

**VERBAL AGGRESSION, PHYSICAL AGGRESSION  
AND INAPPROPRIATE SEXUAL BEHAVIOUR  
FOLLOWING BRAIN INJURY**

**Andrew Ian William James**

**Department of Psychology, University of York**

**Thesis submitted for the degree of**

**Doctor of Philosophy**

**November 2012**

## **ABSTRACT**

Two critical issues concerning serious disorders of interpersonal behaviour following brain injury were investigated over four studies. For the first time, verbal aggression, physical aggression and inappropriate sexual behaviour were shown to be better conceptualised as distinct forms of disordered behaviour, rather than reflecting a single dimension of behavioural dyscontrol. Study 1 demonstrated the psychometric reliability and validity of the BIRT Aggression Rating Scale (BARS) – a new observational tool with which to systematically and contemporaneously record aggressive behaviours. Study 2 used data from the BARS and measures of inappropriate sexual behaviour exhibited by 152 participants with brain injury undergoing residential neurobehavioural rehabilitation. Principal component analysis revealed a clear separation between aggression and inappropriate sexual behaviour. Furthermore, a distinction between verbal and physical aggression was also justified. These results were replicated in study 3 using observed behavioural recordings on the BARS and the St Andrew's Sexual Behaviour Assessment (SASBA) in a separate larger sample. It is recommended that these distinctions be reflected in future research.

The second critical issue was addressed across the four studies and concerned the neurocognitive correlates of each type of behaviour. Males showed an increased risk for all three behaviours, while poorer verbal skills, impaired self-awareness and poorer social participation were consistently associated with both types of aggression only. Other measures of neurocognitive function and emotional status were not significant predictors within multivariate analyses. Study 4 addressed

potential links to executive function in a subsample of 86 participants. Excessive rule violations on the D-KEFS Tower Test were associated with the presence of verbal aggression and inappropriate sexual behaviour. Scores on the D-KEFS Verbal Fluency Test had an unexpectedly positive relationship with the presence of all three behavioural conditions, perhaps indicating the importance of behavioural drive. The implications for clinical work and further research are discussed.

# TABLE OF CONTENTS

<b>CHAPTER 1. INTRODUCTION .....</b>	<b>13</b>
<b>CHAPTER 2. ACQUIRED BRAIN INJURY .....</b>	<b>15</b>
2.1.    The scope of the problem .....	15
2.2.    Traumatic brain injury .....	17
2.2.1.    Definitions and severity.....	18
2.2.2.    Traumatic brain injury epidemiology .....	19
2.2.3.    External causes and demographics.....	20
2.2.4.    Incidence and prevalence rates from the UK.....	21
2.2.5.    Incidence and prevalence rates from elsewhere .....	21
2.3.    Cerebro-vascular accident .....	22
2.3.1.    Cerebral ischaemia .....	23
2.3.2.    Intracranial haemorrhage.....	23
2.3.3.    Risk factors for stroke .....	24
2.3.4.    Incidence and prevalence .....	25
2.4.    Anoxic brain injury .....	26
2.4.1.    Epidemiology of anoxic brain injury .....	27
<b>CHAPTER 3. NEUROBEHAVIOURAL SEQUELAE OF BRAIN INJURY .....</b>	<b>29</b>
3.1.    Behavioural changes associated with brain injury .....	29
3.2.    Disinhibition after brain injury.....	31
3.3.    Aggression .....	32
3.3.1.    Prevalence .....	33
3.3.2.    Methods of measuring aggression.....	33
3.3.3.    Injury factors .....	34
3.3.4.    Emotional factors .....	35
3.3.5.    Cognitive, communication and functional skills .....	35
3.3.6.    Premorbid factors .....	36
3.4.    Inappropriate sexual behaviour .....	36
3.4.1.    Single case reports of inappropriate sexual behaviour.....	37
3.4.2.    Initial group studies of inappropriate sexual behaviour.....	38
3.4.3.    Use of the SASBA .....	39
3.5.    Gaps in the literature .....	40
<b>CHAPTER 4. STUDY 1: DEVELOPMENT OF THE BARS.....</b>	<b>41</b>
4.1.    Introduction .....	41
4.2.    Part 1: Establishing inter-rater reliability .....	46
4.2.1.    Method .....	46
4.2.1.1.    Participants .....	46
4.2.1.2.    Materials .....	46
4.2.1.3.    Procedure .....	49
4.2.1.4.    Statistical analysis .....	50

4.2.2.	Results .....	50
4.3.	Part 2: Investigating concurrent validity .....	51
4.3.1.	Method .....	51
4.3.1.1.	Participants .....	51
4.3.1.2.	Materials .....	52
4.3.1.3.	Statistical analysis .....	53
4.3.2.	Results .....	54
4.3.3.	Discussion .....	55

## **CHAPTER 5. STUDY 2: THE NEUROCOGNITIVE CORRELATES OF VERBAL AGGRESSION, PHYSICAL AGGRESSION AND INAPPROPRIATE SEXUAL BEHAVIOUR 59**

5.1.	Introduction .....	59
5.2.	Method .....	65
5.2.1.	Participants .....	65
5.2.1.1.	Demographics of sample .....	66
5.2.1.2.	Details on brain injuries and illnesses.....	66
5.2.2.	Design and materials .....	68
5.2.2.1.	Demographics and premorbid information.....	68
5.2.2.2.	Injury-related information .....	69
5.2.2.3.	Neurocognitive data .....	69
5.2.2.4.	Behavioural data .....	70
5.2.2.5.	Statistical analyses .....	71
5.2.2.6.	Dealing with excess zeroes .....	72
5.3.	Results .....	74
5.3.1.	Principal component analysis .....	74
5.3.2.	Using independent variables to predict behavioural status .....	77
5.3.3.	The subsample with full neurocognitive data.....	77
5.3.4.	Statistical modelling of group membership .....	79
5.3.4.1.	Verbal aggression .....	80
5.3.4.2.	Physical aggression .....	81
5.3.4.3.	Inappropriate sexual behaviour .....	82
5.4.	Discussion .....	82

## **CHAPTER 6. STUDY 3: TWO-PART REGRESSION MODELLING WITH NEUROBEHAVIOURAL PREDICTORS ..... 89**

6.1.	Introduction .....	89
6.1.1.	St Andrew's Sexual Behaviour Assessment (SASBA) .....	90
6.1.2.	Mayo-Portland Adaptability Inventory (MPAI-4).....	93
6.1.3.	Research questions .....	97
6.2.	Method .....	98
6.2.1.	Participants .....	98
6.2.1.1.	Demographics.....	99
6.2.1.2.	Clinical histories .....	99
6.2.1.3.	Details of brain injuries and illnesses.....	99
6.2.1.3.1	Types of injuries.....	99
6.2.1.3.2	Age at injury/illness and chronicity.....	101
6.2.1.3.3	Injury severity.....	101
6.2.1.3.4	Other relevant clinical information .....	102

6.2.2.	Materials .....	103
6.2.2.1.	Behavioural recordings .....	103
6.2.2.2.	Neurobehavioural function and care/supervision.....	106
6.2.2.3.	Other independent variables.....	107
6.2.2.3.1	Demographics and premorbid information.....	107
6.2.2.3.2	Post-injury clinical information .....	108
6.2.3.	Procedure .....	109
6.2.3.1.	Replication of independent behavioural components .....	109
6.2.3.1.1	Using the same behavioural count variables .....	109
6.2.3.1.2	Using the SASBA variables .....	111
6.2.3.2.	Two-part analyses of behavioural observations .....	111
6.2.3.3.	Re-analysis of MPAI-4 data .....	112
6.3.	Results .....	113
6.3.1.	Principal component analysis of behavioural observations .....	113
6.3.1.1.	PCA with same behavioural variables .....	113
6.3.1.2.	PCA with additional SASBA items .....	115
6.3.2.	Two-part multivariate modelling of behavioural variables .....	119
6.3.2.1.	Final set of predictor variables .....	120
6.3.2.1.1	Type of brain injury .....	120
6.3.2.1.2	Supervision Rating Scale.....	122
6.3.2.2.	Correlations between variables .....	123
6.3.2.2.1	Inter-correlations between independent variables .....	123
6.3.2.2.2	Correlations between independent and dependent variables.....	126
6.3.2.3.	Logistic regression modelling of presence/absence .....	127
6.3.2.3.1	Verbal aggression .....	127
6.3.2.3.2	Physical aggression .....	130
6.3.2.3.3	Inappropriate sexual behaviour .....	132
6.3.2.4.	Linear regression modelling of non-zero values .....	133
6.3.2.4.1	Verbal aggression .....	135
6.3.2.4.2	Physical aggression .....	138
6.3.2.4.3	Inappropriate sexual behaviour .....	140
6.3.3.	Re-analysis of the MPAI-4 .....	142
6.3.3.1.	Principal component analysis of the MPAI-4 items.....	142
6.3.3.2.	Using MPAI-4 components to predict behaviour .....	146
6.4.	Discussion .....	148
6.4.1.	Research questions .....	148
6.4.2.	Replicating behavioural distinctions.....	149
6.4.3.	Two-part regression modelling of behaviour.....	151
6.4.3.1.	Logistic regression models .....	151
6.4.3.2.	Linear regression models .....	152
6.4.3.3.	Individual predictors .....	153
6.4.3.3.1	Sex.....	153
6.4.3.3.2	Education .....	156
6.4.3.3.3	Premorbid aggression.....	157
6.4.3.3.4	Pre-injury substance abuse .....	157
6.4.3.3.5	Type of injury .....	158
6.4.3.3.6	Chronicity .....	160
6.4.3.3.7	MPAI-4 subscales .....	161
6.4.3.3.8	Care and Needs Scale.....	163

6.4.4.	Re-analysis of the MPAI-4 .....	164
6.4.4.1.	MPAI-4 items loading onto the social behaviour component.....	165
6.4.4.2.	Other MPAI-4 components .....	168
6.4.5.	Summary .....	169
<b>CHAPTER 7. STUDY 4: THE ROLE OF EXECUTIVE FUNCTIONS.....</b>	<b>171</b>	
7.1.	Introduction .....	171
7.1.1.	Executive function and aggression .....	172
7.1.2.	Executive function and inappropriate sexual behaviour .....	173
7.1.3.	Results from study 2 (chapter 5).....	174
7.1.4.	Delis-Kaplan Executive Function System (D-KEFS) .....	176
7.1.4.1.	Trail Making Test .....	177
7.1.4.2.	Verbal Fluency Test.....	179
7.1.4.3.	Color-Word Interference Test .....	180
7.1.4.4.	Tower Test.....	181
7.1.5.	Research question .....	183
7.2.	Method .....	183
7.2.1.	Participants .....	183
7.2.2.	Materials .....	185
7.2.3.	Procedure .....	186
7.3.	Results .....	186
7.3.1.	Verbal aggression .....	188
7.3.2.	Physical aggression .....	190
7.3.3.	Inappropriate sexual behaviour .....	191
7.4.	Discussion .....	192
<b>CHAPTER 8. GENERAL DISCUSSION.....</b>	<b>198</b>	
8.1.	Summaries of individual studies .....	198
8.1.1.	Study 1 – Development of the BARS.....	199
8.1.2.	Study 2 – Neurocognitive predictors .....	200
8.1.3.	Study 3 – Neurobehavioural predictors .....	201
8.1.4.	Study 4 – The role of executive function.....	203
8.2.	The distinctions between verbal aggression, physical aggression and inappropriate sexual behaviour .....	205
8.2.1.	Distinguishing between aggression and inappropriate sexual behaviour	206
8.2.2.	Different types of aggression? .....	208
8.3.	Clinical correlates of verbal aggression, physical aggression and inappropriate sexual behaviour .....	209
8.3.1.	Accuracy of models .....	210
8.3.1.1.	Aggression .....	210
8.3.1.2.	Inappropriate sexual behaviour .....	214
8.3.2.	Individual predictor variables .....	215
8.3.2.1.	Verbal skills.....	216
8.3.2.2.	Visuo-spatial skills .....	216
8.3.2.3.	Anterograde memory .....	218
8.3.2.4.	Executive function.....	218
8.3.2.5.	Self-awareness .....	220
8.3.2.6.	Emotional status .....	221

8.4. Limitations and recommendations .....	222
<b>REFERENCES .....</b>	<b>226</b>
<b>APPENDICES .....</b>	<b>275</b>
APPENDIX 1: Histograms of non-zero weighted log-transformed values .....	276
APPENDIX 2: Plots for linear regression residuals – verbal aggression .....	277
APPENDIX 3: Plots for residuals – physical aggression .....	278
APPENDIX 4: Plots for residuals – inappropriate sexual behaviour .....	279
APPENDIX 5: Correlations between MPAI-4 items and outcomes .....	280

## LIST OF FIGURES

Figure 5.1 <i>Eigenvalue screeplot for transformed behavioural counts</i> .....	75
Figure 6.1 <i>Aetiologies of brain injuries in sample for study 3</i> .....	100
Figure 6.2 <i>Eigenvalues after PCA with same variables</i> .....	114
Figure 6.3 <i>Eigenvalues after principal component analysis with six BARS and four SASBA behavioural count variables</i> .....	117
Figure 6.4 <i>Eigenvalues after principal component analysis on the 29 items of the MPAI-4</i> .....	143

## LIST OF TABLES

Table 4.1	<i>Spearman correlations between BARS I/A index and individual Neurobehavioural Rating Scale Items .....</i>	54
Table 5.1	<i>Severity of injury for those participants with TBI .....</i>	68
Table 5.2	<i>Distributions of behavioural data.....</i>	72
Table 5.3	<i>Pattern matrix for three-component structure with oblique rotation .....</i>	76
Table 5.4	<i>Between-group comparisons for neurocognitive data.....</i>	78
Table 5.5	<i>Correlations between predictor variables and behavioural groupings for subsample with complete neurocognitive data .....</i>	80
Table 5.6	<i>Final models of logistic regression analyses.....</i>	81
Table 6.1	<i>Distribution of raw behavioural counts .....</i>	110
Table 6.2	<i>Distribution for raw SASBA observations.....</i>	112
Table 6.3	<i>Pattern matrix for three-component structure of the same behavioural observations.....</i>	115
Table 6.4	<i>Pattern matrix (loadings) for three-component structure of the 10 log-transformed behavioural count items with promax rotation .....</i>	118
Table 6.5	<i>Percentage of sample showing behaviours for each injury type .....</i>	121
Table 6.6	<i>Mean and standard deviation of log-transformed non-zero behavioural values for each type of injury .....</i>	122
Table 6.7	<i>Inter-correlations between independent variables .....</i>	124
Table 6.8	<i>Correlations between predictors and two-part outcome variables .....</i>	126
Table 6.9	<i>Results of logistic regression analyses modelling presence/absence of behavioural disturbance .....</i>	129
Table 6.10	<i>Descriptive statistics for log-transformed weighted non-zero values for each behaviour .....</i>	134
Table 6.11	<i>Predictor coefficients for final model linear regression analyses predicting log-transformed non-zero behavioural values.....</i>	139
Table 6.12	<i>Component matrix of 29 MPAI-4 items following principal component analysis with varimax rotation .....</i>	144
Table 6.13	<i>Component labels and loading MPAI-4 items.....</i>	145
Table 6.14	<i>Correlations between MPAI-4 components and the three behavioural dichotomies (n = 301) .....</i>	146
Table 6.15	<i>Results of logistic regression analyses using MPAI-4 components to predict the presence of behavioural disturbance .....</i>	147
Table 7.1	<i>Between-group comparisons across several descriptors and dependent variables.....</i>	184
Table 7.2	<i>Descriptive statistics for independent variables .....</i>	187
Table 7.3	<i>Correlations between predictor and dependent variables.....</i>	188
Table 7.4	<i>Results of final model logistic regression analyses predicting group membership for presence of behavioural disturbance .....</i>	189

## **ACKNOWLEDGEMENTS**

I would like to acknowledge the assistance that I have received during the course of completing this thesis. Firstly, the encouragement of my good friend and mentor, Dr John Freeland, has been crucial to this adventure. Secondly, I would like to thank my employers, the Brain Injury Rehabilitation Trust, not only for funding my part-time PhD but also for releasing me from clinical responsibilities one day per week over the last six years in order to devote adequate time to my studies. Thirdly, the hard lessons in academic reasoning and writing provided by my supervisor, Prof Andy Young, have been invaluable to me in completing this work. Finally, I would also like to express my gratitude to my colleagues in the various BIRT units for their support in my extensive data collection as well as Ms Charlotte Poyser, who volunteered time to help with the onerous task of data entry.

## **AUTHOR'S DECLARATION**

Chapter 4 details the development of the BIRT Aggression Rating Scale (BARS). Two separate studies were conducted for this: an investigation into the inter-rater reliability of the scale with rehabilitation staff and a separate validity study involving the use of the scale with participants with acquired brain injury. The two studies have been submitted as a single manuscript for publication in a peer-reviewed journal. I would like to make clear that only the second study was my own original work. The first study was conducted in conjunction with my colleagues Dr John Freeland, Ms Tanya Corker and Mr Tom Heritage. I have included this piece of work in my thesis with their generous consent in order to present a fuller account of the development of the BARS as it is used extensively in my subsequent studies in this thesis. The remaining work presented in this thesis is entirely my own contribution.

## CHAPTER 1. INTRODUCTION

Injury to the brain is the leading cause of death and disability in young adults (Fleminger & Ponsford, 2005). In addition to physical problems affecting movement and sensation, survivors of brain injury can suffer from residual neuropsychological sequelae. These include cognitive, emotional and behavioural problems collectively known as *neurobehavioural* (Wood, 2001) deficits. Long-term neurobehavioural problems present a barrier to those with brain injury successfully reintegrating towards their premorbid lifestyles and social roles, thereby adversely affecting their quality of life.

Disturbances in interpersonal behaviour, such as aggression or inappropriate sexual behaviour, occur relatively frequently after brain injury. These behaviours can often be socially disabling or isolating (Wood & Worthington, 2001) and may require intensive, residential treatment programmes. While ultimately cost-effective (Wood, McCrea, Wood, & Merriman, 1999; Worthington, Matthews, Melia, & Oddy, 2006), these programmes are becoming increasingly expensive services to provide (Kreutzer et al., 2001).

Severe instances of aggression or inappropriate sexual behaviour can lead to individuals with brain injury being detained under the Mental Health Act (2007) or brought before the criminal justice system. In order to reduce the impact of these problems, it is important for the medical and rehabilitation sciences to develop greater understanding. This may facilitate better identification of those individuals

who are at risk of exhibiting such behaviours and to effectively plan for and deliver services for rehabilitation and treatment.

While some clinical research has been conducted into aggression following brain injury, there is comparatively little for inappropriate sexual behaviour. The current research jointly investigates verbal aggression, physical aggression and inappropriate sexual behaviour after brain injury for the first time. This approach facilitates an analysis of inter-relationships between these behaviours as well as exploring the potential predictive nature of premorbid psychosocial variables and post-injury clinically relevant variables.

A review of the relevant literature spans the following two chapters. In chapter 2, the basic neurology and epidemiology of brain injury is presented in order to demonstrate the scope and costs of this major health issue. Chapter 3 details the neurobehavioural consequences of brain injury, including the cognitive, emotional and behavioural sequelae. Particular attention is paid to post-injury aggression and inappropriate sexual behaviour. The focus of the present research is on the relationships between these forms of disordered behaviour and on the relationships between cognitive, emotional and behavioural deficits after brain injury. Participants were recruited from residential rehabilitation programmes run by the Brain Injury Rehabilitation Trust (BIRT), a national charity with units based in a number of locations around the UK. Ethical approval was obtained through the University of York, BIRT and a local NHS Trust involved in a partnership with BIRT in one of the programmes.

## **CHAPTER 2. ACQUIRED BRAIN INJURY**

Acquired brain injury is a collective term used to describe damage to the brain that is neither congenital nor progressive in nature. For ease of communication, the term *brain injury* is used throughout this work. The majority of cases of brain injury are made up of specific diagnoses of traumatic brain injury or cerebro-vascular accident (hereafter, referred to as stroke). Although some researchers have classified anoxic brain injury as a sub-category within stroke taxonomy, others have considered it as a separate pathological condition. This chapter addresses these three major types of brain injury in more detail. While there are other, less frequently occurring, neurological conditions that result in brain injury, such as intracranial neoplasm or cerebral infection, these will not be covered further.

### **2.1. The scope of the problem**

The economic costs to society in terms of loss of earning as well as the medical treatment, rehabilitation and ongoing care for survivors of brain injury are high and increasing (Kreutzer et al., 2001). In the United Kingdom, the support required for those young adults with residual disabilities may cost the UK health and social care budget more than 47 million pounds per year (Beecham, Perkins, Snell, & Knapp, 2009). Research from the United States documented that the total lifetime costs for all traumatic brain injury cases treated in one year were estimated to be over 60 billion dollars (Corrigan, Selassie, & Orman, 2010).

There is, unsurprisingly, a growing body of literature on epidemiology and prevention (Abelson-Mitchell, 2008; Steudel, Cortbus, & Schwerdtfeger, 2005). However, information obtained in this area of medicine is often complex and highly nuanced (Corrigan et al., 2010). Additionally, research into brain injury epidemiology requires population-based studies, while the majority of the clinical literature involves studies of hospital case series (Thurman, Coronado, & Selassie, 2007). The majority of these epidemiological studies have looked at specific neurological conditions such as trauma or stroke, rather than brain injury per se.

Two concepts are paramount in epidemiological studies, specifically, prevalence and incidence. Prevalence refers to the total number of cases of a particular condition within a population at a given time, sometimes expressed as lifetime prevalence. Incidence refers to the frequency of occurrence of the particular condition within the population, usually expressed as new cases per year per 100,000 people.

Brain injury contributes significantly to mortality and disability across the lifespan in the United Kingdom, as it does across the world. Recent estimates (Tennant, 2005) indicate that over 100,000 people are admitted to hospital each year in England with a diagnosis of traumatic brain injury, representing an annual incidence rate of 229 new cases each year per 100,000 of the population. A similar incidence rate for first-episode stroke has been estimated in central London (MacDonald, Cockerell, Sander, & Shorvon, 2000).

There have been two studies addressing the prevalence of brain injury, which both utilised postal surveys of general practitioners in Europe. The prevalence of brain

injury in adults was estimated at 183 per 100,000 population in Flanders, Belgium, (Lanoo, Brusselmans, Van Eynde, Van Laere, & Stevens, 2004) and 183.7 per 100,000 in County Mayo, Ireland (Finnerty, Glynn, Dineen, Colfer, & Macfarlane, 2009).

Lanoo et al. (2004) also reported an estimate for the annual incidence of brain injury in their Belgian study of 28 per 100,000 population. No analysis of incidence was reported in the Finnerty et al. (2009) Irish study.

## **2.2. Traumatic brain injury**

Traumatic brain injury has been implicated as the most common cause of death in people under 40 years of age worldwide (Wittenberg, Sloan, & Barlow, 2004). It has also been reported as the most common cause of death and disability in young people (Ghajar, 2000).

The pathophysiological mechanisms involved in traumatic injury to the brain are considered to be two-fold (Kochanek, Clark, & Jenkins, 2007; Lezak, 1995). Initially, a primary injury involves direct disruption to brain tissue, including cortical contusions, axonal injury and vascular damage leading to haemorrhage/infarction. Secondary injury refers to the subsequent physiological processes that cause additional damage, such as oedema (swelling) and the sequelae of vascular disruption.

It is widely accepted that the frontal lobes of the brain are particularly vulnerable to the effects of traumatic brain injury and that the long-term cognitive and behavioural deficits observed after severe injury are largely determined by damage to the prefrontal cortices and their interconnections (Levine, Katz, Dade, & Black, 2002). There is a consistent clinical picture of cognitive impairment after traumatic brain injury, involving slowed and inefficient attention and information processing as well as deficits in memory and executive function, which correlates with the frontal, temporal and diffuse nature of damage following trauma (Lezak, 1995; Ponsford, Sloan, & Snow, 1995).

### **2.2.1. Definitions and severity**

Inconsistencies with definitions and the usage of other terms such as “head injury” have led to complications in comparing research findings across the world. As reported by Thurman, Coronado and Selassie (2007), traumatic brain injury was defined in 1995 by the Centers for Disease Control and Prevention in the United States as “craniocerebral trauma, specifically, an occurrence of injury to the head (arising from blunt or penetrating trauma or from acceleration/deceleration forces) that is associated with any of these symptoms attributable to the injury: decreased level of consciousness, amnesia, other neurologic or neuropsychologic abnormalities, skull fracture, diagnosed intracranial lesions, or death” (p.45). The key point is that the brain insult results from an external mechanical force.

Severity of traumatic brain injury is usually classified as mild, moderate or severe. A number of methods have been used to rate severity, including measuring the depth

of unconsciousness following the injury, with the Glasgow Coma Scale score (Teasdale & Jennett, 1974). Another technique has been to measure the duration of post-traumatic amnesia, which refers to the period of acute confusion, disorientation and behavioural disturbance immediately following the emergence from coma. Instruments used in the measurement of duration of post-traumatic amnesia have included the Galveston Orientation and Amnesia Test (GOAT; Levin, O'Donnell, & Grossman, 1979), the Oxford Scale (Fortuny, Briggs, Newcombe, Ratcliff, & Thomas, 1980) and subsequent adaptations such as the Westmead (Shores, Marosszky, Sandanam, & Batchelor, 1986) and the Modified Oxford (Tate et al., 2006; Tate, Pfaff, & Jurjevic, 2000) scales. Difficulties inherent with the classification of the severity of traumatic brain injury have been noted to impact upon epidemiological studies (Summers, Ivins, & Schwab, 2009).

### **2.2.2. Traumatic brain injury epidemiology**

A World Health Organisation task force recently reported that approximately 70-90% of all TBIs are mild in severity (Cassidy et al., 2004). However, even mild injuries of this nature can result in persisting cognitive, emotional and behavioural changes (Deb, Lyons, & Koutzoukis, 1999; Malec, 1999; McKinlay, Grace, Horwood, Fergusson, & MacFarlane, 2010; McKinlay, Grace, Horwood, Fergusson, & MacFarlane, 2009; Thornhill et al., 2000).

More severe injuries, although in the minority, represent an escalating social problem due to limitations in independence and the associated economic and emotional burdens for caregivers (Lezak, 1995). Risk factors associated with higher incidence

of traumatic brain injury are consistently reported as being male, younger in age and from a lower socio-economic background (Corrigan et al., 2010; Deb, 1999; Harrison, Berry, & Jamieson, 2012; Hillier, Hiller, & Metzer, 1997; Tennant, 2005; Yates, Williams, Harris, Round, & Jenkins, 2006). A consistently higher prevalence of traumatic brain injury has been reported in offender populations for both adults (Shiroma, Ferguson, & Pickelsimer, 2010, 2012; Williams, Mewse, et al., 2010) and adolescents (Davies, Williams, Hinder, Burgess, & Mounce, 2012; Williams, Cordan, Mewse, Tonks, & Burgess, 2010).

### **2.2.3. External causes and demographics**

The major external causes of traumatic brain injury include road traffic accidents, falls and assaults. Although the relative proportions of these causes vary by country, in Europe most research has indicated that traffic accidents are the most frequent events resulting in traumatic brain injury, with falls representing the next most common (Murray et al., 1999; Tagliaferri, Compagnone, Korsic, Servadei, & Kraus, 2006). Studies from the United Kingdom show similar patterns (Wittenberg et al., 2004), although one exception involved a study from Glasgow in which falls represented the most common cause of traumatic brain injury with assaults second most common and traffic accidents much less frequent (Thornhill et al., 2000).

An Australian study (Hillier et al., 1997) reported that the leading cause of traumatic brain injury was traffic accidents (57%), then falls (29%) followed by assaults (9%), similar to the European data. Data from the Centers for Disease Control and Prevention in the United States (Corrigan et al., 2010; Summers et al., 2009) have

shown that falls (28%) are the leading cause followed by traffic accidents (20%), being struck by or against something (19%), and then assaults (11%).

#### **2.2.4. Incidence and prevalence rates from the UK**

The overall incidence rate for admission to hospital in England following a traumatic brain injury has been estimated at 229 per 100,000 (Tennant, 2005). This rate varied by a factor of 4.6 between different regions of the country, with higher proportions of the population travelling to work by public transport predicting fewer cases. Higher incidence rates have also been reported in Exeter (Yates et al., 2006) and Glasgow (Thornhill et al., 2000), with the latter reporting that alcohol was involved in over 60% of hospital admissions. A recent study (Oddy, Moir, Fortescue, & Chadwick, 2012) reported that a 48% prevalence of traumatic brain injury amongst homeless people in the UK was higher than in their control sample of 21%.

#### **2.2.5. Incidence and prevalence rates from elsewhere**

Annual incidence rates for traumatic brain injury have been reported as 506 per 100,000 population in the United States (Corrigan et al., 2010), with approximately 43% of Americans who were hospitalised going on to develop long-term disability (Selassie et al., 2008). Lower TBI incidence rates have also been reported in the literature as 174 per 100,000 in Canada (Phillips, Voaklander, Drul, & Kelly, 2009), 118 per 100,000 (with mortality of 14 per 100,000) in Northern Finland (Winqvist, Lehtilahti, Jokelainen, Luukinen, & Hillbom, 2007) and 322 per 100,000 in Australia (Hillier et al., 1997). A higher incidence rate of 1100 - 2360 per 100,000 was reported

in a comprehensive birth cohort study in New Zealand (McKinlay et al., 2008). More recently, a study looking at Australian youth aged 15-24 years (Harrison et al., 2012) documented an incidence rate of TBI at 169 per 100,000, with 87 per 100,000 noted to have highly life-threatening injuries.

In terms of prevalence, it has been estimated that around 1.1% of the US population were living with long term disability following traumatic brain injury (Zaloshnja, Miller, Langlois, & Selassie, 2008). Birth cohort studies have estimated prevalence in Northern Finland at age 34 years to be 269 per 100,000 (Winqvist et al., 2007) and a remarkably high prevalence of 30% by age 25 years in New Zealand (McKinlay et al., 2008).

### **2.3. Cerebro-vascular accident**

Cerebro-vascular accident, or stroke, is the leading cause of disability and the third leading cause of death behind cancer and heart disease in most countries (Caplan, 2006; Chong & Sacco, 2005). Episodes of stroke have been estimated to represent at least half of the neurological disorders present in a general hospital at any given time (Ropper & Samuels, 2009).

The most recent edition of Adams and Victor's Principles of Neurology defines cerebro-vascular disease as "any abnormality of the brain resulting from a pathologic process of the blood vessels, including occlusion of the lumen by embolus or thrombus, rupture of a vessel, an altered permeability of the vessel wall, or increased viscosity or other change in the quality of the blood flowing through the cerebral

vessels" (Ropper & Samuels, 2009, pp. 746-747). The term therefore refers to brain injury arising from either the obstruction of cerebral blood flow, leading to ischaemia, or haemorrhage from cerebral blood vessels. It has been reported that around 80% of all stroke events are caused by cerebral ischaemia, with 10% caused by primary intracranial haemorrhage, 5% by subarachnoid haemorrhage and the remaining 5% unclear (Warlow et al., 2001).

### **2.3.1. Cerebral ischaemia**

Ischaemia refers to a reduction or blockage in blood supply, which can lead to cell death or infarction. Obstruction of a cerebral artery by thrombosis, in which atherosclerotic deposits build up within the artery walls over time, or embolus, in which a clump of material moves within the circulatory system and lodges within a cerebral artery, is the most frequent cause of focal ischemic damage (Ropper & Samuels, 2009). More than 75% of ischaemic strokes are thrombotic in nature and around 20% embolic (Lezak, 1995). Ischaemic strokes of this nature tend to produce a consistent pattern of neurological deficits, dependent upon the artery that is involved. Less commonly, systemic hypoperfusion can arise from cardiac difficulties, hypotension or problems with oxygenation of the blood. Such a condition is likely to result in a more diffuse pattern of infarction, which is described more fully below.

### **2.3.2. Intracranial haemorrhage**

Haemorrhagic stroke is typically classified in two ways, as either intracerebral or subarachnoid. As the name indicates, intracerebral haemorrhage takes place within

the brain itself with blood leaking from a vessel (usually a small artery) into the parenchymal tissue and developing a haematoma (blood clot), which may spread into the ventricles and then the subarachnoid space. The haematoma can act as a space-occupying lesion, increasing intracranial pressure and causing mechanical disruption to brain tissue. Hypertension is the main risk factor, although poorly monitored and controlled chronic anticoagulant usage may also increase the risk (Lezak, 1995).

Subarachnoid haemorrhage refers to bleeding into the subarachnoid space, which is the space between the arachnoid and the pia mater – two of the three meningeal membranes. This form of haemorrhage typically arises from an aneurysm at branching points of the large cerebral arteries at the circle of Willis, but can also result from congenital arterio-venous malformations. While the blood is largely contained within the subarachnoid spaces, more severe haemorrhaging can lead to vasospasm, or constriction, of the vessels around the circle (Ropper & Samuels, 2009).

### **2.3.3. Risk factors for stroke**

A number of risk factors have been well established in the medical literature. These include smoking and hypertension, although the role of dietary sodium remains controversial (Ebrahim & Harwood, 1999). Diabetes mellitus, atrial fibrillation and hyperlipidaemia have also been implicated (Ropper & Samuels, 2009). Male sex has recently been shown to be a stronger risk factor for stroke (with an overall risk increase of 33% and a prevalence rate 41% higher) than previously reported

(Appelros, Stegmayr, & Terent, 2009). While younger adults are at less risk than those of increasing years, stroke in the young adult population has a large impact upon public health due to lost productivity and living longer with their disabilities (Chong & Sacco, 2005).

Despite a general reduction in mortality rates during the twentieth century (Ebrahim & Harwood, 1999), within-country variations in ethnic minority groups in the risk of stroke continue to be widely reported (Allen, 2009). In the United Kingdom, studies continue to show that there is a higher risk in those of South Asian (Gunarathne et al., 2009) and Afro-Caribbean (Heuschmann, Grieve, Toschke, Rudd, & Wolfe, 2008) descent, compared to whites. There is a similar pattern in the United States – compared to the white US population, there remains a higher risk of stroke amongst African-Americans and Hispanics (Pathak & Sloan, 2009) as well as Asian-Americans (Klatsky et al., 2005). Differences in ethnic risk factors have also been reported elsewhere, including Israel (Telman, Kouperberg, Sprecher, & Yarnitsky, 2010) and New Zealand (Bonita, Broad, & Beaglehole, 1997; Carter et al., 2006; Dyall et al., 2006; Feigin et al., 2006).

#### **2.3.4. Incidence and prevalence**

Similar incidence and prevalence rates for stroke have been reported for Western countries. A recent population-based cohort analysis of the incidence of stroke in England and Wales estimated that over 130,000 people suffer either a first or recurrent stroke each year (Carroll, Murad, Eliahou, & Majeed, 2001). Based on a population of approximately 53 million at the time, this corresponded to an incidence

rate of 245 per 100,000. Over 80% of the recorded strokes in this study were people aged over 65 years.

An approximate incidence rate of 265 per 100,000 has been reported in the United States, with an estimated 795,000 cases of stroke every year (American Heart Association, 2009). Ischemia is more frequent than haemorrhage by a factor of 6:1 (Ropper & Samuels, 2009). Prevalence was approximately 2.9% of the adult population (American Heart Association, 2009). Surveys from Australia estimate the incidence to be 40,000 to 48,000 new cases of stroke each year, 200 to 240 per 100,000 population, and prevalence to be 1.2% (Australian Institute of Health and Welfare, 2004). A recent systematic review of stroke in Arab countries reported lower incidence (ranging from 27.5 to 63 per 100,000) and prevalence (ranging from 42 to 68 per 100,000) than in Western countries (Benamer & Grosset, 2009).

## **2.4. Anoxic brain injury**

Disorders of the circulatory or respiratory systems can lead to a reduction in or total absence of oxygen delivery to brain tissue – referred to respectively as hypoxia and anoxia. These conditions occur with global reduction in blood flow (such as following cardiac arrest or heavy blood loss), suffocation (for example in drowning) and respiratory disorders (such as Guillan-Barre syndrome, which paralyses the muscles required for breathing).

The pathophysiological mechanisms that lead to cerebral damage in anoxia or ischaemia are multiple and complex (Biagas, 1999; Johnston, Nakajima, & Hagberg,

2002; Kuroiwa & Okeda, 1994). There is a selective vulnerability of specific regions in the brain based on the architecture of the cerebro-vascular system and the metabolic requirements of various brain structures. Particularly affected are the so-called cortical “watershed zones” at the extremes of the vascular territories and the basal ganglia (Caine & Watson, 2000). Anoxic or ischaemic conditions produce a cascade of pathophysiological processes leading to neuronal injury and death (Hopkins & Haaland, 2004).

Although many medical texts classify anoxic brain injury within the cerebral ischaemia sub-category of stroke (for example, Bougousslavsky & Hommel, 1993; Caplan, 2006) or as an acquired metabolic disorder (Ropper & Samuels, 2009), other researchers have considered the characteristic neuropathological and neuropsychological sequelae of cerebral anoxia in its own right (Caine & Watson, 2000). The pathophysiological mechanisms of ischaemia and pure anoxia are independent (Vendrame & Azizi, 2007), however, in the majority of clinical cases in which the brain suffers inadequate oxygen supply there is a combination of ischaemia and hypoxia (Ropper & Samuels, 2009).

#### **2.4.1. Epidemiology of anoxic brain injury**

There are a number of published reports on the incidence and prevalence data for peri-natal anoxic brain injury (see, for example, Itoo, Al-Hawsawi, & Khan, 2003; Smith, Wells, & Dodd, 2000), which is often used to rate neonatal hospital services. However, there appear to be no reports for such data in adults. Hopkins and

Haaland (2004) suggest that the potential prevalence of all hypoxic/ischaemic conditions may represent a significant public health issue.

Reports have been mixed regarding functional outcomes following rehabilitation, with one recent study reporting that outcomes for individuals with anoxic brain injury were relatively worse than for traumatic brain injury (Cullen, Crescini, & Bayley, 2009). Others, however, have shown no functional difference between the two groups (Hopkins, Tate, & Bigler, 2005; Shah, Al-Adawi, Dorvlo, & Burke, 2004). It has been suggested that outcomes are determined more by the degree of tissue loss, rather than whether the injury was anoxic or traumatic in nature (Hopkins et al., 2005).

# **CHAPTER 3. NEUROBEHAVIOURAL SEQUELAE OF BRAIN INJURY**

Lezak (1995) noted multiple factors that determine neurobehavioural changes following brain injury. These included premorbid variables (such as age, psychological makeup and life situation at the time of onset of the injury) as well as the size, location, type and severity of brain lesion. The influences of psychosocial and environmental factors after the injury are also important. This chapter reviews the literature on aggression and inappropriate sexual behaviour following brain injury, as well as detailing the clinical correlates of these behaviours.

## **3.1. Behavioural changes associated with brain injury**

There is a long history of investigations into frontal lobe damage in humans that have produced significant changes in behaviour. However, our understanding of how these lesions impact upon personality, emotion and social behaviour is incomplete and largely “unfolding” (Stuss & Knight, 2002).

Surprisingly, focal damage to the prefrontal cortex can sometimes result in no demonstrable impairment in the motor and sensory systems or the major cognitive processes. Indeed, there are published cases that report normal neuropsychological test performance in the context of severely disrupted social behaviours (Eslinger & Damasio, 1985; Wood & Rutherford, 2004). This has led researchers to theorise that the prefrontal cortex acts in an executive rather than an operational capacity and is

required only for adaptive behaviours involving the highest levels of integration, in which ambiguity and contextual factors must be considered (Mesulam, 2002).

Recent progress in neuroscience has demonstrated that the prefrontal cortex is comprised of a number of architectonically distinct regions, whose substantial and reciprocal interconnections with cortical and subcortical (thalamic and basal ganglia) areas differ (Middleton & Strick, 2001; Petrides & Pandya, 2002). A number of discrete frontal-subcortical neuroanatomical circuits have been identified that have profound regulatory influences over emotion and social behaviour.

