University of Windsor
Scholarship at UWindsor

11-7-2015

Emotional Responses Following Sports-Related Concussion; a pilot study with controls

Eva Keatley

University of Windsor

Emotional Responses Following Sports-Related Concussion; a pilot study with controls

By

Eva S. Keatley

A Thesis
Submitted to the Faculty of Graduate Studies
through the Department of Psychology
in Partial Fulfillment of the Requirements for
the Degree of Master of Arts
at the University of Windsor

Windsor, Ontario, Canada

2015

Emotional Responses Following Sports-Related Concussion; a pilot study with controls

by

Eva S. Keatley

APPROVED BY:

_____
C. McGowan
Department of Kinesiology

_____
L. Buchanan
Department of Psychology

_____
C. Abeare, Advisor
Department of Psychology

June 29, 2015

DECLARATION OF ORIGINALITY

I hereby certify that I am the sole author of this thesis and that no part of this thesis has been published or submitted for publication.

I certify that, to the best of my knowledge, my thesis does not infringe upon anyone's copyright nor violate any proprietary rights and that any ideas, techniques, quotations, or any other material from the work of other people included in my thesis, published or otherwise, are fully acknowledged in accordance with the standard referencing practices. Furthermore, to the extent that I have included copyrighted material that surpasses the bounds of fair dealing within the meaning of the Canada Copyright Act, I certify that I have obtained a written permission from the copyright owner(s) to include such material(s) in my thesis and have included copies of such copyright clearances to my appendix.

I declare that this is a true copy of my thesis, including any final revisions, as approved by my thesis committee and the Graduate Studies office, and that this thesis has not been submitted for a higher degree to any other University or Institution.

ABSTRACT

Mild traumatic brain injury is associated with new onset emotional symptoms (Caroll et al., 2014; Konrad et al., 2011). This study will address a gap in the literature by examining the relationship between mTBI, autonomic arousal, and emotional symptoms in the acute phase of a concussion in athletes. In this pilot study, skin conduction and heart rate changes in response to emotional stimuli were measured in 25 undergraduate controls. Results indicate that emotional valence of facial expressions did not elicit the expected differential physiological responses as suggested by previous research, but habituation to an acoustic startle over repeated exposures was observed. Changes to the protocol for future research with concussed athletes are discussed.

DEDICATION

I dedicate this thesis to my mother and father who have gone above and

beyond to support me.

ACKNOWLEDGEMENTS

I want to thank Dr. Christopher Abeare for his continued support and

encouragement with my research endeavors.

TABLE OF CONTENTS

LIST OF TABLES

.

LIST OF FIGURES

LIST OF APPENDICES

CHAPTER I

INTRODUCTION

Mild traumatic brain injury (mTBI) is associated with new onset emotional symptoms (Caroll et al., 2014; Konrad et al., 2011). The neurological underpinnings of these emotional symptoms are poorly understood. Some research suggests that autonomic functioning – a critical component of emotion – is disrupted by TBI and may be the cause of these emotional symptoms (Baker & Good, 2014; Griesbach, Hovda, Tio, & Taylor, 2011; Rushby et al., 2013a). However, this literature is limited to animal studies and to TBI patients in the chronic phase of their injury. The use of these populations makes drawing direct links between the TBI, autonomic functioning, and emotional symptoms challenging; animals cannot report emotional experiences and chronic TBI patients' functioning may be confounded by post-injury factors (e.g., life stressors, disability, neurological atrophy). This study is designed to fill the gap in the literature by exploring the relationship between mTBI, autonomic functioning, and emotional symptoms in a homogenous population in the acute phase of their injury - concussed athletes. This paper consists of the first phase of the study, which pilots the protocol on a control sample.

Background

Definition of mTBI

Mild TBI, also referred to as concussion, is defined as an insult to the head that results in: a) a loss consciousness of 30 minutes or less, b) posttraumatic amnesia not greater than 24 hours, or c) any alteration in mental state at the time of the accident (e.g., feeling dazed, disoriented, or confused; Mild Traumatic Brain Injury Committee, 1993). Mild TBI is a controversial diagnosis because it is characterized by neurological changes that are not readily detectable by traditional imaging techniques, such as computed tomography (CT) and magnetic resonance imaging (MRI) scans. Common symptoms following mTBI include headaches, dizziness, irritability, difficulty concentrating, memory problems, fatigue, visual disturbances, sensitivity to noise, judgment problems, depression, and anxiety (Ryan & Warden, 2003). The majority of individuals who suffer a mTBI recover within a matter of days, however, a minority experience ongoing cognitive, physical, and emotional problems (King, 1996; Meares et al., 2011).

Emotional Changes Following mTBI

Mild TBI is associated with new onset emotional problems - especially depression and anxiety symptoms - among war veterans, athletes, motor-vehicle accident victims, and others (Carroll et al., 2014; Konrad et al., 2011; Mooney & Speed, 2001; Schoenhuber & Gentilini, 1988). In a large-scale TBI study, Fann et al., (2004) found that the incidence of psychiatric illness in the first year post-injury was 49% in moderate to severe TBI, and 34% in mild TBI, as compared to 18% in a control group. These authors found that mTBI was associated with a higher risk of any psychiatric diagnosis even in individuals with no psychiatric history. One meta-analysis of 12 studies found prevalence rates of anxiety following TBI as high as 29% with

the rate dropping slightly to 23% when only mTBI was included in the analysis (Moore et al., 2006). Mooney and Speed (2001) found that among a sample of 80 participants with mTBI enrolled at an outpatient TBI clinic, 24% developed an acquired anxiety disorder and 44% a depressive disorder suggesting high rates of emotional disturbance in this treatment-seeking sample. Research in the past decade has found that veterans who sustained an mTBI are at greater risk for developing posttraumatic stress disorder when compared to their counterparts who sustained other injuries (Riechers, Ruff, Ruff Wang, & Piero, 2012; Vasterling, Verfaellie & Sullivan, 2009). The challenge with this literature is that many populations studied, such as veterans and motor vehicle accident victims, endure their injury during a psychologically stressful event where post-injury factors (e.g., death/injury of a friend, disability) may affect their emotional state. One mTBI population where risk for psychological trauma is low is among athletes.

The nature of sports-related concussion (SRC) is unique from other forms of mTBI in that it is associated with less severe injury, faster recovery, and less disability (Rabinowitz, Li, & Levin, 2014). In addition, athletes are more psychologically prepared for the likelihood of sustaining a head injury and may have lower incidence of pre-existing psychiatric disorders (Rabinowitz et al., 2014).

Given this unique set of protective factors, it is important that studies have found SRC is associated with emotional symptoms. Yang, Peek-Asa, Covassin, and Torner (2015) found that among 67 concussed collegiate athletes 20% reported symptoms of depression, 34% reported anxiety, and 14% reported both depression and anxiety one week after the injury.

Mainwaring and Bischop (2004) studied a sample of concussed collegiate athletes in four sessions; baseline, within 72 hours of concussion, about one week after injury, and 2 weeks after

injury. They found that, as compared to uninjured teammates and undergraduate controls, concussed athletes experienced significantly greater mood disturbance following their injury, especially on scales of depression, confusion, and tension. Levels of mood disturbance decreased over time in the concussed group, returning to baseline levels by the second week, implicating the injury as the cause of the distress. These authors also addressed an important question regarding cause of depression; they found that levels of depression and overall mood disturbance returned to baseline levels prior to athletes returning to play. This suggests the injury itself, and not withdrawal from activity, as the cause of distress.

In a later study, Mainwaring, Hutchison, Bisschop, Comper, and Richards (2010) compared emotional functioning in 16 concussed athletes to 7 with an ACL injury and 28 un-injured athletes. Athletes were scheduled for assessments 1, 4, 8, 15, 22, and 29 days post injury. The concussed athletes demonstrated significantly higher scores in total mood disturbance with larger variability than athletes with an ACL injury at the first assessment following injury. This mood disturbance experienced by the concussed athletes resolved after 7 days while the ACL group's total mood disturbance gradually increased over time reaching peak levels 12 days post-injury. This study provides evidence of an injury specific mood disturbance after concussion that is distinct from other types of injuries and that resolves within a week after the injury. The greater mood disturbance scores within the first week of the concussed athletes as compared to ACL injured athletes suggests that post-concussive emotional distress is not explained by withdrawal from play or anxiety regarding return to play. Importantly, these findings report resolution of emotional disturbances on a similar timeline as neuropsychological (Belanger & Vanderploeg, 2005) and neurophysiological models of concussion (Giza & Hovda, 2001; 2014).

Covassin et al., (2014) used a different measure (State-Trait Inventory) than Mainwaring and colleagues (Profile of Mood States) and found no significant elevation of anxiety in concussed athletes. These authors compared 63 concussion injuries to 63 orthopedic injuries matched for athletes' sex, sport, and playtime loss due to injury. Athletes were tested within 1 week of the injury. These authors found no difference in state or trait anxiety between the concussed and the orthopedic-injury groups. This study also differed from previous studies in that it did not measure depressive symptoms and did not analyze the effect of time since injury, an important variable as evidence suggests SRC mood disturbance is transient (Mainwaring et al., 2010).

These studies provide evidence that mTBI is associated with new onset emotional symptoms although results are mixed depending on the measures used (Covassin et al., 2014). Evidence suggests that changes in mood following an SRC are transient and may be related to the injury itself rather than a reaction to psychological trauma – as suggested in other mTBI populations (King, 2008). Although poorly understood, there is growing evidence that neurological changes following mTBI may cause these emotional symptoms.

Physiological Changes Following mTBI

Modern imaging techniques and animal studies now provide evidence that mTBI results in subtle neurological changes that may be responsible for post-injury cognitive, physical, and emotional problems (Giza & Hovda, 2014; Shenton et al., 2012). In the acute phase, just after concussion, an mTBI causes a neurometabolic cascade involving the release of glutamate, potassium efflux, and sodium and calcium influx. In an effort to restore homeostasis, ionic pumps use up their cells' energy supplies causing diffuse hypoactivity (Giza & Hovda, 2014).

This is evidenced by a low glucose metabolic rate, which can last up to 7-10 days in adult animals. During this period, concussed animals have shown impairments in spatial learning (Yoshino, Hovda, Kawamata, Katayuma & Becker, 1991).

These metabolic changes can also lead to damage to the white matter fibers located in the brain. Diffusion tensor imaging (DTI) is a relatively new imaging technique that is sensitive to diffuse axonal injury and can detect subtle changes to white matter fiber tracts injured by mTBI. These studies provide evidence of two different types of axonal injury: cytotoxic and vasogenic edema (Shenton et al., 2012). Both types of injuries prevent neurons from transmitting messages appropriately. Research has linked white matter abnormalities to post-concussive symptoms, including emotional symptoms (Bazarian et al., 2007), neuropsychiatric symptoms (Sharp & Ham, 2011), and performance on cognitive measures (Miles et al., 2008). It is these changes in the physiology of the brain that are suspected to cause disrupted autonomic functioning – a critical component of emotion.

Autonomic Functioning Following TBI

The review of the literature suggests that a TBI disrupts regular autonomic arousal and responsivity (de Sousa et al., 2012; Rushby et al., 2013a; Fisher et al., 2014). Because autonomic functioning is an important component of emotional experiences it may be that this disruption is associated with emotional symptoms. However, the existing literature does not attempt to draw direct links between TBI, autonomic functioning, and emotional symptoms because of the populations used have their limitations; animals cannot report emotional experiences and chronic TBI patients' functioning may be confounded by post-injury factors (e.g., life stressors, disability, neurological atrophy).

Despite these limitations, the existing literature does indicate TBI disrupts autonomic arousal and responsivity, which has long been a key component in theories of emotion (James, 1884; Lange, 1887; Suchy, 2011). Suchy (2011) defined such physiological processes as Reflexive Emotional Responses that include both 1) involuntary skeletal responses, and 2) autonomic/endocrine responses. According to Suchy, the motor system that produces emotional responses to stimuli reacts automatically and involuntarily and is responsible for such processes as spontaneous facial expressions, increased heart rate, dilated pupils, piloerection, and perspiration. This is evidenced in normal adults who typically demonstrate facial mimicry (Hess & Blairy, 2001), changes in skin conductance (Vrana & Gross, 2004), and changes in cardiac output when viewing facial expressions (Jönsson & Sonnby-Borgström, 2003; Yartz & Hawk, 2002). Neuroanatomic substrates involved in autonomic responses are distributed throughout cortical and sub-cortical regions and include the hypothalamus (emotional relay center), brain stem, and the anterior cingulate cortex (ACC; in sympathetic regulation of cardiac functions; Suchy, 2011). Problems with emotional functioning following TBI can be organized as those associated with hyperarousal (e.g., increased psychological and physiological tension marked by anxiety, exaggeration of startle responses, insomnia, fatigue, and heightened emotional reactivity; Elder et al., 2012; Harbi, 2013; Joiner Jr, et al., 1999) or hypoarousal (e.g. apathy; attenuated emotional responding; flattened affect (Heilman, Schwartz & Watson, 1978).

