MHI students and indeed, in contrast what is typically found, benefit from being activated to a higher level of arousal with respect to cognitive performance (e.g., memory tasks: St. Cyr & Good, 2007; decision-making tasks: Robb & Good, 2012). Baker & Good (2010) found that using a psychosocial stressor to induce arousal had limited effects on cognitive performance perhaps due to the nature of the stressor, the dampened response of students with MHI to the stressor, or the limited duration of the increased arousal following experimental manipulation. As well, persons with a history of head trauma may have a lessened ability to interpret and respond to stressors/emotional events. Another study (Baker & Good, 2012) demonstrated that students with a history of mild head trauma elicited significantly reduced physiological responsivity (i.e., EDA amplitude) to emotionally-evocative stimuli (i.e., positive, negative, and ambiguous pictures) relative to those with no-MHI. Similarly, a study by Bay and colleagues (2009) reported that persons with mild-to-moderate traumatic brain injury (TBI) evidenced hypocortisolemia (i.e., decreased salivary cortisol) and flattened diurnal patterns of cortisol. This finding, in concert with our previous research, suggests that persons with neurological compromise (e.g., TBI) may have dysregulated stress responses. Indices of physiological underarousal, such as hypocortisolemia or salivary alpha-amylase levels, implicate hypothalamic-pituitary-adrenal (HPA) axis involvement. Measures of the cortisol awakening response (CAR) and salivary cortisol would be especially indicative of dysregulated/attenuated stress responses and/or altered diurnal patterns and will be examined in this study. CAR is the increased rise in cortisol immediately following awakening and has been reported to be an index of one's ability to respond to stressors (Clow et al., 2010). Further, numerous studies have demonstrated changes in concentrations of salivary cortisol (e.g., Dickerson & Kemeny, 2004; Kirschbaum & Hellhammer, 2000) and salivary alpha-amylase (e.g., Nater & Rohleder, 2009) following manipulations of arousal (e.g., stressors; emotionally-evocative stimuli).

As such, we have designed a matched-subjects study (N = 60) to examine physiological and self-reported stress responsivity as a function of the severity of neural trauma. This study will examine the *underarousal hypothesis* via the following: a) assessment of underarousal and dysregulated arousal in persons with neurological compromise via physiological (i.e., Cortisol Awakening Response [CAR],

cortisol responsivity, salivary alpha-amylase, EDA, heart rate, and respiration) and self-report indices; b) manipulation of ecologically appropriate ways to induce heightened arousal in persons with neurological compromise (e.g., emotionally-laden visual stimuli; IAPS, Lang et al., 2008); and c) measuring the effects of modified arousal status on cognition (i.e., neuropsychological test battery scores; decision-making task). The subjects will consist of participants who vary in terms of individual differences, particularly severity of neural injury (i.e., no head trauma [*n* = 50], minimal [*n* = 15], mild [n = 15], moderate [*n* =10], or severe [*n* = 10] TBI), but also personality characteristics including optimism, emotional and social competence.

The following hypotheses will be considered: *Hypothesis 1* - Persons with prior head injury will be underaroused as measured through both physiological and self-report measures, despite reporting increased life stressors; *Hypothesis 2* - Indices of dysregulated arousal and stress response (i.e., CAR and cortisol change scores; physiological recordings of heart rate, EDA, and respiration) will vary as a function of neural injury severity (i.e., mild, moderate, and severe) with persons with more severe trauma exhibiting more dysregulated arousal and responsivity; atypical diurnal patterns of cortisol are also expected; *Hypothesis 3* - Induced-stress will heighten physiological arousal and, thereby, improve cognitive performance (e.g., neuropsychological test battery summary scores; decision-making task) for persons with compromised/lower physiological arousal. *Hypothesis 4* - individual differences based on personality characteristics will mirror/be mediated by severity of injury.

This research will improve the understanding of emotional and cognitive functioning of persons with and without a history of neural disruption, particularly with respect to stress responses, and has implications for clinical practice in the fields of neuropsychology and rehabilitative medicine.

10. **Methods:**

Are any of the following procedures or methods involved in this study? Check **all** that apply**.**

- ☐ Questionnaire (mail)
- ☐ Questionnaire (email/web)
- ☒ Questionnaire (in person)
- ☒ Interview(s) (telephone)
- ☐ Interview(s) (in person)
- ☐ Secondary Data
- ☒ Computer-administered tasks
- ☐ Focus Groups
- ☐ Journals/Diaries/Personal Correspondence
- ☐ Audio/video taping specify)
- ☐ Observations
- ☐ Invasive physiological measurements (e.g. venipuncture, muscle biopsies)
- ☒ Non-invasive physical measurement (e.g., exercise, heart rate, blood pressure)
- ☒ Analysis of human tissue, body fluids, etc. (Request for Use of Human Tissue Sample must be completed and attached)
- ☒ Other: (specify) neuropsychological testing

Describe sequentially, and in detail, all of the methods involved in this study and all procedures in which the research participants will be involved (paper and pencil tasks, interviews, questionnaires, physical assessments, physiological tests, time requirements, etc.)
Attach a copy of all questionnaire(s), interview guides or other test instruments. If reference is made to previous protocols, please provide copies of relevant documentation.

University students and persons in the community will be invited to participate in the study and will be asked to contact the researcher for a brief telephone interview. Participants will be read an informed consent telephone script (**see** Appendix) and if he/she agrees to participate he/she will be asked a few screening questions (e.g.,history of medication use, sleep patterns, head injury history, age, level of education, and gender). Participants who meet our inclusion criteria will be invited to participate in two testing sessions and convenient dates/times will be scheduled. The first session will be brief (lasting approximately 10 minutes) during which they will be asked to sign a written informed consent form (**see Appendix**) as well as pick up salivary sample collection materials with instructions (**see Appendix**). The second session will consist of the data collection and should take approximate 2 hours 20 minutes.

Participants will be asked to bring saliva samples (taken at home) to this second session at the university.

Participants will be greeted for both sessions, individually, in a private lab setting in the Jack and Nora Walker Lifespan Development Centre testing facilities at Brock University. The informed consent form will be read aloud to the participant by the researcher for clarification, and the participant can ask any questions at that time or any time throughout the study. Participants will be informed that they will be asked to personally obtain saliva samples taken by themselves the night before and the day of the testing session using the kit provided. They will be advised on how to do this, and provided, explicit simple/straight forward instructions on this procedure. All participants will be advised that when they return for the second session they will be asked to partake in a data collection session. Participants will be asked to engage in a short battery of neuropsychological testing to assess cognitive competency (e.g., memory, attentional tasks) and decision-making. The participants will also be advised that one of the tasks they will be presented involves the viewing pictures of pleasant, unpleasant, or neutral scenes. Participants will be informed that during the testing session physiological measures will be taken for electrodermal responses, heart rate, and respiration (via finger bands around two fingers of the non-dominant hand, pulse oximeter, and respiration bands, respectively). The participant will be given two copies of the written informed consent form to be completed (one copy is given to the participant for his/her records and the other copy is for the researcher—see Appendix). Participants will be informed that their participation in the study is voluntary, and that he or she is free to leave at any time without penalty. Should the participant leave the study early, the researcher will provide him or her with the debriefing form before he or she leaves and any data obtained will not be included in the analysis. In the event of withdrawal from the study, participants will receive research participation hours or monetary compensation for their participation up until that point, will be provided with counseling contact information, and will be invited to speak with the principal investigator who is a Registered Psychologist should s/he prefer.

After the informed consent process is completed, participants will be provided with a saliva collection kit and instructed on how (passive drooling into a plastic tube) and when to collect three saliva samples while at home the night before (at least 2 hours after eating and between 22:00 to 23:30), and the morning of (immediately upon waking, and 45 minutes later), the longer testing session. (See attached instructions which are provided to the participants). Any additional questions the participant may have will be answered, followed by a confirmation of when the participant will be returning for the test session.

