

Exploring the cognitive correlates of boredom in traumatic brain injury (TBI).

by

Yael Goldberg

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, 2012

©Yael Goldberg 2012

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 required final revisions, as accepted by my examiners.

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

Yael Goldberg

Abstract

Boredom is a common human experience, yet little is known about its underlying neural mechanisms. This thesis first set out to investigate the construct of boredom and more closely examined its relationship to phenomenologically similar mood states of depression, apathy and anhedonia. Next, deficits in sustained attention, and novelty seeking were examined in patients with traumatic brain injury (TBI), who are characterized by atypically high levels of boredom. Study 1 established that although related to varying degrees to apathy, anhedonia, and depression, boredom is indeed a distinct emotional experience. Furthermore, two boredom proneness subtypes - agitated and apathetic - were identified which varied in their relationships to depression. The relationship between boredom and depression was found to be high only in the agitated boredom prone subtype, which is characterised by a high degree of motivation to engage in meaningful, stimulating activities despite the fact that all attempts to do so fail to satisfy. In Study 2, the relationship between boredom proneness and depression was found to be greater in TBI patients than in healthy controls. Using a behavioral measure of sustained attention (SART; Robertson et al., 1997), Study 3 demonstrated a relationship between boredom proneness and sustained attention in healthy controls, such that RTs were faster and commission errors more prevalent in the agitated boredom prone subtype. No relationship between boredom proneness and sustained attention was found in TBI patients. So while attention and boredom show a clear relationship in the healthy brain, this relationship may be disrupted in TBI patients. Finally, Study 4 demonstrated an association between agitated boredom proneness and a preference for novel stimuli across participant groups. In addition, patients had a poorer ability to discriminate between similar and dissimilar stimuli than controls, which was more evident in the agitated boredom prone group. It may be the case then that agitated boredom prone individuals fail to satisfy their desire to engage in stimulating activities in part because they fail to accurately identify when something is indeed novel. Taken together, these results highlight important distinctions between apathetic and agitated boredom proneness, and the way in which these subtypes relate to depression, attention, and novelty seeking, in brain injured patients and healthy controls. More work is needed to determine the role played by boredom in TBI, particularly as this evolves from acute to chronic stages of the illness. Importantly, identifying boredom as a key element in depressive mood disorders, attention deficits (e.g., attention deficit hyperactivity disorder), and novelty seeking behaviour, facilitates the design and implementation of appropriate intervention strategies. For example, it will become increasingly important to deal with boredom as a significant component of depression. Thus, the work presented here represents a novel and important contribution to the study of boredom in that it brings the field one step

closer to understanding and treating the experience. Further investigation with greater numbers of patients is necessary to fully explicate the relationship between boredom and depression, attention, and novelty seeking in TBI.

Acknowledgements

I would like to thank my supervisor, James Danckert, for his mentorship and continued support. His assistance with this project has been immeasurable. More importantly, James has been an incredible inspiration throughout my years in graduate school. He has modeled excellence in scholarship, leadership, and parenthood. He has taught me many valuable lessons that will extend well beyond the completion of my doctorate.

Many thanks to Erik Woody and John Eastwood, and to my committee members, Dan Smilek, Liz Nielsen, Mark Fenske, and Tim Kenyon for their insightful contributions to this work, which have greatly improved its quality.

There are many others who have made it possible for me to do this work, several of whom will be particularly mentioned here. Thank you to my parents, for imbuing me with the qualities necessary to reach this stage, and to my family for supporting me along this journey. I am grateful to my colleagues and friends in the Department of Psychology at UW who have helped me navigate this path.

Last, but certainly not least, I extend my heartfelt gratitude to my husband, Mendy, and my daughter Chaya, for their patience, understanding, and unconditional love. Thank you for always reminding me what is truly important, and for inspiring me to rise above obstacles.

Dedication

This thesis is dedicated to my husband, Mendy, and to my daughter, Chaya.

I would not be me if you were not you.

Table of Contents

AUTHOR'S DECLARATION	ii
Abstract	iii
Acknowledgements	v
Dedication	vi
Table of Contents	vii
List of Figures	ix
List of Tables.....	xi
Chapter 1 Introduction.....	1
Chapter 2 Boredom as a distinct construct.....	4
2.1 Introduction	4
2.2 Methods.....	6
2.2.1 Participants and Procedure	6
2.2.2 Measures.....	6
2.2.3 Data Analysis	10
2.3 Results	10
2.4 Discussion	27
Chapter 3 Exploring the relationship between Boredom and Depression in TBI	31
3.1 Introduction	31
3.2 Participant Characteristics	31
3.3 Boredom	34
3.4 Depression.....	35
3.5 Relationship between boredom and depression	36
3.6 Discussion	41
Chapter 4 Exploring the relationship between Boredom and Attention in TBI	43
4.1 Introduction	43
4.2 Methods and Procedure.....	45
4.3 Results	47
4.3.1 Reaction Time	47
4.3.2 Accuracy.....	56
4.3.3 Relationship between sustained attention and boredom proneness.....	58
4.4 Discussion	66

Chapter 5 Exploring the relationship between Boredom and Novelty Preference in TBI.....	70
5.1 Introduction.....	70
5.2 Methods and Procedures	71
5.3 Results.....	74
5.3.1 Performance on the CBT.....	74
5.3.2 Relationship between preference for novelty and boredom.....	77
5.4 Discussion.....	83
Chapter 6 Conclusion.....	86
References.....	92
Appendix A Subscales of the Boredom Proneness Scale (Ahmed, 1990).....	104

List of Figures

<i>Figure 2.1. A 4-Factor model measuring the relationship between boredom, apathy, anhedonia and depression.....</i>	12
<i>Figure 2.2 A 4-factor model depicting the confound between Boredom and the Behaviour subscale of the AES (indicated in grey).</i>	14
<i>Figure 2.3. A 4-factor model depicting the partial correlation between boredom and depression, with apathy and anhedonia held constant.....</i>	18
<i>Figure 2.4. A 4-factor model with 2 subscales of the BPS.</i>	22
<i>Figure 2.5. Correlations between the two subscales of the BPS and the two subscales of the BDI-II across boredom proneness subtypes.</i>	24
<i>Figure 2.6. Correlations between boredom and apathy within the 2 boredom proneness subtypes.</i>	26
<i>Figure 3.1. Relationship between BDI-II and BPS scores across participant groups.</i>	38
<i>Figure 3.2. BDI-II scores across participant group, characterized by the experience of apathetic vs. agitated boredom proneness.</i>	40
<i>Figure 4.1. Schematic representation of trial sequences in the Sustained Attention to Response Task (SART).</i>	46
<i>Figure 4.2. Mean SART response time across participant group.</i>	49
<i>Figure 4.3. Mean reaction time for first half (trials 1-112) and second half (trials 113-225) of the SART task across group.</i>	51
<i>Figure 4.4. Mean reaction time over 225 trials of SART, grouped into 9 blocks of 25 trials each.....</i>	53
<i>Figure 4.5. Mean SART reaction time variance (left) adjusted for age effects (right) across participant group.</i>	55
<i>Figure 4.6. Relationship between age and number of SART omission and commission errors across group.</i>	57
<i>Figure 4.7. SART Reaction time across boredom proneness subtype.</i>	61
<i>Figure 4.8. Difference in reaction time surrounding errors of commission across boredom subtype, calculated by subtracting pre error from post error reaction time.</i>	63
<i>Figure 4.9. Percentage of commission errors across boredom subtype.</i>	65
<i>Figure 5.1. Cognitive Bias Task.</i>	73
<i>Figure 5.2. Scores on the Cognitive Bias Task across group.....</i>	75
<i>Figure 5.3. CBT scores across group in the apathetic and agitated boredom prone subtypes.....</i>	78
<i>Figure 5.4. CBT difference scores across boredom proneness subtype.....</i>	80

Figure 5.5. CBT “like” scores across boredom proneness subtype..... 82

List of Tables

<i>Table 2.1. Descriptive statistics and correlations between measures in Study 1, including reliability estimates</i>	9
<i>Table 3.1. Participant Characteristics of studies 2, 3 & 4</i>	33

Chapter 1

Introduction

We all experience days in which it is nearly impossible to generate interest in activities; nothing maintains our interest, focus, or attention, and everything seems ‘boring’. It is such a universal human experience that we dedicate very little time or attention to actually *defining* what it means to be bored. Indeed, the study of boredom has been broad in scope, yet limited by the absence of a consistent definition of the construct (Darden & Marks, 1999; Geiwitz, 1966; Harris, 2000; Hill & Perkins, 1985; Mikulas & Vodanovich, 1993; Vodanovich, 2003). For example, boredom has been defined as the feeling of having no intention or purpose (Heidegger, 1995; Mills, 1959), as well as a state of “being in limbo” (Heidegger, 1995) in which a *desire* to engage in the world yields little success (Fenichel, 1951). The lack of any clear definition of boredom severely limits the meaningful identification of individuals with atypically high boredom proneness, making it difficult to develop appropriate and effective therapeutic interventions (Binnema, 2004). Boredom can operate adaptively as a personalized signal to discontinue an activity and move on to something new. However, when boredom is experienced at atypically high levels for a majority of the time, its function becomes maladaptive, as it interferes with optimally carrying out day-to-day activities. Indeed, boredom has been shown to be more pervasive in psychiatric and neurological populations, ranging from depression to traumatic brain injury (Binemma, 2004; Cicerone, Levin, Malec, Stuss & Whyte, 2006; Hamilton, Haier, & Buchsbaum, 1984; Seel & Kreutzer, 2003; Vodanovich, 2003; Vodanovich, Verner & Gillbride, 1991). The presence of boredom in these disorders may represent a serious impediment to recovery, as engaging such patients in treatment and rehabilitation becomes extremely difficult.

A range of theories have been proposed to explain the experience of boredom focusing to varying degrees on motivational and cognitive components. There are those who suggest that boredom is externally driven in that it is the affective result of impoverished external stimuli perhaps due to repetition, and a lack of cognitive skills necessary to intrinsically generate interest (Berlyne, 1960; Darden

& Marks, 1999; Geiwitz, 1966; Hebb, 1966; Hill & Perkins, 1985; Mikulas & Vodanovich, 1993; Reid, 1986; Shaw, 1996; Watson, Hickman, Morris, Stutz & Whitting, 1994; Wegner, Flisher, Chikobvu, Lombard, & Kling, 2008). In contrast, others propose that boredom is internally driven, and may be the result of a general lack of meaning and/or spirituality in life (Barbalet, 2000; Binnema, 2004; Csikszentmihalyi, 1975; Eastwood, Cavaliere, Fahlman, & Eastwood, 2007; Hamilton et al., 1984; Leary, Rogers, Canfield, & Coe, 1986; MacDonald & Holland, 2002; Melton & Schulenberg, 2007; Pattyn, Neyt, Heridericlx, & Soetens, 2008; Seib & Vodanovich, 1998; Simmel, 1997; Vodanovich, 2003). More often, boredom has been conceptualized as an emotional state resulting from inefficient or deficient emotional, cognitive or attentional processes (Cheyne, Carriere & Smilek, 2006; Csikszentmihalyi, 1975; Eastwood et al., 2007; Eastwood, Frischen, Fenske, & Smilek, in press; Hamilton et al., 1984; Seib & Vodanovich, 1998), created by suboptimal levels of cortical arousal (Pattyn et al., 2008; Vodanovich, 2003) or “neurasthenia” (overstimulation; Pattyn et al., 2008; Simmel, 1997). Thus, across varied definitions, boredom has been taken to reflect a state of core motivational deficits accompanied by a phenomenological experience of a lack of interest or affective engagement. However, the cognitive and neural correlates of boredom are poorly understood. In short, boredom is a complex, difficult to define construct. Furthermore, it is difficult to distinguish boredom from other, similar mood states including apathy, anhedonia, and depression.

The objective of this thesis was not to generate a comprehensive description of the phenomenon of boredom, or even to define it more fully than has been done in previous literature. Instead, the goal was to first determine whether boredom represents a *distinct* affective construct, second, to examine some of the cognitive correlates of the experience and finally, to explore the role played by boredom in individuals who have suffered traumatic brain injury. This research represents the first steps towards a clearer understanding of boredom and its relationship to cognitive functioning, which will ultimately have implications for treating psychopathological and neurological disorders in which boredom is pervasive. In Chapter 2, the relationship between boredom and other, related mood states was explored. Although found to be related to varying degrees to depression, apathy, and anhedonia, boredom is established as a distinct

emotional construct. Two subtypes of boredom prone individuals were also identified, which are labeled here as agitated and apathetic. Although these labels are necessarily tentative at this point, they have intuitive appeal. The agitated subtype represents individuals who are highly motivated to engage in meaningful and stimulating activities and experience the failure to satisfy that desire as aggressively dissatisfying. In contrast, the apathetic boredom prone individual lacks that motivational component that would provoke a desire to redress the emotional experience of boredom - they are bored, but unmotivated to change. Future work will undoubtedly challenge these definitions and ultimately refine them. They operate here as a useful heuristic to explore differences in cognitive profiles across the two groups. The rest of the thesis is devoted to exploring the cognitive correlates of boredom. This is investigated in individuals with traumatic brain injury (TBI), a population that reportedly experiences atypically high levels of boredom (Al-Adawi, Powell & Greenwood, 1998). Chapter 3 outlines the characteristics of the three groups of individuals that represent the study sample, namely, Severe TBI, Concussion, and Controls. The chapter then describes the relationships between boredom and depression, a common consequence of TBI. The chapters that follow go on to explore the relationships between boredom and deficits commonly found post-injury in the cognitive domains of sustained attention (Chapter 4), and novelty seeking (Chapter 5). The concluding chapter summarizes and highlights the key findings put forth in the dissertation.

Chapter 2

Boredom as a distinct construct

2.1 Introduction

Despite the relatively poor understanding of the concept of boredom, there have been many attempts to empirically measure the construct. However, currently available measures of boredom possess limited amounts of validity and reliability. Furthermore, these measures fail to address the complex nature of the experience. That is, some scales assess only *specific kinds* of boredom (e.g., individual differences in perception of boredom *during leisure*; Leisure Boredom Scale; Iso-Ahola & Weissinger, 1990), whereas others measure only *specific responses* to boredom (e.g., Boredom Coping Scale; Hamilton et al., 1984). To date, the only empirically validated, comprehensive tool for measuring boredom is the Boredom Proneness Scale (BPS; Farmer & Sundberg, 1986). The BPS is a 28-item self-report questionnaire measure of trait susceptibility to the experience of boredom. However, the BPS is not without limitations. First, the factor structure of the BPS remains somewhat controversial, with factor analyses yielding anywhere from two to seven factors (Melton & Schulenberg, 2009; Vodanovich, 2003). It is worth noting that in each of these factor analyses two consistent factors have emerged, measuring respectively the perceived need for excitement or interest in the environment (External Stimulation), and the ability to keep oneself focused, interested and entertained (Internal Stimulation; Ahmed, 1990; Farmer & Sundberg, 1986; Vodanovich & Kass, 1990; Vodanovich, Wallace & Kass, 2005). Notwithstanding, inconsistency in the exact factor structure of the BPS makes it difficult to define subscales and impedes the determination of the specific factors contributing to the experience of boredom (Musharbash, 2007). More importantly, scores on the BPS are significantly and positively correlated with a wide range of other related affective states including depression, hopelessness, loneliness, negative self-awareness, hostility, aggression, apathy, and amotivation (Ahmed, 1990; Farmer & Sundberg, 1986; Gordon, Wilkinson, McGown, & Jovanoska, 1997; Seib & Vodanovich, 1998; Vodanovich et al., 1991; Buss & Perry, 1992). Indeed, Farmer and Sundberg (1986) reported high correlations with a number of distinct scales measuring

depression and prominent symptoms of depression. These associations lead to two critical questions. First, is boredom a distinct experience from these other, phenomenologically related states? Second, can the current measures of boredom adequately distinguish it from these other related affective states?

Apathy, anhedonia, depression and boredom all share, at least on face value, some core motivational and affective components. Apathy is defined as a neuropsychiatric syndrome of primary motivational loss not attributed to emotional distress, intellectual impairment or diminished consciousness (Levy et al., 1998; Marin, 1991). Anhedonia is defined as the loss of capacity or failure to experience pleasure (Klein, 1974; Leventhal, Chasson, Tapia, Miller, & Pettit, 2006; Meehl, 1987; Ribot, 1896; Snaith, 1993) and although the absence of reward properties may in turn lead to a state of amotivation, the construct largely reflects affective phenomena. Nevertheless, the presence of a motivational component across all three constructs – apathy, anhedonia and boredom – makes it difficult to distinguish among them. Finally, depression has been characterized by both significant affective (e.g., loss of interest or pleasure) and motivational dysfunction (e.g., inability to engage in normative activity). Perhaps unsurprisingly then, consistently high correlations have been demonstrated between boredom and depression (Farmer & Sundberg, 1986; Vodanovich, 2003). Although some research suggests the two states differ by virtue of their quality and intensity (Farmer & Sundberg, 1986; Kemper, 1987), the relationship between boredom and depression remains unclear. Despite the fact that previous research has shown a high degree of relationship between boredom and depression (Farmer & Sundberg, 1986, Vodanovich, 2003), the relationship between boredom and apathy or anhedonia has not been examined in detail. However, one would intuitively expect the relationships between boredom, apathy, and anhedonia to be quite high given their shared features.

Given the high degree of conceptual overlap between apathy, anhedonia, boredom and depression, an important first step in understanding the role of boredom in cognitive functioning is to determine the extent to which the construct is independent from related affective states. Study one attempted to do this by contrasting boredom with the related constructs of apathy, anhedonia, and depression in order to establish empirically whether boredom represents a unique mood state.

2.2 Methods

2.2.1 Participants and Procedure

Eight hundred and twenty three undergraduate students (age range 16 to 56) were recruited from the University of Waterloo and York University (males = 243, $M_{age} = 20$ years, $SD = 3.7$). Questionnaires were anonymously administered on-line and participants received course credit or remuneration for their participation. The study received institutional ethics approval from both universities.

2.2.2 Measures

Boredom. Three measures were used as indicators of boredom. The 28-item Boredom Proneness Scale (BPS; Farmer & Sundberg, 1986) measures people's general susceptibility to experiencing boredom (e.g., statements such as "I find it easy to entertain myself" are rated on a 7-point Likert scale). Responses are summed for a total boredom proneness score ranging from 28 to 196 (higher scores indicate greater boredom proneness). Estimates of internal consistency (Cronbach's α) have ranged from .79 to .84 across numerous studies (Vodanovich et al., 2005). The BPS has demonstrated high convergent validity with other boredom measures ($r_s = .25$ or higher; reviewed in Farmer & Sundberg, 1986), as well as measures of personality, mood, negative affect, life satisfaction, cognitive failures, attention, and time perception (Vodanovich, 2003).

The 10-item Boredom Coping scale (BC; Hamilton et al., 1984) assesses the ability to cope with boredom (participants make forced choice judgements between statements such as "I get bored seeing the same old faces" or "I continue to be interested in familiar everyday faces"). Summed scores range from 0 to 10, with higher scores indicating greater ability to cope with boredom. The BC scale has demonstrated high internal consistency ($\alpha = .67$) and test-retest reliability ($r = .64$; Hamilton et al., 1984).

The 29-item Multidimensional State Boredom Scale (MSBS; Fahlman, Mercer-Lynn, Flora, & Eastwood, 2011) measures current feelings of boredom (e.g., participants rate statements such as "Time is passing slower than usual" on a 7-point Likert scale). Scores are summed for a total ranging from 29 to 203 (higher scores indicate higher levels of state boredom). Reliability estimates range from .94 to .96

(Fahlman et al., 2011). Validity was indicated by significant correlations between scores on the MSBS and the BPS, ranging from $r = 0.39$ to 0.63 (Fahlman et al., 2011).

Apathy. The three subscales of the Apathy Evaluation Scale-Self report version (AES-S; Marin, 1990; Marin, 1991; Marin, Biedrzycki, & Firinciogullari, 1991) were used as indicators of apathy. The 18-item questionnaire evaluates the presence of apathy across three domains of goal directed behaviour; overt activity (Behaviour subscale), thought content (Cognition subscale), and emotional responsiveness (Emotion subscale). Participants rate statements such as “I am interested in things” on a 4-point Likert scale based on how much the statement describes their thoughts, feelings, and activities in the past month. Responses within each subscale are summed, and the three subscales are combined to produce a total ranging from 18 to 72 (higher scores indicate greater levels of apathy). The AES has demonstrated high internal consistency ($\alpha = .88$) and test-retest reliability ($r = .78$; Marin, 1991).

Anhedonia. Two measures were used as indicators of anhedonia. The 14-item Snaith-Hamilton Pleasure Scale (SHAPS; Snaith, Hamilton, Morley & Humayan, 1995) measures the capacity to experience pleasure in the last few days (participants rate their agreement with statements such as “I would enjoy my favorite television or radio program” on a 4-point Likert scale). Responses are summed to produce a total ranging from 0 to 14 (higher scores indicate greater capacity to experience pleasure). Internal consistency estimated using Cronbach’s α was reported as $.86$ (Leventhal et al., 2006), and was $.857$ when estimated using the Kuder-Richardson formula applicable to non-parametric data (Guilford, 1954; Snaith et al., 1995).

The 36-item Fawcett-Clark Pleasure Capacity Scale (FCPS; Fawcett, Clark, Scheftner & Gibbons, 1983) measures current ability to experience pleasure (participants rate how pleasurable they find situations such as “listening to beautiful music” on a 5-point Likert scale). Responses to all items are averaged to produce a mean pleasure score ranging from 1 to 5 (higher scores indicate greater capacity to experience pleasure). Across studies (Fawcett et al., 1983; Leventhal et al., 2006), the FCPS has demonstrated high internal consistency ($\alpha s = .92$ to $.96$) and convergent validity with other measures such as the SHAPS, the Beck Depression Inventory-Second Edition (BDI-II; Beck, Steer, & Brown, 1996) and

the pleasure scales of the Weissman Social Adjustment Scale Self-Report (Weissman & Bothwell, 1976), the Beck Hopelessness Scale (Beck & Steer, 1988) and the Chapman Anhedonia Scale (Chapman, Chapman & Raulin, 1976).

Depression. Existence and severity of depression was indicated by the two subscales of the BDI-II (Beck, Steer, & Brown, 1996), a widely used self-report measure of depression. The BDI-II is composed of 21 items, each consisting of four self-evaluative statements scored from 0 to 3. Measures of depression are taken across two major domains of symptomatology: somatic-affective and cognitive, comprising the two subscales of the BDI-II (Beck et al., 1996). Responses from each subscale are tallied, and then combined to produce a total BDI-II score ranging from 0 to 63 (higher scores indicate greater levels of depression). Internal consistency estimates ranged from .92 to .93, and test-retest reliability was reported as .93 (Beck et al., 1996).

Descriptive statistics as well as correlations and current reliability estimates for all scales used are presented in Table 2.1.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. BPS	(.860)													
2. BPS:IN	.687**	(.564)												
3. BPS:EX	.958**	.459**	(.858)											
4. BC	.634**	.423**	.622**	(.652)										
5. MSBS	.711**	.391**	.723**	.523**	(.951)									
6. AES	.259**	.162**	.256**	.146**	.267**	(.931)								
7. AES:B	.357**	.213**	.356**	.197**	.346**	.871**	(.611)							
8. AES:C	.205**	.123**	.205**	.109**	.200**	.968**	.776**	(.891)						
9. AES:E	.177**	.128**	.168**	.100**	.188**	.882**	.692**	.837**	(.505)					
10. SHAPS	.319**	.226**	.269**	.249**	.210**	.229**	.261**	.207**	.205**	(.787)				
11. FCPS	.202**	.193**	.164**	.157**	.126**	.117**	.117**	.086*	.141**	.375**	(.911)			
12. BDI-II	.529**	.321**	.527**	.369**	.646**	.266**	.333**	.213**	.227**	.317**	.148**	(.926)		
13. BDI-II: SA	.515**	.315**	.516**	.389**	.636**	.283**	.328**	.235**	.245**	.321**	.138**	.953**	(.884)	
14. BDI-II: C	.469**	.288**	.464**	.269**	.569**	.202**	.284**	.148**	.176**	.261**	.137**	.911**	.745**	(.872)
Kurtosis ^b	-.48	.043	-.197	-.55	.01	-.890	-.68	-.90	-.76	7.51	.40	1.44	.69	1.90
Skew ^a	-.06	-.117	.057	.25	-.07	.458	.27	.59	.46	2.49	.37	1.18	.98	1.40
SD	19.50	6.15	15.24	2.31	32.63	12.02	2.89	6.24	1.67	1.90	0.44	9.43	5.85	4.32
M	100.54	32.45	65.2	3.95	103.88	37.90	9.89	16.75	4.42	12.91	3.77	11.27	7.24	4.07

Table 2.1. Descriptive statistics and correlations between measures in Study 1, including reliability estimates

Note: Cronbach's α levels are presented on the main diagonal in parentheses. $N = 774$ to 823. M = mean, SD = standard deviation
^a SE = .09, ^b SE = .17, ** $p < .01$, two-tailed. * $p < .05$, two-tailed.

