Understanding boredom proneness: Cognitive and affective correlates in healthy and traumatic brain injured individuals

by

Julia Isacescu

A thesis
presented to the University of Waterloo
in fulfillment of the
thesis requirement for the degree of
Doctor of Philosophy
in
Psychology

Waterloo, Ontario, Canada, 2017

Examining Committee Membership

The following served on the Examining Committee for this thesis. The decision of the Examining Committee is by majority vote.

External Examiner	Andra Smith, PhD Professor
Supervisor	James Danckert, PhD Professor
Internal Member	Evan Risko, PhD Professor
Internal-external Member	Sue Arai, PhD Adjunct Professor
Other Member(s)	Dan Smilek, PhD Professor

Author's Declaration

I hereby declare that I am the sole author of this thesis. This is a true copy of the thesis, including any final revisions, as accepted by my examiners.

I understand that my thesis may be made electronically available to the public.

Abstract

Boredom proneness has been associated with a raft of negative cognitive, affective, and behavioural consequences. Research has sought to better understand boredom proneness from both cognitive and affective perspectives; however, an explanation of its underlying processes is still lacking. First, this thesis explored cognitive and affective factors related to boredom to assess the degree to which levels of self-control could explain these relationships. Next, boredom proneness and self-control were assessed in individuals who had sustained varying degrees of traumatic brain injury (TBI) to explore whether a link exists between boredom proneness, self-control, and head injury severity. Finally, the neural underpinnings of state boredom were explored in healthy controls and a small sample of TBI patients, using functional magnetic resonance imaging. Study 1 showed that boredom proneness was associated with spontaneous mind-wandering, increased depression and hostility, with individual levels of self-control driving these relationships. Study 2 showed that boredom proneness increases as a function of head injury severity. Finally, Study 3 showed recruitment of large-scale default mode network regions (DMN) associated with boredom, with concurrent downregulation of the anterior insula, an area important for switching between default and executive networks. In the TBI patients, results were heterogeneous, with individual patients displaying opposing patterns of activation within and between conditions. Collectively, these results offer insights into the mechanisms of boredom proneness and self-control. Results are discussed in terms of a current definition of boredom which suggests the state represents disengagement from one's environment despite a motivation to engage – an experience that is negatively valenced, and likely represents failures of cognitive and affective self-regulation.

Keywords: boredom proneness, mind-wandering, depression, aggression, self-control, traumatic brain injury

Acknowledgments

I dedicate this dissertation to Gabi and Tudor Isacescu, my dear parents, without whom I would not be where I am today. Cu ajutorul vostru acest proiect a fost posibil. Vă iubesc şi vă mulţumesc din tot sufletul meu. To my supervisor, James, thank you for your vision of this project, for consistently challenging me, and for your encouragement along the way. To my reviewers, Dan and Evan, thank you for the patience and diligence you have put into revising this manuscript. To my lab manager, Nadine, thank you for the months we spent together recruiting participants. To my lab mates, Andriy, Alex, Elisabeth, and Jhotisha, thank you for being as supportive as you have been over the years, your encouragement on my roughest days will never be forgotten. Derick, thank you for your enduring patience and countless technical consultations, I will forever be grateful. And finally, I would to acknowledge every single one of the 2303 participants who took the time to get involved in this research: without you this work would not have come to fruition.

Table of Contents

List of Figures viii

List of Tables ix

List of Abbreviations x

Chapter 1: Introduction 1

Chapter 2: Cognitive and affective predictors of boredom proneness 4

- 2.1 Introduction 4
- 2.2 Method 8
    - Participants 8
    - Self-report measures 8
    - Data Analyses 10
- 2.3 Results 10
- 2.4 Discussion 16

Chapter 3: Exploring the relationship between boredom proneness and self-control in traumatic brain injury (TBI) 20

- 3.1 Introduction 20
- 3.2 Methods 23
    - Participants 23
    - Self-Report Measures 24
    - Data Analyses 24
- 3.3 Results 25
- 3.4 Discussion 32

Chapter 4: Exploring the neural networks associated with boredom in healthy controls and TBI participants. 36

- 4.1 Introduction 36
- 4.2 Method 40
    - Participants 40
    - Self-Report Measures 41
    - fMRI 41
- 4.3 Results 47
    - Self-report measures 47
    - Behavioural Data 51
    - fMRI 53

4.4 Discussion 67
Chapter 5: General Discussion 73
References 79
Appendices 97
  Appendix A: Participant Recruitment 97
  Appendix B: Questionnaires 99
  Appendix C: MRI Screening Form 107

List of Figures

Figure 2.1 Changes in correlation coefficients between boredom and all other measures when controlling for age alone (black bars) or age and self-control (white bars).. 15
Figure 4.1 Boredom, Interest, Resting State and Starry Night Scanning Conditions………. 43
Figure 4.2 Controls' and TBI participants' boredom and mind-wandering ratings between scanning conditions……………………………………………………………… 49
Figure 4.3 Controls: Network activation patterns observed in all four scanning conditions.. 55
Figure 4.4 Controls: Insular activation observed during each scanning condition…………. 56
Figure 4.5 TBIs: BOLD network activation patterns observed for each scanning condition…………………………………………………………………….…. 59
Figure 4.6 Posterior Cingulate Cortex (green) and Precuneus (orange)……………………. 60
Figure 4.7 Boredom: Individual TBI participants' network activation patterns……………. 61
Figure 4.8 Interest: Individual TBI participants' network activation patterns……………... 63
Figure 4.9 Resting State: Individual TBI participants' network activation patterns………... 65
Figure 4.10 Starry Night: TBI participants' network activation patterns. …………………... 66

List of Tables

Table 2.1 Means and standard deviations for all study variables, and within sample differences…….. 10
Table 2.2 Partial correlations for all variables, controlling for age…... 11
Table 2.3 Hierarchical regression analysis statistics for boredom proneness, controlling for gender and age…... 12
Table 2.4 Partial correlations for all variables controlling for age and self-control….. 14
Table 2.5 Partial correlations between boredom proneness and all measures controlling for age, and age and self-control…... 14
Table 3.1 Participant Demographics….. 23
Table 3.2 Descriptive statistics for healthy controls (HC) and Concussed groups….. 26
Table 3.3 Descriptive statistics for age-matched healthy controls (am-HC) and TBI groups.. 26
Table 3.4 Partial correlations: Cognitive and affective measures for Healthy Controls (upper diagonal) and Concussed individuals (lower diagonal)…… 27
Table 3.5 Partial correlations: Cognitive and affective measures for age-matched HC (upper diagonal) and TBI participants (lower diagonal)….. 27
Table 3.6 Head Injury as a predictor of Boredom Proneness…… 29
Table 3.7 Head Injury subgroups as predictors of Boredom Proneness…… 29
Table 3.8 Head Injury as a predictor of Self-Control….. 30
Table 3.9 Head Injury subgroups as predictors of Self-Control….. 30
Table 3.10 Cognitive and affective predictors of Boredom Proneness in individuals with Concussion…… 31
Table 3.11 Cognitive and affective predictors of Boredom Proneness in individuals with TBI. 31
Table 4.1 Control and TBI Behavioural Data…… 52
Table 4.2 Independent component (IC) clusters corresponding to the Boredom scan for Controls…… 56
Table 4.3 Independent component (IC) clusters corresponding to the Interest scan for Control Group…... 57
Table 4.4 Independent component (IC) clusters corresponding to the Resting state scan for Control Group…... 57
Table 4.5 Independent component (IC) clusters corresponding to the Starry Night scan for Control Group…... 58

List of Abbreviations

ADHD = Attention Deficit Hyperactivity Disorder
am-HC = Age-matched Healthy Controls
BA = Brodmann Area
BOLD = Blood Oxygenation-Level Dependent
BPAQ = Buss-Perry Aggression Questionnaire
BSCS = Brief Self-Control Scale
DASS = Depression, Anxiety and Stress Scale
DMN = Default Mode Network
fMRI = Functional Magnetic Resonance Imaging
FOV = Field of View
GCS = Glasgow Coma Scale
HC = Healthy Controls
HI = Head Injury Index
ICA = Independent Components Analyses
IFG = Inferior Frontal Gyrus
IPL = Inferior Parietal Lobule
LG = Lingual Gyrus
LOC = Loss of Consciousness
MFG = Middle Frontal Gyrus
MoCa = Montreal Cognitive Assessment
MOG = Middle Occipital Gyrus
mPFC = medial Prefrontal Cortex
MRI = Magnetic Resonance Imaging
MTG = Middle Temporal Gyrus
MVA = Motor Vehicle Accident MW = Mind-Wandering
OFC = Orbitofrontal Cortex
PCC= Posterior Cingulate Cortex
SBPS = Shortened Boredom Proneness Scale
SFG = Superior Frontal Gyrus
SN = Starry Night
sogICA = Self-Organizing Group Independent Components Analysis
SPL = Superior Parietal Gyrus
STG = Superior Temporal Gyrus
TBI = Traumatic Brain Injury
vmPFC = ventromedial Prefrontal Cortex

Chapter 1: Introduction

The tendency to experience boredom, a state of disengagement from one's environment, on a regular basis has been associated with a raft of negative cognitive, affective, and behavioural consequences. While the antecedents of boredom and the underlying mechanisms common to those who chronically experience the state remain unknown, there is general agreement that boredom is negatively valenced (Eastwood, Frischen, Fenske, & Smilek, 2012). Furthermore, research has shown that those high in boredom proneness demonstrate an increased propensity to take risks, engage in substance abuse and problem gambling, and tend to have poor outcomes in achievement settings (Joireman, Anderson & Strathman, 2003; Kass & Vodanovich, 1990; Mercer & Eastwood, 2010; Pekrun et al., 2014).

One prominent model of boredom suggests that the experience represents a disengaged attentional state (Eastwood et al., 2012). Research has consistently shown that those high in boredom proneness also exhibit difficulties in sustaining attention and show strong positive correlations with attention-related cognitive errors, lapses in attention, and symptoms of attention-deficit hyperactivity disorder (ADHD; Carriere, Cheyne, & Smilek, 2008; Cheyne, Carriere, & Smilek, 2006; Damrad-Frye & Liard, 1989; Hamilton, 1981; Malkovsky, Merrifield, Goldberg & Danckert, 2012).

Boredom proneness has also been associated with a broad range of negatively valenced affective states and syndromes, most notably, depression (Farmer & Sundberg, 1986; Goldberg, Eastwood, LaGuardia & Danckert, 2011). Research has also shown strong associations between boredom and difficulties dealing with and expressing anger (Dahlen, Martin, Regan & Kuhlman, 2004), and exhibiting aggressive tendencies such as physical and verbal aggression and hostility (Fahlman, Mercer-Lynn, Flora & Eastwood, 2013; Rupp & Vodanovich, 1997). In this context, one could consider depression and aggression to represent internalized and externalized affective dysregulation respectively (Dahlen et al., 2004; Fahlman et al., 2013; Rupp & Vodanovich, 1997).

The cognitive and affective dysregulation common to boredom proneness begs the question of a possible common mechanism underlying these relationships. The capacity for effective self-regulation in the pursuit of goals represents one possible mechanism underlying the cognitive and affective correlates of boredom proneness. That is, the bored individual wants to be engaged with their environment in some meaningful and satisfying way (Van Tilburg & Igou, 2011a; Van Tilburg & Igou, 2011b), but all attempts to do so fail. That failure, which in turn leads to the cognitive and affective consequences of boredom, may result from impoverished self-regulatory skills (Denson et al., 2011; Fahlman at el., 2013; Rehm, 1977; Stark, Reynolds & Kaslow, 1987). Indeed, recent research has reported a strong negative relationship between boredom proneness and individual levels of self-control, as well as demonstrating specific relationships with distinct self-regulatory profiles (Struk, Scholer, & Danckert, 2015). In other words, the more adept one is at controlling one's own thoughts, emotions, and actions, the more effective they will be in goal pursuit and the less likely they are to be boredom prone.

Without a better understanding of the underlying psychology of boredom proneness, and a pragmatic explanation of how this trait manifests, it is difficult to suggest potential options for alleviation. This is an important issue given that many clinical syndromes are accompanied by elevated self-reported levels of boredom. As previously mentioned, boredom proneness and depression are strongly correlated in healthy adults – and these relationships are exacerbated as a function of traumatic brain injury (TBI; Goldberg & Danckert, 2013; Seel et al., 2003; Kreutzer, Seel & Gourley, 2001). A persistent state of disengagement – characteristic of boredom – can make the rehabilitation process more difficult than it already is (Seel & Kreutzer, 2003). The suite of deficits common to TBI patients – the so-called dysexecutive syndrome – can in part be thought of as a consequence of impoverished self-control (Arciniegas & Wortzel, 2014; Bailey et al., 2015; Depue et al., 2014; Ham et al., 2014; McDonald et al., 2010; McDonald, Saad & James, 2011; Reeves & Panguluri, 2011; Swick, Honzel, Larsen & Ashley, 2013).

The broad objective of this thesis was to examine the cognitive and affective relationships with boredom proneness through a lens of self-control. Can individual differences in levels of self-control explain the existing relationships between boredom proneness and attentional and affective dysregulation? This was done through surveys of healthy and brain damaged individuals, and a functional neuroimaging study in these same populations. This body of work represents a first step towards building a more comprehensive understanding of boredom proneness both in healthy individuals and as a consequence of TBI by examining the cognitive, affective, and neural underpinnings of boredom.

Study 1 sought to investigate the relationship between cognitive and affective measures related to boredom proneness, and to better understand the degree to which individual levels of self-control can account for these relationships. Study 2 built on this work by assessing the same measures of cognition and affect as they relate to boredom proneness in individuals with varying degrees of traumatic head injury. Finally, Study 3 utilized resting state functional magnetic resonance imaging (fMRI) to examine the relationship between boredom and activity in the default mode network (DMN) in a sample of healthy controls, as well as a small TBI sample. The brain regions that comprise the DMN have been implicated in 'off-task' processing – that is, they represent brain activity when there is no external task to engage with. As such, one might expect to see DMN activity when people are disengaged from an external event. The findings are discussed in terms of a current model of boredom which suggests the state represents disengagement from one's environment despite a motivation for the opposite – a disengagement that is strongly negatively valenced and likely represents a failure of self-control (Eastwood et al., 2012).

Chapter 2: Cognitive and affective predictors of boredom proneness[1]

2.1 Introduction

Boredom is a ubiquitous human experience characterized by a failure to engage with one's environment – a failure that is negatively valenced (Eastwood et al., 2012). Higher levels of boredom proneness can negatively impact attentional capacities, emotional well-being, and have been associated with problematic behavioural consequences. For instance, high boredom-prone individuals are more likely to engage in addictive behaviours such as substance abuse and problem gambling (e.g., Mercer & Eastwood, 2010), impulsive and higher risk-taking behaviours (Joireman, Anderson & Strathman, 2003; Kass & Vodanovich, 1990), and tend to have poorer outcomes associated with achievement settings (Pekrun et al., 2014).

The propensity to experience boredom regularly – that is, trait boredom proneness – has been associated with poor sustained attention, increased attentional lapses, attention-related cognitive errors, and mind-wandering (Cheyne, Carriere & Smilek, 2006; Carriere, Cheyne & Smilek, 2008). Similarly, research has shown that high boredom proneness was associated with poor performance on measures of sustained attention, with individuals who scored high on boredom proneness also demonstrating increased adult symptoms of attention deficit-hyperactivity disorder (ADHD; Malkovsky et al., 2012). Similarly, Gerritsen and colleagues (2014) found that boredom was associated with inattention, hyperactivity, and executive dysfunction. Taken together, this suggests that boredom proneness is associated with dysregulation of attentional control (Eastwood et al., 2012).

While research has demonstrated a clear link between boredom proneness and cognitive difficulties, it has also been associated with negative affective consequences. High boredom

---

[1] A version of this chapter has been published as Isacescu, J., Struk, A.A., & Danckert, J. (2016). Cognitive and affective predictors of boredom proneness. *Cognition & Emotion.* DOI: 10.1080/02699931.2016.1259995.

proneness is associated with feelings of dissatisfaction, frustration, and anger (Dahlen et al., 2004; Goldberg et al., 2011; Cheyne, Carriere & Smilek, 2008; Fahlman et al., 2013). Perhaps the most commonly demonstrated relationship between boredom proneness and affect is with depression (Farmer & Sundberg, 1986; Goldberg et al., 2011). It may be the case that the persistent disengagement from one's environment – characteristic of boredom – in turn leads to feelings of sadness, helplessness, and in more extreme cases, depressive episodes (Smallwood, Fitzgerald, Miles & Phillips, 2009). At least one study provides tentative support for this contention. Using structural equation modelling, these authors suggested that lapses in attention (i.e., disengagement from one's task or environment) do indeed lead to elevated levels of *both* boredom *and* depression (Cheyne, Carriere & Smilek, 2006; Carriere, Cheyne & Smilek, 2008). That is, being disengaged from one's environment may be a precursor to both boredom and depression.

Boredom proneness has also been related to inappropriate expression of anger, and deficits in controlling aggressive feelings (Dahlen et al., 2004). When controlling for sensation seeking and impulsivity, research has demonstrated a strong association between boredom proneness and various measures of aggression including physical and verbal aggression, anger, and hostility (Fahlman et al., 2013; Rupp & Vodanovich, 1997). Collectively, these studies suggest that boredom proneness is associated with a difficulty in self-regulating negative affect.

Research has shown that individuals with high self-control (i.e., the capacity to self-regulate one's cognitions, affect, and behaviours; Tangney, Baumeister & Boone, 2004) show a marked reduction in measures of impulsivity, engage in less risky behaviours such as substance abuse and gambling, and experience reduced negative affective states, such as depression (Rehm, 1977) and aggression (Denson et al., 2011). Recent research showed that those low in general measures of self-control tended to exhibit higher boredom proneness (Struk, Scholer, & Danckert, 2015). In other words, those with *high* self-control represent a kind of mirror-symmetric presentation to what is commonly observed in high boredom-prone individuals (Dahlen et al., 2004; Farmer & Sundberg, 1986). Research on the development of self-control has shown that as we age, our levels of self-control increase (Anderson et al., 2001). With an increase in self-control over time, one would expect to see a decline in boredom proneness; indeed studies have reported such a decrease in boredom proneness for older adults, relative to their younger counterparts (Vodanovich & Kass, 1990).

The current chapter aimed to replicate findings pertaining to boredom proneness and self-control, and to extend our understanding of boredom proneness by further exploring the relation between boredom proneness and measures of cognitive and affective dysregulation. With respect to cognitive dysregulation mind-wandering was chosen as the construct of interest as this represents a kind of 'lapse' in attention. Mind-wandering can be divided into deliberate (i.e., intentionally allowing one's thoughts to shift from a current task to something else) and spontaneous mind-wandering (i.e., unintentional 'off-task' processing; Seli, Carriere & Smilek, 2015). The distinction is not trivial. If boredom proneness is more strongly associated with spontaneous mind-wandering, it would lend support to the notion that this trait is more strongly linked to a failure to self-regulate cognition; however, if boredom proneness is related to both spontaneous *and* deliberate mind-wandering equally, a self-regulatory explanation for boredom proneness may not be appropriate. To date no study has examined the relationship between boredom proneness and deliberate or spontaneous mind-wandering. Regarding affect, depression and aggression were chosen as the constructs of interest to better understand which of these constructs best explain boredom proneness.

A large undergraduate sample was surveyed on measures of boredom proneness, self-control, mind-wandering, depression, and aggression to better understand how boredom proneness is related to these constructs; to assess the degree to which cognitive and affective measures can predict boredom proneness; and to assess the role self-control plays in these relationships. It was considered important to first account for any potential influence of age, even in an undergraduate sample with a relatively restricted range. Thus, the first prediction was that boredom proneness would decrease as individuals age (Prediction 1; Vodanovich & Kass, 1990). Next, a replication of previous findings such that boredom proneness is negatively related to self-control was explored (Struk, Scholer, & Danckert, 2015). Given that spontaneous mind-wandering is indicative of poor cognitive control, it was predicted that boredom proneness would relate most strongly to this subtype of mind-wandering and would show little, if any, relationship to deliberate mind-wandering (Prediction 2). With respect to affective correlates, positive associations were expected between boredom proneness and the measures of depression and aggression (Prediction 3). These two affective states were chosen as depression is possibly the strongest and most reliable affective correlate of boredom in the literature and together, depression and aggression represent internalized vs. externalized affective dysregulation respectively (Dahlen et al., 2004; Fahlman et al., 2013; Rupp & Vodanovich, 1997). With respect to the aggression sub-scales physical and verbal aggression, anger, hostility), any directional hypothesis concerning specific subscales were considered speculative. It was expected that levels of self-control would operate as a negative predictor of boredom proneness (Prediction 4), whereas the cognitive and affective indicators of dysregulation would positively predict boredom proneness (Prediction 5). Finally, regarding the role of self-control, it was expected that self-control would account for a significant portion of covariance in these relationships (Prediction 6).

2.2 Method

Participants

The current sample was recruited in the Fall semester of 2013 to participate online using the University of Waterloo's Research Experiences Group in which undergraduate students participate for course credit. The initial sample was comprised of 3,555 individuals; only those participants who reported no prior history of head injury (with or without a loss of consciousness), or neurological or psychiatric illness were included in the study leading to a final sample of 1,928 participants (1,400 females; *M* age = 19.64 years; *SD* = 1.88; range 15-30 years). Participants gave informed consent prior to completing the questionnaires. The study was approved by the University of Waterloo's Office of Research Ethics. Procedures for determining the sample size and data exclusions, as well as all manipulations and measures used in the study are presented in Appendix A.

Self-report measures

SBPS - Shortened Boredom Proneness Scale

The SBPS is an 8-item questionnaire designed to assess trait propensity for experiencing boredom (Struk, Carriere, Cheyne, & Danckert, 2015; Appendix B). The SBPS includes items such as "I find it hard to entertain myself" measured on a 7-point Likert scale from 1 'Strongly disagree' to 7 'Strongly agree'.

BSCS - Brief Self-Control Scale

The BSCS is a 13-item scale that measures the level of self-control one has over one's cognitions, emotions, and behaviours (Tangney, Baumeister & Boone, 2004; Appendix B). It includes items such as "I am good at resisting temptation" measured on a 5-point Likert scale from 1 'Not at all' to 5 'Very much'.

MW - Mind-Wandering

The MW scale is an 8-item measure of the propensity with which an individual allows his/her mind to wander from topic to topic (Carriere, Seli & Smilek, 2013; Appendix B). The scale is split into deliberate (MW-D) and spontaneous (MW-S) subscales, each with 4 items measured on a 7-point Likert scale from 1 'Extremely inaccurate' to 7 'Extremely accurate'. The MW-D scale includes items such as "I allow my thoughts to wander on purpose," whereas the MW-S scale includes items such as "It feels like I don't have control over when my mind wanders."

DASS - Depression, Anxiety and Stress Scale

The DASS is a 42-item questionnaire designed to measure an individual's general level of depression, anxiety, and stress (Lovibond & Lovibond, 1995; Appendix B). The current study only made use of the Depression sub-scale which includes 14 items such as "I felt that life was meaningless" measured on a 4-point Likert scale from 0 'Did not apply to me at all' to 3 'Applied to me very much, or most of the time'.

BPAQ - Buss-Perry Aggression Questionnaire

The BPAQ is a 27-item measure of an individual's level of aggression. The scale subdivides aggression into four domains: 1) physical aggression; 2) verbal aggression; 3) anger; and 4) hostility (Buss & Perry, 1992; Appendix B). This scale includes items such as "Once in a while I can't control the urge to strike another person;" "I have threatened people I know;" "When frustrated, I let my irritation show;" and "When people are especially nice, I wonder what they want," respectively. It is measured on a 7-point Likert scale from 1 'Extremely uncharacteristic of me' to 7 'Extremely characteristic of me.'

Data Analyses

Statistical analyses were conducted using SPSS Statistics 20 (Armonk, NY). First, possible gender differences and age effects on boredom proneness were examined. Second, while controlling for age, partial correlational analyses were performed to examine the direction and strength of any relations between boredom proneness and self-control, mind-wandering, depression, and aggression. Third, to assess the degree to which the cognitive and affective measures predicted levels of boredom proneness, a hierarchical regression analysis was conducted. Fourth, to assess the degree to which self-control accounts for the relationships between boredom proneness and the measures of cognition and affect, partial correlational analyses were once again calculated, this time controlling for age *and* levels of self-control.

2.3 Results

Descriptive and difference statistics are presented in Table 2.1.

Table 2.1. Means and standard deviations for all study variables, and within sample differences.

	Sample (*N* = 1928)				
	Women (*n*=1,400)		Men (*n*=528)		
	M	*SD*	*M*	*SD*	*t*
Age	19.56	1.85	19.84	1.97	-2.89*
Boredom Proneness	23.21	8.65	25.51	9.13	-5.00**
Self-Control	39.76	8.60	38.52	8.58	2.83*
Deliberate Mind-Wandering	17.91	5.80	18.20	5.72	-0.99
Spontaneous Mind-Wandering	16.93	5.57	16.67	5.80	0.88
Depression	11.58	4.42	11.91	4.68	-1.40
Overall Aggression	80.94	28.83	89.49	27.37	-5.99**
Physical Aggression	17.19	8.06	21.52	8.33	-10.26**
Verbal Aggression	20.29	7.70	23.43	7.73	-7.93**
Anger	19.07	8.16	19.30	7.85	-0.56
Hostility	24.39	11.28	25.32	11.00	-1.65

* $p < 0.01$; ** $p < 0.001$.