Upon return to the testing situation, participants will be asked for their collection kit (and samples - which will be coded alphanumerically without personal identifiers). They will then be connected to physiological recording equipment to collect heart rate, electrodermal activity (EDA), and respiration data via Polygraph Professional (2008) software. In order to decrease contact between researcher and participant during the application of the physiological recording equipment, the researcher will model the application for the participant and ask him/her to make minor adjustments. Note that the physiological recordings of arousal state and self-report of arousal state rating (i.e., participant is asked how he/she feels on a scale of 1 to 10 with 1 being relaxed and 10 being stressed) will be taken intermittently throughout the testing session (e.g., baseline, prior to, during, and after emotional arousal induction). Two more saliva samples will be collected from the participants - one at this point in the session, and another following the emotional arousal induction. All physiological activity recorded and saliva samples will be coded alphanumerically without personal identifiers.

Once the participant is comfortably fitted with the physiological recording equipment, and has provided a saliva sample, he/she will participate in neuropsychological tasks that assess cognitive abilities of memory, executive functioning, and attention (see **Appendix for list and description of Neuropsychological Tasks**). Matched versions of these neuropsychological tasks will be administered prior to, and after, exposure to the emotional arousal induction. For memory and attentional control, subtests of the Wechsler Memory Scale (Logical memory, Letter-Number Sequencing, Mental Control from WMS-IV, The Psychological Corporation, 2009), as well as a nonverbal memory task (Rey Complex Figure - Osterreith, 1944), a nonverbal visual scanning test (Trails - DKEFS, 2002) will be used. To test executive functioning, subtests of the Wechsler Adult Intelligence Scale (Matrix Reasoning,

Social Cognition - WAIS-IV, 2009) and of the Wide Range Achievement Test (Reading - WRAT-IV, 2006) will be used. Emotional competence will be assessed with the Emotional Quotient Inventory (Bar-On, 1997).

Participants will receive instructions regarding the "emotional arousal induction" by viewing and rating a set of pictures derived from the International Affective Picture System (IAPS; Lang et al., 2008) (see Appendix for IAPS Verbal Script). The pictures (approximately 10" x 8" in size) will be presented on a 17" computer screen with a viewing distance of approximately 60 cm and will be displayed for 5 seconds. Each will be rated for intensity, pleasantness, arousal, and empathy elicitation on a scale from 1 (minimal) to 9 (significant) (mimicking the IAPS procedures). The scenes in the pictures involve either neutral, unpleasant, or pleasant stimuli. Physiological activity recordings will be taken throughout the viewing and rating of the pictures and will be segmented to provide indices of sympathetic nervous system activation during anticipatory phases (i.e., prior to viewing of each picture), during presentation of stimuli, and during the response phase (i.e., rating of each picture). Refer to Appendix for Verbal Script of IAPS stimuli presentation and rating instructions. A total of 50 pictures have been selected from the IAPS stimuli and includes pictures of persons, animals, and inanimate objects that had high arousal (i.e., excited), valence (i.e., positive and negative affect), and emotion (e.g., anger, sadness) ratings as indicated by the normative data (Lang et al., 2005; Libkuman et al., 2007; Mikels et al., 2005).

For the second part of the emotional arousal induction, participants will be asked to view these stimuli again, but this time, will be asked to generate emotional descriptors/narratives for the picture (i.e., positive - e.g., happy; negative - e.g., sad) before providing a rating. The purpose of the emotional arousal induction task is to induce heightened emotional arousal (both physiological and self-report) and has been commonly used with university students. The emotional arousal induction will last approximately 30 minutes with continuous physiological recording throughout with variable sampling.

Participants will also complete a computerized decision-making task, the Iowa Gambling Task (IGT; 100 trials, 25 minutes in duration), which consists of being presented four decks of cards (two 'advantageous' and two 'disadvantages') on a computer screen and being asked to select one card at a time from one of the decks with the goal of gaining as many points as possible. Upon completion of the decision-making task the participant will be given a set of questions (developed by Maia & McClelland, 2004 - see Appendix), which measures the subject's awareness and explicit knowledge the strategies s/he used during the IGT.

At this point, the subject will be asked to complete a set of questionnaires that will assess emotional status (BarON Emotional Quotient Inventory, BarON EQ-I, Bar-on, 1997; State-Trait Anxiety Inventory, STAI, Spielberger, 1983; Emotional Intelligence, Barchard, 2001; Symptom Assessment-45 Questionnaire, SA-45, Strategic Advantage, 1998; Toronto Empathy Questionnaire, Spreng et al., 2009) and demographics to gather information on individual differences. The questions should take approximately 30 minutes to complete (see Appendix for a description). After completion of the self-report measures, a final physiological activity recording and self-report of arousal state will be obtained to verify return to baseline status.

Physiological equipment will then be removed by the participant with simulated modeling provided by the experimenter; the participant will be given sanitary wipes to remove any residual electrode gel from his/her hands. They will then be debriefed as to the nature of the study, and thanked for their cooperation (see Appendix for Debriefing form). Overall, participation in this study (including time for acquisition of informed consent and debriefing procedures) will not exceed 2.5 hours. Also included in the debriefing form (see Appendix) will be counselling contact information for Brock University Counselling Services should any negative emotions surface as a result of participating in this study. Participants will also receive contact information for the principal investigator/faculty supervisor. Finally, participants will be thanked for their time and participation in the study, and will be invited to view the results of the study at its completion (by September 2013).

11. **Professional Expertise/Qualifications:**

Does this procedure require professional expertise/recognized qualifications (e.g., registration as a clinical psychologist, first aid certification)?

☒ **Yes** specify: _____ ☐ **No**

The neuropsychological testing materials are protected, standardized psychological tests that require professional expertise for their use. Administration of the tests will be monitored by Dr. Dawn Good (principal investigator) who is a Registered Psychologist.

If **YES**, indicate whether you, your supervisor, or any members of your research team have the professional expertise/recognized qualifications required? ☒ **Yes** ☐ **No**

Dr. Dawn Good (principal investigator) is a Registered Psychologist.

12. **Participants:**

Describe the number of participants and any required demographic characteristics (e.g., age, gender).

One-hundred individuals will participate in this study. The participants will include Brock University students (n = 60; 15 no history of head trauma non-varsity participants, 15 mild head injury non-varsity participants; 15 no history of head trauma varsity participants, 15 mild head injury varsity participants) and members from the Niagara community (n=40; persons who have sustained moderate [n=10] or severe [*n* = 10] brain injuries; 20 persons who have not sustained head/brain injury). Participants will be matched for age, sex, and if possible, level of education.

13. **Recruitment:**

Describe how and from what sources the participants will be recruited, including any relationship between the investigator(s), sponsor(s) and participant(s) (e.g., family member, instructor-student; manager-employee).

Attach a copy of any poster(s), advertisement(s) and/or letter(s) to be used for recruitment.

One hundred participants will be recruited for the study by volunteering their participation through the online Brock University Psychology Department Research Website (i.e., SONA see Appendix for advertisement) and poster advertisements. Poster advertisements for the study will be posted on the Psychology Research Board (see Appendix for poster) and in the Athletic Therapy Building (Harrison Hall), and Trainers Room (Walker Complex) - the latter in an attempt to recruit varsity athletes. Note we will not be asking varsity athletes to participate via individual recruitment, anyone who views the poster advertisement and wishes to participate will be considered for the study with the poster advising them on how to contact us. Community centers that provide support and services to individuals living with acquired brain injury (i.e., the Ontario Brain Injury Association and the Brain Injury Assocation of Niagara) will be contacted to recruit an additional 40 participants (i.e., persons who have sustained moderate or severe traumatic brain injury, and matched community cohorts). Participants will complete a brief informed consent and telephone interview to select participants based on study characteristics, mainly age, sex, and history of neural disruption. Participants will be excluded if they are shift workers. In addition, eligible participants will be excluded once the number of participants for that group (e.g., mild TBI group) has been fufilled.

14. **Compensation:**

a) Will participants receive compensation for participation? ☒ **Yes** ☐ **No**

b) If yes, please provide details.

Participants will have the opportunity to receive research participation hours for applicable courses at the university or receive a small monetary honorarium for their research participation (i.e., $12 per hour of participation). The participants may be credited at the rate of one half credit per half hour of participation which is the standard rate associated with participation. Community participants will receive a small monetary honorarium (i.e., $12 per hour of participation plus transportation).