2.2.3 Data Analysis

Structural equation modeling was applied to the data using Amos 7.0 (Arbuckle, 2006). The construct of *Boredom* was indicated by the three scales described above (BPS, BC, and the MSBS). *Apathy* was indicated by the three subscales (Behaviour [Beh], Cognition [Cog], and Emotional Responses [Emo]) of the AES. *Anhedonia* was indicated by the SHAPS and the FCPS. Finally, *Depression* was indicated by the two subscales of the BDI-II (Somatic-Affective [Sad] and Cognitive [Cd]). Random measurement error was included in the model and missing data (arising from rare cases when a participant declined to answer an item on a given scale), were included in the analysis using the full information maximum likelihood method (Anderson, 1957; Arbuckle, 2006).

Several measures of fit were used to determine the best model for the data. These included Chi-Square (χ^2) – an index of the *lack* of fit of the model to the data (e.g., a perfect fit would yield a chi-square of zero). Given the fact that the chi-square test is sensitive to sample size we also used the comparative fit index (CFI; Bentler, 1990), root-mean-square error of approximation (RMSEA; Browne & Cudeck, 1993), and probability of close fit (pclose; Browne & Cudeck, 1993). Both the CFI and RMSEA index how well the model fits the data. The pclose provides a test of whether the deviation from close fit, defined as RMSEA less than or equal to .05, would likely occur due to sampling error alone. A model with a CFI value greater than .95 and a RMSEA value of less than .08 is considered to represent a very good fit to the data (Bentler, 1990; Browne & Cudeck, 1993). A high (non-significant) pclose value indicates that the observed deviation from fit to the model would likely occur due to sampling error.

2.3 Results

If boredom is indeed distinct from apathy, anhedonia, and depression, then structural modeling of the data should indicate that the best fitting model was one in which all four constructs were psychometrically distinct. To test this hypothesis, a model was estimated in which each construct was represented by a

separate factor. The 4-factor model adequately fit the data, $\chi^2(29, N = 823) = 259.28, p < .001$, CFI = 0.95, RMSEA = 0.10, $p_{close} < .001$ (Figure 2.1).

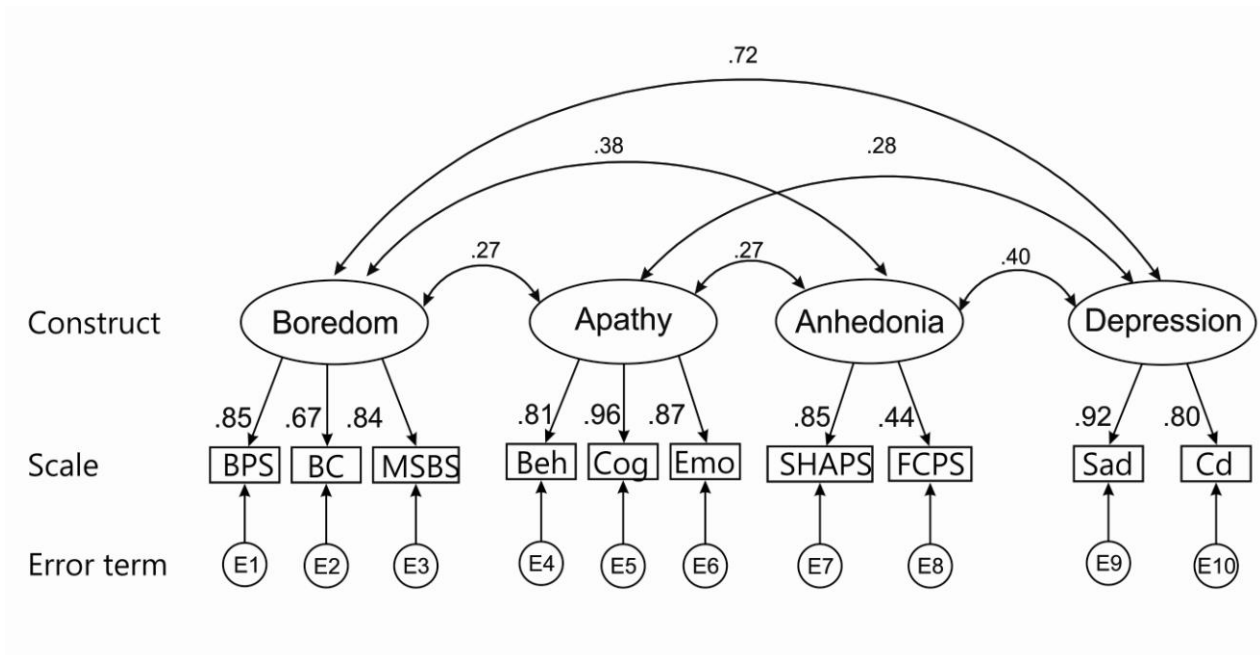


Figure 2.1. A 4-Factor model measuring the relationship between boredom, apathy, anhedonia and depression.

Measures used to evaluate each construct (i.e., scales and/or subscales) are indicated in rectangles (abbreviations as indicated in the text). Error variables (E1 – E10), reflecting imperfect measurement of indicators on latent variables are represented in circles. Correlations between latent constructs are presented above the bidirectional arrows representing the strength of the relationships between those constructs. The strength of the relationships between the constructs and the measures used to assess them is indicated by a standardized regression coefficient (numbers are presented adjacent to the straight line arrows linking constructs and their scales). The proportion of variance in each measure attributable to the latent construct is represented by the square of the standardized regression coefficient.

The RMSEA, however, suggested that the model could be improved. To determine which component of the model reduced the fit, model modification indices were utilized indicating there was one source of lack of fit in the model – the absence of a path from Boredom to the Behaviour subscale of the AES (Beh). A revised model including this path provided a better fit to the data, $\chi^2(28, N = 823) = 170.88, p < .001, CFI = 0.97, RMSEA = 0.08, p_{close} < .001$ (Figure 2.2).

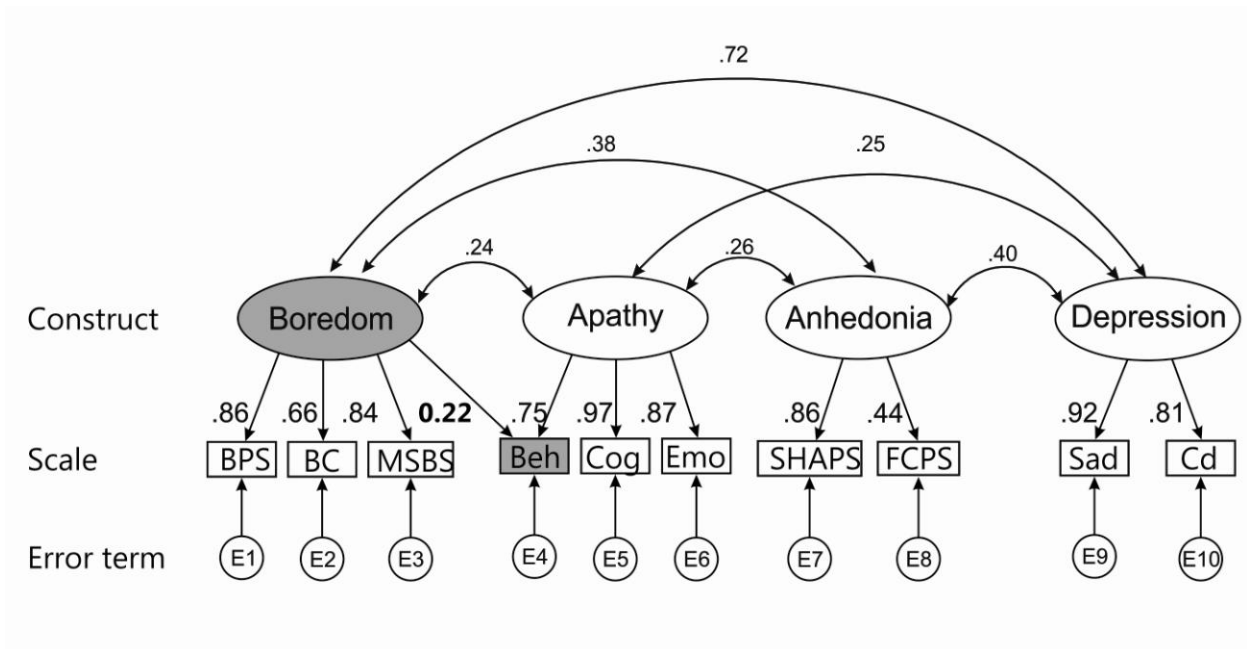


Figure 2.2 A 4-factor model depicting the confound between Boredom and the Behaviour subscale of the AES (indicated in grey).

Note: this model was the final measurement model adopted. All other aspects are as indicated for Figure 2.1.

The degree of the relationship between boredom and the Behavior subscale of the AES was small but significant ($r = 0.22, p < .001$), and indicated that the Behaviour subscale of the AES was not only a measure of apathy, but also measured some components of boredom.

A review of all the individual items from the Behaviour subscale of the AES highlighted the fact that as a measure of the motivational impairment found in apathy, the scale also unavoidably taps into the motivational aspect of boredom. Thus, since motivational impairment plays a role in both boredom and apathy, it is not surprising that measures of a lack of motivation would capture both constructs.

Because the Behaviour subscale of the AES confounded apathy and boredom, a model was estimated from which the subscale had been removed. The resulting model fit the data well, $\chi^2(21, N = 823) = 153.95, p < .001, CFI = 0.96, RMSEA = 0.09, pclose < .001$, without changing the relationships between boredom and the other constructs. Thus, although the Behaviour subscale of the AES measured both apathy and boredom, its presence did not distort the results. Therefore, the final model adopted (Figure 2.2) included this measure.

Although the 4-factor model fit the data reasonably well, the possibility that boredom, apathy, anhedonia and depression were *not* psychometrically distinct was explored further. A model was estimated in which 1 factor represented all 4 constructs. The 1-factor model yielded a very poor fit to the data, $\chi^2(35, N = 823) = 2171.14, p < .001, CFI = 0.5, RMSEA = 0.272, pclose < .001$. This ill-fitting 1-factor model indicated that multiple constructs were required to explain the pattern of correlations among the variables. A hierarchical factor model was also explored to evaluate whether the intercorrelations between the first order factors (boredom, apathy, anhedonia and depression) could be explained in terms of an overarching second order factor (factor *X*). Again, the resulting model yielded a very poor fit to the data, $\chi^2(35, N = 823) = 813.231, p < .001, CFI = 0.818, RMSEA = 0.164, pclose < .001$. The inferior fit of these two alternate designs confirmed that the best fitting model was one in which boredom, apathy, anhedonia and depression were represented by distinct factors.

The final measurement model (Figure 2.2) consisted of high loadings of most measures on the latent variables as indicated by the standardized regression coefficients, representing the strength of the

relationship between the latent constructs and the measures used to evaluate them. The proportion of variance in each measure attributable to the latent construct is represented by squared multiple correlations: the construct of boredom significantly explained 73% ($p < .001$) of the variance in the BPS, 44% ($p < .001$) of the variance in the BC, and 71% ($p < .001$) of the variance in the MSBS. Anhedonia accounted for 73% of the variance in the SHAPS, yet only 19% of the variance in the FCPS suggesting that the FCPS is a poor measure of anhedonia. Apathy, together with a small contribution from boredom, accounted for 69% ($p < .001$) of the variance in the Behaviour subscale of the AES. Apathy explained 93% ($p < .001$) of the variance in the Cognition subscale, and 75% ($p < .001$) of the variance in the Emotional Responses subscale. Depression significantly accounted for 85% ($p < .001$) of the variance in the Somatic-Affective subscale and 65% ($p < .001$) of the variance in the Cognitive subscale of the BDI-II.

An advantage of the structural equation model is that the estimated correlations among the four latent constructs are corrected for random measurement error. Therefore, they should provide a more accurate picture of the relation between boredom and the other constructs than simple correlations between pairs of measures. There were significant yet low estimated correlations between boredom and apathy ($r = 0.24, p < .001$), and boredom and anhedonia ($r = 0.38, p < .001$). The correlation between boredom and depression was substantially higher ($r = 0.72, p < .001$). With such a high correlation between boredom and depression, it was possible that the two constructs overlapped to such a degree that they were psychometrically indistinguishable from one another. That is, they may have been two components of the same construct. One means of testing this possibility was to fix the correlation between boredom and depression at 1.00 to model a scenario in which the two constructs are essentially considered to be identical. The resulting model yielded a very poor fit to the data, $\chi^2 = (32, N = 823) = 536.31, p < .001$, CFI = 0.88, RMSEA = 0.14, $pclose < .001$. Additionally, the difference in fit between the original (Figure 2.2) and current models was significant, $\Delta \chi^2 (3, N = 823) = 277.28, p < .001$, suggesting that although boredom and depression were highly related, they were empirically distinct.

According to the Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition, Text Revision (American Psychiatric Association, 2000) both apathy and anhedonia are considered diagnostic features of depression, however, boredom is not. Therefore, the possibility that the relation between boredom and depression was *mediated* by apathy and anhedonia was investigated via a model in which boredom and depression were partially correlated, with apathy and anhedonia held constant. The resulting model (Figure 2.3) fit the data well, yielding approximately the same fit statistics as the final measurement model, $\chi^2(28, N = 823) = 170.88, p < .001, CFI = 0.97, RMSEA = 0.08, p_{close} < .001$. More importantly, although the correlation between boredom and depression was slightly reduced, it was still significant ($r = .66, p < .001$), indicating that the contribution of apathy and anhedonia to the relationship between boredom and depression was minimal.

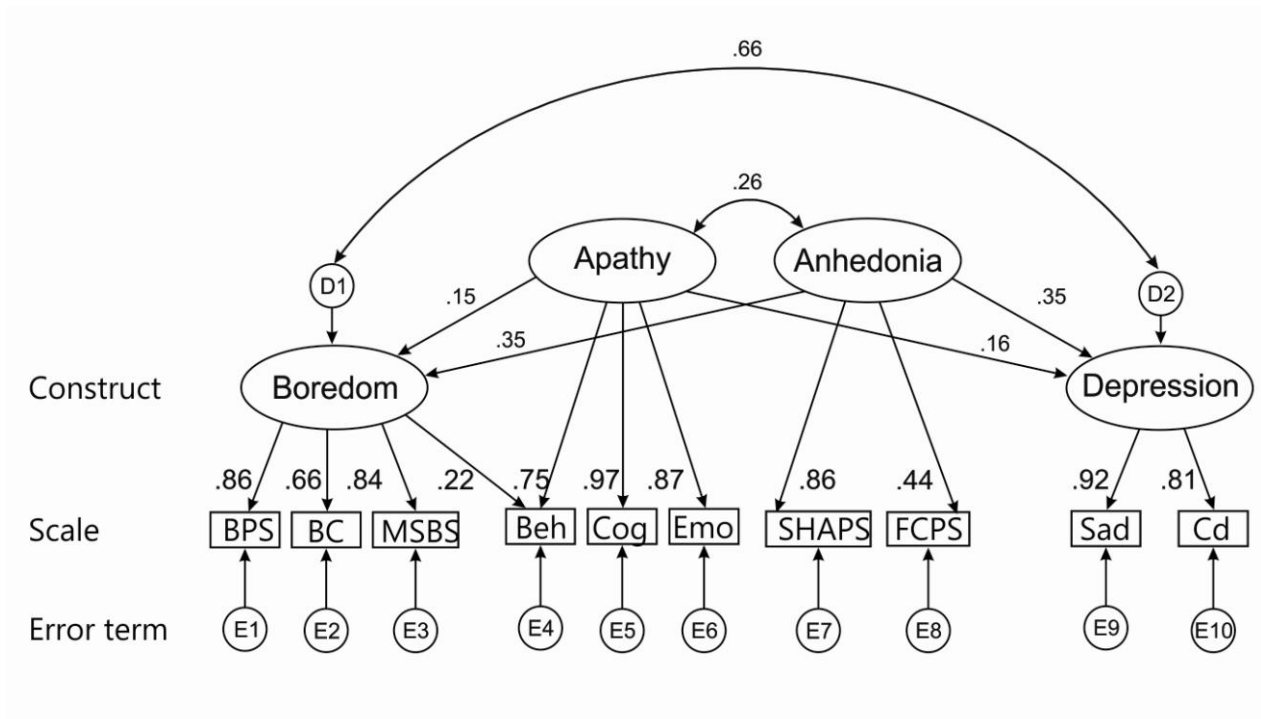


Figure 2.3. A 4-factor model depicting the partial correlation between boredom and depression, with apathy and anhedonia held constant.

The proportion of variance in boredom unexplained by apathy or anhedonia is represented by D1, and the proportion of variance in depression unexplained by apathy or anhedonia is represented by D2. All other aspects of the figure are as indicated in Figure 2.1.

Another possible explanation for the high degree of correlation between boredom and depression came from examining the scales used to measure both constructs. There are a number of highly similar items in the BPS and the BDI-II. More specifically, both scales include questions about one's ability to concentrate, activity level, loss of interest, enjoyment, restlessness and low self-image. It was possible that these similarities in content could account for the high correlation between boredom and depression. Therefore, a model was estimated in which overlapping items in the BPS and BDI-II were removed from the analysis to determine their impact on the relation between the constructs. The resulting model provided similar results when compared to the final model adopted, $\chi^2(28, N = 823) = 163.71, p < .001$, CFI = 0.97, RMSEA = 0.08, $p_{close} < .001$. There were small changes in the correlations between boredom and depression (from $r = .72, p < .001$ to $r = .70, p < .001$), and boredom and apathy (from $r = .24, p < .001$ to $r = .23, p < .001$). The correlation between boredom and anhedonia remained unchanged ($r = .38, p < .001$). Thus, removing the overlapping items from the analysis did not substantially improve the model fit, or change the correlations between the constructs.

To further explore the relationship between boredom and depression, the BPS was subdivided into 2 subscales (Appendix A), representing the two factors which emerged in factor analyses of the BPS (Ahmed, 1990). The Internal Stimulation subscale (IN) indexed the ability to keep oneself focused, interested, and entertained through internal stimulation, and the External Stimulation subscale (EX) indexed the perceived need for excitement, challenge, or interest in the external environment (Ahmed, 1990). Correlations were estimated to determine which component of boredom was most related to the experience of depression. Results showed that the external subscale was more highly correlated with the BDI-II ($r = .527, p < .001$) than the internal subscale ($r = .321, p < .001$). These correlations were significantly different from each other, $z = -6.50, p < .001$ (DeCoster, 2007)¹. Moreover, the external subscale was more highly correlated with both the somatic-affective ($r = .516, p < .001$) and cognition ($r = .464, p < .001$) subscales of the BDI-II than was the internal subscale ($r = .315, p < .001$ and $r = .288, p < .001$ respectively). Most importantly, the squared correlation coefficients (r^2) revealed that 28% ($p < .001$) of the variance in the BDI-II was attributable to the external subscale, while only 10% ($p < .001$) was attributable to the internal subscale. This pattern of correlation suggested that a perceived lack of interest in, or alternatively, a need for excitement from, the environment was most responsible for the relationship between boredom and depression.

Due to the confound between apathy and boredom in the final SEM model adopted (Figure 2.2), correlations were estimated between apathy and the two subscales of the BPS. Results yielded significant correlations between the internal subscale and measures of apathy (Behavior: $r = .213, p < .001$; Cognition: $r = .123, p < .001$; Emotional Responses: $r = .128, p < .001$), with the greatest correlation between the internal subscale and the behavior subscale of the AES. Estimated correlations between the external subscale and apathy followed a similar pattern (Behavior: $r = .356, p < .001$; Cognition: $r = .205, p < .001$; Emotional Responses: $r = .168, p < .001$). Whereas the internal subscale of the BPS explained

¹ Dissattenuated correlations between both subscales of the BPS and the BDI-II were calculated to investigate the impact of subscale content on correlational values. Results showed values that were similarly different in magnitude ($r_1 = .43, r_2 = .57$). Thus, subscale items were not making a significant impact on correlations between measures.

.05% of the variance in the behavior subscale of the AES, the external subscale of the BPS explained 13% of the variance in the behavior subscale of the AES. This suggested that the external subscale of the BPS was most responsible for the relationship between boredom and the *behavioural* component of apathy.

To verify these findings using SEM, a model was estimated in which there were 4 indicators of boredom, namely, the 2 subscales of the BPS (i.e., internal and external), the BC, and the MSBS. The resulting model (Figure 2.4) provided similar results when compared to the final model adopted (Figure 2.2), $\chi^2(37, N = 823) = 188.71, p < .001$, CFI = 0.97, RMSEA = 0.07, $p_{close} < .001$. The correlation between boredom and depression changed slightly (from $r = .72, p < .001$ to $r = .71, p < .001$), as did the correlation between boredom and anhedonia ($r = .37, p < .001$). The correlation between boredom and apathy ($r = .24, p < .001$) remained unchanged. Perhaps more importantly, the construct of boredom significantly explained 75% ($p < .001$) of the variance in the external subscale of the BPS, but only 27% ($p < .001$) of the variance in the internal subscale. This suggested that the external stimulation subscale of the BPS represents a far better measure of boredom in general. Indeed, the reliability estimate for the external subscale was $\alpha = .858$, compared with only $\alpha = .564$ for the internal subscale.

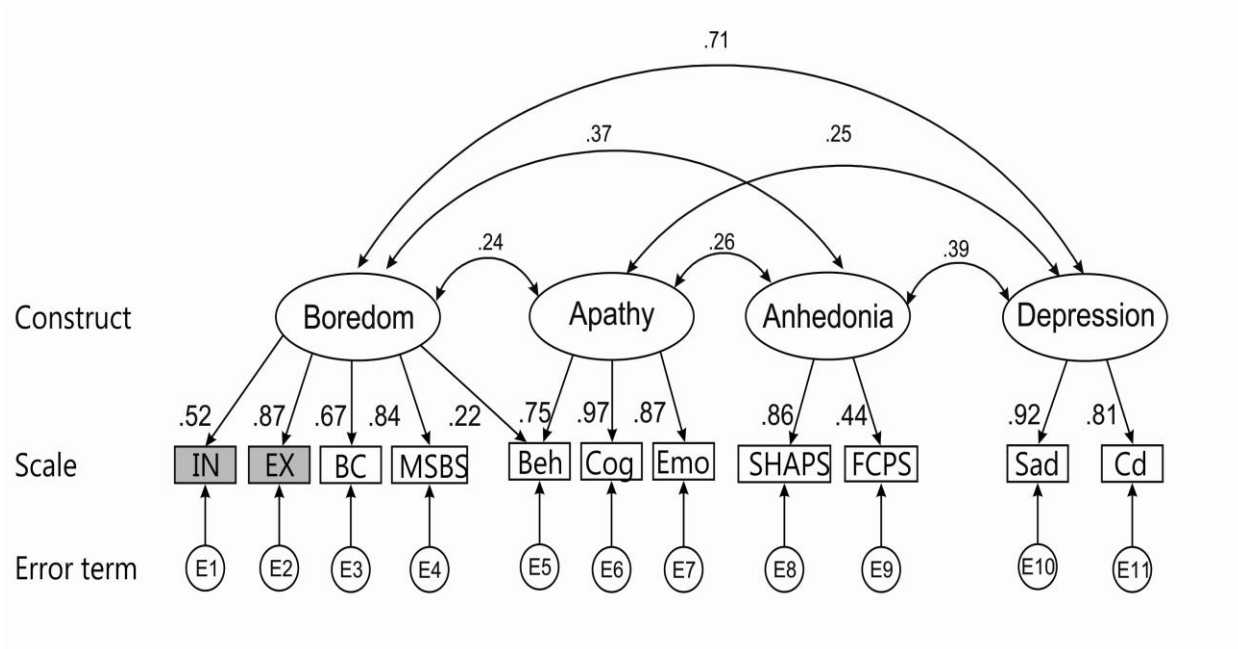


Figure 2.4. A 4-factor model with 2 subscales of the BPS.

IN=internal subscale of the BPS; EX=external subscale of the BPS. All other abbreviations are as for Figure 2.1.

With the emerging differences between the internal and external subscales in the data, it seemed worthwhile to divide the sample based on participants' endorsements of these two factors. The aim was to create two groups of individuals who were characteristically different in their phenomenological experience of boredom, to determine whether these two subtypes of boredom proneness differed in their relationships to depression and apathy. Accounting for reverse scored items (Appendix A), those who scored high on the internal subscale and low on the external subscale ($N = 124$) were characterized as experiencing what could be termed *apathetic boredom proneness*. That is, they had difficulty maintaining focus and keeping themselves interested and entertained through internal stimulation. Those who scored high on the external subscale and low on the internal subscale ($N = 135$) were characterized as experiencing *agitated boredom proneness*, in that they were interested in engaging in the environment but their continued efforts to achieve external stimulation were fruitless, leaving them aggressively dissatisfied. The labels used to describe these two boredom proneness subtypes are necessarily tentative at this stage, and will undoubtedly require refinement through further research. Nevertheless, the key defining feature of these subtypes is the difference in the locus of stimulation being sought (i.e., internal versus external), as well as the degree of motivation for the search (i.e., high versus low).

Correlations between boredom and depression within each subtype of boredom proneness (i.e., agitated and apathetic) yielded the following results. First, the BPS was significantly correlated with the BDI-II in the agitated group ($r = .203, p < .05$), but not in the apathetic group ($r = .136, p = .133$), suggesting that only those defined as experiencing agitated boredom also experienced significant symptoms of depression. This relationship between agitated boredom and depression was even more evident when examining the correlations between the subscales of the BPS and BDI-II (Figure 2.5). That is, in both the agitated and apathetic boredom prone individuals, there was no relationship between the internal subscale of the BPS and either subscale of the BDI-II, however, there was a significant relationship between the external subscale of the BPS and the Cognitive subscale of the BDI-II (agitated: $r = .22, p < .01$; apathetic: $r = .20, p < .01$).