For example, two major subtypes of frontal lobe behavioural syndrome – abulia and disinhibition – have been identified in addition to a cognitive dysexecutive syndrome and these three syndromes have been associated with separate frontal-subcortical dysfunction (for a recent review, see Saint-Cyr, Bronstein, & Cummings, 2002). Abulia, which is characterised by severe apathy, a lack of initiation and emotional blunting, has been also described as a disorder of drive (Wood, 2001) and is associated with lesions to the anterior cingulate circuit. The disinhibition syndrome, characterised by behavioural impulsivity, emotional lability and deficits in judgement and insight, has been labelled a disorder of regulatory control (Wood, 2001). Behavioural disinhibition has been associated with dysfunction of the orbitofrontal-subcortical circuit, which incorporates the orbitofrontal cortex, baso-temporal cortex, related regions of the thalamus and basal ganglia and their various interconnections. It is of note that the orbitofrontal cortex appears to be the only region of the frontal lobe that has a direct connection to the amygdala (Zald & Kim, 1996).

The dysexecutive syndrome (Baddeley, 1986) is a constellation of deficits affecting executive cognitive processes, which organise goal-directed and adaptive behaviour. Impaired initiative, planning, self-monitoring and problem-solving are all considered features of the dysexecutive syndrome (Lezak, 1995; Ponsford et al., 1995). Lesions in the dorsolateral circuit, which includes the dorsolateral prefrontal and inferotemporal cortices, have been associated with such a syndrome.

### **3.2. Disinhibition after brain injury**

As a result of severe brain injury, disorders of behavioural regulation such as aggression and inappropriate sexual behaviour are common. However, there have been inconsistencies in how these behavioural disorders have been defined (Eames, 1990). Methods used in measurement in clinical research have also varied, further complicating comparisons between findings. While a substantial body of work has now been conducted on aggression following brain injury, there has been relatively little investigation into inappropriate sexual behaviour.

Aggression in patients with prefrontal cortex damage has been described as reactive (Blair, 2001) or impulsive (Barratt & Slaughter, 1998; Brower & Price, 2001; Greve et al., 2001; Surius et al., 2004) in nature, as opposed to instrumental or planned. It has been theorised that damage to the ventromedial prefrontal (orbitofrontal and medial frontal) cortex interferes with somatic or emotional processing, leading to severe impairments in the executive control of behaviour (Bechara, Damasio, & Damasio, 2000; Tranel, 2002). Such dysfunction of the executive emotional control over “fight or flight” systems responding to external threat (Blair, 2001) therefore leads to

unplanned, reactive aggression that others consider to be disproportionate to the triggering event (Wood, 2001).

It is important to note that injury to the prefrontal cortex does not always lead to aggression or inappropriate sexual behaviour. The behavioural expression of this injury is considered to be influenced by the type and severity of the lesion as well as the patient's premorbid personality (Golden, Jackson, Peterson-Rohne, & Gontovsky, 1996).

A three year longitudinal study estimated a minimum incidence of 2.5 individuals per 100,000 population exhibiting behavioural disturbances following severe traumatic brain injury (Johnson & Balleny, 1996). Behaviour change, which was noted in 26 of 33 (79%) patients, was measured via a short questionnaire completed by hospital staff and relatives, which addressed 7 typical post-injury behaviours: apathy, restlessness, impulsivity, irritability, aggression and disinhibition (including sexual). It was noted that aggression and disinhibition were more evident once the patients had been discharged home from hospital, in the post-acute phase of their recovery...

### **3.3. Aggression**

Aggression after brain injury has been conceptualised as reflecting both abnormal processes within the brain and interactions between the damaged brain and the external environment (Wood & Liossi, 2006b). Careful behavioural analysis has shown that antecedents and environmental factors, such as the patient being given a verbal instruction, are relevant to aggression occurring (Alderman, 2007). Aggressive

behaviour after brain injury is more likely to be verbal rather than physical in nature (Dyer, Bell, McCann, & Rauch, 2006; Rao et al., 2009). The reported clinical correlates of aggression after brain injury are mixed and have included both physiological and psychological associations.

### **3.3.1. Prevalence**

There have been a number of reports on the prevalence of aggression following the acute phase of recovery after traumatic brain injury. However, differences in sample recruitment, chronicity since injury and methods used to measure aggression have contributed to rates varying between 25% and 38% (Dyer et al., 2006; Johansson, Jamora, Ruff, & Pack, 2008; McKinlay, Brooks, Bond, Martinage, & Marshall, 1981; Rao et al., 2009; Tateno, Jorge, & Robinson, 2003). Importantly, one longitudinal study noted that prevalence remained at around 25% at 5 years post-injury (Baguley, Cooper, & Felmingham, 2006).

### **3.3.2. Methods of measuring aggression**

The majority of studies investigating post-injury aggression have measured aggression via clinical interview and self-report questionnaires, often in a retrospective manner. However, systematic and careful assessment of behaviours is required in order to overcome the biases and distortions inherent in normal human judgement (Kazdin, 2001). A superior method of measuring aggression in a clinical sample, therefore, is to record behaviours in a systematic and objective manner as they occur. This method has been utilised by Alderman and colleagues using a

modified version of the Overt Aggression Scale (Alderman, 2007; Alderman, Knight, & Henman, 2002; Alderman, Knight, & Morgan, 1997). Surius and colleagues (2004), in their review of instruments used to measure aggression in clinical and research settings, recommend that a statistically reliable tool applicable to the intended population is important.

### **3.3.3. Injury factors**

There have been mixed reports regarding the influence of the brain injury itself on aggressive behaviour. In a longitudinal study of 228 patients with moderate to severe traumatic brain injury (Baguley et al., 2006), there were no associations with injury pattern on CT scan. Although relatively common, aggression was transient in nature and related to psychosocial variables rather than the organic nature of the injury. Other studies have also reported no association with presence of abnormality on CT scan with aggression (Wood & Liossi, 2006b).

However, there are studies that have shown a relationship between post-injury aggression and the presence of frontal lobe (Chan, Campayo, Moser, Arndt, & Robinson, 2006; Grafman et al., 1996; Kim, Choi, Kwon, & Seo, 2002; Tateno et al., 2003) and temporal lobe (Pontius & LeMay, 2003; Tonkonogy, 1991) lesions. No effects of size or laterality of lesion were predictive of post-acute stroke aggression (Kim et al., 2002). Severity of injury, as measured by lowest Glasgow Coma Scale score or duration of post-traumatic amnesia, has been reported to be unrelated to aggression (Tateno et al., 2003; Wood & Liossi, 2006b).

### **3.3.4. Emotional factors**

Depression has been reported to predict aggression in some studies (Baguley et al., 2006; Johansson et al., 2008; Rao et al., 2009; Tateno et al., 2003) but not others (Alderman, 2007; Paradiso, Robinson, & Arndt, 1996; Wood & Liossi, 2006b).

Emotional lability and poor frustration tolerance have also been linked with aggression in adolescents (Dooley, Anderson, Hemphill, & Ohan, 2008).

### **3.3.5. Cognitive, communication and functional skills**

Poorer communication, social skills and greater functional impairment have been associated with post-injury aggression (Alderman, 2007; Alderman et al., 2002; Johansson et al., 2008; Rao et al., 2009). Behavioural impulsivity has been also identified as a predictor (Dyer et al., 2006; Greve et al., 2001; Wood & Liossi, 2006b).

In acute stroke patients, aggressive behaviour has been reported to be relatively common and associated with greater cognitive impairment (Chan et al., 2006). No relationship between post-injury cognitive status, as measured by the Mini Mental State Examination, and aggression was reported by Tateno et al. (2003). However, research using more sophisticated cognitive assessments has shown that aggressive patients perform more poorly on tests of verbal memory and visuo-spatial function (Wood & Liossi, 2006b), suggesting that verbal abilities may play a critical modulatory role in aggression.

### **3.3.6. Premorbid factors**

Mixed results have also been reported for the relevance of premorbid factors in aggression following brain injury. A history of substance abuse and prior aggression was found to be predictive in some studies (Greve et al., 2001; Tateno et al., 2003) but not others (Johansson et al., 2008; Rao et al., 2009). In a paediatric traumatic brain injury sample, a premorbid history of aggression or inattention was associated with a greater risk of post-injury aggression (Cole et al., 2008).

## **3.4. Inappropriate sexual behaviour**

There have been a number of case series and individual case studies reported in the literature on inappropriate sexual behaviour after brain dysfunction in dementia and developmental disabilities. The prevalence of inappropriate sexual behaviour in these populations has been reported to range from around 2% to 15% (Alagiakrishnan et al., 2005; Burns, Jacoby, & Levy, 1990; Tsai, Hwang, Yang, Liu, & Lirng, 1999), considerably lower than similar estimates for aggression (Knight et al., 2008). This section will focus on reviewing the research involving patients with acquired brain injury.

Following severe brain injury, it is relatively common for patients to exhibit sexually inappropriate behaviours such as making lewd comments or masturbating (Bezeau, Bogod, & Mateer, 2004). Alterations in sexual preferences, libido and in the regulation of sexual behaviour have been demonstrated after brain dysfunction (Hibbard, Gordon, Flanagan, Haddad, & Labinsky, 2000; Zasler, 1994).

Consequently, many of these people experience breakdown in their personal relationships, may become the target of other people's aggressive reactions and even come to the attention of the criminal justice system.

### **3.4.1. Single case reports of inappropriate sexual behaviour**

Miller and colleagues (1986) described a series of eight individuals who, following differing aetiologies of brain injury, developed changes in their sexual behaviour. Four cases exhibited an increase in sexual activity, or hypersexuality, and displayed this heightened drive with disinhibited sexual and other social behaviours in public. Two of these had demonstrable basal frontal lobe injury, while another had temporal lobe epilepsy. The frontal lobes have been implicated in the mediation of the motor components of sexual behaviour and the control of sexual responses (Baird, Wilson, Bladin, Saling, & Reutens, 2007). Emory, Cole and Meyer (1995) also documented a case series of eight males with traumatic brain injury who developed problematic inappropriate sexual behaviour some years after their initial injury. Similar behaviour has also been reported following traumatic brain injury in a 9 year old boy (Fyffe, Kahng, Fitro, & Russell, 2004) and in three men with frontal encephalomalacia, or brain softening due to haemorrhage or inflammation (Woods, Sigford, & Lanham, 1998).

Other case reports have documented acquired paedophilia with poor control over sexual impulses subsequent to right orbitofrontal tumour (Burns & Swerdlow, 2003) and other neurological conditions (Mendez & Shapira, 2011). Hypersexuality has been described following surgery to remove a left occipital arterio-venous

malformation, which resulted in dysfunction in the left frontal lobe and left hippocampal gyrus (Cao, Zhu, Wang, Wang, & Zhao, 2010). Hypersexuality as part of a Kluver-Bucy syndrome has been reported in numerous cases in the acute stage of neurological illness and temporal lobe epilepsy (for example, Cohen, Park, Kim, & Pillai, 2010; Kusano, Horiuchi, Tanaka, Tsuji, & Hongo, 2012; Lin, Yeh, Chen, Chang, & Chen, 2011; Rashid, Eder, Rosenow, Macken, & Schuele, 2010).

### **3.4.2. Initial group studies of inappropriate sexual behaviour**

There have been fewer group studies of inappropriate sexual behaviour following brain injury. These have focused on records of post-injury sexual “offending” or “aberrant” behaviour (Simpson, Blaszczynski, & Hodgkinson, 1999; Simpson, Tate, Ferry, Hodgkinson, & Blaszczynski, 2001) or on investigating the prevalence of prior brain injury amongst groups of adults who have committed sexual offences (DelBello et al., 1999; Langevin, 2006). One study surveying the staff working in a paediatric residential brain injury rehabilitation setting noted significant inappropriate behaviour (Luiselli, Sherak, Dunn, & Pace, 2005).

The initial Simpson et al. (1999) study documented that 6.5% of 477 consecutive admissions to a hospital-based brain injury rehabilitation unit had committed some form of “sexual offence”. All were male and represented 7.9% of the male sample. Their follow-up study in 2001 included between-group comparisons of 25 of these males showing sexually aberrant behaviour after traumatic brain injury with 25 carefully matched control brain injured cases. No relationships were found between

post-injury sexually aberrant behaviour and premorbid psychosocial disturbance or post-injury radiological, medical, or neuropsychological variables.

Amongst convicted sexual offenders, a substantial prevalence of prior brain injury has been reported. Nine (36%) of the DelBello et al. (1999) sample of 25 male offenders had a history of at least one TBI. Langevin (2006) reported a larger sample of 476 male sexual offenders and noted that 49.3% had sustained a TBI with loss of consciousness, while 22.5% had sustained significant neurological injury.

A recent review paper (Johnson, Knight, & Alderman, 2006) has recommended that continuous assessment of inappropriate sexual behaviours with structured measurement tools is desirable. The same group of researchers subsequently developed a standardised system for continuously recording challenging sexual behaviour, the St Andrew's Sexual Behaviour Assessment (SASBA; Knight et al., 2008).

### **3.4.3. Use of the SASBA**

Two studies have since been published involving the SASBA (Alderman, Knight, & Birkett-Swan, 2009; Stewart, Knight, Alderman, & Haywad, 2010). The former was a clinical audit survey of aggression (as measured by the OAS-MNR) and inappropriate sexual behaviour (as measured by the SASBA) in a sample of 91 inpatients over 3 months in a hospital ward specialising in challenging behaviour following brain injury. It was reported that inappropriate sexual behaviour was observed less frequently than aggression, that the two behaviours showed only a

“modest” correlation and that there were different functional elements to the behaviours. The latter study involved a survey of SASBA recordings exhibited by 97 older adults within a specialised ward and reported a prevalence of 32% exhibiting inappropriate sexual behaviour.

### **3.5. Gaps in the literature**

There have been very few published studies specifically analysing observations of aggressive and inappropriate sexual behaviour following brain injury together within the same sample. The question of whether both types of behaviour may be considered part of the same dimension of poorly regulated, disinhibited and impulsive behaviour has therefore not yet been dealt with adequately. This could be addressed via exploratory factor analysis studies, which might be used to reveal the underlying structure across a range of behavioural observations. Would all instances of disordered interpersonal behaviour load onto the same dimension or would there be evidence for two or more separate clinical entities?

There has been some work published concerning the clinical correlates of aggression following brain injury. However, few of these studies involved contemporaneously and systematically recorded aggressive behaviours. The clinical correlates of inappropriate sexual behaviour following brain injury have not yet been adequately explored. Not surprisingly, these have not both been examined within the same sample of participants.

## **CHAPTER 4. STUDY 1: DEVELOPMENT OF THE BARS**

### **4.1. Introduction**

Among the greatest obstacles to social re-integration following acquired brain injury (ABI) are the neurobehavioural sequelae relating to the regulation of mood, behaviour and executive functioning (Wood et al., 1999; Worthington et al., 2006). Increased irritability and aggressive behaviour are two of the most frequently observed and socially-restrictive of these sequelae, and as such, provide a considerable challenge for neurorehabilitation services (Alderman, 2007; Alderman et al., 2002). These behaviours not only restrict the lives of individuals in neurorehabilitation settings, but can also have a long-term negative impact on their families and care-givers (Kolakowsky-Hayner, Miner, & Kreutzer, 2001; Oddy & Herbert, 2003).

Addressing these issues is therefore a priority for neurorehabilitation services. To gauge the effectiveness of behavioural or psychotropic interventions in a neurorehabilitation setting requires measures be taken of the intensity and nature of aggressive incidents (Chatham Showalter & Kimmel, 2000). Clinical experience indicates that barriers to reliably measuring aggression include staff familiarity with rating systems, staff recall of the rating system and staff training.

Anecdotal measurements of aggressive behaviours, such as review of treatment narratives, are prone to a number of threats to their accuracy, such as the well-documented saliency and recency effects (Miller & Campbell, 1959). In addition, staff

proximity to aggression may result in a distorted interpretation of these events unless a robust system of measurement is used. Historically, hospital incident reports have been used as the primary source of staff-collected information relating to aggressive behaviours. The data obtained from these sources, however, may not accurately reflect actual levels of aggression (Iverson & Hughes, 2000).

A number of different approaches have been used to assess aggressive behaviours in both general psychiatric services and neurorehabilitation facilities. Occasionally, self-report measures will be used to gauge service-user's aggressive feelings. In a recent review on the pharmacological management of aggression (Fleminger, Greenwood, & Oliver, 2003), one out of the six studies evaluated used a self-assessment tool as an outcome measure for assessing treatment (Mooney & Haas, 1993). Historically, one of the more commonly used of these tools has been the Buss-Durkee Hostility Inventory (Buss & Durkee, 1957). Whilst these measures provide valuable information relating to how the service-user views their own behaviour, they are not entirely suitable for individuals with acquired brain injury, where problems such as cognitive impairment and self-awareness deficits are often present (Hart, Seignourel, & Sherer, 2009; Ownsworth et al., 2007; Sherer et al., 2003).

An accurate system of measurement should provide reliable data relating to the nature of aggressive behaviours, from which clinical decisions can be made with confidence. This is especially important for services focussing on challenging behaviours, where the main priority for behavioural interventions is often to address violent and aggressive behaviours (Treadwell & Page, 1996).

The Overt Aggression Scale (Yudofsky, Silver, Jackson, Endicott, & Williams, 1986) was designed as an objective rating of aggressive behaviours in a general psychiatric population. This scale divides sixteen separate incidents of aggression into four categories: verbal aggression, physical aggression against objects, physical aggression against self and physical aggression against others. In addition to this, the scale allows for the recording of eleven specific interventions related to each aggressive event. Alderman and his colleagues developed a modified version of the Overt Aggression Scale (OAS-MNR), designed to enhance the original scale by coding antecedents and reflecting in more detail the range of interventions used in neurorehabilitation services (Alderman et al., 1997). Unfortunately, however, clinical experience has indicated that the complexity of this scale results in poor inter-rater reliability among direct care staff.

The primary aim of study 1 was the development of a system of measurement specific to aggression after ABI, for use by staff providing direct care or treatment to individuals with ABI. As inadequate staff training appears to be a significant source of inaccurate measurement, the scale was designed to be conceptually straightforward and placing as few demands on memory and training. As such, the BIRT Aggression Rating Scale (BARS) utilises commonly held cultural constructs related to aggression and violence that are reflected in western systems of jurisprudence.

One premise of the BARS is that an ordinal scale of measurement may allow for better assessment of overall aggressive patterns than numerous discrete categorical

measures. The scale utilises an ordinal rating scale to categorise verbal and physical aggression at three levels of intensity. The ordinal aspect of the scale was designed to parallel three broad legal categories: socially undesirable but legal, misdemeanour and felony. These categories were designed to increase in intensity in line with the severity of aggression. This scaling upon common legal conventions was developed from a practical and pragmatic perspective in order to allow greater ease of staff training and hence greater reliability. The categories were not derived as theoretical divisions of behaviour based upon neurobiological constructs but rather as a pragmatic and more readily communicated set of constructs that allow for easier staff training and ease of communication among professionals.

An accurate scale in itself is not sufficient to ensure reliable data from which clinical decisions can be made. It is also essential to develop an effective training package to inform staff members about the rationale behind the measures, as well as how to use them accurately (Kobak, Lipsitz, & Feiger, 2003). The BARS was therefore designed for use by staff (both professional and non-professional) who have undergone a standardised training programme provided using video-based training and a brief video-vignette assessment protocol.

To date the scale has been used in three levels of residential post-acute neurobehavioural rehabilitation: supported community living schemes; community reintegration rehabilitation facilities and units designed specifically for challenging behaviours. Use of the BARS with individual cases has allowed for more practical and useful communication of behaviours to oversight authorities at clinical reviews. It

has not been trialled with outpatients and would require trained family or other carers to use the ratings in order to be practical in such a setting.

The BARS has been designed to provide high levels of reliability when measuring irritability and aggression in persons with acquired brain injury or illness. Such aggression is typically unplanned and impulsive in nature (Barratt & Slaughter, 1998; Barratt, Stanford, Kent, & Felthous, 1997). The scale was not intended for use with persons who engage in gratuitous, instrumental violence that has been described as psychopathic in nature (Baron & Richardson, 1997; Blair, 2001). It was also not intended for use with persons who are patently confused and disoriented, whose aggressive behaviour is best characterised as agitated. Such behaviour may be more usefully measured with the Agitated Behavior Scale developed by Corrigan and his associates (Corrigan & Bogner, 1995).

In order for the BARS to be considered an effective tool for measuring aggressive behaviours and informing clinical decisions, it must be shown to possess adequate psychometric properties. This aim was approached in two ways. Firstly, the BARS and a computerised training programme for staff are described in detail; inter-rater reliability was hypothesised to be adequate for a sample of rehabilitation staff after completion of this training. Secondly, it was anticipated that the BARS data on a sample of participants with ABI would show high concurrent validity with items in clinician-rated outcome measures (particularly those items specifically addressing aggressive behaviour) consistent with previously reported correlational studies on aggression after brain injury.

## **4.2. Part 1: Establishing inter-rater reliability**

### **4.2.1. Method**

#### **4.2.1.1. Participants**

A total of 106 volunteer participants (74 females and 32 males) were recruited from the staff of five neurorehabilitation units in England that provide neurobehavioural rehabilitation for persons who have sustained an acquired brain injury. Staff members from a range of educational and professional backgrounds were included in the study, including support workers and specialised therapy staff. Length of employment within the organisation ranged from less than one month to 11 years with a median of 12 months (positively skewed). Most were full-time employees. The range of vocational backgrounds of participants varied and included psychology, psychiatry, nursing and allied health. Nineteen (17.9%) held no relevant academic or vocational experience related to medicine or rehabilitation. Forty-one (38.7%) had experience with the BARS prior to the computer-based training protocol while the remaining 65 participants were naïve to the rating scale.

#### **4.2.1.2. Materials**

The BARS scale is divided into two categories; verbal and physical aggression. These two categories are then sub-divided into three levels of severity:

- **V1 – Non-directed verbal aggression;**
- **V2 – Directed verbal aggression;**

- **V3** – Direct verbal threats of harm;
- **P1** – Non-destructive physical aggression;
- **P2** – Destructive physical aggression directed towards objects; and
- **P3** – Physical aggression directed towards oneself or another.

The term “directed” in the verbal area refers to abuse uttered directly to another person. The term “non-destructive” in the physical area has been operationally defined so as to include destruction of an object which is less than one British pound in value. The ordinal aspect of the scale (1,2,3) was designed to match generally to three broad legal categories: (i) an incident which is not chargeable, (ii) a minor crime (or misdemeanour), (iii) a more serious offence, similar in severity to a felony.

Using these more culturally normative categories of aggression based upon possible legal consequences is intended to allow easier recollection by staff and faster training. The categories of V1 and P1 cover behaviours that are socially inappropriate but not generally treated as illegal acts; V2 and P2 are behaviours which could be construed as minor crimes or misdemeanours, and V3 and P3 are behaviours which could result in a more serious chargeable crime, analogous to a felony. Strictly speaking, harm to oneself is not typically treated as a crime, but this anomaly has not appeared to dilute the utility of the scale. In most instances the scale is used within rehabilitation units and supported houses as part of a general recording procedure that utilises antecedent/behaviour/consequence records. The training was delivered using a computerised automated slide presentation with embedded video presentations and full narration. The training was provided

individually or in small groups. A staff member familiar with the scale was available during all training sessions.

The training programme was developed with several aims. The first aim was to emphasise the purpose and benefits of recording behaviours. Secondly there was a section that emphasised recording measurable, observable behaviours rather than interpretations of behaviour. Thirdly the rationale of the scale was described. Each category was described in detail accompanied by a videotaped vignette in which amateur and professional actors re-enacted recorded clinical behaviours. The training gave directions in the following areas: how to objectively rate each level of aggressive behaviour with narration and supporting text, differentiation between behaviour associated with frustration (which is not rateable on the BARS) and characteristics of aggressive behaviour sufficient to be rated on the scale. Several vignettes illustrated this distinction. The object of the training module was a *turnkey* programme, which could be presented without preparation using ordinary computer equipment widely available within the rehabilitation facilities. The training was 18 minutes in duration. All participants were given the opportunity to ask questions or receive further information from individuals already trained in the BARS.

In developing the training protocols, a total of 51 videotaped vignettes were recorded using both staff volunteers and professional actors. Each vignette was a re-enactment of aggressive behaviours that had been observed within the rehabilitation settings. Five experts (three psychologists and two assistant psychologists) rated the vignettes for the prototypicality of the re-enacted aggressive episodes. There was no variation in ratings among the experts.

From the highest-ranking vignettes, eight were used in the training programme, ten were used in post-test one and ten were used in post-test two. The current study only examines post-test one. In order to ensure realism of the prototypical vignettes, many of the exemplars of physical aggression also presented concomitant verbal aggression. Participants were asked to mark all rateable behaviours in each video-vignette, which again is analogous to typical behaviour patterns found in neurobehavioural rehabilitation units.

The response sheet allowed participants to select one or more of the six categorised forms of aggression, as well as an option of “none” should they consider the behaviour observed to be not rateable on the BARS. The post-test consisted of two vignettes for each category of physical aggression, three for directed verbal aggression (V2 or V3), one for non-directed verbal aggression (V1) and one example with no rateable behaviours. Scores were compared to the agreed expert ratings of the vignettes. Each vignette had one specific target behaviour to rate, even if several other behaviours occurred concomitantly and scoring for each vignette related to the rating of that specific behaviour. An alternate form of the assessment was developed, but was not analysed for the purposes of this study.

#### **4.2.1.3. Procedure**

Participants were all shown the eighteen-minute training presentation describing the BARS system of measurement. This was done either individually or within small groups with a trained facilitator. The participants then viewed and rated each video-

vignette using the BARS on the standardised written response sheet. The vignettes were presented only once on the post-test and were not replayed as this would not have constituted a fair analogue of rating actual behaviours within a neurorehabilitation unit. The post-test required approximately ten minutes per participant. Within a group situation, participants were requested not to discuss the scenes amongst themselves prior to the completion of the study period.

#### **4.2.1.4. Statistical analysis**

Inter-rater reliability of the BARS was assessed using a random effects model of intra-class correlation (ICC), based on the assumption that the ten prototypical video vignettes represented a random selection from the population of possible aggressive incidents and that the participants represented a random selection of potential staff that might utilise the scale. A generalised kappa value is considered applicable when there are more than two observers measuring the same event (Fleiss, 1971; Shrout & Fleiss, 1979).

#### **4.2.2. Results**

Eighty percent of participants achieved a score of 70% correct or greater on the post-test. Tests for differential performance based upon the participant's demographics did not yield significant differences; *p*-values for individual items on the post-test ranged from 0.48 to 0.92. The average ICC of raters was highly significant ( $ICC = .92$ ,  $F_{(9,945)} = 11.71$ ,  $p < .001$ ), indicating that a high degree of

agreement was achieved between the participant raters. The 95% confidence interval for the ICC ranged from .82 to .97.

### **4.3. Part 2: Investigating concurrent validity**

#### **4.3.1. Method**

Organisational outcome data were analysed in order to investigate the concurrent validity of the BARS. Selection criteria included all service users who had completed at least 2 months of rehabilitation prior to discharge from 12 residential rehabilitation programmes during the years 2007 to 2010 inclusive.

##### **4.3.1.1. Participants**

Three hundred and nine service users met the selection criteria. Males were more prevalent in the sample ( $n = 218$ , 71%). Age on admission ranged from 17 to 74 years (mean 42.0 years, SD 14.5 years) and followed a relatively normal distribution. Length of stay ranged from 2.0 to 131.0 months (median 6.7 months) and chronicity (time between injury and admission) ranged from 0.8 to 410.0 months (median 9.8 months). Both length of stay and chronicity were heavily positively skewed.

Diagnosis information was available on 299 of the participants (97%): over half of these (57%) had suffered traumatic brain injury (TBI), while other diagnoses consisted of cerebro-vascular accident (22%), neoplasm (11%), cerebral infection (8%) and cerebral anoxia (2%). Data on severity of injury were not available,

however the nature of the residential programmes was such that a higher representation of severe injuries was expected. A recent survey of similar residential brain injury rehabilitation programmes (Glenn, Rotman, Goldstein, & Selleck, 2005) found a consistently high proportion of severe injuries amongst those service users with TBI.

#### **4.3.1.2. Materials**

BARS data on each participant were collected in the following manner. An overall index score of irritability and aggression was developed in order to efficiently summarise BARS data into one variable. A weighting algorithm was utilised in order to address both frequency and severity of each behavioural recording with physical items double-weighted to represent more serious incidents than verbal aggression. The resulting irritability/aggression (I/A) index was therefore calculated by the following equation:

$$I/A = \text{sumV1} + 2*(\text{sumV2}) + 3*(\text{sumV3}) + 2*(\text{sumP1}) + 4*(\text{sumP2}) + 6*(\text{sumP3}) \quad (1)$$

The highest weekly BARS I/A score recorded during the first 4 weeks of admission was selected for further analysis. Data for each participant were also available on the Neurobehavioural Rating Scale (Levin et al., 1987), which consists of 27 items covering a wide range of symptoms, each of which is rated on a 7-point scale, and the Supervision Rating Scale (Boake, 1996), which is a single 13-point scale

reflecting the level of supervision required by the participant. Rehabilitation clinicians rated both scales during the first four weeks of admission.

#### **4.3.1.3. Statistical analysis**

The heavily skewed variables of length of stay and chronicity were subjected to log normal transformation so that parametric analyses could be performed using these as dependent variables. The BARS I/A index variable had a high proportion (53%) of zero scores. Such variables (also described as zero-clustered) pose a difficulty for further statistical analyses, however various solutions have been published, particularly when the dependent variable is zero-clustered (Chang & Pocock, 2000; Lachenbruch, 1992, 2001a, 2001b, 2002). Huson (2007) ran simulation studies to show that regular parametric correlations were preferable in most instances, although the proportion of zeroes in those studies did not exceed 30%. In the present study, however, it was decided to utilise Spearman non-parametric correlations as the NRS individual item and SRS data were ordinal by nature.

Concurrent validity was therefore investigated with non-parametric correlations between the BARS I/A index score and the 28 items (including total score) from the Neurobehavioural Rating Scale (NRS), as well as the Supervision Rating Scale (SRS), both rated at admission. The relationships that the BARS I/A index admission score had with other demographic (sex, age at admission) and injury-related (diagnosis chronicity) variables were also explored.

### 4.3.2. Results

The BARS weighted index score on admission was significantly correlated with the total NRS score on admission ( $r_s = .15, p < .01$ ), with higher aggression scores being associated with greater neurobehavioural dysfunction. The magnitude of this correlation represents a small effect size (Cohen, 1988). Table 4.1 shows the Spearman correlations for each of the 27 individual items on the NRS with the BARS I/A admission score.

**Table 4.1 Spearman correlations between BARS I/A index and individual Neurobehavioural Rating Scale Items**

Inattention / reduced alertness	.03	Memory deficit	.09	Motor retardation	.38**
Somatic concern	-.01	Agitation	.23**	Unusual thought content	.46**
Disorientation	.05	Inaccurate insight and self-appraisal	.14*	Blunted affect	.31**
Anxiety	.33**	Depressive mood	.15**	Excitement	.01
Expressive deficit	.32**	Hostility / uncooperativeness	.09	Poor planning	.04
Emotional withdrawal	.06	Initiative / motivation	.11*	Labile mood	.16**
Conceptual disorganisation	.07	Suspiciousness	.04	Tension	.16**
Disinhibition	.06	Fatigability	.03	Comprehension	.13*
Guilt feelings	.08	Hallucinatory behaviour	.41**	Speech articulation	.04

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

A number of medium size (between .30 and .50) correlations were observed: “unusual thought content” ( $r_s = .46, p < .01$ ), “hallucinatory behaviour” ( $r_s = .41, p < .01$ ), “motor retardation” ( $r_s = .38, p < .01$ ), “anxiety” ( $r_s = .33, p < .01$ ), “expressive deficit” ( $r_s = .32, p < .01$ ) and “blunted affect” ( $r_s = .31, p < .01$ ). Of the two NRS items most similar in nature to explicitly aggressive behaviour, “agitation” ( $r_s = .23, p < .01$ ) showed a small but significant correlation while “hostility/uncooperativeness” ( $r_s = .09, p = \text{n.s.}$ ) showed no significant association.

The BARS I/A index admission score was also significantly correlated with the Supervision Rating Scale on admission ( $r_s = .22$ ,  $p < .01$ ), again showing a small effect, in which those displaying higher levels of aggression on admission were likely to require a higher degree of external supervision. Neither participant age ( $r = .09$ ,  $p = \text{n.s.}$ ) nor log-transformed chronicity ( $r = .06$ ,  $p = \text{n.s.}$ ) showed a significant parametric association. The relationship between sex of participant and BARS I/A index score on admission was analysed via a two-part model, developed to compare two independent groups with zero-clustered data (Lachenbruch, 1992, 2001a, 2001b, 2002), which produces a  $\chi^2$  statistic with 2 degrees of freedom. Such an analysis revealed no significant relationship ( $\chi^2 = 1.19$ ,  $p = \text{n.s.}$ ).

#### **4.3.3. Discussion**

This chapter presented two studies regarding the psychometric properties of the BIRT Aggression Rating Scale (BARS). The BARS has been developed in order to provide a method of objectively measuring aggression within a brain injury rehabilitation context and to facilitate staff training and understanding of behavioural interventions. Firstly, the BARS and a staff training programme were described and a study performed which established adequate inter-rater reliability of the measure. Secondly, a separate study was conducted into the concurrent validity of the BARS by exploring its relationship with previously published scales.

The inter-rater reliability of the BARS was calculated as high in the first study. A computerised training programme with videotaped vignettes was utilised with 106 rehabilitation staff across five neurobehavioural rehabilitation units in the United

Kingdom. The reliability of the scale was assessed using an intraclass correlation analysis, with an intraclass correlation average for all raters of .92. The results of this analysis indicate that the BARS is a reliable means of categorising and grading aggressive behaviours for persons with acquired brain injury. Following a training programme, it is possible for individuals working within a neurobehavioural rehabilitation facility to reliably identify and score into one of six categories the aggressive behaviours typically displayed by service users. One of the objectives of the BARS package is that a relatively short training programme would lead to reliable use of the scale, due to its internal logic and its relation to standard social constructs.

The second study was designed to address the concurrent validity of the BARS with a sample of 309 participants with acquired brain injury. The highest weighted BARS index during the first four weeks of admission was used for comparison with items from the Neurobehavioural Rating Scale (Levin et al., 1987) and the Supervision Rating Scale (Boake, 1996), two previously published and widely used scales within the field of brain injury rehabilitation. Treating clinicians rated both of these scales during the first four weeks of admission for each participant.

There were significant but small associations between the BARS index and both the total NRS score and the SRS, which are consistent with previously reported relationships between measured aggression and poorer functional status following brain injury (Alderman, 2007; Alderman et al., 2002). Comparisons with individual items on the NRS revealed a number of larger (but still “medium”) size associations with the BARS index. There are two NRS items that would appear to relate to

aggression per se. “Agitation” showed a small relationship with the BARS index, but “hostility/uncooperativeness” did not.

NRS items reflecting typically psychiatric symptoms, such as unusual thoughts or hallucinations, produced the largest correlations with higher aggression scores. Such complications are considered rare following brain injury (McAllister & Ferrell, 2002), however, links between visual hallucinations and aggression have been previously reported following frontal lobe damage (Fornazzari, Farcnik, Smith, Heasman, & Ichise, 1992).

Poorer communication skills following brain injury have been previously related to aggression (Alderman, 2007; Alderman et al., 2002) and this relationship was also observed in the present validation study. This was particularly the case for the NRS item “expressive deficit” (medium size correlation) but also for “comprehension” (small size). No association was found for “speech articulation defect”.

Depression following brain injury has been reported to predict aggression in some studies (Baguley et al., 2006; Johansson et al., 2008; Rao et al., 2009; Tateno et al., 2003) but not others (Alderman, 2007; Paradiso et al., 1996; Wood & Liossi, 2006b). In the current study, “depressive mood” on the NRS showed a small but significant association with higher BARS index scores. “Labile mood” also showed a small but significant correlation with higher aggression, consistent with the finding that emotional lability and poor frustration tolerance has been linked with aggression in adolescents with brain injury (Dooley et al., 2008). Increased anxiety on the NRS showed a medium sized relationship with higher levels of BARS index aggression.

A small but significant association was also observed between higher BARS index scores and an NRS item that reflects poorer self-awareness – “inaccurate insight and self-appraisal”. Impaired insight or self-awareness is a common problem after severe brain injury (Hart et al., 2009; Sherer et al., 2003) and may contain different subtypes (Ownsworth et al., 2007). Further research may be warranted investigating the role of disorders of self-awareness in aggression following brain injury.

With a reliability coefficient of 0.92 and evidence of concurrent validity that is consistent with previous research, the BARS is demonstrably an appropriate tool for measuring aggressive behaviours within a neurorehabilitation setting in persons with acquired neurological impairments. The integration of training protocols with rating scales increases the likelihood of consistency when used outside of the research context. Rehabilitation services which focus on challenging behaviours invariably prioritise behavioural interventions designed to address aggression and irritability. The accurate capturing of behavioural data is essential to monitoring interventions, be they psychopharmacologic or behavioural. The BARS is specifically designed to provide a reliable and valid measure of aggression for persons with acquired neurological impairment.

# **CHAPTER 5. STUDY 2: THE NEUROCOGNITIVE CORRELATES OF VERBAL AGGRESSION, PHYSICAL AGGRESSION AND INAPPROPRIATE SEXUAL BEHAVIOUR**

## **5.1. Introduction**

Disorders of interpersonal behaviour, of which the more serious include aggression and inappropriate sexual behaviour, are relatively common consequences of severe acquired brain injury (Baguley et al., 2006; Johnson & Balleny, 1996; Kelly, Brown, Todd, & Kremer, 2008; McKinlay et al., 1981). These behaviours are often socially disabling or isolating (Wood & Worthington, 2001) and may require intensive residential treatment programmes, which are typically expensive services to provide. It is important, therefore, to have a solid clinical research base so as to understand these behaviours and develop effective treatments.

There is a growing literature on the clinical correlates of aggression after severe acquired brain injury. The findings have been mixed, which may be largely the result of differences in how aggressive behaviours are measured. The gold standard must be to have trained professional staff using a psychometrically reliable and valid tool to record aggressive behaviours as they occur, for instance, within a residential treatment programme or a hospital environment. Some group studies exploring potential relationships between post-injury aggression and neurocognitive status after brain injury have achieved this (Alderman, 2007; Alderman et al., 2002; Tateno

et al., 2003). Others, though, have relied on less direct measures such as semi-structured interviews, typically getting their information from relatives of the injured person (Greve et al., 2001; Kelly et al., 2008; Wood & Liossi, 2006b) or simply by reports from ward staff (Kerr, Oram, Tinson, & Shum, 2011). Although such methods can be useful, it is problematic to rely entirely on retrospective reports by relatives and others who may be unfamiliar with disordered behaviours and inexperienced in describing and evaluating them. Instead, systematic and careful assessment of behaviours is required in order to overcome the biases and distortions inherent in normal human judgement (Kazdin, 2001). Despite the practical difficulties, it is also clearly preferable for measurements of disordered behaviour to be made objectively and systematically as soon as possible following the event.

As well as using different techniques to measure behavioural disturbances, neurocognitive function has also been quantified in various ways. These have included clinician ratings on established scales (Alderman, 2007; Alderman et al., 1997) as well as performances on standardised assessment tools such as the Mini-Mental Status Examination (Folstein, Folstein, & McHugh, 1975), used in some studies (Tateno et al., 2003; Visscher, van Meijel, Stolker, Wiersma, & Nijman, 2011), individual neuropsychological test results (Greve et al., 2001; Wood & Liossi, 2006b) or clinician rating of impairments based on neuropsychological test results (Simpson et al., 2001).

Where significant associations between post-injury aggression and predictor variables have been reported, these have been modest in size. Premorbid psychosocial variables have been found to be associated with post-injury

aggression, such as a history of premorbid violence (Greve et al., 2001; Kerr et al., 2011; Tateno et al., 2003), prior drug and alcohol misuse (Tateno et al., 2003), male sex and lower socio-economic status (Kerr et al., 2011; Wood & Liossi, 2006b). One study (Visscher et al., 2011) found no association with sex or prior drug and alcohol misuse. No previous study has combined systematic measurement of aggression as it occurs with comprehensive neuropsychological data. The current data set achieves this.