SECTION C - DESCRIPTION OF THE RISKS AND BENEFITS OF THE PROPOSED RESEARCH

15. **Possible Risks:**

1) Indicate if the participants might experience any of the following risks:

a) Physical risks (including any bodily contact, physical stress, or administration of any substance)? ☒ **Yes** ☐ **No**

b) Psychological risks (including feeling demeaned, embarrassed worried or upset, emotional stress)? ☒ **Yes** ☐ **No**

c) Social risks (including possible loss of status, privacy, and / or reputation)? ☐ **Yes** ☒ **No**

d) Are any possible risks to participants greater than those that the participants might encounter in their everyday life? ☐ **Yes** ☒ **No**

e) Is there any deception involved? ☒ **Yes** ☐ **No**

f) Is there potential for participants to feel obligated to participate or coerced into contributing to this research (because of regular contact between participants and the researcher, relationships that involve power-dynamics, etc.)? ☐ **Yes** ☒ **No**

2) If you answered **Yes** to any of 1a - 1f above, please explain the risk.

a) Participants will be connected to physiological activity recording equipment to collect physiological data (i.e., heart rate, electrodermal activity, and respiration). To collect this data, two electrodes (placed on separate fingers of the non-dominant hand) will be used to record electrodermal activity, two respiration bands (placed around the participant's chest and lower abdomen), and a pulse oximeter (placed on the participant's finger) to collect heart rate data. Although the equipment is not invasive, the application of the electrodes, pulse oximeter, and respiration bands involves minor physical contact from the researcher to the participant. In order to minimize any discomfort participants may feel during the placement of the physiological recording equipment, participants will be clearly asked for consent and the process of applying the physiological recording equipment will be fully explained and modeled for the participants by the researcher prior to application. In addition, participants will be asked to complete/and directed as to how to make any adjustments of the equipment on his/her body to minimize physical contact between his/herself and the researcher. Participants will be asked to self-identify any dermal sensitivities they may have as it is possible, but unlikely, that participants may have sensitivity to the electrode conductive gel. Participants will be provided with sanitary moist wipes to remove the conductive gel. Participants will also be providing salivary samples via passive drooling into a plastic storage tube for approximately 2 minutes both at their home and in the testing setting (for a total of 5 samples). Explicit instructions for all procedures will be provided to the participant (directly and through modeling in terms of the polygraph equipment; directly and through written instructions for the saliva collection kit) and sanitary procedures will be explained and implemented (e.g., use of gloves, cleansed and disinfected equipment, etc.).

b (i) It is expected that participants will experience mild distress during the heightened arousal induction manipulation due to discomfort of viewing emotionally-arousing stimuli (i.e., emotionally-provocative pictures International Affective Picture System [IAPS]). However, the level of stress experienced is deemed to be no greater, and/or not unlike the type of stress encountered in everyday life (e.g., pictures from news reports, magazines). Arousal manipulations using the IAPS stimuli have shown to be sufficient to produce noncritical physiological changes in heart rate and EDA (e.g., Sanchez-Navarro et

al., 2006) and these stimuli are commonly used with university students. The emotionally-arousing pictures will be used in order to provide, or otherwise produce, a heightened level of stress vigilance which is the precise effect being investigated.

b (ii) Participants often feel the psychological pressure of being evaluated when doing psychological tests (e.g., personality questionnaires, tests of reasoning) due to their association with overall competency. As a result, they may by slightly embarrassed, or disquieted, by their performance or otherwise stressed as to what their performance means in terms of capacity or ability. Participants will be reminded that the researchers are interested in group, rather than individual, responses, and that the cognitive tests are intentionally challenging in order to avoid ceiling effects, rather than it reflecting their cognitive capacity. Participants will have been previously informed during the informed consent process that the questionnaires may involve questions of a sensitive or personal nature and are at liberty to omit any answer/response should they choose.

b (iii) Female participants may feel that their privacy has been invaded due to one of the questions asked during the demographic questionnaire regarding their use of birth control medication. We must ask about this because the literature indicates a relationship between estrogen supplements/medications (such as oral contraceptives) and the elevation of cortisol levels. Participants are informed that they can choose to answer/not answer any question.

e) Finally, informed consent procedures for university students and community participants do not explicitly state the researchers' interests in head injury/brain injury as a primary variable in this study. Research has shown that informing participants that head injury/brain injury is one of the study variables of interest can influence subsequent performance (Suhr & Gunstad, 2002; 2005). Subjects will be fully debriefed upon study completion.

3) Describe how the risks will be managed and include the availability of appropriate medical or clinical expertise or qualified persons. Explain why less risky alternative approaches could not be used.

a) Subjects will be asked about any allergies or skin sensitivities they may have and will be screened to not participate in the study as appropriate. In the unlikely event that participants may have an unknown sensitivity to the electrode conductive gel, participants will be provided with sanitary wipes to remove the gel. Further, to ensure sanitary conditions, the researcher will provide the participant with antibacterial lotion prior to application of electrodes; for any procedures during with the researcher will need to minimally assist the participant through contact (e.g., application of the electrodes), s/he will wear gloves and use sanitary procedures.

b) To manage psychological risk, participants will be fully informed during the informed consent process that they will be asked to view and rate pictures that may induce an emotional response. They will also have knowledge that cognitive/neuropsychological testing will take place. The subject's freedom to withdraw from the study at the time of consent, or any other time throughout testing, will be reinforced. Furthermore, the researcher will answer any questions that the participant may have initially, and throughout the testing session, and participants will be fully debriefed verbally by the researcher at the end of the study. The researcher will reinforce that the tests do not reflect the capacity of the participant and that they are intentionally challenging to ensure the ceiling effects are avoided for data collection. The heightened arousal manipulation and the completion of the cognitive tasks and questionnaires are considered to be of low risk since these tests simulate the experiences students would otherwise have/be familiar with in a university setting (e.g. writing tests, viewing pictures on the news/media, providing demographic information).

During debriefing the participants will be advised of our interest in, amongst other things, head and brain injuries. For persons who have experienced a brain injury, it will be clear to them that they are in the brain injury group, and not alarmed to this fact; for persons who have experienced milder neural complications (impact to the head causing an altered state of consciousness, repeated concussions), it will be clear that they are of particular interest as well, but they be more concerned due to the questions they may have as to 'why' they would be of interest to researchers - is there something permanently

wrong with their brains. We will explain that neural changes after concussions are mostly temporary and otherwise subtle, but can be more permanent, as has been witnessed in the popular press for some sports celebrities. We will reinforce our intention to understand the implications on function (emotional, cognitive), if any, of these possible neural changes, subtle or otherwise, and ultimately, assist/optimize functioning for any person with traumatic injuries to the head and brain.

The researcher will also confirm with the participants their comfort and/or concerns upon testing completion (with confirmation of return-to-baseline physiological indices at test completion) and provided with counseling and research ethics contact information should they feel they have any negative experience or emotion (e.g., feeling uncomfortable, etc.) as a result of participating in the study that would need to be addressed outside of the 'study' setting. Participants will also be provided with resources should they like more information/support regarding head trauma (The Ontario Brain Injury Association (OBIA): www.obia.ca; The Ontario Neurotrauma Foundation (ONF): www.onf.org); Brain Injury Association of Niagara (BIAN): www.**bianiagara**.org).

e) Participants are not informed in advance about head injury/brain injury as a focus for the study because research has shown that informing participants head injury is a study variable of interest can influence subsequent performance (Suhr & Gunstad, 2002; 2005). This phenomenon of 'diagnosis threat' is similar to the social psychological phenomena known as stereotype threat; individuals have schemas and representations of what their group membership involves and may behave in ways that confirm these representations (i.e., head injuries are associated with limitations in functional capacity and this may negatively affect how individuals approach and respond to task demands). However, participants will be fully informed of our interest in head and brain injuries at the completion of the study.

16. **Possible Benefits:**

Discuss any potential direct benefits to the participants from their involvement in the project. Comment on the (potential) benefits to the scientific community/society that would justify involvement of participants in this study.