Women were significantly younger than men in this sample, and reported significantly lower levels of boredom proneness relative to men. Women also reported significantly higher levels of self-control relative to men. Overall levels of aggression were higher in men, as were reports of physical and verbal aggression, relative to women. There were no significant gender differences in reports of deliberate or spontaneous mind-wandering, depression, anger, or hostility (Table 2.1).

A linear regression analysis was employed to determine whether boredom proneness changed as a function of age, while controlling for the effect of gender[22]. Results indicated that age was a significant negative predictor of boredom proneness ($F(2, 1922) = 16.75, p < .001$), indicating that as one ages, boredom scores tend to decline ($\beta = -.06, t = -2.67, p = .008$). This age effect continued to be significant even when the age range was further restricted from 17-22 years ($\beta = -.32, t = -2.20, p = .03$), in line with Prediction 1.

Controlling for age, boredom proneness was negatively correlated with self-control (Table 2.2). Boredom was positively correlated with both deliberate and spontaneous mind-wandering, indicating that mind-wandering of both kinds was more prevalent in those high in boredom proneness. Directly contrasting these two correlations using z-scores for dependent correlations (DeCoster, 2007) demonstrated that the correlation between spontaneous mind-wandering and boredom was significantly larger than the same relationship seen for deliberate mind-wandering ($z = 9.98, p < .001$), in line with Prediction 2.

Table 2.2. Partial correlations for all variables, controlling for age.

	1	2	3	4	5	6	7	8	9	10
1 Boredom Proneness	(.88)	-.542	.200	.426	.574	.429	.263	.262	.336	.475
2 Self-Control		(.84)	-.263	-.469	-.408	-.426	-.287	-.289	-.350	-.419
3 Deliberate Mind-Wandering			(.88)	.422	.116	.143	.117	.095	.104	.135
4 Spontaneous Mind-Wandering				(.87)	.346	.283	.154	.171	.228	.324
5 Depression					(.89)	.426	.202	.250	.337	.520
6 Overall Aggression						(.93)	.741	.811	.857	.817
7 Physical Aggression							(.83)	.528	.519	.406
8 Verbal Aggression								(.80)	.675	.494
9 Anger									(.83)	.611
10 Hostility										(.90)

Chronbach's α levels are presented on the main diagonal in parentheses. All coefficients are significant ($p < .001$).

---

[2] To control for gender, the variable was dummy coded and an unweighted effects code was computed as a ratio between females and males, and then collapsed to assess the effect of age.

Regarding affect, significant positive correlations between boredom proneness and all measures were observed, in line with Prediction 3 (r values ranging from .26 to .57; Table 2.2). Directly contrasting aggression sub-scores and boredom proneness using the DeCoster method indicated that the relationship between boredom proneness and hostility was significantly stronger than the correlations observed for boredom proneness and physical aggression ($z = -9.51$, $p < .001$), verbal aggression ($z = -10.32$, $p < .001$), and anger ($z = -7.76$, $p < .001$). The next largest correlation between boredom and subscales of aggression was observed with anger, which was significantly larger relative to the correlations with physical ($z = 3.47$, $p < .001$) and verbal aggression ($z = 4.26$, $p < .001$). The correlations between boredom and physical and verbal aggression did not differ significantly from one another.

To assess the degree to which boredom proneness is predicted by cognitive and affective measures, a hierarchical regression analysis was conducted (Table 2.3).

Table 2.3. Hierarchical regression analysis statistics for boredom proneness, controlling for gender and age.

						CI (95%)for B			
	B	*SE*	*β*	*t*	*p*	*LB*	*UB*	*sr*	sr^2*(%)*
[1] Gender	1.17	.232	.118	5.062	.000	.719	1.628	.117	.014 (1.37)
Age	-.303	.109	-.065	-2.774	.006	-.517	-.089	-.064	.004 (0.41)
[2] Self-Control	-.279	.022	-.272	-12.971	.000	-.321	-.237	-.219	.048 (4.80)
Deliberate Mind-Wandering	.017	.029	.011	.593	.553	-.040	.074	.010	.000 (0.01)
Spontaneous Mind-Wandering	.208	.033	.132	6.272	.000	.143	.273	.106	.011 (1.12)
Depression	.677	.041	.344	16.563	.000	.597	.757	.280	.078 (7.84)
Physical Aggression	.031	.023	.029	1.371	.171	-.014	.076	.023	.001 (0.05)
Verbal Aggression	-.039	.028	-.034	-1.396	.163	-.094	.016	-.024	.001 (0.06)
Anger	.026	.029	.024	.894	.371	-.031	.084	.015	.000 (0.02)
Hostility	.100	.019	.126	5.152	.000	.062	.138	.087	.008 (0.76)

DV = Boredom Proneness; B = unstandardized beta coefficient; SE = standard error of unstandardized beta coefficient; *β* = standardized beta coefficient; *t* = t-score; *p* = significance value; *CI* = confidence interval; *LB*/*UB* = lower/upper bounds; *sr* = semi-partial correlation; sr^2*(%)* = squared semi-partial correlation (unique variance).

The first step of the regression was used to control for gender and age effects, which accounted for ~2% of the total variance in the model (R^2 = .02, *SE* = 8.81, $p < .001$); the second step included all cognitive and affective measures of interest, which accounted for an additional 46% of variance (ΔR= .46, *SE*= 6.42, $p < .001$). Results showed that self-control was the sole significant *negative* predictor of boredom proneness, in line with Prediction 4. Spontaneous mind-wandering was a significant *positive* predictor of boredom proneness (Prediction 5), whereas deliberate mind-wandering did not reach significance, and failed to improve the fit of the model. Regarding affect, depression and hostility were significant positive predictors of boredom proneness (also evidence for Prediction 5), whereas physical aggression, verbal aggression and anger did not significantly predict boredom proneness and failed to improve the fit of the model. The overall model fit was significant ($F(10,1827) = 167.92$, $p < .001$), with a total of 47.7% of the variance explained (adjusted R^2 = 0.477).

Finally, to assess the degree to which self-control accounts for the relationships between boredom and all measures of mind-wandering, depression, and aggression, partial correlations were conducted controlling for age and self-control. All relationships remained significant (Table 2.4); but decreased in magnitude across the board (Prediction 6; Table 2.5). To illustrate this decrease when taking self-control into account, coefficient difference scores were computed for each r-value and are presented as *percentage decreases* in Table 2.5 and Figure 2.1. These decreases provide a rough indication of the proportion of the relationship between boredom proneness and each variable that can be accounted for by age and levels of self-control.

Table 2.4. Partial correlations for all variables controlling for age and self-control.

	1	2	3	4	5	6	7	8	9
1 Boredom Proneness	(.88)	.071**	.232***	.461***	.261***	.133***	.131***	.185***	.325***
2 Deliberate Mind-Wandering		(.88)	.351***	.009	.035	.045*	.021	.013	.029
3 Spontaneous Mind-Wandering			(.87)	.192***	.104***	.023	.042	.077**	.159***
4 Depression				(.89)	.305***	.097***	.151***	.228***	.421***
5 Overall Aggression					(.93)	.714***	.794***	.836***	.778***
6 Physical Aggression						(.83)	.486***	.466***	.328***
7 Verbal Aggression							(.80)	.640***	.429***
8 Anger								(.83)	.545***
9 Hostility									(.90)

* $p < 0.05$; **0.01; ***0.001.

Table 2.5. Partial correlations between boredom proneness and all measures controlling for age, and age and self-control.

	Boredom Proneness		
	C: Age	*C: Age + Self-Control*	% Change
Deliberate Mind-Wandering	.200	.071	64.5
Spontaneous Mind-Wandering	.426	.232	45.5
Depression	.574	.461	19.7
Total Aggression	.429	.261	39.2
Physical Aggression	.263	.133	49.4
Verbal Aggression	.262	.131	50.0
Anger	.336	.185	44.9
Hostility	.475	.325	31.6

Table 2.5. C = Control variable. All coefficients are significant ($p < .001$).

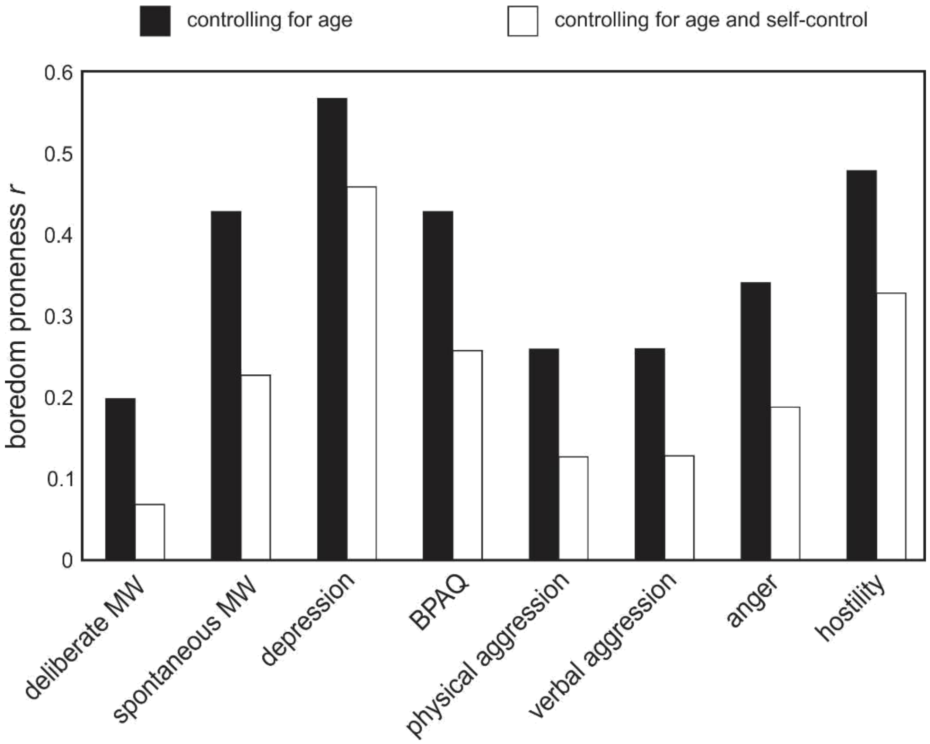


Figure 2.1 Changes in correlation coefficients between boredom and all other measures when controlling for age alone (black bars) or age and self-control (white bars).

Taking into account both age and self-control led to dramatic reductions in the magnitude of initial coefficients. Regarding mind-wandering, the strength of both relationships was reduced; boredom proneness and deliberate mind-wandering was reduced by 64.5% and boredom proneness and spontaneous mind-wandering was reduced by 45.5%. Similarly, for the measures of affect, boredom and depression's relationship was reduced by ~20%, and regarding aggression sub-scores, observed decreases ranged from 31% (hostility) to 49% (physical aggression). When controlling for age alone, the relationships between boredom proneness and measures of cognition and affect were moderate in strength; however, when parsing out the influence of self-control, there was a large shift in how these variables related to each other. The strength of every relationship was decreased by a factor of at least 20%, a substantial decrease across the board. These results suggest that a large proportion of how cognition and affect relate to boredom proneness can be accounted for by varying levels of self-control.

2.4 Discussion

This study sought to better understand boredom proneness by exploring the role played by self-control in the cognitive and affective contributors to this trait. In line with previous findings, gender and age differences were observed, with women reporting lower levels of boredom proneness relative to men, and younger people reporting higher boredom proneness relative to older adults (Vodanovich & Kass, 1990). Age was found to be a significant negative predictor of boredom proneness despite the restricted range of the sample (15 years), and remained significant when this age range was further restricted to just 5 years (17-22 y.o.a.). The tantalizing, although admittedly speculative, hypothesis is that levels of boredom proneness follow to some extent the degree of frontal cortical maturation (Hamilton, 1983). This would be consistent with the role of self-control as a negative predictor of boredom proneness (Table 2.3); as frontal cortex matures in the late teens and early twenties, one expects concomitant improvements in executive functions that in part may result in higher levels of self-control (Anderson et al., 2001; Poletti, 2009). Similarly, frontal maturation is related to increased levels of attentional control perhaps making it easier for people to engage with their environment, leading to lower levels of boredom proneness (Keating, 2012). Future research using structural and functional neuroimaging techniques may help address these hypotheses by examining changes in brain structure (e.g., cortical thickness, grey/white matter ratios; white matter connectivity) and activity (e.g., functional connectivity) as a function of age and self-reported levels of boredom proneness.

Regarding self-report measures of cognition, correlational analyses indicated that boredom proneness was most strongly related to spontaneous mind-wandering (Table 2.2). This is consistent with previous accounts relating boredom proneness to increased difficulties with sustained or directed attention (Eastwood et al., 2012; Malkovsky et al., 2012). Furthermore, hierarchical regression indicated that only spontaneous mind-wandering acted as a significant, positive predictor of boredom proneness (Table 2.3). These findings lend support to the notion that a failure to self-regulate attention is strongly related to boredom proneness. It would be worthwhile for future research to pursue the possibility that *deliberate* mind-wandering, by virtue of successful engagement with one's own thoughts, may act to *prevent* boredom proneness.

With respect to affective measures, correlational analyses indicated that depression and all subscales of aggression were positively correlated with boredom proneness, replicating previous findings (Table 2.2; Dahlen et al., 2004; Rupp & Vodanovich, 1997). The regression analysis similarly replicated previous findings that showed levels of depression to be a significant positive predictor of boredom proneness (Table 2.3; Goldberg et al., 2011). Interestingly, when examining the subscales of aggression, it was shown that only hostility significantly predicted boredom proneness. Both affective states, depression and hostility, would make it difficult to engage with the environment, albeit for potentially different reasons. For the depressed individual, the failure to satisfy the need for external stimulation may result in feelings of helplessness that in turn impede their ability to engage with their environment. On the other hand, increased levels of hostility may be related to a higher tendency to discount, or devalue, potential options for engagement – options that may otherwise alleviate boredom (Stein & Madden, 2013; note: such 'discounting' behavior may also be evident in depressed individuals; see Dennhardt & Murphy, 2011). Indeed, research on boredom and discounting has shown that high boredom-prone individuals will readily discount rewards that are not immediate (Smits et al., 2013). To discount an option before considering it entirely is in essence antagonistic, and may explain why hostility is a strong positive predictor of boredom proneness. Clearly, further research is needed to fully explore these hypotheses.

Interestingly, self-control was the only construct negatively correlated with boredom proneness (Table 2.2); the regression analysis indicated that it was also the only negative predictor of boredom proneness (Table 2.3), replicating previous work (Struk, Scholer, & Danckert, 2015). Furthermore, results showed that individual levels of self-control can partially explain the observed relationships between boredom proneness and measures of cognition and affect. This provides evidence to support the notion that both the cognitive and affective components associated with trait boredom can be explained by failures of self-regulatory control. The results here reflect the relationships between self-control and *trait* propensity to experience boredom. It may also be the case that the intensity and duration of *states* of boredom are also related to levels of self-control and self-regulatory capacity. Further research on state boredom is needed to address this possibility.

This work is not without limitations. First, correlational analyses do not allow us to infer causation. An experimental manipulation using tasks known to require self-control (e.g., Stroop or Go/No-go tasks) would go some way to addressing whether or not low levels of self-control and high levels of boredom proneness have explicit behavioural consequences. Mood inductions may also help address questions concerning the consequences of state boredom for cognitive and behavioural control. Second, the cognitive and affective constructs measured here are unlikely to function in a unidirectional manner. Instead, boredom, depression and aggression likely interact in dynamic ways. It is entirely plausible that the propensity to experience boredom may lead to feelings of depression and vice versa. Finally, the use of a general measure of self-control is associated with inherent limitations; it is not possible with this measure to separately parse out the cognitive, affective, and behavioural aspects of self-control. Future research could utilize more directed measures of self-control that specifically address distinct regulatory modes or foci.

The current findings underline the dynamic interplay between cognitive and affective components of boredom proneness. This is not to suggest that the chosen measures are the *only* factors that can contribute to boredom proneness; for instance, research has demonstrated that motivation and sensation seeking can play an important role in engaging with one's environment (Dahlen et al., 2004). While motivation or sensation seeking were not assessed directly here, the SBPS assesses an individual's need for external stimulation (Struk et al., 2015); presumably, individuals high in boredom proneness *are* motivated to engage with their environments, but when they attempt to do so, they fail. With respect to sensation seeking, research has shown that peak sensation seeking behaviour occurs in mid-adolescence, tapering off after the age of 15, and is strongly related to immature capacities for self-control. As such, the current sample, with a mean age of 20, is beyond that peak age for sensation seeking (Steinberg et al., 2008). Taken together, the current findings suggest that boredom proneness is strongly related to both cognitive and affective dysregulation, and illustrate that differing levels of self-control can explain a substantial proportion of variance in the relationships between boredom proneness, cognition, and affect.

Chapter 3: Exploring the relationship between boredom proneness and self-control in traumatic brain injury (TBI)[3]

3.1 Introduction

As shown in the previous chapter, boredom proneness is associated with cognitive and affective dysregulation. More specifically, boredom proneness was strongly correlated to spontaneous mind-wandering and self-control on one hand, and to negative affective states such as depression and aggression, especially hostility, on the other. It was also found that self-control tempered the relationship between boredom proneness and spontaneous mind-wandering (i.e., those who exhibited low levels of self-control experienced the highest levels of boredom proneness and spontaneous mind-wandering, whereas individuals with high self-control exhibited the lowest boredom proneness scores and spontaneous-mind wandering (Chapter 2; Isacescu, Struk & Danckert, 2016). These findings suggest that one common factor underlying higher levels of boredom proneness relates to individual levels of self-control or self-regulation (Isacescu, Struk & Danckert, 2016; Struk, Scholer & Danckert, 2015; see also Elpidorou, 2014). The raft of negative impacts associated with high boredom proneness can also be recast as *failures* of self-control. That is, increases in impulsivity, risk taking and addictive behaviours ranging from substance abuse to problem gambling, all reflect, at least to some degree, failures of self-control or self-regulation (Fahlman at el., 2013; Stark, Reynolds & Kaslow, 1987). Such failures of self-control are also prominent in individuals who have suffered from traumatic brain injury (TBI; Arciniegas & Wortzel, 2014; Bailey et al., 2015; Depue et al., 2014; Ham et al., 2014; McDonald et al., 2010; McDonald, Saad & James, 2011; Reeves & Panguluri, 2011; Swick et al., 2013).

---

[3] A version of this chapter has been published as Isacescu, J. & Danckert, J. (2016). Exploring the relationship between boredom proneness and self-control in traumatic brain injury (TBI). *Experimental Brain Research*, DOI 10.1007/s00221-016-4674-9.

There is some evidence to suggest that boredom levels are elevated following TBI (Seel & Kreutzer, 2003); however, these authors did not address the distinction between state and trait boredom. It is plausible that the tendency to experience boredom may be a consequence of increased difficulties in sustaining attention – a commonly observed deficit following TBI and a prominent factor related to boredom proneness (Dockree et al., 2004; Dockree et al., 2006; Drew et al., 2007; Konrad et al., 2011; O'Keeffe, Dockree, Moloney, Carton & Robertson, 2007; Whyte et al., 1996; Malkovsky et al., 2012). Alternatively, increases in the propensity to experience boredom, as well as the challenges TBI individuals face with sustaining attention, may both reflect failures of self-control. The current study represents a first step in addressing this hypothesis.

Acceleration-deceleration injuries – the most common causes of TBI – affect the orbitofrontal cortex (OFC), a region of the brain known to represent the reward value of choices and decisions to act (Berlin, Rolls, & Kischka, 2004; Elliott, Newman, Longe & Deakin, 2003; Gottfried, O'Doherty & Dolan, 2003; O'Doherty, Kringelbach, Rolls, Hornak & Andrews, 2001; Rule, Shimamura, & Knight, 2002; Wallis, 2007). Accurately representing the reward value of a given stimulus or action choice is a vital component in regulating goal pursuit (Kruglanski, 2003; Mischel, Shoda & Rodriguez, 1989). In Chapter 2, age functioned as a significant negative predictor of boredom proneness (Isacescu, Struk & Danckert, 2016). This was true even when the age range was dramatically restricted to only include participants aged 17-22 years. Even in such a narrow age range, older participants demonstrated lower levels of trait boredom. This age range represents a critical period of neurodevelopment in which late maturation of the frontal association cortices – including the OFC – occurs (Gogtay et al., 2004). Furthermore, the effect of age on boredom proneness was also tempered by self-control – as we age, we attain higher levels of self-control that in turn enable us to stave off boredom. Injury to the OFC, as often occurs following TBI, may interfere with that relationship, leading to impoverished self-control and increased levels of boredom proneness.

Individuals suffering from TBI experience more than just the cognitive sequelae of poor sustained attention (Andersson, Gundersen & Finset, 1999; de Sousa, McDonald & Rushby, 2012; Ganesalingam, Yeates, Sanson & Anderson, 2007; Jorge & Arciniegas, 2014; Juengst, Arenth, Raina, McCue & Skidmore, 2014; Konrad et al., 2011; Lange, Iverson & Rose, 2011; Rosenberg, McDonald, Dethier, Kessels & Westbrook, 2014; Sigurdardottir, Andelic, Roe & Schanke, 2014). Affective dysregulation is also prominent in TBI with elevated levels of depression, increases in aggression, and difficulties with expressing feelings of anger and frustration, as well as reports of increased levels of hostility (Arciniegas & Wortzel, 2014; Hanks et al., 1999). A common cause for such cognitive and affective dysregulation may be explained by failures of self-control and poor self-regulation in the pursuit of goals.

In this study, self-report measures of cognition and affect in TBI individuals were employed to examine how these measures relate to boredom proneness and self-control. Past research has shown that boredom and depression are more strongly correlated in TBI individuals (Goldberg & Danckert, 2013). It may be the case that the same *exaggeration* of relations is evident in TBI for cognitive (i.e., mind-wandering and self-control) and affective (i.e., aggression) measures as they relate to boredom proneness. It was predicted that boredom proneness would be higher in participants who had suffered moderate to severe TBI when contrasted with those who had suffered mild TBI (i.e., concussion) or healthy controls. It was also predicted that the moderate to severe TBI group would demonstrate the lowest levels of self-control, and that the relationships between boredom and cognitive and affective measures would be strongest in this group.

3.2 Methods

Participants

The current investigation was comprised of three groups of participants: healthy controls, free of neurological and/or psychiatric disorders, with no history of previous head injury; concussed participants, with a history of head injury with or without a loss of consciousness; and, individuals who have sustained a TBI (Table 3.1; For details regarding Participant Recruitment, see Appendix A). All participants gave informed consent prior to completing the questionnaires with one exception. One TBI participant gave assent to participate via permission of their primary care giver (Power of Attorney). This study was approved by the University of Waterloo's Office of Research Ethics (ORE) and the Tri-Hospital Research Ethics Board (THREB).

Table 3.1. Participant Demographics

	Healthy Controls	Concussed	TBI
N (male %)	1928 (27.4%)	340 (38.5%)	35 (74.2%)
Age (SD)	19.64 (1.88)	20.10 (2.67)	43 (13.97)
Education (Myrs)	14	14	10
MoCa *(M)*	-	-	23.9
Head Injury (HI) Index (%)			
0 = No HI	1928 (100%)		
1 = HI no LOC		194 (57.1%)	
2 = HI LOC <1min		82 (24.1%)	
3 = HI LOC 1-5min		40 (11.8%)	
4 = HI LOC 6-15min		11 (3.2%)	
5 = HI LOC 16-30min		2 (.05%)	
6 = HI LOC 30+min		11 (3.2%)	
GCS	N/A	N/A	
Mild (13-15) (n, %)			7 (20%)
Moderate (7-12) (n, %)			7 (20%)
Severe (3-6) (n, %)			18 (51.4%)

*MoCa = Montreal Cognitive Assessment; LOC = Loss of consciousness; GCS = Glasgow Coma Scale; MVA = Motor vehicle accident; HI=Head Injury Index.

Self-Report Measures

All but the DASS scale used in study one (SPBS, BSCS, MW, BPAQ, see Section 2.2) were also employed here, with two additional questionnaires included for the TBI group outlined below.

GCS - Glasgow Coma Scale

The GCS is a neurological scale that assesses consciousness following a head injury, focusing on three elements: 1) responsiveness of the eyes; 2) verbal ability; and 3) motor ability (Teasdale & Jennett, 1974; Appendix B). It is scored on a scale from 3-15, with ranges 3-6 indicating deep unconsciousness (severe brain injury); 7-12 (moderate brain injury) and 13-15 (mild brain injury).

MoCa - Montreal Cognitive Assessment

The MoCa is a 30-point cognitive assessment tool that tests several cognitive domains: short-term memory, visuospatial abilities, multiple aspects of executive functioning, sustained attention, and orientation in time and place (Nasreddine, et al., 2005; Appendix B).

Data Analyses

These data were analyzed in a series of steps. First, independent samples t-tests were conducted to assess differences across measures between groups; controls and concussed were well matched for age, but the TBI group was significantly older. To address this, a subset of age-matched was extracted from the larger control sample, and was used for comparisons with the TBI group. Second, correlational analyses were conducted to assess the degree to which boredom proneness related to all measures of cognitive and emotional functioning in each group; again, age-matched controls[4] and concussed participants were compared directly, and the same relationships were examined separately in the TBI group contrasted with a subset of age-matched controls[5]. Third, multiple regression analyses were conducted: 1) to assess whether the presence and severity of TBI[6] would predict levels of a) boredom proneness, and b) self-control. Fourth, to assess differences in boredom proneness and self-control reports as a function of head injury severity, head injury groups were contrasted (i.e., Controls v. Concussed; Controls v. TBI; Concussed v. TBI[7]). Finally, to assess the degree to which self-control, mind-wandering, and aggression measures predicted boredom proneness in concussed and TBI participants, two multiple linear regression models were conducted, controlling for age and gender.