A number of post-injury clinical variables have also been reported to be associated with post-injury aggression. These have included the presence of frontal lobe lesions (Brower & Price, 2001; Chan et al., 2006; Paradiso et al., 1996; Pardini et al., 2011; Siever, 2008; Tateno et al., 2003), poorer neurocognitive function (Alderman, 2007; Alderman et al., 2002; Wood & Liossi, 2006b) and greater functional impairment (Alderman et al., 2002; Kerr et al., 2011). Two studies (Alderman, 2007; Wood & Liossi, 2006b) reported no relationship between post-injury aggression and current emotional status, although the latter found a link with major depression. The reported relationship between aggression and neurocognitive status in group studies has ranged from none (Greve et al., 2001; Tateno et al., 2003; Visscher et al., 2011) to poor verbal memory and visuo-spatial function (Wood & Liossi, 2006b). Significant associations with impaired language function have also been reported (Alderman, 2007; Alderman et al., 2002). Weaker verbal ability with a history of prior brain injury has been associated with aggression towards an intimate partner in other areas of psychological research (Walling, Meehan, Marshall, Holtzworth-Munroe, & Taft, 2012).

While there have been a number of published studies addressing the clinical correlates of post-injury aggression, there has been much less focus on sexually inappropriate behaviour. Researchers have predominantly focused on the prevalence of prior brain injury amongst groups of adults who have committed sexual offences (DelBello et al., 1999; Langevin, 2006) or individual case reports of inappropriate sexual behaviour following neurological injury/illness (Cao et al., 2010; Emory et al., 1995; Fyffe et al., 2004; Kelly & Simpson, 2011; Miller et al., 1986; Poletti, Lucetti, & Bonuccelli, 2010; Woods et al., 1998). There has been only one study (Simpson et al., 1999) describing the characteristics of inappropriate sexual behaviour (somewhat misleadingly termed “sexual offending”) amongst a large cohort of people with traumatic brain injury. Their procedure involved a retrospective case screening by staff, rather than planned structured recording of behaviours as they occur. A follow up controlled comparison of participants who exhibited these behaviours reported no significant clinical correlates among premorbid psychosocial or post-injury clinical variables (Simpson et al., 2001). A recent review paper (Johnson et al., 2006) has recommended that continuous assessment of sexually inappropriate behaviours with structured measurement tools is desirable. The same group of researchers subsequently developed a standardised system for continuously recording challenging sexual behaviour (Knight et al., 2008).

There have been few reported studies addressing both aggression and inappropriate sexual behaviour following brain injury. The relationship between these maladaptive behaviours therefore remains largely unexplored. The first aim of this study, then, is to fill this gap. Clinical data were obtained during a nine week residential assessment on individuals with acquired brain injury admitted over a six year period in a post-

acute brain injury rehabilitation programme. In this setting, interpersonal behaviours are systematically monitored as part of a holistic neurobehavioural assessment. The presence of premeditated aggression or severely sexually aberrant behaviour would have precluded admission or, if the person had already been admitted, would likely have necessitated a transfer into a specialist facility designed to manage more challenging behaviours.

The key question was whether the range of aggressive and inappropriate sexual behaviours is better understood as both reflecting a single, underlying behavioural disinhibition syndrome arising from impaired self-regulation (Wood, 2001) or as separate clinical phenomena. In order to address this empirically, multivariate statistical modelling of systematic and simultaneous recordings of aggressive and sexually inappropriate behaviour was employed. As there have been a number of significant associations reported between aggression and predictor variables, with none for inappropriate sexual behaviour, it was predicted that the analysis would support the notion of separate phenomena requiring individual analyses. It was less clear, however, whether further subdivisions within each behavioural category (such as verbal comments versus physical actions, non-directed versus directed aggression) would be borne out.

Following clarification of the above issue, the second aim of this study was to investigate the predictive nature of various clinical variables for each category of behavioural disturbance. One important question concerns the relationship between observed behaviour and neurocognitive function, as measured by performance on standardised neuropsychological tests. At one extreme, single case studies have

been reported that demonstrate striking dissociations between neurocognitive status and behavioural disturbance (Eslinger & Damasio, 1985; Wood & Rutherford, 2004). These single case studies show that measurable neurocognitive deficits are not inevitable correlates of disturbed social behaviour. Group studies, however, are required to investigate whether such dissociations are typical following brain-injury, or whether they represent an atypical (albeit theoretically important) pattern. Distinctions have been made between social disinhibition arising from impaired regulatory control of behaviour (Wood, 2001) and a cognitive dysexecutive syndrome (Baddeley, 1986). Separate underlying frontal-subcortical neuroanatomical circuits for a disinhibition syndrome (Starkstein & Kremer, 2001) and cognitive deficits (Salmon, Heindel, & Hamilton, 2001) have been proposed (for a review, see Saint-Cyr et al., 2002).

As the key question is in clinical patterns of disturbed behaviour and their relation to neurocognitive status, a group of participants with brain injury were studied who had been referred to a neurobehavioural rehabilitation centre and were therefore likely to present with behavioural difficulties. It is clear that these participants do not constitute a representative sample of all people with brain injuries. Likewise, the aetiologies underlying their brain lesions are diverse – they were not selected on any neuropathological basis other than they did not have progressive neurological conditions. For the purpose of investigating clinical patterns, it was considered valuable to have a range of acquired injuries and a substantial number of participants with behavioural disturbances. Although the potential associations between these phenomena and the underlying neuropathology are also worth

exploring, the present study was primarily concerned with the relationships between aggressive and inappropriate sexual behaviours.

The specific research questions were therefore as follows. Firstly, it was anticipated that an exploratory factor analytic approach would differentiate between observed aggressive and inappropriate sexual behaviours in the first such study addressing both. Secondly, it was expected to replicate previously reported associations in separate multivariate statistical modelling of aggressive behaviours – specifically poorer language and memory functioning, lower educational achievement, prior history of substance misuse and history of aggression – and a lack of similar associations for inappropriate sexual behaviour.

## **5.2. Method**

### **5.2.1. Participants**

Clinical records were scrutinised for all admissions to a post-acute residential neurobehavioural brain injury rehabilitation programme during the six year period: 2004 to 2009. During this time a total of 174 admissions occurred. However, 22 of these did not complete the initial nine week assessment period due to a variety of reasons. Fifteen service users self-discharged prematurely. Two admissions were readmissions during the sample time period and the participant was therefore already included in the data set. Two others were discharged due to serious transgressions with substance misuse, two were atypical admissions (day only or respite) and a further two were taken to hospital with acute medical issues and did

not return. Unfortunately, one participant died unexpectedly during their assessment period.

#### **5.2.1.1. Demographics of sample**

A total of 152 admitted participants therefore completed an initial nine week residential period, in which continuous and contemporaneous behavioural recordings were made according to standardised protocols. There were 114 (75%) males and 38 (25%) females in the sample. The majority identified themselves as premorbidly right-handed (136, 89%) with 15 (10%) left-handed and one participant reported mixed handedness. The continuous variables of age on admission (range 16 to 72 years, median of 39 years), chronicity of injury (ranging 2 to 468 months with median of 12 months) and years of education (ranging 8 to 15 with median 10) were all non-normally distributed.

#### **5.2.1.2. Details on brain injuries and illnesses**

The majority of diagnoses were traumatic brain injury (101, 66% of total) for which road traffic accidents (54, 53% of TBI) were the most common cause of injury, followed by falls (28, 28% of TBI) and assaults (15, 15% of TBI). There were three (3% of TBI) combat-related injuries (two shrapnel/penetrating and one blast/concussion) and one (1% of TBI) workplace injury.

There were 51 (34% of total) participants whose injuries or illnesses were non-traumatic in nature. Cerebro-vascular accidents (CVA) accounted for 24 (16% of

total), of which 16 (67% of CVA) were haemorrhagic in nature, 7 (29% of CVA) occlusive and one (4% of CVA) radiation-induced vasculitis. Cerebral anoxia was the mechanism of injury for 14 participants (9% of total), with exactly half of these sustained following cardiac arrest, three (21% of anoxic) following drug overdose, two (14% of anoxic) subsequent to hypoglycaemic coma and one (7% of anoxic) sustained in an attempted hanging. The 13 remaining participants (9% of total) had “other” diagnoses of cerebral tumour, encephalitis (each with 4, 31% of “other”), Wernicke’s encephalopathy (3, 23% of “other”) and one (8% of “other”) each following toxic solvent abuse and acute pontine myelinolysis.

Severity of injury was rated for those 101 participants with TBI. Previous clinical research (Braunling-McMorrow, Dollinger, Gould, Neumann, & Heiligenthal, 2010; Glenn et al., 2005) has indicated a consistently high proportion of severe traumatic injuries in similar post-acute brain injury rehabilitation programmes. Data were available for the lowest recorded Glasgow Coma Scale (GCS; Teasdale & Jennett, 1974) prior to sedation for 60 participants and on duration of post-traumatic amnesia (PTA) for 70 participants. Severity ratings are presented in Table 5.1, in which it can be seen that 78.3% of available GCS scores fell in the severe category and 75.7% of available PTA durations were coded as extremely severe, the most severe category for each scale.

**Table 5.1 Severity of injury for those participants with TBI**

Glasgow Coma Scale ( <i>n</i> = 60)		Post-traumatic amnesia ( <i>n</i> = 70)	
Minimum	3	Minimum	1 day
Maximum	14	Maximum	420 days
Severe (GCS ≤ 8)	47	Extremely severe (PTA > 28 days)	53
Moderate (GCS 9-12)	6	Very severe (PTA 7-28 days)	15
Mild (GCS ≥ 13)	7	Severe (PTA 1-7 days)	2
		Moderate (PTA 1-24 hours)	0
		Mild (PTA < 60 minutes)	0

### **5.2.2. Design and materials**

Archived clinical files were searched for data falling in four broad categories: demographic, injury-related, neurocognitive and behavioural. These are discussed in turn.

#### **5.2.2.1. Demographics and premorbid information**

This information is routinely gathered in the service and was collected from all participant clinical files. Continuous variables were created for age at admission (years), age at injury (years), chronicity (months since injury) and education (years of formal schooling). Dichotomous variables were coded for sex, history of prior brain injury requiring hospitalisation, psychiatric history (formal diagnosis and/or treatment), forensic history of aggression, forensic history of sexual offences, drug/alcohol history (which interfered with social functioning) and handedness.

### **5.2.2.2. Injury-related information**

Firstly, the type of acquired brain injury was coded: traumatic brain injury (TBI), cerebrovascular accident (CVA), cerebral anoxia (CA) and other. For any cases of TBI, information on standard measures of severity of injury were also collated – lowest Glasgow Coma Scale (GCS) score prior to sedation and duration of post-traumatic amnesia (PTA) in days. Dichotomous variables were coded for intracranial abnormality on reported neuroimaging (CT or MRI), the requirement for neurosurgical intervention and whether the participant was prescribed psychotropic medication at any point during their residential assessment period. To address the potential influence of any ongoing litigation, participants were also coded for medicolegal status (none, settled or ongoing).

### **5.2.2.3. Neurocognitive data**

Routine neuropsychological evaluation during the assessment period was available for 98 (64%) of the 152 participants. The lack of evaluations for all participants was due to a variety of reasons, with some participants being considered too severely cognitively impaired ( $n = 17$ ) or language-impaired ( $n = 11$ ) for formal testing, others having been recently assessed prior to admission ( $n = 10$ ), ongoing civil litigation assessments taking priority ( $n = 10$ ), test results unable to be located ( $n = 3$ ), physical impairments preventing sufficient formal testing ( $n = 2$ ) and one participant could not be formally assessed due to not being fluent in English premorbidly.

For the 98 participants with a neuropsychological evaluation, it was decided to utilise only those for whom a full six factor structure (Tulsky, 2003; Tulsky & Price, 2003) of the Wechsler Adult Intelligence Scale – Third Edition (Wechsler, 1997a) and the Wechsler Memory Scale – Third Edition (Wechsler, 1997b) could be calculated. The six factors are Verbal Comprehension Index (VCI), Perceptual Organisation Index (POI), Processing Speed Index, Working Memory Index (WMI), Auditory Memory (AM) and Visual Memory (VM). Using this criterion, seventy-seven (51%) of the participants had full neurocognitive data sets.

#### **5.2.2.4. Behavioural data**

Contemporaneous behavioural recordings were made by staff on all service users as part of routine monitoring and analysis of social behaviours. All staff had previously received formal training in behavioural documentation and psychology staff later reviewed the accuracy of codings.

Aggressive behaviour was coded according to the BIRT Aggression Rating Scale (BARS), which is implemented in all of the organisation's residential units around the country and has demonstrated good inter-rater reliability and validity (see previous chapter; Freeland, Corker, Heritage, & James, 2012). The BARS codes aggression into 6 categories reflecting the nature (verbal or physical) and severity (1, 2 or 3) of each episode. Incidents of verbal aggression are scored as V1 for non-directed, V2 for directed at another person or V3 for verbal threats. Similarly, incidents of physical aggression are scored as P1 for non-directed, P2 for damage to property and P3 for violence towards another person or one's self.

Sexually inappropriate behaviour was also coded using another standardised system that is utilised organisation-wide. Incidents of this nature were rated as either S1 for spoken comments (for example, if a client asked a staff member to participate in sexual activity or asked for personal information of a sexual nature) or S2 for inappropriate action (such as attempting to touch a staff member in a sexual manner). Other incidents of overfamiliar or disinhibited behaviour without a sexual component were not included in this analysis.

Typically, a participant's behaviour was observed for the nine weeks of assessment in order to obtain a baseline from which later clinical decisions could be taken. In most cases, no specific intervention was applied during that time. For each participant, total frequencies of recorded incidents across the assessment period were therefore obtained for each of these eight raw behavioural variables (V1, V2, V3, P1, P2, P3, S1, S2). Data recorded in the first week were not included as this was often only a partial week depending on the day of admission.

#### **5.2.2.5. Statistical analyses**

The statistical analyses were conducted using PASW version 18.0. The nature of the behavioural observation data, which were to be utilised as dependent variables, constrained subsequent analyses in several ways. The data contained many zero counts (in which no observations for that particular behaviour were made) and the remaining non-zero values are heavily skewed and leptokurtic. Table 5.2 shows the descriptive statistics for the 8 behavioural variables described above, including the

proportion of zeroes and standardised scores for skewness and kurtosis. It can be seen that the proportion of zeroes was considerable for all categories of behavioural observation and varied from around 47% (V1s) to a little over 90% (P2s).

**Table 5.2 Distributions of behavioural data**

	<b>Min</b>	<b>Max</b>	<b>Mean</b>	<b>SD</b>	<b>Z<sub>skewness</sub></b>	<b>Z<sub>kurtosis</sub></b>	<b>% zero</b>
<b>V1</b>	0	241	5.24	21.55	47.51	250.69	46.71
<b>V2</b>	0	86	4.30	12.36	23.72	60.81	57.24
<b>V3</b>	0	33	0.91	3.34	35.53	152.88	78.29
<b>P1</b>	0	49	1.15	4.35	46.78	251.60	69.74
<b>P2</b>	0	8	0.17	0.75	40.69	201.15	90.13
<b>P3</b>	0	17	0.68	2.11	24.99	74.63	79.61
<b>S1</b>	0	33	1.63	4.24	21.52	58.95	73.68
<b>S2</b>	0	325	3.05	26.43	61.62	380.46	77.63

### **5.2.2.6. Dealing with excess zeroes**

Datasets with “excess zeroes” have been noted as problematic for decades (Lachenbruch, 2002). These data have also been characterised as “zero-clustered” (Huson, 2007) or with “clumping at zero” (Chang & Pocock, 2000) and have been repeatedly found in the medical/healthcare literature (Chang & Pocock, 2000; Delucchi & Bostrom, 2004; Schneider, Tahk, & Krosnick, 2007) as well as other fields such as ecology research (Fletcher, Mackenzie, & Villouta, 2005). Lachenbruch (2001a, 2001b, 2002) has developed two-part models in order that two

such distributions may be compared. The proportion of zeroes is firstly compared and then the difference between the positive values tested; these statistics may then be combined in a  $\chi^2$  test with two degrees of freedom. This two-part approach was to be utilised with the current study when two independent groups of zero-inflated data were to be compared, for example, when analysing whether there was a sex difference for inappropriate sexual behaviour.

The stated first aim with the present study was to determine whether aggressive and inappropriate sexual behaviours observed in the sample could be differentiated or whether they were better represented as reflecting an overall dysregulation of social behaviour. To this end, an exploratory factor analysis was conducted on the six BARS categories of aggression and the two categories of sexually inappropriate behaviour. A principal component analysis (PCA) was carried out, using promax rotation in order to allow any underlying components to correlate. When not testing specific hypotheses, PCA is an acceptable way to explore and describe a complex dataset (Costello & Osborne, 2005; Field, 2005). The concerns raised above about our unusual data structure violating the assumptions of normality are not considered critical in PCA when utilised in this manner (Dudzinski, Norris, Chmura, & Edwards, 1975; Dunteman, 1989; Jackson, 1991; Jolliffe, 2002). Nevertheless, a lognormal transformation –  $\log(x + 1)$  – was used for each behavioural count variable in order to reduce the impact of high frequency counts in the data set. This transformation does not alter the proportion of zeroes.

The PCA was conducted in order to clarify the most appropriate way to further analyse the behavioural data. For instance, it was of particular interest whether it

was preferable to use a single outcome variable of total behavioural incidents (summing across all 8 categories) or, as per the specific research question, whether it would be justified in analysing aggression and sexually inappropriate behaviour separately. In the latter case, it was also of interest whether the six aggression categories should be treated as reflecting one or more underlying components, such as a verbal/physical distinction or, indeed, another distinction such as non-directed aggression (a measure of frustration and irritability) versus more serious aggression directed at self/others. Clarification on this point would then determine the number of logistic regression models required to address the second stated aim, involving the predictive power of the independent variables.

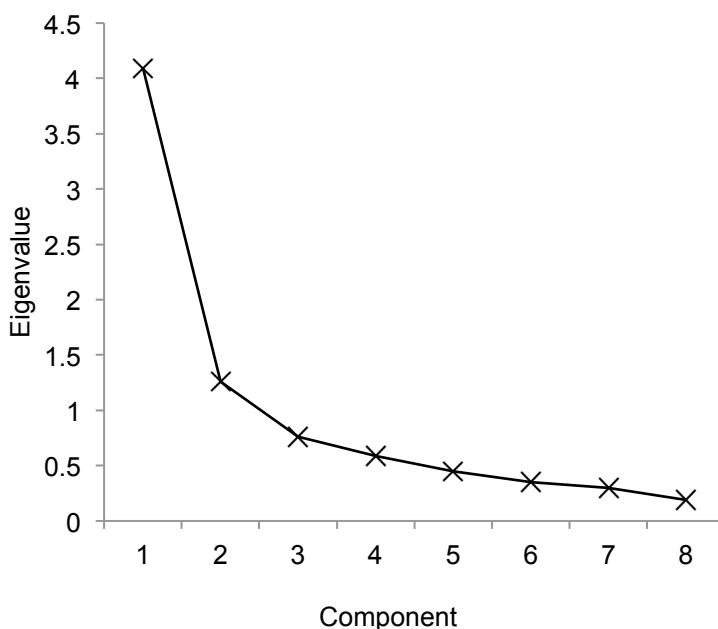
## **5.3. Results**

### **5.3.1. Principal component analysis**

The principal component analysis with promax rotation of the 8 lognormal behavioural count variables produced a component structure with a Kaiser-Meyer-Olkin (KMO) value of 0.81, which is considered acceptable (Field, 2005; Kaiser, 1974). Other measures of the adequacy of using this technique included a highly significant Bartlett's test ( $p < 0.001$ ), all diagonal items of the anti-image correlation matrix were above 0.5 and 50% of non-redundant residuals had absolute values greater than 0.05 (Field, 2005).

The optimal number of components to retain following PCA involves a number of considerations, but the use of eigenvalues and a screeplot has been considered a

“rough guide” (Hutcheson & Sofroniou, 1999, p.229). Using the traditional eigenvalue cut-off score of 1.0, only two components from the present PCA should be retained, explaining 66.8% of the variance. However, employing a more recent (Jolliffe, 2002) criterion of 0.7, three components would be retained explaining 76.3% of the variance. The resulting screeplot (Figure 5.1) shows a reasonably clear inflexion point at the third component with a subsequent straight line, further supporting the conclusion that three components should optimally be retained rather than two.



**Figure 5.1** Eigenvalue screeplot for transformed behavioural counts

Based on this, three components were retained for this data set and the resulting pattern matrix is presented in Table 5.3. Note that any loadings smaller than 0.400 are not included in this table.

It is clear from the pattern matrix that the log-transformed behavioural count variables fall quite neatly into the three components of verbal aggression

(component 1), physical aggression (component 2) and inappropriate sexual behaviour (component 3). Only the lognormal P1 behavioural variable (non-directed physical aggression) showed loadings greater than 0.4 for more than one component. Accordingly, it was decided that these three components would be used for further analysis. Due to the unusual properties of the dataset, the coefficients were not used to compute derived scores for these components. Rather, the sum of all log-transformed counts of verbal aggression ( $\log V1 + \log V2 + \log V3$ ), physical aggression ( $\log P1 + \log P2 + \log P3$ ) and inappropriate sexual behaviour ( $\log S1 + \log S2$ ) were used. For simplicity, the lognormal P1 variable (which loaded marginally more highly on component 2 than 1) was utilised only within the physical aggression component.

**Table 5.3** Pattern matrix for three-component structure with oblique rotation

	Component		
	1	2	3
logV1	0.899		
logV2	0.955		
logV3	0.811		
logP1	0.477	0.495	
logP2		1.035	
logP3		0.452	
logS1			0.853
logS2			0.959

### **5.3.2. Using independent variables to predict behavioural status**

The first research question, regarding whether the recorded behaviours should be treated together or separately, was now determined and it had been demonstrated that three separate components (verbal aggression, physical aggression and inappropriate sexual behaviour) best explained the variance in behavioural data among the 152 participants with brain injury. In addressing the second research question, which was to explore the predictive ability of the independent variables, the size of the sample was limited by those participants for whom a full data set was available. Although data were available for almost all participants in terms of demographics and injury-related variables, the inclusion of robust measures of neurocognitive function limited this subsample to 77 participants.

### **5.3.3. The subsample with full neurocognitive data**

In order to explore any potential bias in this subsample, between-group (complete data versus incomplete data) comparisons were performed on all predictor and outcome variables. These comparisons, which consist of Mann-Whitney tests for the continuous variables (some were non-normally distributed), chi-square tests for dichotomous variables and two-part models for the zero-inflated behavioural component data, are presented in Table 5.4.

**Table 5.4 Between-group comparisons for neurocognitive data**

Variable	Mann-Whitney tests		
	median for incomplete six factors (n = 75)	median for complete six factors (n = 77)	Mann-Whitney Z-score (effect size)
Age at admission	41	37	-1.99 (.01)*
Age at injury	34	35	-1.02 (.01)
Chronicity	13	9	-1.08 (.01)
Education	10	10	-.12 (.00)
GCS (total n = 60)	6 (n = 27)	4 (n = 43)	-.73 (.01)
PTA (total n = 70)	60 (n = 30)	60 (n = 40)	-.03 (.00)
	$\chi^2$ tests		
	incomplete six factors (n = 75)	complete six factors (n = 77)	$\chi^2$ (effect size)
Sex	M53:F22	M61:F16	1.48 (0.10)
Prior ABI	N69:Y6	N64:Y13	2.74 (.13)
Psychiatric history	N57:Y18	N58:Y19	.01 (.01)
Aggression history	N67:Y8	N67:Y10	.20 (.04)
D&A history	N51:Y24	N49:Y28	.32 (.05)
Abnormal imaging (total n = 137)	N5:Y60	N5:Y67	.03 (.01)
Neurosurgery (total n = 143)	N41:Y27	N50:Y25	.63 (.07)
Psychotropic meds	N14:Y61	N28:Y49	5.95 (.20)*
Diagnosis (TBI v non-TBI)	T47:N28	T54:N23	.95 (.08)
	Two-part models for zero-inflated data		
	$\chi^2$ for presence/absence (effect size)	t for non-zero values (effect size)	final $\chi^2$ with 2 df (critical value = 5.99)
Verbal aggression (log-transformed)	.14 (.03)	1.21 (0.13)	1.60
Physical aggression (log-transformed)	1.27 (.09)	.59 (.08)	1.62
Inappropriate sexual behaviour (log-transformed)	.08 (.02)	-.44 (.06)	.27

\*p < .05.

It can be seen from Table 5.4 that there were only two comparisons (age at admission,  $p = .04$ , and psychotropic medication,  $p = .02$ ) that produced a statistically significant difference. Applying any form of adjustment for multiple comparisons at this stage would render both non-significant. Importantly, there were no differences between these two groups on any of the three behavioural component variables of interest. It was therefore felt that this particular subsample was

representative of the total sample and that any relationships uncovered between neurocognitive status and behavioural disturbance should not be obfuscated by any bias, for instance that high levels of behavioural disturbance might have resulted in less complete neurocognitive testing.

#### **5.3.4. Statistical modelling of group membership**

To permit parametric logistic regression modelling of the component variables, several non-normally distributed predictor variables were modified into dichotomies. Education was transformed into “less than 10 years” ( $n = 25$ ) and “10 years or more” ( $n = 52$ ). Chronicity of injury was similarly transformed into “less than 12 months” ( $n = 42$ ) and “12 months or more” ( $n = 35$ ). Diagnosis of injury/illness type was coded into “TBI” ( $n = 54$ ) and “non-TBI” ( $n = 23$ ). Table 5.5 shows the parametric correlations between all predictor variables and the three component behavioural groups.

For consistency, any predictor variable that showed a significant correlation ( $p < .10$ ) with any outcome grouping was included in all three logistic regression models. As such, the following predictor variables were utilised: history of prior ABI; psychiatric history; prior aggression; drug and alcohol history; current use of psychotropic medication; four of the six Wechsler factors (Verbal Comprehension Index, Perceptual Organisation Index, Processing Speed Index and Working Memory Index). A backward stepwise method was utilised in order to minimise suppressor effects (Field, 2005).

**Table 5.5** Correlations between predictor variables and behavioural groupings for subsample with complete neurocognitive data

Predictor variables	Verbal aggression	Physical aggression	Inappropriate sexual behaviour
Demographics			
Age at admission	-.09	-.12	-.04
Education	-.00	-.15	-.01
Sex	-.04	.11	-.07
Clinical history			
Prior ABI	.02	.12	.22*
Psychiatric history	.22*	.23**	.13
Aggression history	.24**	.13	-.09
D&A history	.23**	-.03	.07
Injury details			
Age at injury	-.07	-.10	-.04
Chronicity	.16	.12	.12
Injury type	-.04	-.17	.05
Psychotropic meds	.26**	.31**	.16
Neurocognitive factors			
Verbal comprehension	-.31**	-.31**	-.09
Perceptual organisation	-.21*	-.03	-.14
Processing speed	-.22*	.01	-.10
Working memory	-.26**	-.11	.00
Auditory memory	-.05	-.05	.02
Visual memory	-.19	-.02	-.17

\*  $p < .10$ . \*\*  $p < .05$ .

### 5.3.4.1. Verbal aggression

As 46 of the 77 participants showed some verbal aggression, a null model predicting that each participant is in the “some verbal aggression” group would correctly classify 59.7% of cases. After logistic regression, the final model retained only prior aggression, current use of psychotropic medication and the Verbal Comprehension Index and correctly classified 74% of cases. This model explained 29% of the variance in the data. Table 5.6 shows the results of the logistic regression analyses.

**Table 5.6** Final models of logistic regression analyses

	B (SE)	95% CI for exp <i>b</i>		
		Lower	exp <i>b</i>	Upper
<b>Verbal aggression</b>				
$R^2 = .22$ (Cox & Snell), .29 (Nagelkerke). Model $\chi^2(3) = 18.74, p < .001$ .				
Constant	1.95 (1.30)	-	7.01	-
Aggression history	2.59* (1.18)	1.31	13.33	135.84
Psychotropic meds	1.46* (.57)	1.40	4.29	13.12
Verbal Comprehension Index	-.06* (.03)	.89	.94	.99
<b>Physical aggression</b>				
$R^2 = .17$ (Cox & Snell), .23 (Nagelkerke). Model $\chi^2(2) = 14.23, p = .001$ .				
Constant	1.09 (1.33)	-	2.97	-
Psychotropic meds	1.47* (.63)	1.28	4.37	14.94
Verbal Comprehension Index	-.06* (.03)	.89	.94	.99
<b>Inappropriate sexual behaviour</b>				
$R^2 = .04$ (Cox & Snell), .06 (Nagelkerke). Model $\chi^2(1) = 3.51, p = .06$ .				
Constant	-1.02	-	.36	-
Prior ABI	1.17 (.62)	.95	3.23	10.96

\* $p < .05$

In this model, a positive history of aggression, taking psychotropic medications and poorer Verbal Comprehension Index scores were statistically significant in predicting verbal aggression. A forensic history of aggression increased the proportionate change in odds of being verbally aggressive approximately 13-fold, while the odds were four times higher if the participant was also taking psychotropic medication. With regard to the Verbal Comprehension Index, for every unit increase in score the odds were reduced by .06; the poorer the score, the higher the odds of being verbally aggressive.

#### 5.3.4.2. Physical aggression

A null model predicting that no-one behaved in a physically aggressive manner would successfully predict 66.2% of participants, as 51 of 77 did not show any such

behaviour. After logistic regression, the model successfully predicted 64.9% of participants, slightly worse than the null model, and accounted for 23% of the variance in the data. Nevertheless, it was a statistically significant model in which taking psychotropic medication and poorer Verbal Comprehension Index scores were also predictive of physical aggression. Similar to verbal aggression, the presence of the former increased the odds four-fold, while every unit increase in VCI reduced the odds by .06.

#### **5.3.4.3. Inappropriate sexual behaviour**

For inappropriate sexual behaviour, a null model in which no participants were predicted to exhibit these behaviours successfully classified 68.8% of cases, as 53 participants showed no ISB. No variables significantly ( $p < .05$ ) predicted group membership, although history of having had a prior brain injury approached significance. This variable was retained in Table 5.6, as the removal criterion was  $p < .10$ . Equally, the model did not quite reach significance.

### **5.4. Discussion**

Two critical issues with this data set had been identified. Firstly, as episodes of aggressive and inappropriate sexual behaviour exhibited by participants were systematically recorded as rehabilitation staff observed them, there was an opportunity to analyse relationships between these types of behaviour for the first time. It was anticipated that aggressive behaviours would be distinguished statistically from inappropriate sexual behaviours. This would provide compelling

evidence that they represent separate clinical entities, rather than manifestations of a general behavioural disinhibition syndrome. It was not clear whether the different forms of aggressive behaviour recorded with the BARS would differentiate further. Secondly, in keeping with previous research, it was anticipated that there would be small to medium sized relationships between a number of predictor variables and aggressive behaviour, while no such associations would be present for inappropriate sexual behaviour.

Considering the first research question, as anticipated, principal component analysis provided strong grounds for treating inappropriate sexual behaviour separately from aggressive behaviours in the complete sample of 152 participants. As this study is without precedent, there are no empirical data elsewhere with which to compare. In their single case report of psychosocial treatment of inappropriate sexual behaviour after brain injury, Kelly and Simpson (2011) previously speculated that these behaviours have distinct functional features that differ from those involved in aggression. The current results support such a notion; even when applying conservative criteria for how many components should be retained during principal component analysis there was a clear distinction between aggressive and inappropriate sexual behaviours.

When applying more recent (and less conservative) criteria, three components were retained, as a distinction between verbal aggression and physical aggression emerged in addition to inappropriate sexual behaviour. Others have also reported differences between verbal and physical aggression. A group of people with traumatic brain injury showed higher levels of verbal aggression than control groups

(spinal cord injury and an uninjured group) but not physical aggression (Dyer et al., 2006) and that this was associated with greater impulsivity. Most of the high level of aggressive behaviours recorded by Alderman and colleagues (Alderman, 2007; Alderman et al., 2002) was noted to be verbal rather than physical in nature and they noted that these forms of aggression showed different setting events and antecedents. As such, it was determined here that three separate behavioural components – verbal aggression, physical aggression and inappropriate sexual behaviour – were to be retained for the second research question.

In order to address the second issue, a range of premorbid psychosocial and post-injury clinical variables were used to predict the presence of verbal aggression, physical aggression and inappropriate sexual behaviour. For this, a subset of 77 participants for whom neurocognitive data was available was utilised. This subset did not differ in any meaningful way from those participants without neurocognitive data. Three separate groupings were created for presence/absence of each behavioural category; these were then subjected to logistical regression. Future analyses involving larger sample sizes could also address the degree of behavioural disturbance within each ‘presence’ group, within a two-part process recommended by statisticians (Lachenbruch, 1992, 2001a, 2001b, 2002). This approach would facilitate a more complete analysis of the variance in recorded behavioural disturbances.

Consistent with previously published studies, there were a number of significant correlations between the predictor variables and group membership for both verbal and physical aggression. These were of small to medium effect size (Cohen, 1988,

1992). When subjected to logistic regression, however, only three remained in the final model for verbal aggression and two for physical aggression (explaining 29% and 23% of the variances, respectively). Two predictor variables were the same for each: poorer verbal skills, as measured by the Verbal Comprehension Index (Tulsky & Price, 2003), and the concurrent use of psychotropic medications. Each was of similar predictive power in both models. A premorbid history of aggression was a strong predictor of verbal aggression only.

Poor verbal skills have been previously associated with aggressive behaviours that are commonly seen in brain injury rehabilitation programmes (Alderman, 2007; Alderman et al., 2002). It has been hypothesised that linguistic processing may help in regulating those executive functions such as abstract reasoning, cognitive modulation of emotion and reflection that are necessary to inhibit aggressive impulses (Miller, Collins, & Kent, 2008). Additionally, significant language impairments may interfere with standard non-aversive behavioural treatments for aggression (Alderman, 2007).

Although concurrent use of non-specified psychotropic medication remained in the models for both categories of aggression, there was no information available as to the temporal relationship of when these medications had been introduced. Therefore it was impossible to determine what class of psychotropic medication had been prescribed and at what stage post-injury. While a particular medication may have been introduced in order to treat behavioural disturbance, there are documented adverse effects on social behaviours for some medications – for example, the newer types of antiepileptics (Schmitz, 2006). Future studies of this type should examine

more closely any contribution that psychotropic medications may have in exacerbating or reducing the behavioural disturbance under investigation.

It was unclear why a premorbid history of aggression would be a relatively strong predictor for post-injury verbal aggression (increasing the odds by a factor of 13) but not be associated with post-injury physical aggression. A similar finding (but not specific to verbal or physical) has been previously reported in the literature. Greve et al. (2001) speculated that traumatic brain injury did not cause a fundamental personality change but rather ‘further disinhibited an already impulsive and aggressive individual’ (p. 260) and that this implied that most individuals without such a history will not develop these behaviours post-injury. In the present study, specific reports of pre-injury violence in the records had been coded but potential covariates had not taken into account, such as whether the behaviour had occurred in the context of acute intoxication. This disparity between such a history predicting verbal but not physical aggression requires replication and further scrutiny.

In contrast to some previous studies (Kerr et al., 2011; Tateno et al., 2003; Wood & Liossi, 2006b), this study found no predictive value for post-injury aggression (either verbal or physical) in a history of premorbid drug and alcohol misuse, male sex or lower education/socio-economic status. Differences in anterograde memory function between aggressive and non-aggressive groups have been reported in brain injury (Wood & Liossi, 2006b) but not with a prison population (Barratt et al., 1997). The current study included psychometrically robust measures of both auditory and visual anterograde memory: neither was related to the occurrence of aggressive behaviour.

Associations between poorer executive functioning and a higher risk of aggression have been demonstrated in many groups of people, including clinical (Gansler et al., 2009; Grafman et al., 1996; Hancock, Tapscott, & Hoaken, 2010) and non-clinical groups (Giancola & Zeichner, 1994; Hoaken, Shaughnessy, & Pihl, 2003; Kockler & Stanford, 2008; Lau, Pihl, & Peterson, 1995). Wood and Liossi (2006b), while not finding a relationship between post-injury aggression and performances on neuropsychological tests of cognitive executive function, reported associations with behavioural measures of impulsivity and disinhibition. The present study did not include measures of executive functioning, but these should clearly be incorporated into further research in this area and will be addressed later in this thesis. Neurobiological models of both aggression (Siever, 2008; Volavka, 2002) and sexual behaviour (Rees, Fowler, & Maas, 2007; Spinella, 2007) highlight the importance of so-called “top-down” executive cognitive processes involved in the regulations of these behaviours. The models also detail the relevance of the prefrontal cortex in such processes.

In the separate analysis of inappropriate sexual behaviour, none of the predictor variables were significantly correlated with group membership and a logistic regression model could not be constructed. This was also consistent with the only previously published study looking at these behaviours in a brain injury sample (Simpson et al., 2001), in which a wide range of potential predictors were explored. Due to inconsistencies in the neuropsychological measures available in that study, instead of utilising standardised data these researchers used a classification system across a range of neurocognitive constructs, including various executive processes. The present study used psychometrically robust data for the first time with

inappropriate sexual behaviour – however, the results were remarkably similar. Clearly, attempts must be made to incorporate more reliable measures of executive cognitive functioning into future research.

What do these results mean in the broader clinical context of verbal aggression, physical aggression and inappropriate sexual behaviour? These behaviours are undoubtedly multifactorial, mediated by complex interactions between the damaged brain and the immediate environment. Furthermore, despite relatively sophisticated statistical analyses, causation cannot be implied (Alderman, 2007; Simpson et al., 2001; Wood & Liossi, 2006b). It would seem that for the presence of both forms of aggression, several pre-injury and clinical variables can account for a minority of the variance seen in this sample. The remaining unaccounted variance continues to be worth exploring in order to better understand and treat post-injury aggressive behaviours. For inappropriate sexual behaviour, these results reaffirm Simpson et al.'s (2001) concluding comments that more complex models of sexual behaviour and brain function need to be developed alongside more precise clinical measurement so as to further understanding in this area.

# **CHAPTER 6. STUDY 3: TWO-PART REGRESSION**

## **MODELLING WITH NEUROBEHAVIOURAL PREDICTORS**

### **6.1. Introduction**

For the first time, the previous study (chapter 5 in this thesis) examined verbal aggression, physical aggression and inappropriate sexual behaviour exhibited within the same sample of people with severe acquired brain injury. All 152 participants had been admitted to a single residential neurobehavioural rehabilitation unit in England, considered a community re-integration programme within the continuum of neurobehavioural rehabilitation (Wood & Worthington, 2001). Statistical analyses indicated that these behaviours were better considered as separate entities rather than part of a general behavioural disinhibition syndrome. This finding warranted replication in a larger sample that encompassed a broader range of behavioural disturbances across a variety of brain injury rehabilitation settings.

The clinical correlates of aggression after brain injury are complex and multi-faceted. Previous empirical research has documented associations with premorbid psychosocial variables (Greve et al., 2001; Kerr et al., 2011; Tateno et al., 2003; Visscher et al., 2011; Wood & Liossi, 2006b) as well as post-injury clinical indicators (Alderman, 2007; Alderman et al., 2002; Brower & Price, 2001; Chan et al., 2006; Kerr et al., 2011; Paradiso et al., 1996; Pardini et al., 2011; Siever, 2008; Tateno et al., 2003; Wood & Liossi, 2006b). There has been considerably less research conducted into potential clinical correlates of inappropriate sexual behaviour

following acquired brain injury with the only such research not finding any similar associations (Simpson et al., 1999; Simpson et al., 2001). The previous study (chapter 5 in this thesis) found results in keeping with this pattern, namely that several pre- and post-injury variables increased the likelihood of verbal aggression and physical aggression with no such relationships with inappropriate sexual behaviour. The present study was therefore designed to replicate the statistical distinctions between verbal aggression, physical aggression and inappropriate sexual behaviour with the BARS and a newly available observational tool designed for recording inappropriate sexual behaviour after brain injury. Additionally, a broader range of neurobehavioural symptomatology (as captured by the MPAI-4) was included as predictor variables for the two-part regression modelling (Lachenbruch, 1992, 2001a, 2001b, 2002).

### **6.1.1. St Andrew's Sexual Behaviour Assessment (SASBA)**

A relatively recent review paper concerning defining and evaluating inappropriate sexual behaviour in people after brain injury (Johnson et al., 2006) recommended that continuous assessment of these behaviours with structured measurement tools was desirable. The same group of researchers subsequently developed a standardised system for continuously recording challenging sexual behaviour, the St Andrew's Sexual Behaviour Assessment (SASBA, Knight et al., 2008).