Both student and community participants can benefit from participation in this study by gaining insight into neuropsychological and physiological research and empirical methods relevant to psychology, assessment of capacity, and neuroscience. Additionally, through their efforts, this study will benefit the scientific community by contributing to our knowledge of the possible mechanisms (e.g., underarousal) or correlates (e.g., personality characteristics) associated with one's cognitive and emotional function in individuals with varying amounts of neural disruption (from none, to minimal, to mild, to moderate, to severe injury). In addition, the study will identify how modifying arousal state (i.e., via viewing emotionally-arousing pictures) can influence cognitive abilities. Furthermore, this research will improve the understanding of stress responsivity, and has implications for clinical practice in the fields of neuropsychology and rehabilitative medicine.

SECTION D - THE INFORMED CONSENT PROCESS

17. **The Consent Process:**

Describe the process that the investigator(s) will be using to obtain informed consent. Include a description of who will be obtaining the informed consent. If there will be no written consent form, explain why not.
For information about the required elements in the letter of invitation and the consent form, as well as samples, please refer to: http://www.brocku.ca/researchservices/forms/index.php

If applicable, attach a copy of the Letter of Invitation, the Consent Form, the content of any telephone script, and any other material that will be utilized in the informed consent process.

The participants involved in this study will be invited to participate in the study and will be asked to

contact the researcher for a brief telephone interview. Participants will be read an informed consent telephone script and if he/she agrees to participate he/she will be asked a few screening questions (e.g.,history of medication use, caffeine use, sleep patterns, head injury history, age, level of education, and gender). If participants meet inclusion criteria, they will be invited to participate in a testing session and a date/time will be scheduled. Participants will attend two sessions - one of which they will be asked to sign a written informed consent form (**see Appendix**) and pick up salivary sample collection materials and instructions (**see Appendix**) (this session should only take 10 minutes). The second session will consist of the data collection.

18. **Consent by an authorized party:**

If the participants are minors or for other reasons are not competent to consent, describe the proposed alternative source of consent, including any permission form to be provided to the person(s) providing the alternative consent.

An individual will be presumed to be capable unless the person has been deemed to have incapacity. Both the host organization and/or host facility will know who has capacity (it is their responsibility). Should direct observation (e.g., the person is confused, disoriented, unable to make a decision, etc.) or information provided by caregivers/rehabilitation workers provide evidence of incapacity, then the individual's legally authorized substitute decision-maker or legal guardian will be contacted and/or otherwise the person will not participate in the study (e.g., similar to Ontario's Health Care Consent Act [HCCA]). All participant involvement in the study will be monitored by Dr. Dawn Good, Registered Psychologist who specializes in Neuropsychology, and particularly working with persons who have experienced ABI. All individuals will be reviewed for capacity through their host Association.

19. **Alternatives to prior individual consent:**

If obtaining individual participant consent prior to commencement of the research project is not appropriate for this research, please explain and provide details for a proposed alternative consent process.

N/A

20. **Feedback to Participants:**

Explain what feedback/ information will be provided to the participants after participation in the project. This should include a more complete description of the purpose of the research, and access to the results of the research. Also, describe the method and timing for delivering the feedback.

At the end of testing session, participants will be given a debriefing statement (**see Appendix**) and will also be given a verbal description of the study. The purpose of the study, the manipulation introduced (i.e., viewing of emotionally-arousing pictures), and a verification of the stabilization of their stress state (emotional and physical) will be discussed. It will be explained to participants that the heightened arousal induction was used in order to provide, or otherwise produce, a heightened level of stress vigilance which is the precise effect being investigated. All participants will be informed that the data collected will be summarized, used as thesis data, presented as a publishable report and conference study. All individual data will remain confidential and anonymous. Participants will be invited to view the results of the study by date of completion (September 2013) and may contact the investigators either directly or via e-mail. Contact information will be provided to the participant on the debriefing form should

the participant wish to contact the researchers at any time.

21. **Participant withdrawal:**

a) Describe how the participants will be informed of their right to withdraw from the project. Outline the procedures that will be followed to allow the participants to exercise this right.

Participation in this study is voluntary. Participants can choose to withdraw any time during the telephone interview, brief instructional or experimental sessions. The participants will be informed of their freedom to withdraw in both the verbal and written informed consent processes (**see Appendix**). Also, the consent form will be read aloud to the participants to reiterate their freedom to withdraw without penalty. It will be explained that if the participant should choose to withdraw their participation, they will receive participation credit commensurate with their participation and their data will be destroyed and disposed of in a professional and confidential manner. Participants will be informed that he/she can verbally inform the researcher at any time during the sessions of their choice to withdraw participation. Furthermore, they will be reminded of the services available that they can consult should they have any questions (Brock University Counselling Services; Research Ethics Officer; Principal Investigator).

b) Indicate what will be done with the participant's data should the participant choose to withdraw. Describe what, if any, consequences withdrawal might have on the participant, including any effect that withdrawal may have on participant compensation.

If participants choose to withdraw, the researcher will provide them with a written debriefing form (**see Appendix**), and also answer any questions. If a participant withdraws at any time during the telephone screening, instructional or experimental sessions, any data collected from him or her will be destroyed (shredded; biological measures will be appropriately disposed) and not used in data analysis. If the participant choosing to withdraw is receiving research participation credit, the length of the student's participation will be credited for appropriate participation hours up to the maximum length of the study.

SECTION E - CONFIDENTIALITY & ANONYMITY

***Confidentiality*:** information revealed by participants that holds the expectation of privacy. This means that all data collected will not be shared with anyone except the researchers listed on this application.

***Anonymity of data*:** information revealed by participants will not have any distinctive character or recognition factor, such that information can be matched **(even by the researcher)** to individual participants. Any information collected using audio-taping, video recording, or interview cannot be considered anonymous. **Please note that this refers to the anonymity of the data itself and not the reporting of results.**

22. Given the definitions above:

a) Will the data be treated as confidential? ☒ **Yes** ☐ **No** b)
Are the data anonymous? ☐ **Yes** ☒ **No**

c) Describe any **personal identifiers** that will be collected during the course of the research (e.g., participant names, initials, addresses, birth dates, student numbers, organizational names and titles etc.). Indicate how personal identifiers will be secured and if they will be **retained** once data collection is complete.

Participant names will be collected through the informed consent process, however, informed consent forms are kept entirely separate from collected data. All data collected (questionnaires, test forms, physiological measures) will be alphanumerically coded with no personal identifiers. There will be a master list to which only the principal investigators have access so that we are able to link the participant's data from multiple sessions (e.g., informed consent/info session to testing session) and

from multiple sources (i.e., the saliva collections completed at home, in the lab, the physiological data, the computer responses, and the questionnaires). Informed consent forms will be retained for a period of five years after which time they will be shredded.

d) If any personal identifiers will be **retained** once data collection is complete, provide a comprehensive rationale explaining why it is necessary to retain this information, **including the retention of master lists that link participant identifiers with unique study codes and de-identified data.**

Master lists that link participant identifiers with study codes will be retained until all data has been analyzed. Master lists will be destroyed after such time.

e) State who will have access to the data.

Dr. Dawn Good (principal investigator), Julie Baker (principal student investigator), Sean Robb (student co-investigator) and research assistants associated with Dr. Good's laboratory will have access to the data. Only the principal investigators will have access to the participant identifier master list.

f) Describe the procedures to be used to ensure anonymity of participants and/or confidentiality of data **both during the conduct of the research and in the release of its findings.**

To insure confidentiality, informed consent forms will be kept separate from the data collected from the participants. Also, all data will be alphanumerically coded to ensure confidentiality. No information that could potentially reveal a participant's identity will be used in discussion, or in the reporting, of the findings. Participants will be informed that all data collected will be kept strictly confidential in a locked, safe lab to which only the principal investigator, student investigator and the research assistants will have access. To further ensure confidentiality, researchers and research assistants have signed confidentiality agreements (**see Appendix**).

g) If participant anonymity and/or confidentiality is not appropriate to this research project, explain, in detail, how all participants will be advised that data will not be anonymous or confidential.