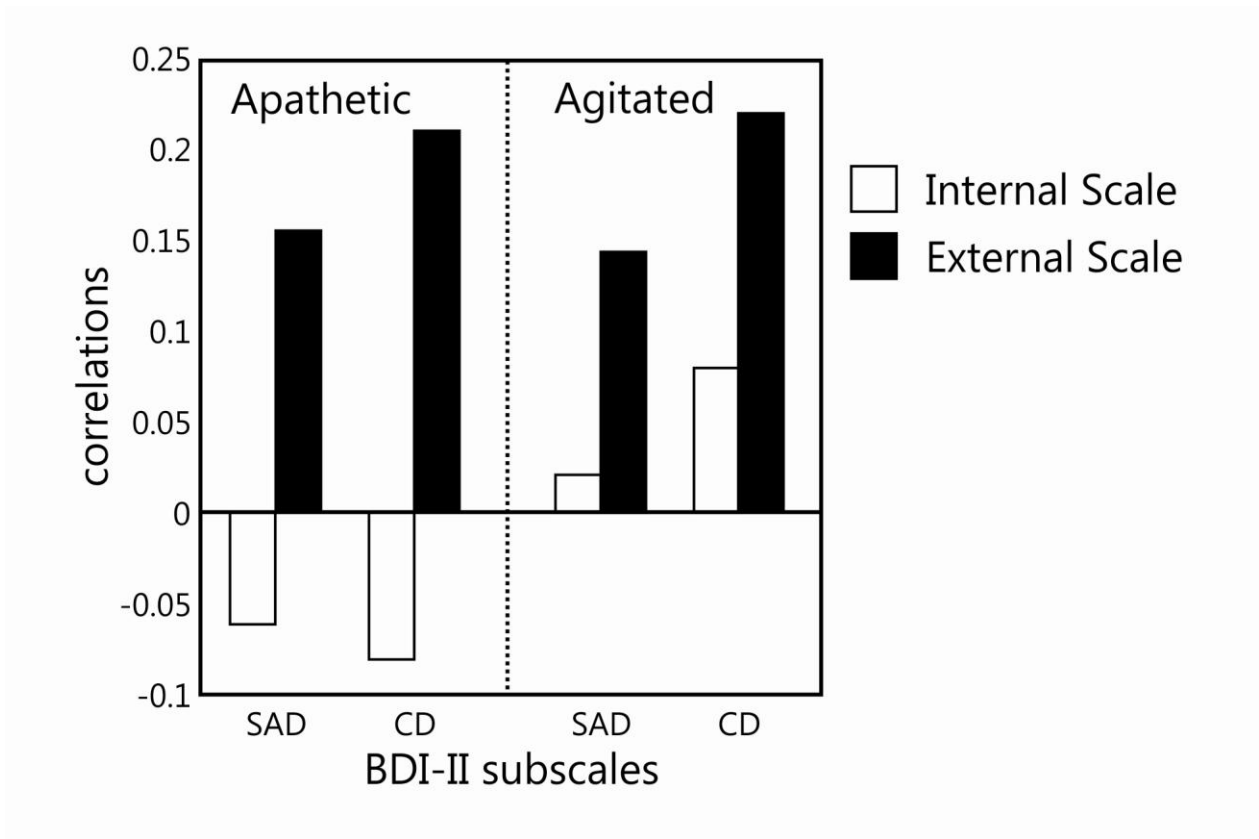


Figure 2.5. Correlations between the two subscales of the BPS and the two subscales of the BDI-II across boredom proneness subtypes.

Left panel=apathetic boredom prone; right panel=agitated boredom prone. CD=Cognitive subscale of the BDI-II; SAD=somatic, affective subscale of the BDI-II.

Interestingly, although non-significant, the internal subscale of the BPS was negatively correlated with measures of depression in the apathetic group, but not in the agitated group. This may suggest that the search for internal stimulation, however unsuccessful, helps to stave off depression in apathetic boredom prone individuals, but not in those prone to experiencing agitated boredom.

Estimated correlations between boredom and apathy within each subtype of boredom proneness revealed a significant positive correlation between the AES and BPS in the apathetic group ($r = .186, p < .05$), yet no correlation was found in the agitated group ($r = -.057, p = .508$). The difference between these correlations trended towards significance ($z = 1.95, p = .051$; DeCoster, 2007). Moreover, in the apathetic group, both subscales of the BPS were positively correlated with subscales of the AES, with the external subscale having a greater degree of correlation than the internal subscale (Figure 2.6). In the agitated group, the BPS subscales were not significantly correlated with the AES, however, there was a positive trend for the Behavior subscale, and a negative trend for the other subscales.

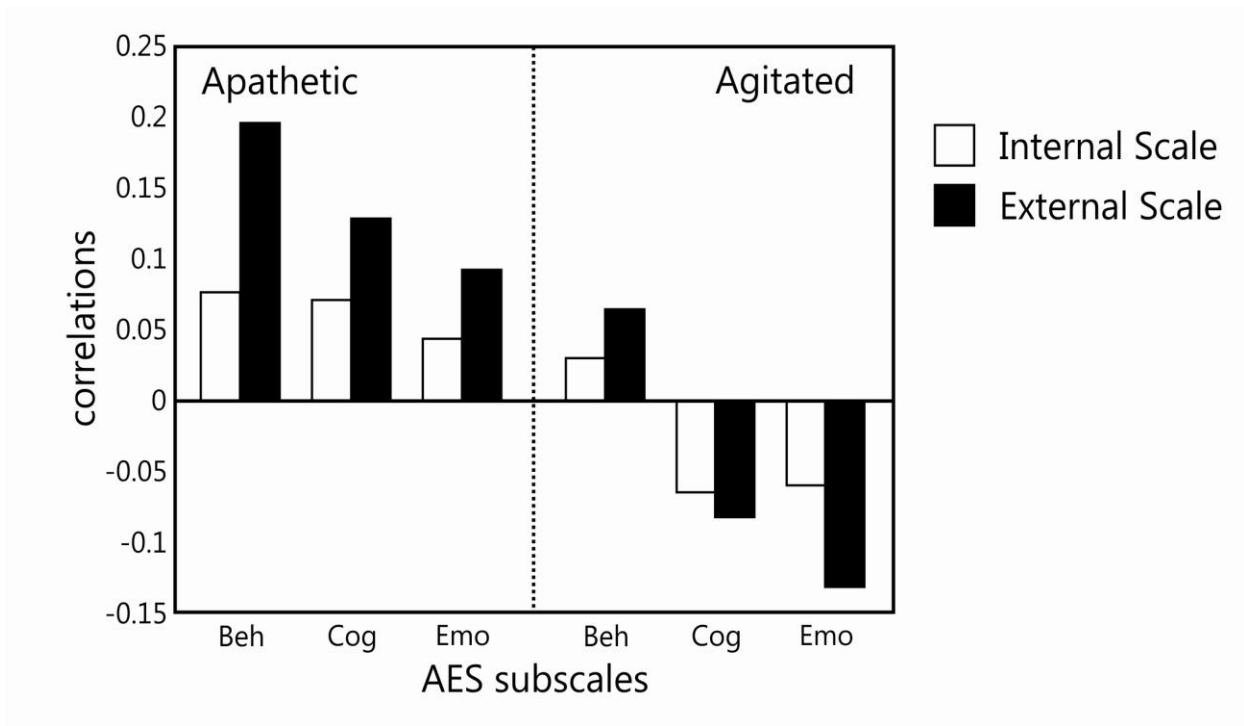


Figure 2.6. Correlations between boredom and apathy within the 2 boredom proneness subtypes.

Beh= Behaviour; Cog=Cognition; Emo=Emotion.

These results suggest that the relationship between boredom and apathy was greatest in individuals with apathetic boredom proneness as one might expect. Despite this, it was the responses on the *external stimulation* subscale of the BPS that continued to drive the relationship between boredom and apathy even within the apathetic boredom prone group. Moreover, the strongest relationship between apathy and boredom was evident in the Behavior subscale of the AES (Figure 2.6; see also Figure 2.2).

2.4 Discussion

The goal of study one was to distinguish the construct of boredom from phenomenologically related states of apathy, anhedonia and depression. Although boredom is correlated to varying degrees with each construct, results clearly showed that boredom represents a distinct affective state.

Although shown here to be statistically independent, boredom and depression were highly correlated, consistent with previous findings (Farmer & Sundberg, 1986; Vodanovich, 2003). Although they appear to share some symptoms (Farmer & Sundberg, 1986), it is unclear why boredom and depression are related to such a high degree. Despite a high degree of overlap in the *content* of individual items in the scales used to measure boredom (BPS) and depression (BDI-II), the current results show that the correlation between the two is *not* solely based on those items. In addition, both scales seem to be measuring similar *elements* of experience. That is, a recent investigation of the factor structure of the BDI-II (Cohen, 2008), suggests that one of the two dimensions of depression measured is symptom expression in terms of *arousal level* (e.g., “loss of energy/pleasure” indicative of low arousal, and “agitation/irritability” indicative of high arousal). Although the factor structure of the BPS remains unspecified, most researchers agree that two of the domains of experience measured by the BPS are a perceived lack of external or internal *stimulation* (Ahmed, 1990; Farmer & Sundberg, 1986; Vodanovich & Kass, 1990; Vodanovich et al., 2005; Melton & Schulenberg, 2009). The current study demonstrated that when boredom was measured by separate external and internal stimulation subscales, it was the perceived lack of *external stimulation* that was most related to depression. This was also true when the sample was split into two separate groups characterized by different phenomenological experiences of

boredom (i.e., apathetic and agitated boredom proneness). That is, an agitated bored state was more related to depression than an apathetic one. Thus, although portions of the BDI-II and the BPS appear to measure similar elements of experience (i.e., arousal and stimulation), this study established that depression and boredom are empirically distinct constructs.

Boredom and depression have also been shown to be distinct in a study of anti-depressant (citalopram) treatment for depression and boredom in cancer patients, where the incidence of depression is high (Theobald et al., 2003). Results showed that over the course of the 8-week study, symptoms of depression and boredom improved at different rates in response to citalopram. That is, above baseline improvements in depression were notable by week two of the study, whereas improvements in boredom were not evident until week six. Moreover, results demonstrated two distinct forms of boredom (labeled by those authors as overt and spirituality), which differed in their relationships to depression. More specifically, overt boredom was correlated with depression, whereas boredom related to meaning and spirituality was not. These findings are consistent with results presented here.

In contrast, a recent study addressing this issue investigated the relationship between boredom and 'life meaning' (Fahlman, Mercer, Gaskovski, Eastwood & Eastwood, 2009; see also Melton & Schulenberg, 2007), defined as an existential awareness of purpose translating into the development of meaningful life goals. Researchers found the relationship between boredom and depression was mediated by 'life meaning'. That is, when the variance associated with life meaning was removed from the analysis, the relation between boredom and depression was minimized. In addition, life meaning predicted levels of boredom weeks later, whereas depression did not. Furthermore, changing perceptions of life meaning caused changes in levels of boredom. Taken together, these findings suggest that the relationship between boredom and depression may be dependent on at least one other factor (i.e., life meaning; Fahlman et al., 2009).

Another recent study investigating the nature of the relationship between boredom and depression found that everyday cognitive failures, such as lapses in attention and memory, play a role in causing both depression and boredom (Carriere, Cheyne, & Smilek, 2008). Although the authors could not determine

whether boredom causes depression or vice versa, they did find that the same attention and memory failures shown to lead to depression may also lead to boredom, as they impact people's ability to sustain interest and maintain engagement with their surroundings. Thus, the relation between boredom and depression seems to be driven in part by common cognitive mechanisms underlying both.

The relationships between measures of boredom and apathy and boredom and anhedonia in the current study were surprisingly lower than expected given their high degree of conceptual overlap. It is unclear why this is the case, especially in light of the fact that boredom and depression were so highly related. Another noteworthy finding relates specifically to the relationship between boredom and apathy. The AES evaluates the presence of apathy across three domains of goal directed behaviour; overt activity (the behaviour subscale), thought content (the cognition subscale), and emotional responsivity (the emotion subscale). When the experimental sample was split into two subtypes of boredom proneness (i.e., apathetic and agitated), results showed, as one would intuitively expect, that the relationships between apathy and boredom were greatest in individuals with apathetic boredom proneness. However, within both the apathetic and agitated groups, it was the perceived lack of external stimulation that was most related to the experience of apathy. On the surface this finding seems counterintuitive, as one would suspect that a lack of internal stimulation would be most related to apathy. Indeed, across a broad range of disciplines, boredom has been thought of as either an *apathetic* state, where one has no intention or purpose (e.g., Heidegger, 1995; Mills, 1959), or an *agitated* state in which a desire to engage in the world yields little success (e.g., Fenichel, 1951). Moreover, boredom and apathy were most strongly related through the Behavior subscale of the AES, which is a measure of goal directed overt activity. Thus, it may be possible that the relationship between boredom and apathy is mediated by a lack of external stimulation.

In conclusion, the present study statistically established that although related to varying degrees to apathy, anhedonia, and depression, boredom represents an independent affective construct. This finding provides clear insight into the nature of boredom by identifying the distinct subtypes of apathetic and agitated boredom proneness. Interestingly, it was the agitated state that was most responsible for the relationships between boredom and the other latent variables. Establishing boredom as a distinct construct

makes an investigation of its cognitive correlates possible as a next step, which has important implications for the identification and treatment of boredom in psychiatric (e.g., depression) and neurological (e.g., traumatic brain injury) populations in which it is most pervasive and disruptive (Binemma, 2004; Cicerone et al, 2006; Hamilton et al. 1984; Seel & Kreutzer, 2003; Vodanovich, 2003; Vodanovich et al., 1991).

Chapter 3

Exploring the relationship between Boredom and Depression in TBI

3.1 Introduction

The second goal of this thesis was to investigate the cognitive correlates of the subjective experience of boredom. Patients who had suffered varying degrees of closed head injuries were examined as this population consistently reports atypically high levels of boredom. In particular, traumatic brain injury (TBI) often leads to pervasive emotional and cognitive changes in the individual, including increases in boredom and depression, and decreases in motivation (Al-Adawi et al., 1998; Chervinsky et al., 1998; Glenn et al., 2002; Mathias, Beall & Bigler, 2004; Seel & Kreutzer, 2003; van Baalen et al., 2006). Frontal cortices represent the most commonly injured brain region in TBI which leads to a range of cognitive difficulties including, but not limited to, poor sustained attention and concentration and a bias toward novelty over familiarity (Catalano et al., 2004; Cicerone, Levin, Malec, Stuss & Whyte, 2006; Dockree et al., 2004; Dockree et al., 2006; Gianotti et al., 2009; Manly et al., 2003; Mathias et al., 2004; O’Keeffe et al., 2007; Robertson et al., 1997; Whyte et al., 2006). The current investigation focused on the subjective experience of boredom in patients with different degrees of TBI, to examine relationships between boredom and depression following TBI.

3.2 Participant Characteristics

There were three groups of participants in this study and for the chapters that follow on the relationship between sustained attention and boredom (Chapter 4) and novelty preference and boredom (Chapter 5). Males who had sustained acceleration/deceleration closed head injuries were recruited from the Kitchener-Waterloo, Hamilton, and Niagara communities, St. Michael’s Hospital in Toronto, and the University of Waterloo’s volunteer subject pool. They were split into two patient groups categorized by injury severity, determined using American Congress of Rehabilitation Medicine (1993) criteria (i.e., Glasgow Coma Scale scores – where available, duration of loss of consciousness, and post traumatic amnesia). One patient group (Concussion) was comprised of 38 males (M age = 24; SD = 9) who had

sustained concussions or other forms of mild TBI. The other patient group (TBI) was comprised of 14 males (M age = 36; SD = 12.1) who had sustained moderate to severe TBI. Eighty-eight males (M age = 23; SD = 7.4) were recruited through the University of Waterloo's undergraduate volunteer subject pool and the local community to form the control group (Controls). Participants either received course credit or remuneration for their participation, and signed a consent form before completing the experiment. All procedures were approved by the Office of Research Ethics at the University of Waterloo. Characteristics of each experimental group are summarized in Table 3.1.

Group (N)	Age M (SD)	Age Range	Years Post Injury	ACRM criteria	BPS Score M (SD)	Internal Subscale score M (SD)	External Subscale score M (SD)	BDI-II Score M (SD)
Controls (88)	23 (7.4)	17-57		NA	93.42 (28.37)	26.58 (15.30)	64.51 (19.68)	11.53 (11.62)
Concussion (38)	24 (9.0)	18-54		GCS 13-15 w/in 30 mins, LOC < 30 mins	101.21 (23.72)	30.37 (4.74)	67.47 (19.57)	10.58 (9.87)
TBI (14)	36 (12.1)	18-56		GCS =<12, LOC > 30 mins, PTA	106.21 (21.93)	34.57 (8.72)	68.43 (17.32)	13.36 (9.96)
Pt 1	42		20	Coma for several weeks	100	35	63	0
Pt 2	36		30	Coma for several months	75	28	50	4
Pt 3	40		20	Coma for 6 weeks	140	48	89	30
Pt 4	56		30	LOC 40 mins	100	16	71	8
Pt 5	25		6	LOC, coma for 6 weeks	119	28	87	18
Pt 6	34		14	Level 3 coma for 6 weeks	123	39	78	16
Pt 7	18		2	LOC, PTA 2 weeks	116	36	77	11
Pt 8	54		10	Coma for 2 weeks	80	27	52	10
Pt 9	23		12	LOC > 2 hours	143	35	102	32
Pt 10	39		<1	LOC, PTA for 8 hours	113	38	74	22
Pt 11	39		2	'Brief' LOC	72	28	42	8
Pt 12	19		10	LOC – unknown length	104	37	63	20
Pt 13	30		11	Come for 2 weeks, PTA	91	40	49	3
Pt 14	49		2	Collapsed with seizure	111	49	61	5

Table 3.1. Participant Characteristics of studies 2, 3 & 4

Note. M=mean; SD=standard deviation; GCS=Glasgow Coma Scale; LOC=loss of consciousness; PTA=post traumatic amnesia.

3.3 Boredom

Susceptibility to experiencing boredom was measured with the Boredom Proneness Scale (BPS; Farmer & Sundberg, 1986; see Section 2.2.2 for description). Analyses of the BPS scores across the three groups were conducted using a one-way analysis of covariance (ANCOVA), with participant's age as a covariate to control for any differences resulting from age.² Although the mean BPS score was highest for the TBI group, next highest for the Concussion group and lowest for the Controls (Table 3.1), results showed that these differences were not significant [$F(2, 136)=1.31, p=.274$]. The three groups also showed no difference in their scores on the internal [$F(2, 134)=1.66, p=.195$], or external subscales of the BPS [$F(2, 134)=.366, p = .694$].

This finding was surprising given the documented subjective reports of increased levels of boredom in individuals with TBI (Al-Adawi et al., 1998; Chervinsky et al., 1998; Glenn et al., 2002; Seel & Kreutzer, 2003; van Baalen et al., 2006). One possible explanation for this discrepancy is that the BPS as a general measure of boredom proneness fails in its ability to measure the experience of boredom in individuals with TBI (i.e., the test lacks appropriate sensitivity). It may also be the case that patients with TBI do in fact show elevated levels of boredom proneness *relative* to premorbid levels. In addition, many of the moderate to severe TBI patients had suffered their injury many years prior to testing (Table 3.1). It may well be the case that boredom levels subside with age in general and with time post injury in particular, both possibilities that would require further research. Also, the sample size tested here is quite small which may have impacted the results. Finally, the reliability of self-report measures is often questioned because they may very well contain responder biases, and reflect poor responder insight. Indeed, TBI patients are known to have poor insight and are unreliable historians when it comes to autobiographical information (Mathias, Beall & Bigler, 2004; McAvinue et al, 2005). Further research is

² Results indicated that the relationship between age and BPS scores did not differ significantly as a function of participant group. That is, there was no significant interaction between age and participant group on the total BPS score [$F(2, 134) = 1.52, p = .222$], the internal subscale score [$F(2, 132) = 2.26, p = .109$], or the external subscale score [$F(2, 132) = 2.47, p = .089$]. Thus, the interaction term was removed from subsequent models analyzing BPS scores.

needed to investigate the inconsistency between subjective reports and empirical evidence regarding boredom levels in traumatic brain injury.

In light of the findings in Study 1 (Chapter 2), the current participant groups were further examined based on the distinct kinds of boredom proneness identified - apathetic or agitated. Those who scored high on the internal subscale of the BPS and low on the external subscale of the BPS were characterised as experiencing *apathetic boredom* (see Section 2.3). Those who scored high on the external subscale of the BPS and low on the internal subscale of the BPS were characterised as experiencing *agitated boredom*. When the sample was characterised according to these two subtypes of boredom proneness, the following profile emerged. Twenty-nine percent of the TBI group were characterised as experiencing apathetic boredom, 14% experienced agitated boredom, and 57% did not fall into a specific subtype. In the Concussion group, 18% were characterised as experiencing apathetic boredom, 5% experienced agitated boredom, and 76% did not fall into either subtype. Finally, in the Control group, 16% were characterised as experiencing apathetic boredom, 16% experienced agitated boredom, and 68% did not fall into a subtype. In other words, the TBI group was twice as likely to present with apathetic boredom proneness than either the Concussion or Control groups. Note, with such small numbers of agitated boredom prone individuals in the concussion and TBI groups, results with these data should be interpreted with caution.

3.4 Depression

Depression severity was indicated by the BDI-II (Beck, Steer, & Brown, 1996; see Section 2.2.2 for description). ANCOVA was performed on the BDI-II scores (Table 3.1) to determine whether there were differences between the participant groups, controlling for age. Results yielded a significant interaction effect between participant group and age [$F(2, 134) = 4.05, p < .05$]. Linear regression lines were fit to the data, yielding significant positive slopes relating age and BDI-II scores in both the control and concussion groups, and a non-significant negative slope in the TBI group. Multiple regression analysis indicated that for the control and concussion groups, greater age was significantly associated with higher levels of

depressive symptoms, whereas for the TBI group, there was no such association. One possibility for the lack of any association between depression and age in the TBI group may relate to the fact that individuals with TBI often have poor insight into their own behaviours and as a consequence may experience lower levels of depressive symptoms (Seel & Kreutzer, 2003). Alternatively, the mean number of years post injury for this group is 12.14 years. It may be the case that any symptoms of depression experienced in the acute phase post injury have subsided by the time of testing in this study.

3.5 Relationship between boredom and depression

Bi-variate correlations were performed between measures of boredom (BPS) and depression (BDI-II) to determine whether there was any relation between the two across the participant groups. Based on previous results (Section 2.3), it was expected that boredom and depression would be highly correlated in all groups. However, it was hypothesized that the relationship between boredom and depression would be the strongest in the TBI group, and greatest among those who were in a state of agitated boredom. Results revealed significant correlations between BPS and BDI-II scores in all three groups (Control: $r = .409, p < .01$; Concussion: $r = .616, p < .01$; TBI: $r = .764, p < .01$). Although the differences between these correlations were non-significant, there was a trend towards a stronger relationship between boredom and depression in the concussion ($z=1.41, p=0.15$) and TBI groups ($z=1.78, p=.07$) than in controls. This is consistent with previous research establishing high levels of boredom and depression in these patient groups (Seel & Kreutzer, 2003).

When correlations were calculated between the BDI-II and the individual subscales of the BPS, no significant relationships were found between the internal subscale and the BDI-II scores in any of the groups (Control: $r = .134, p = .218$; Concussion: $r = .268, p = .104$; TBI: $r = .260, p = .369$). In contrast, the external subscale was significantly correlated in all three groups (Control: $r = .393, p < .001$; Concussion: $r = .662, p < .001$; TBI: $r = .786, p < .001$). Tests of differences between these correlations revealed no significant difference between the concussion and TBI groups, however, there was a significant difference between the TBI and controls ($z = -2.01, p < 0.05$), and the difference between the

concussion and controls approached significance ($z=1.89$, $p=0.057$). Thus, the relationship between the BDI-II and the external subscale scores of the BPS is greater in the TBI and concussion groups than controls (Figure 3.1).