Hyperarousal

Among the TBI literature, evidence of hyperarousal comes from two sources: 1) greater incidence of anxiety symptoms and PTSD following TBI (as discussed above), and 2) increased

stress behaviours in animals immediately following an experimentally induced concussion (Elder et al., 2012; Griesbach et al., 2011; Heldt et al., 2014; Meyer, Davies, Barr, Manzerra & Forster, 2012; Reger et al., 2011) . For instance, Griesbach et al. (2011) employed a technique called lateral fluid percussion injury (FPI) to mimic cerebral concussion in rats. This technique involved applying a brief fluid pressure pulse on the intact dura of the animal. They found that concussed, rats exhibited a heightened stress response compared to non-concussed rats; the concussed rats release of stress hormones (e.g., corticosterone and adrenocorticotropic hormone) in response to restraint positions were more pronounced and longer lasting.   Similarly, Reger et al. (2011) demonstrated that starting two days after a concussion, injured rats showed a significant increase in context and cued fear expression. Anxiety behaviors following concussion were also found in a study by Elder et al. (2012) who demonstrated that even in the absence of psychological stressors, rats developed high anxiety traits after repeated mTBI. These authors rendered unconscious both the mTBI and non-TBI groups before inducing an injury, such that the TBI group would not have any awareness of the injury. Despite this lack of conscious experience of a TBI, only the concussed rats exhibited increased anxiety behaviors. Meyer et al. (2012) findings supported this research and found that rats with mild head injuries had anxiety like behaviours that resolved quickly. Recently Heldt et al. (2014) devised a new form of experimentally induced concussion using pressure blasts. These authors demonstrated that rats with mild blast injuries exhibited anxiety like behaviors, including a heightened acoustic startle response that persisted over several weeks. This research strongly suggests that in rats, concussion causes a time-limited state of hyperarousal.

To date, there have been no studies that have objectively measured indicators of arousal in the acute phase of TBI in humans. Instead, exisiting literature is limited to populations in the

chronic phase of moderate to severe TBI. In contrast to the animal studies, this line of research suggests TBI leads to a state of hypoarousal.

Hypoarousal

Several researchers have examined autonomic functioning in TBI patients. These studies primarily include participants with moderate to severe TBI that occurred years prior to the study. In the only study examining psychophysiology of mild head injury, Baker and Good (2013) demonstrated that college students who self-reported a history of mild head injury were physiologically under-aroused, as measured by electrodermal activity, when compared to their non-head injured controls. However, this study compared athletes to non-athletes and there was significant variability in the time since injury. In a sample of participants with moderate-severe TBI, Rushby et al. (2013b) reported diminished overall skin conductance levels in a sample of TBI patients, as compared to controls, in the absence of any stimuli (η^2=.115). De Sousa et al. (2010; 2012) measured skin conductance to pleasant, neutral, and unpleasant movie clips. These authors found that participants with moderate-severe TBI were significantly less aroused by unpleasant and pleasant clips than were controls, but they produced similar levels of skin conductance response to neutral stimuli. Sánchez-Navarro et al. (2005) used similar pleasant, unpleasant, and neutral images and also reported lower overall skin conductance response in the moderate-severe TBI group as compared to the control group.

The data is somewhat more complicated when facial expressions were used for stimuli. Research using normal controls shows that physiological arousal to angry faces is greater than that to happy faces or neutral faces (Jönsson & Sonnby-Borgström, 2003; Yartz & Hawk, 2002). Theory suggests that angry faces activate the sympathetic nervous system to generate greater

skin conductance, heart rate, and breathing rate in an effort to prepare the system to engage in "fight-or-flight" in response to the threatening stimulus (Kreibig, 2010). Researchers have found that in comparison to controls, persons in the chronic phase of a moderate-severe TBI do not produce the normal sympathetic response to angry faces (De Sousa et al., 2012; Fisher et al., 2014; Hopkins et al., 2002). Hopkins et al. (2002) found that participants with moderate-severe TBI produced similar skin conductance response to happy facial expressions as controls, however, they produce less skin conductance response to angry facial expressions (e.g., $\eta^2=.22$). De Sousa et al. (2011) reported similar results; they found TBI participants had a significantly lower skin conductance response to angry facial expressions than the control group. Fisher et al. (2014) measured both autonomic response and conducted structural MRIs of chronic moderate-severe TBI patients in their study. These authors demonstrated that participants with TBI had significant volume loss in the amygdala and insula, and they showed reduced skin conductance in response to all facial expressions (happy, angry, and neutral). These authors suggested that the TBI caused neuronal cell death in the amygdala and insula, which, once damaged, was not able to produce the appropriate autonomic arousal.

Several studies measured startle response in moderate-severe TBI participants to assess if TBI impacted affective modulation. Startle response, when viewing emotional stimuli, is modulated by the affective state of the subject, which can be experimentally induced by viewing an emotionally valenced images. When a subject is primed by a negatively valenced image (e.g., angry face) the startle response is enhanced compared to those elicited while viewing neutral stimuli. The opposite occurs when viewing pleasant pictures (e.g., happy face); the startle is attenuated. Startle response is commonly measured via skin conductance, heart rate, and blink reflex (Kreibig et al., 2010). The dominant theory of the startle reflex suggests that negatively

valenced images (e.g., angry faces) activate the sympathetic nervous system ("fight-or-flight"), while pleasant pictures (e.g., happy faces) is associated with activation of the parasympathetic system ("rest-and-digest"). As such, startle response is closely tied to the arousal level elicited by the stimuli, and authors have pointed to role of the amygdala in affective modulation and the startle reflex (Lang, Bradley, & Cuthbert, 1997; Angrilli et al., 1996; Buchanan, Tranel & Adolphs, 2004). Williams & Wood (2012), for instance, used pleasant, unpleasant, and neutral images coupled with an acoustic noise to study if participants with TBI would demonstrate the same modulation effect as controls. As expected, the control groups showed a potentiated eye-blink startle response (measured using electromyography) to unpleasant images and an attenuated response to pleasant images. In comparison, participants with TBI had a significantly lower startle response to unpleasant images but had a similar response to pleasant images. This finding suggested that TBI impaired the ability to appropriately increase arousal in the context of fear inducing stimuli, a role often attributed to the amygdala. Further supporting the hypothesis that chronic severe TBI results in disruption of the amygdala is that patients with amygdala damage have depressed startle responses (Angrilli et al., 1996; Buchanan et al., 2004) similar to patients with severe TBI (Sánchez-Navarro et al., 2005; Saunders et al., 2006).

This research provides evidence that in the long-term, moderate-severe TBI may result in hypoarousal, possibly due to permanent brain damage in regions of the brain associated with generating and moderating arousal, such as the amygdala. If these brain regions are damaged in the long-term, it is plausible that they are undergoing some important physiological changes in the acute phase of the inury as well. Such physiological changes may be detected through irregular autonomic arousal but this has yet to be studied in humans.

Several studies showed that participants with chronic moderate-severe TBI had different patterns of habituation to stimuli indicating that the brain systems involved in producing autonomic arousal in response to emotional stimuli were disrupted (de Sousa et al., 2012; McDonald et al. 2011a, 2011b; Rushby et al., 2013). McDonald et al. (2011a, 2011b) showed controls had greater heart rate deceleration in response to repetition of happy faces as compared to angry faces. De Sousa et al., (2012) reported that when exposed to film clips, normal control participants showed skin conductance decreased over time when viewing unpleasant clips, and increasing when viewing pleasant clips; no change over time was evident among the TBI participants. The pattern seen when exposed to repetitions of the same film in Rushby et al. (2013b) was somewhat different. These authors found that skin conductance increased over repeated exposures regardless of emotional valence while heart rate decreased. The later study suggested that repeated exposure increased skin conductance possibly due to the effort involved in sustaining attention across repetitions, but did not offer an explanation as to why heart rate would decrease.

Summary

Together this line of research supports the theory that mTBI causes new onset emotional symptoms (Elder et al., 2012; Heldt et al., 2014; Mainwarring et al., 2010). These emotional symptoms may be associated with hyper- or hypo-arousal. Existing literature suggests a short-term period characterized by hyperarousal (Elder et al., 2012; Heldt et al., 2014), which may lead to chronic hypoarousal (De Sousa et al., 2012; Rushby et al., 2013b; William & Wood, 2012; Yoshino et al., 1991), at least in moderate-severe injury cases where neuronal cell death is likely (Fisher et al., 2013a). This study will address a gap in the literature by examining the

relationship between mTBI, autonomic arousal, and emotional symptoms in the acute phase of the injury. The study will include college athletes as their injuries are less likely to be paired with a psychological trauma (Rabinowitz et al., 2012). Findings from this study may help explain why TBI is associated with emotional symptoms, and in turn inform the development of treatments.

Objectives

This study consisted of two phases. The first phase piloted an experimental protocol designed to demonstrate affective modulation of a startle response with a non-concussed sample of undergraduate participants. Findings from the pilot study were used to improve the protocol, which will then be used on a sample of concussed athletes and their controls.

The following paper reports on Phase I of the study: piloting the protocol. I developed a protocol that we predicted would elicit differential emotional arousal based on the valence of the stimuli presented. We included an acoustic startle to elicit greater physiological responses that are modulated by the affective state of the individual as demonstrated in previous research (Saunders et al., 2006; Williams and Wood, 2012). In this study we use three types of facial expressions; happy, neutral, and angry. Based on previous research we developed three hypotheses: 1) angry faces will elicit significantly greater physiological responses; 2) participants will demonstrate habituation to stimuli over time, and 3) emotional symptoms will be associated with greater autonomic arousal.

CHAPTER II

METHODS

Participants

Thirty-two undergraduate students enrolled in the psychology participant pool were recruited. Exclusion criteria consisted of no history of neurodevelopmental disease (e.g., autism, intellectual disability disorder, brain tumour). Of the thirty-two recruited, only 25 had complete data. The 7 participants removed from the analysis did not have usable physiological data due to technical difficulties with the recording equipment. Among the 25 included, 19 (76%) were female and had an average age was 22.08 (5.94) years. They had completed 1.88 (1.56) years of post-secondary education and the majority (68%) were also working part-time or full-time.

Stimulus Materials

Task 1 Stimuli

The experimental stimuli consisted of facial expressions taken from the NimStim stimulus set, which is a set of 43 male and female actors of various ethnicities, each making eight different expressions. The total number of pictures in the stimulus set is 672. The faces have shown to have good validity based on level of between-subject agreement between the intended expression and participants' ratings, with Cohen's kappa values ranging from .60 to .94 for each of the eight expressions (Tottenham et al., 2009). Photographic images for this study included neutral faces, happy faces, and angry faces. The images were edited to be grayscale as is consistent in previous studies (de Sousa et al., 2010; Saunders et al., 2006). The original coloured images had very good validity: happy ($\kappa = .94$), angry ($\kappa = .92$), and neutral ($\kappa = .87$). The

images have very good reliability according to the within-subject proportion of agreement

values: happy (.98), sad (.77), and neutral (.94; Tottenham et al., 2009).

Task 2 Stimuli

An acoustic startle stimulus of white noise was used to test affective modulation. This

acoustic noise was paired with images of happy, neutral, and angry facial expressions from the

NimStim database described above. The acoustic noise consisted of a 50-ms, 100-dB burst of

white noise with an instantaneous rise/fall time. This is the standard volume of noise used in

startle response research (Williams and Wood, 2012).

Measures

Intake Interview

Participants were asked about their demographics, education, medical and psychiatric

history, and life-long involvement in athletics. The questionnaire was administered by the

researcher conducting the interview (see Appendix A). In the event that a participant endorsed

high levels of psychiatric symptoms, they were provided with a resource sheet for campus and

community mental health resources (see Appendix B).

Brain Injury Symptom Questionnaire-Adapted (BISQ; Gordon et al., 2000)

Following the intake interview, the researcher administered the Brain Injury Symptom

Questionnaire - Adapted (BSIQ-A) to elicit any information regarding history of traumatic brain

injuries and/or head injuries. This form prompts responders to consider lifetime incidents of

head injuries that may or may not have resulted in a TBI in order to develop a more nuanced exposure index for potential damage to neural tissue (see Appendix C).