3.3 Results

Table 3.2 shows the descriptive statistics for the healthy controls and concussed groups on all measures, whereas Table 3.3 shows the descriptive statistics for the TBI participants and their age-matched healthy controls.

---

[4] Given that the TBI sample was so small, direct comparisons to the subset of age-matched controls raised concerns regarding statistical power; to address this, Monte Carlo simulations were run (10,000 iterations for an *n* of 35) to derive 95% Confidence Intervals for each coefficient (Cohen, Cohen, West & Aiken, 2003).

[5] To determine which relationships were strongest within a group, correlations were contrasted using z-scores and the DeCoster test for dependent measures (DeCoster, 2007) with Bonferroni corrections made for the number of comparisons within each group (p=0.05/7; p=0.007).

[6] Head Injury was dummy coded: 0 = No Head Injury to 6 = Head Injury with LOC 30+min; all TBI participants with medical diagnoses received a score of 7 (Table 1).

[7] Again, using the dummy code, the controls (0) were used as the reference group for the first two contrasts, and the concussed group (1-6) were used as a reference group for the final contrast with the TBI participants (7).

Table 3.2. Descriptive statistics for healthy controls (HC) and Concussed groups.

		HC		Concussed			
		N	*M (SD)*	*N*	*M (SD)*	*t*	*p*
Age		1928	19.64 (1.88)	340	20.10 (2.27)	-3.59	0.000
SBPS		1923	23.84 (8.84)	338	25.35 (9.95)	-2.61	0.009
BSCS		1925	39.42 (8.61)	339	38.04 (8.83)	2.67	0.008
MWD		1916	17.99 (5.78)	333	18.14 (5.72)	-0.42	0.674
MWS		1899	16.86 (5.63)	331	17.59 (5.79)	-2.14	0.033
BPAQ		1907	79.58 (27.69)	336	87.35 (29.83)	-4.45	0.000
	PA	1917	23.13 (10.18)	337	27.18 (12.07)	-5.82	0.000
	VA	1919	16.398 (6.25)	338	17.89 (6.54)	-3.89	0.000
	A	1919	15.41 (6.97)	337	16.60 (7.92)	-2.596	0.010
	H	1915	24.64 (11.21)	338	25.88 (11.03)	-1.89	0.059

* t-tests are 2-tailed, independent samples t-tests with unequal variances assumed. SBPS=Short Boredom Proneness Scale; BSCS=Brief Self Control Scale; MWD=Mind-Wandering – Deliberate; MWS=Mind-Wandering – Spontaneous; BPAQ=Buss-Perry Aggression Questionnaire; PA=Physical Aggression; VA=Verbal Aggression; A=Anger; H=Hostility (these last four measures are subscales of the BPAQ).

Independent samples t-tests contrasting the HCs with the Concussed group indicated that the concussed participants had significantly higher levels of boredom proneness, spontaneous mind-wandering and aggression (with all but the hostility subscale reaching significance, the latter scale approaching significance; Table 3.2). In addition, concussed participants demonstrated significantly lower levels of self-control (Table 3.2).

Table 3.3. Descriptive statistics for age-matched healthy controls (am-HC) and TBI groups.

	am-HC		TBI			
	N	*M (SD)*	*N*	*M (SD)*	*t*	*p*
Age	36	39.86 (7.63)	33	43.00 (13.97)	-1.14	0.246
Boredom Proneness	36	17.69 (6.16)	35	32.17 (6.82)	-9.38	0.000
Self-Control	36	43.78 (9.59)	34	41.79 (10.08)	0.843	0.402
Deliberate Mind-Wandering	36	14.94 (5.45)	35	14.51 (6.47)	0.302	0.763
Spontaneous Mind-Wandering	36	13.42 (6.34)	35	18.77 (6.96)	-3.39	0.001
Aggression (Total)	36	62.81 (23.05)	34	94.44 (33.25)	-4.60	0.000
Physical Aggression	36	17.56 (6.81)	34	24.94 (13.28)	-2.90	0.006
Verbal Aggression	36	12.89 (6.54)	35	18.82 (7.26)	-3.62	0.001
Anger	36	14.75 (7.51)	35	22.63 (9.68)	-3.82	0.000
Hostility	36	17.50 (9.68)	35	27.00 (13.89)	-3.41	0.001

* t-tests are 2-tailed, independent samples t-tests with unequal variances assumed.

Similar differences were seen when contrasting the TBI group with their age-matched controls (Table 3.3). Here the TBI participants demonstrated significantly higher levels of boredom proneness, spontaneous mind-wandering, and aggression (for these comparisons all subscales of the BPAQ were significantly higher in the TBI group; Table 3.3).

Next, partial correlations were conducted, controlling for age for the healthy controls and concussed groups (Table 3.4) and the TBI and age-matched healthy controls (Table 3.5).

Table 3.4. Partial correlations: Cognitive and affective measures for Healthy Controls (upper diagonal) and Concussed individuals (lower diagonal).

	1.	2.	3.	4.	5.	6.	7.	8.
1. SBPS		-.536*** (-.727/-.265)	.199*** (.112/.480)	.424*** (.132/.651)	.297*** (-.010/.568)	.199*** (-.124/.481)	.342*** (.030/.590)	.468*** (.184/.680)
2. BSCS	-.504***		-.263*** (-.529/-.050)	-.473*** (-.685/-.189)	-.322*** (-.573/-.013)	-.216*** (-.494/-.105)	-.345*** (-.591/-.037)	-.412*** (-.641/-.102)
3. MWD	.103***	-.183**		.419*** (.123/.649)	.112*** (-.208/.407)	.087 (-.222/.387)	.109*** (-.214/.406)	.131*** (-.192/.419)
4. MWS	.475***	-.468***	.348***		.179*** (.133/.462)	.122*** (-.201/.421)	.224*** (-.095/.496)	.320*** (.012/.569)
5. PA	.251***	-.298***	.156**	.154**		.455*** (.165/.672)	.586*** (.330/.759)	.468*** (.182/.682)
6. VA	.155***	-.227***	.083	.148**	.507***		.571*** (.316/.750)	.395*** (.095/.627)
7. A	.318***	-.325***	.102†	.314***	.522***	.578***		.595*** (.349/.765)
8. H	.459***	-.375***	.144**	.345***	.383***	.382***	.563***	

*=p<0.05; **=p<0.01 ***=p<0.001. †=approached significance with p value = 0.061. Abbreviations as for Table 3.2. Lower and upper bound 95% confidence intervals derived from Monte Carlo simulations (10,000 iterations; *n*=35; Cohen, Cohen, Aiken & West, 2003), shown in parentheses.

Table 3.5. Partial correlations: Cognitive and affective measures for age-matched HC (upper diagonal) and TBI participants (lower diagonal).

	1.	2.	3.	4.	5.	6.	7.	8.
1. SBPS		-.381*	.117	.326†	-.141	-.097	.104	.048
2. BSCS	-.536 **		-.196	-.700***	-.027	-.092	-.141	-.332†
3. MWD	.184	-.260		.351*	.227	-.162	.285†	.174
4. MWS	.309†	-.320 †	.209		-.006	-.156	.067	.325†
5. PA	.466 **	-.528 **	.243	.223		.445 **	.518***	.255
6. VA	.283	-.361	-.021	.354	.381 *		.521 ***	.458 **
7. A	.419 *	-.594 ***	-.056	.440 *	.719 ***	.647 ***		.500 **
8. H	.277	-.560 **	-.018	.091	.385 *	.326 †	.359 *	

*=p<0.05; **=p<0.01 ***=p<0.001. †=approached significance with p values between 0.056 and 0.096. Abbreviations as for Table 3.2.

As was the case for healthy controls in Study 1, correlations in the Concussed group showed that higher levels of boredom proneness were associated with lower levels of self-control, higher levels of mind-wandering (particularly spontaneous mind-wandering) and higher levels of aggression (particularly the hostility and anger subscales; Table 3.2). An almost identical pattern of strength of relationships was observed to that seen in HCs from Study 1. That is, a stronger relationship between boredom and spontaneous vs. deliberate mind-wandering (Z=-6.475, p<0.001); stronger relationship between boredom and hostility vs. all other measures of aggression (all Zs>3.768, all ps<0.001); stronger relationship between boredom and anger vs. verbal aggression (Z=3.362, p<0.001). The relationship between boredom and either physical or verbal aggression was not significantly different. In further support for the similarity of relationships in each group, DeCoster comparisons for independent samples found no significant differences between the groups in the relationships between boredom proneness and either spontaneous mind-wandering, self-control or hostility (i.e., the three most prominent relationships seen within each group).

Given the relatively small samples sizes for the TBI group and their age-matched controls, comparisons within each group did not highlight any significant differences in the relationships observed between boredom proneness and all other measures. Independent samples contrasts revealed that the relationship between boredom proneness and self-control although nominally higher in the TBI group, was not significantly different. However, the relationship between boredom proneness and physical aggression was significantly stronger in the TBI group (Z=-2.607, p=0.009). In addition, the relationships between self-control and both anger (Z=2.184, p=0.0289) and physical aggression (Z=2.258, p=0.023) were significantly stronger in the TBI group.

Next, regression analyses were used to address several key questions. First, does head injury (i.e., presence and severity) predict levels of boredom proneness? Using boredom proneness as the dependent measure the first step in the hierarchical regression was to enter age (β=-0.042, p=0.05) and gender[8] (β=-0.123, p=0.001) as control variables, which accounted for ~2% of the variance in the model (r^2=0.016; Table 3.6). Note that both age and gender were *negative* predictors of boredom proneness.

Table 3.6. Head Injury as a predictor of Boredom Proneness.

		β	t	p	95.0% CI (LB)	95.0% CI (UB)
1	Age	-.042	-2.063	.039	-.155	-.004
	Gender	-.123	-5.971	.000	-2.283	-1.154
2	Head Injury	.108	4.920	.000	1.406	3.269

*DV: Boredom Proneness; CI = Confidence Interval; LB = Lower Bound; UB = Upper Bound.

When Head Injury was entered in the second step of the regression, it functioned as a significant *positive* predictor of boredom proneness (β=0.108, p=0.001, r^2=0.025).

To explore the effects of head injury further, specific contrasts were conducted between the head injury groups. This analysis indicated that when controlling for age and gender, participants who experienced a concussion reported significantly higher levels of boredom proneness relative to healthy controls (β=0.052, p=0.012). Similarly, relative to both healthy controls (β=0.163, p=0.001) and concussed participants (β=0.146, p=0.001), TBI participants reported significantly higher levels of boredom proneness. The model fit in all instances was significant (Table 3.7).

Table 3.7. Head Injury subgroups as predictors of Boredom Proneness.

		β	t	p	95.0% CI (LB)	95.0% CI (UB)
1	Age	-.042	-2.063	.039	-.155	-.004
	Gender	-.123	-5.971	.000	-2.283	-1.154
2	Concussed (v Controls)	.052	2.528	.012	.294	2.331
	TBI (v Controls)	.163	6.595	.000	8.809	16.264
	TBI (v Concussed)	.146	5.837	.000	7.453	14.994

DV: Boredom Proneness; CI = Confidence Interval; LB = Lower Bound; UB = Upper Bound

---

[8] The ratio of men to women was skewed by the number of women in this sample and thus, a weighted effects code was computed to account for this discrepancy prior to including the gender variable in any regression analysis.

Next, the same analyses were run with self-control as the dependent measure. Once again, the first step in the model controlled for age (β=0.098, p=0.001) and gender (β=0.07, p=0.001) which accounted for a little over 1% of the variance (r^2=0.014). Note, as opposed to boredom proneness, these variables now act as *positive* predictors of self-control (Table 3.8).

Table 3.8. Head Injury as a predictor of Self-Control

		β	t	p	95.0% CI (LB)	95.0% CI (UB)
1	Age	.098	4.753	.000	.104	.250
	Gender	.070	3.399	.001	.399	1.488
2	Head Injury	-.054	-2.444	.015	-2.029	-.233

*DV: Self-Control; CI = Confidence Interval; LB = Lower Bound; UB = Upper Bound.

When head injury was entered in the second step of the regression, it functioned as a significant *negative* predictor (β=-0.054, p=0.015) of self-control.

Further examination of the effect of head injury severity on self-control (Table 3.9), indicated that individuals with concussions reported significantly lower levels of self-control (β=-0.05, p=0.016), relative to controls.

Table 3.9. Head Injury subgroups as predictors of Self-Control.

		β	t	p	95.0% CI (LB)	95.0% CI (UB)
1	Age	.098	4.753	.000	.104	.250
	Gender	.070	3.399	.001	.399	1.488
2	Concussed (v. Controls)	-.050	-2.411	.016	-2.204	-.227
	TBI (v. Controls)	-.020	-.815	.415	-5.227	2.158
	TBI (v. Concussed)	-.004	-.168	.867	-4.052	3.414

*DV: Self-Control; CI = Confidence Interval; LB = Lower Bound; UB = Upper Bound.

Surprisingly, no difference was seen between the healthy controls and the TBI participants with respect to self-control. Similarly, there was no difference between the Concussed and TBI groups.

Finally, the cognitive and affective predictors of boredom proneness were investigated in the head injury subgroups. To do this, two linear regression analyses were conducted; in the same manner as in previous analyses, age and gender effects were controlled for, but in this case neither variable reached statistical significance nor improved the fit of the models and were thus removed from further analysis. In the main analyses, measures of self-control, mind-wandering, and aggression were included. Results for the concussed group are presented in Table 3.10; for the TBI group, see Table 3.11.

Table 3.10. Cognitive and affective predictors of Boredom Proneness in individuals with Concussion.

	β	*t*	*p*	95.0% CI (LB)	95.0% CI (UB)
Self-Control	-.331	-6.172	.000	-.487	-.252
Mind-Wandering (Deliberate)	-.069	-1.456	.146	-.279	.042
Mind-Wandering (Spontaneous)	.235	4.259	.000	.213	.580
Physical Aggression	.040	.725	.469	-.057	.124
Verbal Aggression	-.059	-1.030	.304	-.263	.082
Anger	.011	.170	.865	-.145	.172
Hostility	.254	4.514	.000	.129	.328

*DV: Boredom Proneness; CI = Confidence Interval; LB = Lower Bound; UB = Upper Bound.

For the concussed group (Table 3.10), as in the healthy controls presented in Study 1, three significant predictors of boredom proneness emerged: self-control, spontaneous mind-wandering, and hostility. While increases in spontaneous mind-wandering and hostility predicted increases in boredom proneness, increases in self-control negatively predicted boredom proneness. Together, these measures accounted for 39.4% of variance in the model (R^2=0.394, p < 0001.).

Table 3.11. Cognitive and affective predictors of Boredom Proneness in individuals with TBI.

	β	*t*	*p*	95.0% CI (LB)	95.0% CI (UB)
Self-Control	-.287	-1.540	.134	-.458	.064
Spontaneous Mind-Wandering	.137	.807	.426	-.206	.474
Physical Aggression	.300	1.748	.091	-.026	.336

*DV: Boredom Proneness; CI = Confidence Interval; LB = Lower Bound; UB = Upper Bound.

For the TBI group, the results were less clear; overall the model was significant, and explained a total of 30.4% of the variance in boredom proneness scores, but no specific predictor reached statistical significance independently (Table 3.11).

3.4 Discussion

This study examined the relationship between boredom proneness and self-control as a consequence of traumatic brain injury of varying degrees of severity. The relatively large sample sizes in the control and concussed groups yielded significant correlations with boredom proneness and all other measures. A similar pattern of correlations were observed in the Concussed group (Table 3.4), relative to those seen in the healthy sample presented in Study 1 (Table 2.2). Although the Concussed group reported higher levels of boredom proneness, the strength of the relationships between boredom proneness and measures of cognition and affect were not significantly different in this group.

For the first time, a significant rise in boredom proneness as a function of head injury has been demonstrated (Tables 3.2 and 3.3). Head injury operated as a significant positive predictor of boredom across the three groups (Table 3.6 and 3.7), with Concussed participants demonstrating slightly higher levels of boredom proneness than healthy controls, and TBI participants exhibiting the highest levels, even relative to their age-matched controls (Tables 3.2 and 3.3). In addition, TBI participants showed a significant positive correlation between boredom proneness and physical aggression (Table 3.5) not seen in any other group. The regression models showed that presence and severity of head injury, as well as self-control, operated as significant predictors of boredom proneness. This suggests that individuals who have suffered more severe head injuries and who demonstrate lower levels of self-control are more prone to the experience of boredom.

The results with respect to self-control as a function of head injury were somewhat mixed. The Concussed participants reported lower levels of self-control relative to HCs; however, the TBI participants did not show lower levels of self-control relative to age-matched HCs. Both TBI and age-matched control groups had higher levels of self-control relative to the larger healthy control sample, probably reflective of the influence of age (i.e., levels of self-control increase with age). Correlational analyses showed that the nature of the relationships between self-control and the other measures of interest did not differ between control and concussed participants. In the TBI group, results showed that the strength of coefficients was strongest between self-control and measures of aggression (specifically, physical aggression and anger). It may be the case that failures of inhibitory control – a common consequence of TBI – lead this group to express their dissatisfaction with their environment in more direct physical ways. Regression analyses directly contrasting groups showed that the only subgroup which reached statistical significance was the controls v. Concussed group contrast (Table 3.9). The TBI group did not differ significantly from controls or from the Concussed group. There are a few plausible reasons for this: first, the TBI sample is small and variability of responses is high in this population. Second, the TBI sample is older and report generally higher levels of self-control than seen in the younger sample of healthy controls (Tables 3.2 and 3.3). Finally, it is also possible that individuals with TBI over-reported levels of self-control (insight into one's own behaviour is commonly diminished in this group).

With respect to the cognitive and affective predictors of boredom proneness in the concussed and TBI samples, results were somewhat mixed. In the concussed group, results mirrored what was observed in the healthy controls of Study 1 with self-control, spontaneous mind-wandering, and hostility all significant predictors of boredom proneness (Isacescu, Struk & Danckert, 2016). On the other hand, for the TBI group, while the model itself was significant, no individual predictor reached statistical significance independently. It is plausible that the dynamics between these variables of cognition and affect are even more variable among individuals with TBI – the influence of self-control varies even in control populations – and, heterogeneity of data is very common in TBI populations. More research is needed to elucidate the possible mechanisms through which cognitive and affective traits interact to predict boredom proneness in TBI. The current results highlight that boredom proneness is associated with failures of self-control and this is exacerbated in individuals who have suffered varying levels of TBI. The failure to fully engage with a task or environment in this population is likely to have serious consequences for an individual's ability to re-establish independence and to benefit from rehabilitation programs.

There are several key limitations to the current study that warrant mention. First, the TBI group did not differ from their age-matched controls in terms of self-reported levels of self-control. This highlights the challenge of self-report measures in a TBI population (Sherer et al., 2014). That is, individuals with TBI often lack insight into their condition and abilities making their self-reports (for all measures used here) somewhat unreliable. Using care-giver responses will overcome this to some extent. Nevertheless, even if a lack of insight (or the effects of desirability – making responses that put one in a positive light; Dyer, Bell, McCann & Rauch, 2006) influenced reports of levels of self-control, they cannot explain some of the other results. That is, desirability or a lack of insight would presumably lead to under-reporting of levels of physical aggression that were highest in the TBI group. Similarly, group membership (i.e., the presence and severity of a head injury) is not susceptible to these problems and accounted for over half the variance in the model with age-matched controls.

Additionally, age-matched controls for the TBI group were not matched on gender (quite the opposite). The findings here and in Study 1 suggest that while gender does impact upon boredom proneness (with males more prone to the experience than females); this effect is typically small, accounting for only 1-2% of the variance in the model. Nevertheless, this discrepancy was taken into account using weighted effects codes computed. A much bigger contributor, evident when controlling for gender, are levels of self-control which was true in the current samples; while it would have been ideal to match on gender as well, it seems unlikely that it has skewed the results dramatically. Finally, it was not possible to control for general levels of cognitive functioning in the regression analyses as a measure of this (i.e., the MoCA) was only obtained for the TBI sample. When assessing the relationship of participant MoCa scores to boredom proneness in this group, analyses failed to reach significance (correlations as well as regressions). This is important for the TBI group especially, given that a lower cognitive functioning score could suggest that cognitive deficits may contribute in some way to elevated levels of boredom proneness.

Perhaps what is warranted as a next step in exploring the consequences of brain injury for boredom proneness is to develop behavioural assays of the experience – objective measures that distinguish between those high vs. low in boredom proneness. In the past, sustained attention tasks have been used to demonstrate that high boredom prone individuals perform more poorly than their low boredom counterparts (Malkovsky et al., 2012). This behavior is similar to what is seen in TBI individuals (Robertson, Manly, Andrade, Baddeley & Yiend, 1997; Dockree et al., 2004, Dockree et al., 2006; O'Keeffe et al., 2007). While this shows that attention deficits are characteristic of both TBI and healthy individuals highly susceptible to boredom, they do not provide any direct metric of boredom proneness. The proposed cornerstone of boredom proneness – a disengagement from one's environment – may manifest as a failure to *persist* in performing a challenging task. One possibility for future research then would be to employ variants of tasks such as foraging to examine the extent to which high and low boredom prone individuals (and individuals with TBI) engage with a variable and challenging task environment.

Chapter 4: Exploring the neural networks associated with boredom in healthy controls and TBI participants.

4.1 Introduction

The first two studies of this thesis explored the relationship between boredom proneness and cognitive and affective measures as they relate to different levels of self-control. The results support the notion that boredom proneness is related to lower levels of self-control reflective of failures of self-regulation affecting both cognitive and affective domains (Eastwood et al., 2012; Isacescu, Struk & Danckert, 2016; Isacescu & Danckert, 2016; Struk, Scholer & Danckert, 2015). Interestingly, age operated as a significant negative predictor of boredom even when a very narrow age range (17 – 22 y.o.a) was explored (Study 1; Isacescu, Struk & Danckert, 2016). This hints at the possibility that frontal cortical maturation leads to decreased levels of boredom proneness. Finally, boredom proneness was increased as a function of head injury (Study 2; Isacescu & Danckert, 2016), supporting the notion that the integrity of frontal cortex – compromised in TBI – is important in staving off the experience of boredom.

The findings presented thus far underscore the relationship between boredom proneness and dysregulation of attentional control. As mentioned in Chapter 2, this relationship has been repeatedly demonstrated across a range of studies showing that boredom proneness is associated with poor sustained attention, increased attentional lapses, attention-related cognitive errors (Bench and Lench 2013; Cheyne, Solman, Carriere & Smilek, 2009; Cheyne, Carriere & Smilek, 2006; Carriere, Cheyne & Smilek, 2008; Malkovsky, et al. 2012), spontaneous mind-wandering (Isacescu, Struk & Danckert, 2016), and adult symptoms of ADHD (Malkovsky et al, 2012).

Imaging research exploring disengaged attentional states, such as off-task processing and spontaneous mind-wandering, have shown neural activation within a network of brain regions known collectively as the default mode network (DMN; Binder et al. 1999; Bonnelle et al., 2011; Buckner et al., 2008; Christoff , 2011; Christoff, Gordon, Smallwood, Smith & Schooler, 2009; Gusnard & Raichle 2001; Mason et al., 2007; Schooler et al., 2011, Weissman, Roberts, Visscher, & Walforff, 2006). A highly-interconnected set of brain regions, the DMN supports internally-focused thinking (i.e., thinking to oneself, imagining past events, thinking of the future) and is activated when there is no external task or stimulus for the individual to engage with (Buckner, Andrews-Hanna & Schacter, 2008; Gusnard, Akbudak, Shulman & Raichle, 2001; Mason et al., 2007). Structurally, the main hubs of the DMN include the posterior cingulate cortex and precuneus, ventromedial prefrontal cortex and medial, lateral, and inferior parietal cortices (Buckner, Andrews-Hanna & Schacter, 2008). In healthy participants, DMN activation has been shown to increase when at rest or during mind-wandering, and to decrease when one is engaged in an externally-focused task (Greicius, Krasnow, Reiss & Menon, 2003; Mason et al. 2007; Weissman et al. 2006; Gusnard & Raichle 2001).

Research exploring the neural underpinnings of boredom is in its infancy. While imaging studies have assessed boredom levels in passing, few have made it the primary focus. For instance, Ulrich and colleagues assessed the neural networks of an experimentally induced state of “flow” – a state one might consider to be the opposite of boredom, in which the individual is deeply engaged in an activity to the point that all else fades away (Csikszentmihalyi, 1996; Nakamura & Csikszentmihalyi, 2014). In their experiment, participants had to perform mental arithmetic tasks (i.e., summing two or more numbers) of varying levels of difficulty: the task with the lowest degree of difficulty was considered “boring” and was used as a control relative to an adaptive “flow” condition in which difficulty levels were adjusted according to a participant’s performance (Ulrich, Keller, Hoenig, Waller & Gron, 2014). They found that, relative to the boring condition, the state of flow was associated with a decrease in DMN activation, specifically in medial prefrontal cortex and the amygdala. Relatively speaking, posterior portions of the DMN were more active during the boring condition (Ulrich et al., 2014). In a more recent study, the state of boredom was more directly assessed via a mood induction and was contrasted with a resting state, an induction of interest/engagement, and a sustained attention task (Danckert & Merrifield, 2016). Results showed that when people were bored activity was evident in the posterior cingulate and adjacent precuneus, middle and superior temporal gyri – in other words, posterior portions of the DMN. Interestingly, anticorrelated activity was observed in the anterior insula during the boredom mood induction. No such activity was evident for the resting state scan (Danckert & Merrifield, 2016).