The structure of the SASBA was largely based on the same group's earlier work on aggression. Their modification of the original Overt Aggression Scale (OAS, Yudofsky et al., 1986), which largely consisted of extending the range of possible

interventions and introducing a range of antecedents, became known as the Overt Aggression Scale – Modified for Neurorehabilitation (OAS-MNR, Alderman et al., 1997). This scale utilises a 4x4 matrix to classify aggressive behaviours according to four categories of behaviour with four levels of severity. A similar 4x4 matrix was employed with the following categories of inappropriate sexual behaviour: verbal comments, non-contact, exposure and touching others. An “aggregate” score similar to that developed by the authors for the OAS-MNR (Alderman, Knight, Stewart, & Gayton, 2011) is also used with the SASBA (N. Alderman, personal communication, 20<sup>th</sup> June, 2012).

The authors reported adequate validity and reliability analyses, although they recommended that staff training in using the protocol was important. Review of the literature found two subsequent papers on the use of the SASBA, both published by the developers (Alderman et al., 2009; Stewart et al., 2010).

The first of these papers (Alderman et al., 2009) documented a survey of challenging behaviour within a brain injury neurorehabilitation service. The OAS-MNR and the SASBA were employed by staff to record incidents of aggression and inappropriate sexual behaviour among 91 hospitalised participants over a 12 week period. Most of the participants displayed aggression (82.4%) and less than seven percent of all incidents were inappropriately sexual by nature. Less than half of the participants (41.8%) displayed inappropriate sexual behaviour and two of these individuals accounted for nearly half of these SASBA incidents. Only three (0.4%) recordings were attributable to female patients, whereas a higher proportion of aggressive behaviours (11.9%) was seen in females. Amongst the inappropriate sexual

behaviours, verbal comments were the most frequently observed type of incident (47.6%) with exposure the least common type (5.3%).

The authors concluded that, as there was only a modest correlation between aggression and inappropriate sexual behaviour, the two have differing aetiologies. Furthermore, the authors also commented that, from their analysis of antecedents and interventions, they likely have different functions: “aggressive behaviour that of avoidance and escape, whilst ISB may primarily fulfil a social distance reduction function” (p. 218). By the latter they had speculated that, in the context of very limited social engagement, some patients exhibited this as an expression of sexual need.

The second paper involving the SASBA (Stewart et al., 2010) was an observational report of a clinical audit in an older adult hospital ward. During a three month period, 97 inpatients (76 males and 21 females) were audited. Over this sample time period, 225 separate incidents of inappropriate sexual behaviour were documented. These had been exhibited by 32% of the sample, compared to a much higher proportion of the sample displaying aggression.

The SASBA was therefore used in study 3 alongside the BIRT Aggression Rating Scale (BARS, Freeland et al., 2012), which was already used by rehabilitation staff to record observed aggressive behaviours. Using the SASBA in this clinical population would facilitate greater discrimination than had been previously possible, thereby providing an opportunity to further explore the relationships between aggressive and inappropriate sexual behaviours.

### **6.1.2. Mayo-Portland Adaptability Inventory (MPAI-4)**

In order to collect as wide a range as possible of neurobehavioural outcome information, the various treating clinical teams within the multiple rehabilitation centres were also asked to complete the Mayo-Portland Adaptability Inventory (MPAI-4) for all participants in study 3. This also avoided the inconsistency in usage of neuropsychological tests between psychologists (Archer, Buffington-Vollum, Stredny, & Handel, 2006; Lees-Haley, Smith, Williams, & Dunn, 1996; Rabin, Barr, & Burton, 2005; Sullivan & Bowden, 1997) and the potential impact of test revisions (Strauss, Spreen, & Hunter, 2000) over a lengthy data collection phase.

The Mayo-Portland Adaptability Inventory (MPAI) is a clinical rating scale that was designed for the assessment of people with acquired brain injury in the post-acute rehabilitation period. The scale may be completed by clinicians, significant others or self-rated by the person with brain injury. Based upon the original Portland Adaptability Inventory (Lezak, 1987) and, following significant revisions (Bohac, Malec, & Moessner, 1997; Malec et al., 2003; Malec, Moessner, Kragness, & Lezak, 2000; Malec & Thompson, 1994), the MPAI is now in its fourth version (Malec & Lezak, 2003, April).

The MPAI-4 is a 29-item scale covering typical sequelae of brain injury in the physical, cognitive, emotional, behavioural and social domains (Bellon, Malec, & Kolakowsky-Hayner, 2012). Each item is rated on a 5-point Likert scale, with a typical scoring range: 0 (no problem), 1 (mild problem not interfering with activities),

2 (mild problem interfering no more than 24% of the time), 3 (moderate problem interfering 25-75% of the time) and 4 (severe problem interfering more than 75%).

These 29 items are grouped into three subscales: Ability (consisting of 12 items covering sensory, motor and cognitive skills), Adjustment (consisting of 9 items focussing on mood and interpersonal skills) and Participation (consisting of 8 items addressing social contact, initiation and financial management). There are also an additional six items (not part of the subscales) that address pre-existing and associated conditions. The three subscales are summed (subtracting three overlapping items within Adjustment and Participation) to produce a total score, reflecting an overall measure of outcome after acquired brain injury. The developers report that these three subscales were derived “rationally” following Rasch item analysis, despite several exploratory factor analyses indicating that seven or eight underlying factors were present.

The MPAI-4 manual (Malec & Lezak, 2003, April) provides comparative data for these four scales, with reference to either a US national sample of 386 people with acquired brain injury (88% traumatic brain injury) or a Mayo sample of 134 people (65%) traumatic brain injury. T-scores (mean of 50, standard deviation of 10) are derived from these comparative samples, with higher scores representing greater dysfunction. Note that these comparisons scores are made against others with acquired brain injury, not the neurologically healthy population. The following guidance is provided for interpretation of these T-scores:

- Below 30 represents relatively good outcomes
- Between 30 to 40 suggest mild limitations

- Between 40 to 60 considered average or typical –
  - 40-50 mild to moderate range of overall severity compared to others with acquired brain injury
  - 50-60 moderate to severe range
- Above 60 suggest severe limitations even when compared to other people with acquired brain injury

The developers advise that scoring and interpretation of the MPAI-4 requires professional training and experience, and that clinical staff evaluation by consensus leads to better accuracy and reliability (Malec & Lezak, 2003, April). Until recently, the MPAI-4 was considered to have substantial empirical research supporting its validity but limited published information regarding reliability (Tate, 2010). Adequate reliability had been previously reported in a paediatric sample (Oddson, Rumney, Johnson, & Thomas-Stonell, 2006) and this was also later reported in adults (Kean, Malec, Altman, & Swick, 2011).

The MPAI-4 has been widely utilised in clinical research. This has included examining psychological adjustment after brain injury (Beck, Franks, & Hall, 2010; Jacobsson, Westerberg, Malec, & Lexell, 2011; Malec, Brown, Moessner, Stump, & Monahan, 2010; Silva, Ownsworth, Shields, & Fleming, 2011) and evaluating progress within post-acute rehabilitation (Altman, Swick, Parrot, & Malec, 2010; Eicher, Murphy, Murphy, & Malec, 2012) or an adolescent mentoring programme (Fraas & Bellerose, 2010). The scale has also been used in acquired brain injury research investigating anosognosia (Murrey, Hale, & Williams, 2005), hypopituitarism (Srinivasan et al., 2009), acute medical treatment decisions (Malec,

Mandrekar, Brown, & Moessner, 2009) and in examining the ecological validity of a neuropsychological battery (Zgaljardic, Yancy, Temple, Watford, & Miller, 2011).

Most recent research involving the MPAI-4 has used the three subscale and total scores. Regression analyses have been conducted using these subscales of the MPAI-4 as both dependent and independent variables (Eicher et al., 2012; Jacobsson et al., 2011; Zgaljardic et al., 2011). There have, however, also been explorations of the underlying factor structure of the items comprising the MPAI (Bohac et al., 1997; Kean et al., 2011; Malec et al., 2003).

In their principal component analysis of the original 30 item MPAI, Bohac et al. (1997) used data from 189 participants with acquired brain injury. Four items were subsequently excluded from the initial thirty (“audition”, “law violations”, “alcohol use” and “illegal drug use”) as they correlated weakly with other items. Seven factors were indicated using the standard criterion of eigenvalues greater than 1.0, but following examination of the screeplot and the residual correlation matrix the authors felt that 8 factors explaining 64.4% better modelled their data. Orthogonal rotation was used as the factors were not highly inter-correlated even after reanalysing with oblique rotation.

Further exploratory factor analysis of the MPAI was reported in the development of the current, i.e., fourth version (Malec et al., 2003). The 29 items of the MPAI-4 produced 7 factors after principal component analysis using the criterion of eigenvalues greater than 1.0. The authors concluded that although such a multifactorial structure may be useful when investigating outcome after brain injury,

the three rationally-derived subscales provided greater psychometric reliability and practical use.

### **6.1.3. Research questions**

The research questions for study 3 were therefore as follows:

(1) It was anticipated that exploratory factor analysis (principal component analysis) would again show discrimination between verbal aggression, physical aggression and inappropriate sexual behaviour in this larger multi-centre sample. So that direct comparisons could be made, incidents of inappropriate sexual behaviour documented with the SASBA firstly needed recoding to ensure that they were consistent with the previous study. Secondly, utilisation of the original four categories of behaviour within the SASBA would confirm the empirical distinctions between verbal aggression, physical aggression and inappropriate sexual behaviour.

(2) Given a larger sample than the previous study (chapter 5), two-part modelling of the *presence* and *degree* of each behavioural disturbance would be possible. The two-part modelling process (Lachenbruch, 1992, 2001a, 2001b, 2002) involves performing a logistic regression analysis predicting whether the behaviour should be present and then a separate linear regression analysis on the non-zero values to predict the amount of behaviour observed. It was anticipated that, within this two-part modelling of verbal aggression, physical aggression and inappropriate sexual behaviour, the broader range of clinical symptomatology captured by the MPAI-4

would provide greater predictive ability than did the six neurocognitive variables used in the previous study.

(3) It was anticipated that this large dataset would replicate the previously reported underlying factor structures of the MPAI-4. Use of the resulting factors would then permit closer examination of the individual items associated with verbal aggression, physical aggression and inappropriate sexual behaviour.

## **6.2. Method**

### **6.2.1. Participants**

Three hundred and one participants were recruited from a total pool of admissions to seven organisational residential rehabilitation programmes across the UK during the period January 2010 to June 2012. Two of the programmes specialised in challenging behaviour while the remaining five were classed as community re-integration (although one programme within a local hospital ward was also considered sub-acute rather than post-acute). Participants were included if they had completed at least 9 weeks of residential neurobehavioural assessment, which included continuous behavioural observation and recording. Additionally, for inclusion in the study participants needed to have had each of the specified psychometric measures completed on admission by the treating clinical team.

### **6.2.1.1. Demographics**

Two hundred and thirty-five (78%) of the participants were male and 66 (22%) female. Age at admission, which was normally distributed, ranged from 16 to 76 years, with mean of 42.7 years and standard deviation of 14.6 years. Years of formal education ranged from 6 to 18, which was positively skewed and leptokurtic, with a median value of 10 years. The majority of the sample (93%) was identified as predominantly right-handed prior to their injury/illness.

### **6.2.1.2. Clinical histories**

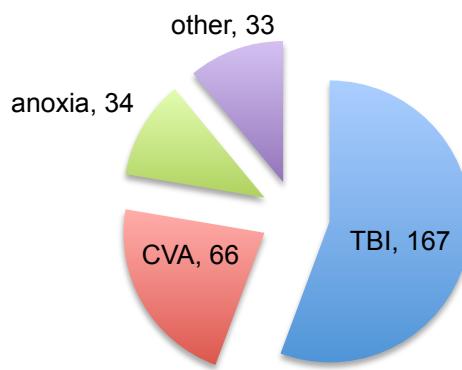
A number of variables were coded relating to a participant's pre-injury clinical history. A history of significant brain injury prior to the injury/illness for which rehabilitation was initiated was present in 39 (13%) participants. Previous psychiatric illness had been documented in 56 (19%). Twenty-eight participants (9%) had a pre-injury history of aggression leading to a criminal conviction, while only 2 (1%) participants had been convicted of a sexual offence premorbidly. Pre-injury substance misuse had been identified in 113 participants (38%).

### **6.2.1.3. Details of brain injuries and illnesses**

#### **6.2.1.3.1 Types of injuries**

Figure 6.1 below shows the types of acquired brain injuries or illnesses in the sample. Note that this information could not be obtained for one participant so the

figure sums to three hundred only. Over half of the brain injuries in this sample were traumatic in nature (56%). These could be further broken down into road traffic accidents ( $n = 77$ ), falls ( $n = 47$ ), assaults ( $n = 34$ ), combat-related TBI ( $n = 4$ ) and other ( $n = 5$ ).



**Figure 6.1** Aetiologies of brain injuries in sample for study 3

The next largest proportion (22%) of injuries was cerebro-vascular accidents. These were made up of a similar number of occlusive- ( $n = 35$ ) and haemorrhagic-type ( $n = 31$ ). Cerebral anoxia made up 11% of the sample, for which the most common mechanism was cardiac arrest ( $n = 14$ ). Other types of injuries or illnesses made up 11% of the sample and included infectious diseases ( $n = 16$ ), cerebral tumour ( $n = 6$ ) and alcohol-related brain damage ( $n = 4$ ).

#### **6.2.1.3.2     *Age at injury/illness and chronicity***

The age at which the participants acquired their brain injuries or illnesses ranged from 1 to 75 years and was normally distributed, with mean age 39.7 years and standard deviation 16.8 years. Chronicity, or time between injury/illness and admission to a rehabilitation programme ranged from 1 month to 636 months. Chronicity was not normally distributed, being positively skewed and leptokurtic. Median chronicity was 5.9 months, consistent with the conceptualisation of the neurobehavioural rehabilitation as post-acute in nature.

#### **6.2.1.3.3     *Injury severity***

Data regarding severity of injury were incomplete. Lowest Glasgow Coma Scale (GCS; Teasdale & Jennett, 1974) prior to sedation was available for 126 (42%) participants. This ranged from the lowest (3) to the highest (15) possible scores and was positively skewed, indicating a more severely injured sample, with a median score of 5. GCS information was most frequently reported for participants with traumatic brain injury (54%), compared with cerebro-vascular accident or cerebral anoxia (both 29%). Over three-quarters (79%) of the available GCS values were 8 or less, generally considered to reflect the most severe category of injury (Lezak, 1995).

For traumatic brain injuries, the duration of post-traumatic amnesia (PTA) is often considered a good indicator of the severity of the injury (Lezak, 1995). This information was reported for 47 participants (28%) with TBI. PTA ranged from 1 day

to 500 days; unsurprisingly this was not normally distributed, being both positively skewed and leptokurtic in nature. Median value was 70 days. Thirty-nine (83%) participants had reported duration of PTA of 28 days or more, which reflects the most severe category of injury (Lezak, 1995).

Although data on severity were incomplete, the available information indicated that this sample had a very high representation of severe brain injuries. This is consistent with previous clinical research (Braunling-McMorrow et al., 2010; Glenn et al., 2005) in similar post-acute brain injury rehabilitation programmes. Such a high proportion of severe injuries is also consistent with the sample in study 2 (chapter 5 in this thesis).

#### **6.2.1.3.4      *Other relevant clinical information***

To further examine how severely injured this sample was, other clinical information was also obtained. Abnormal neuroimaging was reported in 233 out of 242 participants (96.3%), but inconsistencies in reporting permitted no further analysis of this information. Neurosurgery in the acute medical treatment stage was required for 94 of 216 participants (43.5%).

Seventeen of 214 participants (7.9%) were involved in litigation during their residential neurobehavioural assessment. This was most typically a civil compensation case arising from a road traffic accident.

Details of prescribed psychotropic medication were available for 293 of the 301 participants (97.3%). The majority of participants ( $n = 213$ , 72.7%) were taking at least one such medication. The breakdown within four major classes was as follows: anti-depressants (38.2%), anti-convulsants (45.7%), anti-psychotics (19.5%) and anxiolytics (8.9%). Note that any medications prescribed PRN or, as needed only, were not included.

### **6.2.2. Materials**

Data were collected for each participant on their behaviour during the nine week initial assessment period, specifically forms of aggression and inappropriate sexual behaviour. Psychometric measures of neurobehavioural function and levels of care/supervision were completed within the first four weeks of admission.

#### **6.2.2.1. Behavioural recordings**

In keeping with the previous study (see chapter 5), contemporaneous behavioural recordings were made by staff on all service users as part of routine monitoring and analysis of social behaviours. All staff had previously received formal training in behavioural documentation and psychology staff later reviewed the accuracy of codings.

Aggressive behaviour was coded according to the BIRT Aggression Rating Scale (BARS), which has demonstrated good inter-rater reliability and concurrent validity (see chapter 4 in this thesis, Freeland et al., 2012). The BARS codes aggression into

six categories reflecting the nature (verbal or physical) and severity (1, 2 or 3) of each episode. Incidents of verbal aggression are scored as V1 for non-directed, V2 for directed at another person or V3 for verbal threats. Similarly, incidents of physical aggression are scored as P1 for non-directed, P2 for damage to property and P3 for violence towards another person or one's self.

Inappropriate sexual behaviour was coded in a different manner to the previous study. The previous study had utilised the codes S1 for spoken comments (for example, if a client asked a staff member to participate in sexual activity or asked for personal information of a sexual nature) and S2 for inappropriate action (such as attempting to touch a staff member in a sexual manner). For the current study, episodes of inappropriate sexual behaviour were recorded by staff with the St Andrews Sexual Behaviour Assessment – SASBA (Knight et al., 2008). This scale consists of four categories of behaviour (Verbal Comments, Non-Contact, Exposure and Touching Others) with four severity levels within each category. This produces a matrix of 16 specific behaviour codes, although an “aggregate” score combining category and frequency is also used (see equation 4 below).

Participants' behaviours were observed for the nine weeks of assessment in order to obtain a baseline from which later clinical decisions could be taken. In most cases, no specific intervention was applied during that time. Data recorded in the first week were not included as this was often only a partial week depending on the day of admission. Therefore, for each participant, total frequencies of recorded incidents across the assessment period were obtained for each of these 22 raw behavioural variables:

- BARS (6 behavioural codes): V1, V2, V3, P1, P2, P3
- SASBA (16 behavioural codes): VC1, VC2, VC3, VC4, NC1, NC2, NC3, NC4, E1, E2, E3, E4, TO1, TO2, TO3, TO4

Additionally, aggregate scores (combining category and severity of behaviour) were available for:

- BARS Irritability/Aggression Index (I/A)

$$I/A = \text{sumV1} + (2 \times \text{sumV2}) + (3 \times \text{sumV3}) + (2 \times \text{sumP1}) + (4 \times \text{sumP2}) + (6 \times \text{sumP3}) \quad (1)$$

- BARS Verbal Aggregate (VA) score:

$$VA = \text{sumV1} + (2 \times \text{sumV2}) + (3 \times \text{sumV3}) \quad (2)$$

- BARS Physical Aggregate (PA) score:

$$PA = (2 \times \text{sumP1}) + (4 \times \text{sumP2}) + (6 \times \text{sumP3}) \quad (3)$$

- SASBA Aggregate (SA) score:

$$SA = \text{sumVC1} + (2 \times \text{sumVC2}) + (3 \times \text{sumVC3}) + (4 \times \text{sumVC4}) + (2 \times \text{sumNC1}) + (4 \times \text{sumNC2}) + (6 \times \text{sumNC3}) + (8 \times \text{sumNC4}) + (3 \times \text{sumE1}) + (6 \times \text{sumE2}) + (9 \times \text{sumE3}) + (12 \times \text{sumE4}) + (4 \times \text{sumTO1}) + (8 \times \text{sumTO2}) + (12 \times \text{sumTO3}) + (16 \times \text{sumTO4}) \quad (4)$$

As in the previous study reported in this thesis (study 2, chapter 5), each behavioural variable, whether raw or aggregate, was expected to have a highly non-normal distribution with an excess of zero counts. Therefore, the use of specific statistical techniques developed to address this particular issue (Chang & Pocock, 2000; Delucchi & Bostrom, 2004; Fletcher et al., 2005; Huson, 2007; Lachenbruch, 1992, 2001a, 2001b, 2002; Schneider et al., 2007) was considered appropriate.

### **6.2.2.2. Neurobehavioural function and care/supervision**

Within the first two weeks of admission, each participant was rated via consensus of his or her treating clinical team on the following psychometric measures:

- Mayo-Portland Adaptability Inventory 4 – MPAI-4 (Malec & Lezak, 2003, April)
- Care and Needs Scale – CANS (Tate, 2004), an eight category scale utilised to capture the wide range of support needs after brain injury. The current

study has used only the Support Levels extent of support, which ranges from 0 (does not need any contact) to 7 (cannot be left alone).

- Supervision Rating Scale – SRS (Boake, 1996), a thirteen point ordinal scale measuring the level of supervision that an individual receives from caregivers. The SRS can also be grouped into five ranked categories of Independent, Overnight Supervision, Part-Time Supervision, Full-Time Indirect Supervision and Full-Time Direct Supervision. Subsequent work (Hart et al., 2003) has utilised a three-group formation: independent (SRS score 1-2), moderate supervision (SRS score 3-5) and heavy supervision (SRS score 6-13).

#### **6.2.2.3. Other independent variables**

A number of premorbid psychosocial and other post-injury clinical variables were also coded for each of the participants. These variables were scrutinised prior to the two-part statistical modelling

##### **6.2.2.3.1 *Demographics and premorbid information***

Age at admission and age at injury (both measured in years and normally distributed) were highly correlated ( $r = .92$ ) and therefore only one was required for multivariate analyses. Age at injury was selected, as being injured at a younger age has previously been associated with poorer behavioural and psychosocial outcomes (Bedell, 2008; Crowe, Catroppa, Babl, & Anderson, 2012; Donders & Warschausky, 2007; Karver et al., 2012; Leblanc, Chen, Swank, Levin, & Schachar, 2006; Sonnenberg, Dupuis, & Rumney, 2010).

The distributions of chronicity, i.e., time between injury and admission to the rehabilitation unit, and years of formal education could not be normalised following various suggested transformations (Field, 2005). Both were therefore modified into dichotomous variables. Those participants admitted within 6 months of their injury ( $n = 153$ , 50.8%) were split from those admitted 6 months or more after their injury ( $n = 148$ , 49.2%). Similarly, those without the equivalent of UK GCSE education ( $n = 98$ , 32.6%) were separated from those with this level or higher ( $n = 203$ , 67.4%). Lower level of pre-injury education has consistently been associated with poorer functional outcomes after severe brain injury (Draper, Ponsford, & Schonberger, 2007; Ketchum et al., 2012; Kim, 2011; Ponsford, Draper, & Schonberger, 2008; Schonberger, Ponsford, Olver, Ponsford, & Wirtz, 2011; Sigurdardottir, Andelic, Roe, & Schanke, 2009; Spitz, Ponsford, Rudzki, & Maller, 2012).

Other dichotomous variables consisted of sex and the clinical history variables of prior brain injury, psychiatric illness, criminal convictions for aggression and prior substance misuse. These variables were described in more detail above. A criminal conviction for sexual offences was not included as a variable given that only two participants had such a history. Interestingly, neither of these participants exhibited any inappropriate sexual behaviour on the SASBA but both had recordings for aggressive behaviour made on the BARS.

#### 6.2.2.3.2 *Post-injury clinical information*

The type of acquired brain injury had been initially encoded as a categorical variable with four groups (traumatic brain injury, cerebro-vascular accident, cerebral anoxia,

other). Once the outcome behavioural variables (to be used as dependent variables) were finalised, it would then need to be determined whether type of injury would be dichotomised (for example, TBI versus non-TBI) or dummy variables for the four initial types would be utilised for regression analyses.

### **6.2.3. Procedure**

#### **6.2.3.1. Replication of independent behavioural components**

##### **6.2.3.1.1 *Using the same behavioural count variables***

In order to address the first research question in this study, the first step was to examine the structure of the behavioural observations. Study 2 (chapter 5 in this thesis) utilised the 6 categories of aggressive behaviour of the BARS with two measures of observed inappropriate sexual behaviour – S1 and S2. In order to be able to compare directly, SASBA data in the new sample required conversion into equivalents for S1 and S2, reflecting the way that the behavioural observations had been coded in the previous study. Consequently, any SASBA Verbal Comments recordings (VC1 through VC4) were summed as S1 and the remaining SASBA recordings (NC1 through TO4) were summed as S2. This produced the same 8 behavioural count variables as previously. Table 6.1 below shows these behavioural variables and it is clear that they each contain a high proportion of zero values and are non-normally distributed.

**Table 6.1** Distribution of raw behavioural counts

	<b>Min</b>	<b>Max</b>	<b>Mean</b>	<b>SD</b>	<b>Z<sub>skewness</sub></b>	<b>Z<sub>kurtosis</sub></b>	<b>% zero</b>
<b>V1</b>	0	1042	8.9	61.4	114.4	960.5	56.1
<b>V2</b>	0	348	7.7	27.8	58.3	299.9	54.5
<b>V3</b>	0	1036	9.9	68.0	90.3	639.6	68.8
<b>P1</b>	0	268	3.6	19.7	75.7	454.2	72.8
<b>P2</b>	0	147	0.9	8.7	114.3	957.6	89.4
<b>P3</b>	0	1219	12.3	81.1	86.6	603.3	72.1
<b>S1</b>	0	780	6.7	50.1	95.4	700.9	73.8
<b>S2</b>	0	500	5.5	41.2	77.7	440.5	78.7

These variables were then subjected to the same logarithmic transformation:  $\log(x + 1)$ , in order to reduce the impact of high frequency counts in the data set. Again, this transformation does not alter the proportion of zeroes, but it is likely to normalise the distribution of the positive behavioural counts for each variable. This is important for regression modelling in the second part of the two-part analyses.

The eight log-transformed variables were then subjected to principal component analysis in PASW version 18.0, using promax rotation in order to allow any underlying components to correlate. When not testing specific hypotheses, PCA is an acceptable way to explore and describe a complex dataset (Costello & Osborne, 2005; Field, 2005). Concerns raised about the unusual data structure violating the assumptions of normality are not considered critical for PCA when utilised in this manner (Dudzinski et al., 1975; Duntzman, 1989; Jackson, 1991; Jolliffe, 2002).

#### **6.2.3.1.2 Using the SASBA variables**

Once the eight variables were subjected to PCA in order to replicate the previous component structure, another PCA was conducted which included a greater range of SASBA categories. Table 6.2 shows the descriptive statistics for the raw SASBA recordings. It can be seen that the proportion of zeroes is extremely high for some of these items, up to 98.7% in the case of the E4 item. As such, the decision was taken to sum each category of SASBA item instead for PCA. The proportion of zeroes for each was: Verbal Comments 73.8%; Non-Contact 87.0%; Exposure 94.4%; Touching Others 83.7%. These four SASBA categories in addition to the six BARS categories were then subjected to log transformation followed by further principle component analysis with promax rotation in order to see if the component structure continued to hold.

#### **6.2.3.2 Two-part analyses of behavioural observations**

The nature of this component structure would then provide the basis for the dependent variables subjected to the subsequent two-part regression analyses. This was the essence of the second research question. Firstly, logistic regression would be used to determine a predictive model for the *presence* of that behaviour. Secondly, linear regression would then be used with the subset of those participants showing the behaviour to model the *degree* of behavioural disturbance present. It was anticipated that the log-transformation would have normalised the distribution of all positive behavioural counts.

**Table 6.2 Distribution for raw SASBA observations**

	<b>Min</b>	<b>Max</b>	<b>Mean</b>	<b>SD</b>	<b>Z<sub>skewness</sub></b>	<b>Z<sub>kurtosis</sub></b>	<b>% zero</b>
<b>VC1</b>	0	776	4.3	45.4	118.3	1006.9	81.1
<b>VC2</b>	0	47	0.6	3.5	69.8	407.5	88.7
<b>VC3</b>	0	72	0.6	4.6	94.6	692.1	89.4
<b>VC4</b>	0	125	1.2	8.8	85.6	550.2	88.7
<b>NC1</b>	0	101	1.0	7.6	82.3	497.0	90.7
<b>NC2</b>	0	99	0.7	6.7	87.6	584.5	95.7
<b>NC3</b>	0	9	0.1	0.7	72.7	379.7	98.3
<b>NC4</b>	0	18	0.1	1.0	121.8	1048.8	99.3
<b>E1</b>	0	33	0.2	2.3	91.9	607.3	98.3
<b>E2</b>	0	61	0.2	3.5	123.6	1071.0	98.3
<b>E3</b>	0	15	0.1	1.0	86.1	589.4	96.0
<b>E4</b>	0	17	0.1	1.0	114.6	954.3	98.7
<b>TO1</b>	0	464	2.2	27.1	118.7	1009.3	89.0
<b>TO2</b>	0	29	0.3	2.0	86.0	589.6	93.4
<b>TO3</b>	0	21	0.2	1.4	87.5	636.9	93.0
<b>TO4</b>	0	47	0.3	3.0	97.7	734.0	95.7

#### 6.2.3.3. Re-analysis of MPAI-4 data

The MPAI-4 provides clinical comparison data for the three rationally-derived subscales – Abilities, Adjustment and Participation – as well as the Total score.

However, the psychometric development of the MPAI-4 had shown a different underlying factor structure (Bohac et al., 1997; Malec et al., 2003). The third research question in this study was whether an empirically derived arrangement of items would provide further information regarding the clinical correlates of verbal aggression, physical aggression and inappropriate sexual behaviour.

The 29 individual items used to derive the three subscale scores were firstly subjected to principal component analysis with orthogonal (varimax) rotation. The resulting components were then used in separate logistic regressions for verbal aggression, physical aggression and inappropriate sexual behaviour.

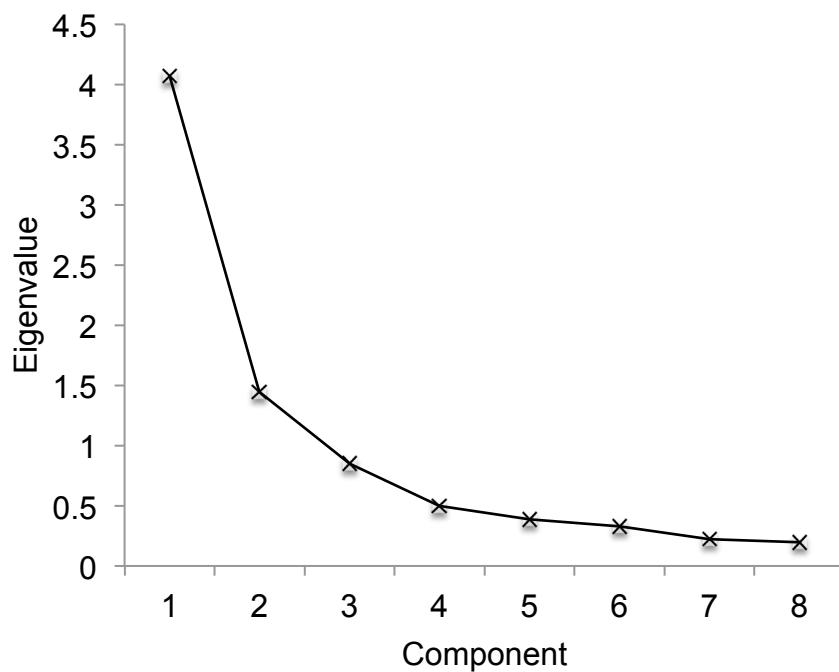
## **6.3. Results**

### **6.3.1. Principal component analysis of behavioural observations**

#### **6.3.1.1. PCA with same behavioural variables**

Principal component analysis with promax rotation of the 8 log-transformed behavioural variables (six BARS categories, S1 and S2) produced a component structure with a Kaiser-Meyer-Olkin (KMO) value of .79, which is considered acceptable (Field, 2005; Kaiser, 1974). Other measures of the adequacy of using this technique included a highly significant Bartlett's test ( $p < .001$ ), all diagonal items of the anti-image correlation matrix were above .5 and only 28% of non-redundant residuals had absolute values greater than .05 (Field, 2005).

According to the “rough guide” (Hutcheson & Sofroniou, 1999) of using eigenvalues and the screeplot to determine the optimal number of components to retain, three components again were produced by the principal component analysis. Figure 6.2 below shows the resulting screeplot, which is less clear than the previous study (Chapter 5). From this plot two, three or even four components may meet this visual inflexion point criterion.



**Figure 6.2** Eigenvalues after PCA with same variables

However, again using the less conservative eigenvalue criterion of .7 (Jolliffe, 2002) rather than the more traditional 1.0, it was clear that 3 components should be retained. A three component model explained 79.6% of the variance, while retaining only two components explained only 69.0%. It was therefore decided that 3 components again fit this data structure best and the resulting pattern matrix from retaining three components is presented in Table 6.3 below. Any loadings smaller

than .400 are not included in this table. This pattern loading is nearly identical to that in the previous study, providing strong evidence that the underlying structure of behavioural observations was essentially the same.

**Table 6.3 Pattern matrix for three-component structure of the same behavioural observations**

	Component		
	1	2	3
logV1	.988		
logV2	.688		
logV3	.865		
logP1		.698	
logP2		.974	
logP3		.810	
logS1			.948
logS2			.933

### 6.3.1.2. PCA with additional SASBA items

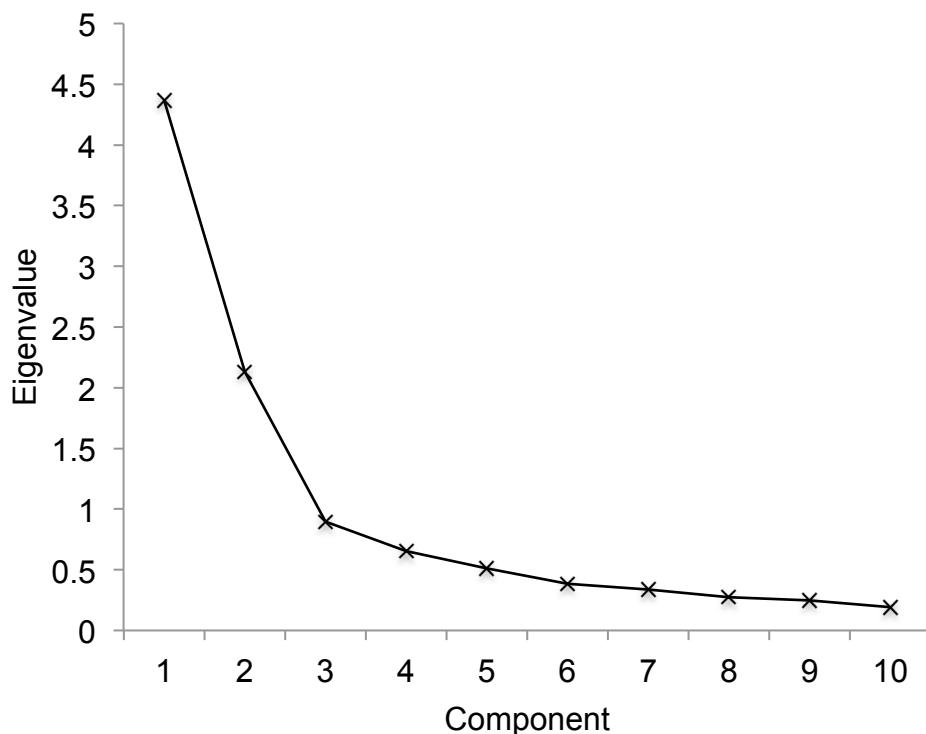
It was clear that, when the SASBA data were divided into the same two categories of inappropriate sexual behaviour as the previous study, the same three components emerged. However, the SASBA provides more subtle differentiation of behaviours than a simple dichotomy of verbal comments (S1) and physical actions (S2). There are four categories with four levels of severity, providing a 16 item matrix. It was therefore considered worthwhile to re-examine the behavioural data structure, this time including a greater range of inappropriate sexual behaviours. The major concern regarding this was the exceedingly high proportion of zeroes among the 16 items. From Table 6.3 above, it can be seen that for 11 out of the 16 items (68.8%) the proportion of zeroes exceeds 90%.

Although it is not crucial that the data utilised in principal component analysis be normally distributed (Dudzinski et al., 1975; Dunteman, 1989; Jackson, 1991; Jolliffe, 2002), such high frequency zero scores warrant caution. There appears to be no specific guidance within the relevant literature for this particular challenge. It was therefore decided to minimise the effect of the frequent zeroes while still expanding the items of inappropriate sexual behaviour to be included in a further principal component analysis. This was done by summing each severity score within each SASBA category, that is, creating a Verbal Comments item which summed VC1, VC2, VC3 and VC4. This was also performed for the other three SASBA categories of Non-Contact, Exposure and Touching Others. Such an approach resulted in only one out of four categories having a proportion of zeroes exceeding 90%: Verbal Comments (73.8%); Non-Contact (87.0%); Exposure (94.4%); Touching Others (83.7%). These four SASBA items were then subjected to principal component analysis with the six BARS items (V1, V2, V3, P1, P2, P3).

The component structure that emerged from this analysis produced a Kaiser-Meyer-Olkin (KMO) value of .81 and a highly significant Bartlett's test ( $p < .001$ ). The diagonal items of the anti-image correlation matrix were all well above the recommended .5 and only 37% of non-redundant residuals had an absolute value in excess of .05.

Although an additional two items of inappropriate sexual behaviour were included in this analysis, the same component structure was revealed. A very clear inflexion point was evident at component 3 in the screeplot, which is presented in Figure 6.3

below. Three components were also retained with eigenvalues greater than 0.7, explaining 74.0% of the variance.



**Figure 6.3** Eigenvalues after principal component analysis with six BARS and four SASBA behavioural count variables

The log-transformed behavioural count items again loaded neatly onto the three components of verbal aggression, physical aggression and inappropriate sexual behaviour. The resulting pattern matrix is shown in Table 6.4 below. As the three component structure of the behavioural observation data was again borne out even with the additional SASBA items, it was determined that the subsequent multivariate modelling of the behavioural data would be differentiated again between verbal aggression, physical aggression and inappropriate sexual behaviour.

**Table 6.4 Pattern matrix (loadings) for three-component structure of the 10 log-transformed behavioural count items with promax rotation**

	Component		
	1	2	3
logV1		.945	
logV2		.748	
logV3		.827	
logP1			.663
logP2			.939
logP3			.792
logVC	.780		
logNC	.910		
logE	.819		
logTO	.797		

Given that there was already a precedent for using a weighted algorithm for both the BARS Irritability/Aggression Index (Freeland et al., 2012) and the SASBA Aggregate Score (N. Alderman, personal communication, 20<sup>th</sup> June, 2012), weighted equations were used for verbal aggression (see Equation 2 above), physical aggression (see Equation 3 above) and inappropriate sexual behaviour (see Equation 4 above). Raw behavioural counts were used for the equations and then the resulting value subjected to  $\log_{10}$  transformation, i.e.,  $y = \log(x + 1)$ . Again, such transformation does not alter the proportion of zeroes and does not, therefore, influence logistic regression modelling of presence versus absence of behavioural disturbance. However, such a transformation has the benefit of altering the non-normally distributed positive behavioural counts into a more normalised distribution, which is essential for linear regression modelling.

In order to determine if using the weighted equations was an appropriate method with which to proceed, rather than simply using summed items as with the previous

study, simple parametric correlations were performed. The Pearson correlations between weighted and summed variables were as follows: verbal aggression  $r = .995$ ; physical aggression  $r = .997$ ; inappropriate sexual behaviour  $r = .918$ . Parametric correlation has previously been considered appropriate with data involving excess zeroes (Huson, 2007). However, as Huson's work involved datasets with the proportion of zeroes not exceeding 30%, non-parametric Spearman correlations were also calculated: verbal aggression  $r_s = .997$ ; physical aggression  $r_s = .998$ ; inappropriate sexual behaviour  $r_s = .996$ .

All correlations (both parametric and non-parametric) were highly significant ( $p < .001$ ) and it was therefore felt that either method would be valid. It was decided to utilise the weighted variables as this appears to be the most commonly used method within the clinical setting.

### **6.3.2. Two-part multivariate modelling of behavioural variables**

The three final behavioural variables (weighted and log-transformed) were firstly subjected to logistic regression modelling for the *presence* of behavioural disturbance. The second part of the two-part model process required linear regression modelling for the *degree* of behavioural disturbance when present.