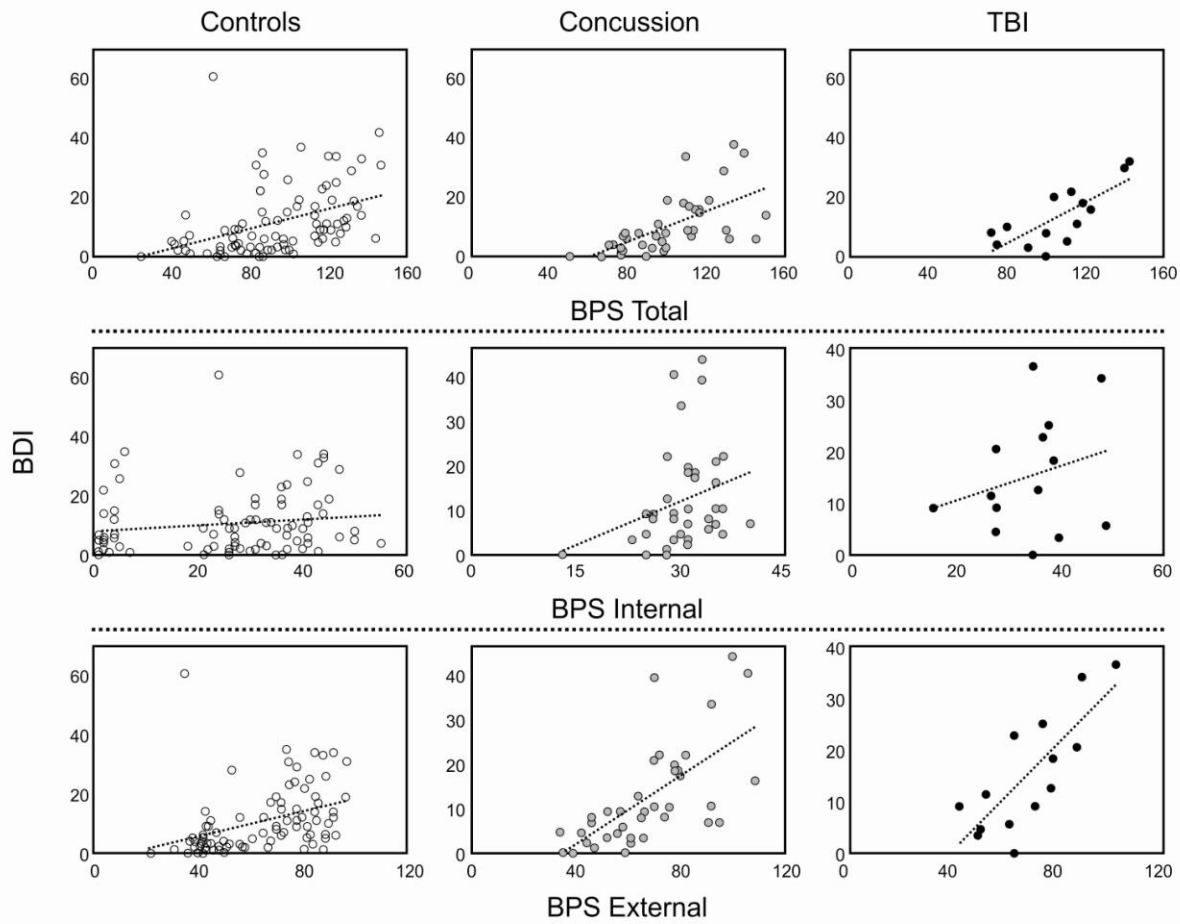


Figure 3.1. Relationship between BDI-II and BPS scores across participant groups.

These findings are consistent with the results of Study 1 (Section 2.3), which found the external subscale of the BPS to be the driving force behind the relationship between the BPS and BDI-II, and suggest that this relationship is magnified following traumatic brain injury.

When the study sample was characterized based on whether individuals were experiencing *apathetic* or *agitated* boredom proneness, some of the resultant categories had very few participants. Thus, the following analyses are meant only to be descriptive and to highlight possible trends, and should be interpreted with caution until larger sample sizes can be obtained. Analysis revealed a greater degree of depression in those with agitated boredom proneness than apathetic boredom proneness (Figure 3.2). This difference was significant in the control group ($t=-3.05$, $p<.01$), indicating that agitated controls had higher scores on the BDI-II than apathetic controls. This is consistent with the hypothesis, and earlier findings in Study 1 (Section 2.3).

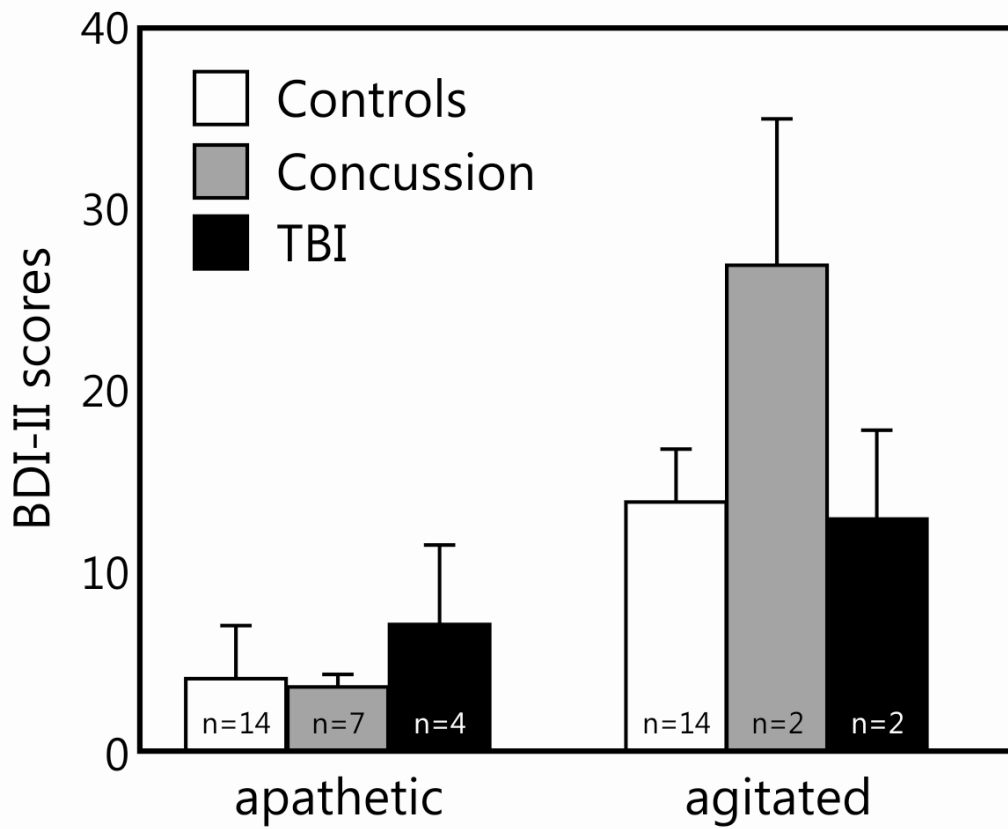


Figure 3.2. BDI-II scores across participant group, characterized by the experience of apathetic vs. agitated boredom proneness.

Note: Error bars represent the standard error of the mean.

3.6 Discussion

The goal of Study 2 was to investigate the relationship between depression and boredom in patients with varying degrees of TBI, where boredom has been reported to be pervasive (Al-Adawi et al., 1998; Chervinsky et al., 1998; Glenn et al., 2002; Seel & Kreutzer, 2003; van Baalen et al., 2006). Results showed that individuals with TBI were more likely to experience apathetic boredom proneness (as opposed to agitated boredom proneness) than the other two groups of participants. Apathetic boredom proneness describes a lack of desire to seek external stimulation, and a difficulty maintaining interest in seeking internal stimulation. Although the literature on apathy following TBI is scarce, researchers have found a high incidence of apathy post injury, which increases with age and time since injury, and has a negative impact on motivation for rehabilitation (Al-Adawi, Powell, & Greenwood, 1998; Chervinsky et al., 1998; Glenn, 2002; Kant, Duffy, & Pivovarnik, 1998). Given that the majority of the TBI participants in the current study were older and further from the time of injury, it is perhaps not surprising that the incidence of apathy was high. It may be the case that the prevalence of boredom proneness subtypes differs over the course of recovery from TBI. Immediately post injury patients may show a tendency towards agitated boredom as they attempt to piece together their lives. This propensity may be expected to lead to the increased novelty seeking and risk taking behaviour characteristic of TBI patients, at least in the acute phases post injury (Gianotti et al., 2009). Any tendencies of this kind may then be expected to diminish over time in concert with other changes including age. In other words, it is possible that agitated boredom in the early phases post TBI gives way to apathetic boredom in more chronic phases. Certainly the group tested here were a long time post injury. Since different mechanisms may be at play depending on age and time since injury (Al-Adawi, Powell, & Greenwood, 1998; Chervinsky et al., 1998; Glenn, 2002; Kant, Duffy, & Pivovarnik, 1998), this speculative time course of post-injury boredom would be worthy of further investigation to determine appropriate interventions that are tailored to each individual's needs, in order to optimize recovery and motivation for recovery.

Results also demonstrated a trend towards a stronger relationship between boredom and depression in the concussion and TBI groups than in the controls. This is not surprising given the high

incidence of post-injury depression in patient groups (Glenn, 2002; Kant, Duffy & Pivovarnki, 1998; Seel & Kreutzer, 2003). Furthermore, the relationship between boredom and depression was found to be largely driven by the external subscale of the BPS in both the TBI and concussion groups, but not the controls. However, when the sample was characterized by boredom proneness subtype, all participants (control, concussion, and TBI) in the agitated boredom prone group were more likely have higher levels of depressive symptoms than those in the apathetic boredom prone group. These findings are consistent with Study 1, and have important implications for the rehabilitation and recovery of TBI patients. It is well known that the incidence of depression and boredom are both high following TBI (Al-Adawi et al., 1998; Seel & Kreutzer, 2003), and that symptoms of boredom and depression have responded differently to treatment in a patient population (Theobald et al., 2003; Section 2.4). The findings presented here suggest that the co-morbid expression of boredom and depression is greatest in individuals with agitated boredom proneness, suggesting the need for individualized treatment approaches based on boredom proneness subtype. Based on the present findings, intervention strategies designed to specifically target features of agitated boredom proneness will likely have the most successful outcomes in treating post-injury depression in TBI patients. Further research with more participants is needed to explicate the relationship between agitated boredom and depression in patient populations.

Chapter 4

Exploring the relationship between Boredom and Attention in TBI

4.1 Introduction

Deficits in attention represent perhaps the most common cognitive consequence of TBI (Catalano et al., 2006; Cicerone, Levin, Malec, Stuss & Whyte, 2006; Dockree et al., 2004; Dockree et al., 2006; Manly et al., 2003; Mathias et al., 2004; McMillan et al., 2002; O’Keeffe et al., 2007; Robertson et al., 1997; Whyte et al., 2006). While the field of brain injury research currently lacks an adequate characterization of these attention deficits, one common tool used to measure sustained attention is the Sustained Attention to Response Task (SART; Robertson et al., 1997). In this task, participants are randomly presented with numbers from ‘1’ to ‘9’, and are required to respond with a button press to every number except one (e.g., the number ‘3’). In an improvement over traditional vigilance tasks which require monitoring a long stream of information for the occurrence of a particular target to respond to, the SART task requires monitoring for a period of less than 5 minutes for the appearance of a target to *withhold* a response from. This forces an individual to more actively attend to the task in order to overcome the tendency to automatically respond. Thus, errors on this task are thought to be more reflective of an impaired ability to sustain attention (for alternate versions of the SART see Dockree et al., 2006; Manly et al., 2003; Robertson & O’Connell, 2010).

Previous research has shown that injury severity is directly related to performance on tasks of sustained attention such as the SART, with severely injured patients demonstrating the worst performances (Robertson et al., 1997; Manly et al., 2003; Dockree et al., 2006). In addition, results typically show that TBI patients make significantly more errors of commission (i.e., erroneously responding to a ‘3’), and while there is no difference in overall response times (RT) between the two groups, TBI patients show more variability in RT (Robertson et al., 1997; Stuss et al., 1999; Manly, 2000; Manly et al., 2003; Dockree et al., 2006). Furthermore, in an alternate version of the SART in which the numbers are presented in a fixed ascending sequence (i.e., allowing individuals to *predict* when the no-go

trials will occur) control participants slow down for the trials immediately preceding a no-go trial, whereas TBI patients do not show any such signs of preparatory slowing (Manly et al., 2003; Dockree et al., 2004; Dockree et al., 2006; O’Keeffe et al., 2007). In fact, in various versions of the SART, the RT of TBI patients gets shorter on trials just preceding an error, suggesting that the patients have stopped paying attention to the stimuli and are instead automatically responding to the trials based on their habitual and most frequent response. That is, errors are considered the result of a change in the approach to the task from a controlled, effortful processing of information, to an automatic mode of responding (Robertson et al., 1997; Dockree et al., 2004; Manly et al., 1999; Cheyne et al., 2006; Smallwood et al., 2007; Cheyne et al., 2009).

Many researchers have proposed that this attentional ‘drift’ from controlled to automatic responding in *healthy individuals* is a consequence of cognitive underarousal and boredom (Fisher, 1993; Hamilton, 1981; Hamilton, Haier & Bauchsbaum, 1984; Robertson et al., 1997; Manly et al., 1999; Wallace, Vodanovich, & Restino, 2003; Kass, Wallace & Vodanovich, 2003; Kass, Vodanovich, Stanny & Taylor, 2001; Cheyne, Carriere & Smilek, 2006 & 2009; Carriere, Cheyne & Smilek, 2008; Cheyne et al., 2009; Pattyn et al., 2008). However, this association between attention and boredom has been established using only *questionnaire* measures. That is, many studies of healthy individuals have demonstrated that significant relationships exist between high levels of boredom proneness and what could be considered failures or ‘lapses’ of attention (e.g., putting milk in the pantry instead of the fridge; Wallace, Kass & Stanny, 2002; Wallace, Vodanovich & Restino, 2003; Cheyne, Carriere & Smilek, 2006; Carriere, Cheyne & Smilek, 2008). Thus, while previous research has demonstrated a link between lapses in attention and boredom in healthy individuals using questionnaire measures, the relationship between attention and boredom has not been investigated using behavioural measures of attention, nor has this relationship been studied in TBI patients where boredom is reportedly more pervasive.

In the present study, the relationship between boredom proneness and sustained attention was examined in patients with varying degrees of TBI. If, as previous research suggests, sustained attention deficits are related to injury severity, one would expect to find that the severely injured TBI patients yield

the worst performance on the SART. Additionally, if, as previous research suggests, poor sustained attention is reflective of higher levels of boredom, one would expect to see the greatest attentional deficits to be evident in individuals with high BPS scores. However, if impaired sustained attention is the result of something other than cognitive underarousal and/or boredom, no differences should be found in SART performance based on level of boredom proneness. Finally, if sustained attention was found to be related to boredom, one would expect to see differences in performance based on the subtype of boredom proneness being experienced (Malkovsky, Merrifield, Goldberg & Danckert, 2012). That is, individuals with *apathetic* boredom proneness who lack the desire to engage in the external environment would be expected to demonstrate poorer performance than individuals with *agitated* boredom proneness, who are highly motivated to engage in their surroundings in their search for stimulation.

4.2 Methods and Procedure

The three groups of participants in the present study have been described elsewhere (Chapter 3, Section 3.2). The SART (Robertson, et al., 1997) was used to measure sustained attention. In the current version of the SART, 225 single digits (25 of each of the 9 digits) were randomly presented in white on a black background over a 4.3 minute period. The font size of the digits varied across trial presentation, with equal random presentation of 48, 72, 94, 100 and 120 point size Symbol font. Each digit was presented for 250 msec, followed by a 900 msec mask consisting of a ring with a diagonal cross in the middle (Figure 4.1).

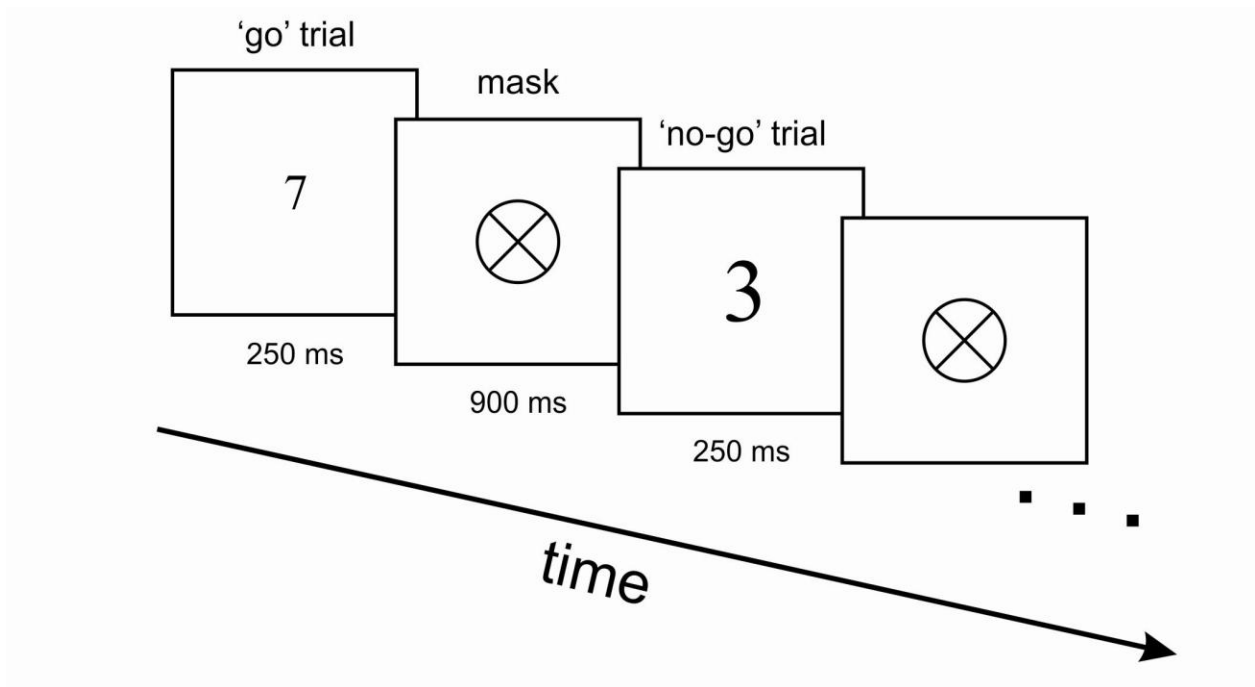


Figure 4.1. Schematic representation of trial sequences in the Sustained Attention to Response Task (SART).

Participants were required to respond as quickly and as accurately as possible to the appearance of each digit by pressing the computer's spacebar, unless the digit 3 was presented. On the 25 occasions that the digit 3 was presented, participants were required to withhold a response. These 25 'no-go' trials were randomly presented throughout the 225 trials. Eighteen practice trials were presented prior to the start of the task. The SART was created in E-prime, and presented to participants on a 15.4 inch computer screen with 1024 X 768 pixels resolution and an NVIDIA 512 MB Quadro NVS 140M video card, Intel Core 2 Duo CPU at 2.60 GHz with a refresh rate of 60 Hz.

All participants completed the SART in the lab. Data collected from the SART was processed prior to analysis as follows: Across all participant groups, individual trials that had response times less than 100 ms were removed, as they represented anticipatory responses made prior to the presentation of the stimulus. For controls, individual trials with response times greater than 2 *SD* from the individual's grand mean were also removed, as these represented uncharacteristically slow responses. This resulted in the elimination of an average of seven trials (3.11%) per individual (*SD*=3.12) from further analysis. An average of seven trials (3.11%) per individual in the concussion group (*SD*=3.23) and eight trials (3.55%) per individual in the TBI group (*SD*=3.52) were greater than 2 *SD* from the individual's grand mean. However, given that increased variability is a hallmark of TBI (Dockree et al., 2006), these trials were not eliminated from further analysis. One control participant had a rate of 100% commission errors (i.e., 25 out of a possible 25), which exceeded our pre-determined accuracy cut-off value of 88% errors (i.e., 22 out of 25). This participant was removed from the data set. One concussion patient was excluded from the analysis due to missing data. Thus, in total, two participants (one control and one concussion patient) were excluded from further analysis. Data was analyzed using ANCOVA, with age as the covariate.

4.3 Results

4.3.1 Reaction Time

Analysis of RT revealed a significant main effect of age [$F(1, 134)=32.62, p<.001$]. Correlations between age and RT were significant in the TBI ($r=.724, p<.01$) and control ($r=.445, p<.001$) groups, and trended

towards significance in the concussion group ($r=.296, p=.075$). These results indicate that across the groups, reaction time increased with age. No interaction was observed between age and participant group. Therefore, all further analyses of RT contrasted group performance without controlling for age.

Planned comparisons of the RT means revealed significant differences between the TBI and control groups ($t=-4.75, p<.001$), and the TBI and concussion groups ($t=3.0, p<.001$). There was no significant difference between the concussion and control groups ($t=-.811, p=.421$). Thus, individuals in the TBI group were slower to respond than those in the concussion and control groups (Figure 4.2).

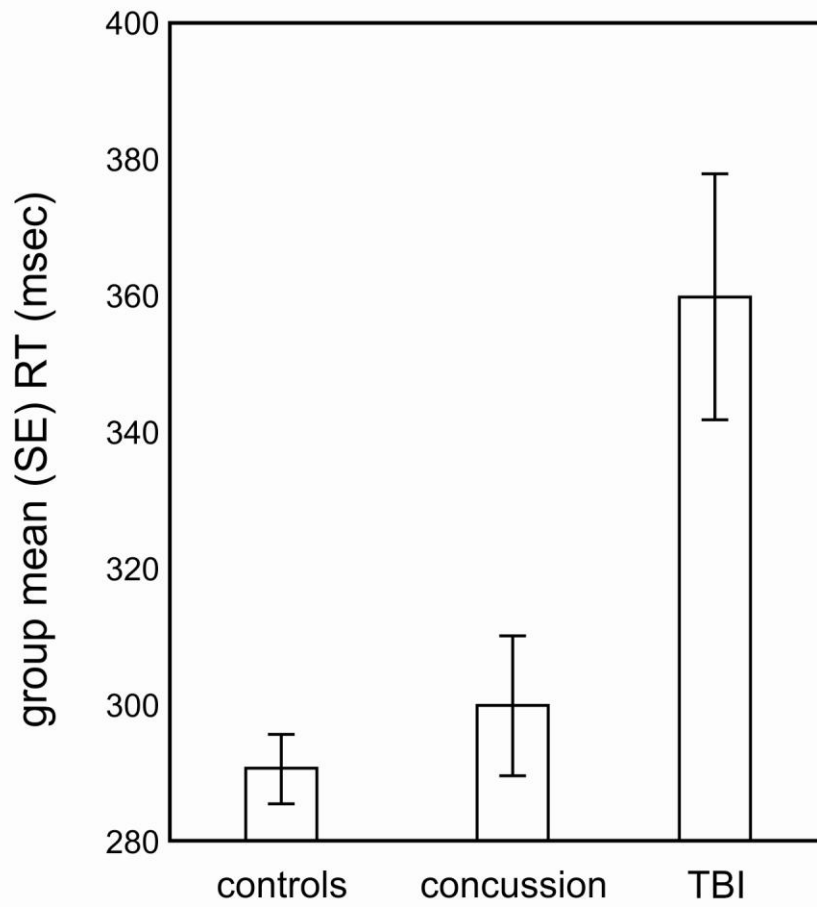


Figure 4.2. Mean SART response time across participant group.

Note: Error bars represent the standard error of the mean.

Next, to determine whether each group slowed down during performance of the SART, RTs for the first half of the task (trials 1-112) were directly contrasted against the last half (trials 113-225) in a mixed design repeated measures ANOVA with group as the between subject variable and first versus last half RTs as the within subjects variable. Results revealed a main effect of RT [$F(1,135)=4.44, p<.05$] and a non-significant trend towards an interaction between RT and group [$F(2, 135)=2.15, p=.120$]. Thus, RTs tended to be slower in the last half of the task, a difference that appeared to be more pronounced in the two patient groups (Figure 4.3).

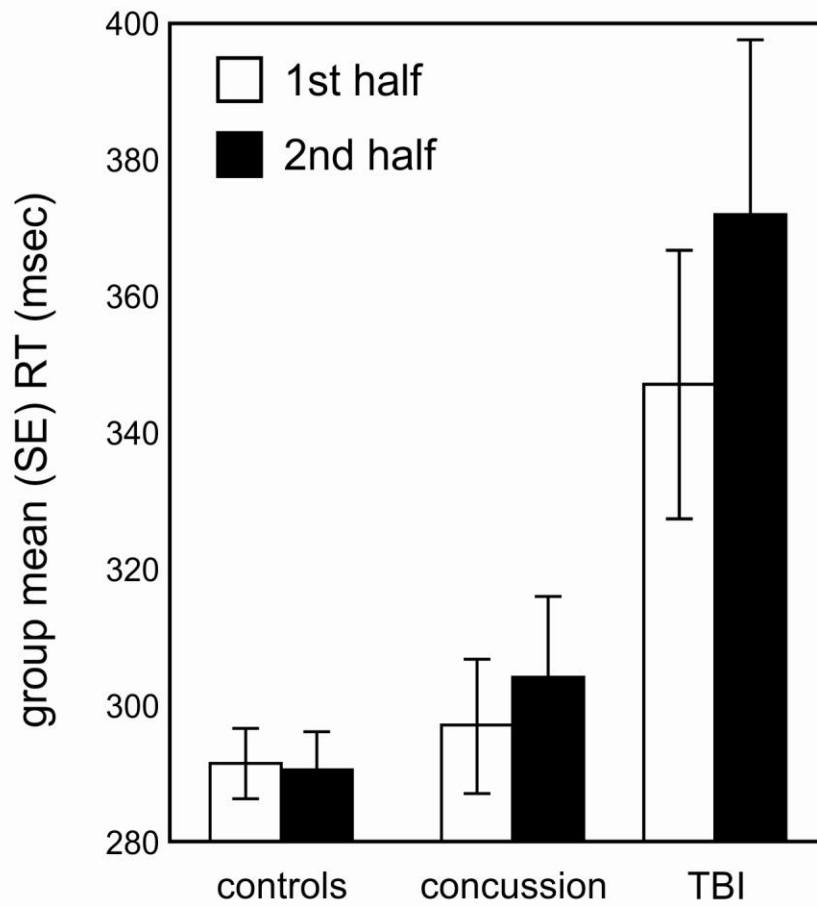


Figure 4.3. Mean reaction time for first half (trials 1-112) and second half (trials 113-225) of the SART task across group.