Note that because participants are initially screened via telephone interview, the data is not considered to be anonymous. The researcher will code each telephone screening interview alphanumerically and should the participant meet the inclusion criteria and consent to participate, this code will be used for all future data collection references. During the consent and debriefing sessions, participants will be advised that while anonymity will not be preserved due to the fact that there will be a Master list advising the Principal Researchers of the participants' identity (having their name and their assigned alphanumeric code) due to the multiple contacts (phone, two test sessions) and multiple sources of data collection (home, lab; saliva collections, physiological measures, task performance), this list will be held in a separate, secure and locked location away from any data per se - with access restricted to only the Principal Investigators. During these times, the confidentiality of their data will be confirmed - that it will be secured, it will be coded alphanumerically in a database, and it will never be used individually, but instead will be used only within the context of group statistical findings.

h) Explain how written records, video/audio tapes, and questionnaires will be secured, and provide details of their final disposal or storage, including how long they will be secured and the disposal method to be used.

All raw data collected will be kept in the secure and locked file in the Principal Investigator's lab (PL 621) for a period of five years. Note that saliva samples will be disposed of into the general waste system after enzyme-linked-immunoassays have been conducted. After the five year period, data will be shredded and/or destroyed.

SECTION F -- SECONDARY USE OF DATA

23.

a) Is it your intention to reanalyze the data **for purposes other than described in this application**?

☐ **Yes** ☒ **No**

b) Is it your intention to allow the study and data to be reanalyzed by colleagues, students, or other researchers outside of the original research purposes? If this is the case, explain how you will allow your participants the opportunity to choose to participate in a study where their data would be distributed to others (state how you will contact participants to obtain their re-consent)

N/A

c) If there are no plans to reanalyze the data for secondary purposes and, yet, you wish to keep the data indefinitely, please explain why.

N/A

SECTION G -- MONITORING ONGOING RESEARCH

It is the investigator's responsibility to notify the REB using the "Renewal/Project Completed" form, when the project is completed or if it is cancelled.
http://www.brocku.ca/researchservices/forms/index.php

24. **Annual Review and Serious Adverse Events (SAE):**

a) **MINIMUM REVIEW REQUIRES THE RESEARCHER COMPLETE A "RENEWAL/PROJECT COMPLETED" FORM AT LEAST ANNUALLY**.
Indicate whether any additional monitoring or review would be appropriate for this project.

Additional review may be required for this project depending on the subject response, but it is intended for the study to be completed by September 1, 2013. REB will be notified when the final research report is completed.

***Serious adverse events** (negative consequences or results affecting participants) **must be reported** to the Research Ethics Officer and the REB Chair, **as soon as possible** and, in any event, no more than 3 days subsequent to their occurrence.

25. **COMMENTS**

If you experience any problems or have any questions about the Ethics Review Process at Brock University, please feel free to contact the Research Ethics Office at (905) 688-5550 ext 3035, or reb@brocku.ca

Informed Consent - Emotion & Cognition Study 2012

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INVITATION
You are invited to participate in a study that involves research. The purpose of this study is examine emotional experiences and emotional functioning and how these may contribute to differences in cognitive abilities or overall emotional functioning.

WHAT'S INVOLVED
Participation will take approximately 2.5 hours of your time. As a participant in this study we will ask you to be involved in providing us with physiological measures (i.e., saliva samples, measures of heart rate and skin response) and self-report measures (i.e., questionnaires) of emotional responses. We will ask you to meet with us for a brief instruction session to obtain saliva collection kits prior to your testing session. **You will be provided with saliva sample kits to take home in order to provide two samples: one prior to bedtime on the night prior to your testing session, and another sample when you wake up in the morning on the day of your testing session.** Instructions for providing the saliva samples will be provided - i.e., passively drool into a plastic tube. *Please bring saliva samples to your testing session.* We will be measuring hormones such as cortisol from your saliva samples.

During the testing session, we will collect physiological measures of emotional responses which will be recorded via electrodes and other recording equipment. The application of the recording equipment will be described to you during the application process and will involve the placement of two electrodes on your fingers, placement of a pulse oximeter on your finger to record your heart rate, and respiration bands will be placed on your upper chest and lower abdomen. Your hands and the researcher's hands will be cleansed prior to, and after, electrode placement. Please advise the researcher if you have any skin sensitivity. In order to reduce physical contact between yourself and the researcher you will be asked to assist in the placement and adjustment of the physiological recording equipment.

You will be asked to participate in cognitive tasks (e.g., paper and pencil tasks; computerized tasks), to view pictures on a computer and provide ratings of these pictures, and to complete questionnaires. Each task will be described in detail as they are introduced. You will be asked to complete tasks that involve memory abilities and cognitive skills (e.g., switching between two tasks, reading words aloud, drawing items on paper, and so forth). Two of the tasks will be viewed on a computer and will involve you making responses with a mouse. You will be asked to view and rate pictures that will include pleasant, unpleasant, and neutral scenes. You will also be asked to complete various questionnaires. Some of the questions are personal and sensitive in nature. You will be asked to provide background information about yourself such as sex, age, and level of education. Once you have completed the tasks, the specific purposes of the study will be explained to you by the researcher and you will be provided a debriefing form.

POTENTIAL BENEFITS AND RISKS
Possible benefits of participation include providing a better scientific understanding of factors involved in emotional experiences. There also may be risks associated with participation Although there are no foreseeable risks for participating in this study it is possible that you may feel uncomfortable during the testing session. For example, you may experience test performance anxiety or you may feel uncomfortable when viewing pictures of an unpleasant nature. You are welcome to ask the researcher questions, or you may contact any of the counselling contact services (listed on your debriefing form), or contact the principal investigator, Dr. Dawn Good, Registered Psychologist, should you choose.

CONFIDENTIALITY
Your name will be associated only with this form. All information collected will be confidential and kept separately from this consent form, and coded by a number assignment. A master list will be kept linking data codes to individuals until data collection for the study is complete (September 2013) after which time the master list will be destroyed. Only Dr. Dawn Good and the principal student investigator will have access to this the master list. The master list is necessary to link participant's data as we are using clinical measures that may require follow-up if scores on any measures indicate that the individual is at risk of self-harm we will need to follow appropriate procedures which involve contacting the participant. Other task data and notes taken will be kept in a locked, secure lab at all times and will be destroyed after 5 years. Only Julie Baker, Dr. Good, and research assistants will have access to this data. All research assistants have completed confidentiality agreements. In addition, any information gathered from this study that is presented at conferences or is published is summarized and group results (rather than individuals) are emphasized which preserves anonymity.

VOLUNTARY PARTICIPATION
Participation in this study is voluntary. If you wish, you may decline to answer any questions or participate in any component of the study. Further, you may decide to withdraw from this study at any time and may do so without any penalty or loss of benefits to which you are entitled. If you choose to withdraw at any time please verbally inform the researcher.

PUBLICATION OF RESULTS
This study forms part of a Ph.D. research project, a Master's project, and an undergraduate thesis. Results of this study may be published in professional journals and presented at conferences. Feedback about this study will be available after September 2013. Please contact the principal faculty or student investigators (Dr. Dawn Good or Julie Baker) via the contact information provided on this form.

CONTACT INFORMATION AND ETHICS CLEARANCE
If you have any questions about this study or require further information, please contact Dr. Dawn Good or Julie Baker at Brock University using the contact information provided above. This study has been reviewed and received ethics clearance through the Research Ethics Board at Brock University. If you have any comments or concerns about your rights as a research participant, please contact the Research Ethics Office at (905) 688-5550 Ext. 3035, reb@brocku.ca.

Thank you for your assistance in this project. Please keep a copy of this form for your records.

CONSENT FORM
I agree to participate in this study described above. I have made this decision based on the information I have read in the Information-Consent Letter. I have had the opportunity to receive any additional details I wanted about the study and understand that I may ask questions in the future. I understand that I may withdraw this consent at any time.