A host of imaging research has been conducted to better understand the neurocognitive consequences of TBI, with results indicating that TBI survivors are left with pervasive structural damage to the neocortex and white matter connective tissue (Hulkower, Poliak, Rosenbaum, Zimmerman & Lipton, 2013; Kraus et al., 2007; Liu, Maldjian, Bagley, Sinson & Grossman, 1999; Mayer, Bellgowan & Hanlon, 2015; Peduzzi, Eleftheriou & Novack, 2001; Scheid, Preul, Gruber, Wiggins & Von Cramon, 2003; Sharp, Scott & Leech, 2014; Smith & Meaney, 2000). The consequences from such diffuse damage can range from difficulties with concentration (Drew et al., 2007; Kim et al., 2007), orienting attention (Nicholl & LaFrance, 2009; Rabinowitz & Levin, 2014; Rao & Lyketsos, 2000; Riggio, 2010), disinhibition (poor control of both emotions and cognitions; Fischer et al., 2014; Hart et al., 2011; Jorge & Arciniegas, 2014; Kim, 2002; Konrad et al., 2014; Wood & Thomas, 2013), and as shown in Study 2, increased levels of boredom proneness (Isacescu & Danckert, 2016). Studies investigating the integrity of connectivity between brain regions after TBI have implicated the involvement of the DMN in attentional tasks, and suggest that network connectivity of the DMN in TBI can predict sustained attention deficits. That is, those individuals with more damage present to tracts that connect the DMN (to each other and to other parts of the brain), present with greater impairments of sustained attention (Bonelle et al., 2011).

Downregulation of the DMN is typically accompanied by upregulation of the salience and central executive networks (i.e., cortical areas typically involved in change detection such as the anterior cingulate cortex (ACC), presupplementary motor areas (preSMA) and anterior insula (AI); Bucknor et al., 2008). One study investigating the structural integrity of salience networks in TBI, demonstrated a failure to *deactivate* the DMN during tasks that required sustained attention and would presumably also require upregulation of the central executive and salience networks. In other words, TBI patients exhibited impaired co-ordination of large scale neural networks (Bonnelle et al., 2012). This failure to downregulate the DMN may be in part responsible for the inefficient cognitive control evident in TBI (Bonnelle et al., 2012).

The aim of the current study was two-fold: first, to replicate previous findings concerning the neural networks associated with state boredom in healthy adults and second, to extend this investigation to include individuals with TBI. A total of 13 controls and four TBI participants[99] were scanned in a series of conditions: a boredom mood induction, a resting state scan, a video intended to induce a state of 'interest', and a sustained attention task (i.e., using the same scanning protocol as in Danckert & Merrifield, 2016; see Methods). The interest mood induction was intended as a kind of 'opposite' state to boredom and the sustained attention task was included to determine whether the neural underpinnings of a disengaged attentional state would be broadly similar across this and the boredom mood induction. As already mentioned, previous research has shown an increase in DMN activation in TBI individuals while performing sustained attention tasks and during resting states (Bonnelle et al., 2011; Bonnelle et al., 2012), however the neural underpinnings of boredom in this population have not yet been explored. If TBI individuals upregulate the DMN (or, have trouble downregulating it, as other researchers have found), it is plausible that the same can be

---

[9] An extremely conservative approach was taken with respect to selection of individuals with TBI to participate in the fMRI portion of this research. In study 2, there was a TBI sample of 35 and while most of them were interested in participating, a total of 31 were excluded due to a wide range of reasons (Appendix A).

expected for the boredom mood induction. For instance, in healthy controls, DMN structures showed correlated activation when participants were bored – with anticorrelated activation present in the anterior insula (Danckert & Merrifield, 2016). It is plausible then, that the TBI participants will show greater activity in the DMN when bored than is observed in healthy controls. It is also possible that, unlike controls, individuals with TBI may fail to downregulate the salience network. That is, where activity in the anterior insular is anticorrelated with the DMN in healthy controls, it may show the opposite pattern in TBI.

4.2 Method

Participants

This study was conducted with neurologically healthy participants and individuals who had sustained a traumatic brain injury (TBI). Healthy and TBI participants were recruited from the Kitchener-Waterloo community (for recruitment details see Appendix A). All control participants were free of neurological and/or psychiatric disorders, with no history of previous head injury and reported having normal or corrected to normal vision and hearing. The control sample was comprised of 15 healthy adults (9 women, all right handed) and ranged in age from 21-61 (M_{age} = 29.33, *SD* = 11.64). The TBI sample was a subset of the sample studied in Study 2 who agreed to participate in this follow-up fMRI investigation. The sample was comprised of four adults (all male, all right handed) and ranged in age from 26-59 (M_{age}=40.5, *SD* = 15.33)[1010]. This project was reviewed by and received ethics clearance through the University of Waterloo Research Ethics Committee and the Tri-Hospital Research Ethics Board (THREB).

---

[10] From the initial 35 individuals who participated in Study 2, a subset agreed to participate in this fMRI follow-up study. An extremely conservative approach was taken with respect to inclusion criteria for TBI participants in this investigation (Appendix A).

Data from two control participants were not included in the fMRI analyses for the following reasons: data from one participant was removed due to excessive motion; data from a second participant was discarded as preliminary analyses failed to illuminate any significant regions of activation across all four conditions[11]. Therefore, the following results are based on the remaining control sample of 13 participants (M_{age}= 30.13, *SD* = 12.29; 8 females, all right-handed) and four TBI participants.

Self-Report Measures

The SBPS and MW scales, used in the first two studies, were employed here as trait measures of boredom proneness and mind-wandering. In addition, retrospective state measures of boredom and mind-wandering were taken after each functional scan. That is, following each scan participants were asked two questions: 'How bored are you right now?' and "While [watching the video/resting with your eyes open/completing the Starry Night task], how much did your mind wander?" Each question was accompanied by a visual Likert scale ranging from 0 (*not at all*) to 9 (*extremely*) presented onscreen. The questions were posed verbally by the experimenter through the scanner's intercom with participants' responses recorded manually.

fMRI

Four scans were completed by each participant; two conditions involved videos intended to induce either boredom or a state of interest, a third scan required participants to perform a sustained attention task, and the final scan consisted of a standard resting state session (see below).

---

[11] Any speculation as to why this happened was deemed impossible to validate. Several attempts to run the single-subject ICA on this data set found the same (lack of) result suggesting it was not a computer glitch.

Boredom Mood Induction

To induce a state of boredom, a video that portrayed two men hanging clothes on a drying rack, occasionally asking each other for a clothes pin was presented for 8 minutes. In prior work, at a variety of durations (from 171 to 341s), this video has been shown to reliably induce intense feelings of boredom (Merrifield & Danckert, 2014). The 8-minute version of the video used here was constructed by looping a shorter 240s version once (Figure 4.1).

Interest Mood Induction

To induce a state of interest/engagement, an excerpt from the British Broadcasting Company's (BBC) documentary film, *Planet Earth* (Fothergill et al., 2007) depicting exotic animals, landscapes, and vegetation was used. Prior research has shown that this clip elicits a strong state of interest (Merrifield & Danckert, 2014). An 8-minute version of this movie was used here (Figure 4.1).

Resting State Scan

Participants were presented with a visual display consisting of a grey background with a centrally located black fixation cross for 8 minutes. They were instructed to relax with eyes open and remain as still as possible for the duration of the scan.

Sustained Attention: the Starry Night Task

Participants were presented with a visual display consisting of a black background with ~250 white target dots (approximately 0.5° of visual angle, maximal contrast) randomly distributed onscreen, intended to represent a starry night sky (Rizzo & Robin, 1990; Figure 4.1). At pseudorandom temporal intervals a 'star' could either appear or disappear with participants asked to press a button when they detected either event type. Appearances and disappearances were equally likely, and occurred with an ISI ranging from 2000ms to 7000ms (Figure 4.1). Parameters for each event were as follows: if the number of stars on-screen was between 248 and 252, a random event occurred (i.e., appearance or disappearance); if the number of stars on the screen was 247, an appearance event occurred; and if the number of stars on-screen was 253, a disappearance event occurred. These rules ensured that the number of stars on the screen at any one time ranged between 247 and 253 (i.e., 250 +/-3). Participants were presented with 160 trails (80 Appearances, 80 Disappearances) over a span of 8 minutes.

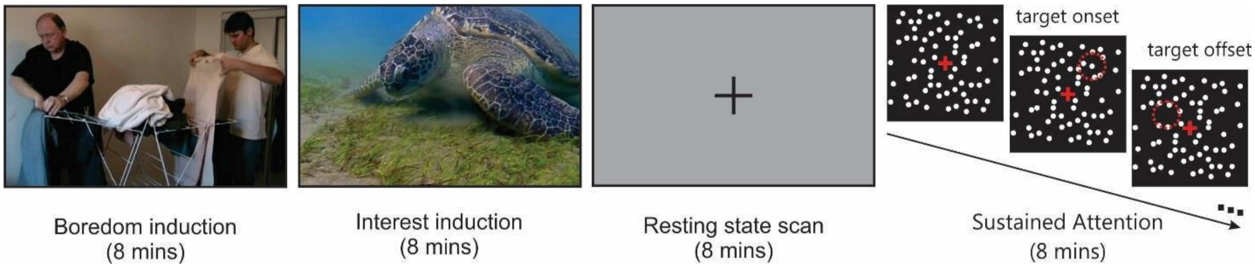


Figure 4.1. Boredom, Interest, Resting State and Starry Night Scanning Conditions.

Apparatus

All stimuli were presented on an Avotec Silent Vision^{TM} (Model SV-7021) fibre-optic visual presentation system with binocular projection glasses controlled by a computer-running E-Prime software (version 1.1, Psychology Software Tools, Pittsburgh, PA) synchronized to trigger-pulses from the magnet.

Procedure

Participants first underwent an anatomical scan prior to the four functional runs: 1) boredom mood induction, 2) interest mood induction, 3) resting state, and 4) the Starry Night task. The four functional runs occurred in random order. During the boredom and interest mood induction scans, participants were instructed to watch the video and to remain still; during the resting state scan, participants were instructed to keep their eyes open, relax, and remain still; finally, during the Starry Night scan, participants were instructed to respond, via button press, as quickly and accurately as possible, to the appearance or disappearance of a 'star' while maintaining their gaze at fixation.

Following each functional run, participants were asked how bored they were and how much their minds had wandered on a scale of 0 (not at all) to 9 (extremely). After exiting the MRI scanner, participants completed the SBPS and Mind-Wandering questionnaires. The experiment lasted approximately 50 minutes.

fMRI Data Acquisition

At the beginning of the session, a whole-brain T1-weighted anatomical image was collected for each participant (TR = 7.5 ms; TE = 3.4 ms; voxel size, 1 x 1 x 1 mm^3; FOV, 240 x 240 mm^2; 150 slices; no gap; flip angle, 8°).

Functional data were collected using gradient echo-planar T2*-weighted images acquired on a Philips 1.5 Tesla machine (TR = 2000 ms; TE = 40 ms; slice thickness = 5 mm with no gap, 26 slices; FOV = 220 x 220 mm^2; voxel size = 2.75 x 2.75 x 5 mm^3; flip angle = 90°). An experimental run consisted of 26 slices/volume and 240 volumes (8 minutes).

fMRI Preprocessing and Statistical Analyses

All MRI data preprocessing and analyses were completed using Brain Voyager QX (version 2.1, Brain Innovation B.V., Maastricht, the Netherlands). First, each participant's anatomical data was transformed into standard stereotaxic space (Talairach & Tournoux, 1988) and co-registered to their functional data. All functional runs were visually inspected for motion artefacts by playing a virtual movie of each volume in sequence (Culham, 2003). Next, preprocessing of all functional data was completed using the following procedures: slice-time correction, linear trend removal, and three cycles of temporal high pass filtering. For all 17 participants, trilinear/sinc interpolation was used to correct for motion artefacts in functional runs. Spatial smoothing using a Gaussian kernel (4 mm Full Width Half Maximum) was applied (Mason et al., 2007). Finally, segmentation of the cortical sheet was carried out and cortex-based volumetric time course (VTC) masks were created for each participant prior to carrying out the Independent Components Analyses (ICA).

Blood oxygenation level dependent (BOLD) signals from each functional run were analyzed using independent components analysis (ICA) to examine network connectivity (DeMartino et al., 2007; Esposito et al., 2005). ICA allows for the identification of sets of voxels with similar spatial patterns in different participants, even if the voxels are distributed in different parts of the brain, are influenced by different sources of noise, and have different time courses in different participants. In this way, temporal and spatial properties can be used to identify task-unrelated noise and components that reflect functional networks in the brain (Beckmann, DeLuca, Devlin, & Smith 2005). In addition, ICA identifies distinct functional networks without relying on a priori hypotheses regarding network anatomy. Functional data was analyzed using the following ICA procedure.

First, single-subject ICAs were conducted for each participant using the fastICA algorithm, once for each of the four scanning conditions (Hyvarienen, Hoyer, & Inki, 2001). Next, 30 Independent Components (ICs; spatial maps) were extracted for each participant, per condition. Individual IC 'fingerprints' were then visually inspected to determine which of these components related to BOLD responses. A fingerprint characterizes each IC along eleven temporal and spatial features, enabling the classification of ICs as related to BOLD responses, motion artefacts, vasculature, etc. (DeMartino et al., 2007).

Next, for the controls and then TBI participants separately, group-level ICAs were conducted using the self-organizing group ICA algorithm (sogICA), once for each scanning condition (DeMartino et al, 2007; Esposito et al., 2005). For each condition, all single-subject IC maps for a particular functional run were clustered at the group level (e.g., for the boredom sogICA, the 30 components extracted for each participant from the boredom functional run were clustered), matching the most similar spatial patterns across participants. From this, 30 group-averaged clusters were extracted and an average spatial map was computed and assumed to be representative for the cluster. The consistency of the clusters across participants was expressed in terms of a similarity mean (*s*), which is defined as the average of the pair-wise spatial correlations between the constituting single-subject IC maps and is based on a hierarchical clustering procedure. That is, the sogICA algorithm converted similarity measures to Euclidean distances and these were used to fill a matrix of distances. Based on this distance matrix, a supervised hierarchical clustering procedure was run, with the supervising constraint consisting of accepting only one component per participant in each cluster formed by the hierarchical procedure. Each of the spatial maps was then visually inspected to identify any major network components. Potential networks and network components were then examined to determine whether they corresponded to BOLD responses, by examining their single-subject maps and fingerprints and only clusters of 100 contiguous voxels and above were considered meaningful. Clusters that were identified as artifacts through this procedure were eliminated from further exploration.

4.3 Results

Self-report measures

Control Participants

Trait Measures

Boredom Proneness

Control participants' mean score on the SBPS was 22.33 (*SD*=7.34, range=10-33), which is on par with the SBPS scores obtained in the larger pool of control participants described in Chapters 2 and 3 (*M* = 23.84, *SD* = 8.84, n = 1928; *t-difference* = -.74, *p* =.76).

Mind-Wandering

Control participants' mean score on the deliberate mind-wandering scale was 21.00 (*SD*=1.95, range=7-17), relative to *M*= 17.99 (*SD* = 5.78; range = 4-28) of the larger control group (*t-difference* = 5.41, *p* = .99). Regarding spontaneous mind-wandering, controls had a mean of 15.08 (*SD* = 4.81, range 8-26), relative to the larger sample that scored *M* = 16.86 (*SD* = 7.72, range = 4-28), *t-difference* = -1.33, *p* = .11).

State Measures

Boredom Probes

Control participants reported feeling most bored in the boredom (*M* = 6.85, *SD* = 1.82; Figure 4.2) and resting state conditions (*M* = 7.54, *SD* = 1.71), which did not differ significantly from each other (*t* = -1.74, *p* = .108). Both conditions were significantly more boring than the interest condition (*M* = 5.15, *SD* = 2.34; *t* = 2.48, *p* = .029 and *t* = 3.48, *p* = .005, respectively) and the Starry Night task (*M* = 4.69, *SD* = 2.53; *t* = 3.542, *p* =.004 and *t* = 4.258, *p* = .001, respectively).

Boredom levels across the Interest and Starry Night conditions did not differ from each other (*t* =.43, *p*=.676).

Mind-Wandering Probes

A similar pattern emerged with respect to the mind-wandering probes: participants mind-wandered the most during the boredom (*M* = 6.23, *SD* = 2.35; Figure 4.2) and resting state (*M* = 6.92, *SD* = 1.66) scans. Again, these two conditions did not differ significantly from each other (*t* = -.987, *p* = .343). Participants mind-wandered significantly less in the Interest and Starry Night conditions relative to the boredom condition (*t* = 3.811, *p* = .002; *t* = 2.509, *p* =.027, respectively), and significantly less than the resting state (*t* = 4.251, *p* = .001 and *t* = 4.530, *p* = .001, respectively). Participants mind-wandered the least during the Starry Night task (*M* = 4.15, *SD* = 2.38), followed by the Interest condition (*M* = 4.54, *SD* = 2.18), but these two conditions did not differ significantly from each other (*t* = .540, *p*=.599).

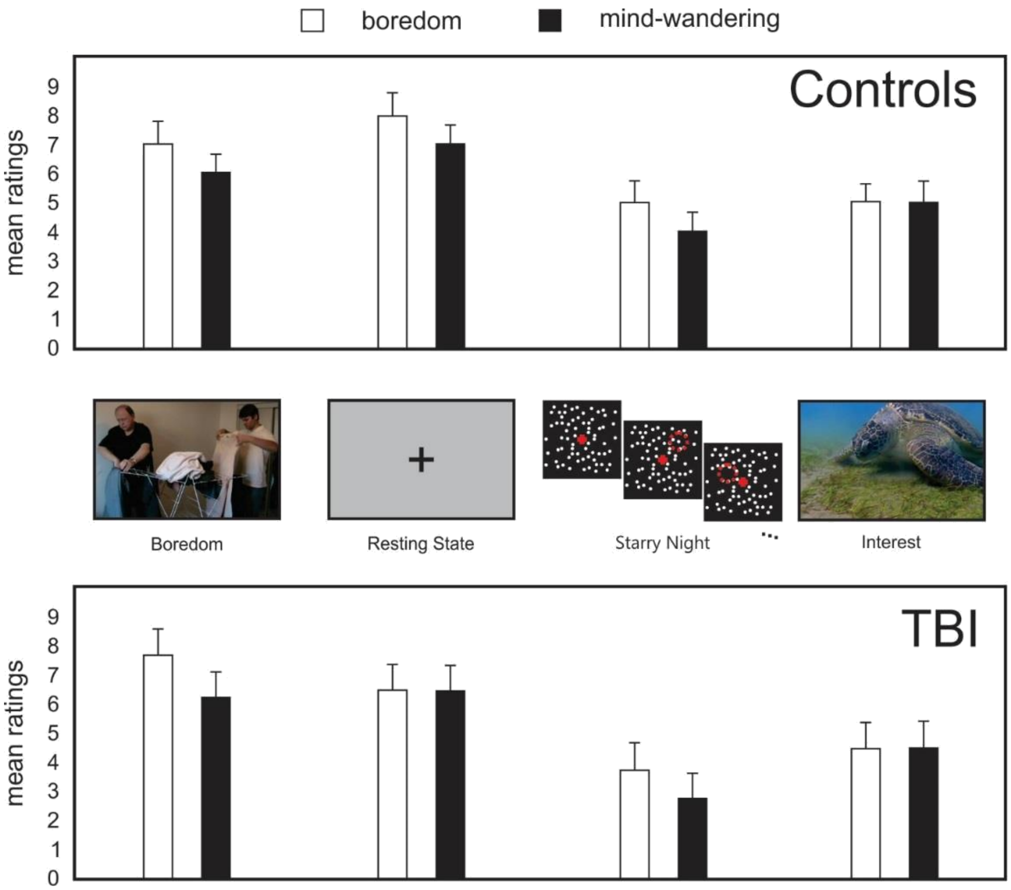


Figure 4.2. Controls' (upper panel) and TBI (lower panel) boredom (white bars) and mind-wandering (black bars) ratings between scanning conditions. Error bars represent SE +/-1.

TBI Participants

Statistics regarding trait and state measures are presented here for the sake of comparison with controls, but should be taken with some caution given the extremely small sample size.

Trait Measures

Boredom Proneness

For the TBI group, mean SBPS score was 22.00 (*SD* = 8.04, range = 16-33). SBPS score observed here are lower than those observed for the larger TBI sample described in Study 2 (*M* = 32.17, *SD* = 6.82, range = 14-50); however, this difference was not statistically significant probably due to the large range of scores in this small sample: *t-difference* = -2.77, *p* = .45). The two samples were similar in age ($M_{Age\ =}$ 40.50, *SD* = 15.33); Study 2 M_{Age} = 43 (*SD* = 13.97).

Mind-Wandering

The TBI group had a Deliberate MW mean of 15.00 (*SD* = 5.83, range = 9-20), relative to the larger TBI sample in Study 2 (*M* = 14.51, *SD* = 6.47, range = 4-27); and spontaneous *M* = 16.25 (*SD* = 8.65, range = 4-24), relative to the larger sample of mean of 18.77 (*SD* = 6.96; range = 4-28).

MoCa and GCS

The TBI group showed similar scores on the GCS (*M* = 6.25; *SD* = 5.85) relative to the larger TBI group from Study 2 (*M* = 5.72, *SD* = 4.50; *t-difference* = .175, *p* = .547). Scores on the MoCa were relatively high for this subsample (*M* = 27, *SD* = 1.41), compared to the larger group from Study 2 (*M* = 23.06, *SD* = 3.99; *t-difference* = 4.038, *p* = .998). Although these scores do not differ statistically, the four individuals selected here represent a high functioning subset of the larger sample in Study 2.

State Measures

Boredom Probes

TBI participants reported feeling most bored in the boredom (*M* = 7.75, *SD* = 1.23; Figure 4.2) and resting state conditions (*M* = 6.50, *SD* = 3.79), which did not differ significantly from each other (*t* = .95, *p* = .412). The boredom condition approached significance relative to the interest condition (*M* = 4.50, *SD* = 3.32; *t* = 2.93, *p* = .061), whereas the rest condition was not significantly different from interest (*t* = 1.63, *p* = .201). Participants found the Starry Night condition (*M* = 3.75, *SD* = 1.26) the least boring, differing significantly from the boredom condition (*t* = 3.542, *p* =.004), whereas the difference in levels of boredom between the resting state and Starry Night conditions was not significantly different (*t* = 1.92, *p* = .151). Boredom levels between the Interest and Starry Night conditions also did not differ significantly from each other (*t* = .52, *p* =.638).

Mind-Wandering Probes

A similar pattern emerged with respect to the mind-wandering probes: TBI participants mind-wandered the most during the boredom (*M* = 6.25, *SD* = .96; Figure 4.2) and resting state (*M*= 6.50, *SD* = 2.52) scans. Again, these two conditions did not differ significantly from each other (*t* = -.293, *p* = .789). Participants mind-wandered the least during the Starry Night task (*M* = 2.75, *SD* = 3.10), which approached statistical significance relative to the boredom condition (*t* = 2.782, *p* =.069), was significantly lower than the rest condition (*t* = 3.174, *p* = .05), as well as the interest condition (*t* = 7.00, *p* = .006). The boredom and interest conditions did not differ significantly from each other (*t* = 1.698, *p* = .188), and neither did the interest and resting conditions (*t* = 1.852, *p* =.161).

Behavioural Data

To assess performance on the Starry Night task, mean reaction times within and between groups were compared, and accuracy proportions and sensitivity ratings (*d'*; proportion of Hit rate – proportion of False alarms; Swets, 1964) were calculated for both groups[12]. Results are presented in Table 4.1. Once again, caution must be taken in interpreting these results given the small sample sizes.

---

[12] Anticipatory responses (<150ms) and abnormally slow responses (>2000ms) were removed from the above analyses.

Table 4.1 Control and TBI Behavioural Data

		Controls		TBI			
		M	*SD*	*M*	*SD*	*t*	*p*
Reaction Time (ms)	A	526.5	102.51	457.39	60.47	1.60	.143
	D	550.16	115.04	444.71	52.19	2.43	.03
Accuracy (prop. correct)	A	.56	.19	.67	.18	-1.02	.328
	D	.48	.14	.72	.08	-3.12	.002
Sensitivity (*d'*)	A	2.06	.61	2.45	.43	-1.17	.204
	D	1.88	.44	2.52	.72	-2.14	.167

Note: A = Appearances; D = Disappearances

Reaction Time

Control participants were faster to detect appearance events relative to disappearance events, but the difference was not statistically significant (*t* = -1.315, *p* =.218; Table 4.1). TBI participants' results were in the opposite direction: they were faster to detect disappearances than appearances, but the difference was not statistically significant (*t* = .413, *p* = .707. Overall, the TBI group had shorter RTs for both appearances and disappearances; the difference between control and TBI RTs was significant for disappearances, but did not reach statistical significance for appearances.

Accuracy

Control participants were numerically more accurate in detecting appearance events relative to disappearance events, but the difference did not reach statistical significance (*t* = 1.458, p = .176. The TBI group showed the opposite pattern, with accuracy scores being the highest for disappearance relative to appearance events, however this difference did not reach statistical significance (*t* = -.521, *p* = .638). Between groups, TBI participants were more accurate in detecting both types of targets: while the proportions for appearance events did not differ significantly, the proportion of accurately detected disappearance events did.