Depression Anxiety and Stress Scale (DASS; Lovibond & Lovibond, 1995)

The DASS is a 42-item scale developed to measure depression, anxiety and stress in the general population. It has excellent psychometric properties including high internal consistency on the three subscales (depression, α=.91; anxiety, α=.84; stress, α=.90). The measure has been used in research in both clinical and non-clinical populations (Antony, Beliing, Cox, Enns, & Swimson, 1998; Henry & Crawford, 2005), as well as TBI populations (de Sousa et al., 2012; Wong, Dahm & Ponsford, 2013). Responses are rated on a 4-point Likert scale ranging from 0 (did not apply to me at all) to 3 (applied to me very much, or most of the time). Factor analysis supports a 3-factor solution with between factor correlations ranging from .28 to .53 (Antony et al., 1998) and is consistent in TBI populations (Wong et al., 2013). In a clinical sample correlations of the DASS with the Beck Depression Inventory, Beck Anxiety Inventory, and the STAI-T indicate good validity (BDI and DASS-D, r=.77; BAI and DASS-A, r=.84; STAI-T and DASS-S, r=.59) with the exception that the STAI-T loaded better on the DASS-D scale (r=.65). Patients with major depressive disorder scored highest on the Depression and Stress Subscales, whereas individuals in the panic disorder group scored highest on the Anxiety subscale (Antony et al., 1998). Scores are calculated by summing the scores for relevant items on each subscale; each score is then categorized as either normal, mild, moderate, severe, or extremely severe (Appendix D). If a participant scores in the moderate range or above on any of the three subscales, they will be provided with the campus and community mental health resource sheet (See Appendix B).

Physiological Measures

Barry et al. (2005) argue that skin conductance, which increases with subtle changes in perspiration, is the most robust objective measure of state changes associated with arousal (Barry et al., 2005; see Fisher et al., 2014). Participants were connected to the PowerLab data acquisition (DAQ) system to measure heart rate/pulse and skin conductance. The PowerLab device is an AD Instruments product used in a variety of research applications including human physiology. To measure cardiovascular output, 2 bipolar limb leads were placed next to the collarbones and one lead was placed on the torso to ground the signal. As the measures were sensitive to movement, participants were instructed to keep their hand still for the duration of the baseline period and tasks. To measure skin conductance, bipolar finger electrodes were connected to the palmar surfaces of the pointer and ring finger of the participant's non-dominant hand. All transducers were attached firmly with Velcro straps. The data acquired through the PowerLab DAQ was analyzed by the accompanying LabChart software; cardiovascular output was analyzed as beats per minute and skin conductance was analyzed in microsiemens (μs). After completion of the tasks, all electrodes were removed.

Procedure

Participants were recruited using the University of Windsor psychology participant pool. The recruitment advertisement requested that participants refrain from eating or drinking caffeine four hours prior to attending their appointment. When participants arrived for their appointment they reviewed the REB consent form and the researchers demonstrated to the participants how the physiological equipment would be attached. Once participants signed the

consent form they were asked to use the washroom and to wash their hands. They were then seated while the researcher administered the intake questionnaire and the BISQ. The participants were then asked to sit in front of the computer that was used for all tasks and complete an electronic version of the DASS. Once finished, the electrocardiogram bipolar electrodes were placed on the participants' torso and the finger electrodes were placed on the index and fourth finger of the non-dominant hand. The participants were asked to sit quietly in front of the blank computer screen for 10 minutes. After 10 minutes the researcher initiated the computer based protocol using DirectRT. Participants were tested on a computer with a fifteen-inch monitor and a resolution of 1024 X 768 pixels.

Task 1 Procedure

Participants were given the following instructions, "Please sit quietly while some images are displayed on the screen. You do not need to respond, just sit as still as possible." Fifteen facial expressions (5 angry, 5 happy, 5 neutral) were shown in random order. Each face was displayed for 6 seconds followed by a fixation cross on a blank screen for 12 seconds. This interval duration is necessary as both skin conductance and heart rate changes take time to dissipate (McDonald et al., 2011). No response was required.

Task 2 Procedure

After a break of at least 30 seconds and up to five minutes, participants were given noise-cancelling headphones and were introduced to the loud acoustic noise twice for habituation. Participants were given the following instructions, "You will now hear a loud noise two times through the headphones. After the second time you will be prompted to begin the task. During

this task you will be presented with some more faces. This time some of the faces will be

coupled with the noise you just heard. You do not need to provide any response, just sit as still as

possible." Facial expressions consisted of 45 images divided into three blocks: each block

consisted of 15 facial expressions (5 happy, 5 neutral, 5 angry) presented in random order. Each

picture was presented for 8 seconds with inter-trial intervals of 12 seconds. A fixation cross was

displayed during inter-trial intervals. The acoustic startle probe was administered 4-6 seconds

($M = 5$ s) following the picture onset. To decrease predictability of the startle stimulus, 3 (1

happy, 1 neutral, 1 angry) of the 45 faces displayed were presented without a startle probe.   The

ratio of slides with and without startle probes was similar to a previous study (Williams & Wood,

2012).

Data reduction

Six-second epochs were selected following stimulus presentation for Task 1 and four-

second epochs following the acoustic probe in Task 2 (Williams and Wood, 2012). Skin

conductance level (SCL) was determined by calculating the average galvanic skin response

during the epoch. Skin conductance response (SCR) was calculated for each epoch by

subtracting the average galvanic skin response of 500ms prior to the stimulus presentation from

the maximum galvanic skin response following stimulus presentation as recommended by

Boucsein (1992) and Mathersul et al., (2013).   Average heart rate (HR) was calculated for each

epoch and presented in beats per minute (bpm).

Data Analysis

Qualitative review of the physiological data was conducted to identify any changes that were caused by movement. Movement was identified by comments inserted into the file by the researcher administrating the protocol. Average SCL, SCR, and HR for each emotion on each task was examined for normality. The data fell within normative range for skewness and kurtosis (|2| and |3|, respectively) with the exception of SCR Neutral for Task 1 (Kurtosis=7.01), SCR Happy for Task 2 (Kurtosis=14.20), SCR Neutral for Task 2 (Kurtosis=16.66), and SCR Angry for Task 2 (Kurtosis=14.01). The SCL and HR measurements had Shapiro-Wilk significance scores greater than 0.5 suggesting normality, but SCR Shapiro-Wilk scores were significant suggesting these were not normally distributed. Log transformation of the data did not improve normality and so data was kept as is. Z-scores were calculated and no outliers ($z>2.5$) were identified.

Repeated measure ANOVAs were conducted for each physiological measure separately (SCL, SCR and HR) for each task (Task 1 and Task 2). Task 1 repeated measure ANOVAs analyzed change over the trials (Trial 1-5) while task 2 analyzed change over blocks (Block 1-3); each block included 5 different facial expressions of an emotion. Sphericity was examined for each analysis using Mauchly's W test of sphericity. When data violated sphericity Greenhouse-Geisser statistic was used. In order to examine whether response patterns habituated to repetitions, simple planned contrasts examined linear and quadratic trends over time (Rushby et al., 2013). Post-hoc simple contrasts were used to examine if participants produced greater arousal to angry facial expression (angry vs. neutral, angry vs. happy).

Mental health rating scores (DASS total, DASS depression, DASS anxiety, and DASS stress) were analyzed for normality. The data fell within normative range for skewness and

20

kurtosis (|2| and |3|, respectively), but Shapiro-Wilk statistics were significant (<.05) suggesting

the data was not normally distributed. To identify outliers z-scores were calculated. One outlier

was identified (z-score >3.00) and was removed but this did not improve normality of the data so

the outlier was retained and the data was log transformed to improve normality. Following the

transformation only the DASS anxiety and DASS depression scores had significant Shapiro-

Wilk values but qualitative review of the data histograms showed that the normality was

improved. Bivariate associations between autonomic and symptom scores were examined using

Pearson's correlation where appropriate (SCL and HR) and Spearman's rank order for data that

was not normally distributed (SCR).

CHAPTER III

RESULTS

Sample Characteristics

The majority (76%) reported at least one past head injury with an average of 2.64 (3.53) head injuries per participant. Of those who had experienced head injuries, 3 had a resulting loss of consciousness less than 30 minutes. Participants were questioned regarding their activities in the 24 hours prior to participation in the study. On average participants had slept 7.28 (2.02) hours the night before testing, 8 (32%) had exercised, 1 (4%) had consumed alcohol, 14 (56%) had consumed caffeine, and 4 (16%) had eaten within four hours of participation.

Possible confounding variables

The role of possible confounding variables to physiological responses was examined. Variables of interest included demographic variables, history of prior head injuries, number of hours of sleep, intake of food prior to participation and intake of caffeine prior to testing. No significant differences existed across gender, age, history of head injuries, number of hours of sleep the previous night, and consumption of caffeine to SCR, SCL, or HR. Consumption of food in the last four hours prior to testing was significantly associated with greater SCL in Task 2 (see Table 1).

Table 1
Associations between Sample Characteristics and Physiological Data

		Gender (t)	Age (r)	Number of Head Injuries (r)	Number of hours of sleep (r)	Consumption of caffeine (t)	Consumption of food (t)
Task 1 Average SCL (µs)	Happy	1.417	-0.276	-0.039	0.042	-0.516	-0.516

	Neutral	-0.067	-0.176	0.297	-0.091	-0.321	-0.424
	Angry	0.871	-0.022	0.22	-0.299	0.627	0.627
Average SCR (μs)	Happy Neutral	1.12	-0.069	0.1	-0.059	-1.902	-1.103
	Angry	1.147	0.129	0.102	-0.227	-1.722	-1.265
ECG RR (bpm)	Happy Neutral	1.235	-0.052	-0.01	0.293	-2.04	-1.095
	Angry	-0.135	0.174	0.037	-0.215	-1.752	-1.752
Task 2							
Average SCL (μs)	Happy	-1.192	-0.105	-0.016	-0.098	-1.021	**-1.004***
	Neutral	-0.589	0.006	-0.176	-0.036	-1.312	**-1.254***
	Angry	-0.463	0.016	-0.11	-0.085	-0.1314	**-1.162***
Average SCR (μs)	Happy Neutral	0.836	0.139	0.286	-0.044	-0.922	-2.363
	Angry	0.876	0.132	0.104	0.355	-0.945	-2.419
ECG RR (bpm)	Happy Neutral	0.849	0.148	1.78	-0.249	-1.039	-2.422
	Angry	-1.49	0.215	-0.17	-0.231	-1.125	-1.516

*p<.05

Skin Conductance Level (SCL)

Task 1. A two-way within group repeated measures ANOVA was conducted to examine differences in SCL across the three picture valences (happy, neutral, angry), which were each presented five times. There was no main effect of emotion but there was a main effect for trial number ($F(1.17, 26.91)=6.46$, $p=.014$, $\eta^2=.219$).

Contrast analyses found a linear trend among trial number ($F(1, 23)=7.08$, $p=.014$, $\eta^2=.235$) such that SCL decreased with each trial. There was a near significant interaction between responses to angry and happy faces such that physiological responses to happy faces decreased more across trials than responses to angry faces ($F(1, 23)=3.73$, $p=.066$, $\eta^2=.140$).

Figure 1. SCL across trials on Task 1.