### **6.3.2.1. Final set of predictor variables**

Before the two-part regression modelling of the dependent variables could be attempted, a final set of predictor variables was required. Two variables still required clarification: the type of brain injury and Supervision Rating Scale score.

#### *6.3.2.1.1 Type of brain injury*

The type of brain injury had been initially encoded as a categorical variable with four groups (traumatic brain injury, cerebro-vascular accident, cerebral anoxia and other). Now that the dependent behavioural variables had been determined, a question arose regarding this variable. Should it be included in the modelling via dummy variables or would it be better to recode as a dichotomous variable – traumatic brain injury versus non-traumatic brain injury – as TBI was the most common type of injury in this sample? Table 6.4 below shows the percentage of each type of injury showing some recordings for each behavioural outcome variable. It can be seen clearly from this table that those participants with traumatic brain injury showed the highest presence of verbal aggression, physical aggression and inappropriate sexual behaviour. Based on this, it was decided that a dichotomous variable (TBI versus non-TBI) should be included in the regression modelling. This would allow the multivariate testing of, when controlling for other influencing variables, whether a participant having a traumatic brain injury (as opposed to any other injury) significantly increases the risk of behavioural disturbance.

**Table 6.5 Percentage of sample showing behaviours for each injury type**

Injury Type	Verbal Aggression	Physical Aggression	Inappropriate Sexual Behaviour
Traumatic brain injury (n = 167)	69.5%	45.5%	37.7%
Cerebro-vascular accident (n = 66)	51.5%	30.3%	25.8%
Cerebral anoxia (n = 34)	41.2%	23.5%	26.5%
Other (n = 33)	27.3%	18.2%	12.1%

It is also clear from Table 6.5 that, within each type of injury, the same pattern of relative frequency of each behavioural disturbance is present. Specifically, for all types of injury, *some* verbal aggression was observed in more participants than *some* physical aggression, which in turn was observed in more participants than *some* inappropriate sexual behaviour.

A similar comparison was performed on all non-zero behavioural observations and can be seen in Table 6.6 below. The pattern evident above is not repeated here; while those participants with traumatic brain injury showed the highest *degree* of verbal aggression, it was those with cerebral anoxia that exhibited the highest *degree* of physical aggression and inappropriate sexual behaviour.

However, Kruskall-Wallis tests showed no differences between groups on non-zero log-transformed verbal aggression ( $H_{(3)} = 5.59, p > .05$ ), physical aggression ( $H_{(3)} = 6.70, p > .05$ ) or inappropriate sexual behaviour ( $H_{(3)} = 0.23, p > .05$ ) scores. Therefore, continuing to utilise a dichotomous grouping of traumatic brain injury versus non-traumatic brain injury was considered the most appropriate way to

include type of injury into the regression modelling of non-zero behavioural component scores.

**Table 6.6** Mean and standard deviation of log-transformed non-zero behavioural values for each type of injury

Injury Type	Verbal Aggression (n = 173)	Physical Aggression (n = 110)	Inappropriate Sexual Behaviour (n = 93)
Traumatic brain injury	1.41 (.68) (n = 116)	1.52 (.82) (n = 76)	1.39 (.62) (n = 63)
Cerebro-vascular accident	1.19 (.59) (n = 34)	1.43 (.58) (n = 20)	1.37 (.80) (n = 17)
Cerebral anoxia	1.30 (.87) (n = 14)	2.03 (.48) (n = 8)	1.45 (.93) (n = 9)
Other	.98 (.56) (n = 9)	1.23 (.59) (n = 6)	1.38 (.72) (n = 4)

#### 6.3.2.1.2 Supervision Rating Scale

The Supervision Rating Scale (Boake, 1996) utilises 13 hierarchical categories, which can be reduced to five levels. These are: independent, overnight supervision, part-time supervision, full-time indirect supervision and full-time direct supervision. Subsequent work (Hart et al., 2003) has also used a three level categorisation. However, for the current study, the raw scores on the Supervision Rating Scale were used.

### **6.3.2.2. Correlations between variables**

#### *6.3.2.2.1 Inter-correlations between independent variables*

The 15 independent variables were grouped according to their nature: six were considered pre-injury (sex, education, history of prior brain injury, psychiatric history, criminal history of aggression, substance abuse history); three were related to the injury itself (type of injury, age at injury, chronicity); six were measures of current function (MPAI-4 Ability, MPAI-4 Adjustment, MPAI-4 Participation, MPAI-4 Total, Care and Needs Scale, Supervision Rating Scale). Parametric correlations between these 15 independent variables are shown in Table 6.7 below

Of particular note from this table is that there is a tendency for the premorbid psychosocial and injury-related variables to produce significantly high inter-correlations amongst themselves and a distinct lack of association with measures of current neurobehavioural function and care/supervision requirements. Similarly, the latter variables showed high-intercorrelations between themselves only. These are discussed below.

**Table 6.7** *Inter-correlations between independent variables*

	Sex	Educ	PriorB	Psych	PriorA	SA	Type	Age	Chron	Ability	Adjust	Part	Total	CANS	SRS
Sex	-														
Education	.09	-													
Prior ABI	-.13*	-.11	-												
Psych history	.08	-.09	.17**	-											
Prior aggression	-.17**	-.19**	.15**	.17**	-										
Substance abuse	-.06	-.30**	.25**	.26**	.32**	-									
Injury type	.24**	.08	.01	.07	-.13*	-.12*	-								
Age at injury	.16**	.14*	.11	.07	-.10	.09	.42**	-							
Chronicity	-.17**	-.01	-.14*	-.11	.05	-.05	-.23**	-.37**	-						
MPAI Ability	.01	-.04	.08	-.08	-.01	-.05	.04	.08	-.01	-					
MPAI Adjustment	-.07	-.05	.01	-.08	.07	.07	-.05	-.01	.13*	.58**	-				
MPAI Participation	-.01	-.03	.08	-.02	-.04	-.02	.16**	.23**	-.02	.66**	.62**	-			
MPAI Total	-.03	-.06	.06	-.08	.02	.01	.03	.09	.05	.88**	.86**	.80**	-		
CANS	.07	-.08	-.03	-.07	-.05	-.06	.05	.06	-.03	.46**	.48**	.54**	.57**	-	
SRS	-.04	-.01	.02	.05	-.03	.05	.13*	.20**	-.13*	.31**	.31**	.44**	.41**	.47**	-

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

There were a number of significant associations with sex. For instance, males were more likely than females to have had a prior brain injury ( $\chi^2_{(1)} = 5.30, p < .05$ ), a premorbid criminal conviction for violence ( $\chi^2_{(1)} = 8.67, p < .01$ ), sustained a traumatic brain injury ( $\chi^2_{(1)} = 16.78, p < .001$ ) and to have been admitted at least six months post-injury ( $\chi^2_{(1)} = 8.48, p < .01$ ). Males were also typically younger than females when they were injured ( $t_{(299)} = -2.76, p < .01$ ). No sex differences were evident in educational group, having a pre-existing psychiatric diagnosis or history of substance abuse, any of the MPAI-4 subscales, the CANS or SRS scores.

In addition to being related to male sex, participants having sustained a traumatic brain injury was also significantly correlated with having had a prior brain injury ( $\chi^2_{(1)} = 5.30, p < .05$ ), a history of substance abuse ( $\chi^2_{(1)} = 5.30, p < .05$ ), younger age when injured ( $t_{(299)} = -2.76, p < .01$ ) and being admitted at least six months post-injury ( $\chi^2_{(1)} = 5.30, p < .05$ ). Having sustained a traumatic brain injury was also associated with better MPAI-4 Participation scores ( $t_{(299)} = -2.76, p < .01$ ) and less required supervision on the SRS ( $t_{(299)} = -2.76, p < .01$ ) than having had a non-traumatic brain injury. Of these differences, only a history of substance abuse ( $r = -.17, p < .01$ ) remained significant once the correlations were controlled for age at injury.

Given the very high correlations between the MPAI-4 scores for Total and Ability, Adjustment, Participation ( $r = .88, .86$  and  $.80$  respectively), the potential for multicollinearity in subsequent regression models was high. When using the three MPAI-4 subscale and Total scores in a linear regression analysis predicting life satisfaction after brain injury, Jacobsson et al. (2011) dealt with the threat of

multicollinearity by running the regression four separate times, including one MPAI-4 variable each time in the list of predictors. Given the number of regression models already planned in the current study, it was felt that the best solution would be to respecify the model without the MPAI-4 Total score, as it was comprised of the other three MPAI-4 scores.

### 6.3.2.2.2 *Correlations between independent and dependent variables*

Parametric correlations were also calculated between the final 14 independent (or predictor) variables and the dependent variables of verbal aggression, physical aggression and inappropriate sexual behaviour. Table 6.8 shows separate correlations calculated on the data set for presence/absence and on non-zero cases within each behavioural component.

**Table 6.8 Correlations between predictors and two-part outcome variables**

Predictor variables	Verbal aggression		Physical aggression		Inappropriate sexual behaviour	
	group n = 301	nonzero n = 173	group n = 301	nonzero n = 110	group n = 301	nonzero n = 93
<b>Pre-injury</b>						
Sex	.28**	.14	.22**	.03	.21**	.11
Education	.11	.20**	.06	.08	.06	.02
Prior ABI	.05	.05	.01	.10	.02	-.12
Psychiatric history	-.07	.01	-.03	.15	-.10	.07
Violence conviction	.04	.04	.04	.03	-.02	.17
Substance misuse	.11	.17*	.08	-.04	.09	-.10
<b>Injury</b>						
Injury type	.27**	.16*	.21**	.01	.17**	.00
Age at injury	-.20**	-.05	-.17**	-.01	-.19**	.02
Chronicity	.23**	.14	.23**	.05	.15*	.23*
<b>Current function</b>						
MPAI-4 Ability	.13*	.19*	.17**	.45**	.02	.15
MPAI-4 Adjustment	.36**	.37**	.37**	.42**	.18**	.38**
MPAI-4 Participation	.10	.29**	.18**	.49**	.04	.18
CANS	.17**	.20**	.22**	.35**	.14*	.16
SRS	.13*	.18*	.15**	.29**	.06	.12

\* p < .05. \*\* p < .01.

It can be seen that, with the exception of sex, the pre-injury variables were largely unrelated to the behavioural dependent variables. All three injury-related variables were significantly related to the behavioural dichotomous groupings and much less so with the non-zero log-transformed behavioural values. The majority of the included measures of neurobehavioural function and care/supervision requirements were significantly related to behavioural outcomes.

### **6.3.2.3. Logistic regression modelling of presence/absence**

As per the two-part model for datasets with excessive zero counts, the first stage is to model the *presence* or *absence* of the particular behavioural disturbance via logistic regression. The 14 predictor variables were used in a hierarchical forced entry method, using three blocks: pre-injury, injury and current function. The same method was applied for each behavioural component grouping and the results will be discussed separately below.

#### **6.3.2.3.1 Verbal aggression**

The null model for verbal aggression, namely that every participant showed some verbal aggression, correctly classified 57.5% of cases. Following the first step (pre-injury variables only), sex ( $p < .001$ ) was the only significant predictor and the classification rose to 65.4%. After the second step (adding injury variables), sex ( $p < .001$ ), type of injury ( $p < .05$ ) and chronicity ( $p < .05$ ) were significant predictors and the classification rose further to 68.8%.

Following completion of the third step (adding current function variables), this final model was highly significant ( $\chi^2_{(14)} = 102.49$ ,  $p < .001$ ) and accounted for 38.8% of the variance in the verbal aggression dichotomous grouping. The correct classification of participants was 76.1%. Male sex (increasing the odds approximately three-fold;  $p < .01$ ), having had a traumatic brain injury (increasing the odds approximately two-fold;  $p < .05$ ), chronicity of 6 months or more (increasing the odds approximately two-fold;  $p < .05$ ), higher MPAI-4 Adjustment scores (with higher scores reflecting poorer adjustment;  $p < .001$ ) and lower MPAI-4 Participation (with lower scores reflecting greater social participation;  $p < .05$ ) were all significant predictors of the presence of verbal aggression. The final model for verbal aggression is presented below in Table 6.9, which also includes the final models for physical aggression and inappropriate sexual behaviour, in order to facilitate comparisons between these models.

Examination of residuals revealed that there were only 5 cases (2%) with an absolute standardised residual value greater than 2. There were no particularly large residuals that might indicate outlying participants. DFBeta values (measures of the influence of a case on the value of  $b$ ) for all predictors were less than 1 (maximum value .11). There were no problematically high values of Cook's distance (maximum value .45) and, although around 40% of leverage values were greater than expected ( $((k + 1)/N = ((14 + 1)/301) = .0498$ ), Field (2005) suggests that this is unlikely to be concerning given that the other residual statistics were acceptable.

**Table 6.9 Results of logistic regression analyses modelling presence/absence of behavioural disturbance**

	B (SE)	95% CI for exp b			
		Lower	exp b	Upper	
<b>Verbal aggression</b>					
$R^2 = .27$ (H & L), .29 (C & S), .39 (N); Model $\chi^2(14) = 102.49, p < .001$ .					
Constant	-7.43*** (1.35)	-	0.01	-	
Sex	1.12** (0.36)	1.52	3.07	6.23	
Education	0.35 (0.33)	0.74	1.42	2.71	
Prior ABI	0.44 (0.44)	0.66	1.56	3.65	
Psychiatric history	-0.23 (0.37)	0.38	0.79	1.64	
Prior violence conviction	-0.86 (0.52)	0.15	0.42	1.17	
Substance abuse	0.33 (0.36)	0.70	1.38	2.72	
Injury type	0.74* (0.31)	1.14	2.10	3.85	
Age at brain injury	-0.01 (0.01)	0.97	0.99	1.01	
Chronicity	0.64* (0.31)	1.04	1.91	3.49	
MPAI-4 Ability	-0.01 (0.02)	0.95	0.99	1.03	
MPAI-4 Adjustment	0.15*** (0.03)	1.10	1.16	1.23	
MPAI-4 Participation	-0.05* (0.02)	0.91	0.95	0.99	
Care and Needs Scale	0.11 (0.12)	0.88	1.11	1.41	
Supervision Rating Scale	0.14 (0.07)	0.99	1.15	1.33	
<b>Physical aggression</b>					
$R^2 = .22$ (H & L), .24 (C & S), .33 (N); Model $\chi^2(14) = 83.62, p < .001$ .					
Constant	-8.31*** (1.34)	-	0.00	-	
Sex	1.09* (0.42)	1.30	2.97	6.79	
Education	-0.08 (0.32)	0.49	0.92	1.72	
Prior ABI	0.02 (0.43)	0.44	1.02	2.35	
Psychiatric history	0.20 (0.38)	0.58	1.23	2.57	
Prior violence conviction	-0.44 (0.48)	0.25	0.64	1.65	
Substance abuse	0.35 (0.33)	0.73	1.41	2.72	
Injury type	0.57 (0.32)	0.95	1.77	3.30	
Age at brain injury	-0.01 (0.01)	0.97	0.99	1.01	
Chronicity	0.69* (0.30)	1.10	1.98	3.58	
MPAI-4 Ability	-0.01 (0.02)	0.95	0.99	1.03	
MPAI-4 Adjustment	0.11*** (0.03)	1.06	1.12	1.17	
MPAI-4 Participation	-0.01 (0.02)	0.95	0.99	1.03	
Care and Needs Scale	0.20 (0.13)	0.95	1.22	1.56	
Supervision Rating Scale	0.10 (0.08)	0.95	1.10	1.28	
<b>Inappropriate sexual behaviour</b>					
$R^2 = .15$ (H & L), .15 (C & S), .22 (N); Model $\chi^2(14) = 49.90, p < .001$ .					
Constant	-3.56** (1.11)	-	0.03	-	
Sex	1.33** (0.44)	1.60	3.77	8.90	
Education	-0.15 (0.31)	0.47	0.86	1.59	
Prior ABI	0.25 (0.42)	0.56	1.28	2.93	
Psychiatric history	-0.51 (0.40)	0.28	0.60	1.31	
Prior violence conviction	-0.85 (0.50)	0.16	0.43	1.14	
Substance abuse	0.71* (0.33)	1.06	2.03	3.88	
Injury type	0.20 (0.31)	0.66	1.22	2.24	
Age at brain injury	-0.02* (0.01)	0.96	0.98	1.00	
Chronicity	0.23 (0.30)	0.70	1.26	2.26	
MPAI-4 Ability	-0.03 (0.02)	0.93	0.97	1.01	
MPAI-4 Adjustment	0.05* (0.02)	1.01	1.05	1.10	
MPAI-4 Participation	-0.01 (0.02)	0.95	0.99	1.03	
Care and Needs Scale	0.29* (0.02)	1.04	1.34	1.71	
Supervision Rating Scale	0.03 (0.07)	0.90	1.03	1.19	

H & L = Hosmer & Lemeshow; C & S = Cox & Snell; N = Nagelkerke.

\*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$ .

In order to examine the possibility of multicollinearity, the same predictors and independent variable were entered into a standard linear regression analysis. This provided values for tolerance and variance inflation factor (VIF), for which a “commonly used rule of thumb” (p.423, Cohen, Cohen, West, & Aiken, 2003), is that scores less than .10 or greater than 10 (respectively) are evidence of serious multicollinearity.

No independent variables came close to these criteria, with the lowest tolerance score .38 and highest VIF of 2.63 (both for MPAI-4 Participation). Examination of the smallest eigenvalues produced by PASW 18.0 collinearity diagnostics did not reveal any problematic variables. Therefore, it was concluded that this logistic regression model for presence of verbal aggression provided a good fit for the data. As the same independent variables were to be used for the subsequent two logistic regression models for physical aggression and inappropriate sexual behaviour, multicollinearity was not re-examined.

#### 6.3.2.3.2 *Physical aggression*

The null model (that no participants showed any physical aggression) correctly classified 63.5% of cases. After entering in the first block of predictors, the first model’s accuracy improved to 66.4% with sex ( $p < .001$ ) emerging as the only significant predictor. Model 2, incorporating both the initial pre-injury (block 1) and injury-related (block 2), correctly classified 67.8% with sex ( $p < .01$ ) and chronicity ( $p < .01$ ) both significant. The final model (including the variables of current function)

had classification accuracy of 74.4% with significant predictors of sex ( $p < .05$ ), chronicity ( $p < .05$ ) and MPAI-4 Adjustment ( $p < .001$ ).

The values of  $\exp b$  are shown above in Table 6.9. For the physical aggression analysis, male sex increased the odds of some physical aggression having been recorded by approximately 3 and having had an injury more than 6 months ago increased the odds by almost 2. Higher scores on the MPAI-4 Adjustment subscale (relating to poorer adjustment) also indicated a greater likelihood of physical aggression.

Examination of the residuals following logistic regression for presence of physical aggression showed that only 5 cases (2%) had a standardised residual greater than 2. Two participants had particularly high standardised residuals (both predicted as “none” but with “some” physical aggression observed) of 6.43 and 5.75. Closer examination of these particular cases showed that although they both differed in sex and chronicity, both had particularly low scores on the MPAI-4 Adjustment subscale (T-scores of 35 and 38, respectively) indicating that they had few of these difficulties. Although both of these outlying cases were observed to have shown some physical aggression, one exhibited only one episode of non-directed behaviour (P1 on the BARS) and the other two episodes of behaviour directed towards others (P3 on the BARS).

DFBeta values for all predictors were less than 1 (maximum value .16). There were no problematically high values of Cook’s distance (maximum value .60) and approximately 42% of leverage values were greater than expected ( $(k + 1)/N = ((14$

$+ 1)/301 = .0498$ ). Again, this indicated that the logistic regression model for physical aggression was a good fit with the data.

#### 6.3.2.3.3 *Inappropriate sexual behaviour*

The null model for this behaviour, namely that no participant showed any inappropriate sexual behaviour, correctly classified 69.1% of cases. Following the first step (pre-injury variables only), sex ( $p < .001$ ) was the only significant predictor and the classification dropped to 67.4%. After the second step (adding injury variables), sex ( $p < .01$ ), a history of substance abuse ( $p < .05$ ) and age at injury ( $p < .05$ ) were significant predictors and the classification rose back to same value as the null model at 69.1%.

The final model correctly classified 70.1% of participants; only a marginal improvement on the null model. Significant predictors in the final model were sex ( $p < .01$ ), a history of substance abuse ( $p < .05$ ), age at injury ( $p < .05$ ), MPAI-4 Adjustment ( $p < .05$ ) and CANS ( $p < .05$ ).

The values of  $\exp b$  are shown above in Table 6.9. For this analysis, male sex increased the odds of some inappropriate sexual behaviour having been recorded by almost 4 and having had a history of substance abuse increased the odds by 2. Higher scores on the MPAI-4 Adjustment subscale (relating to poorer adjustment) and higher CANS ratings also indicated a greater likelihood of inappropriate sexual behaviour having been documented.

Examination of residuals revealed that there were only 5 cases (2%) with an absolute standardised residual value greater than 2. There were no particularly large residuals that might indicate outlying participants. DFBeta values for all predictors were less than 1 (maximum value 0.20). There were no problematically high values of Cook's distance (maximum value 0.49) and around 39% of leverage values were greater than expected ( $((k + 1)/N = ((14 + 1)/301) = 0.0498)$ ). Therefore, although the classification accuracy was only marginally better than the null model for inappropriate sexual behaviour, the final model provided a reasonably good fit for the data.

#### **6.3.2.4. Linear regression modelling of non-zero values**

The second part of the two-part regression model involves linear regression of the non-zero values for each behaviour. Values had been log-transformed in an attempt to normalise the distribution of these non-zero values and can be seen below in Table 6.10. However, it is clear from the rightmost column (showing significant Kolmogorov-Smirnov test statistics) that true normalisation was not achieved in any of the three subsamples. Despite all three distributions showing no significant kurtosis, they remained significantly positively skewed. Nevertheless, these transformed non-zero scores were used as the dependent variables for subsequent linear regression analyses. Histograms for these transformed scores can be seen in Appendix 1.

**Table 6.10 Descriptive statistics for log-transformed weighted non-zero values for each behaviour**

	<i>n</i>	min.	max.	mean	<i>SD</i>	<i>Z<sub>skewness</sub></i>	<i>Z<sub>kurtosis</sub></i>	K-S
Verbal aggression	173	0.30	3.62	1.33	0.68	3.04**	0.01	.08**
Physical aggression	110	0.48	3.92	1.52	0.76	3.04**	0.01	.09*
Inappropriate sexual behaviour	93	0.30	3.53	1.39	0.68	3.08**	1.71	.10*

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

Importantly, the number of participants for each non-zero subsample was now substantially reduced from the total sample of 301. When considering an adequate sample size for linear regression models, both the number of predictor variables and the effect sizes involved are crucial. As effect sizes for this area of research have been medium at best, recent recommendations (Miles & Shevlin, 2001) would suggest that, for an acceptable level of power (.80), a model with 14 predictors of medium effect size would require a sample size of between 150 and 200 participants. Consequently, the subsample size of 173 for verbal aggression would be acceptable. However, the smaller subsamples for physical aggression and inappropriate behaviour would not meet this requirement.

Taking the smallest of the three subsamples (non-zero inappropriate sexual behaviour  $n = 93$ ), the guidance from Miles & Shevlin (2001) would be to limit the number of predictors to a maximum of 5. For consistency, the same predictor variables would need to be used in all three models. Therefore, the original 14 predictor variables were entered into a regression model for verbal aggression with a backwards stepwise method. The significant individual predictors remaining after this

process (criterion for removal  $p < .10$ ) could then be utilised for the regression models for both physical aggression and inappropriate sexual behaviour.

#### 6.3.2.4.1 *Verbal aggression*

Verbal aggression was exhibited by 173 (57.5%) of the participants. The initial model, containing the same 14 predictor variables as the previous logistic regression models, was highly significant ( $F_{(14,158)} = 3.97, p < .001$ ) although only type of injury ( $p < .05$ ) and MPAI-4 Adjustment ( $p < .01$ ) were significant individual predictors of the degree of verbal aggression. This initial model explained around a quarter of the variance in non-zero verbal aggression scores ( $R^2 = .26$ , adjusted  $R^2 = .19$ ).

Following the backwards stepwise procedure, a final model was derived that was also highly significant ( $F_{(4,168)} = 11.30, p < .001$ ) and contained four predictor variables that remained significant at  $p < .10$ . Higher levels of recorded verbal aggression were associated with not having a GCSE level of education ( $p < .05$ ), having sustained a traumatic brain injury ( $p < .01$ ) and higher scores on MPAI-4 Adjustment ( $p < .01$ ), reflecting poorer adjustment, and Participation ( $p < .10$ ), reflecting less social participation, subscales. This final model still explained around a fifth of the variance in non-zero verbal aggression scores ( $R^2 = .21$ , adjusted  $R^2 = .19$ ) and produced a Durbin-Watson statistic of 2.05, well within acceptable limits (Field, 2005), confirming the independence of errors in the model.

Multicollinearity assumptions were previously discussed with regard to regression models involving this particular set of 14 independent variables. With the reduction in

sample size and predictors in the final model, these were rechecked. Lowest tolerance was .51 and highest VIF 1.95 (MPAI-4 Participation), well within acceptable limits (Cohen et al., 2003; Field, 2005).

Outlier analysis for this final model revealed that nine participants (5.2%) had an absolute standardised residual greater than 2.0 and that two (1.2%) were greater than 2.5, which is as expected from chance. One case produced a standardised residual of 3.51 and required closer scrutiny. This participant was the most verbally aggressive in this sample (2078 incidents of verbal aggression on the BARS, which produced a log-transformed weighted verbal aggression score that was 3.38 standard deviations greater than the mean). They had less than a GSCE education and had sustained a non-traumatic brain injury. The MPAI-4 Adjustment score ( $T = 59$ ) and particularly the Participation score ( $T = 74$ ) were relatively high when compared with the clinical comparison group.

Cohen et al. (2003) provide recommendations and cut-off values for a number of case statistics when performing regression diagnostics. These include centred leverage (a measure of extremity on the independent variables), *DFFIT* (measuring the change in predicted *Y* if that case was removed), Cook's *D* (measuring the influence of a particular case across the set of *Bs*) and *DFBETA* (measuring the influence of a particular case on a specific *B*).

Three (1.7%) cases had a centred leverage value greater than 0.069 ( $3k/n$  where  $k =$  number of predictors and  $n =$  sample size), within the 5% expected by chance. No

case was deemed unduly influential as values for Cook's Distance, *DFBETA* and *DFFIT* were all well below the cut-off of 1.0.

When assessing the fit of a regression model, it is also recommended that the plot of standardised residuals versus predicted values is visually inspected (Field, 2005). This addresses the assumption of homoscedacity, which holds that the residual values have constant variance at each point along the predictor variables. The plot of standardised residuals versus predicted values following the linear regression model of non-zero verbal aggression scores bore some resemblance to the "shape of a funnel" (Field, 2005, p. 203), which is indicative of heteroscedacity. This occurs when there is increasing variance across the residuals and is a violation of the assumption of homoscedacity. Further examination of the relationship of each independent variable with the residuals was therefore warranted.

Serial Levene tests confirmed that there were no significant differences in variance of standardised residuals for either the dichotomous variables of education group ( $F_{(1,171)} = 0.00, ns$ ) or type of injury ( $F_{(1,171)} = 0.02, ns$ ). Following the recommendation of Cohen et al. (2003), both of the continuous independent variables were then subjected to a median split ("low" versus "high" groups) so that a Levene test could be applied to these variables as well. These also revealed no significant between-group differences in the variance MPAI-4 Adjustment ( $F_{(1,171)} = 2.63, ns$ ) and MPAI-4 Participation ( $F_{(1,171)} = 1.76, ns$ ).

Visual inspection of the plots (see Appendix 2) of each individual predictor variable against the dependent variable of log-transformed weighted verbal aggression

revealed no obvious source of inconstant variance, although MPAI-4 Adjustment may have had some decreasing variance with higher scores. Given that Cohen et al. (2003) advise that remedial action for heteroscedacity is only necessary if the problem is “large” (p. 146), no further steps were taken.

Finally, the assumption of normality of residuals may be examined with a histogram and normal probability plot of the standardised residuals (see Appendix 2). Neither these plots nor a Kolmogorov-Smirnov test ( $D_{(173)} = 0.06$ , ns) indicated problems with the assumption of normality of residuals. The coefficients for this final model of verbal aggression are presented below in Table 6.11.

#### 6.3.2.4.2 *Physical aggression*

Physical aggression was recorded in 110 (36.5%) of the participants. The four predictor variables were entered into a linear regression model for this physically aggressive subsample, which was highly significant ( $F_{(4,105)} = 10.58$ ,  $p < .001$ ) and accounted for a little more than a quarter of the variance ( $R^2 = .29$ ; adjusted  $R^2 = .26$ ). The model produced a Durbin-Watson statistic of 2.24, within acceptable limits (Field, 2005), confirming the independence of errors. The only significant individual predictor was MPAI-4 Participation ( $p < .001$ ), in which higher scores (reflecting less social participation) were associated with higher levels of physical aggression.

**Table 6.11 Predictor coefficients for final model linear regression analyses predicting log-transformed non-zero behavioural values**

	B	SE B	$\beta$
<b>Verbal aggression (n = 173)</b>			
$R^2 = .21$ , adjusted $R^2 = .19$			
Constant	-0.21	0.32	-
Education	-0.24	0.10	-.17*
Injury type	-0.28	0.10	-.19**
MPAI-4 Adjustment	0.02	0.01	.27**
MPAI-4 Participation	0.01	0.01	.17
<b>Physical aggression (n = 110)</b>			
$R^2 = .29$ , adjusted $R^2 = .26$			
Constant	-0.87	0.43	-
Education	-0.18	0.13	-.12
Injury type	-0.24	0.14	-.14
MPAI-4 Adjustment	0.01	0.01	.14
MPAI-4 Participation	0.03	0.01	.45***
<b>Inappropriate sexual behaviour (n = 93)</b>			
$R^2 = .15$ , adjusted $R^2 = .11$			
Constant	-0.22	0.47	-
Education	0.05	0.14	.04
Injury type	-0.03	0.15	-.02
MPAI-4 Adjustment	0.03	0.01	.42**
MPAI-4 Participation	-0.01	0.01	-.07

\*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$ .

There were no concerns with multicollinearity as the lowest tolerance and highest VIF values were identical to the model for verbal aggression. Outlier analysis for this final model revealed that six participants (5.4%) had an absolute standardised residual greater than 2.0 and that only one (0.9%) was greater than 2.5, which is as expected from chance. No cases had a standardised residual above 3.0.

Three (2.7%) cases had a centred leverage value greater than  $0.109$  ( $3k/n$  where  $k$  = number of predictors and  $n$  = sample size), within the 5% expected by chance. No case was deemed unduly influential as values for Cook's Distance,  $DFBETA$  and  $DFFIT$  were all well below the cut-off of 1.0.

Again the plot of standardised residuals versus predicted values bore some resemblance to the “shape of a funnel” (Field, 2005, p. 203), which is indicative of heteroscedacity. Serial Levene tests confirmed that there were no significant differences in variance of standardised residuals for either the dichotomous variables of education ( $F_{(1,108)} = 2.01$ , ns), type of injury ( $F_{(1,108)} = 1.21$ , ns), or MPAI-4 Adjustment ( $F_{(1,108)} = 2.63$ , ns). However, MPAI-4 Participation ( $F_{(1,108)} = 6.17$ ,  $p < .05$ ) did show a significant difference, which can be seen in Appendix 3.

Neither the histogram and normal probability plot of the standardised residuals nor a Kolmogorov-Smirnov test ( $D_{(110)} = 0.07$ , ns) indicated problems with the assumption of normality of residuals. These can also been seen in Appendix 3. The coefficients for this final model of physical aggression are presented above in Table 6.11.

#### 6.3.2.4.3 *Inappropriate sexual behaviour*

Inappropriate sexual behaviour was observed in 93 (30.9%) participants. The four predictor variables were entered into a linear regression model for this subsample, which was significant ( $F_{(4,88)} = 3.77$ ,  $p < .01$ ) but did not account for much of the overall variance ( $R^2 = .15$ ; adjusted  $R^2 = .11$ ). The model produced a Durbin-Watson statistic of 1.94, within acceptable limits (Field, 2005), confirming the independence of errors. The only significant individual predictor was MPAI-4 Adjustment ( $p < .001$ ), in which higher scores (reflecting poorer adjustment) were associated with higher levels of inappropriate sexual behaviour.

There were no concerns with multicollinearity as the lowest tolerance and highest VIF values were identical to the models for verbal aggression and physical aggression. Outlier analysis for this final model revealed that four participants (4.3%) had an absolute standardised residual greater than 2.0 and that only one (1.1%) was greater than 2.5, which is as expected from chance. Again no cases had a standardised residual above 3.0.

Two (2.1%) cases had a centred leverage value greater than  $0.129$  ( $3k/n$  where  $k$  = number of predictors and  $n$  = sample size), within the 5% expected by chance. No case was deemed unduly influential as values for Cook's Distance, *DFBETA* and *DFFIT* were all well below the cut-off of 1.0.

For this model of inappropriate sexual behaviour, the plot of standardised residuals versus predicted values bore less resemblance to the "shape of a funnel" (Field, 2005, p. 203), which would have been indicative of heteroscedacity. Serial Levene tests confirmed that there were statistically significant differences in variance of standardised residuals for both education ( $F_{(1,91)} = 5.19, p < .05$ ) and type of injury ( $F_{(1,91)} = 4.04, p < .05$ ) but not for MPAI-4 Adjustment ( $F_{(1,91)} = 1.49, ns$ ) or MPAI-4 Participation ( $F_{(1,91)} = 0.80, ns$ ). The plots can again be seen in Appendix 4.

Neither the histogram and normal probability plot of the standardised residuals nor a Kolmogorov-Smirnov test ( $D_{(93)} = 0.20, ns$ ) indicated problems with the assumption of normality of residuals. These plots can also been seen in Appendix 4. The coefficients for this final model of physical aggression are presented above in Table 6.11.

### **6.3.3. Re-analysis of the MPAI-4**

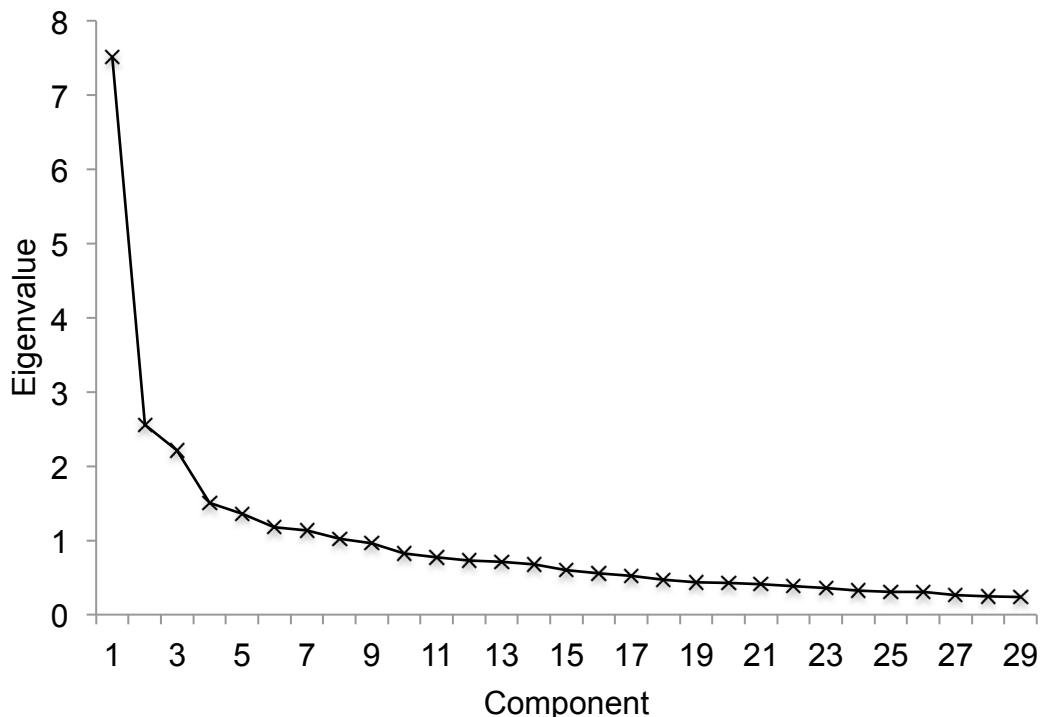
Use of the three rationally-derived subscales of the MPAI-4 had resulted in some modest regression models predicting verbal aggression, physical aggression and inappropriate sexual behaviour. Given that the psychometric development of the MPAI-4 had involved factor analysis (Bohac et al., 1997; Malec et al., 2003), it was important to investigate the underlying factor structure of the items within this dataset and what information this structure would provide regarding the behavioural dependent variables.

#### **6.3.3.1. Principal component analysis of the MPAI-4 items**

Following the previous methods used in the literature (Bohac et al., 1997; Malec et al., 2003), principal component analysis was conducted with varimax rotation of the 29 items of the MPAI-4. The rescored items for audition (item 4), communication (item 7), pain and headache (item 16), transportation (item 27) and employment (item 28) were used instead of the original items.

This produced a component structure with a Kaiser-Meyer-Olkin (KMO) value of .87, which is considered more than acceptable (Field, 2005; Kaiser, 1974). Other measures of the adequacy of using this technique included a highly significant Bartlett's test ( $p < .001$ ), all diagonal items of the anti-image correlation matrix were above .5 and only 33% of non-redundant residuals had absolute values greater than .05 (Field, 2005).

Using an eigenvalue cut-off point of 1.0, consistent with the previous literature, eight components would be retained. There was a clear inflexion point in the screeplot of the resulting eigenvalues (see Figure 6.4 below) that would suggest retaining only four components would be optimal.



**Figure 6.4** Eigenvalues after principal component analysis on the 29 items of the MPAI-4

However, four components explained less than half the variance (47.5%) and, as such, were not considered adequate. It was, therefore, decided to retain eight components, which explained almost two-thirds (63.7%) of the variance. The resulting rotated component matrix is presented below in Table 6.12.

**Table 6.12 Component matrix of 29 MPAI-4 items following principal component analysis with varimax rotation**

Item	Component							
	1	2	3	4	5	6	7	8
1. Mobility	.829							
2. Use of hands	.776							
3. Vision						.773		
4. Audition								.795
5. Dizziness								.512
6. Motor speech	.658							
7. Communication		.439	.478					
8. Attention / concentration		.740						
9. Memory		.824						
10. Fund of information		.598						
11. Novel problem-solving		.683						
12. Visuospatial						.703		
13. Anxiety					.738			
14. Depression					.701			
15. Irritability, anger, aggression			.750					
16. Pain and headache							-.462	.459
17. Fatigue	.672							
18. Sensitivity to mild symptoms					.700			
19. Inappropriate social interaction			.819					
20. Impaired self-awareness			.538					
21. Family / signif. relationships							.710	
22. Initiation		.537						
23. Social contact							.529	
24. Leisure and recreation	.433							
25. Self-care	.660							
26. Residence	.436	.402		.539				
27. Transportation	.429			.446				
28. Employment				.617				
29. Money management		.513		.540				

Any loadings smaller than .4 (explaining less than 16% of the variance of that component) are not included in this table. Table 6.13 below shows the MPAI-4 items loading most strongly for each component. The components have been labelled according to the nature of the items loading onto them. Each item showed a loading of .4 or greater on at least one component, with some items having this magnitude of loading on more than one component. When this occurred, the item was assigned to the component for which it had the highest loading.

**Table 6.13 Component labels and loading MPAI-4 items**

<b>Physical/ADLs</b>	<b>Cognition</b>	<b>Social</b>	<b>Community</b>
		<b>Behaviour</b>	<b>Independence</b>
mobility (1)	attention (8)	communicat. (7)	residence (26)
use of hands (2)	memory (9)	anger (15)	transport (27)
motor speech (6)	fund of info (10)	inappropriate (19)	employment (28)
fatigue (17)	prob-solve (11)	self-aware (20)	money (29)
leisure (18)	initiation (22)		
self-care (25)			

<b>Emotion</b>	<b>Visual</b>	<b>Social</b>	<b>Vestibular</b>
		<b>Relationships</b>	
anxiety (13)	vision (3)	family/other (21)	audition (4)
depression (14)	visuo-spatial (12)	social cont (23)	dizziness (5)
sensitivity (18)		pain (16)	

### 6.3.3.2. Using MPAI-4 components to predict behaviour

The raw items within each component were summed to form eight new MPAI-4 independent variables. The correlations between these components and the three behavioural dichotomies are presented below in Table 6.14.