Sensitivity

Control participants were numerically more sensitive to detecting appearance events relative to disappearance events, although they were not significantly different (t =1.096, p = .299). The TBI group's results showed a similar pattern to that seen above in the RT data: they were numerically more sensitive to detecting disappearance events relative to appearance events, although this difference was not statistically significant either (t = -.23, p = .833). Contrasting groups, TBIs showed numerically higher sensitivity ratings for both appearances and disappearances relative to controls: for appearances, this difference was not statistically significant; and for disappearances, the difference in sensitivity between controls and TBI was not significant either.

fMRI

Control Participants

Three independent component (IC) clusters corresponding to BOLD signals were identified for each of the Boredom, Resting State, Starry Night and Interest scans. Spatial patterns for these clusters were consistent across the majority of participants (Figures 4.3 and 4.4; Tables 4.2 – 4.5). All four scanning conditions revealed a cluster containing regions associated with DMN structures, a cluster comprised of visual network structures, and a third cluster comprised of (mainly) executive network structures, although these clusters were not consistent across conditions. Regarding the DMN cluster, a large, bilateral region of activation in the posterior cingulate and adjacent precuneus was observed. Smaller regions of activation in superior and inferior regions of parietal cortex were also evident, along with bilateral medial prefrontal gyrus (Tables 4.2-4.5, Figure 4.3). Anticorrelated activation was also observed in this cluster, mainly in dorsal portions of frontal cortex and less consistently in the parietal cortices, across all functional runs (Figure 4.3). It was also observed that bilateral insular cortex was anticorrelated in the boredom, resting state, and in the Starry Night task, and correlated for the Interest condition[1313] (no insular activation was observed for the Starry Night scan; Figure 4.4). All four conditions also revealed a cluster comprised of activation of visual networks: these clusters revealed bilateral activation of the occipital lobe, including the cuneus and adjacent lingual gyri (Tables 4.2-4.5). The third cluster observed for each functional scan, albeit more inconsistently across conditions, was comprised mainly of bilateral frontal regions with smaller, unilateral parietal, temporal, and occipital regions (e.g., in the boredom scan regions comprised superior frontal gyrus, superior parietal lobule and occipital cortex, in the interest and resting state scans clusters involved central executive regions, and in the Starry Night scan clusters included prefrontal and middle temporal gyrus). Details pertaining to each cluster and each condition are presented in Tables 4.2 to 4.5.

---

[13] As depicted in Figure 4.4, for the interest condition, insular activation was largest in the L hemisphere, about 4 times the size of activation in the R hemisphere.

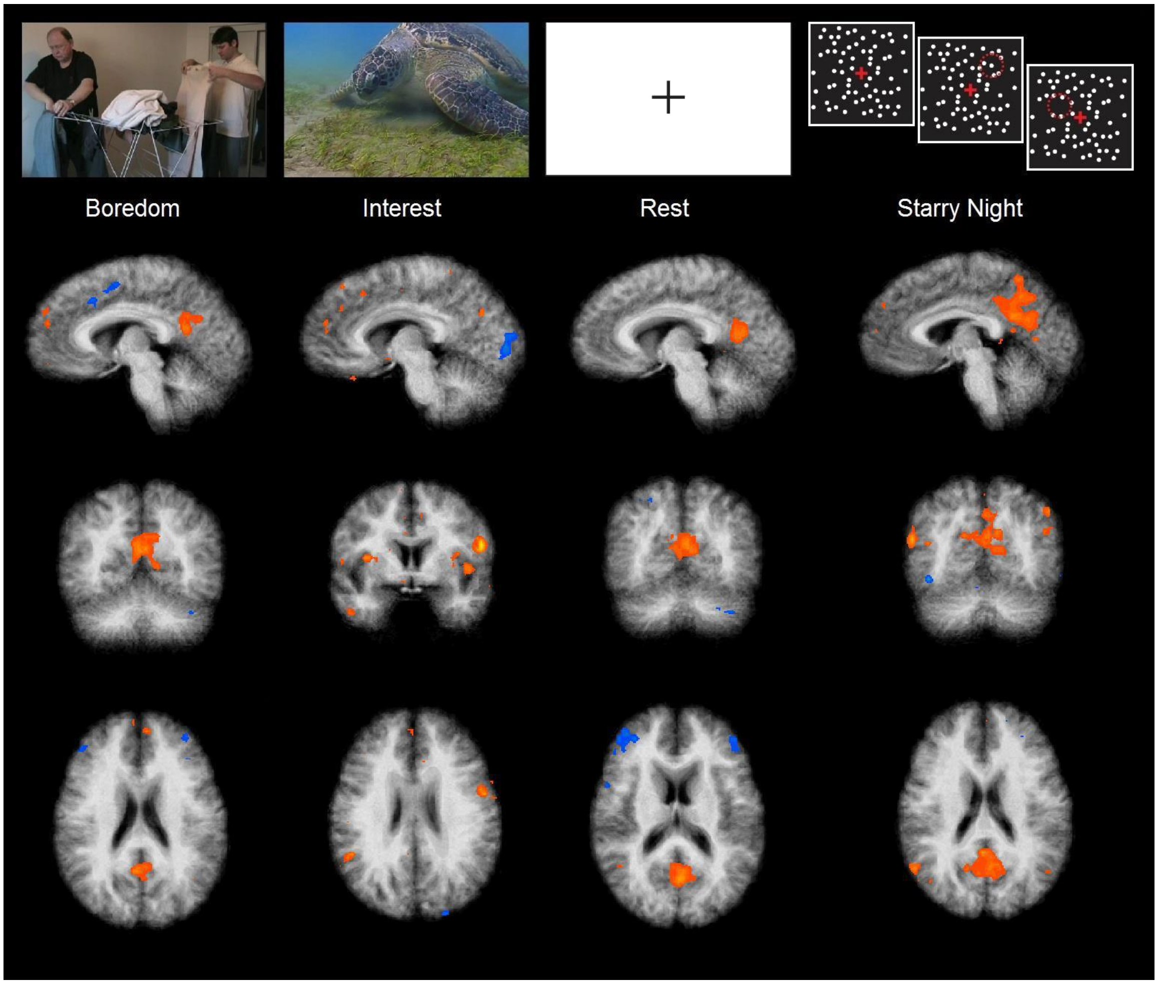


Figure 4.3. Controls: Network activation patterns observed in all four scanning conditions.

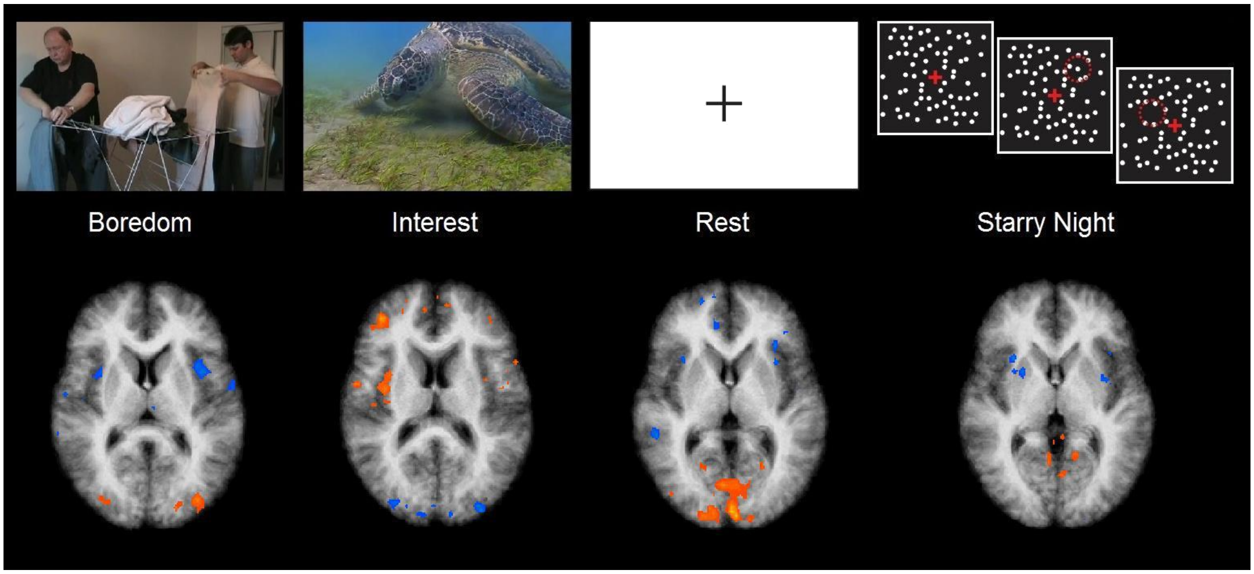


Figure 4.4. Controls: Insular activation observed during each scanning condition.

Table 4.2. Independent component (IC) clusters corresponding to the Boredom scan for the Control group

				Centroid			Size	
Network	Polarity	Region	BA	x	y	z	(voxels)	Similarity M
Visual	Correlated	Cuneus/LG	19/18	0	-69	4	24650	0.29
Default	Correlated	PCC	23	-3	-53	19	3234	0.19
	Correlated	IPL	40	-39	-37	40	887	
	Anticorrelated	R SFG	8	20	57	10	841	
	Anticorrelated	Insula	13	35	1	2	912	
	Anticorrelated	MFG	46	32	30	37	328	
	Anticorrelated	mPFC	10	0	9	45	1628	
	Anticorrelated	mPFC	9	39	31	31	635	
Executive/Visual	Correlated	MOG	19	36	-69	7	7900	.21
	Correlated	SPL	7	-27	-61	49	3135	
	Anticorrelated	SFG	8				328	

Note: IPL=inferior parietal lobule; LG = lingual gyrus; MFG = middle frontal gyrus; mPFC = medial prefrontal cortex; PCC=posterior cingulate cortex; SFG = superior frontal gyrus; SPL = superior parietal gyrus. Neural activation is bilateral unless otherwise indicated.

Table 4.3. Independent component (IC) clusters corresponding to the Interest scan for the Control group

Network	Polarity	Region	BA	Centroid x	 y	 z	Size (voxels)	Similarity M
Visual	Anticorrelated	MOG, LG	19/18	30	-85	-1	3636	0.29
Default	Correlated	SPL	7	30	-71	35	2607	0.16
	Correlated	Precuneus	31	-1	-59	30	1136	
	Correlated	Insula	13	13	0	11	900	
	Correlated	MFG	46	38	41	13	809	
	Correlated	IPL	40	50	-45	28	474	
	Correlated	R mPFC	10	4	44	27	455	
	Correlated	R Precentral Gyrus	4	53	-7	18	333	
	Anticorrelated	R Transverse Temporal Gyrus	41	53	-8	8	1652	
Executive	Correlated	R mPFC	9	5	45	29	917	0.16
	Correlated	mPFC	10	2	51	14	301	
	Anticorrelated	SPL	7	-24	-48	54	564	

Note: MOG = middle occipital gyrus

Table 4.4. Independent component (IC) clusters corresponding to the Resting state scan for the Control group.

System	Polarity	Region	BA	Centroid x	 y	 z	Size (voxels)	Similarity M
Visual	Correlated	Cuneus/LG	19/18	0	-72	-1	15892	0.22
Default	Correlated	PCC	23	0	-56	13	4500	0.22
	Anticorrelated	MFG	46	41	38	20	2928	
	Anticorrelated	R IPL	40	50	-37	45	583	
	Anticorrelated	R IFG	44	52	8	19	541	
	Anticorrelated	R MTG	21	57	-50	-8	405	
	Anticorrelated	Insula	13	-34	19	5	376	
Executive	Correlated	mPFC	9	3	50	17	5488	0.15
	Correlated	MFG	46	-28	33	39	701	

Note: IFG =inferior frontal gyrus; MTG = middle temporal gyrus

Table 4.5. Independent component (IC) clusters corresponding to the Starry Night scan for the Control group.

Network	Polarity	Region	BA	Centroid x	 y	 z	Size (voxels)	Similarity M
Visual	Correlated	Cuneus/LG	18/19	0	-60	-3	15137	0.25
Default	Correlated	PCC/Precuneus	23, 31, 7	-3	-53	25	10780	0.24
	Correlated	L Angular Gyrus	39	-46	-66	30	1428	
	Anticorrelated	R IFG	11	50	6	28	491	
	Correlated	STG	22	53	-58	18	486	
Executive	Anticorrelated	MTG	21	-60	-36	1	995	0.20
	Correlated	mPFC	11	-4	41	-12	828	
	Correlated	IFG	47	21	23	-16	467	

Note: IFG = Inferior Frontal Gyrus; STG = Superior Temporal Gyrus

TBI Participants

First, a group-level ICA was conducted for the TBI participants (DeMartino et al., 2007). Contrary to controls, IC clusters and spatial patterns for those clusters were heterogeneous across conditions in the four TBI participants, likely due to individual differences not evident in controls. Relative to the large areas of activation evident in the control sample (Figures 4.3 and 4.4), the TBI group demonstrated many more regions of activations of much smaller volumes across conditions at the same threshold (Figures 4.5). For instance, the largest clusters in the control data showed contiguous regions of activation ranging from 2500-24000 voxels; contiguous regions of activation in the TBI brains across conditions were much smaller, ranging from a few hundred to ~1000 voxels in size (contrast Figures 4.3 and 4.5).

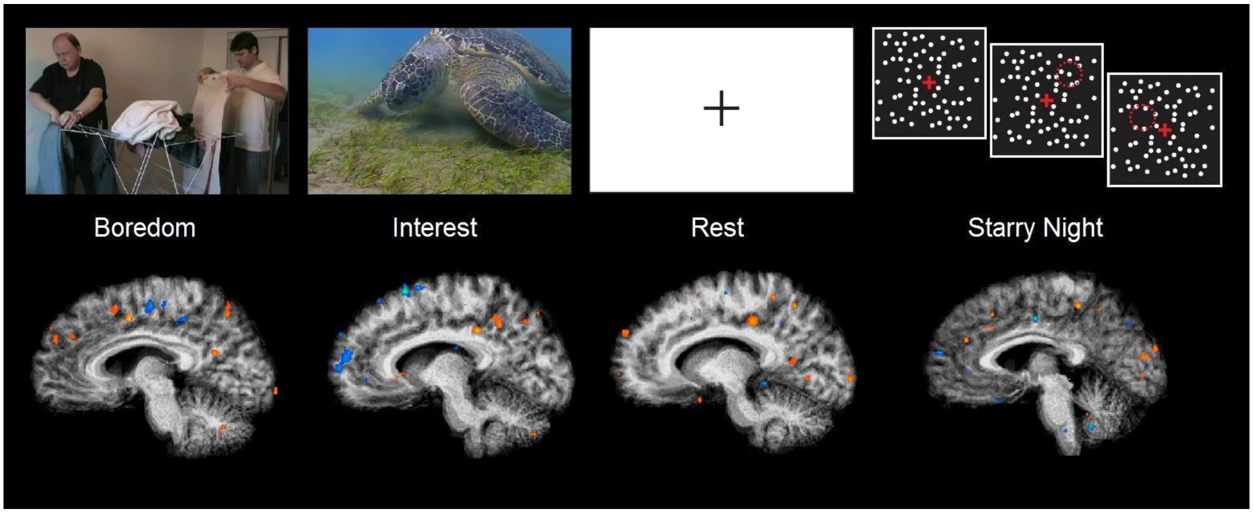


Figure 4.5. TBIs: BOLD network activation patterns observed for each scanning condition.

Given the heterogeneous nature of TBI injuries, and the small sample size, each individual's single-subject ICAs were inspected separately to determine whether or not this 'patchy' pattern of activation observed at the group level was evident at the individual level. Below, results are reported descriptively (but not exhaustively), focusing on two major posterior components of the DMN evident in the controls: the PCC and the precuneus (Figure 4.6) as well as the insular cortex. Individual neural activation patterns are presented in Figures 4.7-4.10 for each scanning condition[14].

---

[14] At the same threshold of activation used in the control sample, individual TBI participants still presented with a far higher number of active regions of generally smaller size. For example, TBI participant 1 demonstrated 54 regions of activity in the boredom scan, where only 11 regions were active for the control group. For this reason, I have chosen not to present exhaustive tables of active regions per TBI participant.

Figure 4.6. Posterior Cingulate Cortex (green) and Precuneus (orange).

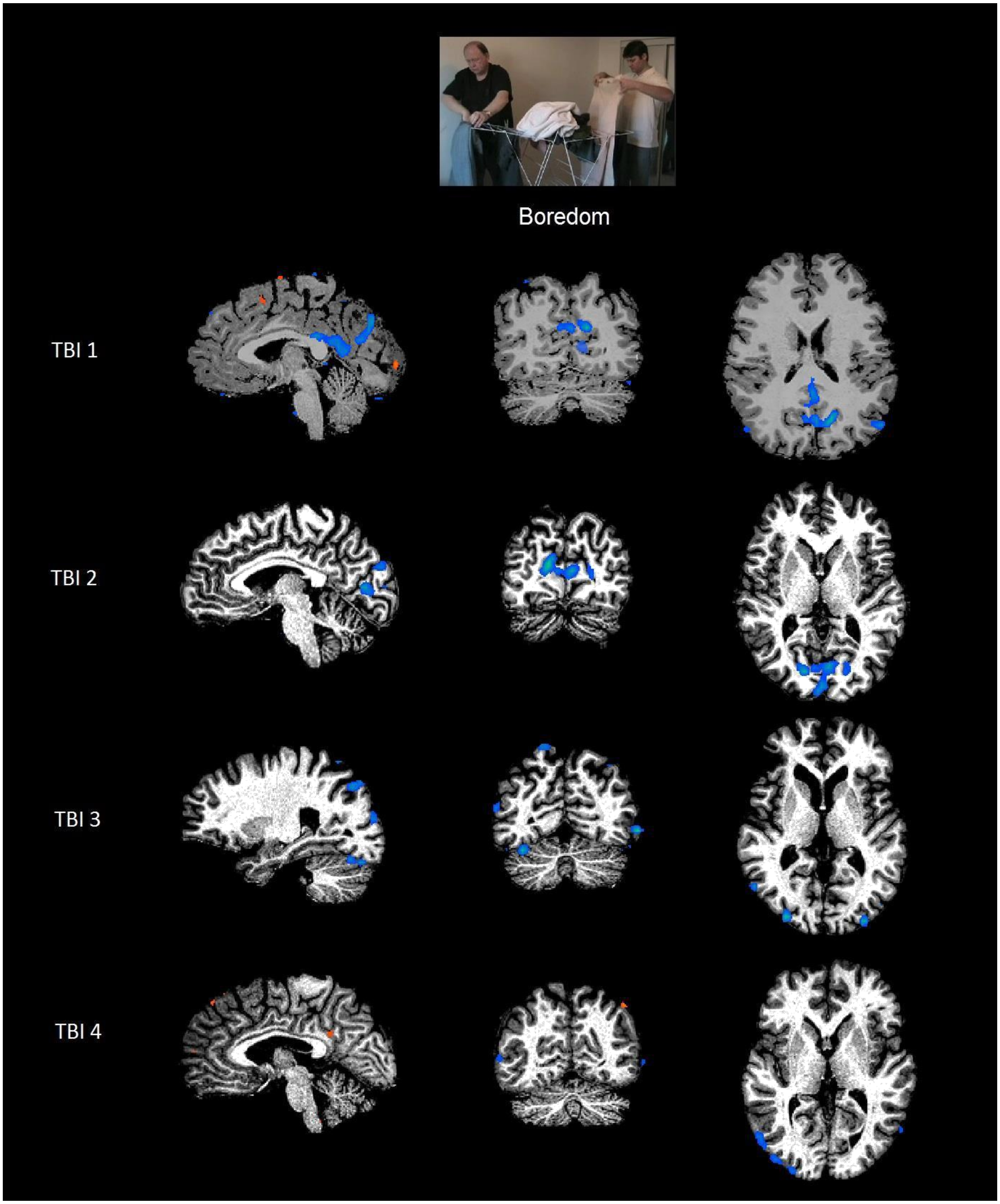


Figure 4.7. Boredom: Individual TBI participants' network activation patterns.

In the boredom condition (Figure 4.7), the TBI participants showed differential neural activation patterns in DMN regions. The PCC was anticorrelated in participant 1 (2035 voxels), and not evident in any other participant. The precuneus was anticorrelated in participants 1 (2274 voxels) & 3 (293 voxels), and in the former the region of activation is 7x larger than the activation seen in participant 3. Pertaining to the insula, only participant 3 showed a very small (115 voxels; L hemisphere) region of anticorrelated activation; participants 1, 2 and 4 displayed no insular activation (or deactivation) in the boredom condition.

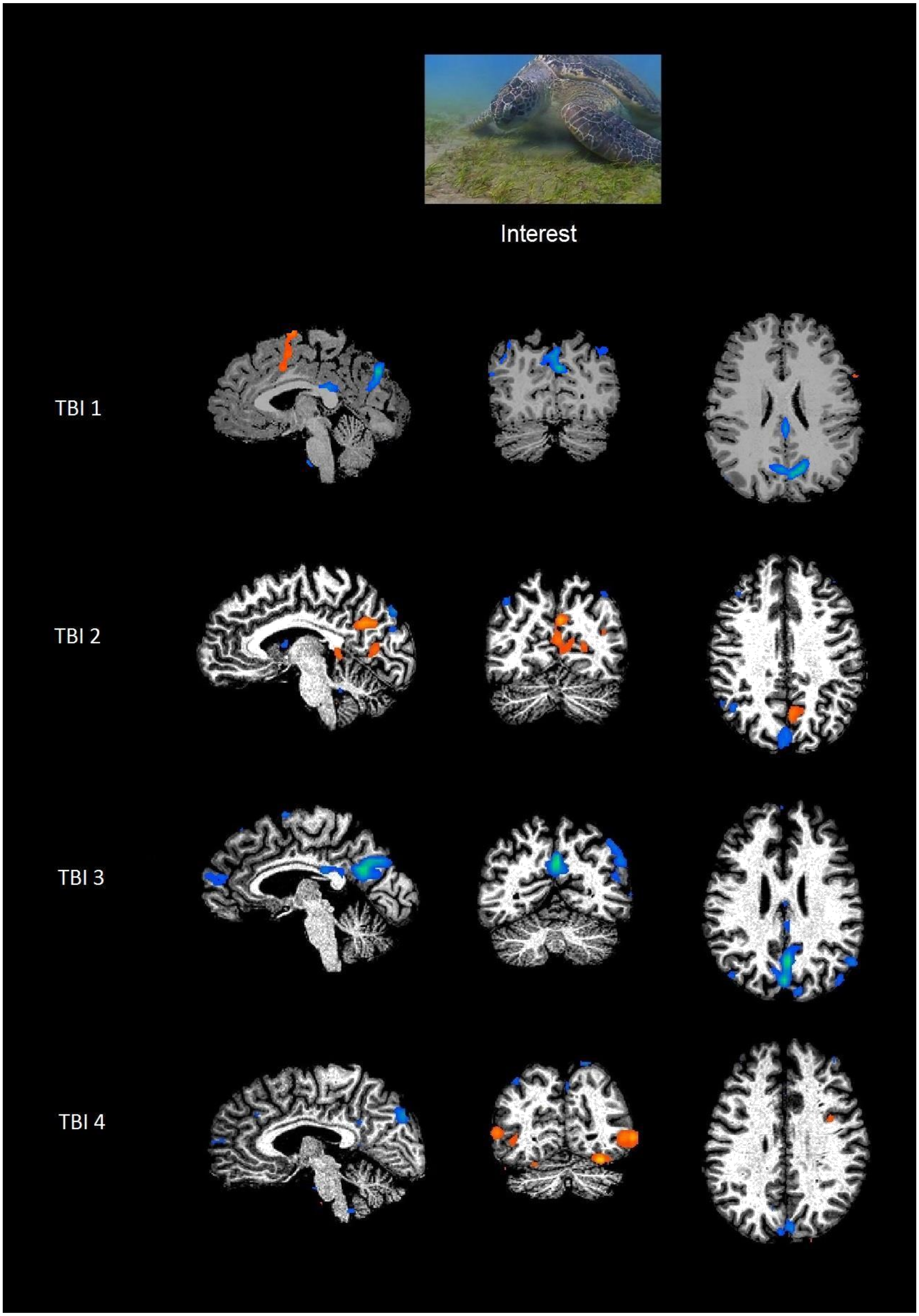


Figure 4.8. Interest: Individual TBI participants’ network activation patterns

In the interest condition (Figure 4.8), the PCC showed correlated activation in participant 2 (319 voxels), anticorrelated activation in participants 1 (276 voxels) and 3 (318 voxels), and no activation in participant 4. The precuneus showed equally heterogeneous activation patterns: participants 1 (2853 voxels) and 3 (3518 voxels) displayed anticorrelated activation, whereas participant 2 showed both correlated (1209 voxels; L hemisphere) *and* bilateral anticorrelated (722 voxels) activations in distinct subregions of the precuneus. No precuneus activation (or deactivation) was observed in participant 4. No insular activation (or deactivation) was observed for any of the participants in this condition.