Name: _____

Signature: _____ **Date:** _____

[ ] I acknowledge that I am participating in this study for a maximum of 2.5 research participation hours in a psychology course (see below)

COURSE (please circle only one course):

PSYC 1F90 2P12 2P20 2F23 2P36 2P37 3P39 Other: _____

O

R

[ ] I acknowledge that I am receiving monetary compensation for participation in this study

[ ] I have explained this study to the participant

Researcher's signature _____ **Date:** _____

THANK YOU FOR YOUR TIME AND PARTICIPATION IN THIS STUDY!!!!

Emotion & Cognition Study

DEBRIEFING FORM

PURPOSE

Thank you for your participation in this research study. As you are aware, this research study was conducted by Dr. Dawn Good, and her students, in the Psychology Department at Brock University. The purpose of this study is to examine individual differences in emotional experience and how this may vary for persons with a history of head trauma relative to persons with no history of head trauma. We are also examining stress responses of persons with and without a history of head trauma. To induce heightened arousal, you were asked to view and rate pictures of an unpleasant, pleasant, or neutral nature. We will investigate responses to life stressors and laboratory stressors as a function of a history of head trauma via physiological (i.e., cortisol response, electrodermal activation, and heart rate) and self-reported indices (e.g., questionnaires). Cortisol, a stress hormone measured from saliva, is hypothesized to be different for those with and without a history of head trauma. We are collecting data from persons who have no history of head trauma and those with mild, moderate, or severe traumatic brain injury. We did not tell you about our interest in whether or not there is any indication of you having sustained a previous head trauma (e.g., concussion, moderate brain injury) because there is published research that suggests that informing participants that head trauma is a study variable of interest can influence subsequent performance (Suhr & Gunstad, 2002; 2005) i.e., may negatively affect how individuals approach and respond to task demands. As a result, we did not advertise our interest in a history of head trauma, nor tell you about it prior to your participation.

BACKGROUND

Previous research has shown that between 25% and 45% of undergraduate students have sustained a mild head injury. Research from our lab (Brock University Neuropsychology Cognitive Research Lab) has shown that individuals with mild head injury demonstrate 'underarousal' (i.e., they are less stressed) relative to their peers, despite reporting increased life stressors such as financial or relationship difficulties. Our research has suggested that when higher levels of arousal are reported by individuals with mild head injury, their cognitive performance has shown to be optimally enhanced. Thus, we are examining whether certain stimuli can modify emotional arousal levels in persons who have/have not sustained previous head trauma and if cognitive performance can be modified.

Therefore we modified arousal through a 'stressor' - type task in which we asked you to view and rate pictures that were highly arousing. You completed various neuropsychological tests and psychological questionnaires were administered to examine cognitive, emotional, social, personality and health factors. For example, the questionnaires you completed provided indices of anxiety, optimism, empathic abilities, emotional-social capabilities, and morningness-eveningness traits. The standardized neuropsychological tests chosen for this study were subtests of the Wechsler Memory Scale-IV (2009), Wechsler Adult Intelligence Scale - IV (2009), the Delis Kaplan Executive Function System (2002), and the Iowa Gambling Task (Bechara et al., 1994). These tests were used as they involve executive functions including abstract reasoning, decision making, memory, cognitive flexibility, attention, and planning.

FINAL REPORT

Your participation is important for us to be able to understand the relationships between subtle brain functions and everyday responses to social/environmental stimuli. This research will improve the understanding of emotional functioning of persons with and without a history of neural disruption, particularly with respect to stress responses, and has implications for clinical practice in the fields of neuropsychology and rehabilitative medicine.

You are invited to view the results of the study by its completion (September, 2013). Findings from this research study form parts of a Ph.D. thesis, a M.A. thesis, as well as an undergraduate research project and may be presented at conferences and/or in published format. Group, not individual, responses will be emphasized. It is important that you not discuss the procedures of participating in this study (until the end of term academic year 2012-2013) with other students or friends as it may effect our results. We appreciate your cooperation.

If you are interested in obtaining a copy of the final report of this study, contact the NCR lab at Brock University (905) 688-5550 ext. 3556, or 5523 - the lab offices of the primary investigator, Dr. Dawn Good (dawn.good@brocku.ca).

CONTACT

If you have *any* questions regarding this study, its purpose or procedures, please feel free to contact us!

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** or the principal investigator Dr. Dawn Good, Registered Psychologist**.**

Should you like more information regarding head trauma please visit the following websites: The Ontario Brain Injury Association (OBIA): http://www.obia.ca/ , The Ontario Neurotrauma Foundation (ONF): http://www.onf.org/ or the Brain Injury Association of Niagara (BIAN): www.**bianiagara**.org).

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

Thank you again for your time and participating in this study!!!
If you have any questions or concerns please feel free to contact us at the Brock University Neuropsychology Cognitive Research Lab:

Principal Student Investigator:
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Principal Investigator:
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(905) 688-5550, ext. 3869

Participants Needed!!!

For research investigating

EMOTION AND COGNITION

As a participant you will be eligible for:
2.5 research participation hours or monetary compensation

Participation in this study will involve:

- Completion of questionnaires and cognitive tasks during different arousal states (i.e. increased vigilance)
- Physiological measurement recording such as heart rate, blood pressure, and electrodermal response. Saliva samples will be collected.
- To participate in this study you must be fluent in English and meet eligibility requirements via a short telephone interview.

For more information or to participate please contact:

Julie Baker
Ph.D. Candidate
Psychology Department
(js01cb@brocku.ca)

Supervisor: Dr. Dawn Good Dawn.Good@brocku.ca (ext. 3556)

This study has been reviewed by and received ethics clearance through the Office of Research Ethics, Brock University 905-688-5550 ext. 3035

Emotion and Cognition Study
Call ext. 3556 for Julie Baker
Email: js01cb@brocku.ca

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Everyday Living Questionnaire - Revised (2012)

Please fill in or circle an answer for each of the following. If you have any questions regarding clarification please ask the researcher. Thank you for your time and effort:

1. How old are you? ____
2. Gender? M___ F___
3. What is the highest level of education you have presently completed?
    a. Less than high school
    b. High School/Grade 12
    c. College 1 2 3 4 5+ (Years)
    d. University 1 2 3 4 5+ (Years)
4. What is the highest level of education your **father** has received?
    a. Less than high school
    b. High School/Grade 12
    c. College 1 2 3 4 5+ (Years)
    d. University 1 2 3 4 5+ (Years)
5. What is the highest level of education your **mother** has received?
    a. Less than high school
    b. High School/Grade 12
    c. College 1 2 3 4 5+ (Years)
    d. University 1 2 3 4 5+ (Years)
6. What is the overall average income your parents/guardians (If divorced, income of both parents combined)?
    a. Under $25,000
    b. $25,000 - $49,999
    c. $50,000 - $74,999
    d. $75,000 - $99,999
    e. $100,000 - $124,999
    f. $125,000 - $149,000
    g. $150,000 or more
7. What ethnicity do you identify most with:
    a. Hispanic
    b. Caucasian
    c. European
    d. African
    e. Chinese
    f. East Indian
    g. West Indian
    h. Japanese
    i. Other

        Specify: _____
8. In elementary school what were your career goals (what did you want to be when you grow up)? _____

9. In high school what were your career goals?
   _____
10. Have you switched your major during university? Yes No
    If yes, please describe the change _____
11. What is your major affiliated with (eg, Social Science, Humanities, etc.)
    a. Social Science
    b. Humanities
    c. Maths and Sciences
    d. Education
    e. Applied Health Science
    f. Business
    g. Undeclared
12. Are you currently pursuing an undergraduate or graduate degree?
    a. Undergraduate
    b. Graduate
13. **If you answered 'a' to question 12**, are you planning on pursuing graduate studies after your undergraduate degree?
    a. Yes
    b. No
    c. Not sure yet
14. What do you currently hope to achieve with your education/what career do you want to pursue?
    _____
    _____
15. Are you currently working while attending school? Yes No
16. **If you answered yes to question 15,** how many hours per week do you work?
    a. Less than 5
    b. 6 to 10
    c. 11 to 15
    d. 16 to 20
    e. More than 20 per week
17. **If you answered yes to question 15**, why do you work during school?
    a. Need the money
    b. Because you enjoy the job
    c. To fill spare time
    d. Other _____
18. Do you work a summer job when school is not in session? Yes No
19. **If you answered yes to question 18**, how many years have you had this job? _____
20. Which hand is your dominant hand (i.e. are you right or left-handed)?
    a. Right
    b. Left
    c. Both
21. Have you ever been hospitalized for (circle any that apply):

a. Fractures Y N
b. Illness Y N
c. Surgery Y N
d. Neurological Complications Y N
e. Other Y N