Task 2. A two-way within group repeated measures ANOVA was conducted to examine differences in SCL across the three picture valences (happy, neutral, angry) across the 3 blocks. Results revealed no main effect for emotion or block and no significant interaction. Trend analysis revealed a quadratic trend across blocks $F(1, 24)=5.385$, $p=.029$, $\eta^2=.183$ such that SCLs were smaller in block 2 and greater in block 3.
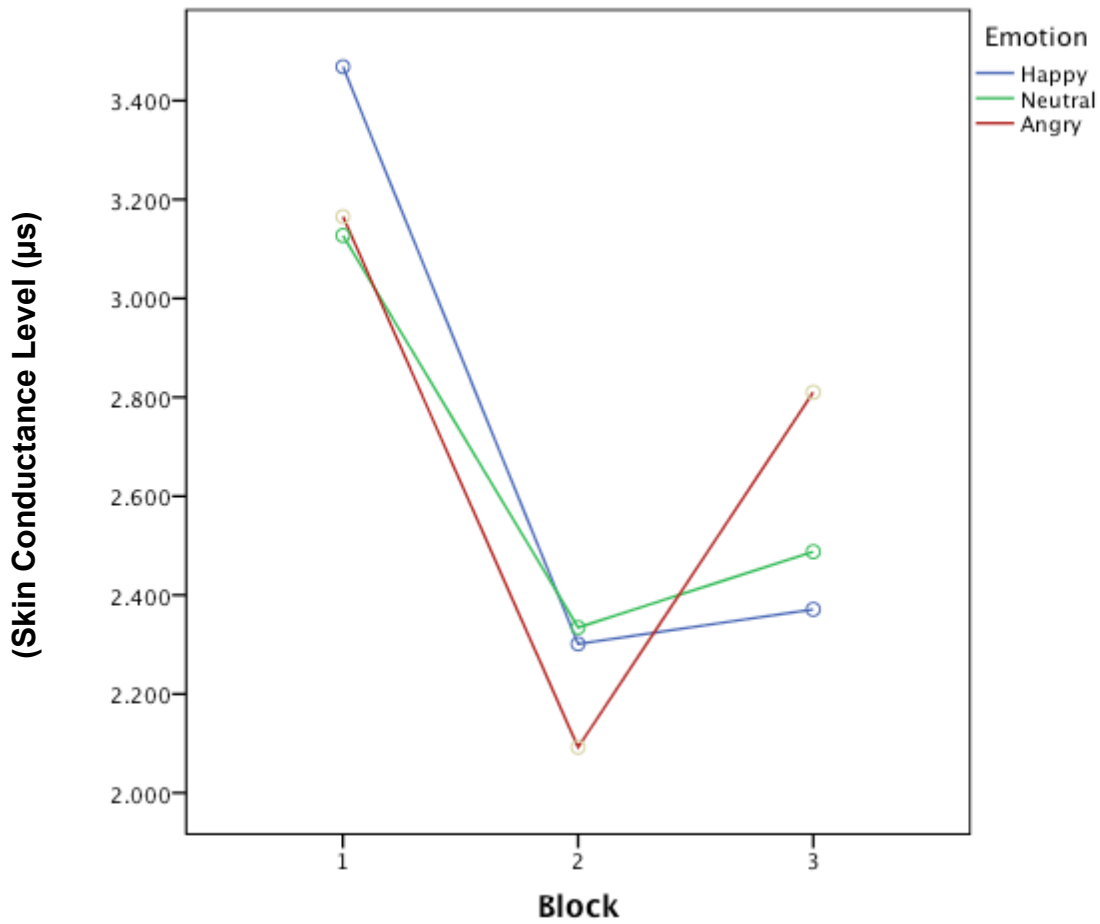
Figure 2. SCL across trials on Task 2.

Skin Conductance Responses (SCR)

Task 1. A two-way within group repeated measures ANOVA was conducted to examine differences in SCR across the three picture valences (happy, neutral, angry), which were each presented five times. There was no main effect of emotion or trial number and no significant interaction.
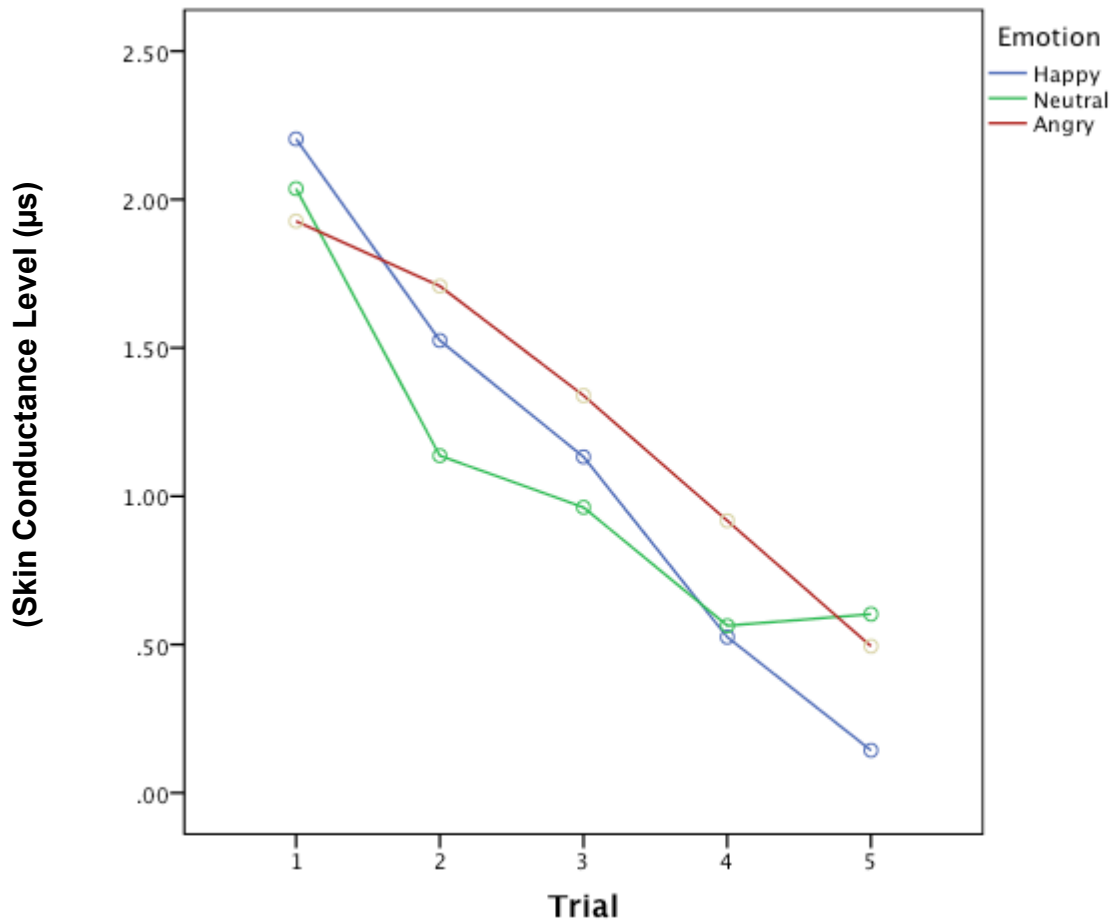
Figure 3. SCR across trials on Task 1.

Task 2. A two-way within group repeated measures ANOVA was conducted to examine differences in SCR across the three picture valences (happy, neutral, angry) across the 3 blocks. Results revealed no main effect for emotion but did find a main effect for block $F(2, 48)=3.99$, $p=.025, \eta^2=.142$.

Additional contrasts revealed no significant difference in SCR between angry or happy faces and angry and neutral faces. Trend analysis identified a significant linear trend across blocks $F(1,24)=6.97, p=.014, \eta^2=.233$ such that responses decreased across blocks. There was also a significant difference between the interaction changes across blocks among angry faces as

compared to changes across block to happy faces ($F(1,24)=4.69$, $p=.040$, $\eta^2=.163$). Given that there appeared to be habituation over time across blocks a post-hoc analysis was conducted to compare responses to angry faces with responses to happy faces in block 1. This revealed that responses to angry faces in block 1 were significantly greater than responses to happy faces $t(24)=-2.45$, $p=.022$.
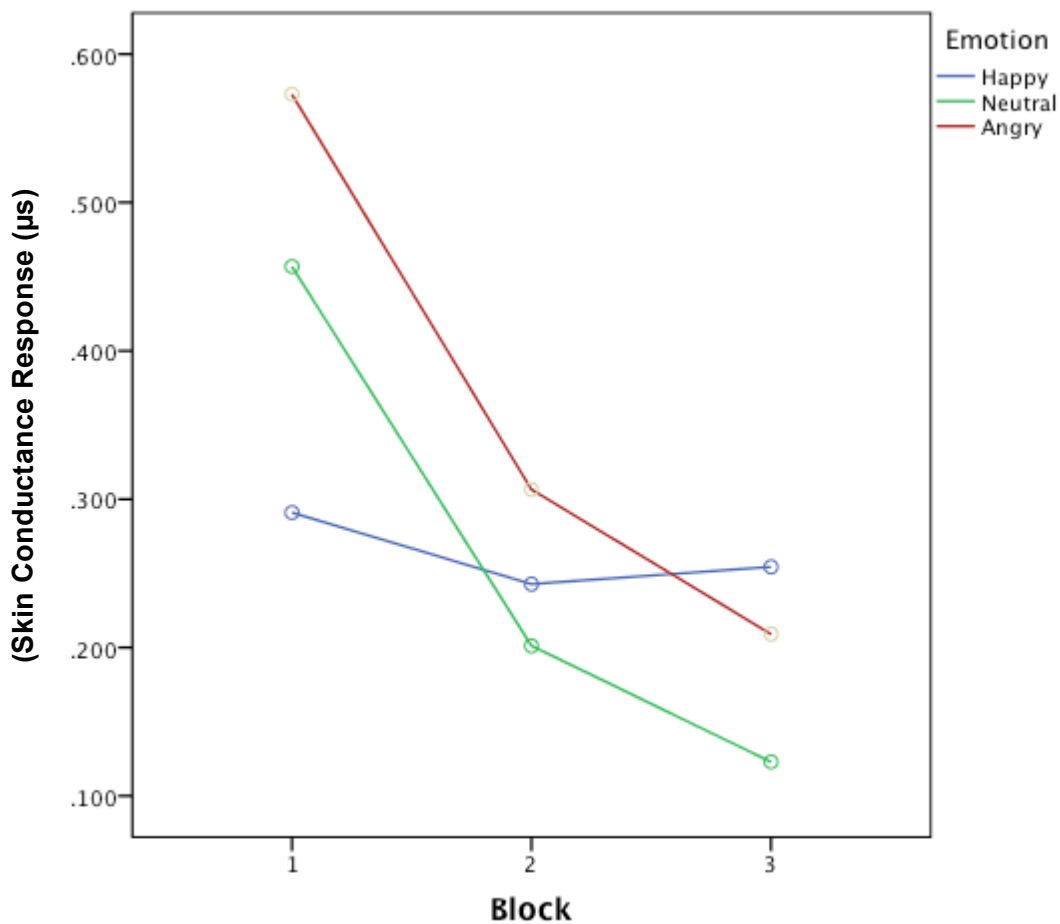
Figure 4. SCR across blocks on Task 2.

Heart Rate (bpm)

Task 1. A two-way within group repeated measures ANOVA was conducted to examine differences in HR across the three picture valences (happy, neutral, angry), which were each presented five times. There was no main effect for emotion but there was a main effect for trial number ($F(4,92)=4.193$, $p=.004$, $\eta^2=.154$). There was no significant interaction.