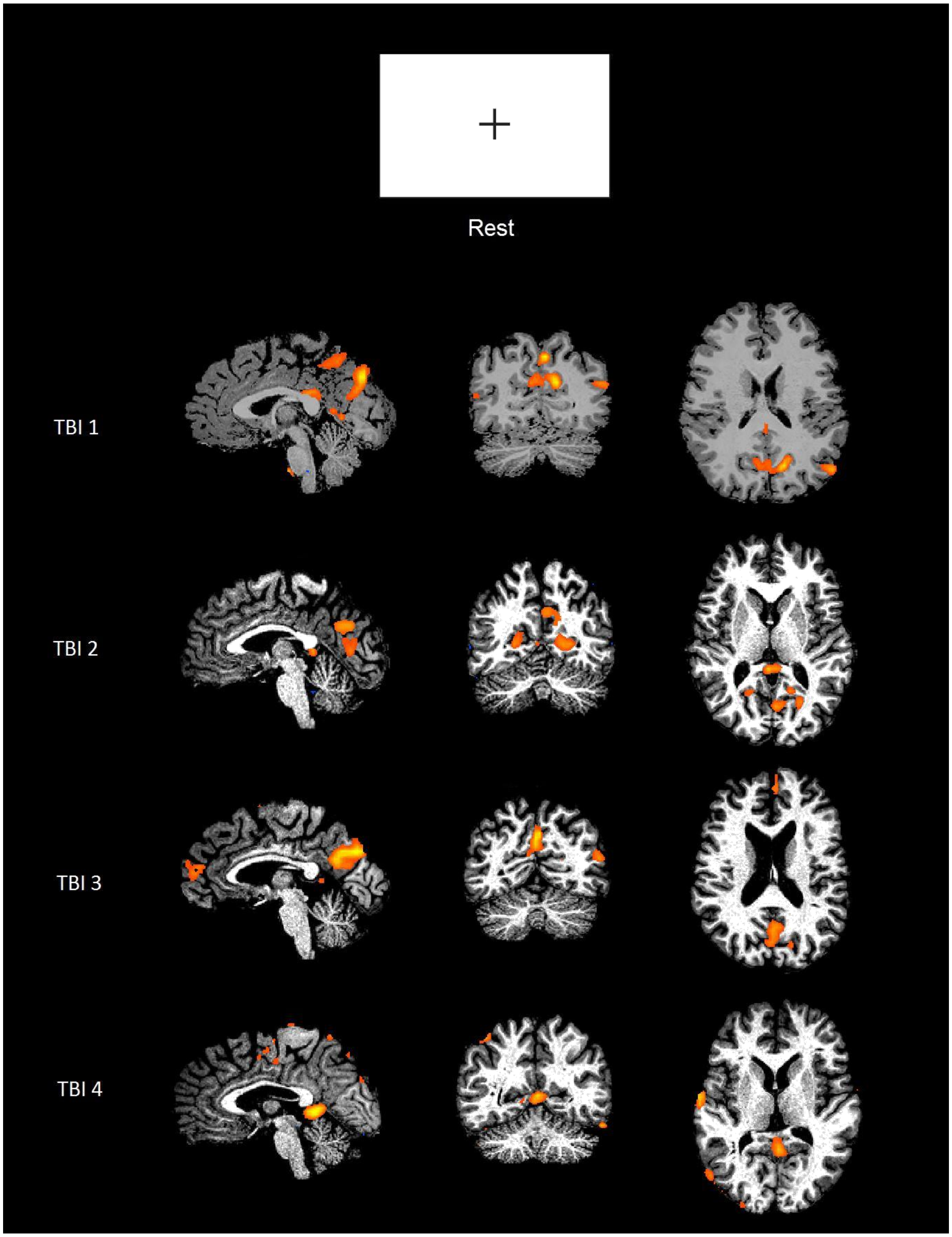


Figure 4.9. Resting State: Individual TBI participants' network activation patterns.

In the resting state condition (Figure 4.9), activation patterns for the TBI participants more closely resembled those of controls (Figure 4.3). Participant 1 was the only TBI individual to show correlated activation in the PCC (345 voxels), whereas for the precuneus large areas of correlated activation were observed in all but participant 4 (ranging from 1438 voxels in participant 2 to 6702 voxels in participant 1). No insular activation (or deactivation) was observed for any of the participants in this condition.

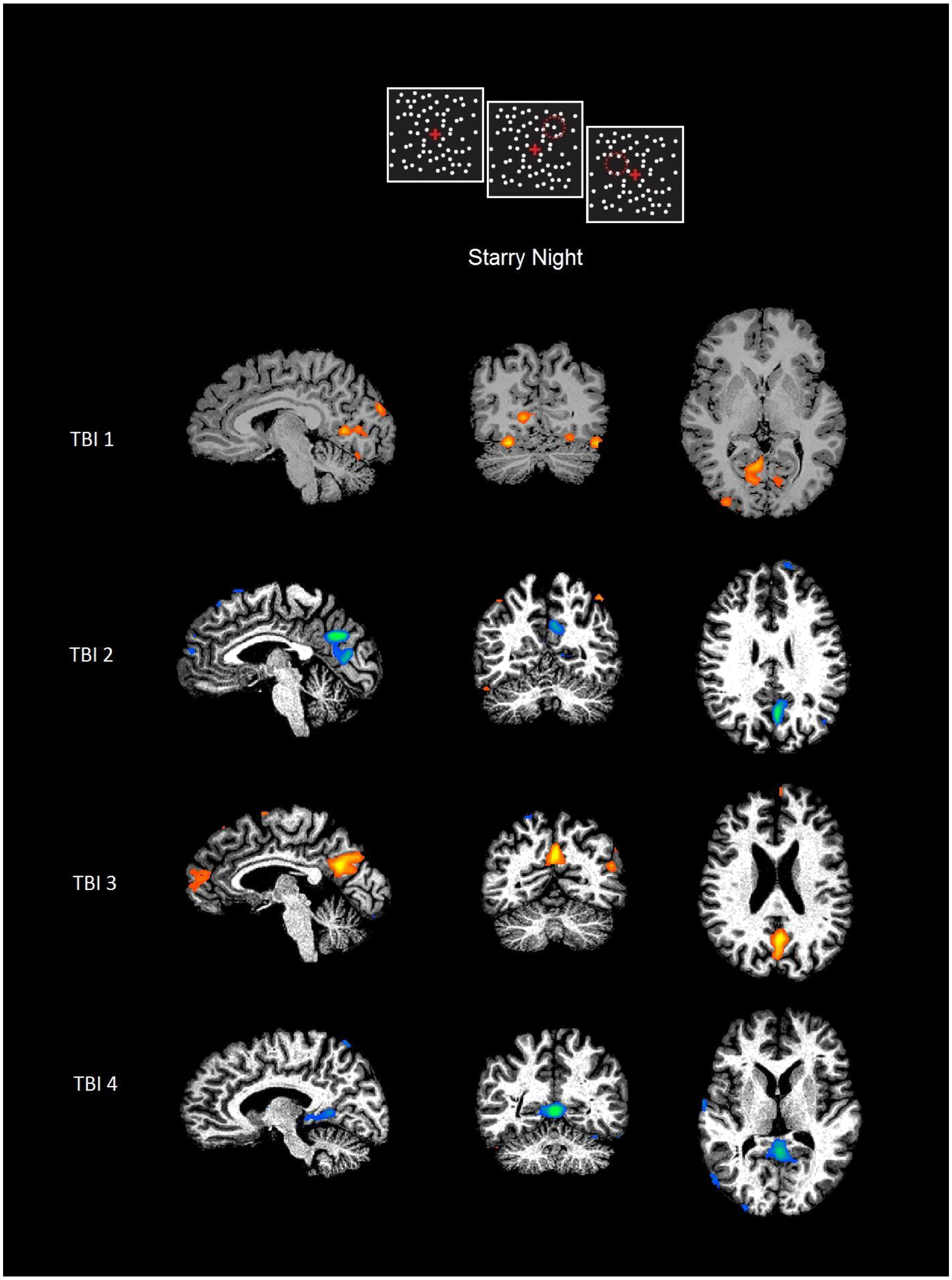


Figure 4.10. Starry Night: TBI participants' network activation patterns.

Finally, in the Starry Night condition (Figure 4.10), again there were notable differences between TBI participants. No activation (or deactivation) of the PCC was observed in any of the participants. Regarding the precuneus, correlated activation was observed in participant 1 (121 voxels) and 3 (3984 voxels), and participant 4 showed anticorrelated activation (100 voxels). Participant 2 showed both correlated activation (254 voxels; R hemisphere) *and* anticorrelated (2458; L hemisphere) in distinct subregions of the precuneus. No insular activation (or deactivation) was observed for any of the participants in this condition.

4.4 Discussion

The aims of this study were twofold: first, to replicate previous findings concerning the neural networks associated with state boredom in healthy adults; and second, to extend this investigation to include individuals with TBI. While bored, control participants exhibited correlated activation in DMN regions including the PCC, precuneus, and medial prefrontal cortex, with concurrent anticorrelated regions including the anterior insula. These results replicate those observed in a previous investigation (Danckert & Merrifield, 2016). When exploring the patterns seen in the other conditions there were some notable differences between the two studies. For instance, in the previous study, anticorrelated insular activation of approximately equal magnitudes was observed in the Boredom and Starry Night Scan, with no insular activation (or deactivation) in the resting state scan. Here, the largest anticorrelated insular region was observed in the boredom scan (912 voxels), followed by the resting state (376 voxels), and Starry Night conditions (77 voxels[15]). The interest condition here revealed correlated insular activation (900 voxels), in the opposite direction, but of roughly equal magnitude as that seen in the boredom scan. The widespread activation in DMN regions across conditions suggests that this network is strongly

---

[15] Insular activation for the Starry Night task was not included in Table 4.5 as it was below a threshold set for meaningful activation. That threshold was a contiguous cluster of 100 voxels.

involved in the experience of boredom, whereas the differential insular activations suggest potential varying levels of disengagement across conditions.

In TBI participants, results of the group analysis were heterogeneous. Overall, there was not the same level of consistency in the large scale networks that were evident in the healthy brain. Instead, numerous, very small regions of activation that spanned most of the brain were observed across all conditions (Figure 4.5). When viewed at the individual level, regions of activation were more robust in terms of the magnitude of activations (i.e., the size of contiguous clusters were larger at the individual level), but continued to exhibit considerable heterogeneity in terms of the precise regions activated and the direction of that activation. For example, although all TBI participants showed activation of DMN regions, the precise locations of those activations and whether they exhibited correlated or anti-correlated activity varied widely (Figures 4.7-4.10). This level of heterogeneity was evident in all conditions with the possible exception of the resting state scan. Here, individual TBI activation patterns more closely resembled those of controls, exhibiting large-scale network connectivity in posterior regions of the DMN (Figure 4.9).

Both control and TBI participants reported being significantly more bored during the boredom and resting state scans, relative to the interest mood induction and sustained attention task. In a similar fashion, mind-wandering was highest during the boredom and resting state scans, relative to the interest and sustained attention task. This positive correlation is in line with what was observed in Studies 1 and 2 of the relationship between boredom and mind-wandering. Despite reporting lower boredom and mind-wandering during the sustained attention task, when behavioural data was examined, data showed that controls performed more poorly than the TBI participants. TBI participants in this investigation were more accurate in detecting appearing and disappearing stimuli, and their sensitivity measures were higher than those for controls (Table 4.1). The Starry Night represents a difficult vigilance task in which attentional capacities are taxed as stimuli are small and difficult to detect. However, for healthy adults to take between 500-600ms per target is slow relative to other published work using this task (e.g., Deouell, Sacher & Soroker, 2005 demonstrate typical RTs around 350-450ms). While performing tasks in the magnet may add some amount of non-specific processing time simply due to the unusual nature of the environment, this would not explain the difference between controls and TBI individuals seen here. It is plausible that the difference in reaction times between controls and TBI participants can be attributed to motivation: TBI participants were potentially more motivated to perform well related to a phenomenon known as 'diagnosis threat' (Ozen & Fernandes, 2011). That is, TBI participants are motivated to perform as well as possible to avoid any diagnosis of impairment. Clearly, more research with a larger sample of TBI participants is warranted and perhaps some external motivation for controls to ensure optimal performance.

In healthy brains, when one is unengaged with external stimuli and engaged with internally-generated thoughts, DMN structures have been shown to become active while regions responsible for executive functions deactivate (Greicius et al., 2003; Mason et al., 2007; Sherman et al., 2014). The fact that large-scale correlated activation in DMN regions with simultaneous anticorrelated executive network regions were observed in the most boring conditions is perhaps not surprising. Of note is that similar large-scale DMN regions were found in the Starry Night condition, the condition in which participants reported being bored the least. Had participants been as engaged in the task as they reported having been, one would expect to see more executive or attentional network activation – instead, at the group level for controls, the Starry Night condition showed large DMN network activation, similar to the scope of contiguous voxels observed in the resting state scan. So while this condition was rated the least boring it may nonetheless have been insufficiently engaging to demonstrate upregulation of attentional and executive control networks.

One of the more interesting findings in this investigation pertains to the differential insular cortex activation across conditions. The insular cortex has been thought to be essential in switching between internally and externally generated cognition, playing a crucial part in recruiting brain networks necessary for these functions (Gao & Lin, 2012; Menon & Uddin, 2010; Seeley, et al., 2007; Spreng, Sepulcre, Turner, Stevens, & Schacter, 2013; Sridharan, Levitin, & Menon, 2008). Furthermore, the insula has been implicated in switching between executive and default-mode networks in the presence of salient stimuli (Menon & Uddin, 2010). In controls, bilateral anticorrelated activation of the insular cortex with concurrent activation of DMN structures were observed in the boredom, resting state, and minimally in the sustained attention task. Intriguingly, the area of insular activation observed in the boredom induction was more than double that seen in the resting state, and more than 10 times the area activated in the sustained attention task. In the interest condition, activation of the insula was of a comparable size to the boredom condition, but in the opposite direction. The deactivation of the insular during the boredom and resting state scans may be unsurprising given that both conditions involve little (boring movie) to nothing (resting state fixation screen) for the participant to engage with – in other words, there is nothing salient to respond to. On the other hand, the Starry Night task, where participants ought to have been engaged with the demands of the task, showed minimal insular activation. Given that participants reported less boredom in the sustained attention task, a minimally deactivated insula might reflect successful engagement, although not to the degree of engagement observed in the interest condition. Here, the dynamic and changing nature of the interesting video provided ample content to engage with leading to prominent insular and executive network activations.

The original study examining the networks of brain activity associated with boredom found no insular activity in the resting state scan, and comparable deactivation of the insular in the boredom and Starry Night conditions (Danckert & Merrifield, 2016). It is plausible that the differences observed between these two samples could be attributed to sex. A recent meta-analysis investigating insular activation with respect to affective processing has shown differential activation and lateralization between the sexes. For instance, women tend to activate bilateral anterior insula and the left mid and posterior insula when presented with emotional stimuli, whereas males primarily activate the left anterior/mid-insula and right posterior insula (Duerden, Arsalidou, Lee & Taylor, 2013). The current sample tested here was largely comprised of women (8:5), whereas the previous study predominantly involved males (8:2; Danckert & Merrifield, 2016). Perhaps the discrepancy in insular activation while bored between these two samples is reflective of differences in how men and women respond to boredom. As seen in Studies 1 and 2, sex differences were also observed with respect to boredom proneness – with women exhibiting lower levels of boredom proneness. Perhaps one of the key differences in the experience of boredom between women and men pertains to the way in which the insular cortex is recruited during monotonous experiences. Clearly, more research is necessary to investigate this hypothesis.

With respect to individual activation patterns in TBI patients, results were quite heterogeneous. The TBI group showed a large degree of variation with respect to number and size of regions activated relative to the control sample. Furthermore, individual data also showed dramatic differences in which regions showed correlated or anticorrelated activity across conditions, true of all but the resting state, which most closely resembled the pattern observed for the control group. Regarding insular activation, only one TBI patient showed any activation – anticorrelation within a small portion (115 voxels) of the left insular cortex – during the boredom mood induction. Relative to controls, where robust deactivation of the insular cortex in the boredom induction and activation in interest condition, very little evidence for either was observed in the TBI patients. Clearly, more research is needed to better understand the role the insular cortex plays in boredom in TBI. Collectively, these results suggest that analyzing TBI participants at the group level might prove problematic given the heterogeneity of TBIs with respect to neuropathology. Such heterogeneity in imaging data for TBI patients is reported often in a variety of other paradigms (Chiou, Genova & Chiaravalloti , 2015; Zhang, Puvenna & Janigro, 2016). The injuries themselves tend to be heterogeneous, with such variance only compounded by variability in patterns and capacity for recovery (Andruszkow et al., 2014; Carney et al, 1999; Cassidy et al., 2014; Di Battista, Godfrey, Soo, Catroppa & Anderson, 2014; Kim et al, 2007; Nicholl & LaFrance, 2009)

This investigation sought to replicate previous findings concerning the neural networks associated with state boredom in healthy adults, and to extend this investigation to include individuals with TBI. Regarding the first aim, data largely replicated previous findings that large-scale DMN regions are activated during a boredom induction task, a resting state, as well as a sustained attention task. In these conditions, regions within the DMN showed widespread activation that coincided with deactivation of executive regions including the insular cortex, most prominently in the boredom induction. Regarding the second aim, this investigation represents the first steps in assessing the neural underpinnings of boredom in TBI. Results were largely heterogeneous across participants with DMN activation differing not only between conditions, but between patients as well, highlighting the difficulty in assessing functional neural networks in this population. Future investigations, perhaps utilizing an event-related design and a larger patient sample could better elucidate the temporal dynamics of how DMN structures activate in conjunction with executive regions over time, and how these de/activation patterns differ as a function of head injury.

Chapter 5: General Discussion

Boredom proneness is a ubiquitous human experience that has the potential to impact one's cognitions, affect, and behaviour. The work presented in this thesis examined the cognitive and affective components of boredom through a lens of self-control, to better understand the underlying process that makes boredom proneness such a pervasive problem. This line of investigation is especially important in individuals who have suffered from traumatic brain injuries in whom boredom is likely to represent a serious impediment to rehabilitation.

Study 1 explored a series of cognitive and affective factors shown to be involved in boredom proneness, and sought to better understand the degree to which individual levels of self-control influence the relationships between these factors. Results showed that in healthy individuals, boredom proneness was strongly positively correlated with spontaneous mind-wandering, depression, and hostility, which in turn all acted as significant positive predictors of boredom proneness. Deliberate mind-wandering, on the other hand, did not significantly predict boredom proneness scores, hinting at an underlying problem with lack of control over one’s own cognitions. That is, the less control one has over one’s own cognitions (i.e., the more spontaneous mind-wandering one experiences), the more likely one is be boredom prone. Albeit speculative, it may be the case that, deliberate mind-wandering represents coping mechanism to avoid boredom. That is, by deliberately engaging with one’s own cognitions, one prevents boredom proneness. Indeed, individual differences in self-reported levels of self-control functioned as the only negative predictor of boredom proneness; and, when individual levels of self-control were taken into account there was a substantial drop in the magnitude of the relationships observed between boredom and the cognitive and affective predictors of the trait – a drop of between ~20 and 65%.

Collectively, these results suggest that the more adept one is at controlling one's own thoughts, emotions, and actions in general, the more effective they will be in goal pursuit and the less likely they are to be boredom prone.

Study 2 built on this work by extending the investigation to include individuals who have sustained varying levels of TBI. The hallmark of pathology in TBI pertains to extensive axonal injury, severing vital connections throughout the brain. As a result of such pervasive damage, TBI patients experience a host of cognitive and affective dysfunctions post-injury. In this investigation, results showed, for the first time, that boredom proneness also increases as a function of head injury. That is, relative to healthy controls, individuals with a past history of concussion showed elevated levels of boredom proneness, with TBI patients demonstrating the highest levels of boredom proneness relative to both groups. Results pertaining to individual levels of self-control were more variable. For instance, while individuals with a history of concussion reported lower levels of self-control relative to controls, TBI reports did not follow suit. That is, the most impaired individuals reported having more self-control than individuals with less brain damage (i.e., concussion). This may reflect differences in age (i.e., the TBI group were in general older than either the controls or concussed group) or may be reflective of differences in insight. That is, given what is known in the literature about the difficulties TBI patients face with various aspects of self-regulation, perhaps it is the case that individuals with the most severe head injuries lack the most insight into their own deficits, which may partially explain the discrepancies in self-reported levels of self-control. Indeed, a lack of insight has been reported often in the TBI literature (McAvinue, O'Keeffe, McMackin& Robertson, 2005; O'Callaghan, McAllister & Wilson, 2012; O'Keeffe, Dockree & Robertson, 2004; O'Keeffe et al., 2007; Prigatano, 1996), rendering self-report data difficult to interpret in this population. There may also be demand characteristics at play. Patients may be motivated to present themselves in a positive light. While this might explain the self-control data it would fail to capture the self-reports of levels of boredom proneness if one assumes that it is always desirable to report *low levels* of any kind of negative affective state. Despite this, investigations on the impact of boredom proneness in this demographic is of vital importance as disengagement from one's environment is likely to undermine any efforts a patient invests into one's own rehabilitation, *especially* if insight into one's own deficits is lacking. Therefore, this line of investigation ought to be addressed in future research studies, where more objective metrics of self-control can be employed (e.g., using tasks such as the Stroop or go/no-go tasks), or with the addition of care-giver/spousal reports that could corroborate (or not) the patient data.

Finally, Study 3 examined the relationship between boredom and activity in the DMN in a sample of healthy controls, and a small sample of TBI patients. The DMN is a set of brain regions that have been consistently linked off-task processing and internally generated cognitions (Binder et al., 1999; Buckner et al., 2008; Christoff, 2011; Christoff et al., 2009; Gusnard & Raichle, 2001; Mason et al., 2007; Schooler et al., 2011). In healthy controls, consistent large-scale activation of the DMN was observed in a series of boring tasks, except the interest scan in which the DMN exhibited smaller-scale activation patterns. Even the sustained attention task, a task in which participants ought to be engaged with onscreen stimuli showed systematic DMN activation in controls. It could be the case the difficult nature of this attentional task, in which changes are very small and difficult to detect, despite requiring participants to engage, fails to provide enough content for participants to engage with. Given that these regions are implicated in states of disengagement and mind-wandering, this finding is in line with those of Studies 1 and 2, suggesting that individuals who tend to engage in more mind-wandering and have difficulty sustaining their attention when bored. In addition, bilateral activation in the insular cortex was observed to be anticorrelated with DMN activity during the boredom, resting state, and sustained attention scans, whereas in the interest condition showed correlated insular activation. These findings could be indicative of boredom interfering with the normal function of the insula which is to switch between the DMN and executive networks when attention is required to be directed externally (Gao & Lin, 2012; Menon & Uddin, 2010; Seeley et al., 2007; Spreng et al., 2012; Sridharan et al., 2008). Regarding the sustained attention task, had participants engaged with the task, one would expect insular activation, not deactivation to occur; however, as mentioned previously, it is plausible that the monotonous nature of the task renders it difficult to engage with. In TBI patients, results were much more heterogeneous. DMN structures were observed to be active (and inactive) for patients between conditions, albeit inconsistently so. For instance, in the boredom condition, while some patients showed activation of DMN structures, other showed deactivation of the same regions. The only condition in which activation patterns of TBI individuals resembled those of controls was the resting state scan. This level of heterogeneity was evident in all scanning conditions and highlights the difficulty of imaging a very heterogeneous sample of individuals.

This body of work is not without limitations. In studies 1 and 2, correlational analyses were utilized and the results do not allow one to infer causation. Utilizing experimental manipulations that require self-control on the part of the participant may shed some light on whether or not low levels of self-control and high levels of boredom proneness have explicit behavioural consequences. For instance, Go/No Go tasks that require participants to inhibit responses (Robertson, Manly, Andrade, Baddeley & Yiend,

1997) may shed light on behavioural tendencies of those prone to boredom prone: it is possible that high boredom prone individuals struggle to inhibit responses and make more errors relative to individuals who are not chronically bored (Hunter & Eastwood, 2016; Malkovsky et al., 2012). Second, the cognitive and affective constructs measured throughout this thesis are unlikely to function in a unidirectional manner. Instead, boredom proneness and measures of cognition and affect likely interact in dynamic ways. For instance, it is possible that the propensity to experience boredom may lead to attentional difficulties, as well as negative affective states. Furthermore, the controls in study 3 were not matched on sex (quite the opposite) with the control sample of the previous imaging study. This difference in ratio between women and men could explain the differential insular activation patterns observed in the two studies. This shortcoming provides an opportunity for future investigations to assess sex differences of cognitive and affective contributors to boredom proneness more directly. Finally, the use of a general measure of self-control is associated with inherent limitations; it is not possible with this measure to separately parse out the cognitive, affective, and behavioural aspects of self-control, nor does it provide any information pertaining to how high levels of self-control are achieved. It could be the case that individuals who exhibit high levels of self-control also exhibit differing neural patterns relative to individuals with poor self-control. Future research could address these potential differences by using self-control measures specifically designed to assess the cognitive, affective, and behavioural subcomponents of self-regulation, experience sampling in fMRI, caregiver reports in TBI to account for self-control discrepancies, or a longitudinal approach investigating the developmental trajectories of self-control and boredom proneness across the lifespan.

Despite the above shortcomings, data presented in this thesis provide new insights to understanding boredom proneness. Collectively, these data suggest that boredom proneness is strongly related to dysregulation of cognition and affect, and that individual levels of self-control can account for a significant proportion of variance in these relationships. That is, the higher an individual's capacity to regulate one's own cognitions, emotions, and behaviours, the less prone the individual is to boredom. The same relationships held true in a sample of individuals with varying degrees of head injury, and for the first time, empirical data has focused on exploring the problem of boredom proneness in this population. Results suggest that the presence and severity of head injury will likely be associated with increased susceptibility to disengage from one's environment, further complicating a patient's road to recovery. This work represents some of the first steps towards better understanding the underlying mechanisms in boredom proneness in healthy and traumatic brain injured people, and suggests that, in general, boredom proneness is associated with difficulties in regulating one's own cognitions, emotions, and behaviours.