If you answered Y to any of the above, briefly please provide details (e.g. How old were you? How did it happen)

_____

_____

_____

22. Have you ever been diagnosed with a neurological condition? Y N
23. Have you ever been diagnosed with a psychiatric condition? Y N
24. Are you currently taking any prescribed medications for a neurological or psychiatric condition? Y N
    a. If Yes, if you wish to disclose what medications please do so: _____
25. 6. [If female] Are you currently taking contraceptive pills or contraceptive injections (e.g., birth control pill or Depo-Provera)? Y [ ] N [ ]
26. Have you ever sustained an injury to your head with a force sufficient to alter your consciousness (e.g. dizziness, vomiting, seeing stars, or loss of consciousness, or confusion)? Y N

    [If you answer **No** to this question you may move ahead to **question 40**]

If yes to question 26, please answer the following questions (if you have had more than one injury, please refer to the *most recent* time you injured your head):

27. If you answered yes to question 26, did you experience these symptoms for more than 20 minutes? Y N
28. Did you experience a loss of consciousness associated with the head injury? Y N
    i. If so, how long was the loss of consciousness?
        1. [ ] <5 minutes
        2. [ ] <30 minutes
        3. [ ] <24 hours
        4. [ ] <1 week
        5. [ ] <1 month
        6. [ ] >1 month
29. If applicable, where did you strike your head?
    a. Front of the head
    b. Right side of the head
    c. Left side of the head
    d. Other Provide brief details: _____
    e. I can't remember
30. How did you injure your head?
    i. [ ] Motor vehicle collision

ii. [ ] Sports-related injury
iii. [ ] Falling
iv. [ ] Other Please Specify: _____

31. Please briefly describe the incident during which the head injury occurred:

_____
_____
_____

32. Please answer the following questions:
    a. Did the head injury result in a concussion? Y N
    b. Did it require stitches? Y N
    c. Did you receive medical treatment for your injury? Y N
    d. Did you stay overnight at a medical care facility? Y N
    e. Approximately how old were you at the time? _____
    f. How many months or years have passed since you hit your head? _____
33. Have you sustained more than one injury to your head with a force sufficient to alter your consciousness (e.g. dizziness, vomiting, seeing stars, or loss of consciousness, or confusion)? Y N
34. **If you answered yes to question 33**, did you experience these symptoms for more than 20 minutes? Y N

If you responded yes to question 33, please answer the following with respect to your *least recent* head injury:

35. Did you experience a loss of consciousness associated with the head injury? Y N
    i. If so, how long was the loss of consciousness?
        1. [ ] <5 minutes
        2. [ ] <30 minutes
        3. [ ] <24 hours
        4. [ ] <1 week
        5. [ ] <1 month
        6. [ ] >1 month
36. If applicable, where did you strike your head?
    a. Front of the head
    b. Right side of the head
    c. Left side of the head
    d. Other Provide brief details: _____
    e. I can't remember
37. How did you injure your head?
    i. [ ] Motor vehicle collision
    ii. [ ] Sports-related injury
    iii. [ ] Falling
    iv. [ ] Other Please Specify: _____
38. Please briefly describe the incident during which the head injury occurred:

_____

_____
_____

39. Please answer the following questions:
    a. Did the head injury result in a concussion? Y N
    b. Did it require stitches? Y N
    c. Did you receive medical treatment for your injury? Y N
    d. Did you stay overnight at a medical care facility? Y N
    e. Approximately how old were you at the time? _____
    f. How many months or years have passed since you hit your head? _____

**********_If you were instructed to move ahead to question 40 please begin here_**********

40. Have you ever experienced any other neural trauma (e.g. stroke, anoxia)? Y N
    a. **If yes,** please explain
       _____
       _____
41. Do you smoke cigarettes? Y N
    a. **If yes,** approximately how many a day? _____
42. Do you regularly engage in consuming alcohol? Y N
    a. If yes, how many drinks per week do you consume? _____
    b. On average how many drinks would you consume in one outing? _____
43. Do you engage in recreational drug use (e.g. smoke marijuana, drop ecstasy, etc.)? Y N
44. Did you consume caffeine today (e.g. coffee, tea, energy drink, chocolate)? Y N
    a. **If yes**, how much?
       1 2 3 more than 3
    b. **If yes**, how much time has passed since you last consumed caffeine today?
       Less than 1 hour More than 1 hour
45. Do you have sensitivity to perfume or scents? Y N
    If yes, please rate your sensitivity:
    Not at all Very
    1 2 3 4 5 6 7 8 9
46. Do you have a valid driver's license? Y N
    a. **If yes**, how long have you had a driver's license? 1-3 years 4-6 years 7+ years
47. Do you wear glasses or contacts? Y N
48. Do you live: on your own with roommates other
    with parents/guardians with partner
49. During elementary school, what were your average grades?
    a. A- to A+
    b. B- to B+
    c. C- to C+
    d. D- to D+
    e. Other _____
50. During high school, what were your average grades?
    a. A- to A+

b. B- to B+
c. C- to C+
d. D- to D+
e. Other _____

51. Currently in University, what are your average grades?
    a. 90 to 100
    b. 80 to 89
    c. 70 to 79
    d. 60 to 69
    e. 50 to 59
    f. Other _____
52. How many university credits are you taking this semester?
    0 0.5 1 1.5 2 2.5 3 3.5 4 4.5 5 5.5 6
53. How many hours per week (on average) do you attend lectures/seminars/tutorials?
    Less than 5 6 – 9 10 – 12 13 – 16 17 – 20 21+
54. How many hours per week (on average) do you spend doing course readings for lecture/seminar/tutorial?
    Less than 3 4-6 7-9 10+
55. How many hours per week (on average) do you spend doing homework/assignments for lecture/seminar/tutorial?
    Less than 3 4-6 7-9 10+
56. 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
57. Have you ever received any extra assistance during your educational history? Y N
    Please circle any that apply and indicate when you received the assistance:
    E = Elementary school H = High school U = University
    a. Learning resource teacher E H U
    b. Tutor E H U
    c. Educational assistant E H U
    d. Speech language pathologist E H U
    e. Occupational therapist E H U
    f. Physical therapist E H U
    g. Other: Please Specify _____ E H U
58. Have you ever been diagnosed or classified as having a Learning Disorder? Y N
59. Do you consider yourself a musician? Y N
60. Have you ever considered yourself to be a musician? Y N
61. If you answered yes to either question 41 or 42, did you play/perform:
    a. Professionally
    b. Recreationally
62. If you answered yes to either question X or X, how long did you play/perform for? _____
63. What age did you start playing/performing at: _____ years
64. How often do you listen to music? _____ hours per week

65. Please indicate the type of music you listen to most often (can circle more than one):
    a. Country
    b. Classical
    c. Rock
    d. R&B
    e. Blues
    f. Independent
    g. Jazz
    h. Pop
    i. Electronic (house/dance)
    j. Folk
    k. Opera
    l. Other: Provide brief details: _____
66. 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
67. On a scale of 1 to 9 how stressful would you rate your day-to-day life:

    Not at all Very

    1 2 3 4 5 6 7 8 9
68. Do you consider yourself to be an athlete? Y N
69. What extracurricular sport(s) did you play in:
    a. Elementary school:
        i. Please describe (e.g. skating, baseball, etc.) – indicate if it was recreational (R) or competitive (C)

_____

        ii. How often did you play sports (per week)? _____
    b. High school:
        i. Please describe (e.g. skating, baseball, etc.) – indicate if it was recreational (R) or competitive (C)