**Table 6.14 Correlations between MPAI-4 components and the three behavioural dichotomies ( $n = 301$ )**

MPAI-4 component	Verbal aggression	Physical aggression	Inappropriate sexual behaviour
1: physical/ADLs	.05	.12*	-.01
2: cognition	.18**	.19**	.07
3: social behaviour	.51**	.49**	.31**
4: community independence	.08	.16**	.05
5: emotional status	.13*	.12*	.01
6: visuo-perceptual	.03	.05	-.02
7: social relationships	.14*	.17**	.05
8: vestibular function	.10	.10	.03

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

It can be seen that both the visuo-perceptual (6) and vestibular function (8) components did not produce any significant associations. While there were a number of significant correlations for both verbal aggression and physical aggression, only social behaviour (3) was significant for the inappropriate sexual behaviour dichotomous grouping. These eight MPAI-4 components were then used as

independent predictor variables in three separate logistic regression analyses to model verbal aggression, physical aggression and inappropriate sexual behaviour.

Table 6.15 below shows the final models after a forced entry procedure.

**Table 6.15 Results of logistic regression analyses using MPAI-4 components to predict the presence of behavioural disturbance**

	B (SE)	95% CI for exp b			
		Lower	exp b	Upper	
<b>Verbal aggression</b>					
$R^2 = .24$ (H & L), $.28$ (C & S), $.37$ (N); Model $\chi^2(8) = 97.31, p < .001$ .					
Constant	-1.25 (0.71)	-	0.29	-	
1: physical/ADLs	-0.01 (0.03)	0.93	0.99	1.04	
2: cognition	-0.01 (0.04)	0.91	0.99	1.08	
3: social behaviour	0.40*** (0.05)	1.34	1.49	1.65	
4: community independence	-0.13 (0.08)	0.75	0.88	1.02	
5: emotional status	-0.01 (0.05)	0.89	0.99	1.10	
6: visuo-perceptual	0.03 (0.07)	0.90	1.03	1.17	
7: social relationships	0.03 (0.09)	0.86	1.03	1.23	
8: vestibular function	-0.04 (0.11)	0.78	0.96	1.19	
<b>Physical aggression</b>					
$R^2 = .21$ (H & L), $.245$ (C & S), $.34$ (N); Model $\chi^2(8) = 84.86, p < .001$ .					
Constant	-3.53*** (0.87)	-	0.03	-	
1: physical/ADLs	0.01 (0.03)	0.95	1.01	1.07	
2: cognition	-0.03 (0.04)	0.89	0.97	1.05	
3: social behaviour	0.37*** (0.05)	1.30	1.44	1.59	
4: community independence	-0.01 (0.08)	0.84	0.99	1.17	
5: emotional status	-0.01 (0.05)	0.89	0.99	1.10	
6: visuo-perceptual	-0.01 (0.07)	0.87	1.00	1.14	
7: social relationships	0.03 (0.10)	0.85	1.03	1.23	
8: vestibular function	-0.08 (0.10)	0.76	0.93	1.13	
<b>Inappropriate sexual behaviour</b>					
$R^2 = .10$ (H & L), $.11$ (C & S), $.16$ (N); Model $\chi^2(8) = 36.50, p < .001$ .					
Constant	-1.75** (0.74)	-	0.17	-	
1: physical/ADLs	-0.02 (0.03)	0.93	0.98	1.03	
2: cognition	-0.03 (0.04)	0.90	0.97	1.06	
3: social behaviour	0.24*** (0.05)	1.16	1.27	1.39	
4: community independence	-0.02 (0.08)	0.85	0.99	1.15	
5: emotional status	-0.07 (0.05)	0.85	0.94	1.03	
6: visuo-perceptual	-0.02 (0.07)	0.86	0.98	1.12	
7: social relationships	-0.02 (0.09)	0.82	0.98	1.17	
8: vestibular function	-0.02 (0.10)	0.81	0.98	1.19	

H & L = Hosmer & Lemeshow; C & S = Cox & Snell; N = Naglkerke.

\*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$ .

It is clear from Table 6.15 that the MPAI-4 component “social behaviour” was the only significant predictor for all three models. Classification accuracies for verbal aggression, physical aggression and inappropriate sexual behaviour were 75.4%,

73.4% and 70.8%, respectively. Pearson correlations for each MPAI-4 item and behavioural groupings are presented in Appendix 5.

## **6.4. Discussion**

### **6.4.1. Research questions**

Three questions were addressed with this study. Firstly, it was predicted that the previously demonstrated distinctions between verbal aggression, physical aggression and inappropriate sexual behaviour would be replicated in this new, larger, multi-centre dataset. Replication would add substance to the conclusion that these behavioural disorders should not be considered part of an overriding general behavioural disinhibition syndrome following brain injury and that they represent separate clinical entities.

Secondly, it was anticipated that using wider-ranging measures of neurobehavioural function as independent predictor variables would lead to more complete regression modelling of verbal aggression, physical aggression and inappropriate sexual behaviour. The MPAI-4 (Malec & Lezak, 2003, April) captures a broader range of neurobehavioural symptomatology than did the neurocognitive measures used in the previous study. Additionally, the larger dataset permitted a two-part approach to modelling the behaviours – a logistic regression analysis of the presence or absence of the behaviour, followed by a linear regression analysis of the amount of behaviour when present.

Thirdly, it was also anticipated that this relatively large dataset would show an underlying factor structure of the MPAI-4 that was similar to previous reports. This would provide a further opportunity to explore the relationships between MPAI-4 items and verbal aggression, physical aggression and inappropriate sexual behaviour.

#### **6.4.2. Replicating behavioural distinctions**

Addressing the first research question, as expected, principal component analysis of the new dataset replicated the separation of verbal aggression, physical aggression and inappropriate sexual behaviour amongst individuals with severe brain injury. Replication is considered a fundamental part of the scientific method but has sometimes been overlooked within the field of psychology (Schmidt, 2009). In the present case, replication was achieved in a separate sample that was drawn from multiple sources, exhibiting a broader range of neurobehavioural presentation and behavioural disturbance. Additionally, while the BARS (Freeland et al., 2012) was again used to measure various forms of aggressive behaviours exhibited by participants, episodes of inappropriate sexual behaviour were recorded in a different way, with the recently developed SASBA (Knight et al., 2008).

Distinctions between verbal aggression, physical aggression and inappropriate sexual behaviour have proven robust. They were present for this sample both when the same eight behavioural variables (log-transformed) were used as in the previous study and when additional variables were included from the SASBA. Due to the infrequency with which some of the SASBA codes were observed within the sample,

as low as 0.7%, it was not possible to include all sixteen SASBA codes. Consequently, only the four different categories of SASBA recordings were used. Further research incorporating participants with higher frequency and more varied inappropriate sexual behaviour that would permit utilisation of all sixteen SASBA codes is warranted.

There have been few empirical studies reported in the literature that investigated forms of aggressive and inappropriate sexual behaviour in the same sample following brain injury. In their single case report of psychosocial treatment of inappropriate sexual behaviour after brain injury, Kelly and Simpson (Kelly & Simpson, 2011) have previously speculated that these behaviours have distinct functional features that differ from those involved in aggression. The only other similar study addressing both aggression and inappropriate sexual behaviour in the same sample (Alderman et al., 2009) showed only a modest correlation between aggression and inappropriate sexual behaviour, with the authors concluding that the two have differing aetiologies.

Others have reported differences in the relative frequencies between verbal and physical aggression. A group of people with traumatic brain injury showed higher levels of verbal aggression than control groups (spinal cord injury and an uninjured group) but not physical aggression (Dyer et al., 2006) and this was associated with greater impulsivity. Most of the high level of aggressive behaviours recorded by Alderman and colleagues (Alderman, 2007; Alderman et al., 2002) was noted to be verbal rather than physical in nature and that the two showed different setting events and antecedents.

### **6.4.3. Two-part regression modelling of behaviour**

In addressing the second research question, measures of current neurobehavioural function and care/supervision requirements were used alongside pre-injury and injury-related data as independent variables for two-part regression modelling of verbal aggression, physical aggression and inappropriate sexual behaviour displayed by the 301 participants with brain injury. Clinical comparison T-scores for the three subscales (Ability, Adjustment and Participation) of the MPAI-4 (Malec & Lezak, 2003, April) were used with raw scores for the Care and Needs Scale (Tate, 2004) and the Supervision Rating Scale (Boake, 1996) as the predictor variables for current functioning. The relatively large dataset permitted a two-part approach (Lachenbruch, 1992, 2001a, 2001b, 2002) that is useful when attempting to model dependent variables with excessive proportions of zero values. This was achieved by performing serial logistic regression analyses of the presence or absence of the behaviour for all 301 participants, followed by serial linear regression analyses to predict the amount of behaviour when present.

#### **6.4.3.1. Logistic regression models**

Logistic regression with 14 predictor variables was used to separately model the presence or absence of the three behavioural outcomes. Valid models were constructed for all three behavioural dichotomies that accounted for 39%, 33% and 22% of the variance in verbal aggression, physical aggression and inappropriate sexual behaviour, respectively. Final model classification accuracies were improved for verbal aggression (76.1% from an initial 57.5%) and for physical aggression

(74.4% from an initial 63.5%) but not for inappropriate sexual behaviour (69.1% for both). Male sex and higher scores on the Adjustment subscale of the MPAI-4 (reflecting poorer psychological adjustment) were significant predictors for all three behaviours. Being admitted six months or more post-injury was predictive of both verbal aggression and physical aggression but not inappropriate sexual behaviour. Having sustained a traumatic brain injury (as opposed to any other non-traumatic type) predicted the presence of verbal aggression only. Having a premorbid history of substance abuse, being injured at a younger age and having higher care needs were all associated with the presence of inappropriate sexual behaviour only.

#### **6.4.3.2. Linear regression models**

Linear regression analyses were then performed on the subsample of participants displaying each category of behaviour. The subsequent reduction in  $n$  meant that the entire set 14 independent variables could only be utilised for modelling the amount of verbal aggression ( $n = 173$ ). A backwards method led to a significant final model (accounting for 21% of the variance) with four predictors: educational level below GCSE, sustaining a traumatic brain injury, higher scores on MPAI-4 Adjustment and higher scores on MPAI-4 Participation (reflecting poorer social participation). Of these, only the last was not a significant individual predictor of greater verbal aggression.

These four remaining independent variables were then used for subsequent linear regression models (with forced entry procedure) for both physical aggression ( $n = 110$ ) and inappropriate sexual behaviour ( $n = 93$ ), explaining 29% and 15% of the

variance, respectively. Regression diagnostics indicated that a degree of heteroscedacity was present for both and that future efforts in this area should employ even larger sample sizes. With that caveat in place, only one of the four predictors were significant in each model: MPAI-4 Participation for physical aggression and MPAI-4 Adjustment for inappropriate sexual behaviour. Due to the limitations in sample size, these two models could not include the other ten predictor variables.

#### **6.4.3.3. Individual predictors**

The fourteen independent variables that were used in the logistic regression analyses and in the linear regression analysis for log-transformed non-zero verbal aggression values consisted of six premorbid psychosocial variables (sex, education, history of prior brain injury, psychiatric history, conviction for violence and substance abuse), three injury-related variables (type of injury, age at injury and chronicity) and five variables relating to current neurobehavioural function and care/supervision requirements (MPAI-4 Ability, MPAI-4 Adjustment, MPAI-4 Participation, CANS and SRS). The relationships between these predictor variables and the dependent variables of verbal aggression, physical aggression and inappropriate sexual behaviour are discussed below in turn.

##### **6.4.3.3.1 Sex**

Male sex increased the odds for all three behaviours occurring, by around three-fold for both verbal aggression and physical aggression and almost four-fold for

inappropriate sexual behaviour. However, in looking at only those participants who were verbally aggressive, sex did not predict how much verbal aggression was observed. No comparisons could be made for the remaining two behavioural subgroups as sex was subsequently not included in those models.

This finding is discrepant with the previous study (chapter 5), in which sex was not correlated with any of the three behavioural dichotomies and was not retained in each logistic regression model. The current sample ( $n = 301$ ) had identified 21/66 (37.9%) of the female participants as showing some verbal aggression (compared with 64.7% of males), 11/66 (16.7%) of females showing some physical aggression (compared with 42.1% males) and 8/66 (12.1%) of females showing some inappropriate sexual behaviour (compared with 36.2% of males). The  $\chi^2$  tests were all significant and the resulting effect sizes were presented in Table 6.6. As for the previous study ( $n = 152$ ), 22/38 (57.9%) of the female participants showed some verbal aggression (compared with 62.3% of males), 18/38 (47.4%) of females showed some physical aggression (compared with 35.1% of males) and 8/38 (21.1%) showed some inappropriate sexual behaviour (compared with 36.0% of males). None of the sex-based comparisons of these proportions were significant.

Clearly then, female participants displayed more verbal aggression, physical aggression and inappropriate sexual behaviour in study 2 (chapter 5) than in the current study. Why might this have been the case? Participants in study 2 were all drawn from a single rehabilitation unit, while in the current study (study 3) participants were drawn from seven different units (but including the same one as study 2). Recruiting participants from more than one unit, which includes a broader

range of presentations, is likely to reduce any sampling bias present in study 2. It may be concluded then, that the sample in the current study is more similar to the population of people surviving brain injury than the sample from study 2. Consequently, more confidence may be drawn in the finding from this current study, that males are indeed more likely than females to exhibit verbal aggression, physical aggression and inappropriate sexual behaviour following brain injury.

Previous studies have shown mixed results regarding the relationship between male sex and aggression after brain injury. In their sample of 89 consecutive patients admitted to hospital with traumatic brain injury, Tateno et al. (2003) reported that the proportion of males did not differ between their aggressive ( $n = 30$ ) and non-aggressive ( $n = 59$ ) groups. While Visscher et al. (2011) noted that their males were both more aggressive and more representative in the aggressive group than females in their small sample with acquired brain injury ( $n = 57$ ), sex did not prove to be a significant predictor of aggression within a logistic regression model. In their larger sample of consecutive referrals for neuropsychological assessment following traumatic brain injury ( $n = 287$ ), Wood and Liotti (2006b) noted that males were over-represented in their aggression group ( $n = 134$ ), although the reported effect size was small (.12).

Regarding inappropriate sexual behaviour, Alderman et al. (2009) reported that, in their survey of hospitalised inpatients with brain injury, only a small fraction (0.4%) of the 699 SASBA recordings were made by females (24.2% of 91 participants). Previously, Simpson et al. (1999) reported that, of the 29 of their cohort of 445 participants that were identified to have engaged in some form of “sex offending”

following brain injury, all were male. It would seem that the current method of using a standardised measurement tool (such as the SASBA) with which trained staff record inappropriate sexual behaviours as they occur after brain injury, as opposed to the retrospective reports employed by Simpson et al. (1999), revealed that some females do indeed exhibit these behaviours, albeit less commonly than males.

#### 6.4.3.3.2 *Education*

In this study, the level of education obtained by participants prior to their brain injury was shown to have limited ability to predict post-injury behavioural disturbance. The only significant correlation between a dichotomous grouping of educational level (at least GSCE versus below GCSE) and the various behavioural outcome variables was with the log-transformed non-zero values of verbal aggression ( $n = 173$ ); within the verbally aggressive subsample, having less than GCSE level education was associated with higher levels of post-injury verbal aggression. This association remained significant following linear regression modelling. The previous study (study 2, chapter 5) also did not find a relationship between education and the presence of behavioural disturbance.

Lower levels of education have previously been associated with aggression following acquired brain injury (Greve et al., 2001; Kerr et al., 2011; Wood & Liossi, 2006b) but not universally (Tateno et al., 2003). The only similar study addressing inappropriate sexual behaviour (Simpson et al., 2001) did not specifically address education in their sample.

#### **6.4.3.3.3      *Premorbid aggression***

Only 28 of 301 (9.3%) participants in this sample had a history of criminal conviction for aggression or violence prior to the brain injury that was documented in the clinical records. This prevalence is relatively consistent with the previous study (chapter 5), in which 18 of 152 (11.8%) participants had such a history, as well as those reported by Tateno et al. (2003) – 6.7% of 89 participants. Kerr et al. (2011), although reporting that a history of aggression was a significant predictor of post-injury aggression, did not state how many of their participants had such a history.

Somewhat surprisingly, having a premorbid history of aggression was not associated with any behavioural outcome measure in study 3. In study 2 (chapter 5), this variable proved to be a significant predictor of the presence of post-injury verbal aggression (but not physical aggression or inappropriate sexual behaviour). Other studies that have included this variable in their analyses have also found a significant relationship with aggression (Greve et al., 2001; Kerr et al., 2011; Tateno et al., 2003).

#### **6.4.3.3.4      *Pre-injury substance abuse***

Having a premorbid history of drug and/or alcohol abuse was associated with a participant being twice as likely to have exhibited inappropriate sexual behaviour during their residential assessment period. This variable was not significant in the logistic regression models for verbal aggression or physical aggression, or indeed for the linear regression model for non-zero verbal aggression.

This finding is in contrast with that of Simpson et al. (2001), who reported no differences in having a pre-injury history of substance misuse between their group of males showing post-injury inappropriate sexual behaviour ( $n = 25$ ) and their similarly sized clinical control group who did not show these behaviours. This variable was also not a significant predictor of the presence of inappropriate sexual behaviour in the study 2 (chapter 5).

It must be noted, however, that although this variable was a significant predictor within the current logistic regression model for the presence of post-injury inappropriate sexual behaviour, this model was no better than the null model at classifying cases. Moreover, the relationship between this variable and the dichotomous grouping of the behaviour was non-significant prior to the modelling. Given these points, this is a finding that requires confirmation in further work.

Considering the two forms of aggressive behaviour in this study, previous work that included a history of substance abuse in their analyses reported no relationship with aggression following brain injury (Kerr et al., 2011; Visscher et al., 2011). This is in keeping with the current findings and with study 2 (chapter 5).

#### 6.4.3.3.5 *Type of injury*

The type of brain injury or illness that each participant had sustained was initially coded as one of four types: traumatic brain injury, cerebro-vascular accident, cerebral anoxia and “other”. A diagnosis of traumatic brain injury showed the highest presence of verbal aggression, physical aggression and inappropriate sexual

behaviour in the sample. For those participants who did show these behaviours, analyses of the non-zero values revealed no differences between the four types. As such, this variable was collapsed into a dichotomy of traumatic versus non-traumatic brain injury for ease of analysis.

The current results indicated that having sustained a traumatic brain injury increased the likelihood of post-injury verbal aggression approximately two-fold. For those participants that were verbally aggressive, having a traumatic brain injury predicted higher levels of verbal aggression. This effect of type of injury was not seen in physical aggression or inappropriate sexual behaviour. In study 2 (chapter 5), dichotomous type of injury was not related to any of the three behavioural groupings.

While a number of studies in this area specifically sampled traumatic brain injury only (Greve et al., 2001; Tateno et al., 2003; Wood & Liossi, 2006b), two studies that utilised a more heterogeneous sample of acquired brain injury (as in this study) showed contrasting results. Kerr et al. (2011) used the same dichotomous variable for injury type and reported no relationship to group membership for generic aggression rated retrospectively by staff; of note is that their recording system separated verbal from physical aggression but this distinction was not used in their analysis. Visscher et al. (2011) reported that hypoxia as a cause of brain injury was associated with the presence of aggression and that this remained a significant predictor following logistic regression, however it should be noted that only 9 of their 52 participants (16%) had this diagnosis.

#### 6.4.3.3.6 *Chronicity*

Chronicity, or the time between injury/illness and admission to rehabilitation, was measured in months and the final variable was dichotomised. Participants admitted within six months of their injury were differentiated from those admitted six months or more since their injury. In this sample, the latter increased the odds of a participant having exhibited some verbal aggression or physical aggression by around two-fold. There was no association with the non-zero values of either, nor was there a relationship with the presence of inappropriate sexual behaviour.

Previous research has consistently shown that this variable was not related to aggression (Greve et al., 2001; Tateno et al., 2003; Visscher et al., 2011; Wood & Liossi, 2006b) or inappropriate sexual behaviour (Simpson et al., 2001). It seems most likely that the contrasting results found in this study are related to a bias in referral. The present sample was not designed to be representative of all brain injuries, or even all severe brain injuries. The rehabilitation programmes, particularly the two challenging behaviour units, were founded on a neurobehavioural ethos, which means that a common referral problem concerns post-injury aggressive behaviour. Effectively, admissions within six months of injury may be more related to independent living skills and transfers from acute hospitals. Later admissions (people who may well have already left hospital and returned to living in the community) are likely to have been referred for continuing problematic interpersonal behaviour such as verbal and/or physical aggression that was presenting barriers for community-based rehabilitation and support.

#### **6.4.3.3.7 MPAI-4 subscales**

The MPAI-4 subscale of Adjustment was a significant predictor of verbal aggression, physical aggression and inappropriate sexual behaviour having been recorded by staff. Specifically, higher Adjustment scores (reflecting poorer adjustment after brain injury) were strongly associated with the presence of behavioural disorder and for the degree of verbal aggression and inappropriate sexual behaviour recorded. This is not surprising, as in addition to items concerning anxiety and depression, this subscale also includes items for agitation, anger and aggression (item 15) and inappropriate social interaction (item 19).

The guidance in the MPAI-4 manual for the former item is to rate “verbal or physical expressions of anger” (Malec & Lezak, 2003, April, p. 20). The rater is instructed to make a distinction on the 5-point Likert scale scoring system from 0 (“normal control of aggressive impulses”) through 4 (“severe lack of control of aggressive impulses”). Although such clinical judgements will be prone to cognitive bias, clearly this item will overlap with the dependent variables of verbal aggression and physical aggression, as measured by the BARS.

Similarly, the latter item involving inappropriate social interaction concerns social behaviours displayed by the person with brain injury. For this item, raters are instructed to make a judgement ranging from 0 (normal behaviour in social situations) through to 4 (disinhibited behaviour is apparent almost continuously). The item is defined rather loosely – “acting childish, silly, rude; behaviour is not

consistently fitting to the time and place" (Malec & Lezak, 2003, April, p. 24).

Instances of inappropriate sexual behaviour would likely be included within this item.

The MPAI-4 subscale of Participation was associated with the presence of verbal aggression but not physical aggression or inappropriate sexual behaviour. However, this relationship was in an unexpected direction. Higher scores on the subscale (reflecting poorer participation in social roles such as leisure and recreational activities, self-care and employment) were associated with no verbal aggression being present. This appears to make less intuitive sense than the finding that higher scores on this subscale were strongly associated with a greater degree of physical aggression when present (but not verbal aggression or inappropriate sexual behaviour). Those participants who were physically aggressive tended to show significantly more physically aggressive behaviour if they were also socially isolated and or heavily reliant upon others for engaging in social roles. This may have been related to the frequency with which caregivers needed to be within close proximity and providing guidance and direction for meaningful activities, thereby providing those participants with more opportunity to engage in physically aggressive behaviours. Alderman and colleagues (Alderman, 2007; Alderman et al., 2002) had previously reported that environmental triggers such as staff prompting were common antecedents to severe aggression.

More interestingly, the subscale of Abilities (comprised of items addressing physical status and cognitive skills) had no predictive relationship with the presence of verbal aggression, physical aggression or inappropriate sexual behaviour. This may not be surprising when it is recalled that this subscale, along with the other two, were

“rationally-derived” by the developers and that the underlying factor structure of the MPAI-4 has turned out to be quite different in nature (Bohac et al., 1997; Malec et al., 2003). This is discussed further in 6.4.4 below.

#### 6.4.3.3.8 *Care and Needs Scale*

Scores on the CANS (Tate, 2004), which reflect the need for external care and support, showed no relationship with verbal or physical aggression. Higher scores were significantly associated with the presence of inappropriate sexual behaviour. Methodological limitations prevented further analysis of the non-zero inappropriate sexual behaviours. Previously, the clinical profile of a sample of people with brain injury referred to a community behavioural management service had been described with the CANS (Kelly et al., 2008). However, individual scores had not then been compared with the challenging behaviour recorded, including verbal aggression and inappropriate sexual behaviour.

Greater care needs inevitably lead to more frequent contact by caregivers, thereby providing numerous opportunities for misreading of social cues on the part of the person with brain injury. The majority of people within post-acute residential rehabilitation programmes are male and, clinical experience is that the overwhelming majority of rehabilitation support workers are female. It is, therefore, not surprising that greater care needs provide the environmental setting within which such behaviours may be exhibited. Further research interest in this area could also address the non-zero values of inappropriate sexual behaviour, that is, whether

greater care needs also result in more frequent and/or severe inappropriate sexual behaviour.

#### **6.4.4. Re-analysis of the MPAI-4**

The third research question posed by this study concerned the structure of the MPAI-4 itself. It will be recalled that the manual (Malec & Lezak, 2003, April) provides standardised clinical comparison T-scores for three “rationally-derived” subscales – Ability, Adjustment, Participation – as well as a Total score. T-scores on the three subscales were utilised in addressing the second research question in this study. However, previous empirical studies (Bohac et al., 1997; Malec et al., 2003) have shown that the underlying structure of this instrument is not consistent with these three constructs. Would re-analysing the MPAI-4 data in the current sample replicate this previous work? What would this say about the dependent variables of verbal aggression, physical aggression and inappropriate sexual behaviour?

Principal component analysis employing a similar methodology to previous studies revealed an eight component structure that explained 63.7% of the variance observed in the sample of 301 participants with acquired brain injury. This was consistent with an early study of the original MPAI (Bohac et al., 1997), in which eight factors explained 64.4% of the variance among 189 participants with acquired brain injury. A similar study with the current version of the MPAI-4 had then been conducted with 386 participants with acquired brain injury, revealing seven factors (Malec et al., 2003), although variance accounted for was not reported.

For the present study, labels were applied to each component in an attempt to provide a description of the MPAI-4 items that loaded onto it. The resulting components were: physical/activities of daily living; cognition; social behaviour; community independence; emotion; visual perception; social relationships; vestibular function. These eight components were then used as independent predictor variables in separate logistic regression models for verbal aggression, physical aggression and inappropriate sexual behaviour. While the overall models for each were statistically significant with modest classification accuracy, only the social behaviour component was a significant individual predictor.

This is an important finding. The strength of the social behaviour component should not be surprising, given that items 15 (irritability, anger and aggression) and 19 (inappropriate social interaction) both loaded specifically onto this component. However, the other items that loaded onto this social behaviour component were also clearly related to the dependent variables and are therefore deserving of further discussion. Equally, those components (and items comprising those components) that did not predict verbal aggression, physical aggression and inappropriate sexual behaviour are also informative.

#### **6.4.4.1. MPAI-4 items loading onto the social behaviour component**

In addition to items 15 and 19, both communication (item 7) and impaired self-awareness (item 20) also loaded onto the component labelled social behaviour. Poorer communication skills have previously been related to aggressive behaviours following brain injury, in the literature (Alderman, 2007; Alderman et al., 2002) and in

study 2 here (chapter 5). A link between self-awareness and aggression has been reported in the clinical literature for psychosis (Alia-Klein, O'Rourke, Goldstein, & Malaspina, 2007; Bo, Abu-Akel, Kongerslev, Haahr, & Simonsen, 2011; Schaub, Brune, Bierhoff, & Juckel, 2012) and alcohol-related violence (Giancola, Duke, & Ritz, 2011; Giancola, Josephs, DeWall, & Gunn, 2009) but not yet within brain injury.

The importance of disorders of self-awareness after brain injury has been known for some time and is well documented (Bach & David, 2006; Prigatano, 2005a, 2005b; Prigatano & Altman, 1990; Stuss & Anderson, 2004). A number of theoretical and conceptual models have been developed (Crosson, Barco, & Velozo, 1989; Schacter, 1990; Stuss & Levine, 2002) and self-awareness per se is now frequently a goal for rehabilitation (Goverover, Johnston, Toglia, & Deluca, 2007; Lundqvist, Linnros, Orlenius, & Samuelsson, 2010; Medley & Powell, 2010; Ownsworth, Turpin, Andrew, & Fleming, 2008; Schmidt, Lannin, Fleming, & Ownsworth, 2011).

Given this, it is surprising that measures of self-awareness have not been utilised in research addressing aggression and inappropriate sexual behaviour following brain injury. Perusal of the literature revealed no empirical study in which self-awareness or insight was used in a statistical analysis of aggression. Clearly, there is a need for further efforts in this area of research to further explore this relationship. Regarding inappropriate sexual behaviour, Simpson et al. (2001) reported no difference in clinician-rated awareness between their “sexually aberrant” group and the comparison group not exhibiting such behaviours. Both were equally impaired.

How do the present results compare with the previous factor analyses of the MPAI? The Bohac et al. (1997) study used the original version of the MPAI and, although there are some minor differences between this and the current version, comparisons may still be drawn. In that study, the items “irritability to aggression” (corresponding to the current item 15) and “appropriate social interaction” (corresponding to the current item 19) loaded with “family/significant relationships” onto a separate factor that the authors labelled “social skills/support”. A communication item loaded onto a factor labelled “cognition”, along with items for memory, fund of information and novel problem-solving. There was no single item labelled “self-awareness”, however, “indifference” loaded along with “anxiety to agitation”, “pain” and “depression” onto a factor that was labelled “impaired self-awareness”. It is also of note that their sample of 189 consecutive US outpatients with brain injury (80% traumatic brain injury) was more highly educated and less severely injured than the current sample. Although not explicitly stated, it is also most likely that the outpatient sample exhibited less verbal aggression, physical aggression and inappropriate sexual behaviour than the current residential sample.

The Malec et al. (2003) revision study, on the other hand, employed the MPAI-4. Their reported principal component analysis produced seven factors that were not given specific labels. “Irritability, anger and aggression” (item 15) loaded onto the same factor as “sensitivity to mild symptoms”, “anxiety” and “depression”, while “inappropriate social interaction” (item 19) loaded onto a different factor along with “family/significant relationships”, “social contact” and “leisure and recreational activities”. Both “communication” and “self-awareness” loaded onto a factor that might be reasonably have been labelled “cognition”, along with the other items

“memory”, “novel problem-solving”, “fund of information”, “attention/ concentration” and “initiation”. Again, the sample used in this study was not equivalent with the current study; the participants were a consecutive series that had been recruited from outpatient, community-based and residential facilities from across the US. The sample was again comparatively highly educated with lower injury severity ratings. No data were provided regarding specific behavioural disturbances.

#### **6.4.4.2. Other MPAI-4 components**

The remaining seven components were not predictive of verbal aggression, physical aggression or inappropriate sexual behaviour in the logistic regression models. This may not be surprising, given that individual MPAI-4 items would likely have loaded more heavily onto the social behaviour component during the principal component analysis were they more strongly related to the dependent variables.

A lack of association between the dependent behavioural variables and the “cognition” factor identified during principal component analysis is consistent with other studies employing overall measures of cognitive function (Alderman, 2007; Alderman et al., 2002; Tateno et al., 2003; Visscher et al., 2011). In contrast to the results in this study, impoverished self-care and independent living skills (related to components “physical/ADLs” and “social independence” in this study) were related to aggression in some similar studies (Alderman, 2007; Kerr et al., 2011) but not universally (Tateno et al., 2003).

#### **6.4.5. Summary**

The discussion presented here has been lengthy and the reader would likely benefit from a summary at this point. As such, it will be recalled that this study addressed three specific questions concerning verbal aggression, physical aggression and inappropriate sexual behaviour after brain injury.

Firstly, the previously identified (chapter 5) distinctions between verbal aggression, physical aggression and inappropriate sexual behaviour were replicated here in a separate and larger sample of participants with severe acquired brain injury that was drawn from multiple rehabilitation centres. Little work has been conducted in this area, particularly involving inappropriate sexual behaviour. This important replication provides further compelling evidence that these behaviours should be considered as separate clinical entities in further applied research.

Secondly, the relatively large size of the current sample permitted two-part modelling of verbal aggression, physical aggression and inappropriate sexual behaviour after brain injury. This statistical technique can deal with the problems that arise from excess zeroes in the dependent variables. The presence or absence of behaviour was modelled via logistic regression while the degree of behaviour (when present) was modelled via linear regression. A number of independent variables relating to premorbid psychosocial, injury-related and current functioning (including the MPAI-4) were able to predict stable but modest models. However, smaller subsample sizes within the linear modelling for physical aggression and inappropriate sexual behaviour led to limitations in the available predictors as well as concerns over

possible heteroscedacity, or inconstant variance. Further efforts with larger subsamples of participants exhibiting these two behaviours are therefore warranted.

Thirdly, exploratory factor analysis of the MPAI-4 data revealed a number of underlying factors that were broadly consistent with previously published research. Only one of the eight identified factors was predictive of the presence of behavioural disturbance and this same factor was significant for verbal aggression, physical aggression and inappropriate sexual behaviour. Taking into account the complicating issue of two of the MPAI-4 items that likely shared an overlap with the dependent variables, items for communication and impaired self-awareness also loaded onto this factor. While an association between post-injury aggression and poorer communication skills has been previously reported in the literature, there has been a conspicuous gap regarding self-awareness after brain injury.

# **CHAPTER 7. STUDY 4: THE ROLE OF EXECUTIVE FUNCTIONS**

## **7.1. Introduction**

The term “executive functions” is typically used when referring to higher order mental processes that have a regulatory role over lower order cognition and self-directed adaptive behaviours. Important early models of cognition (for example, Baddeley & Hitch, 1974; Norman & Shallice, 1986) built on Luria's (1966, 1973) conception of the role of the human frontal lobes in organising, programming and checking of meaningful activities. While various aspects of executive function have been linked with specific neuroanatomical frontal lobe regions (for example, Stuss et al., 2002), overall the results of research in this area have generally been mixed (Alvarez & Emory, 2006).

Attempts have been made to refine understanding of the executive functions. Lezak (1983) considered them distinct from cognitive functions, and initially speculated on several separable processes: volition, planning, purposive action and effective performance. In a recent review of executive functions (Jurado & Rosselli, 2007), it was noted that research into numerous integrated processes including planning and organising, initiation, implementing problem-solving strategies, self-control, flexibility in thinking and self-monitoring has weakened the concept of a single executive system. Factor analytic studies have indicated that the key underlying components of

executive function seem to be inhibition/switching, working memory and sustained/selective attention (Alvarez & Emory, 2006).

Given the established regulatory role of cognitive executive functions over purposeful behaviour, clearly they are of critical interest when examining socially maladaptive behaviours such as aggression and inappropriate sexual behaviour. Neurobiological models of both aggression (Siever, 2008; Volavka, 2002) and sexual behaviour (Rees et al., 2007; Spinella, 2007) highlight the importance of so-called “top-down” executive cognitive processes involved in the regulations of these behaviours. What has previous research reported about this relationship?

### **7.1.1. Executive function and aggression**

Despite difficulties with definitions, a consistent relationship between cognitive executive function and aggression has been documented (for reviews, see Hawkins & Trobst, 2000; Morgan & Lilienfeld, 2000). Subsequent work has continued to document this link in neurologically healthy children (Ellis, Weiss, & Lochman, 2009; Raaijmakers et al., 2008) and university students within laboratory models of aggressive behaviour (Denny & Matthias, 2012; Kramer, Kopciok, Richter, Rodriguez-Fornells, & Munte, 2011; Sprague, Verona, Kalkhoff, & Kilmer, 2011). Other populations in which this relationship has been reported include patients with psychosis (Hanlon, Coda, Cobia, & Rubin, 2012; Harris et al., 2010; Krakowski & Czobor, 2012; Naudts & Hodgins, 2006; Song & Min, 2009) and violent offenders (Hancock et al., 2010; Hanlon, Rubin, Jensen, & Daoust, 2010; Hoaken, Allaby, & Earle, 2007; Ross & Hoaken, 2011). Executive function has been implicated as a

mediator in alcohol-related aggression (Giancola, 2000, 2004; Giancola, Godlaski, & Roth, 2012; Heinz, Beck, Meyer-Lindenberg, Sterzer, & Heinz, 2011) and highlighted as a risk factor in intimate partner aggression (Howard, 2012; Walling et al., 2012).

There has been limited research into the relationship between executive function and aggression in people with acquired brain injury. Neither Greve et al. (2001) nor Wood and Liossi (2006b) found significant differences between an aggressive and a control group on neuropsychological measures of executive function. In the study reported by Greve et al. (2001), both groups were equally impaired in their performances on the Wisconsin Card Sorting Test, a verbal fluency test and the Trail Making Test. The authors concluded that impulsive aggression after brain injury was not closely associated with executive function as measured by traditional neuropsychological tests. Similarly, Wood and Liossi (2006b) reported that their groups did not differ in performances on the Hayling & Brixton tests, the Zoo Map test from the Behavioural Assessment of the Dysexecutive Syndrome (BADS) and Trails B; indeed the authors reported that the participants within their aggressive group performed within the average range on the executive tests, with the exception of Trails B. Clinician-rated incidence of impulsivity and disinhibition (via the Neurobehavioral Rating Scale) were significantly higher in the aggressive group. It was concluded that these symptoms were related to executive deficits, even if the test results showed no difference.

### **7.1.2. Executive function and inappropriate sexual behaviour**

Little work has again been conducted in the area of inappropriate sexual behaviour. Disinhibition of sexual impulses has been shown to be impaired following

neurological injury and illness, particularly implicating the frontal lobes (Baird et al., 2007; Burns & Swerdlow, 2003; Cao et al., 2010; Emory et al., 1995; Fyffe et al., 2004; Woods et al., 1998). Studies of sex offenders have also shown weaker executive function than other offenders (Dolan, Millington, & Park, 2002; Eastvold, Suchy, & Strassberg, 2011; Joyal, Black, & Dassylva, 2007; Kelly, Richardson, Hunter, & Knapp, 2002; Suchy, Whittaker, Strassberg, & Eastvold, 2009).

In the one group study involving people with acquired brain injury, Simpson et al. (2001) found no difference between their “sexually aberrant” group and a clinical control group in terms of executive function. Due to the variety of neuropsychological measures that had been administered, the researchers used the various test results to form clinician-ratings of executive (and cognitive) function. As such they did not use the test results in a more direct manner.

### **7.1.3. Results from study 2 (chapter 5)**

The second research question in study 2 (reported in chapter 5 in this thesis) involved the predictive relationships of neurocognitive independent variables on verbal aggression, physical aggression and inappropriate sexual behaviour in logistic regression modelling. This was performed on a subset of 77 from the total sample of 152 participants with severe brain injury that had full data for the six factor structure (Tulsky, 2003; Tulsky & Price, 2003) of the Wechsler Adult Intelligence Scale – Third Edition (Wechsler, 1997a) and the Wechsler Memory Scale – Third Edition (Wechsler, 1997b). The six factors were Verbal Comprehension Index (VCI),

Perceptual Organisation Index (POI), Processing Speed Index, Working Memory Index (WMI), Auditory Memory (AM) and Visual Memory (VM).

It will be recalled that for verbal aggression, despite significant correlations with Verbal Comprehension, Perceptual Organisation, Processing Speed and Working Memory, only the Verbal Comprehension Index proved to have any individual predictive ability after logistic regression. For physical aggression, only the Verbal Comprehension Index was correlated with the dichotomous behavioural grouping and this measure remained in the logistic regression model as a significant individual predictor. Regarding inappropriate sexual behaviour, none of the six measures were significantly correlated with behavioural grouping and a significant logistic regression model could not be produced.

Due to the limited sample size, measures of cognitive executive function could not be included for analysis in study 2, even though they were available for a number of participants. The option of combining the samples from study 2 ( $n = 152$ ) and study 3 ( $n = 301$ ) would allow a greater subset of participants for whom both six factor neurocognitive and executive function data were available. The current study was designed to realise this option. In addition to the same six measures of neurocognitive function employed in study 2, four individual tests of the Delis-Kaplan Executive Function System (D-KEFS; Delis, Kaplan, & Kramer, 2001) were also included: Trail Making Test, Verbal Fluency Test, Color-Word Interference Test and Tower Test.