References

Amyot, F., Arciniegas, D. B., Brazaitis, M. P., Curley, K. C., Diaz-Arrastia, R., Gandjbakhche, A. …& Razumovsky, A. (2015). A review of the effectiveness of neuroimaging modalities for the detection of traumatic brain injury. *Journal of Neurotrauma*, *32*(22), 1693-1721.

Anderson, V.V., Anderson, P., Northam, E., Jacobs, R. & Catroppa, C. (2001). Development of executive functions through late childhood and adolescence in an Australian Sample. *Developmental Neuropsychology*, 20, 385-406. doi: 10.1207/S15326942DN2001_5.

Andersson S, Gundersen PM, Finset A (1999) Emotional activation during therapeutic interaction in traumatic brain injury: effect of apathy, self-awareness and implications for rehabilitation. *Brain Inj* 13: 393–404.

Andruszkow, H., Deniz, E., Urner, J., Probst, C., Grün, O., Lohse, R., ... & Hildebrand, F. (2014). Physical and psychological long-term outcome after traumatic brain injury in children and adult patients. *Health and Quality of Life Outcomes*, *12*(1), 1.

Arciniegas DB, Wortzel HS (2014) Emotional and behavioral dyscontrol after traumatic brain injury. *Psychiat Clin Nth America* 37: 31–53.

Bailey NW, Hoy KE, Maller JJ, Upton DJ, Segrave RA, Fitzgibbon BM, Fitzgerald PB (2015) Neural evidence that conscious awareness of errors is reduced in depression following a traumatic brain injury. *Biol Psych* 106: 1–10.

Beckmann, C. F., DeLuca, M., Devlin, J. T., & Smith, S. M. (2005). Investigations into resting-state connectivity using independent component analysis. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, *360*(1457), 1001-1013.

Bench SW, Lench HC (2013) On the function of boredom. *Behav Sci* 3: 459–472.

Berlin HA, Rolls ET, Kischka U (2004) Impulsivity, time perception, emotion and reinforcement sensitivity in patients with orbitofrontal cortex lesions. 127: 1108–1126.

Binder, J. R., Frost, J. A., Hammeke, T. A., Bellgowan, P. S. F., Rao, S. M., & Cox, R. W. (1999). Conceptual processing during the conscious resting state: a functional MRI study. *Journal of Cognitive Neuroscience*, *Brain 11*(1), 80-93.

Bonnelle, V., Ham, T. E., Leech, R., Kinnunen, K. M., Mehta, M. A., Greenwood, R. J., & Sharp, D. J. (2012). Salience network integrity predicts default mode network function after traumatic brain injury. *Proceedings of the National Academy of Sciences*, *109*(12), 4690-4695.

Bonnelle, V., Leech, R., Kinnunen, K. M., Ham, T. E., Beckmann, C. F., De Boissezon, X., ... & Sharp, D. J. (2011). Default mode network connectivity predicts sustained attention deficits after traumatic brain injury. *The Journal of Neuroscience*, *31*(38), 13442-13451.

Buckner, R. L., Andrews-Hanna, J. R., & Schacter, D. L. (2008). The brain's default network. *Annals of the New York Academy of Sciences*, *1124*(1), 1-38.

Buss, A.H. & Perry, M. (1992). The aggression questionnaire. *Journal of Personality and Social Psychology,* 63, 452-459.

Carney, N., Chesnut, R. M., Maynard, H., Mann, N. C., Patterson, P., & Helfand, M. (1999). Effect of cognitive rehabilitation on outcomes for persons with traumatic brain injury: A systematic review. *The Journal of Head Trauma Rehabilitation*, *14*(3), 277-307.

Carriere, J.S.A., Cheyne, J.A., & Smilek, D. (2008). Everyday attention lapses and memory failures: The affective consequences of mindlessness. *Consciousness and Cognition,* 17, 835-847.

Carriere, J.S.A., Seli, P. & Smilek, D. (2013). Wandering in both mind and body: Individual differences in mind wandering and inattention predict fidgeting. *Canadian Journal of Experimental Psychology,* 67, 19-31.

Cassidy, J. D., Cancelliere, C., Carroll, L. J., Côté, P., Hincapié, C. A., Holm, L. W., ... & Borg, J. (2014). Systematic review of self-reported prognosis in adults after mild traumatic brain injury: results of the International Collaboration on Mild Traumatic Brain Injury Prognosis. *Archives of Physical Medicine and Rehabilitation*, *95*(3), S132-S151.

Cheyne, J.A., Carriere, J.S.A. & Smilek, D. (2006). Absent-mindedness: Lapses of conscious awareness and everyday cognitive failures. *Consciousness and Cognition,* 15, 578-592.

Cheyne, J.A.., Solman, G.C., Carriere, J.S. & Smilek, D. (2009). Anatomy of an error: a bidirectional state model of task engagement/disengagement and attention-related errors. *Cognition*, 111, 98-113.

Chiou, K. S., Genova, H. M., & Chiaravalloti, N. D. (2015). Structural white matter differences underlying heterogeneous learning abilities after TBI. *Brain Imaging and Behavior*, 1-6.

Christoff , K. (2011). Undirected thought: neural determinants and correlates. *Brain Research, 1428*, 51-59. doi: 10.1016/j.brainres.2011.09.060

Christoff, K., Gordon, A. M., Smallwood, J., Smith, R., & Schooler, J. W. (2009). Experience sampling during fMRI reveals default network and executive system contributions to mind wandering. *Proceedings of the National Academy of Science, 106*, 8719-8724. doi: 10.1073/pnas.0900234106

Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). Applied multiple correlation/regression analysis for the behavioral sciences. *UK: Taylor & Francis*

Csikszentmihalyi, M. (1996). Flow and the psychology of discovery and invention. *New Yprk: Harper Collins.*

Culham, J. (2003). Attention-grabbing motion in the human brain. *Neuron, 40*, 451-452. doi: 10.1016/S0896-6273(03)00689-5

Dahlen, E.R., Martin, R.C., Regan, K. & Kuhlman, M.M. (2004). Boredom proneness in anger and aggression: effects of impulsiveness and sensation seeking. *Personality and Individual Differences,* 37, 1615-1627.

Damrad-Frye, R., & Liard, J. D. (1989). The experience of boredom: The role of the self-perception of attention. *Journal of Personality and Social Psychology, 57*, 315-320

Danckert, J., & Merrifield, C. (2016). Boredom, sustained attention and the default mode network. *Experimental brain research*, 1-12

De Martino, F., Gentile, F., Esposito, F., Balsi, M., Di Salle, F., Goebel, R., & Formisano, E. (2007). Classification of fMRI independent components using IC-fingerprints and support vector machine classifiers. *NeuroImage, 34,* 177-194. doi: 10.1016/j.neuroimage.2006.08.041

de Sousa A, McDonald S, Rushby J (2012) Changes in emotional empathy, affective responsivity, and behavior following severetraumatic brain injury. *J Clin Exp Neuropsychol.* 2012 34: 606–623. doi: 10.1080/13803395.2012.667067.

DeCoster, J. (2007). Applied Linear Regression Notes set 1. Retrieved from http://www.stat-help.com/notes.html

Dennhardt, A.A. & Murphy, J.G. (2011). Associations between depression, distress tolerance, delay discounting, and alcohol-related problems in European American and African American college students. *Psychology and Addictive Behaviors,* 25, 595-604.

Denson, T.F., Pedersen, W.C., Friese, M., Hahm, A. & Roberts, L. (2011). Understanding impulsive aggression: Angry rumination and reduced self-control capacity are mechanisms underlying the provocation-aggression relationship. *Personality and Social Psychology Bulletin,* 37, 850-862.

Deouell, L. Y., Sacher, Y., & Soroker, N. (2005). Assessment of spatial attention after brain damage with a dynamic reaction time test. *Journal of the International Neuropsychological Society*, *11*(06), 697-707

Depue BE, Olson-Madden JH, Smolker HR, Rajamani M, Brenner LA, Banich MT (2014) Reduced amygdala volume is associated with deficits in inhibitory control: a voxel- and surface-based morphometric analysis of comorbid PTSD/mild TBI. *BioMed Res Int*: doi: 10.1155/2014/691505.

Di Battista, A., Godfrey, C., Soo, C., Catroppa, C., & Anderson, V. (2014). Depression and health related quality of life in adolescent survivors of a traumatic brain injury: a pilot study. *PloS one*, *9*(7), e101842

Dockree PM, Bellgrove MA, O'Keeffe FM, Moloney P, Aimola L, Carton S, Robertson IH (2006) Sustained attention in traumatic brain injury (TBI) and healthy controls: enhanced sensitivity with dual-task load. *Exp Brain Res* 168: 218–229.

Dockree PM, Kelly SP, Roche RA, Hogan MJ, Reilly RB, Robertson IH (2004) Behavioural and physiological impairments of sustained attention after traumatic brain injury. *Brain Res Cogn Brain Res* 20: 403–414. doi:10.1037/0022-3514.63.3.452

Drew AS, Langan J, Halterman C, Osternig LR, Chou LS, van Donkelaar P (2007) Attentional disengagement dysfunction following mTBI assessed with the gap saccade task. *Neurosci* Lett 417: 61–65.

Duerden, E. G., Arsalidou, M., Lee, M., & Taylor, M. J. (2013). Lateralization of affective processing in the insula. *Neuroimage*, *78*, 159-175

Dyer KF, Bell R, McCann J, Rauch R (2006) Aggression after traumatic brain injury: Analysing socially desirable responses and the nature of aggressive traits. *Brain Injury* 20: 1163–1173.

Eastwood, J.D., Frischen, A., Fenske, M.J. & Smilek, D. (2012). The unengaged mind: Defining boredom in terms of attention. *Perspectives on Psychological Science,* 7, 482-495.

Elpidorou A (2014) The bright side of boredom. *Front Psych* 5: doi: 10.3389/fpsyg.2014.01245.

Esposito, F., Scarabino, T., Hyvarinen, A., Himberg, J. Formisano, E., Comani, S., … Di Salle, F. (2005). Independent components analysis of fMRI group studies by self-organizing clustering. *NeuroImage, 25*, 193-205. doi:10.1016/j.neuroimage.2004.10.042

Fahlman, S.A., Mercer-Lynn, K.B., Flora, D.B. & Eastwood, J. (2013). Development and validation of the multidimensional state boredom scale. *Assessment*, 20, 68-85.

Farmer, R. & Sundberg, N.D. (1986). Boredom proneness: The development and correlates of a new scale. *Journal of Personality Assessment,* 50, 4-17.

Fischer, B. L., Parsons, M., Durgerian, S., Reece, C., Mourany, L., Lowe, M. J., ... & Scheibel, R. S. (2014). Neural activation during response inhibition differentiates blast from mechanical causes of mild to moderate traumatic brain injury. *Journal of Neurotrauma*, *31*(2), 169-179

Fothergill, A, (director), Berlowitz, V., Malone, S., & Lemire, M. (producers). (2007). *Planet Earth* [film]. Warner Home Video, Burbank, CA, USA.

Ganesalingam K, Sanson A, Anderson V, Yeates KO (2007) Self-regulation as a mediator of the effects of childhood traumatic brain injury on social and behavioral functioning. *J Int Neuropsychol Soc* 13: 298–311.

Gao, W., & Lin, W. (2012). Frontal parietal control network regulates that anticorrelated default and dorsal attention networks. *Human Brain Mapping, 33*, 192-202. doi: 10.1002/hbm.21204

Gerritsen, C.J., Toplak, M.E., Sciaraffa, J. & Eastwood, J. (2014). I can't get no satisfaction: Potential causes of boredom. *Consciousness and Cognition*, 27, 27-41.

Gogtay N, Giedd JN, Lusk L, Hayashi KM, Greenstein D, Vaituzis AC, Thompson PM (2004) Dynamic mapping of human cortical development during childhood through early adulthood. *Proc Natl Acad Sci* USA 101: 8174–8179.

Goldberg Y, Danckert J (2013) Traumatic brain injury, boredom and depression. *Behav Sci* 3: 434– 444.

Goldberg, Y.K., Eastwood, J.D., LaGuardia, J. & Danckert, J. (2011). Boredom: An emotional experience distinct from apathy, anhedonia, or depression. *Journal of Social and Clinical Psychology*, 30, 647-666.

Gottfried, J. A., O'Doherty, J., & Dolan, R. J. (2003). Encoding predictive reward value in human amygdala and orbitofrontal cortex. *Science*, *301*(5636), 1104-1107.

Greicius, M. D., Krasnow, B., Reiss, A. L., & Menon, V. (2003). Functional connectivity in the resting brain: a network analysis of the default mode hypothesis. *Proceedings of the National Academy of Sciences*, *100*(1), 253-258

Gusnard, D. A., & Raichle, M. E. (2001). Searching for a baseline: functional imaging and the resting human brain. *Nature Reviews Neuroscience, 2*, 685-694. doi: 10.1038/35094500

Gusnard, D. A., Akbudak, E., Shulman, G. L., & Raichle, M. E. (2001). Medial prefrontal cortex and self-referential mental activity: relation to a default mode of brain function. *Proceedings of the National Academy of Science U.S.A.*, *98*, 4259–64.

Ham TE, Bonnelle V, Hellyer P, Jilka S, Robertson IH, Leech R, Sharp DJ (2014) The neural basis of impaired self-awareness after traumatic brain injury. *Brain* 137: 586–597. doi: 10.1093/brain/awt350.

Hamilton, J.A. (1983). Development of interest and enjoyment in adolescence. Part II. Boredom and psychopathology. *Journal of Youth and Adolescence,* 12, 355-362.

Hanks RA, Temkin N, Machamer J, Dikmen SS (1999) Emotional and behavioral adjustment after traumatic brain injury. *Arch Phys Med Rehabil* 80: 991–997.

Hart, T., Brenner, L., Clark, A. N., Bogner, J. A., Novack, T. A., Chervoneva, I., ... & Arango-Lasprilla, J. C. (2011). Major and minor depression after traumatic brain injury. *Archives of physical medicine and rehabilitation*, *92*(8), 1211-1219

Hulkower, M. B., Poliak, D. B., Rosenbaum, S. B., Zimmerman, M. E., & Lipton, M. L. (2013). A decade of DTI in traumatic brain injury: 10 years and 100 articles later. *American Journal of Neuroradiology*, *34*(11), 2064-2074.

Hunter, A., & Eastwood, J. D. (2016). Does state boredom cause failures of attention? Examining the relations between trait boredom, state boredom, and sustained attention. *Experimental Brain Research*, 1-10.

Hyvarinen, A., Hoyer, P. O., & Inki, M. (2001). Topographic independent components analysis. *Neural Computation, 13*, 1527-1558. doi: 10.1162/089976601750264992

Isacescu, J. & Danckert, J. (2016). Exploring the relationship between boredom proneness and self-control in traumatic brain injury (TBI). *Experimental Brain Research*, DOI 10.1007/s00221-016-4674-9.

Isacescu, J., Struk, A.A., & Danckert, J. (2016). Cognitive and affective predictors of boredom proneness. *Cognition & Emotion.* DOI: 10.1080/02699931.2016.1259995

Joireman, J., Anderson, J. & Strathman, A. (2003). The aggression paradox: understanding links among aggression, sensation seeking, and the consideration of future consequences. *Journal of Personality and Social Psychology,* 84, 1287-1302.

Jorge RE, Arciniegas DB (2014) Mood disorders after TBI. *Psychiatr Clin North Am* 37: 13–29. doi: 10.1016/j.psc.2013.11.005.

Juengst SB, Arenth PM, Raina KD, McCue M, Skidmore ER (2014) Affective state and community integration after traumatic brain injury. *Am J Phys Med Rehabil* 93: 1086–1094.

Kass, S.J. & Vodanovich, S.J. (1990). Boredom proneness: Its relationship to Type A behaviour pattern and sensation seeking. *Psychology: A Journal of Human Behaviour,* 27, 7-16.

Keating, D.P. (2012). Cognitive and brain development in adolescence. *Enfance*, 64, 267-279. http://dx.doi.org.proxy.lib.uwaterloo.ca/10.4074/S0013754512003035.

Kim, E. (2002). Agitation, aggression, and disinhibition syndromes after traumatic brain injury. *NeuroRehabilitation*, *17*(4), 297-310

Kim, E., Lauterbach, E. C., Reeve, A., Arciniegas, D. B., Coburn, K. L., Mendez, M. F., ... & Coffey, E. C. (2007). Neuropsychiatric complications of traumatic brain injury: a critical review of the literature (a report by the ANPA Committee on Research). *The Journal of Neuropsychiatry and Clinical Neurosciences*, *19*(2), 106-127

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*(06), 1197-1211

Kraus, M. F., Susmaras, T., Caughlin, B. P., Walker, C. J., Sweeney, J. A., & Little, D. M. (2007). White matter integrity and cognition in chronic traumatic brain injury: a diffusion tensor imaging study. *Brain*, *130*(10), 2508-2519

Kreutzer, J. S., Seel, R. T., & Gourley, E. (2001). The prevalence and symptom rates of depression after traumatic brain injury: a comprehensive examination. *Brain injury*, *15*(7), 563-576.

Kruglanski AW (2003). Goal systems theory: Integrating the cognitive and motivational aspects of self-regulation. In Motivated social perception: The Ontario symposium (Vol. 9, p. 247). Psychology Press.

Lange RT, Iverson GL, Rose A (2011) Depression strongly influences postconcussion symptom reporting following mildtraumatic brain injury. *J Head Trauma Rehabil* 26: 127–137.

Liu, A. Y., Maldjian, J. A., Bagley, L. J., Sinson, G. P., & Grossman, R. I. (1999). Traumatic brain injury: diffusion-weighted MR imaging findings. *American Journal of Neuroradiology*, *20*(9), 1636-1641.

Lohmann H (2011) Long-term cognitive and emotional consequences of mild traumatic brain injury. *Psychol Med* 41: 1197–1211. doi: 10.1017/S0033291710001728.

Lovibond, P.F. & Lovibond, S.H. (1995). The structure of negative emotional states: Comparison of the depression anxiety stress scales (DAAS) with the Beck Depression and Anxiety Inventories. *Behaviour Research and Therapy,* 33, 335-343.

Malkovsky, E., Merrifield, C., Goldberg, Y. & Danckert, J. (2012). Exploring the relationship between boredom and sustained attention. *Experimental Brain Research*, 221, 59-67.

Mason, M. F., Norton, M. I., Van Horn, J. D., Wegner, D. M., Grafton, S. T., & Macrae, C. N. (2007). Wandering minds: the default network and stimulus-independent thought. *Science, 315*, 393-395. doi: 10.1126/science.1131295

Mayer, A. R., Bellgowan, P. S., & Hanlon, F. M. (2015). Functional magnetic resonance imaging of mild traumatic brain injury. *Neuroscience & Biobehavioral Reviews*, *49*, 8-18.

McAvinue, Laura, Fiadhnait O'Keeffe, Deirdre McMackin, and Ian H. Robertson. "Impaired sustained attention and error awareness in traumatic brain injury: Implications for insight." *Neuropsychological Rehabilitation* 15, no. 5 (2005): 569-587.

McDonald S, Hunt C, Henry JD, Dimoska A, Bornhofen C (2010) Angry responses to emotional events: The role of impaired control and drive in people with severe traumatic brain injury. *J Clin Exp Neuropsych* 32: 855–864.

McDonald S, Saad A, James C (2011) Social dysdecorum following severe traumatic brain injury: Loss of implicit social knowledge or loss of control?. *J Clin Exp Neuropsych* 33: 619–630.

Menon, V., & Uddin, L. Q. (2010). Saliency, switching, attention and control: a network model of insula function. *Brain Structure and Function, 214*, 5-6. doi: 10.1007/s00429-010-0262-0

Mercer, K.B. & Eastwood, J.D. (2010). Is boredom associated with problem gambling behaviour? It depends on what you mean by 'boredom'. *International Gambling* Studies, 10, 91-104.

Merrifield, C., & Danckert, J. (2014). Characterizing the psychophysiological signature of boredom. *Experimental brain research*, *232*(2), 481-491.

Meythaler, J. M., Peduzzi, J. D., Eleftheriou, E., & Novack, T. A. (2001). Current concepts: diffuse axonal injury–associated traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, *82*(10), 1461-1471

Mischel W, Shoda Y, Rodriguez MI (1989) Delay of gratification in children. Science 244: 933–938. Moreno-López, Sahakian, Manktelow, Menon & Stamatakis (2016) Depression following traumatic brain injury: A functional connectivity perspective, *Brain Injury*, 30:11, 1319-1328, DOI: 10.1080/02699052.2016.1186839

Nakamura, J., & Csikszentmihalyi, M. (2014). The concept of flow. In *Flow and the foundations of positive psychology* (pp. 239-263). Springer Netherland

Nasreddine ZS, Phillips NA, Bédirian V, Charbonneau S, Whitehead V, Collin I, Cummings JL, Chertkow H. (2005) The Montreal Cognitive Assessment (MoCA): A Brief Screening Tool For Mild Cognitive Impairment. *J Am Geriatrics Soc* 53: 695–699.

Nicholl, J., & LaFrance, W. C. (2009, July). Neuropsychiatric sequelae of traumatic brain injury. In *Seminars in neurology* (Vol. 29, No. 03, pp. 247-255). © Thieme Medical Publishers.

O'Callaghan, A., McAllister, L., & Wilson, L. (2012). Insight vs readiness: Factors affecting engagement in therapy from the perspectives of adults with TBI and their significant others. *Brain injury*, *26*(13-14), 1599-1610.

O'Doherty J, Kringelbach ML, Rolls ET, Hornak J, Andrews C (2001) Abstract reward and punishment representations in the human orbitofrontal cortex. *Nature Neurosci* 4: 95–102.

O'Keeffe FM, Dockree PM, Moloney P, Carton S, Robertson IH (2007) Characterising error-awareness of attentional lapses and inhibitory control failures in patients with traumatic brain injury. *Exp Brain Res* 180: 59–67.

O'Keeffe, F. M., Dockree, P. M., & Robertson, I. H. (2004). Poor insight in traumatic brain injury mediated by impaired error processing?: Evidence from electrodermal activity. *Cognitive Brain Research*, *22*(1), 101-112.

Ozen, L. J., & Fernandes, M. A. (2011). Effects of “diagnosis threat” on cognitive and affective functioning long after mild head injury. *Journal of the International Neuropsychological Society*, *17*(02), 219-229

Pekrun, R., Hall, N.C., Goetz, T. & Perry, R.P. (2014). Boredom and academic achievement: Testing a model of reciprocal causation. *Journal of Educational Psychology*, 106, 696-710.

Poletti, M. (2009). Adolescent brain development and executive functions: A prefrontal framework for developmental psychopathologies. *Clinical Neuropsychiatry: Journal of Treatment Evaluation*, 6, 155-165.

Prigatano, G. P. (1996). Behavioral limitations TBI patients tend to underestimate: A replication and extension to patients with lateralized cerebral dysfunction. *The Clinical Neuropsychologist*, *10*(2), 191-201.

Rabinowitz, A. R., & Levin, H. S. (2014). Cognitive sequelae of traumatic brain injury. *Psychiatric Clinics of North America*, *37*(1), 1-11

Rao, V., & Lyketsos, C. (2000). Neuropsychiatric sequelae of traumatic brain injury. *Psychosomatics*, *41*(2), 95-103

Reeves RR, Panguluri RL (2011) Neuropsychiatric complications of traumatic brain injury. *J Psychosoc Nursing Mental Health Services* 49: 42.

Rehm, L.P. (1977). A self-control model of depression. *Behavior Therapy,* 8, 787-804.

Riggio, S. (2010). Traumatic brain injury and its neurobehavioral sequelae. *Psychiatric Clinics of North America*, *33*(4), 807-819

Rizzo, M., & Robin, D. A. (1990). Simultanagnosia – a defect of sustained attention yields insights on visual information-processing, *Neurology, 40*, 447-455 Robertson IH, Manly T, Andrade J, Baddeley BT, Yiend J (1997) 'Oops!': performance correlates of everyday attentional failures in traumatic brain injured and normal subjects. *Neuropsychologia* 35: 747–758.

Robertson, I. H., Manly, T., Andrade, J., Baddeley, B. T., & Yiend, J. (1997). Oops!': performance correlates of everyday attentional failures in traumatic brain injured and normal subjects. *Neuropsychologia*, *35*(6), 747-758

Rosenberg H, McDonald S, Dethier M, Kessels RP, Westbrook RF (2014) Facial emotion recognition deficits following moderate-severe Traumatic Brain Injury(TBI): re-examining *the valence effect and the role of emotion intensity. J Int Neuropsychol Soc* 20: 994–1003.

Rule RR, Shimamura AP, Knight RT (2002) Orbitofrontal cortex and dynamic filtering of emotional stimuli. *Cogn Affect Behav Neurosci* 2: 264–270.

Rupp, D.E. & Vodanovich, S.J. (1997). The role of boredom proneness in self-reported anger and aggression. *Journal of Social Behaviour & Personality,* 12, 925-936.

Scheid, R., Preul, C., Gruber, O., Wiggins, C., & Von Cramon, D. Y. (2003). Diffuse axonal injury associated with chronic traumatic brain injury: evidence from T2*-weighted gradient-echo imaging at 3 T. *American Journal of Neuroradiology*, *24*(6), 1049-1056

Schooler, J. W., Smallwood, J., Christoff, K., Handy, T. C., Reichle, E. D., & Sayette, M. A. (2011). Meta-awareness, perceptual decoupling and the wandering mind. *Trends in Cognitive Sciences*, *15*(7), 319-326.