Note: Error bars represent the standard error of the mean.

To further examine how SART performance evolved over the course of the task, the 225 trials of the SART were sorted into nine blocks of 25 trials each. Linear curve estimates (on the RT for each bin) were calculated to determine whether there were differences in response trends across groups. Analysis revealed no significant linear trend for controls ($R^2=.000$, $p=.603$), or the concussion group ($R^2=.002$, $p=.459$). The curve of the TBI group approached significance ($R^2=.022$, $p=.097$), suggesting a trend towards slowing down over time (Figure 4.4).

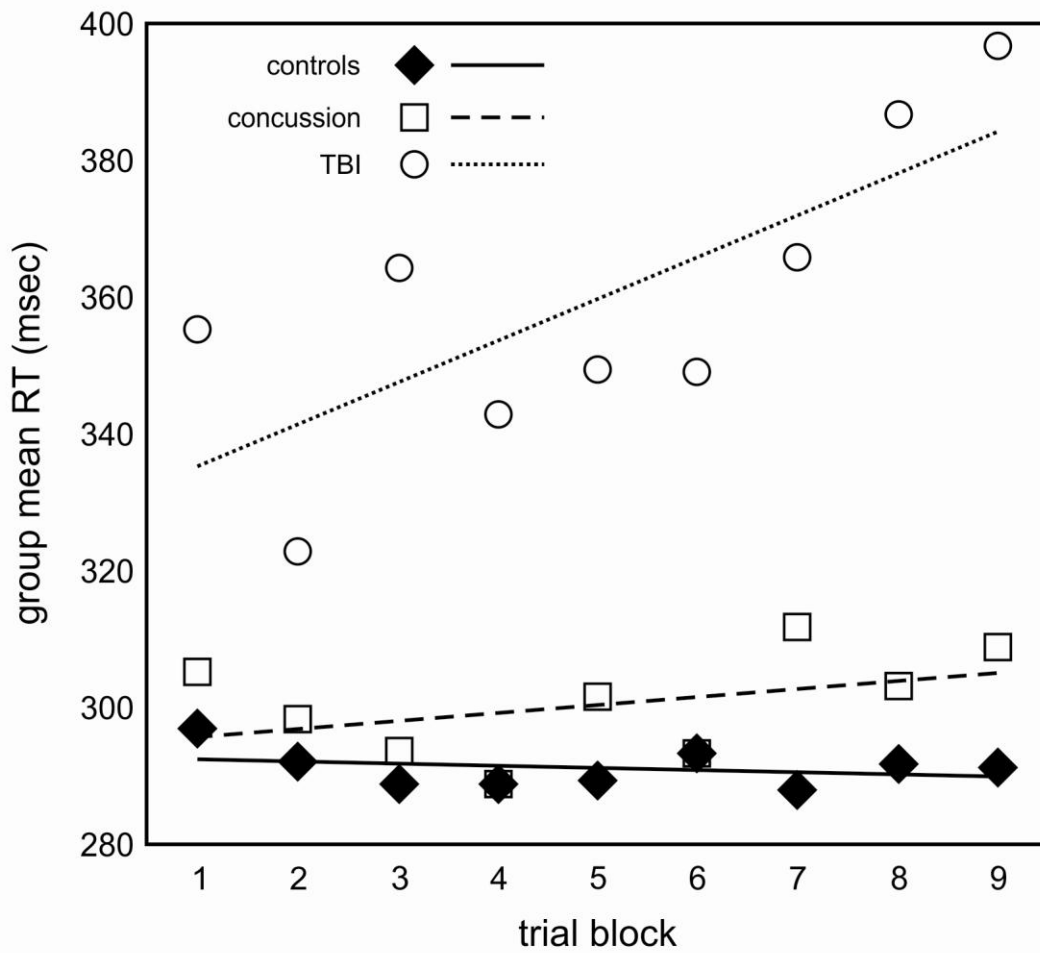


Figure 4.4. Mean reaction time over 225 trials of SART, grouped into 9 blocks of 25 trials each.

To examine variance in performance across the groups, each individual's standard deviation across the whole task was used to generate a group mean variance score as a dependent variable. Analysis of RT variance controlling for age, yielded a significant interaction effect between participant group and age [$F(2, 132) = 6.44, p < .01$]. Linear regression lines were fit to the data, yielding significant positive slopes relating age and RT variance in both the concussion and TBI groups, with no significant relation evident in controls. Multiple regression analysis indicated that both the concussion and TBI groups had significantly larger slopes than controls, while the slopes of the TBI and concussion groups were not significantly different from each other. These results indicated that as they age, the patient groups became increasingly variable in their performance relative to controls.

It was hypothesized that the TBI group would have the most variable performance, followed by the concussion group, with controls showing the least amount of variability. Although the analysis of mean reaction time variance, did not reveal a main effect of participant group [$F(2,132)=1.56, p=.213$], the pattern of response variability was as hypothesized (Figure 4.5). Therefore, planned comparisons were executed on the adjusted means (controlling for age). Results revealed a significant difference between the control and the concussion groups ($t=-3.68, p<.001$). The difference between the TBI group and controls approached significance ($t=1.88, p=.063$). There was no significant difference between the TBI and concussion groups ($t=-.03, p=.975$; Figure 4.5).

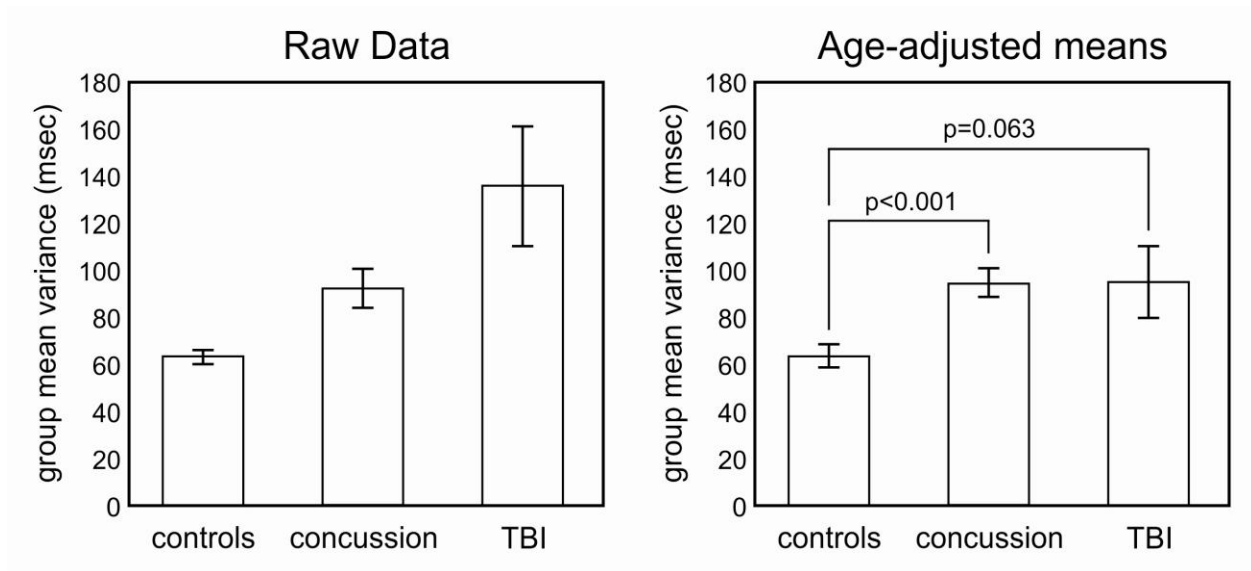


Figure 4.5. Mean SART reaction time variance (left) adjusted for age effects (right) across participant group.

Note: Error bars represent the standard error of the mean.

The mean RT surrounding errors of commission was calculated for each individual (using the three trials immediately preceding and anteceding an erroneous response on a no-go trial) to determine whether there was a relationship between RT and accuracy. It was hypothesized that controls would slow down after making an error, but patients would not (Robertson et al., 1997; Chan, 2002). To test this, pre and post error RTs were analyzed in a mixed design ANOVA with group as the between subjects variable and pre versus post error RT as within subjects variables. No significant interaction effect was found between RT and group [$F(2,135)=.237, p=.790$]. Thus, contrary to predictions based on previous literature, none of the groups showed any post error slowing in RT.

4.3.2 Accuracy

Failing to respond to a ‘go’ trial was considered an error of omission, while erroneously responding to a ‘no-go’ trial was considered an error of commission. Analysis of the rate of omission errors, yielded a significant interaction effect between participant group and age [$F(2, 132) = 5.89, p < .01$]. Linear regression lines were fit to the data, yielding a significant positive slope relating age and accuracy in the concussion group, with no significant relation evident in the TBI or control groups. These results indicated that as the concussion group aged, they made more errors of omission (Figure 4.6).

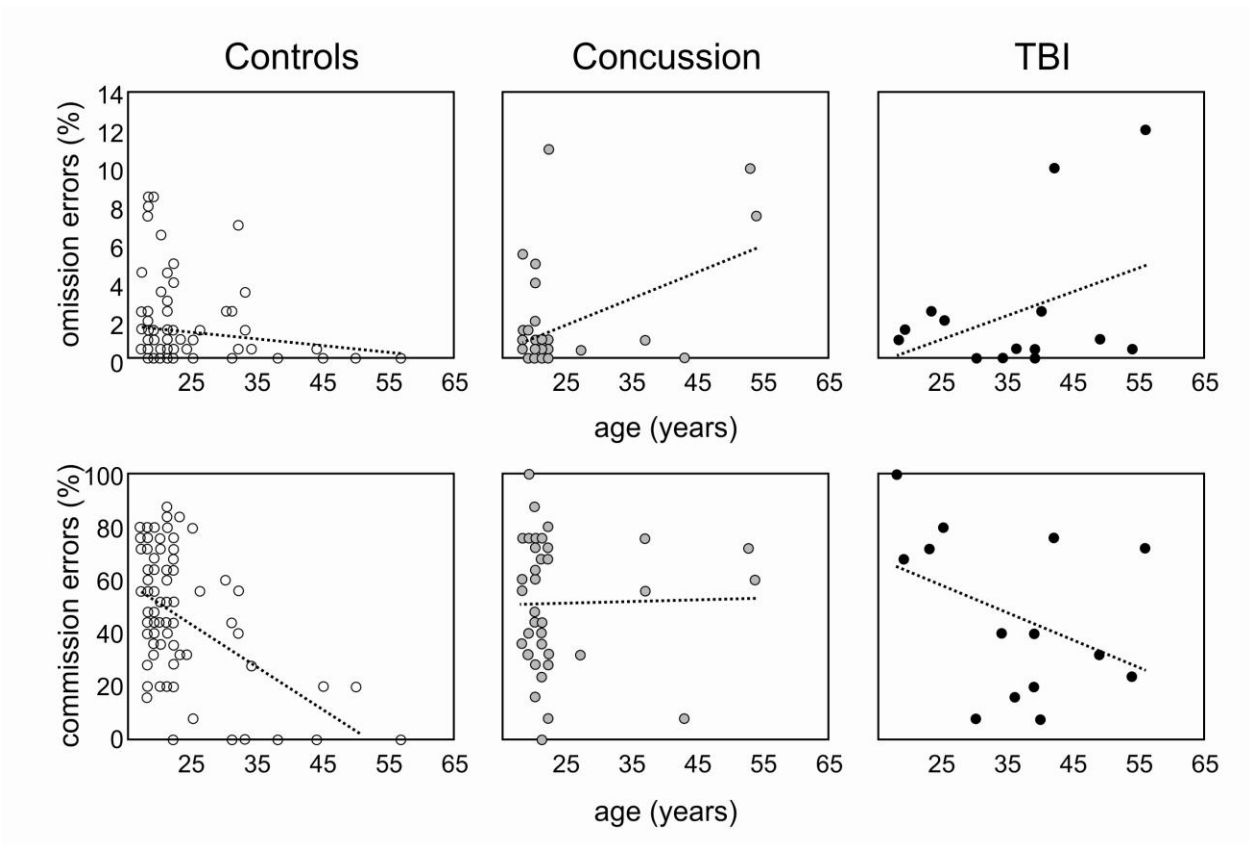


Figure 4.6. Relationship between age and number of SART omission and commission errors across group.

Analysis of the rate of commission errors, also yielded a significant interaction effect between participant group and age [$F(2, 132)=4.62, p<.05$]. Linear regression lines were fit to the data, yielding a significant negative slope relating age and rate of commission errors in the control group, with no significant relation evident in the TBI or concussion groups. These results indicated that as individuals in the control group aged, they were less likely to make errors of commission (Figure 4.6).

Planned comparisons on the adjusted means (controlling for age) yielded no significant differences between the groups in the number of omission or commission errors made. Taken together, the pattern of accuracy results suggests that although there were no significant group differences, the concussion and control groups shifted their approach to the task as they aged. The concussion group became more conservative in their strategy, evident in the increase of omission errors. That is, as they got older, the concussion group were more reluctant to respond on go trials (i.e., resulting in higher omission error rates) perhaps in an attempt to minimize commission errors for the infrequent no-go trials. As they aged the control group became more vigilant in their approach to the task, evident in the decrease in commission errors. No change in strategy was evident in the TBI group, who may be less aware of their errors (McAvinue et al., 2005; O'Keefe, 2007), or less able to cognitively shift their approach to the task.

4.3.3 Relationship between sustained attention and boredom proneness

Analysis of the relationship between boredom and attention was conducted with bi-variate correlations between the BPS and SART variables. Estimated correlations between the full-scale BPS (as well as its individual subscale scores) and SART RTs were non-significant across all groups (Control: $r=-.041, p=.704$; Concussion: $r=.185, p=.274$; TBI: $r=-.225, p=.439$). There were also no significant correlations between BPS scores and SART RT variance across groups. However, analysis of pre and post error RTs revealed significant correlations with the internal subscale of the BPS in the control group (pre-error: $r=.232, p<.05$; post-error: $r=.240, p<.05$). That is, higher scores on the internal subscale were associated with slower RTs immediately preceding and antecedent errors of commission in controls. Of note, the correlation between the external subscale and post-error reaction times approached significance in the

control group, $r=-.208$, $p=.056$. Again, this relationship suggests that greater proneness to boredom was related to slower RTs surrounding errors of commission in controls.

Correlations between BPS scores and the rate of SART omission errors were non-significant across groups. However, a significant correlation was found between the external subscale of the BPS and the rate of SART commission errors in controls ($r=.301$, $p<.01$). That is, higher scores on the external subscale were associated with higher commission error rates in controls. No significant correlations were found between commission errors and BPS scores in the patient groups.

In light of the effects of age within the SART variance and accuracy measures, the associations between the BPS and these specific SART variables were examined using partial correlations to control for age. Correlations were found to be significant in the control group between the SART commission error rate and the BPS full-scale ($r=.225$, $p<.05$) as well as BPS external subscale ($r=.302$, $p<.01$). Thus, when controlling for age, there is a significant positive association between BPS scores and commission errors in controls, with higher levels of boredom proneness related to increased commission error rates.

The study sample was then analyzed based on subtypes of boredom proneness (i.e., apathetic vs. agitated; see Chapter 2 for definitions). In the patient groups this subdivision yielded very small sample sizes with respect to agitated boredom proneness (TBI: $n=2$; Concussion: $n=2$). Therefore, the data could not be analyzed quantitatively. Instead, t-tests were used to compare the means of the control group across boredom subtypes, and individual data points from the patient groups were examined qualitatively, relative to the controls. In addition, t-tests were used to compare groups within the apathetic subtype, which had large enough samples in each group to make meaningful comparisons.

For the apathetic subtype, there was no difference in RT between the concussion and control groups ($t=.898$, $p=.381$; Figure 4.7). However, there were significant differences between the TBI and control groups ($t=-2.3$, $p<.05$), and the TBI and concussion groups ($t=2.406$, $p<.05$), suggesting that the apathetic TBI group were slower to respond than both the concussion and control groups. This is consistent with the above mentioned finding that the TBI group had the slowest overall RT. When the control group was compared across boredom subtype, the difference in RT between apathetic ($M=300.86$)

and agitated ($M=273.43$) boredom prone controls approached significance ($t=-1.86, p=.075$), such that agitated boredom prone controls were faster to respond than apathetic boredom prone individuals. No discernible pattern was evident in the patient groups (Figure 4.7).

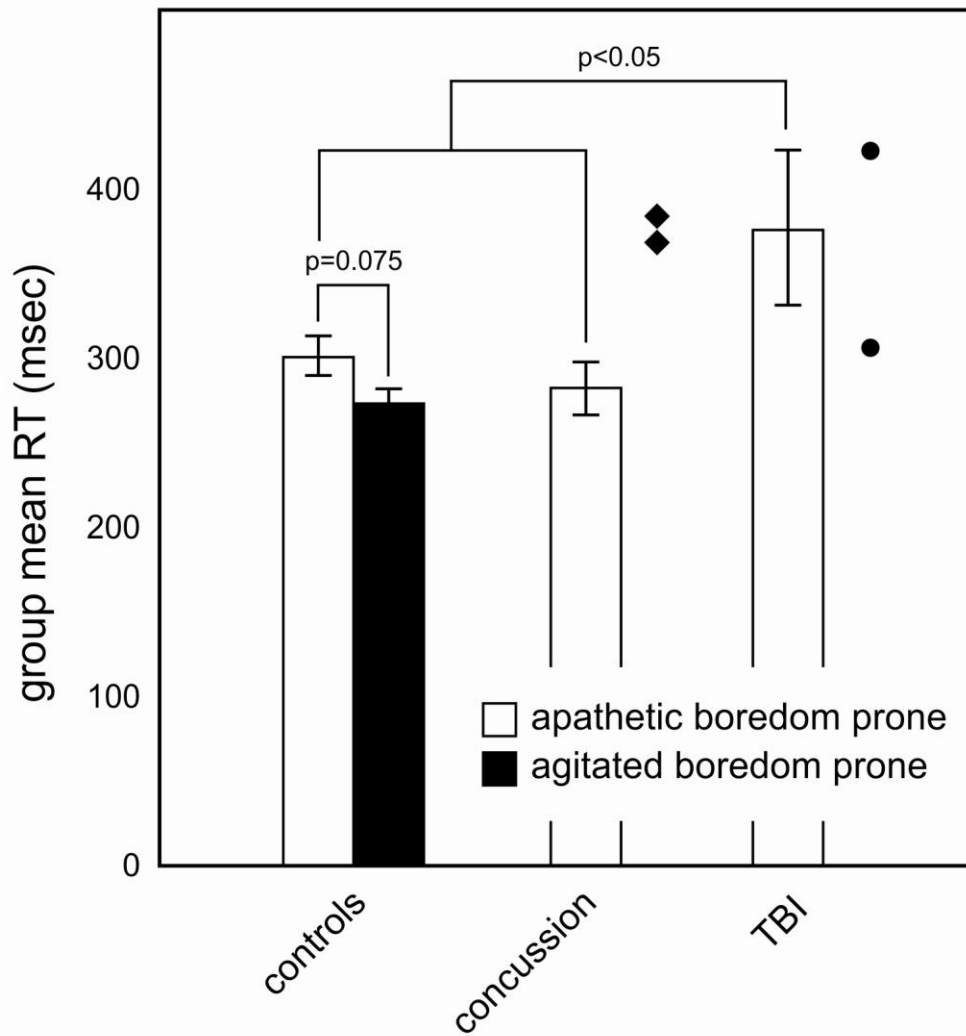


Figure 4.7. SART Reaction time across boredom proneness subtype.

Note: agitated boredom prone patients are represented as individual data points (diamond = concussed individuals; circle = TBI). Error bars represent the standard error of the mean.

To examine RT surrounding errors of commission, a difference score was calculated by subtracting the pre-error RT from the post-error RT (averaged over the three preceding and antecedent trials in each individual). Thus, a positive difference score would reflect post-error slowing. In the apathetic boredom subtype, there were no significant differences between the groups on this measure. However, when comparing the control group across the two subtypes, there was a significant difference ($t=-2.08$, $p<.05$), indicating that apathetic boredom prone controls slowed down after commission errors ($M=5.11$), a pattern which was reversed in the agitated boredom prone controls ($M=-23.78$; Figure 4.8).

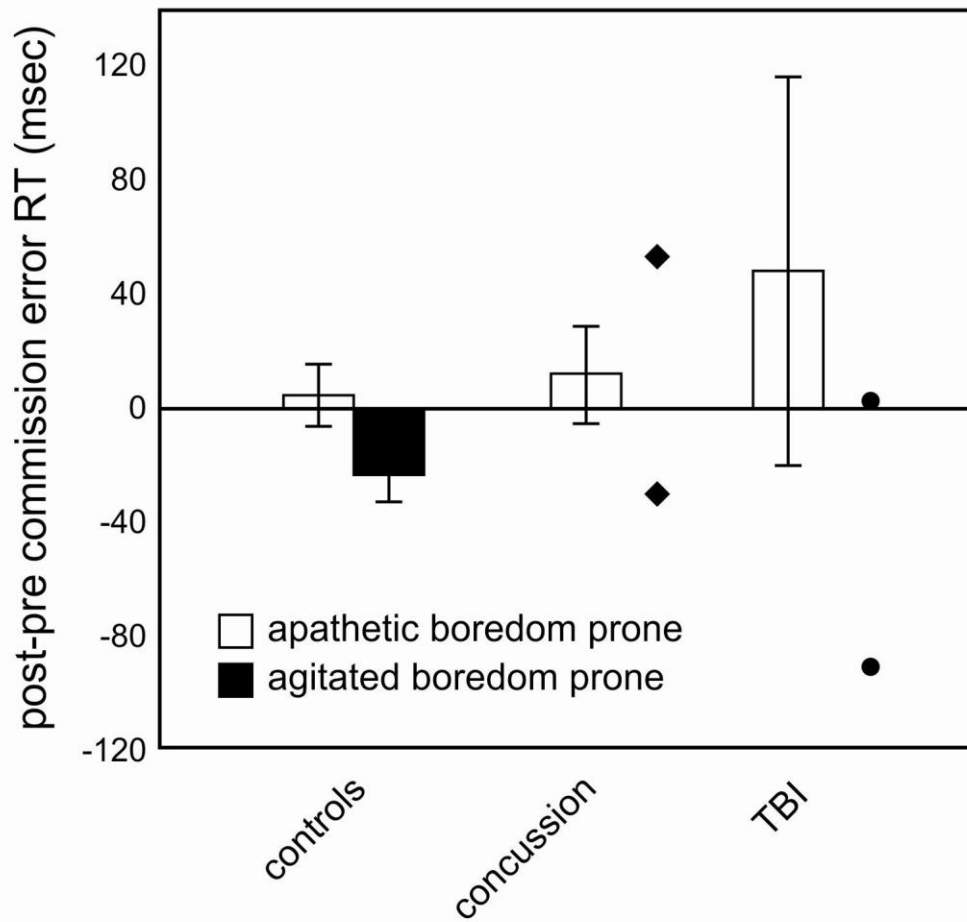


Figure 4.8. Difference in reaction time surrounding errors of commission across boredom subtype, calculated by subtracting pre error from post error reaction time.

Note: agitated boredom prone patients are represented as individual data points (diamond = concussed individuals; circle = TBI). Error bars represent the standard error of the mean.

When examining commission errors, there were no significant differences between the groups within the apathetic boredom subtype. However, when comparing the controls across boredom subtype, a significant difference was found between apathetic and agitated controls ($t=2.13, p<.05$), indicating that agitated boredom prone controls made more errors of commission ($M=15.0$) than apathetic boredom prone controls ($M=10.86$; Figure 4.9). No significant differences were found in the number of omission errors made either within the apathetic subtype (across patient groups), or across boredom subtypes (within the control group).