Trend analysis revealed a significant linear trend for trial with HR increasing across trials ($F(1,23)=5.88$, $p=.024$, $\eta^2=.204$).
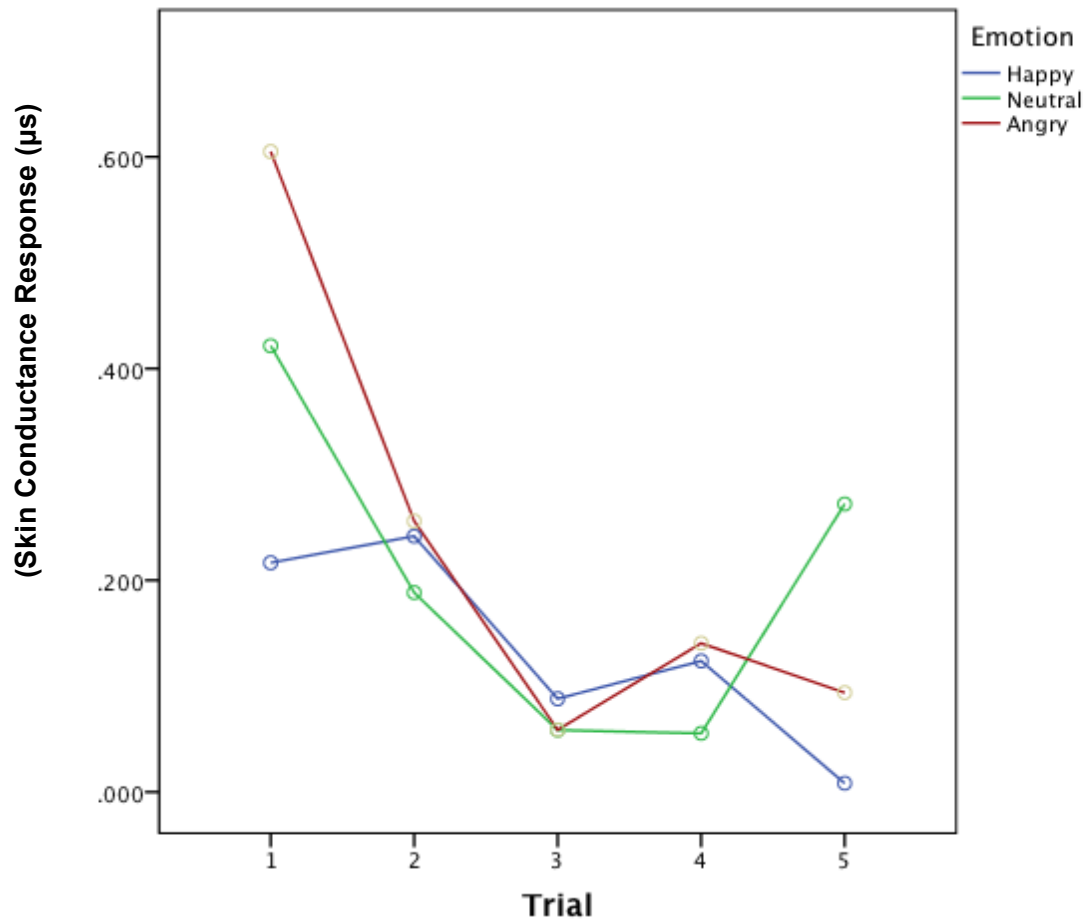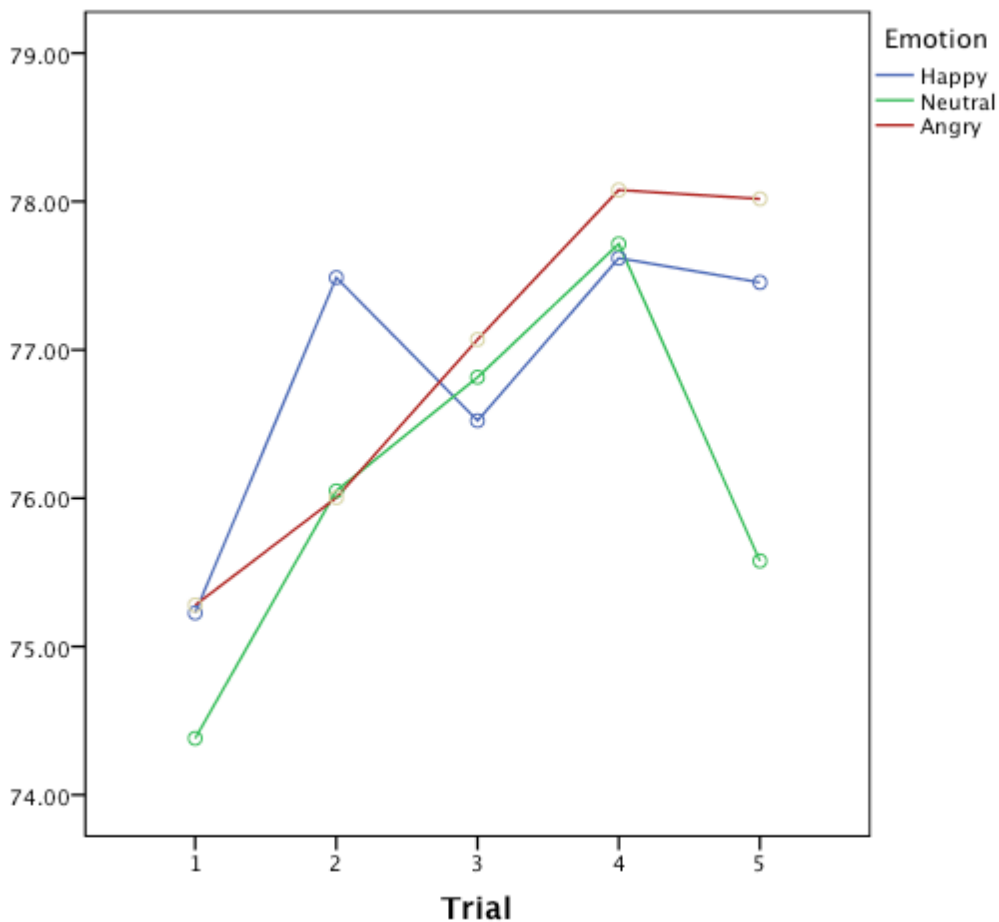

Figure 5. HR across trials on Task 1.

Task 2. A two-way within group repeated measures ANOVA was conducted to examine differences in HR across the three picture valences (happy, neutral, angry) across three blocks. Results revealed a trend towards significance for main effect for emotion ($F(2,46)=2.73$, $p=.076$, $\eta^2=.106$) and a significant main effect for block ($F(1.36,31.34)=6.55$, $p=.009$, $\eta^2=.222$) such that heart rate increased across blocks. There was no significant interaction between emotion and block.

Additional contrasts revealed no significant difference in HR between angry and neutral faces or angry and happy faces. Trend analysis revealed that HR increased linearly across blocks ($F(1, 23)=6.41$, $p=.019$, $\eta^2=.218$). There were no significant differences among the interaction contrasts.
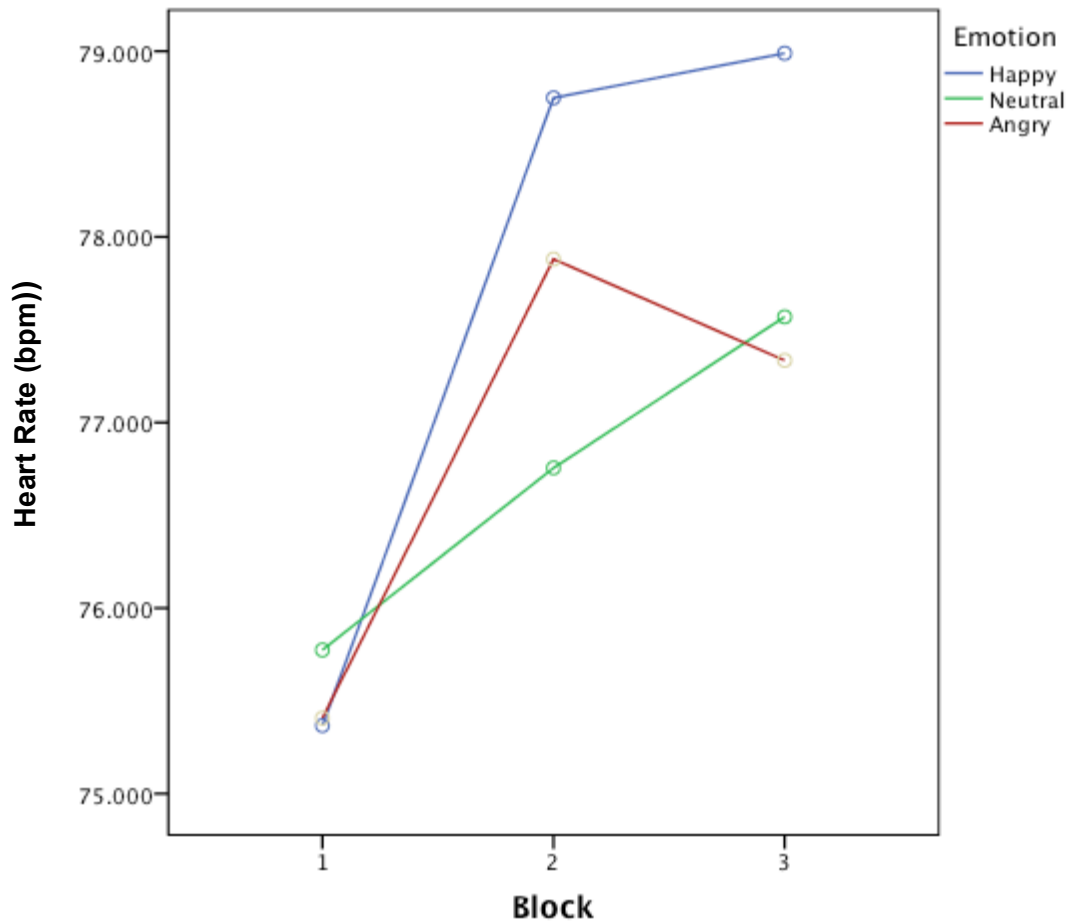
Figure 6. HR across blocks on Task 2.

Symptom Correlates

The average scores on measures of mental health are reported in Table 2 for men and women. Women scored on average higher than men on all measures but because data were not normally distributed and the male group was less than half that of the female group, significance tests were not used. Symptom scores were not significantly associated with number of past head injuries. The relationship between symptom scores and physiological responses to emotional stimuli were explored (see Table 3). Because this analysis was exploratory and we did not control for multiple comparisons, significant correlations were further explored using scatter

plots. Heart rates across all stimuli valences were significantly correlated with DASS anxiety, DASS stress (with the exception of neutral valence), and DASS total scores. Scatter plots confirmed that clients with more anxiety and stress symptoms had higher HRs across emotional valences during the first task.

Table 2
Average scores on measures of mental health

	Gender	N	Mean	Std. Deviation	Std. Error Mean
DASS Total	Male	6	7.667	6.088	2.486
	Female	18	23.556	26.046	6.139
DASS Depression	Male	6	0.833	1.329	0.543
	Female	18	7.389	10.308	2.430
DASS Anxiety	Male	6	2.500	0.548	0.224
	Female	18	5.722	6.702	1.580
DASS Stress	Male	6	4.333	4.803	1.961
	Female	18	10.444	10.601	2.499

Table 3
Correlations between physiological measures and measures of mental health

			DASS Total	DASS Depression	DASS Anxiety	DASS Stress
Task 1						
Average SCL (µs)	Happy	Pearson	0.141	0.101	0.116	0.196
	Neutral	Pearson	0.2	0.147	0.174	0.258
	Angry	Pearson	0.123	0.088	0.12	0.173
Average SCR (µs)	Happy	Spearman's	-0.096	-0.109	0.013	-0.189
	Neutral	Spearman's	.001	-0.135	0.125	-0.057
	Angry	Spearman's	0.109	0.153	0.298	0.078
Average HR (bpm)	Happy	Pearson	**.461***	0.257	**.459***	**.422***
	Neutral	Pearson	**.432***	0.27	**.415***	0.394
	Angry	Pearson	**.460***	0.268	**.435***	**.422***
Task 2						
Average SCL (µs)	Happy	Pearson	0.235	0.154	0.227	0.286
	Neutral	Pearson	0.205	0.128	0.196	0.253
	Angry	Pearson	0.21	0.129	0.203	0.257
Average SCR (µs)	Happy	Spearman's	-0.081	-0.320	0.278	-0.032
	Neutral	Spearman's	-0.161	-0.244	0.042	-0.113

	Angry	Spearman's	-0.051	-0.160	-0.048	-0.027
Average HR (bpm)	Happy	Pearson	0.234	0.136	0.276	0.149
	Neutral	Pearson	0.197	0.079	0.233	0.119
	Angry	Pearson	0.206	0.113	0.246	0.135

*p<.05

CHAPTER IV

DISCUSSION

The present study was a pilot study aimed at determining if repeated exposure to emotionally valenced facial expressions could evoke patterns of arousal in a control group consistent with previous research (de Sousa et al., 2012; Fisher et al., 2013; Jönsson et al., 2003; Williams & Wood, 2012; Yartz & Hawk, 2002). In addition, we examined the relationship between sample characteristics, self-reported mental health symptoms and physiological arousal. Findings from this pilot study will be used for future research aimed at testing the differences in psychophysiological arousal in concussed athletes as compared to controls.

Limited differential physiological responses to repeated presentation of emotionally valenced facial expressions was found as seen in previous research (Jönsson et al., 2003; Rushby et al., 2013; Vrana et al., 2004; Williams & Wood, 2012). Measurement of HR on Task 2 detected a near significant main effect for emotion. Closer inspection of this finding indicated that while HR was similar for all emotions at block 1 of the task, HR showed a greater increase in response to happy faces than neutral or angry faces. This finding is consistent with previous research that found happiness is characterized by increased cardiac activity due to vagal withdrawal (Kreibig , 2010). Heart rate in response to angry faces was similar to or slightly less than responses to neutral faces. This finding contradicted our hypothesis that anger would elicit greater arousal, however, prior research has identified similar patterns of response when participants are presented with expressions of anger, rather than responding to harassing material (e.g., movie clips that elicit anger). Specifically, picture viewing of facial expressions of anger decelerates HR instead of accelerates (Johnosson et al., 2003; Vrana et al., 2004). These researchers proposed that the emotional response to anger expressions is related to fear, rather

than anger itself. This finding may indicate a sympathetic-parasympathetic cardiac deactivation in response to threatening stimuli that is associated with a withdrawal-oriented response rather than the approach-oriented response associated with anger. Future research will need to account for the differences between anger and fear responses.