_____

        ii. How often did you play sports (per week)? _____
    c. University:
        i. Please describe (e.g. skating, baseball, etc.) – indicate if it was recreational (R) or competitive (C)

_____

        ii. How often did you play sports (per week)? _____

70. In university, do you participate in any organized teams/sports? Y N
    If no, please skip to question 81
    If yes, please list the sports below and indicate if they are:

Community/Recreational	Intermural	Varsity
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____

71. How many consecutive years, including the current season, have you participated in each sport?
    _____
    _____
    _____
72. How many practices do you attend per week (per sport)?
    _____
    _____
    _____
73. How long in duration is the average practice (per sport)?
    _____
    _____
    _____
74. What does the typical practice consist of?
    _____
    _____
    _____
    _____
    _____
75. In the last season, did you participate in any organized tournaments? Y N
    If yes, for which sport(s)? _____
    If no, why not? _____
76. Do you plan to attend any organized tournaments this season? Y N
    If yes, for which sport(s)? _____
    If no, why not? _____
77. Do any of your sports continue over the summer months when school is not in session? Y N
    If yes, please describe any differences between the in season (school year) and off season (summer months) practices or workouts.
    _____
    _____
    _____
    _____
78. Do you exercise on a regular basis? Y N

79. Outside of organized practices for sports/teams, how many times per week do you exercise/work out? _____
80. Outside of organized practices for sports/teams, how long in duration is the average exercise/work out? _____
81. Outside of organized practices for sports/teams, what types of activities do you typically do to exercise/work out?

    _____

    _____

    _____
82. Do you participate in any non-athletic extracurricular activities, clubs or groups?

    _____

    _____

    _____

    If yes, how many hours a week (combined) do you spend at these activities? _____
83. When you ride a bike/skate/etc. do you wear a helmet? Y N not applicable
84. Do you regularly engage in relaxation techniques (e.g. deep breathing or yoga): Y N
    a. **If yes**, how many times a week do you engage in relaxation methods? _____
    b. Please describe: _____
85. Was last night's sleep typical for you? Y N

    If No, what was different (better, worse)? _____

    Why was it different? (stress, room temperature, noise, etc.)

    _____
86. Please indicate how well you slept last night by circling a number:

    Worse Possible 1 2 3 4 5 6 7 Best Possible

    Sleep Sleep
87. Please indicate how you feel right now by circling a number

    Very Sleepy 1 2 3 4 5 6 7 Very Alert
88. Have you had anything out of the ordinary occur in the past day or so? Y N

    If yes, please explain:

    _____

    _____
89. 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
    Disrupted Sleep

90. Please indicate how your day has been so far by circling a number:

Calm	1	2	3	4	5	6	7	8	9	10 Busy
Pleasant	1	2	3	4	5	6	7	8	9	10 Unpleasant
NOT Stressful	1	2	3	4	5	6	7	8	9	10 VERY Stressful

91. Please rate each of the following symptoms based on how you may have been affected during the past 2 months according to the following scale.

FREQUENCY	INTENSITY	DURATION
1 = Not at all	1 = Not at all	1 = Not at all
2 = Seldom	2 = Seldom	2 = A Few Seconds
3 = Often	3 = Clearly Present	3 = A Few Minutes
4 = Very often	4 = Interfering	4 = A Few Hours
5 = All of the time	5 = Crippling	5 = Constant

	FREQUENCY	INTENSITY	DURATION
Headache			
Dizziness			
Irritability			
Memory Problems			
Difficulty Concentrating			
Fatigue			
Visual Disturbance			
Aggravated by Noise			
Judgment Problems			
Anxiety			

Question 89 format adapted from Holmes & Rahe (1967); Question 91 from Gouvier et al. (1992).

Thank you for your time and consideration in completing this questionnaire! ☺

EK QUESTIONNAIRE

1. Rate, on a scale of -10 to +10, how good or bad you think **deck A** is, where -10 means that it is terrible and +10 means that it is excellent. _____
2. Why did you rate **deck A** with...(your rating from question 1)?

_____

_____

3. Rate, on a scale of -10 to +10, how good or bad you think **deck B** is, where -10 means that it is terrible and +10 means that it is excellent. _____
4. Why did you rate **deck B** with...(your rating from question 3)?

_____

_____

5. Rate, on a scale of -10 to +10, how good or bad you think **deck C** is, where -10 means that it is terrible and +10 means that it is excellent. _____
6. Why did you rate **deck C** with...(your rating from question 5)?

_____

_____

7. Rate, on a scale of -10 to +10, how good or bad you think **deck D** is, where -10 means that it is terrible and +10 means that it is excellent. _____
8. Why did you rate **deck D** with...(your rating from question 7)?

_____

_____

9. In answering the questions that follow, consider the following definitions. Your "**winning amount**" for a trial is the amount you won that trial. Your "**loss**" on a trial is the amount you lost on that trial. Your "**net result**" for a trial is the amount you won minus the amount you lost on that trial. Do you understand these definitions and the differences between the three terms? If not please contact the research assistant.

> *Winning Amount* – amount you won during that trial
>
> *Loss* – amount you lost on that trial
>
> *Net Result* – the amount you won minus the amount you lost on that trial

   a. Now suppose you were to select 10 cards from **deck A**
      i. What would you expect your average **net result** to be? _____
      ii. What would you expect your average **winning amount** to be? _____
      iii. In how many of the 10 trials would you expect to get a **loss** (not necessarily a net loss)? _____
      iv. For those trials in which you would get a loss, what would you expect the **average loss** to be? _____
   b. Now suppose you were to select 10 cards from **deck B**
      i. What would you expect your average **net result** to be? _____
      ii. What would you expect your average **winning amount** to be? _____
      iii. In how many of the 10 trials would you expect to get a **loss** (not necessarily a net loss)? _____
      iv. For those trials in which you would get a loss, what would you expect the **average loss** to be? _____
   c. Now suppose you were to select 10 cards from **deck C**

i. What would you expect your average **net result** to be? _________________

ii. What would you expect your average **winning amount** to be? _________________

iii. In how many of the 10 trials would you expect to get a **loss** (not necessarily a net loss)? _________________

iv. For those trials in which you would get a loss, what would you expect the **average loss** to be? _________________

d. Now suppose you were to select 10 cards from **deck D**

i. What would you expect your average **net result** to be? _________________

ii. What would you expect your average **winning amount** to be? _________________

iii. In how many of the 10 trials would you expect to get a **loss** (not necessarily a net loss)? _________________

iv. For those trials in which you would get a loss, what would you expect the **average loss** to be? _________________

10. On a scale of 0 to 100, how much do you think that you know what you should do in this game in order to win as much money as possible (or, if you can't win, to avoid losing money as much as possible)? **0** means that you have no idea of what you should do and feel that you still need to explore the game more and **100** means that you know exactly what you should do and have no doubts that would be the best strategy. _________________

0 -100

No idea what I should do and feel that I still need to explore the game more		I know exactly what I should do and have no doubt that it is the best strategy

11. Now suppose I told you that you could only select cards from one of the decks until the end of the game, but that you were allowed to choose now the deck from which you draw your cards. Which of the four decks would you pick (A, B, C, or D)? _____

Participants Ratings of Conscious knowledge of advantageous strategy Scoring Key:

- Question 9 (Ai), (Bi), (Ci) and (Di) – participants provide the highest average expected net (The amount you won minus the amount you lost averaged across trials) to one of the two best decks
- Question 9 (Aii-iv), (Bii-iv), (Cii-iv) and (Dii-iv) – provides an indication of participant's knowledge about outcomes for each deck in terms of each deck's reward value, probability of getting a loss and mean loss value.
    - The respective questions allow one to calculate the mean net that a participant should expect, based on their knowledge about outcomes of each deck.
    - Formula
        - **Calculated net = Q9 (Aii) + (Q9 (Aiii) / 10) × Q9 (Aiv)**
            - Q9 (Aii) – Asks about average winnings
            - Q9 (Aiii) – in 10 trials, how many times would one expect a loss (not necessarily a net loss)
            - Q9 (Aiv) – Asks about average loss
        - Please note that the A represents the respective deck