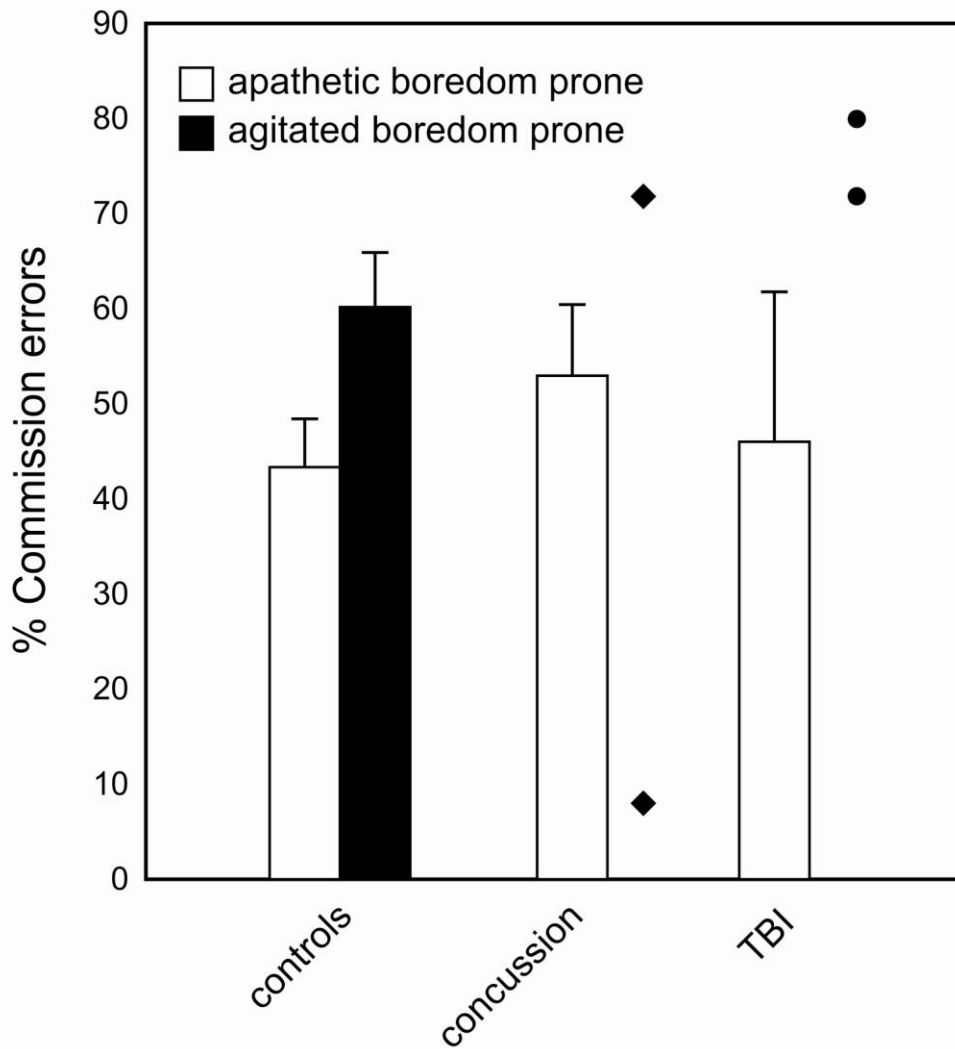


Figure 4.9. Percentage of commission errors across boredom subtype.

Note: agitated boredom prone patients are represented as individual data points (diamond = concussed individuals; circle = TBI). Error bars represent the standard error of the mean.

In sum, these results indicate that although no differences in RT were seen when the samples were examined as a whole, the apathetic boredom prone TBI individuals were slower to respond than agitated boredom prone individuals in both the concussion and control groups. Moreover, these results indicate that healthy individuals characterized as prone to experiencing agitated boredom were faster to respond, made more errors of commission, and sped up after making errors of commission relative to their apathetic boredom prone counterparts.

4.4 Discussion

Results from the current study revealed that RT on the SART increased with age across all participant groups. Contrary to previous findings (Robertson et al., 1997; Stuss et al., 1999; Manly, 2000; Manly et al., 2003; Dockree et al., 2006), results also demonstrated that individuals with TBI were slower to respond than the other groups. In addition, the TBI group demonstrated a trend towards slowing down over the course of the task suggesting that for this group, sustained attention waned over time, even though that time span was relatively short (~5 minutes). Both patient groups were also found to be more variable in their performance as they aged, and trended towards greater overall variability compared to controls. This suggests that brain injured individuals have difficulty sustaining attention over a short time period, a problem which becomes even more difficult as they age.

There were no differences between the groups in post error RT. Previous research has shown that healthy individuals will tend to slow down after making an error (Robertson et al., 1997; Chan, 2002) whereas brain injured patients will show no such sensitivity to having made an error. In other words, the failure to show post error slowing in our patients is consistent with previous literature. Why the control group in the present study were similarly insensitive to having made an error is more difficult to determine. It may be that our (mostly undergraduate) sample was not well motivated to perform the task and did not therefore make any attempts to optimize performance. Further research directly manipulating motivation levels would be needed to investigate this possibility. In one such study, Borman & Danckert (under consideration) added incentives to the SART by having a 'health bar' much like you would see on

first-person shooter video games. The bar tracked correct responses and penalized commission and omission errors. With respect to post error slowing only those in the low boredom proneness group showed significant slowing post errors. In addition, only the apathetic boredom prone group showed any relationship between motivation levels and errors of commission. This group, but not the agitated boredom prone group, showed a negative relationship between motivation levels and commission errors (i.e., higher levels of motivation were associated with lower commission error rates). So at least in this one study, motivation levels do influence sustained attention performance, with distinct influences for apathetic and agitated boredom prone individuals (Borman & Danckert, under consideration).

When accuracy was examined, results showed that all participant groups were committing errors at the same rate. Thus, although they were not making more errors than controls, the patient groups were slower to respond, suggesting a speed-accuracy trade-off. That is, patients may have needed to respond more slowly in order to achieve the same level of accuracy as controls. Results also indicated a shift in task strategy with age in both the concussion and control groups, but not the TBI group. More specifically, the concussion group became more conservative in their approach, with increased reluctance to respond on 'go' trials resulting in more errors of omission (Figure 4.6). On the other hand, the control group became more vigilant with age, evidenced by a decrease in commission error rate (Figure 4.6). The TBI group showed no shift in strategy with age, possibly owing to a lack of awareness of errors (McAvinue et al., 2005; O'Keefe, 2007), or difficulty in making shifts in cognitive strategy (Cicerone et al., 2006).

When the relationship between boredom proneness and attention was examined, significant correlations were found between the BPS and some SART variables in the control group. More specifically, greater boredom proneness was associated with pre and post error slowing, and increased commission error rate in controls. No significant relationships were found between the BPS and *any* of the SART variables in the patient groups. These analyses did not distinguish between agitated and apathetic boredom. When the sample *was* characterized by boredom proneness subtype, significant differences were found in SART performance between apathetic and agitated controls. That is, agitated boredom

prone controls were faster to respond (Figure 4.7), made more errors of commission (Figure 4.9), and sped up after making commission errors (Figure 4.8) when compared to apathetic boredom prone controls. Apathetic boredom prone controls were slower to respond and demonstrated the expected post error slowing. Thus, in healthy individuals, there was a pattern of more conservative responding among the apathetic boredom prone group, and a more impulsive response style among the agitated boredom prone group. These distinct patterns suggest different attentional profiles depending on boredom proneness subtype. Thus, claims that lapses in attention lead to boredom (Carriere et al., 2008) should be re-examined in the context of the boredom proneness subtypes. Indeed, recent work (Malkovsky et al., 2012) measuring lapses in everyday attention found that only those characterized as being prone to apathetic boredom showed such lapses. Of note, the post error slowing shown by the apathetic boredom prone controls is what would have been expected from the entire control sample (Robertson et al., 1997; Chan, 2002). When the conservative strategy of the apathetic boredom prone group is coupled with the impulsive style of the agitated boredom prone group any evidence of post error slowing was cancelled out.

Due to the small sample sizes of agitated boredom prone individuals in the patient groups, only qualitative comparisons between apathetic and agitated boredom prone patients could be made. Nevertheless, individuals in the TBI group appeared to generate approximately the same RTs in both boredom subtypes. However, in contrast to controls, agitated concussed individuals were slower to respond than their apathetic counterparts. Agitated boredom prone patients made more commission errors than their apathetic counterparts, which is consistent with the performance of healthy controls and previous research on sustained attention deficits in TBI (Robertson et al., 1997; Manly, 2000; Manly et al., 2003; Dockree et al., 2006). Perhaps previous studies of sustained attention in TBI have investigated only those TBI patients who could be characterized as experiencing boredom as an agitated state. It may also be the case that it is these individuals who demonstrate increased impulsivity, the often cited symptom of dysexecutive syndrome. With the distinct attentional profiles of the two boredom proneness

subtypes presented here - at least in the controls - it seems prudent to examine the relationship between sustained attention and boredom within this context in future work.

Chapter 5

Exploring the relationship between Boredom and Novelty Preference in TBI

5.1 Introduction

Sensation seeking - the propensity to seek varied, novel, complex, and intense sensations and experiences - is often marked by increased risk-taking behaviour, and boredom susceptibility (Zuckerman, 1971; Zuckerman, 2005; Joseph, Liu, Jiang, Lynam, & Kelly, 2009; Lawson et al., 2012; Zheng et al., 2010). While the cognitive correlates of sensation seeking are still under investigation, some have thought that sensation seeking behaviour may be related to achieving and maintaining a high level of arousal (e.g., Zuckerman & Como, 1983). This same need for arousal has been thought by many to underlie the experience of boredom (Pattyn et al., 2008; Vodanovich, 2003; Zuckerman, 2005). This is perhaps most prominent within the agitated boredom prone subtype - individuals characterized as motivated to engage in stimulating, meaningful activities. It may also be the case that the increased sensation seeking and risk taking behaviour commonly reported in TBI patients (Gianotti et al., 2009), is a consequence of boredom and the need to maintain a higher level of arousal.

The present study set out to investigate the relationship between boredom and an individual's preference for novel versus familiar stimuli. An individual with a high need for seeking novel, complex and intense sensations (i.e., a sensation seeker) would be expected to also demonstrate a preference for novelty over familiarity on the task employed here. The Cognitive Bias Task (CBT; Goldberg & Podell, 2000) was designed to examine adaptive decision making in patients with focal frontal brain lesions. In this task, participants are required to choose one of two targets that they liked best in relation to a probe. The probe differed from the target along five binary dimensions (see below for details). Targets could then be assigned a score determined by how many of those binary dimensions were shared with the probe - a high score indicating a high degree of similarity between the target and probe and vice versa. Thus, by

asking the individual to indicate which of two targets they *liked* best in relation to the probe, the task provides an index of the individual's preference for either novel (i.e., low similarity targets) or familiar (i.e., high similarity targets) stimuli (Goldberg & Podell, 2000). Results showed that males with right frontal lesions had a greater preference for familiarity, while males with left frontal lesions had a weaker preference for familiarity than males with posterior lesions and healthy controls (Goldberg & Podell, 2000).

Previous research used the CBT to examine the relationship between an individual's preference for novelty and boredom proneness (Malkovsky, et al., 2012). Results indicated that higher levels of boredom proneness were associated with a weaker preference for familiarity in healthy individuals, especially in those with *agitated* as opposed to *apathetic* boredom proneness. The present study aimed to replicate these findings, and to examine the relationship between novelty preference and boredom in patients with TBI who are commonly characterized by both increased boredom proneness and sensation seeking (Al-Adawi, Powell & Greenwood, 1998; Gianotti et al., 2009; Catalano et al., 2004). It was hypothesized that higher levels of boredom proneness would be associated with a preference for novelty, and that this would be most evident in individuals with agitated boredom proneness, who are, by the definition used here, more inclined to seek out arousing stimuli in the external environment.

5.2 Methods and Procedures

The three groups of participants in the present study have been described elsewhere (Chapter 3, Section 3.2). Preference for novel over familiar stimuli was assessed using the Cognitive Bias Task (CBT; Goldberg & Podell, 2000). This forced-choice task examines personal preference for stimuli that can be classified as either 'novel' or 'familiar' with respect to a probe stimulus. Participants are first shown a probe which varies along five binary dimensions: color (red/blue), shape (circle/square), number (1 or 2), size (large/small), and contour (outline/filled; Figure 5.1). Two target stimuli are then presented which can be characterized according to a "similarity index" - a score out of five representing the degree of similarity between the target and probe; higher scores indicate greater similarity. Each trial consists of the

presentation of one probe and two target stimuli which vary on their degree of similarity to the target (Figure 5.1).

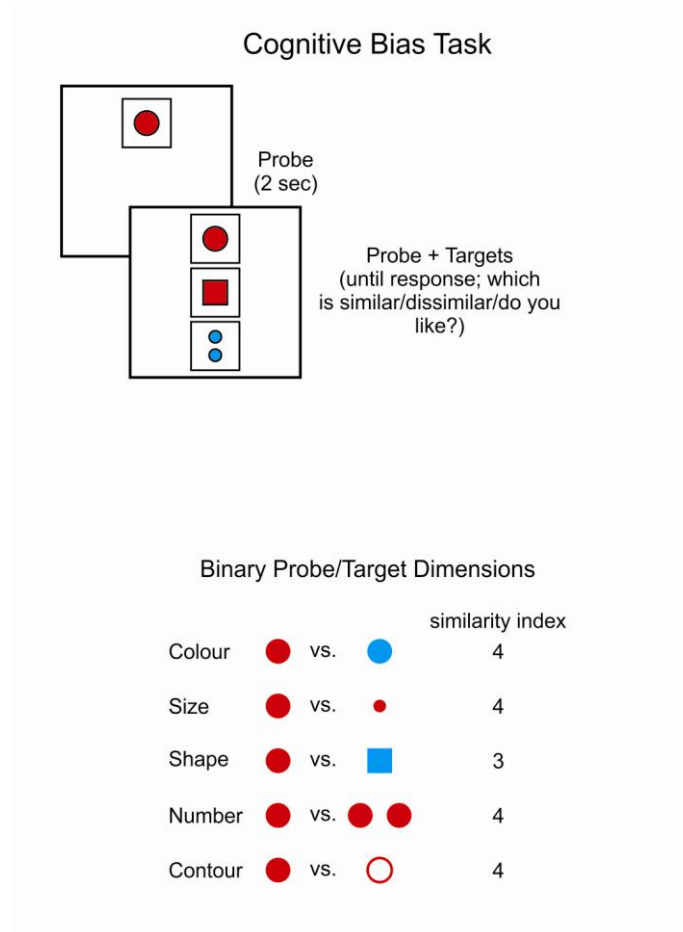


Figure 5.1. Cognitive Bias Task.

A probe stimulus is presented at the top of the screen for 2 seconds, followed by two target stimuli, which vary in their degree of relatedness to the probe. In the example given above, target 1 has four features in common with the target (colour, size, number and contour) leading to a similarity index of 4, whereas target 2 has two features in common with the target (shape and contour) leading to a similarity index of 2. Thus, if asked which target is most *similar* to the probe, the correct response would be target 1. If, when asked which stimulus do you like most, the participant chooses target 2, this would be an indication of a preference for novelty.

In three separate conditions, participants are instructed to make judgments concerning the target items with respect to their relationship to the probe. In the first two conditions participants first look at the probe and then select which of the two targets they consider to be either most similar (similar judgment) or dissimilar (dissimilar judgment) to the probe. The final condition required the participant to indicate which target they *liked* best. Similarity scores for the target stimuli are summed across trials to generate a cumulative score ranging from 80 to 220 for each condition (i.e., similar, dissimilar, and preference; optimal performance in the dissimilar condition would yield a score of 80, whereas optimal performance in the similar condition would yield a score of 220). Thus, the task yields indicators of each individual's judgments of similarity and dissimilarity, which are used as a baseline when considering their preference scores. Those with a preference for familiarity will generate a similarity index that approximates the score obtained when making similarity judgments. Conversely, those with a preference for novelty will generate a similarity index that approximates the score obtained when they made dissimilarity judgments.

The CBT task was created in SuperLabPro, and presented to participants on a 15.4 inch computer screen with a resolution of 1024 X 768 pixels. An NVIDIA 512 MB Quadro NVS 140M video card was used, with an Intel Core 2 Duo CPU at 2.60 GHz, with a refresh rate of 60 Hz. All participants completed the CBT in the lab. There were 60 trials in each of the three experimental conditions. Similar and dissimilar conditions were presented in alternating order to counterbalance effects of primacy and recency. The preference condition was always presented last.

5.3 Results

5.3.1 Performance on the CBT

Across all three groups, participant similarity indices approximated optimal performance in the dissimilar and similar conditions, and demonstrated a preference for similarity in the 'like' condition (Figure 5.2).

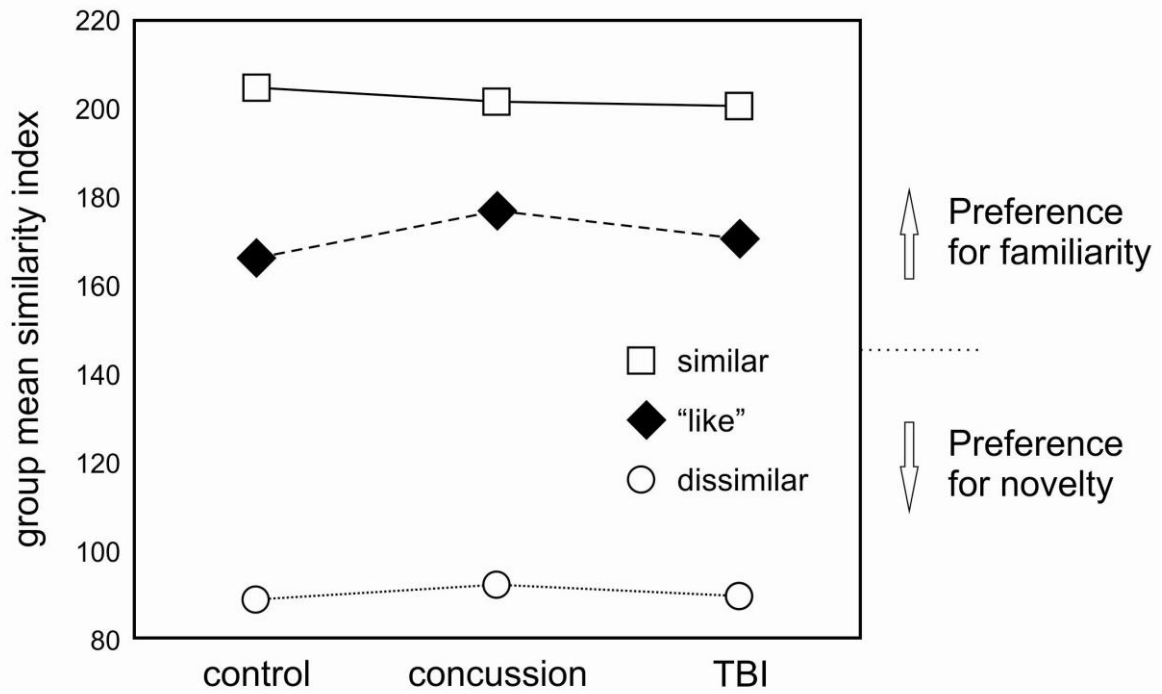


Figure 5.2. Scores on the Cognitive Bias Task across group.

Optimal similar indices were 220 in the similar condition and 80 in the dissimilar condition. Scores in the “like” condition that were above 150 indicated a preference for familiarity, whereas scores below 150 indicated a preference for novelty.

CBT scores were analyzed using ANCOVA to determine whether there were any differences between the participant groups, controlling for age. There were no significant main effects of age on dissimilar scores [$F(1,136)=.126, p=.723$], similar scores [$F(1, 136)=.220, p=.640$], or like scores [$F(1, 136)=.561, p=.455$]. Nor were there any significant interaction effects between participant group and age for the dissimilar condition [$F(2, 134)=1.73, p=.181$], the similar condition [$F(2, 134)=.310, p=.734$], or the “like” condition [$F(2, 134)=1.35, p=.262$]. Thus, the interaction term was removed from the model.

A repeated measures ANOVA with group as the between-subjects factor and similarity indices (similar and dissimilar) as the within-subjects factor revealed a significant interaction effect [$F(2, 137)=3.65, p=.028$]. Post-hoc tests of the similar condition revealed a significant difference between the TBI and control groups ($t=2.55, p=.012$), indicating that the TBI group had a significantly lower similarity score. Recall that similarity scores should optimally be approaching the *highest* possible score. Thus, the TBI group performed more poorly than controls in making similarity judgments. No significant differences were found between the TBI and concussion groups ($t=.278, p=.782$), or the concussion and control groups ($t=-1.61, p=.114$) in making judgments in the similar condition. In the dissimilar condition, results showed a significant difference between the concussion and control groups ($t=2.08, p=.040$), indicating that the concussion group had a significantly higher similarity score. Recall that similarity scores in the dissimilar condition should be optimally approaching the *lowest* possible score. Thus, the concussion group performed more poorly than controls in making dissimilarity judgments. No significant differences were found between the TBI and concussion groups ($t=.911, p=.367$), or the TBI and controls ($t=-.413, p=.681$), in making judgments in the dissimilar condition. Taken together, these results indicated that although they could do the task, patients in both groups had greater difficulty discriminating between similar and dissimilar stimuli when compared to controls.

Analysis of the “like” condition (Figure 5.2) revealed a significant difference between the concussion and control groups ($t=2.11, p=.037$), with the concussion group having higher scores and thus greater preference for familiarity than controls. No significant differences were found between the TBI and concussion groups ($t=.739, p=.464$) or the TBI and control groups ($t=.564, p=.574$). Taken together,

these results suggested that all participants demonstrated a preference for familiarity, which was greater in the concussion group relative to controls.

5.3.2 Relationship between preference for novelty and boredom

Across all three groups, analysis revealed non-significant correlations between measures of boredom (the full-scale BPS, and both of its subscales) and cognitive style (all three conditions of the CBT). However, when CBT performance was examined across boredom subtype, correlations were found in the apathetic concussion group. More specifically, there was a significant negative correlation found between the CBT dissimilar condition and the BPS external subscale ($r=-.764$, $p<.05$). That is, higher scores on the BPS external subscale were associated with lower scores on the dissimilar condition of the CBT. Results also showed a trend towards a positive correlation between the CBT dissimilar condition and the BPS internal subscale ($r=.753$, $p=.051$), with higher scores on the BPS internal subscale being associated with higher scores on the CBT dissimilar condition. These results indicated that in concussed individuals who were apathetically boredom prone, higher scores on the *internal* subscale were related to poorer performance, whereas higher scores on the *external* subscale were related to better performance in discriminating between targets and probes (i.e., dissimilarity judgments). No significant correlations were evident in the apathetic TBI or control groups. In the agitated subtype, no significant correlations were found between the BPS (full-scale and subscales) and CBT (all three conditions) in the control group. Given the small sample of agitated boredom prone patients (Concussion $N=2$, TBI $N=2$), correlations were not considered meaningful.

To further examine the relationship between CBT performance and boredom proneness subtype (Figure 5.3), separate repeated measures ANOVAs were conducted on the apathetic and agitated subtypes, with group as the between-subjects factor, and similarity indices (similar and dissimilar) as the within-subjects factor. No interaction was found in the agitated subtype [$F(2, 15)=1.35$, $p=.290$], however, a significant interaction effect was found in the apathetic subtype [$F(2,22)=4.039$, $p=.032$].

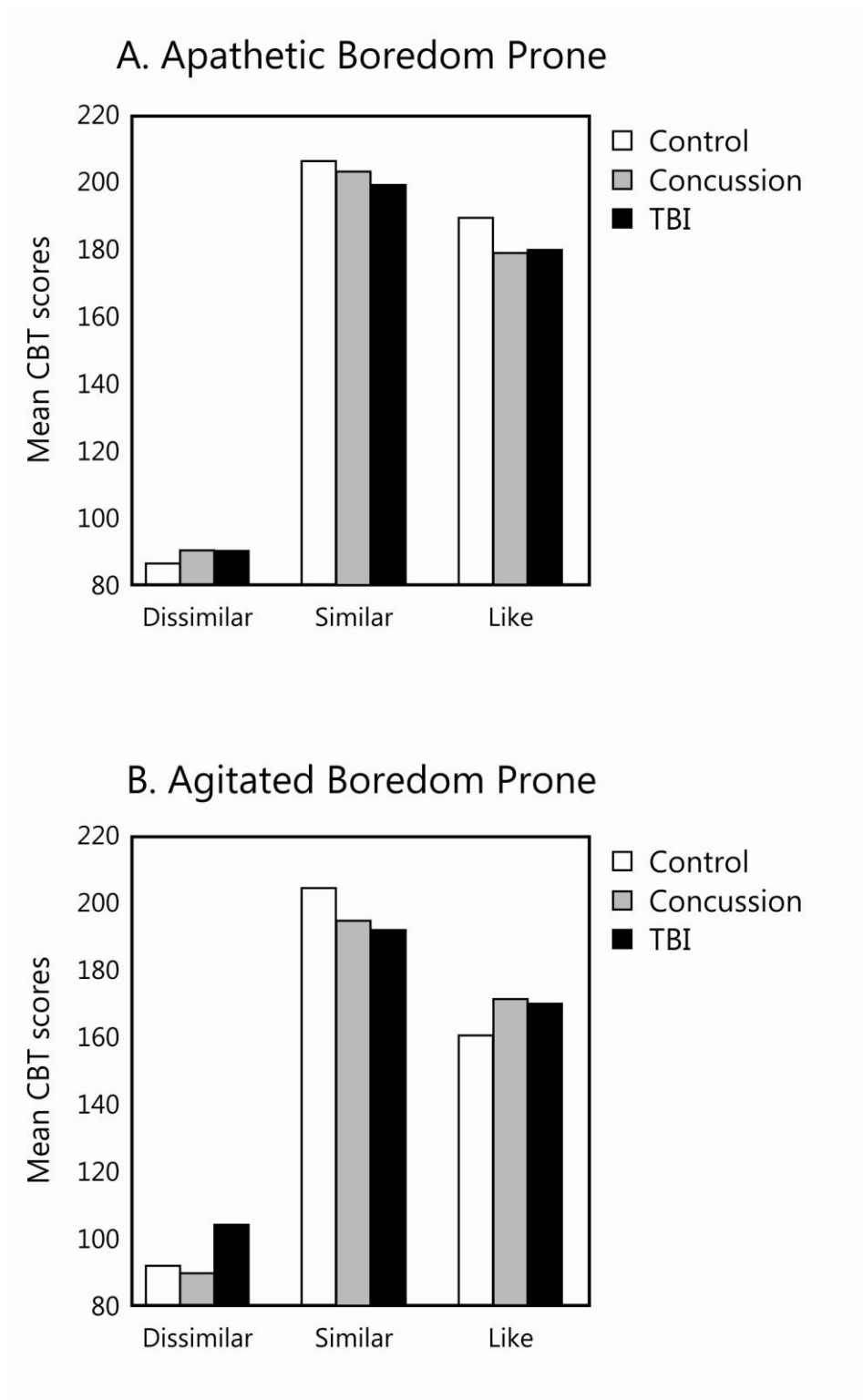


Figure 5.3. CBT scores across group in the apathetic and agitated boredom prone subtypes.