There was no main effect for emotion detected for either task by either SCR or SCL. Linear contrasts did reveal a significant difference in the interaction of response to angry and happy faces across blocks as measured by SCR in Task 2. As hypothesized, an acoustic startle in the context of an angry face elicited a greater SCR than that in the context of happy faces in block 1, however, the difference in responses decreased across blocks 2 and 3, suggesting that habituation may have muted the effect. This finding provides evidence of affective modulation of the startle response (Williams & Wood, 2012).

We found that at different time points angry faces elicited greater skin conductance and caused the heart to decelerate. Several studies have found decreased HR and increased SCR in response to picture presentation of threatening material (e.g., pictures of faces, snakes, spiders). Kreibig, 2010 hypothesized that such material may involve a later stage along the "fear continuum," which is characterized by immobilization rather than an active coping response. Such immobilization-response could include a sharp rise in electrodermal activity while also increasing vagal restriction of heart rate.

Patterns of habituation varied across measures of physiological arousal. On both tasks, heart rate increased over time across all emotions. That heart rate would increase with repeated exposure to stimuli would suggest increased withdrawal of vagus nerve activation or increased sympathetic activity and/or release of catecholamines (Clifton, Zieger, & Grossman, 1981). As discussed above, HR deceleration is observed in response to fear inducing simuli. It is possible

that as participants habituated to the acoustic startle deceleration of the heart lifted and so an increase in HR was observed.

There was a main effect for trial number for SCL on Task 1. SCL decreased over time suggesting habituation to the stimuli. This finding is inconsistent with de Sousa et al., (2012) and Rushby et al., (2013) who found that among controls, SCL remained constant or increased over multiple exposures to emotional stimuli. These authors suggested SCL may have increased as a result of the effort needed in sustaining attention. It is possible that our study did not show this effect because our stimuli were less complex and therefore required less effort to attend to; we presented facial expressions while the above authors used film clips. Habituation was also evident with SCL between block 1 and block 2 during Task 2 but SCL rose again during Task 3. Although data was screened for large movements, we believe that the increase in block 3 may be due to more subtle movements towards the end of the experiment, which was observed by researchers. These movements were characterized by changing position in chair and foot tapping and may not have been detected by the screening process. Despite removing large movements, SCL is known to be sensitive to movement and will increase with subtle changes of position. This finding suggests that participants may benefit from a shorter protocol.

Habituation was evident among the SCR in Task 2 but not Task 1. While the overall average SCL was decreasing in Task 1, the change in SCL before and after the stimulus presentation (SCR) was negligible and so did not change much over time. On Task 2, when the responses to the acoustic noise were greater, habituation over time was evident. This finding may suggest that SCR is useful to demonstrate habituation only when there is sufficient reactivity to the stimuli while SCL is a useful measure to track arousal levels over time.

There was little evidence that patterns of habituation differed according to emotion. During Task 1, the decrease in SCL over time to angry facial expressions was significantly less than that to happy faces. This may be indication that the response to angry facial expressions is more resilient to habituation over multiple exposures than that to happy faces; however, this finding should be interpreted with caution as it was only near significant and was not present in Task 2.

Finally, we explored the relationship between SCL, SCR, HR, and symptom measures. Findings suggest that higher scores on the DASS total, DASS anxiety, and DASS stress scales were moderately correlated with increased HR during Task 1. This finding did not hold true for Task 2. It is possible that participants with higher stress and anxiety exhibited greater sympathetic activity towards the beginning of their participation due to participation anxiety, which normalized over time. If the hypothesis that fear causes HR to decelerate holds true, the fear of the acoustic startle may have suppressed HR among anxious individuals. That HR is associated with DASS anxiety and stress scores suggests that it may be a useful measure for identifying participants with elevated levels of stress and anxiety and it should be retained in the amended protocol.

We found no association between gender, age, history of head injuries, number of hours of sleep the previous night, and consumption of caffeine and physiological responsivity. This is consistent with previous research (Williams & Wood, 2012). We did find that consumption of food in the last four hours prior to testing was significantly associated with greater SCL in Task 2. This finding emphasizes the importance of controlling for food consumption in future research. That number of head injuries were not associated with arousal or self-reported stress is inconsistent with a previous study by Baker et al. (2013) who suggested history of head injuries

was associated with muted arousal in response to stress. However, due to significant differences in protocols, differences in findings should be interpreted with caution; Baker et al. (2013) administered a stress induction protocol while this study involved affective modulation of startle response.

There were important limitations to this study. First, the sample size was small given that the range of effect sizes for physiological arousal between emotionally valenced stimuli are small. That said, prior research has demonstrated significant findings with sample sizes that ranged between 9-30 participants (Vrana et al., 2004; Jönsson et al., 203) and so it is unlikely that sample size is the only limiting factor. The length of the study may have masked some findings as participants habituated to the stimuli and they engaged in more movement as the experiment progressed. This movement may have affected results in unexpected ways, as demonstrated by the increase in SCL in block 3 of Task 2. Finally, there were significant problems with equipment that resulted in the necessity of throwing out the data of 7 participants. Future research will need to implement safe guards to protect against any more failures to record data properly. Finally, due to the exploratory nature of this pilot study we did not control for multiple comparisons and future research will be needed to confirm findings.

Future Research

Research has indicated several regions in the brain susceptible to injury due to TBI are also involved in the monitoring and modulating sympathetic and parasympathetic tone such as the orbital surface of the frontal lobe (Yamour et al., 1980) and the insular cortex (Groswasser, Reider-Groswasser, Soroker & Machtey, 1987). Research from animal studies (Elder et al., 2012; Heldt et al., 2014) and chronic TBI (De Sousa et al., 2012; Williams & Wood, 2012)

suggest that these injuries cause changes in physiological arousal, which may contribute to disrupted emotional functioning. Understanding how concussion affects physiological arousal may inform us why some mTBI patients experience changes in emotional functioning following their injury.

The first goal of phase 2 of this study is to demonstrate that concussed athletes report more psychological symptoms as compared to their baseline symptom scores and compared to controls. Although higher incidence of depression and anxiety has been reported following a mild TBI, these studies are often confounded by the psychological trauma during which the injury occurred. To date, few studies have documented psychological distress following SRC, which is unique in that the injury is not typically coupled with a psychological trauma. The few studies that have explored psychological symptoms following SRC demonstrate a week-long emotional state that is characterized by depression and anxiety (Mainwaring et al., 2004; 2010; Yang et al., 2015). Using the Depression, Anxiety, and Stress Scale (DASS), scores of concussed athletes will be compared to: 1) scores of controls matched on age and gender, and 2) their own baseline scores taken as part of standard protocol for all athletes undergoing pre-season baseline cognitive testing. Based on previous studies of self-report symptoms following SRC, we predict that athletes will report elevated levels of depression, stress, and anxiety (Mainwaring et al., 2004; 2010; Yang et al., 2015).

The second goal is to demonstrate that concussed athletes will have significantly different psychophysiological arousal than controls as is suggested by animal research (Elder et al., 2012; Heldt et al., 2014; Regers et al., 2011). Findings from the pilot study found that controls did not produce clear differential patterns to facial expressions as expected and there was no main effect for emotion across physiological measures. The only evidence of affective modulation was that

SCR was significantly greater to angry faces in the first block of the 2nd task than that to happy faces. Due to this lack of robust findings we will consider simplifying the task to measure pure startle response, as done in animal studies, and not startle response modulated by affective state, as is common in human TBI research. Exaggerated startle is reputed to be one of the cardinal symptoms of posttraumatic stress disorder (Morgan et al., 1995) and is evidenced in experimentally induced fear (Kreibig, 2010) as well as persons with clinical anxiety disorders (Grillon, 2002). Differences in raw baseline measures as well as response to an acoustic startle probe will be compared across concussed and control participants.

As HR was significantly associated with higher self-reported anxiety and stress, the relationship between self-reported symptoms and physiological arousal at baseline and in response to a startle will be compared across groups. We predict that higher DASS anxiety and stress scores will be associated with greater HR at baseline and greater SCR to the startle probe.

The final goal of the study will be to draw a direct link between the concussive injury and emotional functioning. We are interested in examining whether a concussion leads to dysregulated arousal (hyper- or hypoarousal) and that this in turn results in greater self-reported symptoms. Although causation cannot be demonstrated, we can examine if injury characteristics will predict physiological arousal and/or self-reported symptoms. Pre-injury, injury, and post-injury factors will be entered into a regression model in order to determine which may be associated with emotional outcomes. Outcome variables will consist of self-reported symptoms, baseline arousal, and startle response. Predictors will include: number of prior concussions, time (in hours) since injury, and change in ImPACT cognitive efficiency index.

Identifying elevated psychological symptoms and dysregulated psychophysiological responses following concussion is an initial step in determining if concussions lead to neurophysiological changes that disrupt emotional functioning.

REFERENCES

Al Harbi, A. (2013). Prevalence of Psychological Disorders among Patients with Mild vs Severe Traumatic Brain Injury: The Devil in the Details (P05. 117).*Neurology*, *80*(Meeting Abstracts 1), P05-117.

Angrilli, A., Mauri, A., Palomba, D., Flor, H., Birbaumer, N., Sartori, G., & di Paola, F. (1996). Startle reflex and emotion modulation impairment after a right amygdala lesion. *Brain*, *119*(6), 1991-2004.

Antony, M. M., Bieling, P. J., Cox, B. J., Enns, M. W., & Swinson, R. P. (1998). Psychometric properties of the 42-item and 21-item versions of the Depression Anxiety Stress Scales in clinical groups and a community sample.*Psychological assessment*, *10*(2), 176.

Baker, J. M., & Good, D. E. (2014). Physiological emotional under-arousal in individuals with mild head injury. *Brain Injury*, *28*(1), 51–65.

Barry, R. J., Clarke, A. R., McCarthy, R., Selikowitz, M., & Rushby, J. A. (2005). Arousal and activation in a continuous performance task. *Journal of psychophysiology*, *19*(2), 91-99.

Bazarian, J. J., Zhong, J., Blyth, B., Zhu, T., Kavcic, V., & Peterson, D. (2007). Diffusion tensor imaging detects clinically important axonal damage after mild traumatic brain injury: a pilot study. *Journal of neurotrauma*, *24*(9), 1447-1459.

Belanger, H. G., & Vanderploeg, R. D. (2005). The neuropsychological impact of sports-related concussion: a meta-analysis. *Journal of the International Neuropsychological Society: JINS*, *11*(4), 345–357.

Boucsein, W. (1992). Electrodermal Indices of Emotion and Stress, chapter 3.*Electrodermal Activity*, 369-391.

Buchanan, T. W., Tranel, D., & Adolphs, R. (2004). Anteromedial temporal lobe damage blocks

startle modulation by fear and disgust. *Behavioral Neuroscience*, *118*(2), 429–437.

Carroll, L. J., Cassidy, J. D., Cancelliere, C., Côté, P., Hincapié, C. A., Kristman, V. L., …

Hartvigsen, J. (2014). Systematic Review of the Prognosis After Mild Traumatic Brain

Injury in Adults: Cognitive, Psychiatric, and Mortality Outcomes: Results of the

International Collaboration on Mild Traumatic Brain Injury Prognosis. *Archives of

Physical Medicine and Rehabilitation*, *95*(3, Supplement), S152–S173.

Clifton, G. L., Ziegler, M. G., & Grossman, R. G. (1981). Circulating catecholamines and

sympathetic activity after head injury. *Neurosurgery*, *8*(1), 10-14.

Covassin, T., Crutcher, B., Bleecker, A., Heiden, E. O., Dailey, A., & Yang, J. (2014). Postinjury

Anxiety and Social Support Among Collegiate Athletes: A Comparison Between

Orthopaedic Injuries and Concussions. *Journal of Athletic Training*. doi:10.4085/1062-

6059-49.2.03

De Sousa, A., McDonald, S., Rushby, J., Li, S., Dimoska, A., & James, C. (2010).

Understanding deficits in empathy after traumatic brain injury: The role of affective

responsivity. *Cortex; a Journal Devoted to the Study of the Nervous System and

Behavior*, *47*(5), 526–535.

De Sousa, A., McDonald, S., & Rushby, J. (2012). Changes in emotional empathy, affective

responsivity, and behavior following severe traumatic brain injury. *Journal of Clinical

and Experimental Neuropsychology*, *34*(6), 606–623.