Seel RT, Kreutzer JS (2003) Depression assessment after traumatic brain injury: An empirically based classification method. *Arch Phys Med Rehab* 84: 1621–1628. doi: 10.1053/S0003-9993(03)00270-3

Seel, R. T., Kreutzer, J. S., Rosenthal, M., Hammond, F. M., Corrigan, J. D., & Black, K. (2003). Depression after traumatic brain injury: a National Institute on Disability and Rehabilitation Research Model Systems multicenter investigation. *Archives of Physical Medicine and Rehabilitation*, *84*(2), 177-184.

Seeley, W. W., Menon, V., Schatzberg, A. F., Keller, J., Glover, G. H., Kenna, H., ... & Greicius, M. D. (2007). Dissociable intrinsic connectivity networks for salience processing and executive control. *The Journal of neuroscience*, *27*(9), 2349-2356

Seli, P., Carriere, J.S., & Smilek, D. (2015). Not all mind wandering is created equal: dissociating deliberate from spontaneous mind wandering. *Psychological Research*, 79, 750-758. doi: 10.1007/s00426-014-0617-x.

Sharp, D. J., Scott, G., & Leech, R. (2014). Network dysfunction after traumatic brain injury. *Nature Reviews. Neurology*, *10*(3), 156

Sherer M, Davis LC, Sander AM, Caroselli JS, Clark AN, Pastorek NJ (2014) Prognostic importance of self-reported traits/problems/strengths and environmental barriers/facilitators for predicting participation outcomes in persons with traumatic brain injury: a systematic review. *Arch Phys Med Rehabil* 95: 1162–1173.

Sherman, L. E., Rudie, J. D., Pfeifer, J. H., Masten, C. L., McNealy, K., & Dapretto, M. (2014). Development of the default mode and central executive networks across early adolescence: a longitudinal study. *Developmental cognitive neuroscience*, *10*, 148-159

Sigurdardottir S, Andelic N, Roe C, Schanke AK. (2014) Identifying longitudinal trajectories of emotional distress symptoms 5 years after traumatic brain injury. *Brain Inj* 28: 1542–1550.

Smallwood, J., Fitzgerald, A., Miles, L.K. & Phillips, L.H. (2009). Shifting moods, wandering minds: Negative moods lead the mind to wander. *Emotion*, 9, 271-276.

Smith, D. H., & Meaney, D. F. (2000). Axonal damage in traumatic brain injury. *The neuroscientist*, *6*(6), 483-495

Smits, R.R., Stein, J.S., Johnson, P.S., Odum, A.L. & Madden, G.J. (2013). Test-retest reliability and construct validity of the experimental discounting task. *Experimental and Clinical Psychopharmacology,* 21, 155-163.

Sozda, C. N., Larson, M. J., Kaufman, D. A., Schmalfuss, I. M., & Perlstein, W. M. (2011). Error-related processing following severe traumatic brain injury: an event-related functional magnetic resonance imaging (fMRI) study. *International Journal of Psychophysiology*, *82*(1), 97-106

Spreng, R. N., Sepulcre, J., Turner, G. R., Stevens, W. D., & Schacter, D. L. (2013). Intrinsic architecture underlying the relations among the default, dorsal attention, and frontoparietal control networks of the human brain. *Journal of cognitive neuroscience*, *25*(1), 74-86;

Sridharan, D., Levitin, D. J., & Menon, V. (2008). A critical role for the right fronto-insular cortex in switching between central-executive and default-mode networks. *Proceedings of the National Academy of Sciences*, *105*(34), 12569-12574

Stark KD, Reynolds WM, Kaslow NJ (1987) A comparison of the relative efficacy of self-control therapy and a behavioral problem-solving therapy for depression in children. *J Abn Child Psych* 15: 91–113.

Stein, J.S. & Madden, G.J. (2013). Delay discounting and drug abuse: Empirical, conceptual, and methodological considerations. MacKillop, J (Ed); de Wit, H. (Ed). The Wiley-Blackwell handbook of addiction psychopharmacology. Wiley-Blackwell. http://dx.doi.org.proxy.lib.uwaterloo.ca/10.1002/9781118384404.ch7.

Steinberg, L, Albert, D., Cauffman, E., Banich, M., Graham, S. & Woolard, J. (2008). Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: evidence for a dual systems model. *Developmental Psychology,* 44, 1764-1778.

Struk AA, Scholer AA, Danckert J (2015) A self-regulatory approach to understanding boredom proneness. *Cogn Emot* 29: 1–14. doi: 10.1080/02699931.2015.1064363.

Struk, A.A., Carriere, J., Cheyne, A.J., & Danckert, J. (2015). A short Boredom Proneness Scale (sBPS): Psychometric properties and validation. *Assessment*, 1-14. doi: 10.1177/1073191115609996.

Swets, J. A. (1964). Signal Detection and Recognition in Human Observers: Contemporary Readings Swick D, Honzel N, Larsen J, Ashley V (2013) Increased response variability as a marker of executive dysfunction in veterans with post-traumatic stress disorder. *Neuropsychologia* 51: 3033–3040.

Talairach, J., & Tournoux, P. (1988). Co-planar stereotaxic atlas of the human brain. 3-Dimensional proportional system: an approach to cerebral imaging

Tangney, J.P., Baumeister, R.F. & Boone, A.L. (2004). High self-control predicts good adjustment, less pathology, better grades, and interpersonal success. *Journal of Personality,* 72, 271-322.

Teasdale, G. & Jennett, B. (1976). Assessment and prognosis of coma after head injury. *Acta neurochirurgica*, *34*(1-4), 45-55.

Ulrich, M., Keller, J., Hoenig, K., Waller, C., & Gron, G. (2014). Neural correlates of induced flow experiences. *NeuroImage, 86*, 194-202. doi: 10.1016/j.neuroimage.2013.08.019

van Tilburg, W. A., & Igou, E. R. (2011). On boredom and social identity: A pragmatic meaning-regulation approach. *Personality and Social Psychology Bulletin*, 0146167211418530.

Van Tilburg, W. A., & Igou, E. R. (2011). On the meaningfulness of existence: When life salience boosts adherence to worldviews. *European Journal of Social Psychology*, *41*(6), 740-750.

Vodanovich, S.J. & Kass, S.J. (1990). Age and gender differences in boredom proneness. *Journal of Social Behaviour & Personality,* 5, 297-307.

Wallis JD (2007) Orbitofrontal cortex and its contribution to decision-making. *Annu Rev Neurosci* 30: 31–56.

Weissman, D. H., Roberts, K. C., Visscher, K. M., & Woldorff, M. G. (2006). The neural bases of momentary lapses in attention. *Nature neuroscience*, *9*(7), 971-978

Whyte J, Polansky M, Cavallucci C, Fleming M, Lhulier J, Coslett HB (1996) Inattentive behavior after traumatic brain injury. *J Int Neuropsychol Soc* 2: 274–281.

Wood, R. L., & Thomas, R. H. (2013). Impulsive and episodic disorders of aggressive behaviour following traumatic brain injury. *Brain injury*, *27*(3), 253-261

Zhang, J., Puvenna, V., & Janigro, D. (2016). Biomarkers of Traumatic Brain Injury and Their Relationship to Pathology.

Appendix A: Participant Recruitment

Study 1

Time and Place:

Fall of 2013; University of Waterloo

Procedure:

Using the University of Waterloo's Research Experiences Group (REG), undergraduate students participate for course credit by completing surveys online.

Inclusion Criteria:

Completed all Questionnaires.

Exclusion Criteria:

History of head injury, with or without loss of consciousness (LOC).
Diagnosis of neurological/psychiatric condition(s), with or without medication(s).

Initial participants: 3555

Did not complete all questionnaires: 1079 History of
head injury, with or without LOC: 355
Diagnosis of neurological/psychiatric condition(s), with or without medication(s): 193
Final sample: 1928

Study 2

Time and Place:

Summer of 2014; University of Waterloo & Kitchener Waterloo community

Procedure:

Controls & Concussed: Using the University of Waterloo's Research Experiences Group (REG), undergraduate students participate for course credit by completing surveys online.
TBI: Participants recruited from local outreach community centers; completed questionnaires in person.

Initial Control participants: 3555

Did not complete all questionnaires: 1079 History of
head injury, with or without LOC: 355
Diagnosis of neurological/psychiatric condition(s), with or without medication(s): 193
Final sample: 1928

Initial Concussed participants: 355

Did not complete all questionnaires: 15
Final sample: 340

Initial TBI participants: 36

Did not complete all questionnaires: 1

Final sample: 35

Study 3

Time and Place:

June – December, 2015; Grand River Hospital

Procedure:

Controls: Participants recruited from Kitchener-Waterloo community.
TBI: Participants from Study 2 who agreed to participate in fMRI follow-up were contacted.

Exclusion Criteria:

MRI Screening form (see Appendix C)

Initial Control participants: 20
- Time conflict: 2
- Changed their mind: 3
- *Final sample: 15*

Initial TBI participants: 36
- Metal implants: 13
- Spinal cord stimulator: 1
- Claustrophobia: 3
- Hemiparesis: 2
- Vision impairment: 8
- Could not be reached: 2
- Changed their mind: 2
- *Final sample: 5*

Appendix B: Questionnaires

Short Boredom Proneness Scale

SBPS (Struk, Carriere, Cheyne, & Danckert, 2015)

Instructions:

The following are some statements that may or may not describe you, in general, on a typical day. Please rate each statement using the 7-point scale above by circling the number that corresponds to how much you do or do not feel like the sentence describes you. Remember to rate each statement based on how much it describes you **in general**.

1 Strongly Disagree	2 Disagree	3 Somewhat Disagree	4 Neutral	5 Somewhat Agree	6 Agree	7 Strongly Agree

1. I often find myself at ‘loose ends’, not knowing what to do.	1	2	3	4	5	6	7
2. I find it hard to entertain myself.	1	2	3	4	5	6	7
3. Many things I have to do are repetitive and monotonous.	1	2	3	4	5	6	7
4. It takes more stimulation to get me going than most people.	1	2	3	4	5	6	7
5. I don’t feel motivated by most things I do.	1	2	3	4	5	6	7
6. In most situations, it is hard for me to find something to do or see to keep me interested.	1	2	3	4	5	6	7
7. Much of the time I just sit around doing nothing.	1	2	3	4	5	6	7
8. Unless I am doing something exciting, even dangerous, I feel half-dead and dull.	1	2	3	4	5	6	7

Brief Self-Control Scale

BSCS (Tangney, Baumeister & Boone, 2004)

Instructions: Using the scale provided, please indicate how much each of the following statements reflects how you typically are.

1	2	3	4	5
Not at all		Neutral		Very much

1.	I am good at resisting temptation.	1	2	3	4	5
2.	I have a hard time breaking bad habits.	1	2	3	4	5
3.	I am lazy.	1	2	3	4	5
4.	I say inappropriate things.	1	2	3	4	5
5.	I do certain things that are bad for me, if they are fun.	1	2	3	4	5
6.	I refuse things that are bad for me.	1	2	3	4	5
7.	I wish I had more self-discipline.	1	2	3	4	5
8.	People would say that I have iron self-discipline.	1	2	3	4	5
9.	Pleasure and fun sometimes keep me from getting work done.	1	2	3	4	5
10.	I have trouble concentrating.	1	2	3	4	5
11.	I am able to work effectively toward long-term goals.	1	2	3	4	5
12.	Sometimes I can't stop myself from doing something, even if I know it is wrong.	1	2	3	4	5
13.	I often act without thinking through all the alternatives.	1	2	3	4	5

Depression, Anxiety and Stress Scale

DAAS (Lovibond & Lovibond, 1995)

Instructions:

Please read each statement and circle a number 0, 1, 2, or 3 which 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 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, lifts, 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, sweaty hands).	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
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, send 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

Mind-Wandering

MW (Carriere, Seli & Smilek, 2013)

Instructions: For the following statements please select the answer that most accurately reflects your everyday mind-wandering.

1 Extremely Inaccurate	2 Inaccurate	3 Somewhat Inaccurate	4 Neutral	5 Somewhat Accurate	6 Accurate	7 Extremely Accurate

1.	I allow my thoughts to wander on purpose.	1	2	3	4	5	6	7
2.	I enjoy mind-wandering.	1	2	3	4	5	6	7
3.	I find mind-wandering is a good way to cope with boredom.	1	2	3	4	5	6	7
4.	I allow myself to get absorbed in pleasant fantasy.	1	2	3	4	5	6	7
5.	I find my thoughts wandering spontaneously.	1	2	3	4	5	6	7
6.	When I mind-wander my thoughts tend to be pulled from topic to topic.	1	2	3	4	5	6	7
7.	It feels like I don't have control over when my mind wanders.	1	2	3	4	5	6	7
8.	I mind wander even when I'm supposed to be doing something else.	1	2	3	4	5	6	7

Buss-Perry Aggression Questionnaire

BPAQ (Buss & Perry, 1992)

Instructions:

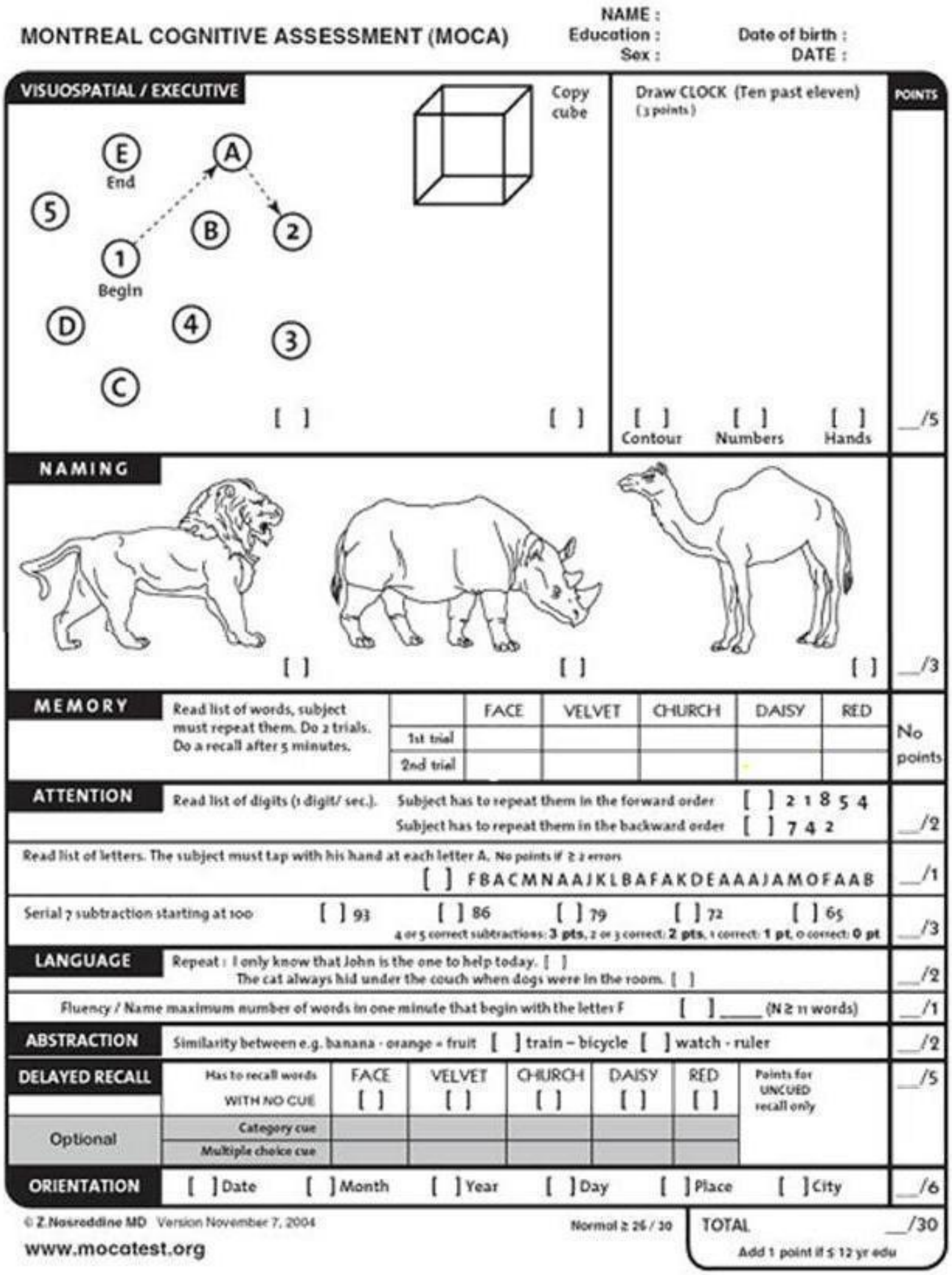
Please rate each of the following items in terms of how characteristic they are of you. Use the following scale for answering these items.

1	2	3	4	5	6	7
Extremely Uncharacteristic	Uncharacteristic	Somewhat Uncharacteristic	Neutral	Somewhat Characteristic	Characteristic	Extremely Characteristic

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

Montreal Cognitive Assessment

(Nasreddine, et al., 2005)



MONTREAL COGNITIVE ASSESSMENT (MOCA)

NAME :
Education :
Sex :
Date of birth :
DATE :

POINTS

VISUOSPATIAL / EXECUTIVE

[ ]

Copy cube [ ]

Draw CLOCK (Ten past eleven) (3 points)

[ ] Contour [ ] Numbers [ ] Hands

___/5

NAMING

[ ] [ ] [ ]

___/3

MEMORY

Read list of words, subject must repeat them. Do 2 trials. Do a recall after 5 minutes.

	FACE	VELVET	CHURCH	DAISY	RED
1st trial					
2nd trial					

No points

ATTENTION

Read list of digits (1 digit/ sec.).
Subject has to repeat them in the forward order [ ] 2 1 8 5 4
Subject has to repeat them in the backward order [ ] 7 4 2

___/2

Read list of letters. The subject must tap with his hand at each letter A. No points if ≥ 2 errors

[ ] FBACMNAAJKLBAFAKDEAAAJAMOFAAB

___/1

Serial 7 subtraction starting at 100 [ ] 93 [ ] 86 [ ] 79 [ ] 72 [ ] 65

4 or 5 correct subtractions: **3 pts**, 2 or 3 correct: **2 pts**, 1 correct: **1 pt**, 0 correct: **0 pt**

___/3

LANGUAGE

Repeat : I only know that John is the one to help today. [ ]
The cat always hid under the couch when dogs were in the room. [ ]

___/2

Fluency / Name maximum number of words in one minute that begin with the letter F [ ] ____ (N ≥ 11 words)

___/1

ABSTRACTION

Similarity between e.g. banana - orange = fruit [ ] train – bicycle [ ] watch - ruler

___/2

DELAYED RECALL

	FACE	VELVET	CHURCH	DAISY	RED
Has to recall words WITH NO CUE	[ ]	[ ]	[ ]	[ ]	[ ]
Optional: Category cue					
Optional: Multiple choice cue					

Points for UNCUED recall only

___/5

ORIENTATION

[ ] Date [ ] Month [ ] Year [ ] Day [ ] Place [ ] City

___/6

© Z.Nasreddine MD Version November 7, 2004

www.mocatest.org

Normal ≥ 26 / 30

TOTAL ___/30

Add 1 point if ≤ 12 yr edu

Glasgow Coma Scale

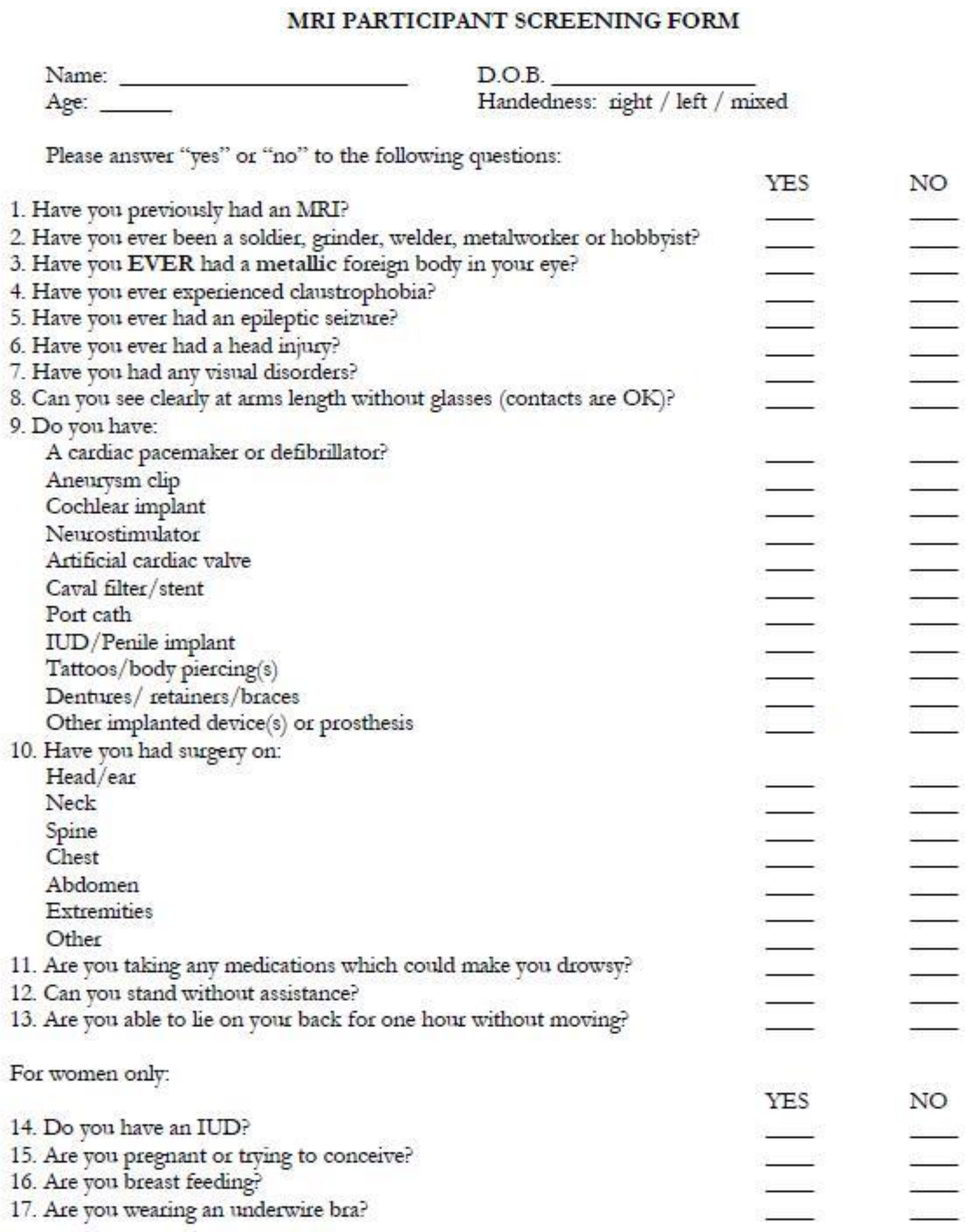
GCS (Teasdale & Jennett, 1974)

Test	Score	Condition
Eye Opening	4	The patient can open his eyes spontaneously
	3	The patient can open his eyes on verbal command
	2	The patient open his eyes only in response to painful stimuli
	1	The patient does not open his eyes in response to any stimulus
Best Verbal Response	5	The patient is oriented and can speak coherently
	4	The patient is disoriented but can speak coherently
	3	The patient uses inappropriate words or incoherent language
	2	The patient makes no verbal response at all
	1	The patient gives no verbal response at all
Best Motor Response	6	The patient can move his arms and legs in response to verbal commands
	2-5	The patient shows movement in response to a variety of stimuli, including pain
	1	The patient shows no movement in response to stimuli

The results of the three tests are added up to determine the patient's overall condition

Total Score	Scale
13-15	Mild Head Injury
9-12	Moderate Head Injury
3-8	Severe Head Injury

Appendix C: MRI Screening Form



MRI PARTICIPANT SCREENING FORM

Name: ______________________ D.O.B. ________________
Age: ______ Handedness: right / left / mixed

Please answer "yes" or "no" to the following questions:

	YES	NO
1. Have you previously had an MRI?	____	____
2. Have you ever been a soldier, grinder, welder, metalworker or hobbyist?	____	____
3. Have you **EVER** had a **metallic** foreign body in your eye?	____	____
4. Have you ever experienced claustrophobia?	____	____
5. Have you ever had an epileptic seizure?	____	____
6. Have you ever had a head injury?	____	____
7. Have you had any visual disorders?	____	____
8. Can you see clearly at arms length without glasses (contacts are OK)?	____	____
9. Do you have:		
A cardiac pacemaker or defibrillator?	____	____
Aneurysm clip	____	____
Cochlear implant	____	____
Neurostimulator	____	____
Artificial cardiac valve	____	____
Caval filter/stent	____	____
Port cath	____	____
IUD/Penile implant	____	____
Tattoos/body piercing(s)	____	____
Dentures/ retainers/braces	____	____
Other implanted device(s) or prosthesis	____	____
10. Have you had surgery on:		
Head/ear	____	____
Neck	____	____
Spine	____	____
Chest	____	____
Abdomen	____	____
Extremities	____	____
Other	____	____
11. Are you taking any medications which could make you drowsy?	____	____
12. Can you stand without assistance?	____	____
13. Are you able to lie on your back for one hour without moving?	____	____

For women only:

	YES	NO
14. Do you have an IUD?	____	____
15. Are you pregnant or trying to conceive?	____	____
16. Are you breast feeding?	____	____
17. Are you wearing an underwire bra?	____	____

Additional comments:

____
____
____
____
____

Before entering the magnet room, please:

	DONE
Remove all jewellery	____
Remove your wristwatch	____
Remove *everything* from your pockets	____
Keep your credit cards in the control room	____

Participant's signature: ______________________________ Date: _______________

Screening individual signature: ________________________ Date: _______________