Post hoc tests of the apathetic subtype revealed the following. In the similar condition, a significant difference was found between the TBI and control groups ($t=-3.11$, $p=.007$), with lower scores in the TBI group. As similarity scores should optimally be approaching the *highest* possible score, results indicated that the TBI group performed more poorly than controls in making similarity judgments. No significant differences were found between the TBI and concussion group ($t=1.09$, $p=.306$), or concussion and control group ($t=-1.67$, $p=.111$). In the dissimilar condition, no significant differences were found between the TBI and concussion group ($t=.146$, $p=.887$), or the TBI and control group ($t=1.43$, $p=.171$). However, the difference between the concussion and control groups approached significance ($t=2.08$, $p=.052$), with higher scores in the concussion group. As optimal scores in the dissimilar condition should be approaching the *lowest* possible score, results indicated that the concussion group trended towards poorer performance than controls when making judgments of dissimilarity. Taken together, these results indicated that apathetic boredom prone patients had difficulty discriminating between similar and dissimilar stimuli when compared to apathetic boredom prone controls.

To further examine the ability to make similarity judgments between the two boredom subtypes, a difference score was calculated by subtracting the dissimilar score from the similar score (Figure 5.4). A higher score on this measure represents greater ability to discriminate between similar and dissimilar stimuli. Comparison of the means of these difference scores across boredom proneness subtype suggested that apathetic boredom prone individuals had higher difference scores than agitated boredom prone individuals in each group. This difference only approached significance in the TBI group (Controls: $t=-1.41$, $p=.179$; Concussion: $t=-.807$, $p=.725$; TBI: $t=-2.38$, $p=.076$; Figure 5.4).

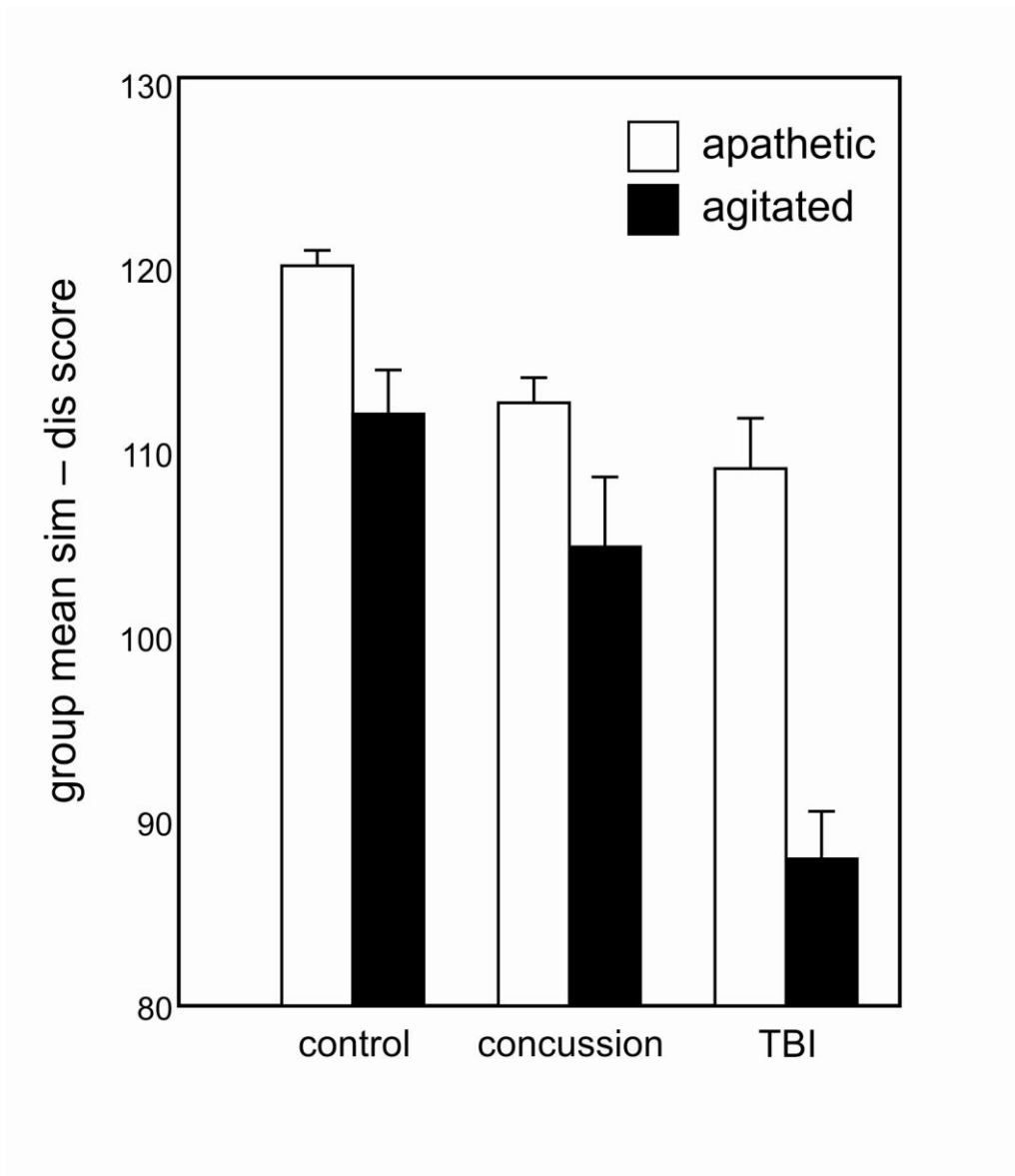


Figure 5.4. CBT difference scores across boredom proneness subtype.

Note: Difference scores were calculated by subtracting dissimilar from similar scores. Given that an optimal score on the dissimilar condition was 80, and an optimal score on the similar condition was 220, a difference score of 150 represented a perfect ability to make similarity judgments. Error bars represent the standard error of the mean.

Thus, this pattern of results suggests that individuals with agitated boredom proneness had greater difficulty discriminating target stimuli based on their similarity/dissimilarity to the probe, when compared to their apathetic counterparts. This was especially true of individuals in the TBI group.

In sum, when comparing individuals *within* the apathetic boredom prone subtype, patients had more difficulty discriminating between similar and dissimilar stimuli than controls. However, when comparing individuals *between* the two boredom prone subtypes, all participants with agitated boredom proneness (especially in the TBI group), had more difficulty discriminating between similar and dissimilar stimuli than participants with apathetic boredom proneness.

Analysis of the “like” condition across boredom subtype revealed that in all three participant groups, agitated boredom prone individuals had lower scores than their apathetic counterparts (Figure 5.5). This suggested a weaker preference for familiarity in the agitated boredom prone individuals across groups - a difference that was significant only in controls (controls: $t=3.42$, $p<.01$; concussion: $t=-.379$, $p=.716$; TBI: $t=-.248$, $p=.816$).

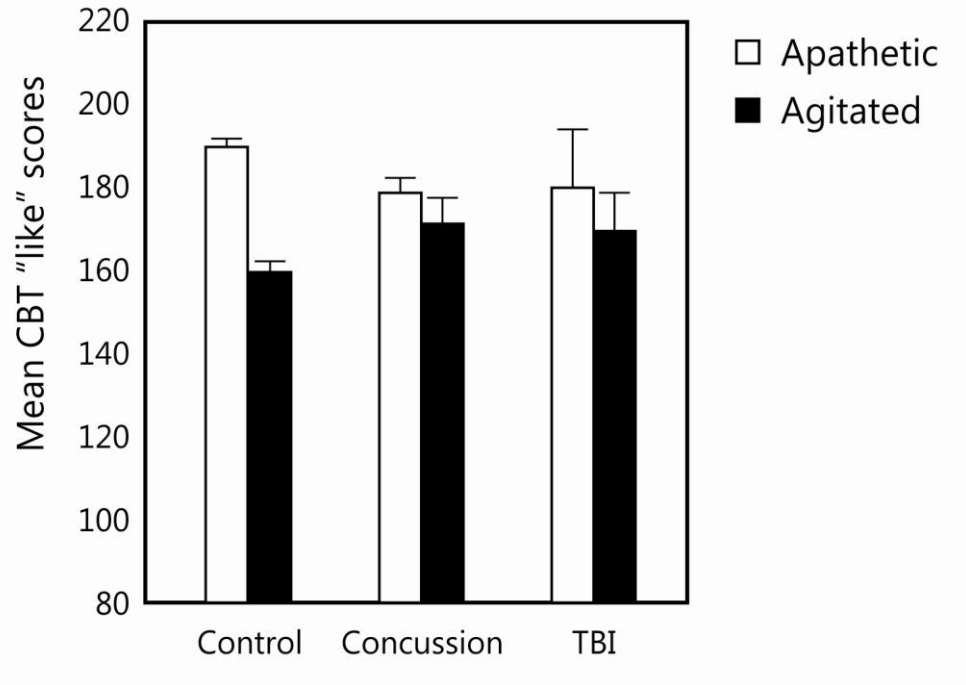


Figure 5.5. CBT "like" scores across boredom proneness subtype.

Note: Error bars represent the standard error of the mean.

Thus, controls with agitated boredom proneness had a significantly weaker preference for familiarity when compared against controls with apathetic boredom proneness, replicating previous results (Malkovsky et al., 2012).

5.4 Discussion

The goal of the present study was to examine the relationship between boredom and novelty preference. It was hypothesized that there would be a high degree of relationship between boredom and novelty preference, which would be most prominent in the TBI patient groups who are known to have increased levels of sensation seeking and risk taking behaviour (Gianotti et al., 2009). This hypothesis was not substantiated. Instead, results from the present study demonstrated that patients had more difficulty discriminating between similar and dissimilar stimuli compared to controls, a difficulty which was most prominent in the agitated as opposed to apathetic boredom prone subtype (Figures 5.3 and 5.4). It is possible that while making similarity judgments, patients were not paying as much attention to the context of the decision at hand, which led to more errors (Goldberg & Podell, 2000). That is, during the task, participants were asked to choose between two targets *relative* to a probe. If patients tended to ignore the probe, or at least processed it in a less efficient manner than controls, they would not have been as successful in making the correct decisions about the targets. Using the CBT, Goldberg & Podell (2000) found that compared to those with right frontal lesions, males with left frontal lesions ignored the probe when choosing targets that they liked the best, indicative of a difficulty in making context-dependent decisions. With respect to boredom, this would lead to a difficulty in recognizing when a stimulus or event was in fact novel. Given the poorer discrimination performance in the agitated boredom prone groups it may be the case that one potential *cause* for their boredom is a difficulty in determining what is in fact a novel event. More specifically, it is quite possible that agitated boredom proneness is rooted in a disconnection between expected and actual reward. That is, an agitated boredom prone individual may expect an event or a stimulus to be exciting, but the actual experience of the event is perceived as less exciting than predicted, yielding a feeling of agitation. Thus, it may be the *perception* of the event after

the fact that leads to the experienced discrepancy between expected and actual reward. Given the difficulties with novelty processing in agitated boredom proneness, it is possible that the inability to perceive an event or stimulus as novel or exciting is responsible for the experience of boredom. In apathetic boredom proneness, there is no expectation of reward from the external environment, so the experience of an event is not being compared to a predicted experience, and thus perception (or misperception) of novelty may not be responsible for the experience of boredom. Future studies should investigate the relationship between novelty processing and the experience of reward across boredom proneness subtypes to determine whether this is indeed the case.

All participants in the current study showed a preference for familiarity. As hypothesized, this preference was weaker in individuals with agitated boredom proneness (Figure 5.5). This finding suggests a positive association between a preference for novelty and agitated boredom proneness, in that individuals who are by nature more likely to seek external stimulation, are also more likely to prefer novel stimuli (Malkovsky et al., 2012). Surprisingly, patients with TBI did not show a stronger preference for novelty than the other groups. One possibility is that the TBI patients did not attend to the novel stimuli as efficiently. This is substantiated by the finding that both the TBI and concussion groups performed more poorly than controls when discriminating between targets and probes (Figure 5.4). That is, the patient groups may have had a harder time detecting when something was in fact novel. An alternate possibility is that TBI patients may have formed a negative emotional association with the stimuli. When faced with a choice between stimuli, TBI patients may fail to show any novelty preference because they do not see stimuli as being novel, or because that novel information also functions as distracting information relative to the probe, making it less likely to be selected. This would be consistent with previous research that shows impoverished attention to novelty and impaired novelty processing in patients with frontal lobe damage (Daffner et al., 2000; Yamagata, Yamaguchi, & Kobayashi, 2004), and research showing that previously ignored visual stimuli are rated negatively (Raymond, Fenske, & Tavassoli, 2003). Perhaps then the difficulties assimilating and accommodating novel information serve as a catalyst for the high rate of sensation-seeking behaviour commonly found in TBI patients. That is, individuals with TBI may

not be able to feel stimulated because they fail to process the stimuli that would be expected to produce stimulation. This leads to continuous search for something that will produce the desired stimulation, and leaves them feeling frustratingly bored.

Chapter 6

Conclusion

Boredom is a common human experience that is poorly understood. Various theories have been proposed to define boredom, implicating deficits in attention, motivation, arousal and/or reward circuitry. To date, there is no universal theory or definition of boredom, making it difficult to identify and treat. This thesis set out to examine the relationship between boredom, depression and attention, particularly in a population reported to experience high levels of boredom - traumatic brain injured (TBI) patients.

Research has shown that boredom is highly related to other affective mood states, most conspicuously depression, leading to the assertion that boredom is merely a by-product or symptom of affective mood states. In Chapter 2, results established boredom as an affective state that is empirically distinct from depression, apathy, and anhedonia. While related to varying degrees to these other affective states potentially due to a common *motivational* deficit, boredom was identified as a distinguishable construct. Although independent, boredom and symptoms of depression are likely to interact in complex ways. Previous research showing different rates of recovery from depression and boredom in response to pharmacotherapy (Theobald et al., 2003), highlights the need to better understand how the two constructs interact, particularly in the context of psychological disorders and neurological impairments.

Data from Chapter 2 also demonstrated that boredom cannot be considered a unitary construct and instead is comprised of two distinct subtypes which differ in their phenomenological experience, and the level of motivation to redress the experience. *Apathetic boredom proneness* is described here as a state in which one has little motivation to seek stimulation from the outside world. Instead, the individual focuses inward in attempts to achieve stimulation, however, with low motivation levels any failure to redress boredom is not perceived as being overly distressing. *Agitated boredom proneness* is described here as a desire to seek external stimulation, whereby engagement in the environment is repeatedly unsuccessful, leading to aggressive dissatisfaction. These boredom proneness subtypes were found to be differentially associated with apathy and depression, with agitated boredom proneness demonstrating a

stronger relationship to depression, whereas apathetic boredom proneness, as one would expect, showed a stronger relationship to apathy. The categorical labels of boredom proneness subtypes presented here are necessarily speculative and not without limitations. For example, the apathetic boredom prone subtype was highly related to apathy (as measured by the AES), but this relationship was driven by responses on the external subscale of the BPS, the subscale used here as an index of *agitated* boredom proneness. The behaviour subscale, which is a measure of overt goal-directed behaviour, could thus be taken as a measure of one's level of motivation to engage with the environment. Indeed, this subscale was shown to contribute to both apathy and boredom in the structural model (Figure 2.2). Intuitively then, one would expect to see a negative correlation between the behaviour subscale of the AES and the internal subscale of the BPS, representative of low motivation to engage with the external environment. This was not the case. Both the internal and external subscales of the BPS were positively correlated with the behaviour scale of the AES. However, when looking only at the agitated boredom prone group there was no relationship between the AES and BPS. Finally, one would expect to see lower scores on the behaviour subscale in individuals with apathetic boredom proneness, and higher scores in individuals with agitated boredom proneness. This was in fact the case, as individuals with apathetic boredom proneness scored significantly lower ($M=9.38$, $SD=2.7$) than individuals with agitated boredom proneness ($M=10.64$, $SD=2.7$; $t=3.82$, $p<.001$). These results suggest that the two subtypes of boredom proneness proposed here do in fact differ in terms of their endorsement of apathy. The picture is somewhat complicated by the fact that for both depression and apathy, it is the external stimulation subscale of the BPS (over the whole sample) that drives the observed relationships (and not necessarily in the expected directions). This may prompt a debate concerning the labels chosen here, or alternatively, may reflect the imprecise labeling of the subscales of the BPS. Further research is needed, perhaps with a new scale intended to specifically address the motivational difference proposed here between agitated and apathetic boredom proneness, to describe in more detail the different boredom prone subtypes.

The relationship between boredom and depression, sustained attention, and novelty seeking was examined in patients with TBI. Results showed that TBI patients were more likely to be characterized as apathetic than agitated boredom prone. This was perhaps not surprising given previous research showing a high incidence of apathy in chronic stages of TBI (Al-Adawi et al., 1998; Chervinsky et al., 1998; Glenn, 2002; Kant, Duffy, & Pivovarnik, 1998; most patients tested here were at least 11 years post injury). Interestingly, the relationship between boredom and depression was found to be greatest in patients with agitated boredom proneness, irrespective of age. Several possibilities warrant further research. Does boredom proneness subtype change over time or in response to different kinds of psychological and neurological injury? An important step in this regard would be to determine how boredom proneness changes over the lifetime in healthy individuals. Just as the elderly have been shown to have a positivity bias in recollection memory (Gallo et al., 2011; Werheid et al., 2011), it may be the case that boredom levels are lower in the elderly - that is, as a negative affective evaluation, boredom may diminish with age in those individuals demonstrating a strong positivity bias. Any diminution of boredom proneness with age may differ according to the subtype of boredom proneness. The same may turn out to be true for patients with TBI. Boredom proneness levels were not higher in the TBI group in the current thesis (although without premorbid measures it is impossible to know whether boredom proneness levels had *increased* in these patients post injury). However, given that most of those individuals were characterised as apathetically boredom prone, it may be that this subtype of boredom proneness is more likely to diminish over time than is agitated boredom proneness. Further research would be needed to test this hypothesis. Certainly, given that the agitated boredom prone individuals also had higher levels of depressive symptoms, one might hypothesise that this subtype is experienced as a more intense negative affective experience and so may have greater consequences for the individual's mental health. A recent study examining cardiac health in relation to boredom, found that chronic boredom led to increased cardiovascular disease (Britton & Shipley, 2010). Again, such a relationship may be modified by boredom proneness subtype. Given the description put forth here of agitated boredom proneness, one could hypothesise that this subtype would be most likely to suffer cardiovascular problems associated with

increased stress levels. Recent work has shown that inducing a state of boredom does in fact lead to increased cortisol levels and increased heart rates - a finding that is most prevalent in high boredom prone individuals (Merrifield & Danckert, under consideration).

The current study demonstrated that sustained attention deficits were related to boredom in healthy controls, but not in TBI patients (Chapter 4). However, across all participants, distinct attentional profiles were evident depending on boredom proneness subtype. That is, in healthy controls, agitated boredom prone individuals were faster to respond, made more errors of commission, and sped up after making commission errors, whereas their apathetic boredom prone counterparts were slower to respond and showed post-error slowing. In the patient groups, agitated boredom prone individuals trended towards more commission errors than apathetic boredom prone patients, a pattern which is consistent with the performance of the healthy controls and patients with frontal lesions (Robertson et al., 1997; Manly, 2000; Manly et al., 2003; Dockree et al., 2006). However, in contrast to controls, agitated boredom prone patients trended towards slower responding than apathetic boredom prone patients. Thus, evidence presented here highlights the need to examine the relationship between boredom and sustained attention within the context of boredom proneness subtypes, while also considering age and time since injury.

In the current investigation of novelty seeking, TBI patients had more difficulty discriminating between similar and dissimilar stimuli compared to controls. However, the TBI patients did not show a greater preference for novelty than controls. In light of the established sensation seeking behaviour of TBI patients, the latter finding at first glance appears counterintuitive. However, previous research showing impoverished attention to novelty and impaired novelty processing in patients with frontal lobe damage supports the idea that perhaps TBI patients have deficits of selective attention that impede their ability to recognize novelty (Daffner et al., 2000; Yamagata, Yamaguchi, & Kobayashi, 2004). Distinct novelty seeking profiles also emerged based on boredom proneness subtypes across participants. Individuals characterized as agitated boredom prone demonstrated poorer ability to discriminate between similar and dissimilar stimuli, and showed a weaker preference for familiarity than their apathetic boredom prone

counterparts. As with TBI patients, the difficulty in discriminating between novel and familiar stimuli may be indicative of deficits in processing novelty within the agitated boredom prone subtype. Given the model of boredom proposed here - i.e., that boredom arises as a consequence of a disconnection between expected and actual reward values of stimuli/actions - the failure to discriminate what is truly novel may be at the heart of that disconnection. In other words, any given stimulus or event would need to have an extreme 'novelty' value to be perceived as interesting by an agitated boredom prone individual. Further research is necessary to examine this relationship. Event-related potentials (ERP) represent an intriguing methodology in this respect given the wealth of research demonstrating distinct ERP signatures for novelty processing (Jiang et al., 2009; Løvstad et al., 2012; Optiz et al., 1999; Ruhnau et al., 2010; Sokhadze et al., 2009; Wetzel, Widmann, & Schroger, 2012). If novelty detection is impaired on a visual discrimination task in individuals with agitated boredom proneness, this would be reflected in reduced amplitude of the P3 response (Jiang et al., 2009; Løvstad et al., 2012; Wetzel et al., 2012). Certainly, evidence presented here suggests that it would be worthwhile to investigate the possibility that poor ability to recognize novelty plays a role in the maintenance of sensation seeking behaviour in both TBI patients and the agitated boredom prone subtype.

In sum, data presented here highlights the need to consider boredom in terms of two different subtypes, the phenomenological experience of which may change over the course of aging, and/or neurological insult. Although commonly experienced, boredom has significant cognitive and emotional consequences especially in neurological and psychiatric populations where boredom may compromise engagement in rehabilitation and recovery, and impede a successful return to normal social and occupational functioning. The identification of apathetic or agitated boredom proneness becomes especially necessary then in the context of treatment planning, where interventions will need to target specific symptoms of the boredom subtype being experienced in order to be most effective. For example, apathetic boredom prone individuals would benefit from treatments that focus on behavioural activation in order to increase motivation, whereas these treatments would likely be ineffectual if employed with agitated boredom prone individuals who already have high levels of motivation. Instead, agitated boredom

prone individuals would benefit from treatments that focus on improving novelty detection (such as modified visual or auditory discrimination tasks) as a means to experiencing desired stimulation. That is, if one can recognize and acknowledge novelty when it occurs, there may be a greater likelihood of feeling stimulated. Thus, learning how to feel stimulation may decrease the continuous, unsuccessful, and often detrimental sensation seeking behaviour formerly employed in attempts to achieve external stimulation. Along these lines, another focus of treatment for agitated boredom prone individuals should be on interpretation or appraisal of events in order to rehabilitate the disconnect between expected and actual reward. Perceiving an event as the exciting event it truly is would bridge the gap between the expectation and the outcome.

References

- Ahmed, S.M.S. (1990). Psychometric properties of the boredom proneness scale. *Perceptual and Motor Skills, 71*, 963-966.
- American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, Text Revision. Washington, DC: American Psychiatric Association.
- Anderson, T.W. (1957). Maximum likelihood estimates for a multivariate normal distribution when some observations are missing. *Journal of the American Statistical Association, 52*, 200-203.
- Arbuckle, J.L. (2006). *Amos 7.0 user's guide*. Chicago: SPSS.
- Al-Adawi, S., Powell, J.H., & Greenwood, R.J. (1998). Motivational deficits after brain injury: a neuropsychological approach using new assessment techniques. *Neuropsychology, 12* (1), 115-124.
- Barbalet, J. (2000). Boredom and social meaning. *Journal of Sociology, 50*(4), 631-646.
- Beck, A.T., Kovacs, M., & Weissman, A. (1979). Assessment of suicidal intention: The scale for suicide ideation. *Journal of Consulting & Clinical Psychology, 47*(2), 343-352.
- Beck, A.T., & Steer, R.A. (1988). *Manual for the Beck Hopelessness Scale*. San Antonio, TX: The Psychological Corporation.
- Beck, A.T., & Steer, R.A. (1990). *Manual for the Beck Anxiety Inventory*. San Antonio, TX: The Psychological Corporation.
- Beck, A.T., Steer, R.A., & Brown, G.K. (1996). *Manual for the Beck Depression Inventory-II*. San Antonio, TX: The Psychological Corporation.
- Beck, A.T., Ward, C.H., Mendelson, M., Mock, J.E., & Erbaugh, J.K. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4*, 561-571.
- Beck, A.T., Weissman, A., Lester, D., & Trexler, L. (1974). The measurement of pessimism: The hopelessness scale. *Journal of Consulting and Clinical Psychology, 42*, 861-865.
- Bentler, P. (1990). Comparative fit indexes in structural models. *Psychological Bulletin, 107*, 238-246.