Elder, G. A., Dorr, N. P., De Gasperi, R., Gama Sosa, M. A., Shaughness, M. C., Maudlin-

Jeronimo, E., … Ahlers, S. T. (2012). Blast exposure induces post-traumatic stress

disorder-related traits in a rat model of mild traumatic brain injury. *Journal of Neurotrauma*, *29*(16), 2564–2575.

Fann, J. R., Burington, B., Leonetti, A., Jaffe, K., Katon, W. J., & Thompson, R. S. (2004). Psychiatric Illness Following Traumatic Brain Injury in an Adult HealthMaintenance Organization Population. *Archives of General Psychiatry*,*61*(1), 53-61.

Fisher, A. C., Rushby, J. A., McDonald, S., & Parks, N. (2014). Neurophysiological correlates of dysregulated emotional arousal in severe traumatic brain injury. *Clinical Neurophysiology*. doi: 10.1016/j.clinph.2014.05.033

Giza, C. C., & Hovda, D. A. (2001). The Neurometabolic Cascade of Concussion. *Journal of Athletic Training*, *36*(3), 228–235.

Giza, C. C., & Hovda, D. A. (2014). The new neurometabolic cascade of concussion. *Neurosurgery*, *75 Suppl 4*, S24–33.

Griesbach, G. S., Hovda, D. A., Tio, D. L., & Taylor, A. N. (2011). Heightening of the stress response during the first weeks after a mild traumatic brain injury. *Neuroscience*, *178*, 147–158.

Grillon, C. (2002). Startle reactivity and anxiety disorders: aversive conditioning, context, and neurobiology. *Biological psychiatry*, *52*(10), 958-975.

Groswasser Z, Reider-Groswasser I, Soroker S, Machtey Y. Magnetic resonance imaging in head injured patients with normal late computed tomography. Surgeral Neurology 1987;27:331–7.

Al Harbi, A. (2013). Prevalence of Psychological Disorders among Patients with Mild vs Severe Traumatic Brain Injury: The Devil in the Details (P05.117). *Neurology*, *80*(Meeting Abstracts 1), P05.117.

Heilman, K. M., Schwartz, H. D., & Watson, R. T. (1978). Hypoarousal in patients with the

    neglect syndrome and emotional indifference. *Neurology*,*28*(3), 229-229.

Heldt, S. A., Elberger, A. J., Deng, Y., Guley, N. H., Del Mar, N., Rogers, J., … Reiner, A.

    (2014). A novel closed-head model of mild traumatic brain injury caused by primary

    overpressure blast to the cranium produces sustained emotional deficits in mice.

    Frontiers in Neurology, *5*, 2.

Henry, J., & Crawford, J. R. (2005). A meta-analytic review of verbal fluency deficits in

    depression. *Journal of Clinical and Experimental Neuropsychology*, *27*(1), 78–101.

Hess, U., & Blairy, S. (2001). Facial mimicry and emotional contagion to dynamic emotional

    facial expressions and their influence on decoding accuracy.*International journal of

    psychophysiology*, *40*(2), 129-141.

Hopkins, M. J., Dywan, J., & Segalowitz, S. J. (2002). Altered electrodermal response to facial

    expression after closed head injury. *Brain Injury: [BI]*, *16*(3), 245–257.

James, W. (1884). What is an emotion?. *Mind*, (34), 188-205.

Joiner, T. E., Jr, Steer, R. A., Beck, A. T., Schmidt, N. B., Rudd, M. D., & Catanzaro, S. J.

    (1999). Physiological hyperarousal: construct validity of a central aspect of the tripartite

    model of depression and anxiety. *Journal of Abnormal Psychology*, *108*(2), 290–298.

Jönsson, P., & Sonnby-Borgström, M. (2003). The effects of pictures of emotional faces on tonic

    and phasic autonomic cardiac control in women and men. *Biological Psychology*, *62*(2),

    157-173.

King, N. S. (1996). Emotional, neuropsychological, and organic factors: their use in the

    prediction of persisting postconcussion symptoms after moderate and mild head injuries.

    Journal of Neurology, Neurosurgery, and Psychiatry, *61*(1), 75–81.

King, N. (2008). PTSD and traumatic brain injury: Folklore and fact? *Brain Injury: [BI]*.

Retrieved from

http://informahealthcare.com.ezproxy.uwindsor.ca/doi/abs/10.1080/02699050701829696

Konrad, C., Geburek, A. J., Rist, F., Blumenroth, H., Fischer, B., Husstedt, I., … Lohmann, H.

(2011). Long-term cognitive and emotional consequences of mild traumatic brain injury.

Psychological Medicine, *41*(6), 1197–1211.

Lange, K. (1887). *Ueber Gemüthsbewegungen: eine psychophysiologische Studie*. Thomas.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1997). Motivated attention: Affect, activation,

and action. *Attention and orienting: Sensory and motivational processes*, 97-135.

Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states:

Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression

and Anxiety Inventories. *Behaviour research and therapy*,*33*(3), 335-343.

Mainwaring, L., & Bischop, S. (2004). Emotional Reaction of Varsity Athletes to Sport-Related

Concussion. *Journal of Sport & Exercise Psychology, 26*, 119-135.

Mainwaring, L. M., Hutchison, M., Bisschop, S. M., Comper, P., & Richards, D. W. (2010).

Emotional response to sport concussion compared to ACL injury. *Brain Injury*, *24*(4),

589–597.

Mathersul, D., McDonald, S., & Rushby, J. A. (2013). Psychophysiological correlates of social

judgement in high-functioning adults with autism spectrum disorder. *International

Journal of Psychophysiology: Official Journal of the International Organization of

Psychophysiology*, *87*(1), 88–94.

Mild Traumatic Brain Injury Committee, A. C. o. R. M., Head Injury Interdisciplinary Special

    Interest Group. Definition of mild traumatic brain injury. Journal of Head Trauma

    Rehabilitation 1993;8(3):86-87.

McDonald, S., Rushby, J., Li, S., de Sousa, A., Dimoska, A., James, C., … Togher, L. (2011).

    The influence of attention and arousal on emotion perception in adults with severe

    traumatic brain injury. *International Journal of Psychophysiology*, *82*(1), 124–131.

Meares, S., Shores, E. A., Taylor, A. J., Batchelor, J., Bryant, R. A., Baguley, I. J., …

    Marosszeky, J. E. (2011). The prospective course of postconcussion syndrome: the role

    of mild traumatic brain injury. *Neuropsychology*, *25*(4), 454–465.

Meyer, D. L., Davies, D. R., Barr, J. L., Manzerra, P., & Forster, G. L. (2012). Mild traumatic

    brain injury in the rat alters neuronal number in the limbic system and increases

    conditioned fear and anxiety-like behaviors. *Experimental Neurology*, *235*(2), 574–587.

Miles, L., Grossman, R. I., Johnson, G., Babb, J. S., Diller, L., & Inglese, M. (2008). Short-term

    DTI predictors of cognitive dysfunction in mild traumatic brain injury. *Brain*

    Injury, *22*(2), 115-122.

Mooney, G., & Speed, J. (2001). The association between mild traumatic brain injury and

    psychiatric conditions. *Brain Injury*, *15*(10), 865–877.

Moore, E. L., Terryberry-Spohr, L., & Hope, D. A. (2006). Mild traumatic brain injury and

    anxiety sequelae: a review of the literature. *Brain Injury: [BI]*, *20*(2), 117–132.

Rabinowitz, A. R., Li, X., & Levin, H. S. (2014). Sport and nonsport etiologies of mild traumatic

    brain injury: similarities and differences. *Annual Review of Psychology*, *65*, 301–331.

Reger, M. L., Poulos, A. M., Buen, F., Giza, C. C., Hovda, D. A., & Fanselow, M. S. (2011). Concussive brain injury enhances fear learning and excitatory processes in the amygdala. *Biological Psychiatry*, *71*(4), 335–343.

Riechers, R., Ruff, R., Ruff, S., Wang, X. F., & Piero, T. (2012). Neurological Deficits (ND) and Post Traumatic Stress Disorder (PTSD) Are Related to the Number of Episodes of Mild Traumatic Brain Injury (mTBI) in US Combat Veterans (S49. 003). *Neurology*, *78*(Meeting Abstracts 1), S49-003.

Rushby, J. A., Fisher, A. C., McDonald, S., Murphy, A., & Finnigan, S. (2013a). Autonomic and neural correlates of dysregulated arousal in severe traumatic brain injury. *International Journal of Psychophysiology*, *89*(3), 460–465.

Rushby, J. A., McDonald, S., Randall, R., de Sousa, A., Trimmer, E., & Fisher, A. (2013b). Impaired emotional contagion following severe traumatic brain injury. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology*, *89*(3), 466–474.

Ryan, L. M., & Warden, D. L. (2003). Post concussion syndrome. *International Review of Psychiatry* , *15*(4), 310–316.

Sánchez-Navarro, J. P., Martinez-Selva, J. M., & Román, F. (2005). Emotional response in patients with frontal brain damage: effects of affective valence and information content. *Behavioral neuroscience*, *119*(1)*,* 87.

Saunders, J. C., McDonald, S., & Richardson, R. (2006). Loss of emotional experience after traumatic brain injury: findings with the startle probe procedure. *Neuropsychology*, *20*(2), 224–231.

Schoenhuber, R., & Gentilini, M. (1988). Anxiety and depression after mild head injury: a case control study. *Journal of Neurology, Neurosurgery, and Psychiatry*, *51*(5), 722–724.

Sharp, D. J., & Ham, T. E. (2011). Investigating white matter injury after mild traumatic brain injury. *Current Opinion in Neurology*, *24*(6), 558–563.

Shenton, M. E., Hamoda, H. M., Schneiderman, J. S., Bouix, S., Pasternak, O., Rathi, Y., … Zafonte, R. (2012). A review of magnetic resonance imaging and diffusion tensor imaging findings in mild traumatic brain injury. *Brain Imaging and Behavior*, *6*(2), 137–192.

Suchy, Y. (2011). *Clinical neuropsychology of emotion*. Guilford Press.

Tottenham, N., Tanaka, J. W., Leon, A. C., McCarry, T., Nurse, M., Hare, T. A., … Nelson, C. (2009). The NimStim set of facial expressions: judgments from untrained research participants. *Psychiatry Research*, *168*(3), 242–249.

Vasterling, J. J., Verfaellie, M., & Sullivan, K. D. (2009). Mild traumatic brain injury and posttraumatic stress disorder in returning veterans: perspectives from cognitive neuroscience. *Clinical Psychology Review*, *29*(8), 674–684.

Vrana, S. R., & Gross, D. (2004). Reactions to facial expressions: effects of social context and speech anxiety on responses to neutral, anger, and joy expressions. *Biological Psychology*, *66*(1), 63-78.

Williams, C., & Wood, R. L. (2012). Affective modulation of the startle reflex following traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, *34*(9), 948–961.

Wong, D., Dahm, J., & Ponsford, J. (2013). Factor structure of the Depression Anxiety Stress Scales in individuals with traumatic brain injury. *Brain injury*,*27*(12), 1377-1382.

Yang, J., Peek-Asa, C., Covassin, T., & Torner, J. C. (2015). Post-concussion symptoms of depression and anxiety in division I collegiate athletes. *Developmental Neuropsychology*, *40*(1), 18–23.

Yartz, A. R., & Hawk, L. W. (2002). Addressing the specificity of affective startle modulation: Fear versus disgust. *Biological psychology*, *59*(1), 55-68.

Yoshino, A., Hovda, D. A., Kawamata, T., Katayama, Y., & Becker, D. P. (1991). Dynamic changes in local cerebral glucose utilization following cerebral concussion in rats: evidence of a hyper- and subsequent hypometabolic state. *Brain Research*, *561*(1), 106–119.

APPENDICES

Appendix A

Intake Interview Form

U WINDSOR: SPORTS CONCUSSION CLINIC

INTAKE EVALUATION FORM

First name:

Surname:

Interviewer:

Date of Interview: M M D D Y Y Y Y

Participant Group:
- Not participating in reseach
- Referred to concussion clinic
- Referred from undergrduate participant pool

Participant ID:

Section I: Demographics

Date of Birth:

M	M	D	D	Y	Y	Y	Y

Dominant Hand:
☐ Right
☐ Left
☐ Ambidextrous

Primary (native) language: _____

Secondary language(s): _____

Country of birth: _____

Prompt: What is your gender?
Gender:
☐ Male
☐ Female
☐ Other

Marital status: (check one)
☐ Single, never married
☐ In relationship, not married
☐ Married
☐ Separated
☐ Divorced
☐ Widowed
☐ Other

Ethnicity: _____

Living Situation:
☐ In parents home
☐ Alone
☐ With roommates
☐ In residence
☐ With partner
☐ Other

Section II: Education

How many years of post-secondary education have you **completed**?