#### **7.1.4. Delis-Kaplan Executive Function System (D-KEFS)**

Within clinical neuropsychological practice, specific tests of executive function are routinely used alongside other tests of neurocognitive function (Lees-Haley et al., 1996; Rabin et al., 2005). While there is no gold standard test, there are a number of tests that are considered to measure particular aspects of executive function (Chan, Shum, Toulopoulou, & Chen, 2008). The lack of a comprehensive neuropsychological theory of executive function (Jurado & Rosselli, 2007) has led some to recommend that a process-driven approach is the best method for clinical assessment (Lezak, 1995; Miyake, Emerson, & Friedman, 2000; Strauss, Sherman, & Spreen, 2006). One such assessment tool developed from this approach is the Delis-Kaplan Executive Function System (D-KEFS; Delis et al., 2001).

The D-KEFS is a set of nine standalone neuropsychological tests of executive function that is widely used in clinical and research settings. All D-KEFS tests were co-normed and standardised on a US representative sample of 1750 neurologically healthy people aged 8-89. The nine tests are considered refinements of prior clinical or experimental tests (Strauss et al., 2006) and, rather than being derived from a theoretical model of executive functions, involve a process-oriented approach. Each test produces multiple scores that were intended to enable the separation of specific executive deficits from primary cognitive deficits.

Independent published reviews (Baron, 2004; Homack, Lee, & Riccio, 2005) concluded that the inherent cognitive-process approach of the D-KEFS was particularly useful in the assessment of executive functions, although cautions were

raised about a perceived lack of reliability and validity studies. A subsequent recommendation was made to avoid using the contrast measures due to low reliability and measurement error (Crawford, Sutherland, & Garthwaite, 2008). A three factor structure of the D-KEFS tests has been reported, consisting of conceptual flexibility, monitoring and inhibition (Latzman & Markon, 2010). There has also been recent work developing supplementary methods of analysing base rate information regarding score discrepancies (Crawford, Garthwaite, Sutherland, & Borland, 2011).

For the current study, four of the D-KEFS standalone tests of executive function were used: Trail Making Test, Verbal Fluency Test, Color-Word Interference Test and Tower Test. These tests were selected as they are the most frequently administered D-KEFS tasks within the dataset. The primary measures from these four tasks all load onto a reported inhibition factor (Latzman & Markon, 2010). Sample size restrictions meant that only one measure from each selected D-KEFS test could be included as variables of executive function. The criteria for single measure selection are explained below.

#### **7.1.4.1. Trail Making Test**

Lezak (1995) noted that the original version of this task was part of the Army Individual Test Battery in 1944. It was comprised of two parts: in part A the person taking the test draws a line that connects consecutively numbered circles on a single sheet, and in part B the person then draws a line that alternates between the sequences of numbered circles and lettered circles. The D-KEFS version of the Trail

Making Test has five parts, in an attempt to isolate set-shifting (Condition 4: Number-Letter Sequencing, equivalent to part B) from other basic cognitive processes such as visual scanning, number sequencing (Condition 2, equivalent to part A), letter sequencing and motor speed.

Clinical research involving the original version of the Trail Making test has indicated that part B is a valid measure of aspects of executive function, particularly in terms of task-switching or cognitive flexibility (Ashendorf et al., 2008; Kortte, Horner, & Windham, 2002; Lange, Iverson, Zakrzewski, Ethel-King, & Franzen, 2005; Sanchez-Cubillo et al., 2009). Functional neuroimaging and lesion studies have also implicated the role of the prefrontal cortex in part B performance (Allen, Owens, Fong, & Richards, 2011; Jacobson, Blanchard, Connolly, Cannon, & Garavan, 2011; Moll, de Oliveira-Souza, Moll, Bramati, & Andreuolo, 2002; Stuss et al., 2001; Zakzanis, Mraz, & Graham, 2005).

The D-KEFS version of the Trail Making Test has also been utilised in clinical research. Patients with lateral prefrontal cortex lesions performed more poorly than controls on several conditions of the task, including both slower completion times and higher error rates on Condition 4 (Yochim, Baldo, Nelson, & Delis, 2007). In an ecological study involving four D-KEFS tasks – Trail Making Test, Verbal Fluency Test, Design Fluency Test and Tower Test – only Condition 4 (Number-Letter Sequencing) of the Trail Making Test accounted for unique variance in functional status in older adults (Mitchell & Miller, 2008). The same key measure was selectively impaired in children with foetal alcohol syndrome (Mattson, Goodman, Caine, Delis, & Riley, 1999), patients with temporal lobe epilepsy (McDonald, Delis,

Norman, Tecoma, & Iragui-Madozi, 2005) and older adults with poor cardiovascular function (Jefferson, Poppas, Paul, & Cohen, 2007). Recent normative adjustments for education and vocabulary skills have also been published (Fine, Delis, & Holdnack, 2011).

It was, therefore, decided that the scaled score of the Condition 4: Number-Letter Sequencing of the D-KEFS version of the Trail Making Test was to be included in the subsequent analyses. This is the replica of part B of the original version of the test.

#### **7.1.4.2. Verbal Fluency Test**

The D-KEFS Verbal Fluency Test is based on the original Controlled Oral Word Association (Benton & Hamsher, 1976), which requires the person taking the test to say as many words beginning with a particular letter of the alphabet within a 60 second time limit (verbal generativity) and while obeying certain conditions (requiring the inhibition of errors). Lezak (1995) summarised the initial clinical research with the task, concluding that lesions to either frontal lobe caused selective reduction in performance, but particularly so for the left frontal lobe. The left inferior frontal gyrus has been selectively implicated in a systematic review of functional neuroimaging studies involving verbal fluency tasks (Costafreda et al., 2006). Recent meta-analytic reviews of verbal fluency tasks have been shown to be particularly sensitive to the effects of traumatic brain injury (Henry & Crawford, 2004b) and frontal lesions (Henry & Crawford, 2004a).

Research involving the D-KEFS version of the task, which consists of the sub-tasks letter fluency (condition 1), category fluency (condition 2) and switching (condition 3), has been consistent with previous incarnations. Both the letter and category fluency conditions were reported to differentiate participants with traumatic brain injury from a matched healthy control group (Strong, Tiesma, & Donders, 2011). Category fluency was shown to be reduced in recurrent major depression (Schmid, Strand, Ardal, Lund, & Hammar, 2011) and associated with anosmia following traumatic brain injury (Sigurdardottir, Jerstad, Andelic, Roe, & Schanke, 2010). A group of patients with frontal lobe lesions performed more poorly on all conditions of the D-KEFS Verbal Fluency Test, particularly the letter fluency condition (Baldo, Shimamura, Delis, Kramer, & Kaplan, 2001). The scaled score for condition 1: Letter Fluency, which best corresponds to the original task, was selected for inclusion in the current study.

#### **7.1.4.3. Color-Word Interference Test**

The original version of this task (Stroop, 1935) was named after the developer and there have been many versions in clinical use since that time. The essence of the task is that it takes people longer to name colour patches than it does to read words, and longer again when attempting to name the colour of the ink when a colour word is printed in a contrasting colour ink (Lezak, 1995). It is generally considered a measure of selective attention, although other cognitive processes may be involved (Ben-David, Nguyen, & van Lieshout, 2011; Dimoska-Di Marco, McDonald, Kelly, Tate, & Johnstone, 2011). A recent systematic review of functional neuroimaging

studies concluded that fronto-parietal neural circuits are consistently implicated in performance of this task (Roberts & Hall, 2008).

The D-KEFS version of this task – the Color-Word Interference Test – includes an inhibition/switching task in addition to the initial simpler tasks of naming colour patches (condition 1), reading aloud colour names (condition 2) and the traditional Stroop-task of naming the colour of the ink of a contrasting colour word (condition 3). Condition 4 requires the person taking the test to switch between responding as in condition 3 and reading aloud the colour words. This requires the person to repeatedly switch between these two different response styles. Both the inhibition and inhibition/switching conditions were performed more slowly by children with foetal alcohol syndrome in an early D-KEFS validity study (Mattson et al., 1999), patients with frontal lobe epilepsy (McDonald, Delis, Norman, Wetter, et al., 2005) and a group of older adults with subcortical lacunar infarcts (Kramer, Reed, Mungas, Weiner, & Chui, 2002). One study has suggested that the switching condition may not be any harder than the inhibition condition (Lippa & Davis, 2010). For the current study, therefore, the scaled score for the inhibition condition of the CWIT was used.

#### **7.1.4.4. Tower Test**

The D-KEFS Tower Test is a development of earlier versions of visuo-spatial and motor planning tasks such as the Tower of Hanoi (Simon, 1975) and the Tower of London (Shallice, 1982). Performance on these tasks has been associated with frontal lobe function in neuropsychological lesion studies (Morris, Miotti, Feigenbaum, Bullock, & Polkey, 1997; Owen, Downes, Sahakian, Polkey, &

Robbins, 1990; Shallice, 1982; Shallice & Burgess, 1991) and with dorsolateral prefrontal cortex in functional neuroimaging studies (Baker et al., 1996; Crescentini, Seyed-Allaei, Vallesi, & Shallice, 2012; Lazonen et al., 2000; Owen, Doyon, Petrides, & Evans, 1996; van den Heuvel et al., 2003).

Research involving the D-KEFS version of the Tower Test has shown that it probably does not measure exactly the same aspects of executive function as its predecessors (Larochette, Benn, & Harrison, 2009; McKinlay, Grace, Kaller, et al., 2009). In older adult samples, the overall achievement score for the test was not associated with overall daily functioning (Jefferson, Paul, Ozonoff, & Cohen, 2006; Mitchell & Miller, 2008) but was linked with poor cardiovascular output (Jefferson et al., 2007). The total score was lower for children with autistic spectrum disorders than controls (Lopez, Lincoln, Ozonoff, & Lai, 2005) and has also been linked with insight in psychosis patients (Lysaker, Whitney, & Davis, 2006).

The D-KEFS Tower Test incorporates a rule violation measure in addition to the total score and more traditional variables of time taken and number of moves made. Importantly, excessive Tower Test rule violation errors (a marker of impulsivity and difficulty in inhibiting errors) has been associated with decreased bilateral frontal lobe volume (Carey et al., 2008) and localised lesions in the prefrontal cortex (Yochim, Baldo, Kane, & Delis, 2009). In an early D-KEFS development study, children with foetal alcohol syndrome also made more rule violations than controls (Mattson et al., 1999). It was therefore decided that the rule violation ratio score (scaled score) would be included in the analyses.

### **7.1.5. Research question**

The predictive ability of the six neurocognitive measures in study 2 (chapter 5) was relatively modest, but no measures of cognitive executive function were included. This study therefore added robust neuropsychological tests of executive function to the previous measures. It was anticipated that poorer scores on the executive function tests would account for additional variance in the probability of having exhibited verbal aggression, physical aggression or inappropriate sexual behaviour.

## **7.2. Method**

The separate datasets from the previous two studies (chapters 5 and 6) were combined in order that measures of executive function could be obtained from a large enough sample. There was no overlap between these two samples and both had the same behavioural observation timeframe of nine weeks from admission. It will be recalled that the first week of behavioural observations was not included in the analysis due to variability in the admission day of the week.

### **7.2.1. Participants**

There were a total of 453 participants in the combined datasets from studies 2 and 3. The characteristics of these participants are described in detail in the previous two chapters of this thesis. For the current study, only those participants who had complete data available for the selected neurocognitive measures, which are described below, were selected.

This selection criterion resulted in a subsample of 86 (19.0%) participants, of which 70 (81.4%) were male and 16 (18.6%) female. Table 7.1 below shows the results of statistical tests comparing this subsample with the remainder of the participants.

**Table 7.1** Between-group comparisons across several descriptors and dependent variables

Variable	Mann-Whitney tests		
	EF subset (n = 86) median	remainder (n = 367) median	Z-score (effect size)
Age at admission	35	43	3.25** (.15)
Age at injury	34	40	2.20* (.10)
Chronicity	9	7	1.29 (.06)
Education	10	10	0.84 (.04)
GCS (n = 195)	5 (n = 43)	4 (n = 152)	0.95 (.07)
PTA (n = 117)	70 (n = 37)	60 (n = 49)	0.14 (.01)
$\chi^2$ tests			
	EF subset (n = 86)	remainder (n = 367)	$\chi^2$ (effect size)
Sex	M70:F16	M279:F88	1.14 (.05)
Type (TBI v non-TBI)	T57:N29	T211:N156	2.23 (.07)
Verbal aggression	Y43:N43	Y223:N144	3.33 (.09)
Physical aggression	Y22:N64	Y146:N221	6.02* (.12)
Inappropriate sexual behaviour	Y19:N67	Y123:N244	4.22* (.10)

\* p < .05. \*\* p < .01.

Of particular note is that, while there was no difference between the two groups in terms of the presence of verbal aggression, the subsample with executive function data had a lower proportion of participants exhibiting physical aggression and inappropriate sexual behaviour. The two groups did not differ in the ratio of sex or type of injury, nor did they differ on chronicity or years of education. Importantly, the two groups did not differ on severity of injury, as measured by Glasgow Coma Scale (GCS) score or duration of post-traumatic amnesia (PTA) when this information was available. However, the group with data was both younger at admission and injured

at an earlier age than the group without data. The effect sizes of these statistically significant differences were small.

### **7.2.2. Materials**

The BARS (Freeland et al., 2012) had been used in both samples to record episodes of verbal and physical aggression during the assessment period. Although inappropriate sexual behaviour had been recorded with different methods, it will be recalled that the SASBA (Knight et al., 2008) data used in the larger sample could be recoded in a manner consistent with the smaller sample. As the current study was only addressing the *presence* of the dependent behavioural variables, differences between the two studies in categorising forms of inappropriate sexual behaviour when they were present were irrelevant.

Regarding the neurocognitive data, the six factor structure (Tulsky, 2003; Tulsky & Price, 2003) of the Wechsler Adult Intelligence Scale – Third Edition (Wechsler, 1997a) and the Wechsler Memory Scale – Third Edition (Wechsler, 1997b) were again used, in keeping with the earlier study (chapter 5). Normative T-scores, with a mean of 50 and standard deviation of 10, are produced. As discussed above, measures of executive function were also obtained from the D-KEFS tasks of Trail Making Test (condition 4: Number-Letter Sequencing), Verbal Fluency Test (condition 1: Letter Fluency), Color-Word Interference Test (condition 3: Inhibition) and Tower Test (Rule-Violations-Per-Item-Ratio). The executive measures produce scaled scores with mean 10 and standard deviation 3.

### **7.2.3. Procedure**

Due to sample size restrictions in this study, the premorbid psychosocial and injury-related independent variables were not included in the logistic regression models. These had been adequately addressed in the previous two studies in this thesis. For the ten variables in this study (six neurocognitive and four executive), the criterion for inclusion into the final set of predictor variables for logistic regression analyses was that of being significantly correlated with any of the three dependent variables. A conservative level of significance was set ( $p < .10$ ). The final set of predictor variables were then to be used to create separate logistic regression models (forced entry method) for verbal aggression, physical aggression and inappropriate sexual behaviour.

## **7.3. Results**

Table 7.2 below shows the descriptive statistics for the ten independent predictor variables. As a group, the subsample showed considerable cognitive impairments. Particularly impaired were the Processing Speed Index, Auditory Memory and Visual Memory from the WAIS-III/WMS-III six factors, consistent with well-established patterns of cognitive impairment following acquired brain injury (Lezak, 1995) and the Trail Making Test condition 4 from the D-KEFS.

**Table 7.2 Descriptive statistics for independent variables**

	n	min	max	mean	SD
Verbal Comprehension Index	86	21	73	45.70	10.51
Perceptual Organisation Index	86	20	65	41.53	10.28
Processing Speed Index	86	7	64	32.84	10.06
Working Memory Index	86	17	78	41.42	10.48
Auditory Memory	86	14	71	37.40	14.44
Visual Memory	86	13	60	32.06	11.66
Trail Making Test	86	1	11	4.26	3.48
Verbal Fluency Test	86	1	15	6.35	3.35
Color-Word Interference Test	86	1	13	6.92	4.05
Tower Test	86	1	12	8.50	3.22

Correlations between the independent and dependent variables for this subsample from the combined datasets are presented in Table 7.3. It can be seen that, when using a significance level of  $p < .10$ , there were five dependent variables that produced significant correlations. For four of these (Verbal Comprehension Index, Perceptual Organisation, Working Memory and Tower Test), poorer scores were associated with presence of the outcome behaviour. However, the correlations between Verbal Fluency Test scores and all three behavioural groupings were in the opposite direction, in which better scores were related to the presence of outcome behaviour. This relationship was significant for the inappropriate sexual behaviour group. The sizes of these correlations were small but close to medium (Cohen, 1988, 1992).

**Table 7.3 Correlations between predictor and dependent variables**

	n	Verbal aggression	Physical aggression	Inappropriate sexual behaviour
Verbal Comprehension Index	86	-.11	-.19*	.00
Perceptual Organisation Index	86	-.19*	-.06	-.01
Processing Speed Index	86	-.03	.13	.04
Working Memory Index	86	-.19*	.04	.09
Auditory Memory	86	-.06	-.17	.10
Visual Memory	86	-.09	.04	.03
Trail Making Test	86	-.11	.09	.12
Verbal Fluency Test	86	.11	.13	.20*
Color-Word Interference Test	86	-.06	-.02	-.04
Tower Test	86	-.25**	-.12	-.28**

\*  $p < .10$ . \*\*  $p < .05$ .

The five predictor variables that produced significant correlations were then used to produce serial logistic regression models for verbal aggression, physical aggression and inappropriate sexual behaviour. These are reported separately below.

### 7.3.1. Verbal aggression

As there were an equal number of participants in both groups for verbal aggression, the null model was 50% accurate. The resulting logistic regression model was significant ( $p < .05$ ) and the classification accuracy increased to 66.3%. In terms of individual predictors, only the Verbal Fluency Test and Tower Test proved significant. The predictive relationships of these two variables were the same as in the univariate correlations: the odds of verbal aggression having been observed were greater with higher scores on the Verbal Fluency Test and lower scores on the Tower Test. The final model for verbal aggression is presented below in Table 7.4, which also includes the models for physical aggression and inappropriate sexual behaviour for ease of comparison.

There were no residual outliers in this model, defined as absolute standardised residual values greater than 2. DFBeta values (measures of the influence of a case on the value of  $b$ ) for all predictors were less than 1 (maximum value .04). There were no problematically high values of Cook's distance (maximum value .36) and, although around 42% of leverage values were greater than expected ( $(k + 1)/N = 0.0698$ ), this was not considered problematic given the acceptability of the other residual statistics (Field, 2005).

**Table 7.4 Results of final model logistic regression analyses predicting group membership for presence of behavioural disturbance**

	B (SE)	Lower	95% CI for exp $b$	
			exp $b$	Upper
<b>Verbal aggression</b>				
$R^2 = .12$ (H & L), .14 (C & S), .19 (N); Model $\chi^2(5) = 13.20, p < .05$ .				
Constant	3.72* (1.52)	-	41.32	-
Verbal Comprehension Index	-0.03 (0.03)	0.93	0.97	1.03
Perceptual Organisation Index	-0.02 (0.03)	0.93	0.99	1.04
Working Memory Index	-0.04 (0.03)	0.91	0.96	1.02
Verbal Fluency Test	0.19* (0.09)	1.02	1.21	1.43
Tower Test	-0.17* (0.09)	0.72	0.85	0.99
<b>Physical aggression</b>				
$R^2 = .11$ (H & L), .11 (C & S), .16 (N); Model $\chi^2(5) = 9.62, n.s.$				
Constant	1.19 (1.53)	-	3.27	-
Verbal Comprehension Index	-0.07* (0.03)	0.87	0.93	0.99
Perceptual Organisation Index	-0.01 (0.03)	0.93	0.99	1.06
Working Memory Index	0.02 (0.03)	0.96	1.02	1.09
Verbal Fluency Test	0.19* (0.09)	1.01	1.21	1.44
Tower Test	-0.08 (0.08)	0.78	0.92	1.08
<b>Inappropriate sexual behaviour</b>				
$R^2 = .16$ (H & L), .13 (C & S), .21 (N); Model $\chi^2(5) = 12.41, p < .05$ .				
Constant	-1.05 (1.58)	-	0.35	-
Verbal Comprehension Index	-0.02 (0.03)	0.92	0.98	1.05
Perceptual Organisation Index	-0.01 (0.04)	0.92	0.99	1.07
Working Memory Index	0.04 (0.04)	0.96	1.04	1.12
Verbal Fluency Test	0.19* (0.09)	1.00	1.20	1.45
Tower Test	-0.24** (0.09)	0.66	0.79	0.94

H & L = Hosmer & Lemeshow; C & S = Cox & Snell; N = Naglkerke.

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

In order to examine the possibility of multicollinearity, the same predictors and independent variable were entered into a standard linear regression analysis. This

provided values for tolerance and variance inflation factor (VIF), for which a “commonly used rule of thumb” (p.423, Cohen et al., 2003), is that scores less than .10 or greater than 10 (respectively) are evidence of serious multicollinearity. No independent variables came close to these criteria, with the lowest tolerance score .60 and highest VIF of 1.66 (for Working Memory Index). Examination of the smallest eigenvalues produced by PASW 18.0 collinearity diagnostics did not reveal any problematic variables. Multicollinearity was not re-examined for the other models as the same set of predictor variables was used.

### **7.3.2. Physical aggression**

The null model that no participants in this subsample exhibited any physical aggression had an accuracy rate of 74.4%. Following logistic regression, a model was produced that was not statistically significant and the classification accuracy was unchanged. However, two of the independent variables were significant predictors: Verbal Comprehension Index and Verbal Fluency Test. The directions of these relationships were as in the correlations, in which a greater likelihood of physical aggression was associated with poorer scores on Verbal Comprehension Index and better scores on Verbal Fluency Test. The results of the logistic regression model for physical aggression are presented above in Table 7.4.

Examination of the residuals revealed a single outlier (1.2%) with a standardised residual of 2.91. DFBeta values (measures of the influence of a case on the value of  $b$ ) for all predictors were less than 1 (maximum value .03). There were no

problematically high values of Cook's distance (maximum value .62) and around 34% of leverage values were greater than expected ( $(k + 1)/N = 0.0698$ ).

### **7.3.3. Inappropriate sexual behaviour**

The null model that no participants exhibited inappropriate sexual behaviour successfully classified 77.9% of cases. The logistic regression model was significant, although the classification accuracy dropped slightly to 76.7%. There were two significant individual predictors that raised the likelihood of inappropriate sexual behaviour having been observed: higher scores on Verbal Fluency Test and poorer scores on Tower Test. Indeed, scores on the Tower Test were significant at  $p < .01$ .

The model is presented above in Table 7.4.

There were three outliers with one (3.19) having a standardised residual value exceeding 3. Inspection of this case revealed that, while in the observed inappropriate sexual behaviour group, the case had been predicted to be in the no behaviour group. Scores on Verbal Comprehension Index ( $T = 31$ ), Perceptual Organisation ( $T = 42$ ), Working Memory Index ( $T = 38$ ) and Verbal Fluency Test (scaled score = 3) were all low average or below average, while Tower Test (scaled score = 10) was well within the average range. Essentially, while this participant exhibited inappropriate sexual behaviour (verbal comments only), their scores on the two significant predictor variables were both in the opposite direction from the model.

DFBeta values (measures of the influence of a case on the value of  $b$ ) for all predictors were less than 1 (maximum value .08). Around 34% of leverage values were greater than expected ( $(k + 1)/N = 0.0698$ ).

One case had a value of Cook's distance that exceeded the recommended (Cohen et al., 2003) cut-off of 1.0 (actual value 1.43), indicating that this single case may have been exerting undue influence on the regression model. Closer inspection of this case revealed that it had been incorrectly predicted to be in the inappropriate sexual behaviour group. The participant had scored in the average range or better on Verbal Comprehension ( $T = 73$ ), Perceptual Organisation Index ( $T = 51$ ) and Working Memory Index ( $T = 50$ ). On the two significant predictor variables, this participant produced the highest observed score on the Verbal Fluency Test (scaled score = 15) and the lowest possible score on the Tower Test (scaled score = 1). The effect of these extreme scores may well have been lessened in a larger sample.

## 7.4. Discussion

This study used a subsample of 86 participants from the combined datasets of studies 2 and 3 (chapters 5 and 6 in this thesis) to investigate the relationship between executive function and observed verbal aggression, physical aggression and inappropriate sexual behaviour. Specifically, it was anticipated that poorer scores on the selected measures of executive function would be associated with a greater likelihood of behavioural disturbance being observed, even when measures of lower-order neurocognitive function were included.

The results were not entirely as anticipated. Two of the four included measures of executive function proved to be significant predictors in the logistic regression models. The D-KEFS Tower Test rule violations, considered a measure of impulsivity and inhibition, significantly predicted both verbal aggression and inappropriate sexual behaviour in the anticipated direction. That is, poorer performances on the measure were associated with the presence of those behaviours. However, for the letter fluency condition of the D-KEFS Verbal Fluency Test, which predicted group membership in all three dependent variables, the relationship was in the opposite direction. Better performance on the task was associated with the presence of behavioural disturbance. The Verbal Comprehension Index was associated with the presence of physical aggression but not verbal aggression or inappropriate sexual behaviour. The other neurocognitive and executive measures of Perceptual Organisation Index, Processing Speed Index, Working Memory Index, Auditory Memory, Visual Memory, Trail Making Test and Color-Word Interference Test had no significant relationships with the behavioural outcomes.

Relatively high numbers of rule violation errors on the Tower Test (equating with low scaled scores on the variable) were a significant predictor of both verbal aggression and inappropriate sexual behaviour. Interestingly, neither of the studies by Greve et al. (2001) or Wood and Liossi (2006b) had included a similar task in their measures of executive function. Excessive rule violations on versions of the Tower Test have been associated with focal frontal lobe lesions in both adults (Yochim et al., 2009) and children (Jacobs & Anderson, 2002; Levin et al., 1993). Excessive rule violations on the Tower Task differentiated a group of patients with frontotemporal dementia

from both control and Alzheimer's Disease groups (Carey et al., 2008), when both clinical groups performed equally poorly on the overall achievement scores.

In order to avoid making rule violations on the Tower Test, the examinee must be able to keep in mind the specific rules, monitor their own performance and then inhibit any potential rule violations. A trade-off between speed and accuracy is required, for which error analysis is crucial to understanding some neuropsychological impairments. This was well illustrated in the case reported by Cato, Delis, Abildskov, and Bigler (2004), in which ventromedial prefrontal damage produced marked impairments in error production in the context of average to superior results on most traditional neuropsychological tests.

Reduced inhibition as evidenced by poor Tower Test performance has been related to poor self-monitoring skills and less awareness in patients with schizophrenia (Lysaker et al., 2006), although they did not specifically analyse the rule violations measure. The notion that poor self-monitoring/self-awareness could be related to the behavioural outcomes in this analysis is consistent with the findings from studies 1 and 3 (chapters 4 and 6 in this thesis). Items reflecting self-awareness from the NRS (study 1) and the MPAI-4 (study 3) were associated with behavioural outcome. Prior studies outside the brain injury literature have also linked impairments in self-awareness to aggression (Alia-Klein et al., 2007; Bo et al., 2011; Giancola et al., 2011; Giancola et al., 2009; Schaub et al., 2012). However, regarding inappropriate sexual behaviour after brain injury, Simpson et al. (2001) reported that both their "sexually aberrant" group and a comparison group not exhibiting such behaviours were equally impaired in terms of clinician-rated awareness. The current findings

therefore require independent repetition in terms of post-injury inappropriate sexual behaviour.

Scores on the letter fluency condition of the D-KEFS Verbal Fluency Test were associated with the presence of all three behaviours. However, this was not in the anticipated direction, as higher scores on the test (reflecting greater generativity) were significantly related to the presence of the behavioural disturbances. This finding is in contrast to previous comparable studies in aggression, in which performances on verbal fluency tasks did not differ between aggressive and non-aggressive groups (Greve et al., 2001; Wood & Liossi, 2006b).

At first, this result may seem surprising. However, positive correlations between poorer performances on verbal fluency tasks and poorer general behavioural output have been reported previously. For instance, negative symptoms of schizophrenia (including a general lack of behaviour) have been consistently associated with poorer verbal fluency scores (Basso, Nasrallah, Olson, & Bornstein, 1998; Cochrane, Petch, & Pickering, 2012; Liddle & Morris, 1991; O'Leary et al., 2000; Szulc et al., 2012; Woodward, Ruff, Thornton, Moritz, & Liddle, 2003).

The only study in the literature reporting an association between better performance on a verbal fluency task and the presence of aggression (verbal but not physical) was in seven year old children (Tacher & Readdick, 2006). It was suggested that the more creative children (in terms of verbal fluency ability) were able to utilise verbal skills (including verbal aggression) when reacting to and resolving difficult situations within the school environment. It is tempting to speculate that behavioural drive,

which may be altered following acquired brain injury (Lezak, 1995; Wood, 2001) is an important factor in determining whether a person will exhibit post-injury verbal aggression, physical aggression and inappropriate sexual behaviour. For instance, people with impaired drive after brain injury may be less likely to exhibit these behaviours. As such, it may be that a person's performance on letter fluency tasks is an indirect measure of behavioural drive. Clearly, these unexpected findings require replication elsewhere.

The Simpson et al. (2001) study reported that clinician-rated variables of generativity and drive did not differ between their "sexually aberrant" group and matched controls. No other studies could be found in the literature that specifically addressed executive function, let alone verbal fluency performance, in a clinical sample of people displaying inappropriate sexual behaviour.

Poor verbal skills, as measured by the Verbal Comprehension Index of the WAIS-III, were associated specifically with the presence of physical aggression but not verbal aggression or inappropriate sexual behaviour. The Verbal Comprehension Index was predictive for both verbal aggression and physical aggression in study 2 (chapter 5 in this thesis) and the communication item of the MPAI-4 loaded onto the only factor that significantly predicted the presence of the three behavioural outcomes in study 3 (chapter 6 in this thesis). This effect remained with the combining of the two datasets. Poor verbal skills have been previously associated with aggressive behaviours that are commonly seen in brain injury rehabilitation programmes (Alderman, 2007; Alderman et al., 2002). It has been hypothesised that linguistic processing may help in regulating those executive functions such as abstract

reasoning, cognitive modulation of emotion and reflection that are necessary to inhibit aggressive impulses (Miller et al., 2008).

The multivariate models for presence of verbal aggression, physical aggression and inappropriate sexual behaviour formed in this study were rather modest, explaining only 19%, 16% and 21% of the variance in the sample, respectively. It has been known for some time that executive function tests show limited ecological validity to observations outside the testing laboratory (Chaytor, Schmitter-Edgecombe, & Burr, 2006; Cripe, 1996; Odhuba, van den Broek, & Johns, 2005; Wood & Liossi, 2006a). This study also suggests that neuropsychological measures of executive function are only small factors in determining which people exhibit these behavioural disturbances following severe brain injury.

## **CHAPTER 8. GENERAL DISCUSSION**

This thesis had two main aims. Firstly, in order to address a major gap in the brain injury literature, analyses of contemporaneously recorded verbal aggression, physical aggression and inappropriate sexual behaviour were to be conducted within the same samples of participants. These analyses would provide crucial evidence as to whether these serious disorders of interpersonal behaviour following brain injury should be conceptualised within a general behavioural dysregulation or whether they represented separate clinical entities. Secondly, the clinical correlates of verbal aggression, physical aggression and inappropriate sexual behaviour were also to be explored. Previous research had indicated that forms of aggressive behaviour after brain injury were indeed related to various clinical variables, but little work had been done in the area of inappropriate sexual behaviour and no such relationships had yet been reported. This chapter summarises the separate studies conducted towards these two aims and then integrates the work as a whole with previous research. Finally, recommendations for clinical practice and further research efforts are made.

### **8.1. Summaries of individual studies**

Four separate pieces of empirical research were undertaken and are documented in this thesis. In this section, the results of each study will be briefly summarised.

### **8.1.1. Study 1 – Development of the BARS**

The first study involved the development of the BIRT Aggression Rating Scale (BARS; Freeland et al., 2012), an observational rating scale specifically designed to provide a reliable and valid measure of aggression for persons with acquired brain injury. The BARS differentiates between verbal and physical aggression, with three levels of severity of behaviour within each. It was developed to objectively record various forms of aggressive behaviour that reflect both legal concepts and common understanding of aggression. In order to minimise human error associated with recalling previous incidents from memory, recordings of behaviour are made by trained staff in a structured manner as soon as possible after the incident. The BARS is intended to be used at an individual level for clinical purposes as well as at group level for organisational audit and clinical research.

Two separate pieces of work were presented in chapter 4. The first established adequate inter-rater reliability in a large sample of rehabilitation staff. The author was not primarily involved in this part of the study, as declared on page 11, although it was included in the thesis to provide context for the second part of the study. This was concerned with the concurrent validity of the BARS and was conducted solely by the author. Organisational outcome data were analysed in a sample of 309 participants with acquired brain injury who had been involved in residential neurobehavioural rehabilitation. The highest aggregate aggression score during the first four weeks of admission showed small but significant correlations with the total score from the Neurobehavioural Rating Scale (Levin et al., 1987) and the Supervision Rating Scale (Boake, 1996), consistent with previous work showing that

post-injury functional status was related to aggression (Alderman, 2007; Alderman et al., 2002; Kerr et al., 2011). A number of individual Neurobehavioural Rating Scale items produced small to medium sized significant correlations with aggression scores. These included the presence of psychiatric symptoms such as unusual thoughts or hallucinations and depressive or labile mood and anxiety, as well as poor communication skills and impaired self-awareness.

### **8.1.2. Study 2 – Neurocognitive predictors**

The second study addressed two specific research questions. Firstly, separation of verbal aggression, physical aggression and inappropriate sexual behaviour was indicated following exploratory factor analysis of the observed behaviours demonstrated by a sample of 152 participants with severe acquired brain injury. Secondly, potential clinical correlates of verbal aggression, physical aggression and inappropriate sexual behaviour were investigated with separate logistic regression models in a subsample of 77 participants.

Independent predictors consisted of premorbid psychosocial, injury-related and neurocognitive variables. Only one (Verbal Comprehension Index) of six measures of neurocognitive function proved to have any predictive relationship in logistic regression modelling for the presence of verbal aggression and physical aggression. Prescribed use of psychotropic medication was also associated with both verbal and physical aggression. A premorbid history of aggression was related to verbal aggression only. A logistic regression model could not be formed for inappropriate sexual behaviour and there were no significant individual predictors. Limitations in

the sample size of participants for whom neuropsychological test scores were available meant that measures of cognitive executive function could not be included in that study.

### **8.1.3. Study 3 – Neurobehavioural predictors**

The third study in this thesis was conducted with a larger sample of 301 participants with acquired brain injury. The participants were recruited from seven different rehabilitation centres around the UK and presented with a wider range of neurobehavioural presentation than was captured in the previous study. The study involving this larger sample was designed to address three specific research questions. Firstly, the prior behavioural separation between verbal aggression, physical aggression and inappropriate sexual behaviour was replicated in this sample. Replication of previous results is a fundamental part of the scientific process (Schmidt, 2009). As such, studies 2 and 3 provided compelling evidence that these distinctions were valid and, therefore, needed to be considered in further work involving verbal aggression, physical aggression and inappropriate sexual behaviour.

Secondly, the larger sample size of study 3 permitted the use of two-part regression modelling that has been recommended when analysing data sets with excessive zero counts (Lachenbruch, 1992, 2001a, 2001b, 2002). In this method, presence or absence of the dependent variables is initially modelled via logistic regression and then the non-zero values of the dependent variable are separately modelled via linear regression. Although this technique has been used previously in healthcare research (Chang & Pocock, 2000; Delucchi & Bostrom, 2004; Schneider et al.,

2007), it is the first time that it has been applied in either brain injury research or in studies addressing forms of aggression or inappropriate sexual behaviour.

Two-part modelling was successfully achieved for verbal aggression. However, only a truncated version of the linear regression models could be achieved for physical aggression and inappropriate sexual behaviour. A range of independent predictor variables were used: premorbid psychosocial, injury-related and measures of current functioning. Current functioning was measured with the fourth version of the Mayo-Portland Adaptability Inventory (Malec & Lezak, 2003, April), the Care and Needs Scale (Tate, 2004) and the Supervision Rating Scale (Boake, 1996).

The presence of verbal aggression was associated with male sex, having sustained a traumatic brain injury, being admitted six months or more post-injury and higher scores on MPAI-4 subscales of Adjustment and Participation (reflecting poor adjustment and poorer social participation, respectively). When present, the degree of verbal aggression exhibited by participants was associated with poorer educational attainment (below GCSE level), having sustained a traumatic brain injury and higher scores on the Adjustment subscale.

The presence of physical aggression was associated with male sex, having been admitted six months or more post-injury and MPAI-4 Adjustment. When physical aggression was exhibited, higher frequency and/or severity of behaviour was strongly associated (medium effect size) with higher scores on the Participation subscale of the MPAI-4.

For inappropriate sexual behaviour, presence was associated with male sex, a premorbid history of substance abuse, having sustained brain injury at a younger age, higher MPAI-4 Adjustment scores and higher CANS ratings. When present, higher frequency and/or severity of inappropriate sexual behaviour was associated with higher MPAI-4 Adjustment scores only.

The third and final research question for study 3 concerned the structure of the MPAI-4 itself and what additional information could be obtained by utilising the underlying factor structure of the 29 items, rather than the 3 rationally derived subscales, in the development of models of behaviour. Principal component analysis revealed an 8 factor structure, similar to previous research (Bohac et al., 1997; Malec et al., 2003). Importantly, this analysis revealed that only one factor was associated with the presence of all three behavioural outcomes. Loading alongside individual items for “irritability, anger and aggression” and “inappropriate social interaction” (which should have associated strongly with outcomes) were the items for “communication” and “impaired self-awareness”, consistent with the findings from studies 1 and 2. Other items representing cognition and emotion loaded on separate factors that were not associated with the presence of verbal aggression, physical aggression or inappropriate sexual behaviour.

#### **8.1.4. Study 4 – The role of executive function**

The research question posed by study 4 was to explore the relationships between executive function and the presence of verbal aggression, physical aggression and inappropriate sexual behaviour. Although consistent relationships had been reported

in other fields, there has been limited work conducted on this within the brain injury literature. It was anticipated that the presence of these behaviours would be associated with poorer performances on neuropsychological measures of executive function.

Despite using different sets of predictor variables in studies 2 and 3, the structure of the behavioural observations was shown to be essentially the same in both samples. Therefore, study 4 combined the two separate datasets from studies 2 and 3 to form a larger dataset of 453 participants with acquired brain injury. This allowed the selection of a subsample of 87 participants for whom complete data on measures of neurocognitive (six variables) and executive function (four variables) were available. There was no difference between this subsample and the remaining participants in the proportion who exhibited verbal aggression, however, the participants in the subsample showed lower proportion of physical aggression and inappropriate sexual behaviour. The two groups did not differ in education level, chronicity or severity of injury (as measured by GCS and PTA where available), although the subsample was significantly younger at both age when injured and age at admission than were the remaining participants.

For the subsample of 86 participants, separate logistic regression models were constructed using five predictor variables selected from the univariate correlations. Consistent with the previous studies in this thesis and with prior published work, poorer verbal skills were again associated with the presence of physical aggression, although not for verbal aggression. No other measures of neurocognitive function (visuo-spatial reasoning, speed of information processing, working memory or

anterograde memory) were associated with any behavioural outcome. Regarding the independent variables of executive function measures, greater numbers of rule violations on the D-KEFS Tower Test (thought to reflect poorer inhibition of impulses) was associated with the presence of verbal aggression and particularly strongly with the presence of inappropriate sexual behaviour. No such relationship was found for physical aggression. Additionally, and contrary to expectations, the presence of all three behaviours were associated with better performances on the letter fluency condition of the D-KEFS Verbal Fluency Test. While requiring independent replication, it was suggested that the participants' performances on this measure may have reflected behavioural drive in this sample with brain injury.

## **8.2. The distinctions between verbal aggression, physical aggression and inappropriate sexual behaviour**

One of the main aims of this thesis was to explore the relationships between verbal aggression, physical aggression and inappropriate sexual behaviour following brain injury. There is a growing literature in this area involving group studies of aggression (Alderman, 2007; Alderman et al., 2002; Baguley et al., 2006; Greve et al., 2001; Kerr et al., 2011; Tateno et al., 2003; Visscher et al., 2011; Wood & Liossi, 2006b). However, far less empirical work has been conducted with inappropriate sexual behaviour (Alderman et al., 2009; Simpson et al., 1999; Simpson et al., 2001).