- Berlyne, D.E. (1960). *Conflict, arousal, and curiosity*. New York: McGraw-Hill.
- Binnema, D. (2004). Interrelations of psychiatric patient experiences of boredom and mental health. *Issues in Mental Health Nursing, 25*(8), 833-842.
- Borman, E., & Danckert, J. (submitted). Boredom, sustained attention and the effects of motivation. *Frontiers in Cognition*.
- Britton, A., & Shipley, M.J. (2010). Bored to death? *International Journal of Epidemiology, 39* (2), 370-371.
- Browne, M.W., & Cudeck, R. (1993). Alternative ways of assessing model fit. In K.A. Bollen, & J.S. Long, (Eds.) *Testing structural equation models* (pp.136-162). Newbury Park, CA: Sage Publications.
- Buss, A.H., & Perry, M. (1992). The Aggression Questionnaire. *Journal of Personality and Social Psychology, 63*, 452-459.
- Catalano, D., Pereira, A.P., Wu, M., Ho, H., & Chan, F. (2006). Service patterns related to successful employment outcome of persons with traumatic brain injury in vocational rehabilitation. *NeuroRehabilitation, 21*, 279-293.
- 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.
- Chan, R.C.K. (2002). Attention deficits in patients with persisting postconcussive complaints: A general deficit or specific component deficit? *Journal of Clinical and Experimental Neuropsychology, 24* (8), 1081-1093.
- Chapman, L., Chapman, J., & Raulin, M. (1976). Scales for physical and social anhedonia. *Journal of Abnormal Psychology, 85*, 374-382.
- Cheyne, J.A., Carriere, J.S.A., & Smilek, D. (2006). Absent-mindedness: Lapses of conscious awareness and everyday cognitive failures. *Consciousness and Cognition, 15*(3), 578-592.
- Cheyne, J.A., Carriere, J.S.A., & Smilek, D. (2009). Absent minds and absent agents: Attention-lapse induced alienation of agency. *Consciousness and Cognition, 18*, 481-493.

- Cheyne, J.A., Solman, G.J.F., Carriere, J.S.A., & Smilek, D. (2009). Anatomy of an error: A bidirectional state model of task engagement/disengagement and attention-related errors. *Cognition, 111*, 98-113.
- Chervinsky, A.B., Ommaya, A.K., deJonge, M., Spector, J., Schwab, K., & Salazar, A. M. (1998). Motivation for traumatic brain injury rehabilitation questionnaire (MOT-Q): Reliability, factor analysis and relationship to MMPI-2 variables. *Archives of Clinical Neuropsychology, 13* (5), 433-446.
- Cicerone, K.D., Levin, H., Malec, J., Stuss, D., & Whyte, J. (2006). Cognitive rehabilitation interventions for executive function: Moving from bench to bedside in patients with traumatic brain injury. *Journal of Cognitive Neuroscience, 18*(7), 1212-1222.
- Cohen, A. (2008). The underlying structure of the Beck Depression Inventory II: A multidimensional scaling approach. *Journal of Research in Personality, 42*, 779-786.
- Csikszentmihalyi, M., Larson, R., & Prescott, S. (1977). The ecology of adolescent activity and experience. *Journal of Youth and Adolescence, 6*, 281-294.
- Csikszentmihalyi, M. (1975). *Beyond boredom and anxiety*. San Francisco, CA: Jossey-Bass.
- Darden, D.K., & Marks, A.H. (1999). Boredom: A socially disvalued emotion. *Sociological Spectrum, 19*, 13-37.
- Daffner, K.R., Mesulam, M.M., Scinto, L.F.M., Calvo, V., Faust, R., Chabrierie, A., Kennedy, B., & Holcomb, P. (2000). The central role of the prefrontal cortex in directing attention to novel events. *Brain, 123*, 927-939.
- DeCoster, J. (2007). *Applied Linear Regression Notes set 1*. Retrieved 06/01/ 2012 from <http://www.stat-help.com/notes.html>
- Dockree, P.M., Bellgrove, M.A., O'Keefe, F.M., Moloney, P., Aimola, L., Carton, S., & Robertson, I.H. (2006). Sustained attention in traumatic brain injury (tbi) and healthy controls: enhanced sensitivity with dual-task load. *Experimental Brain Research, 168*, 218-229.
- Dockree, P.M., Kelly, S.P., Roche, R.A.P., Hogan, M.J., Reilly, R.B., & Robertson, I.H. (2004).

- Behavioural and physiological impairments of sustained attention after traumatic brain injury. *Cognitive Brain Research*, 20, 403-414.
- Eastwood, J.D., Cavaliere, C., Fahlman, S.A., & Eastwood, A.E. (2007) A desire for desires: Boredom and its relation to alexithymia. *Personality and Individual Differences*, 42, 1035–1045.
- Eastwood, J.D., Frischen, A., Fenske, M.J., & Smilek, D. (in press). The unengaged mind: Defining boredom in terms of attention. *Perspectives on Psychological Science*.
- Fahlman, S.A., Mercer-Lynn, K.B., Flora, D.B., & Eastwood, J.D. (2011). Development and validation of the Multidimensional State Boredom Scale (MSBS). *Assessment*, 1-18.
- Fahlman, S.A., Mercer, K.B., Gaskovski, P., Eastwood, A.E., & Eastwood, J.D. (2009). Does a lack of life meaning cause boredom? Results from psychometric, longitudinal, and experimental analyses. *Journal of Social and Clinical Psychology*. *Journal of Social and Clinical Psychology*, 28(3), 307-340.
- Farmer, R., & Sundberg, N.D. (1986). Boredom proneness-The development and correlates of a new scale. *Journal of Personality Assessment*, 50, 4-17.
- Fawcett, J., Clark, D.C., Scheftner, W.A. & Gibbons, R.D. (1983). Assessing anhedonia in psychiatric patients: The Pleasure Scale. *Archives of General Psychiatry*, 40, 79-84.
- Fenichel, O. (1951). On the psychology of boredom. In D. Rapaport (Ed.), *Organization and pathology of thought* (pp. 349-361). New York: Columbia University Press.
- Fisher, C.D. (1993). Boredom at work: A neglected concept. *Human Relations*, 46, 395-417.
- Gallo, D.A., Korthauer, L.E., McDonough, I.M., Teshale, S., & Johnson, E.L. (2011). Age-related positivity effects and autobiographical memory detail: Evidence from a past/future source memory task. *Memory*, 19 (6), 641-652.
- Geiwitz, P. (1966). Structure of boredom. *Journal of Personality and Social Psychology*, 3(5), 592-600.
- Gianotti, L.R.R., Knoch, D., Faber, P.L., Lehmann, D., Pascual-Marqui, R.D., Diezi, C., Schoch, C., Eisenegger, C., & Fehr, E. (2009). Tonic activity level in the right prefrontal cortex predicts individuals' risk taking. *Psychological Science*, 20 (1), 33-38.

- Goldberg, E., & Podell, K. (2000). Adaptive decision making, ecological validity, and the frontal lobes. *Journal of Clinical and Experimental Neuropsychology*, 22 (1), 56-68.
- Gordon, A., Wilkinson, R., McGown, A., & Jovanoska, S. (1997). The psychometric properties of the Boredom Proneness Scale: An examination of its validity. *Psychological Studies*, 42, 85-97.
- Glenn, M.B., Burke, D.T., O'Neil-Pirozzi, T., Goldstein, R., Jacob, L., & Kettell, J. (2002). Cutoff score on the apathy evaluation scale in subjects with traumatic brain injury. *Brain Injury*, 16 (6), 509-516.
- Guilford, J.P. (1954). *Psychometric Methods 2nd ed.* New York: McGraw Hill.
- Hamilton, M. (1959). The assessment of anxiety states by rating. *British Journal of Medical Psychology*, 32, 50-55.
- Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology and Neurosurgical Psychiatry*, 23, 56-62.
- Hamilton, M. (1981). Attention, personality, and the self-regulation of mood: Absorbing interest and boredom. *Progress in Experimental Personality Research*, 10, 281-315.
- Hamilton, J.A., Haier, R.J., & Buchsbaum, M.S. (1984). Intrinsic enjoyment and boredom coping scales: Validation with personality, evoked potential, and attention measures. *Personality and Individual Differences*, 5, 183-193.
- Harris, M.B. (2000). Correlates and characteristics of boredom proneness and boredom. *Journal of Applied Social Psychology*, 30(3), 576 –598.
- Hebb, D.O. (1966). *A textbook of psychology*. Philadelphia, PA: Saunders.
- Heidegger, M. (1995). *The Fundamental Concepts of Metaphysics: World, Finitude, Solitude.* (W. McNeill & N. Walker, Trans). Bloomington and Indianapolis, IN: Indiana University Press.
- Hill, A.B. & Perkins, R.E. (1985). Towards a model of boredom. *British Journal of Psychology*, 76, 235-240.
- IBM Inc. (2011). SPSS Base 20.0 for Windows User's Guide. Chicago, IL: IBM Inc..
- Iso-Ahola, S.E., & Weissinger, E. (1990). Perceptions of boredom in leisure: Conceptualisation, reliability

- and validity of the Leisure Boredom Scale. *Journal of Leisure Research*, 22, 1-17.
- Jiang, Y., Lianekhammy, J., Lawson, A., Guo, C., Lynam, D., Joseph, J.E., Gold, B.T., & Kelly, T.H. (2009). Brain responses to repeated visual experience among low and high sensation seekers: Role of boredom susceptibility. *Psychiatry Research: Neuroimaging*, 173, 100-106.
- Joseph, J.E., Liu, X., Jiang, Y., Lynam, D., & Kelly, T.H. (2009). Neural correlates of emotional reactivity in sensation seeking. *Psychological Science*, 20 (2), 215-223.
- Kant, R., Duffy, J.D., & Pivovarnik, A. (1998). Prevalence of apathy following head injury. *Brain Injury*, 12 (1), 87-92.
- Kass, S.J., Wallace, J.C., & Vodanovich, S.J. (2003). Boredom proneness and sleep disorders as predictors of adult attention deficit scores. *Journal of Attention Disorders*, 7, 83-91.
- Kass, S.J., Vodanovich, S.J., Stanny, C.J., & Taylor, T.M. (2001). Watching the clock: Boredom and vigilance performance. *Perceptual and Motor Skills*, 92 (3), 969-976.
- Kemper, T.D. (1987). How many emotions are there? Wedding the social and the autonomic components. *American Journal of Sociology*, 93, 263-289.
- Klein, D.F. (1974). Endogenomorphic depression. *Archives of General Psychiatry*, 31, 447-454.
- Lawson, A.L., Liu, X., Joseph, J., Vagnini, V.L., Kelly, T.H., & Jiang, Y. (2012). Sensation seeking predicts brain responses in the old-new task: Converging multimodal neuroimaging evidence. *International Journal of Psychophysiology*.
- Leary, M.R., Rogers, P.A., Canfield, R.W., & Coe, C. (1986). Boredom in interpersonal encounters: Antecedents and social implications. *Journal of Personality and Social Psychology*, 51, 968-975.
- Leventhal, A.M., Chasson, G.S., Tapia, E., Miller, E.K., & Pettit, J.W. (2006). Measuring hedonic capacity in depression: A psychometric analysis of three anhedonia scales. *The Journal of Clinical Psychology*, 62, 1545-1558.
- Levy, M.L., Cummings, J.L., Fairbanks, L.A., Masterman, D., Miller, B.L., Craig, A.H. . . . Litvan, I. (1998). Apathy is not depression. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 10(3), 314-319.

- Løvstad, M., Funderud, I., Lindgren, M., Endestad, T., Due-Tønnessen, P., Meling, T., . . . Solbakk, A. (2012). Contribution of subregions of human frontal cortex to novelty processing. *Journal of Cognitive Neuroscience, 24*(2), 378-395.
- MacDonald, D.A., & Holland, D. (2002). Spirituality and boredom proneness. *Personality and Individual Differences, 32*(6), 1113-1119.
- Malkovsky, E., Merrifield, C., Goldberg, Y., & Danckert, J. (2012). Exploring the relationship between boredom and sustained attention. *Experimental Brain Research, 221*, 59-67.
- Manly, T., Owen, A.M., McAvinue, L., Datta, A., Lewis, G.H., Scott, S.K., Rorden, C., Pickard, J., & Robertson, I.H. (2003). Enhancing the sensitivity of a sustained attention task to frontal damage: Convergent clinical and functional imaging evidence. *Neurocase, 9* (4), 340-349.
- Manly, T., Robertson, I.H., Galloway, M., & Hawkins, K. (1999). The absent mind: further investigations of sustained attention to response. *Neuropsychologia, 37*, 661-670.
- Marin, R.S. (1990). Differential diagnosis and classification of Apathy. *American Journal of Psychiatry, 147*, 22-30.
- Marin, R.S. (1991). Apathy: a neuropsychiatric syndrome. *Journal of Neuropsychiatry and Clinical Neuroscience, 3*(3), 243-54.
- Marin, R.S., Biedrzycki, R.C., & Firinciogullari, S. (1991). Reliability and validity of the apathy evaluation scale. *Psychiatry Research, 38*, 143-162.
- Mathias, J.L., Beall, J.A., & Bigler, E.D. (2004). Neuropsychological and information processing deficits following mild traumatic brain injury. *Journal of the International Neuropsychological Society, 10*, 286-297.
- McAvinue, L., O'Keefe, F., McMackin, D., & Robertson, I.H. (2005). Impaired sustained attention and error awareness in traumatic brain injury: Implications for insight. *Neuropsychological Rehabilitation, 15* (5), 569-587.
- McKinley, J.C., & Hathaway, S.R. (1944). A multiphasic personality schedule (Minnesota): V. Hysteria, Hypomania, and Psychopathic Deviate. *Journal of Applied Psychology, 28*, 153-174.

- McMillan, T., Robertson, I.H., Brock, D., & Chorlton, L. (2002). Brief mindfulness training for attentional problems after traumatic brain injury: A randomized control treatment trial. *Neuropsychological Rehabilitation, 12* (2), 117-125.
- Meehl, P.E. (1987). 'Hedonic capacity' ten years later: some clarifications. In D.C. Clark and J. Fawcett (Eds.) *Anhedonia and Affect Deficit States* (pp. 33-47). New York, NY: PMA Publishing.
- Melton, A.M.A., & Schulenberg, S.E. (2007). On the relationship between meaning in life and boredom proneness: Examining a logotherapy postulate. *Psychological Reports, 101*(2):3, 1016-1022.
- Melton, A.M.A. & Schulenberg, S.E. (2009). A confirmatory factor analysis of the boredom proneness scale. *The Journal of Psychology, 143*(5), 493-508.
- Merrifield, C., & Danckert, J. (submitted). Characterising the psychophysiological signature of boredom. *Motivation & Emotion*.
- Mikulas, W.L., & Vodanovich, S.J. (1993). The essence of boredom. *The Psychological Record, 43*, 3-12.
- Mills, C.W. (1959). *The Sociological Imagination*. New York: Oxford University Press.
- Montgomery, S.A., & Asberg, M. (1979). A new depression scale designed to be sensitive to change. *British Journal of Psychiatry, 134*, 382-289.
- Musharbash, Y. (2007). Boredom, time, and modernity: An example from aboriginal Australia. *American Anthropologist, 109*(2), 307.
- Opitz, B., Mecklinger, A., Friederici, A.D., & von Cramon, D.Y. (1999). The functional neuroanatomy of novelty processing: Integrating ERP and fMRI results. *Cerebral Cortex, 9*, 379-391.
- O'Keefe, F.M., Dockree, P.M., Moloney, P., Carton, S., & Robertson, I.H. (2007). Characterising error-awareness of attentional lapses and inhibitory control failures in patients with traumatic brain injury. *Experimental Brain Research, 180*, 59-67.
- Pattyn, N., Neyt, X., Herdericlx, D., & Soetens, E. (2008). Psychophysiological investigation of vigilance decrement: Boredom or cognitive fatigue? *Physiology and Behaviour, 93*(1-2), 369-378.
- Raymond, J.E., Fenske, M.J., & Tavassoli, N.T. (2003). Selective attention determines emotional responses to novel visual stimuli. *Psychological Science, 14*, 537-542.

- Radloff, L.S. (1977). The CES-D scale: A self report depression scale for research in the general population. *Applied Psychological Measurement, 1*, 385-401.
- Reid, K. (1986). *Disaffection from school*. London: Methuen.
- Ribot, T. (1896). *La Psychologie des Sentiments*. Paris: Felix Alcan.
- 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.
- Rosvold, H.E., Mirsky, A.F., Sarason, I., Bransome, E.D., & Beck, L.H. (1956). A continuous performance test of brain damage. *Journal of Consulting Psychology, 20*(5), 343-350.
- Ruhnau, P., Wetzel, N., Widmann, A., Schröger, E. (2010). The modulation of auditory novelty processing by working memory load in school age children and adults: a combined behavioral and event-related potential study. *BMC Neuroscience, 11* (126), 1-14.
- Russell, D., Peplau, L.A., & Ferguson, M.L. (1978). Developing a measure of loneliness. *Journal of Personality Assessment, 42*, 290-294.
- Smallwood, J., McSpadden, M., & Schooler, J.W. (2007). The lights are on but no one’s home: Meta-awareness and the decoupling of attention when the mind wanders. *Psychonomic Bulletin & Review, 14* (3), 527-533.
- Seel, R.T., & Kreutzer, J.S. (2003). Depression assessment after traumatic brain injury: An empirically based classification method. *Archives of Physical Medicine and Rehabilitation, 84*, 1621-1628.
- Seib, H.M. & Vodanovich, S.J. (1998). Cognitive correlates of boredom proneness: The role of private self-consciousness and absorption. *The Journal of Psychology, 132*(6), 642-652.
- Shaw, S. (1996). Boredom, stress and social control in the daily activities of adolescents. *Journal of Leisure Research, 28*(4), 274-292.
- Simmel, G. (1997). The Metropolis and Mental Life. In D. Frisby & M. Featherstone (Eds.), *Simmel on Culture* (pp. 174–186). London: Sage Publications.
- Snaith, P. (1993). Anhedonia: a neglected symptom of psychopathology. *Psychological Medicine, 23*,

957-966.

- Snaith, R.P., Hamilton, M., Morley, S., & Humayan, A. (1995). A scale for the assessment of the hedonic tone: The Snaith-Hamilton Pleasure Scale. *British Journal of Psychiatry, 167*, 99-103.
- Sokhadze, E., Baruth, J., Tasman, A., Sears, L., Mathai, G., El-Baz, A., Casanova, M.F. (2009). Event-related potential study of novelty processing abnormalities in autism. *Applied Psychophysiology and Biofeedback, 34* (1), 37-51.
- Spitzer, R., Endicott, J., & Robins, E. (1978). Research Diagnostic Criteria: Rationale and reliability. *Archives of General Psychiatry, 35*(6), 773-782.
- Stuss, D.T., Toth, J.P., Franchi, D., Alexander, M.P., Tipper, S. & Craik, F.I.M. (1999). Dissociation of attentional processes in patients with focal frontal and posterior lesions. *Neuropsychologia, 37* (9), 1005-1027.
- Theobald, D.E., Kirsh, K.L., Hotsclaw, E., Donaghy, K., & Passik, S.D. (2003). An open label pilot study of citalopram for depression and boredom in ambulatory cancer patients. *Palliative and Supportive Care, 1*, 71-77.
- Van Baalen, B., Odding, E., van Woensel, M.P.C., van Kessel, M.A., Roebroek, M.E., & Stam, H.J. (2006). Reliability and sensitivity to change of measurement instruments used in a traumatic brain injury population. *Clinical Rehabilitation, 20*, 686-700.
- Vodanovich, S.J. (2003). Psychometric measures of Boredom: A review of the literature. *The Journal of Psychology, 137*(6), 569-595.
- Vodanovich, S.J. & Kass, S.J. (1990). A factor analytic study of the Boredom Proneness Scale. *Journal of Personality Assessment, 55*(1&2), 115-123.
- Vodanovich, S.J., Verner, K.M., & Gillbride, T.V. (1991). Boredom proneness: Its relationship to positive and negative affect. *Psychological Reports, 69*, 1139-1146.
- Vodanovich, S.J., Wallace, J.C., & Kass, S.J. (2005). A confirmatory approach to the factor structure of the boredom proneness scale: Evidence for a two-factor short form. *Journal of Personality Assessment, 85*(3), 295-303.

- Wallace, J.C., Kass, S.J., & Stanny, C.J. (2002). The cognitive failures questionnaire revisited: Dimensions and correlates. *The Journal of General Psychology, 129* (3), 238-256.
- Wallace, J.C., Vodanovich, S.J., & Restino, B.M. (2003). Predicting cognitive failures from boredom proneness and daytime sleepiness scores: an investigation within military and undergraduate samples. *Personality and Individual Differences, 34*, 635-644.
- Watson, P.J., Hickman, S.E., Morris, R.J., Stutz, N.L., & Whitting, L. (1994). Complexity of self-consciousness subscales: Correlations of factors with self-esteem and dietary restraint. *Journal of Social Behaviour and Personality, 9*, 761-774.
- Wegner, L., Flisher, A.J., Chikobvu, P., Lombard, C., & King, G. (2008). Leisure boredom and high school dropout in Cape Town, South Africa. *Journal of Adolescence, 31*, 421-431.
- Werheid, K., McDonald, R.S., Simmons-Stern, N., Ally, B.A., & Budson, A.E. (2011). Familiar smiling faces in Alzheimer's disease: Understanding the positivity-related recognition bias. *Neuropsychologia, 49* (10), 2935-2940.
- Wetzel, N., Widmann, & Schröger, E. (2012). Distraction and facilitation – two faces of the same coin? *Journal of Experimental Psychology, 38* (3), 664-674.
- Weissman, M.M., & Bothwell, S. (1976). Assessment of social adjustment by patient self-report. *Archives of General Psychiatry, 33*, 1111-1115.
- Whyte, J., Grieb-Neff, P., Gantz, C., & Polansky, M. (2006). Measuring sustained attention after traumatic brain injury: Differences in key findings from the sustained attention to response task (SART). *Neuropsychologia, 44*, 2007-2014.
- Yamagata, S., Yamaguchi, S., & Kobayashi, S. (2004). Impaired novelty processing in apathy after subcortical stroke. *Stroke, 35*, 1935-1940.
- Zheng, Y., Xu, J., Jin, Y., Sheng, W., Ma, Y., Zhang, X., & Shen, H. (2010). The time course of novelty processing in sensation seeking: An ERP study. *International Journal of Psychophysiology, 76*, 57-63.
- Zuckerman, M. (1971). Dimensions of sensation seeking. *Journal of Consulting and Clinical Psychology, 39*, 109-120.

26, 45-52.

Zuckerman, M. (1979). *Sensation seeking: Beyond the optimal level of arousal*. Hillsdale, NJ: Erlbaum.

Zuckerman, M. (2005). The neurobiology of impulsive sensation seeking: Genetics, brain physiology, biochemistry, and neurology. In Stough, C. (Ed.) *Neurobiology of exceptionalty* (pp. 31-52). New York, NY: Kluwer Academic/Plenum Publishers.

Zuckerman, M. & Como, P. (1983). Sensation seeking and arousal systems. *Journal of Personality and Individual Differences*, 4 (4), 381-386.

Appendix A

Subscales of the Boredom Proneness Scale (Ahmed, 1990)

Perceived Lack of Internal Stimulation (note: many of these items are reverse scored to determine level of boredom proneness - e.g., item 11 would be reverse scored)

2. Frequently when I am working I find myself worrying about other things.
7. I have projects in mind all the time, things to do.
8. I find it easy to entertain myself.
11. I get a kick out of most things I do.
18. I often wake up with a new idea.
22. Many people would say that I am a creative or imaginative person.
23. I have so many interests, I don't have time to do everything.
24. Among my friends, I am the one who keeps doing something the longest.
27. It seems that the same things are on television or the movies all the time; it's getting old.

Perceived Lack of External Stimulation

1. It is easy for me to concentrate on my activities.
3. Time always seems to be passing slowly.
4. I often find myself at "loose ends", not knowing what to do.
5. I am often trapped in situations where I have to do meaningless things.
6. Having to look at someone's home movies or travel slides bores me tremendously.
9. Many things I have to do are repetitive and monotonous.
10. It takes more stimulation to get me going than most people.
12. I am seldom excited about my work.
13. In any situation I can usually find something to do or see to keep me interested.
14. Much of the time I just sit around doing nothing.
15. I am good at waiting patiently.
16. I often find myself with nothing to do – time on my hands.
17. In situations where I have to wait, such as a line or queue, I get very restless.
19. It would be very hard for me to find a job that is exciting enough.
20. I would like more challenging things to do in life.
21. I feel that I am working below my abilities most of the time.
25. Unless I am doing something exciting, even dangerous, I feel half-dead and dull.
26. It takes a lot of change and variety to keep me really happy.