☐ 1   ☐ 4
☐ 2   ☐ 5
☐ 3   ☐ 6+

What is your major? _____

Are you a full time or part-time student?

☐ Full time
☐ Part-time

Having you ever been diagnosed with any of the following:

☐ ADHD
☐ Learning disability

When were you diagnosed and by whom?

Have you ever received extra assistance in school (e.g., IEP, modified curriculum, allowed extra time)?

☐ Yes   Describe:
☐ No

Aside from being a student, are you currently working?

☐ Yes, full time   Describe:
☐ Yes, part time
☐ No

*** SKIP SECTION V for CONTROLS ***

Section III: Current Concussion

When was your most recent injury (date)?

| M | M | D | D | Y | Y | Y | Y |

How did it happen?

What did you experience directly after the injury? (approx the following 24 hours)

	Yes	No
Loss of consciousness	☐	☐
Feeling confused	☐	☐
Loss of memory for things that happened directly after the injury	☐	☐
Loss of memory for things that happened directly before the injury	☐	☐

Describe:

Did you receive medical treatment?   ☐ Yes
                                      ☐ No

Describe:

What have you been doing since the injury?

Physical activities   Describe:

☐ Yes ☐ No

Cognitive activities   Describe:

☐ Yes ☐ No

Change in concentration   Describe:

☐ Yes ☐ No

Stressors   Describe:

☐ Yes ☐ No

Rate your level of distress on a scale from 0-9: ☐

Change in appetite   Describe:

☐ Yes ☐ No

Change in sleep   Describe:

☐ Yes ☐ No

Section IV: Medical/Psychiatric

Current medical problems and services received(ing) for them (e.g., medications, treatment):
(other than recent head injury/concussion)

Condition 1:

Received(ing) treatment: ☐ Yes
                           ☐ No

Describe treatment

Medication(s)

Condition 2:

Received(ing) treatment: ☐ Yes
                           ☐ No

Describe treatment

Medication(s)

Condition 3:

Describe treatment

Received(ing) treatment: ☐ Yes
☐ No

Medication(s)

Are you currently taking any medications including over the counter medications? Please list:

Past medical problems and services received for them (e.g., medications, treatment):

Prompt: Are you currently experiencing any pain (in the past 7 days)?

☐ Yes
☐ No

Rating of most severe location:
0=none   4=moderate
2=slight   7=significant
9=severe

Prompt: Have you ever been diagnosed or sought treatment for any of the following:

			Desc:
Depression:	☐ Never	☐ Past ☐ Present	
Anxiety:	☐ Never	☐ Past ☐ Present	
Bi-polar Disorder:	☐ Never	☐ Past ☐ Present	
Psychosis: (e.g., Schizophrenia)	☐ Never	☐ Past ☐ Present	
Thoughts of hurting yourself:	☐ Never	☐ Past ☐ Present	

If Yes: *Do you have a specific plan? Access to means?*
Do you intend on following through?
Do you have support systems?
Do you feel hopeless about the future?

AUGUST 2014   5

54

Prompt: Do you currently use any of the following substances:

Substance use: Alcohol   ☐ Never   ☐ Present
                Drugs   ☐ Never   ☐ Present
                Tobacco   ☐ Never   ☐ Present

Prompt: if yes to substance use; identify how much and how often.

Section V: Athletic History

What sports team(s) are you currently a member of or have been in the past 10 years:

# of Years

☐	Football	☐	Collegiate Varsity	☐	High-school	☐	Recreational	
☐	Soccer	☐	Collegiate Varsity	☐	High-school	☐	Recreational	
☐	Baseball/softball	☐	Collegiate Varsity	☐	High-school	☐	Recreational	
☐	Hockey	☐	Collegiate Varsity	☐	High-school	☐	Recreational	
☐	Basketball	☐	Collegiate Varsity	☐	High-school	☐	Recreational	
☐	Tennis	☐	Collegiate Varsity	☐	High-school	☐	Recreational	
☐	Other	☐	Collegiate Varsity	☐	High-school	☐	Recreational	

Ask only if participant is not on a varsity team:

What kind of exercise do you do and how often?

Section VI: Physiological Status

How many hours of sleep did you get last night?

In the last 24 hours: *Since the concussion:*

Did you do any physical activity?   ☐ Yes   ☐ Yes   Describe:
                                    ☐ No   ☐ No

Did you take any medications?   ☐ Yes   ☐ Yes   Describe (name and dosage)
(prescription, over-the counter, illegal)   ☐ No   ☐ No

Did you drink any alcohol? ☐ Yes ☐ Yes Describe (how many and what kind)
☐ No ☐ No

Did you drink any caffeinted beverages? ☐ Yes ☐ Yes Describe (how many and what kind)
(coffee, tea, energy drink, soda/pop) ☐ No ☐ No

Prompt: As part of this research study we may want to contact you regarding some follow-up information. Would it be alright contact you at a later date (in 3-6 months) regarding more research?

☐ No   If Yes: Telephone: H
☐ Yes
                                  M

Email:

Section VII: Brain Injury Symptom Questionnaire (BISQ)
Administer BISQ

****TO COMPLETE FOLLOWING INTERVIEW****

CURRENT MENTAL STATUS

Appearance:	☐ well-groomed	☐ disheveled	☐ bizarre	☐ inappropriate
Attitude:	☐ cooperative	☐ guarded	☐ suspicious	☐ uncooperative ☐ belligerent
Motor Activity:	☐ calm	☐ hyperactive	☐ agitated	☐ tremors/tics ☐ muscle spasm
Affect:	☐ appropriate	☐ labile	☐ expansive	☐ constricted/flat ☐ blunted
Mood:	☐ calm	☐ depressed	☐ anxious	☐ angry ☐ euphoric
Speech:	☐ normal	☐ delayed ☐ pressured	☐ soft ☐ loud	☐ slurred ☐ excessive ☐ incoherent ☐ perserverating
Thot. Process:	☐ normal	☐ circumstantial	☐ loose assoc.	☐ tangential ☐ flight of ideas
Thot. Content:	☐ normal	hallucinations: ☐ auditory	☐ visual	☐ olfactory
		delusions: ☐ persecutory	☐ being contrld	☐ thought insert. ☐ bizarre
Self-Perception:	☐ normal	☐ depersonalizn.	☐ derealization	
Orientation:	☐ normal	☐ disoriented:	☐ time	☐ place ☐ person
Memory:	☐ intact	☐ impaired:	☐ immediate	☐ recent amnes: amnesia ☐ remote
Judgement:	☐ intact	☐ impaired:	☐ minimal	☐ moderate ☐ severe
Insight:	☐ intact	☐ impaired	☐ minimal	☐ moderate ☐ severe
Risk Factors:	☐ none reported	☐ suicidality ☐ alcohol abuse	☐ homicidality ☐ substance abuse	☐ medical risk ☐ victim of abuse ☐ impulse control ☐ perpetrator of abuse

AUGUST 2014   7

56

Appendix B
Campus and Community Mental Health Resources

University of Windsor Campus Resources

Student Counselling Centre
Room 293 CAW Centre
519-253-3000 ext. 4616
Monday - Friday 8:30am – 4:30pm

Windsor-Essex County Community Resources

Canadian Mental Health Association
Windsor-Essex County Branch
1400 Windsor Avenue
(519)-255-7440

Teen Health Centre
1585 Ouellette Avenue
(519)-253-8481

Windsor Regional Hospital
Ouellette Campus Emergency Department

Community Crisis Centre
Walk-In Service
Windsor Regional Hospital - Ouellette Campus Jeanne Mance Building, 1st Floor
Monday - Friday; regular business hours

Community Crisis Centre
24-Hour Crisis Telephone Line
(519) 973-4435

APPENDIX C
Brain Injury Symptom Questionnaire

Brain Injury Screening Questionnaire – Adapted for SCC		Version 7.8.2012							
Column A		**Column B**							
For each event listed, record the number of times you have ever experienced the following situations..		Ever lose consciousness?		Ever dazed and confused?		Posttraumatic Amnesia?		Retrograde Amnesia?	
	How many times?	How many times?	Longest period?	How many times?	Longest period?	How many times?	Longest period?	How many times?	Longest period?
1. In a motor vehicle crash (e.g., car, motorcycle)?									
2. A pedestrian hit by a vehicle?									
3. Running into or being hit by an object (e.g., equipment)?									
4. Falling, fainting or slipping?									
5. During a drug or alcohol blackout?									
6. While biking?									
7. While roller balding/skateboarding?									
8. While horseback riding?									
9. While skiing/snowboarding?									
10. In sports (football, baseball basketball)?									
11. While on the playground?									

12. While diving into water?								
13. Being assaulted or mugged?								
14. Being physically abused?								
15. Other?								

Ever been hospitalized or seen in the emergency room for any of the following?	How many times?	Ever lose consciousness?		Ever dazed and confused?		Posttraumatic Amnesia?		Retrograde Amnesia?	
		How many times?	Longest period?	How many times?	Longest period?	How many times?	Longest period?	How many times?	Longest period?
1. Concussion?									
2. Fracture to the head, neck or face?									
3. Seizures?									
4. High fever?									
5. Near drowning?									
6. Poisoning?									
7. Hit by lightening?									
8. Electrical power injury?									
9. Gun shot injury?									
10. Stroke/brain hemorrhage?									
11. Brain infection?									
12. Other injury?									

APPENDIX D
Depression, Anxiety and Stress Scale

DASS

Name: *Date:*

Please read each statement and circle a number 0, 1, 2 or 3 that indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

1	I found myself getting upset by quite trivial things	0	1	2	3
2	I was aware of dryness of my mouth	0	1	2	3
3	I couldn't seem to experience any positive feeling at all	0	1	2	3
4	I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
5	I just couldn't seem to get going	0	1	2	3
6	I tended to over-react to situations	0	1	2	3
7	I had a feeling of shakiness (eg, legs going to give way)	0	1	2	3
8	I found it difficult to relax	0	1	2	3
9	I found myself in situations that made me so anxious I was most relieved when they ended	0	1	2	3
10	I felt that I had nothing to look forward to	0	1	2	3
11	I found myself getting upset rather easily	0	1	2	3
12	I felt that I was using a lot of nervous energy	0	1	2	3
13	I felt sad and depressed	0	1	2	3
14	I found myself getting impatient when I was delayed in any way (eg, elevators, traffic lights, being kept waiting)	0	1	2	3
15	I had a feeling of faintness	0	1	2	3
16	I felt that I had lost interest in just about everything	0	1	2	3
17	I felt I wasn't worth much as a person	0	1	2	3

18	I felt that I was rather touchy	0	1	2	3
19	I perspired noticeably (eg, hands sweaty) in the absence of high temperatures or physical exertion	0	1	2	3
20	I felt scared without any good reason	0	1	2	3
21	I felt that life wasn't worthwhile	0	1	2	3

Reminder of rating scale:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

22	I found it hard to wind down	0	1	2	3
23	I had difficulty in swallowing	0	1	2	3
24	I couldn't seem to get any enjoyment out of the things I did	0	1	2	3
25	I was aware of the action of my heart in the absence of physical exertion (eg, sense of heart rate increase, heart missing a beat)	0	1	2	3
26	I felt down-hearted and blue	0	1	2	3
27	I found that I was very irritable	0	1	2	3
28	I felt I was close to panic	0	1	2	3
29	I found it hard to calm down after something upset me	0	1	2	3
30	I feared that I would be "thrown" by some trivial but unfamiliar task	0	1	2	3
31	I was unable to become enthusiastic about anything	0	1	2	3
32	I found it difficult to tolerate interruptions to what I was doing	0	1	2	3
33	I was in a state of nervous tension	0	1	2	3
34	I felt I was pretty worthless	0	1	2	3
35	I was intolerant of anything that kept me from getting on with what I was doing	0	1	2	3
36	I felt terrified	0	1	2	3
37	I could see nothing in the future to be hopeful about	0	1	2	3

38	I felt that life was meaningless	0	1	2	3
39	I found myself getting agitated	0	1	2	3
40	I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
41	I experienced trembling (eg, in the hands)	0	1	2	3
42	I found it difficult to work up the initiative to do things	0	1	2	3

VITA AUCTORIS

NAME: Eva Keatley

PLACE OF BIRTH: Brussels, Belgium

YEAR OF BIRTH: 1986

EDUCATION: Washington International School, Washington, D.C. 2004

                  University of California, San Diego, B.Sc., San Diego, CA, 2009