### **8.2.1. Distinguishing between aggression and inappropriate sexual behaviour**

Only one published paper has addressed both aggression and inappropriate sexual behaviour in the same sample of participants with acquired brain injury (Alderman et al., 2009). The authors reported a clinical audit of inappropriate sexual behaviour and aggression observed over a three month period within a neurobehavioural treatment hospital. It was noted that far fewer patients exhibited inappropriate sexual behaviour than aggression and that there were far fewer incidents of inappropriate sexual behaviour in total. Although the authors did not conduct rigorous analyses of both forms of behaviour together, they did report that there was only a “modest” correlation between the two, concluding “at a broad level, the two categories of behavior have different aetiologies” (p. 218). Further, examination of setting events and antecedents suggested that there were different functional elements to the behaviours. Specifically, the authors felt that while aggressive behaviour operated as an avoidance or escape mechanism by patients, inappropriate sexual behaviour served to initiate contact with others as a way of expressing sexual needs given the limited opportunities that their circumstances allowed. A difference in functional analysis between aggression and inappropriate sexual behaviour was also proposed in a single case report of a person displaying both forms of behaviour (Kelly & Simpson, 2011).

The results from the current studies 2 and 3 indicated a clear distinction between aggression and inappropriate sexual behaviour, in keeping with the two published reports above. Both of the current studies used the BARS (Freeland et al., 2012) to record aggressive behaviours as they occurred during rehabilitation, however,

sexually inappropriate behaviour was recorded differently for each. In Study 2, initiated prior to the publication of the SASBA (Knight et al., 2008), incidents of inappropriate sexual behaviour were coded as S1 for verbal comments and S2 for actions. Study 3 was able to incorporate the SASBA into the design and therefore 16 separate codes (4 categories by four severity levels) were available for inappropriate sexual behaviour. When the 16 SASBA codes were recoded into an S1 and S2 format, the underlying factor structure following principal component analysis was the same. Consistent with the audit conducted by Alderman et al. (2009), some of the 16 individual cells were not observed frequently. For instance, although all 16 SASBA codes were recorded at least once in the whole sample, NC4 (referring to Non-Contact category of the highest severity) was recorded for only 2 (0.7%) of 301 participants and E4 (Exposure category of the highest severity) was recorded for 4 (1.3%) of participants. As a result, all 16 SASBA codes could not be included in a second principal component analysis. Instead, all recordings within a category were summed to leave four SASBA variables rather than the two variables for inappropriate sexual behaviour used in study 2. Nevertheless, all four SASBA variables loaded onto a single factor, separate to the BARS aggression variables.

These results provided strong evidence that, although some participants exhibited both aggressive and inappropriate sexual behaviours, the two should be considered fundamentally distinct clinical entities. The concept of a general disinhibition syndrome (Kim, 2002; Starkstein & Kremer, 2001) to account for incidents of aggression and inappropriate sexual behaviour was not supported by these findings. Further research incorporating participants with higher frequency and more varied

inappropriate sexual behaviour that would permit utilisation of all sixteen SASBA codes is warranted.

### **8.2.2. Different types of aggression?**

Little previous work has been conducted on this issue. A group of people with traumatic brain injury showed higher levels of verbal aggression than control groups (spinal cord injury and an uninjured group) but not physical aggression (Dyer et al., 2006) and this was associated with greater impulsivity. Only one of 19 aggressive participants studied by (Rao et al., 2009) exhibited physical aggression. Most of the high level of aggressive behaviours recorded by Alderman and colleagues (Alderman, 2007; Alderman et al., 2002) was noted to be verbal rather than physical in nature and these showed different setting events and antecedents.

The principal component analyses conducted in studies 2 and 3 both concluded that, in addition to differentiating between aggression and inappropriate sexual behaviour, a distinction between verbal aggression and physical aggression was also indicated. This was based on specific decisions made within the methodology of the analyses. For instance, using a traditional eigenvalue cut-off of 1.0 would have suggested that only a distinction between aggression and inappropriate sexual behaviour was valid. However, more recent statistical advice considers this to be a rather conservative criterion (Jolliffe, 2002). Use of a cut-off value of 0.7, along with visual inspection of the screeplot (Hutcheson & Sofroniou, 1999) clearly indicated that three components should be retained. As the same results were obtained for both studies 2 and 3, it

was therefore determined that there was empirical justification for treating verbal aggression and physical aggression separately in the current set of studies.

As these analyses are unprecedented, independent verification of these distinctions is required in different samples. It will be interesting to see whether a verbal/physical distinction regarding aggression is supported with the use of instruments other than the BARS that are in common use in brain injury rehabilitation, such as the OAS-MNR (Alderman et al., 1997).

### **8.3. Clinical correlates of verbal aggression, physical aggression and inappropriate sexual behaviour**

With the above distinctions determined, the second major aim of this thesis was to investigate the clinical correlates of verbal aggression, physical aggression and inappropriate sexual behaviour. The identification of predisposing factors and clinical variables that lead to disorders of interpersonal behaviour is crucial in establishing which particular individuals may be at risk and how best to provide specialist services that will meet their needs. Such specialist services are increasingly expensive to provide (Kreutzer et al., 2001) and cost-effectiveness is key in the current healthcare climate (Wood et al., 1999; Worthington et al., 2006).

Investigation of clinical correlates formed a core component for each of the four studies. The first study was limited in using non-parametric correlations to explore the relationship between a generic aggression measure generated by the BARS and individual items from the Neurobehavioural Rating Scale (Levin et al., 1987).

However, subsequent studies were designed so that regression techniques could be used to model the observed behaviours. Regression is a more powerful tool than univariate correlations in that multivariate models may be constructed that allow predictions of the outcome variables (Field, 2005). The contribution of individual predictor variables to the dependent variables can also be tested in the presence of other predictors.

### **8.3.1. Accuracy of models**

The majority of studies addressing the clinical correlates of aggression have used univariate between-group analyses to differentiate participants who behaved aggressively from those that did not. However, there have been several previous studies that used regression modelling to predict aggression following acquired brain injury. Typically these have involved logistic regression, in which the dependent variable is dichotomous – aggressive group versus non-aggressive group. The studies presented in this thesis represent the first time that any researcher has attempted to model post-injury inappropriate sexual behaviour. How well do regression models predict aggressive behaviour after brain injury and how do the present results compare with these?

#### **8.3.1.1. Aggression**

The three previously published logistic regression studies (Kerr et al., 2011; Rao et al., 2009; Visscher et al., 2011) showed similar rates of overall classification. Some of the key indicators when weighing up how well a logistic regression model fits the

data include the base rate of the condition (i.e., the null model), the classification accuracy of the final model and the total variance explained by the model. Unfortunately these have not been consistently reported.

Rao et al. (2009) conducted a logistic regression model to predict group membership in a sample for which 19 out of 67 (28.4%) participants exhibited aggression after traumatic brain injury. Seven independent variables were used, of which five were reported as being significant individual predictors of group membership. The null model of no one exhibiting any aggression would have successfully predicted 71.6%; the authors did not report the classification accuracy or the variance explained of their final model.

Visscher et al. (2011) created a logistic regression model of the aggressive behaviour displayed by 24 (42.1%) of their 57 participants with brain injury. Four independent predictors were used and the final model was reported to correctly classify 82% of participants into the correct groups. The amount of variance explained by this model was not reported.

Kerr et al. (2011) also performed a logistic regression analysis with their sample of 46 participants with acquired brain injury. The sample was evenly split between those showing aggression and those not so that the null model correctly classified 50%. Five predictor variables were used and the final model classification accuracy was reported to be 82.8% with 61.4% of the total variance explained by the model.

In this thesis, the separate logistic regression modelling of verbal aggression and physical aggression was conducted four separate times over three chapters. The weakest performing models were those that included only measures of neurocognitive and executive function (study 4): verbal aggression group membership showed 66.3% correct classification (null model of 50%), explaining only 19% of the variance; physical aggression group membership showed 74.4% correct classification (unchanged from the null mode), explaining only 16% of the variance. The best performing models were those including the MPAI-4 (study 3): verbal aggression was correctly classified in 76.1% of cases (null of 57.7%), explaining 37% of the variance; physical aggression was correctly classified in 74.4% of cases (null of 63.5%), explaining 34% of the variance.

The best performing models presented in this thesis appear to fall short of two of the three studies published in the literature. However, the samples used in all three of those studies were relatively small in size, indeed smaller than any study reported in this thesis. No information was presented about outliers and residual diagnostics. It is, therefore, difficult to draw firm conclusions about how well those models performed. Of note is that, even if the Kerr et al. (2011) results are taken at face value, there remains a relatively large proportion of variance in the outcome variable that remains unexplained. Additionally, these studies involved samples of people with acquired brain injury that appeared to be less severely injured and less severely impaired in terms of functional skills. It may be that the variables that predicted outcome in this thesis have different effects across the ranges of severity of injury.

Alderman (2007) published what appears to be the only linear regression model of aggressive behaviour after brain injury. Four neurobehavioural predictor variables were used in a series of models created to predict the frequency and severity of the four categories of aggressive behaviour present in the OAS-MNR (Alderman et al., 1997). In his sample of 108 participants,  $R^2$  did not exceed 0.24, indicating that the majority of variance remained unexplained.

In this thesis, linear regression modelling of verbal aggression and physical aggression in the subsets of participants showing these behaviours was also conducted in study 3. The amount of variance explained by a model with four predictor variables was 21% for verbal aggression ( $n = 173$ ) and 29% for physical aggression ( $n = 110$ ). These values are very similar to those reported by Alderman (2007). Although that sample involved participants with behaviour challenging enough to warrant hospitalisation in a specialist secure setting, perhaps it was the sample most similar to those utilised in this thesis in terms of severity of injury and functional impairments.

In conclusion, the regression studies of verbal aggression and physical aggression presented in this thesis produced statistically significant models that, in the case of logistic regression, were typically superior to the null models. However, the included predictor variables (premorbid psychosocial, injury-related and neuropsychological / neurobehavioural) were, at best, only able to account for a little over a third of the observed variance in the behavioural observations. Clearly, other factors mediating the expression of these aggressive behaviours after brain injury were present.

### **8.3.1.2. Inappropriate sexual behaviour**

There have been no prior regression studies of inappropriate sexual behaviour following brain injury. The one group study in this area (Simpson et al., 2001) looked at between-group differences on a number of variables. No significant differences were reported.

This thesis presents a series of regression studies modelling, for the first time, inappropriate sexual behaviour after acquired brain injury. In the second study, a logistic regression model could not be formed from a range of predictor variables in a sample of 77 participants (24 of whom had exhibited this behaviour). In the third study, a larger sample size ( $n = 301$ ) was obtained, measures of neurobehavioural function were used and a significant model constructed that accounted for 22% of the observed variance. Study 3 also constructed a linear regression model of those participants who had exhibited inappropriate sexual behaviour, designed to predict the degree of behaviour observed. This model was statistically significant, although it only accounted for 15% of the variance. Finally, study 4 involved logistic regression modelling of the presence of inappropriate sexual behaviour in a subsample of 86 participants for whom neuropsychological measures of neurocognitive and executive function were available. Using five predictor variables, a significant model was achieved accounting for 21% of the variance. However, this was not a particularly good classification model as the final accuracy was marginally below the null model.

In conclusion, this thesis presents successfully constructed regression models of inappropriate sexual behaviour after brain injury. This is unprecedented work and,

given the modest levels of explained variance, further efforts in this area are clearly warranted.

### **8.3.2. Individual predictor variables**

Multivariate regression analyses of verbal aggression, physical aggression and inappropriate sexual behaviour produced significant, albeit modest, models. Within this thesis, potential correlates have been grouped into: premorbid psychosocial variables; injury-related variables; neuropsychological and neurobehavioural variables. The first two groups of predictor variables were discussed in detail in study 4 (chapter 7). This next section will focus on the range of neuropsychological and neurobehavioural measures utilised across the four studies. What relationships emerged regarding individual predictor variables?

The Tulsky six factor structure (Tulsky, 2003; Tulsky & Price, 2003) of the WAIS-III and WMS-III was used to assess cognitive function from measures corresponding to a Verbal Comprehension Index, Perceptual Organisation Index, Processing Speed Index, Working Memory Index, Auditory Memory and Visual Memory. Measures of executive function were taken from four tests from the D-KEFS. A broader range of neurobehavioural symptoms had been captured by the Neurobehavioural Rating Scale (study 1) and the Mayo-Portland Adaptability Inventory (study 3). The following section involves a synthesis of the relevant findings for verbal skills, visuo-spatial skills, anterograde memory, executive function, self-awareness and emotional status.

### **8.3.2.1. Verbal skills**

Poorer verbal skills were consistently shown to be a risk for post-injury aggression and to have no association with inappropriate sexual behaviour in the studies reported in this thesis. The Verbal Comprehension Index was a significant predictor of the presence of both verbal and physical aggression in study 2 as well as physical aggression in study 4. Items representing verbal skills from both the NRS (“expressive deficit” and “comprehension of speech) in study 1 and MPAI-4 (“communication”) in study 3 also were strongly associated with the presence of verbal and physical aggression.

Impairments in verbal skills have previously been associated with aggressive behaviours that are commonly seen in brain injury rehabilitation programmes (Alderman, 2007; Alderman et al., 2002). It has been hypothesised that linguistic processing may help in regulating those executive functions such as abstract reasoning, cognitive modulation of emotion and reflection that are necessary to inhibit aggressive impulses (Miller et al., 2008). The lack of association with inappropriate sexual behaviour is consistent with the Simpson et al. (2001) study, which reported no differences in clinician-rated language functioning between their “sexually aberrant behaviour” group and a clinical comparison group.

### **8.3.2.2. Visuo-spatial skills**

Visuo-spatial skills, as measured by the Perceptual Organisation Index, were found to be unrelated to the presence of verbal aggression, physical aggression and

inappropriate sexual behaviour in studies 2 and 4. Similarly, in study 3, the MPAI-4 items of “vision” and “visuo-spatial abilities” are included in the Abilities subscale, which was not predictive of either the presence or degree of any behavioural outcome. Further, in the factor analysis of the MPAI-4 reported in study 3, these two items formed their own factor, consistent with previously reported factor structures of this tool (Bohac et al., 1997; Malec et al., 2003). This factor was not a significant predictor of the presence of verbal aggression, physical aggression and inappropriate sexual behaviour. There were no measures of visuo-spatial function within the items of the Neurobehavioural Rating Scale that was used in study 1.

This consistent lack of a relationship between visuo-spatial skills and behavioural outcome does not support the earlier finding by Wood and Liossi (2006b). These authors reported that, when present with impairments in executive-attention function, deficits in verbal memory and visuo-spatial abilities differentiated the aggressive from non-aggressive groups in a sample of people with traumatic brain injury. Visuo-spatial ability had also been measured with performance subtests from the WAIS-III.

Consistent with the present results, a lack of relationship between visuo-spatial skills and post-injury inappropriate sexual behaviour was also reported by Simpson et al. (2001). In that case, the authors had used clinician rating of abilities, as the neuropsychological tests had been varied.

### **8.3.2.3. Anterograde memory**

Measures of anterograde memory function were not related to the presence of verbal aggression, physical aggression and inappropriate sexual behaviour. Studies 2, 3 and 4 consistently showed that memory was not predictive of behavioural group membership, either with the Auditory Memory and Visual Memory measures in study 2 (chapter 5), the MPAI-4 memory item in study 3 (chapter 6) or with the Auditory Memory and Visual Memory measures in study 4 (chapter 7). These findings are in contrast with two previous studies that reported a significant relationship between memory function and aggression (Kockler & Stanford, 2008; Wood & Liossi, 2006b). The Kockler and Stanford (2008) study involved a sample of aggressive outpatients with no control group, so direct comparisons between the results are not applicable. Wood and Liossi (2006b), on the other hand, suggested that specific impairments in verbal memory and visuospatial abilities were associated with aggression after brain injury when these deficits were present in addition to impoverished executive-attention functioning. The results of the present studies do not support this contention. Rather, they are consistent with the lack of between-group differences across a range of memory measures reported by Rao et al. (2009).

### **8.3.2.4. Executive function**

Neurobiological models of both aggression (Siever, 2008; Volavka, 2002) and sexual behaviour (Rees et al., 2007; Spinella, 2007) highlight the importance of so-called “top-down” executive cognitive processes involved in the regulations of these

behaviours. The results from study 4 supported this contention, although not in a straightforward manner.

Excessive rule violation errors on the D-KEFS Tower Test was a significant predictor of both verbal aggression and inappropriate sexual behaviour, consistent with other research showing the utility of this variable as a measure of impulsivity or disinhibition (Carey et al., 2008; Jacobs & Anderson, 2002; Levin et al., 1993; Yochim et al., 2009). Performance on the letter fluency condition of the D-KEFS Verbal Fluency Test was also associated with the presence of all three behavioural outcomes. However, surprisingly, this was in the opposite direction than expected, as better performance on the task predicted behavioural disturbance. It was considered that this variable may have reflected a more general measure of behavioural drive rather than of behavioural control (Lezak, 1995; Wood, 2001). As such, perhaps a lack of behavioural drive in the form of post-injury adynamia or abulia results in a reduction in the likelihood of expressing verbal aggression, physical aggression or inappropriate sexual behaviour. These unexpected results clearly require independent verification.

Neither the switching task from the D-KEFS Trail Making Test or the inhibition condition from the D-KEFS Color-Word Interference Test had any significant relationships with the three behavioural outcomes. This is consistent with the reports from previous studies (Greve et al., 2001; Rao et al., 2009; Wood & Liossi, 2006b).

### **8.3.2.5. Self-awareness**

Deficits in insight or self-awareness have been reported as a common problem after acquired brain injury (Bach & David, 2006; Hart et al., 2009; Ownsworth et al., 2007; Prigatano, 2005a, 2005b; Prigatano & Altman, 1990; Sherer et al., 2003; Stuss & Anderson, 2004) and various models of awareness have been developed (Crosson et al., 1989; Schacter, 1990). The development of greater self-awareness is frequently an explicit goal within brain injury rehabilitation (Goverover et al., 2007; Lundqvist et al., 2010; Medley & Powell, 2010; Ownsworth et al., 2008; Schmidt et al., 2011).

For the two studies in this thesis that included measures of participant self-awareness, poorer scores were significantly associated with the presence of behavioural disturbance. Surprisingly, despite the importance of disorders of self-awareness after brain injury, this has not yet been included in studies involving post-injury aggression. The Simpson et al. (2001) study addressing inappropriate sexual behaviour after brain injury had reported that clinician ratings of self-awareness had not differed between their two groups. Further research in this area would do well to include robust measures of self-awareness. Importantly, specific techniques addressing the remediation of deficits of self-awareness may prove beneficial in the treatment of these behavioural disorders and this represents a particularly fruitful area for further work.

### **8.3.2.6. Emotional status**

Univariate correlations between current emotional status and aggressive behaviour were consistently significant, however, following multivariate analyses these relationships were not borne out. In study 1, NRS items of “anxiety”, “depressive mood”, “blunted affect” and “labile mood” all produced significant Spearman correlations with an aggregate aggression score on the BARS (Table 4.1). Within the eight factor structure of the MPAI-4 reported in study 3, the items “anxiety”, “depression” and “sensitivity to mild symptoms” loaded onto a single factor that showed small but significant correlations with both verbal aggression and physical aggression but not inappropriate sexual behaviour (Table 6.13). However, this factor was not significant following multivariate logistic regression (Table 6.14).

The literature regarding a potential relationship between emotional status and aggressive behaviour following brain injury is inconsistent. Univariate between-group analyses have been reported to show conflicting associations between depression and aggression, with some studies reporting a significant relationship (Baguley et al., 2006; Johansson et al., 2008; Tateno et al., 2003) but not others (Paradiso et al., 1996; Wood & Liotti, 2006b). Some previous multivariate analyses (Kerr et al., 2011; Visscher et al., 2011) did not include measures of current emotional functioning. The linear regression models of Alderman (2007) documented that ratings of mood and self-esteem had no predictive power within their aggressive sample. Rao et al. (2009) reported that a diagnosis of mood disorder at 3 months post-TBI was a significant predictor of aggression within a logistical regression model, however, a measure of self-awareness was not included as a covariate. It is

not yet clear how the complex relationship between self-awareness and mood after brain injury (Fleminger, Oliver, Williams, & Evans, 2003) may influence the expression of post-injury aggression and inappropriate sexual behaviour. This is clearly an area for further research to address.

#### **8.4. Limitations and recommendations**

The series of studies within this thesis is not without limitations. Although the sizes of the samples recruited for each study were large in comparison with previously published studies in this area, they were still too small for all planned analyses. For example, a large sample of people with acquired brain injury undergoing neurobehavioural assessment was obtained for study 3 and this permitted two-part regression modelling of observed verbal aggression. Logistic regression was used to model the presence of verbal aggression in all 301 participants and then linear regression was successfully used to model the log-transformed weighted verbal aggression scores in the 173 (57.5%) participants that showed some verbal aggression. However, due to the lower prevalence of physical aggression ( $n = 110$ , 36.5%) and inappropriate sexual behaviour ( $n = 93$ , 30.1%) in the sample, use of all intended predictor variables was still not possible and problems with heteroscedacity emerged. This tempers conclusions drawn from the linear regression models of the non-zero values for those behaviours.

Additionally, there were some predictor variables that were not included in the studies. Previous work has shown the relationship between neuroradiological information regarding the site and extent of brain injury, particularly involving the

prefrontal cortex. Due to a lack of consistency in the clinical records of the participants involved in these studies, no such variables could be included. While it was clear from other clinical variables that the samples obtained had large proportions of the most severe brain injuries, a lack of neuroradiological information represents a limitation to the work.

Future research that incorporates detailed neuroimaging information regarding the site of brain lesion(s) may reveal the importance of differential frontal lobe networks (for example, Starkstein & Kremer, 2001; Stuss et al., 2002) within the expression of forms of aggressive and inappropriate sexual behaviours. This would be particularly interesting in relation to individual cases within the current studies that showed the highest frequencies of aberrant behaviour. Closer inspection of these cases is warranted in order to explore the possibility that the relationships found in the current studies arise primarily from relatively few cases.

It is also important to re-emphasise that the participants involved in the studies were not representative of the population of people who acquire neurological injury or illness. Rather, they were something of a sample of convenience as they were all involved in residential neurobehavioural rehabilitation. Nevertheless, this also provided some benefits. It was possible to obtain extensive behavioural observations in a controlled setting as well as a range of independent variables. Additionally, higher frequencies of behavioural disturbances than would be present in the population permitted sophisticated multivariate statistical analyses. Little may be inferred from these studies regarding the prevalence of these behavioural disturbances within the population. There is a particular need for more work

documenting the prevalence of inappropriate sexual behaviour measured with the SASBA in a population-based study.

The significance of individual predictor variables may have implications for clinical practice. Treatment for post-injury aggression and inappropriate sexual behaviour within rehabilitation should include establishing effective communication strategies as well as improvements in self-awareness and behavioural self-monitoring. Goals involving these areas of function are central to the neurobehavioural model of brain injury rehabilitation (Wood & Worthington, 2001) and the current results emphasise this. In addition to rehabilitation strategies addressing cognitive impairments including memory functioning, the treatment of clinically significant anxiety and mood disorders is important within a holistic rehabilitation programme. However, the current results suggest that these may not be of crucial importance when devising treatment protocols for verbal aggression, physical aggression and inappropriate sexual behaviour.

A number of recommendations for further research work have been made through the course of this thesis and these will be briefly reiterated here. Firstly, independent replication of the distinctions between (i) aggression and inappropriate sexual behaviour, and (ii) verbal aggression and physical aggression, are required preferably involving different observation scales to create convergent validity. Further work with participants displaying inappropriate sexual behaviour after brain injury should clarify whether there is indeed only one dimension to consider within this label.

Secondly, there are several recommendations for further investigation into predictor variables for these behavioural disorders. Neuropsychological variables reflecting error analysis were shown to be important in examining the role of executive function in verbal aggression, physical aggression and inappropriate sexual behaviour. Previous studies looking at this question reporting no associations have typically relied on performances variables other than error analysis. It is recommended that, where possible, measures of participants' error production and inhibition is included in future research of this kind. Additionally, the relationships between disorders of self-awareness and behavioural disorders after brain injury have been heretofore neglected. This may well prove a fruitful area for further work.

Thirdly, research into length of stay in hospital/rehabilitation is of increasing importance in the brain injury literature (Arango-Lasprilla et al., 2010; Avesani, Carraro, Armani, & Masiero, 2012; Tooth et al., 2001; Wagner et al., 2003), however, aggressive and inappropriate sexual behaviours have not been factored into these studies to date. Additionally, the effect of these behaviours while in rehabilitation has not been adequately explored in terms of outcomes or efficacy of treatment. These remain significant gaps in the literature and worthy of future attention.

## REFERENCES

- Abelson-Mitchell, N. (2008). Epidemiology and prevention of head injuries: literature review. *Journal of Clinical Nursing*, 17(1), 46-57.
- Alagiakrishnan, K., Lim, D., Brahim, A., Wong, A., Wood, A., Senthilselvan, A., Chimich, W. T., & Kagan, L. (2005). Sexually inappropriate behaviour in demented elderly people. *Postgraduate Medical Journal*, 81(957), 463-436.
- Alderman, N. (2007). Prevalence, characteristics and causes of aggressive behaviour observed within a neurobehavioural rehabilitation service: predictors and implications for management. *Brain Injury*, 21(9), 891-911.
- Alderman, N., Knight, C., & Birkett-Swan, L. (2009). Inappropriate sexual behaviour and aggression observed within a neurobehavioral rehabilitation service: SASBA and OAS-MNR outcomes over a three-month period. *Journal of CyberTherapy & Rehabilitation*, 2(3).
- Alderman, N., Knight, C., & Henman, C. (2002). Aggressive behaviour observed within a neurobehavioural rehabilitation service: utility of the OAS-MNR in clinical audit and applied research. *Brain Injury*, 16(6), 469-489.
- Alderman, N., Knight, C., & Morgan, C. (1997). Use of a modified version of the Overt Aggression Scale in the measurement and assessment of aggressive behaviours following brain injury. *Brain Injury*, 11(7), 503-523.
- Alderman, N., Knight, C., Stewart, I., & Gayton, A. (2011). Measuring behavioural outcome in neurodisability. *British Journal of Neuroscience Nursing*, 7(6), 691-695.
- Alia-Klein, N., O'Rourke, T. M., Goldstein, R. Z., & Malaspina, D. (2007). Insight into illness and adherence to psychotropic medications are separately associated

with violence severity in a forensic sample. *Aggressive Behaviour*, 33(1), 86-96.

Allen, M. D., Owens, T. E., Fong, A. K., & Richards, D. R. (2011). A functional neuroimaging analysis of the Trail Making Test-B: implications for clinical application. *Behav Neurol*, 24(2), 159-171.

Allen, N. (2009). Racial/ethnic differences in stroke in young adults. *Neuroepidemiology*, 32(4), 312.

Altman, I. M., Swick, S., Parrot, D., & Malec, J. F. (2010). Effectiveness of community-based rehabilitation after traumatic brain injury for 489 program completers compared with those precipitously discharged. *Archives of Physical Medicine and Rehabilitation*, 91(11), 1697-1704.

Alvarez, J. A., & Emory, E. (2006). Executive function and the frontal lobes: a meta-analytic review. *Neuropsychological Review*, 16(1), 17-42.

American Heart Association. (2009). *Heart Disease and Stroke Statistics - 2009 Update*. Dallas, Texas: American Heart Association.

Appelros, P., Stegmayr, B., & Terent, A. (2009). Sex differences in stroke epidemiology: a systematic review. *Stroke*, 40(4), 1082-1090.

Arango-Lasprilla, J. C., Ketchum, J. M., Cifu, D., Hammond, F., Castillo, C., Nicholls, E., Watanabe, T., Lequerica, A., & Deng, X. (2010). Predictors of extended rehabilitation length of stay after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 91(10), 1495-1504.

Archer, R. P., Buffington-Vollum, J. K., Stredny, R. V., & Handel, R. W. (2006). A survey of psychological test use patterns among forensic psychologists. *Journal of Personality Assessment*, 87(1), 84-94.

- Ashendorf, L., Jefferson, A. L., O'Connor, M. K., Chaisson, C., Green, R. C., & Stern, R. A. (2008). Trail Making Test errors in normal aging, mild cognitive impairment, and dementia. *Archives of Clinical Neuropsychology*, 23(2), 129-137.
- Australian Institute of Health and Welfare. (2004). *Heart, stroke and vascular diseases - Australian facts 2004* (Vol. 22). Canberra: AIHW and National Heart Foundation of Australia.
- Avesani, R., Carraro, E., Armani, G., & Masiero, S. (2012). Exploring variables associated with rehabilitation length of stay in brain injuries patients. *Eur J Phys Rehabil Med*, 48(3), 433-441.
- Bach, L. J., & David, A. S. (2006). Self-awareness after acquired and traumatic brain injury. *Neuropsychological Rehabilitation*, 16(4), 397-414.
- Baddeley, A.D. (1986). *Working memory*. Oxford: Clarendon Press.
- Baddeley, A.D., & Hitch, G.J. (1974). Working memory. In G. Bower (Ed.), *The psychology of learning and motivation: advances in research and theory* (Vol. 8, pp. 47-90). New York: Academic Press.
- Baguley, I. J., Cooper, J., & Felmingham, K. (2006). Aggressive behavior following traumatic brain injury: how common is common? *Journal of Head Trauma Rehabilitation*, 21(1), 45-56.
- Baird, A. D., Wilson, S. J., Bladin, P. F., Saling, M. M., & Reutens, D. C. (2007). Neurological control of human sexual behaviour: insights from lesion studies. *Journal of Neurology, Neurosurgery & Psychiatry*, 78(10), 1042-1049.
- Baker, S. C., Rogers, R. D., Owen, A. M., Frith, C. D., Dolan, R. J., Frackowiak, R. S., & Robbins, T. W. (1996). Neural systems engaged by planning: a PET study of the Tower of London task. *Neuropsychologia*, 34(6), 515-526.

- Baldo, J. V., Shimamura, A. P., Delis, D. C., Kramer, J., & Kaplan, E. (2001). Verbal and design fluency in patients with frontal lobe lesions. *Journal of the International Neuropsychological Society*, 7(5), 586-596.
- Baron, I.S. (2004). Delis-Kaplan Executive Function System. *Child Neuropsychology*, 10(2), 147-152.
- Baron, R.A., & Richardson, D.R. (1997). *Human Aggression*. New York: Plenum Press.
- Barratt, E. S., & Slaughter, L. (1998). Defining, measuring, and predicting impulsive aggression: a heuristic model. *Behavioral Sciences and the Law*, 16(3), 285-302.
- Barratt, E. S., Stanford, M. S., Kent, T. A., & Felthous, A. (1997). Neuropsychological and cognitive psychophysiological substrates of impulsive aggression. *Biological Psychiatry*, 41(10), 1045-1061.
- Basso, M. R., Nasrallah, H. A., Olson, S. C., & Bornstein, R. A. (1998). Neuropsychological correlates of negative, disorganized and psychotic symptoms in schizophrenia. *Schizophr Res*, 31(2-3), 99-111.
- Bechara, A., Damasio, H., & Damasio, A. R. (2000). Emotion, decision making and the orbitofrontal cortex. *Cereb Cortex*, 10(3), 295-307.
- Beck, K. D., Franks, S. F., & Hall, J. R. (2010). Postinjury personality and outcome in acquired brain injury: the Millon Behavioral Medicine Diagnostic. *Physical Medicine and Rehabilitation*, 2(3), 195-201.
- Bedell, G. M. (2008). Functional outcomes of school-age children with acquired brain injuries at discharge from inpatient rehabilitation. *Brain Injury*, 22(4), 313-324.

- Beecham, J., Perkins, M., Snell, T., & Knapp, M. (2009). Treatment paths and costs for young adults with acquired brain injury in the United Kingdom. *Brain Injury*, 23(1), 30-38.
- Bellon, K., Malec, J. F., & Kolakowsky-Hayner, S. A. (2012). Mayo-portland adaptability inventory-4. *Journal of Head Trauma Rehabilitation*, 27(4), 314-316.
- Ben-David, B. M., Nguyen, L. L., & van Lieshout, P. H. (2011). Stroop effects in persons with traumatic brain injury: selective attention, speed of processing, or color-naming? A meta-analysis. *Journal of the International Neuropsychological Society*, 17(2), 354-363.
- Benamer, H. T., & Grosset, D. (2009). Stroke in Arab countries: a systematic literature review. *Journal of the Neurological Sciences*, 284(1-2), 18-23.
- Benton, A.L., & Hamsher, K. (1976). *Multilingual Aphasia Examination*. Iowa City: University of Iowa.
- Bezeau, S. C., Bogod, N. M., & Mateer, C. A. (2004). Sexually intrusive behaviour following brain injury: approaches to assessment and rehabilitation. *Brain Injury*, 18(3), 299-313.
- Biagas, K. (1999). Hypoxic-ischemic brain injury: advancements in the understanding of mechanisms and potential avenues for therapy. *Current Opinion in Pediatrics*, 11(3), 223-228.
- Blair, R. J. (2001). Neurocognitive models of aggression, the antisocial personality disorders, and psychopathy. *Journal of Neurology, Neurosurgery & Psychiatry*, 71(6), 727-731.

- Bo, S., Abu-Akel, A., Kongerslev, M., Haahr, U. H., & Simonsen, E. (2011). Risk factors for violence among patients with schizophrenia. *Clin Psychol Rev*, 31(5), 711-726.
- Boake, C. (1996). Supervision rating scale: a measure of functional outcome from brain injury. *Archives of Physical Medicine and Rehabilitation*, 77(8), 765-772.
- Bohac, D. L., Malec, J. F., & Moessner, A. M. (1997). Factor analysis of the Mayo-Portland Adaptability Inventory: structure and validity. *Brain Injury*, 11(7), 469-482.
- Bonita, R., Broad, J. B., & Beaglehole, R. (1997). Ethnic differences in stroke incidence and case fatality in Auckland, New Zealand. *Stroke*, 28(4), 758-761.
- Bougousslavsky, J., & Hommel, M. (1993). Ischaemic stroke syndromes: clinical features, anatomy, vascular territories. In H. P. Adams (Ed.), *Handbook of Cerebrovascular Diseases* (pp. 51-94). New York: Marcel Dekker, Inc.
- Braunling-McMorrow, D., Dollinger, S. J., Gould, M., Neumann, T., & Heilgenthal, R. (2010). Outcomes of post-acute rehabilitation for persons with brain injury. *Brain Injury*, 24(7-8), 928-938.
- Brower, M. C., & Price, B. H. (2001). Neuropsychiatry of frontal lobe dysfunction in violent and criminal behaviour: a critical review. *Journal of Neurology, Neurosurgery and Psychiatry*, 71(6), 720-726.
- Burns, A., Jacoby, R., & Levy, R. (1990). Psychiatric phenomena in Alzheimer's disease. IV: Disorders of behaviour. *British Journal of Psychiatry*, 157, 86-94.
- Burns, J. M., & Swerdlow, R. H. (2003). Right orbitofrontal tumor with pedophilia symptom and constructional apraxia sign. *Archives of Neurology*, 60(3), 437-440.

- Buss, A. H., & Durkee, A. (1957). An inventory for assessing different kinds of hostility. *Journal of Consulting and Clinical Psychology*, 21(4), 343-349.
- Caine, D., & Watson, J.D.G. (2000). Neuropsychological and neuropathological sequelae of cerebral anoxia: a critical review. *Journal of the International Neuropsychological Society*, 6, 86-99.
- Cao, Y., Zhu, Z., Wang, R., Wang, S., & Zhao, J. (2010). Hypersexuality from resection of left occipital arteriovenous malformation. *Neurosurgical Review*, 33(1), 107-114.
- Caplan, L.R. (2006). *Stroke*. New York: Demos Medical Publishing.
- Carey, C. L., Woods, S. P., Damon, J., Halabi, C., Dean, D., Delis, D. C., Miller, B. L., & Kramer, J. H. (2008). Discriminant validity and neuroanatomical correlates of rule monitoring in frontotemporal dementia and Alzheimer's disease. *Neuropsychologia*, 46(4), 1081-1087.
- Carroll, K., Murad, S., Eliahoo, J., & Majeed, A. (2001). Stroke incidence and risk factors in a population-based prospective cohort study. *Health Statistics Quarterly*, 12(Winter 2001), 18-26.
- Carter, K., Anderson, C., Hacket, M., Feigin, V., Barber, P. A., Broad, J. B., & Bonita, R. (2006). Trends in ethnic disparities in stroke incidence in Auckland, New Zealand, during 1981 to 2003. *Stroke*, 37(1), 56-62.
- Cassidy, J. D., Carroll, L. J., Peloso, P. M., Borg, J., von Holst, H., Holm, L., Kraus, J., & Coronado, V. G. (2004). Incidence, risk factors and prevention of mild traumatic brain injury: results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine*, 43(Supplement), 28-60.

- Cato, M. A., Delis, D. C., Abildskov, T. J., & Bigler, E. (2004). Assessing the elusive cognitive deficits associated with ventromedial prefrontal damage: a case of a modern-day Phineas Gage. *Journal of the International Neuropsychological Society*, 10(3), 453-465.
- Chan, K. L., Campayo, A., Moser, D. J., Arndt, S., & Robinson, R. G. (2006). Aggressive behavior in patients with stroke: association with psychopathology and results of antidepressant treatment on aggression. *Archives of Physical Medicine and Rehabilitation*, 87(6), 793-798.
- Chan, R. C., Shum, D., Toulopoulou, T., & Chen, E. Y. (2008). Assessment of executive functions: review of instruments and identification of critical issues. *Archives of Clinical Neuropsychology*, 23(2), 201-216.
- Chang, B. H., & Pocock, S. (2000). Analyzing data with clumping at zero. An example demonstration. *Journal of Clinical Epidemiology*, 53(10), 1036-1043.
- Chatham Showalter, P. E., & Kimmel, D. N. (2000). Agitated symptom response to divalproex following acute brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences*, 12(3), 395-397.
- Chaytor, N., Schmitter-Edgecombe, M., & Burr, R. (2006). Improving the ecological validity of executive functioning assessment. *Archives of Clinical Neuropsychology*, 21(3), 217-227.
- Chong, J. Y., & Sacco, R. L. (2005). Epidemiology of stroke in young adults: race/ethnic differences. *Journal of Thrombosis and Thrombolysis*, 20(2), 77-83.
- Cochrane, M., Petch, I., & Pickering, A. D. (2012). Aspects of cognitive functioning in schizotypy and schizophrenia: evidence for a continuum model. *Psychiatry Res*, 196(2-3), 230-234.

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

Cohen, Jacob. (1988). *Statistical power analysis for the behavioral sciences* (Second ed.). Hillsdale, New Jersey: Lawrence Erlbaum Associates.

Cohen, Jacob. (1992). A power primer. *Psychological Bulletin*, 112(1), 155-159.

Cohen, M. J., Park, Y. D., Kim, H., & Pillai, J. J. (2010). Long-term neuropsychological follow-up of a child with Kluver-Bucy syndrome. *Epilepsy & Behaviour*, 19(4), 643-646.

Cole, W. R., Gerring, J. P., Gray, R. M., Vasa, R. A., Salorio, C. F., Grados, M., Christensen, J. R., & Slomine, B. S. (2008). Prevalence of aggressive behaviour after severe paediatric traumatic brain injury. *Brain Injury*, 22(12), 932-939.

Corrigan, J. D., & Bogner, J. A. (1995). Assessment of agitation following brain injury. *NeuroRehabilitation*, 5(3), 205-210.

Corrigan, J. D., Selassie, A. W., & Orman, J. A. (2010). The epidemiology of traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 25(2), 72-80.

Costafreda, S. G., Fu, C. H., Lee, L., Everitt, B., Brammer, M. J., & David, A. S. (2006). A systematic review and quantitative appraisal of fMRI studies of verbal fluency: role of the left inferior frontal gyrus. *Hum Brain Mapp*, 27(10), 799-810.

Costello, A.B., & Osborne, J.W. (2005). Best practices in exploratory factor analysis: Four recommendations for getting the most from your analysis. *Practical Assessment, Research & Evaluation*, 10(7), 1-9.

- Crawford, J. R., Garthwaite, P. H., Sutherland, D., & Borland, N. (2011). Some supplementary methods for the analysis of the Delis-Kaplan Executive Function System. *Psychological Assessment*, 23(4), 888-898.
- Crawford, J. R., Sutherland, D., & Garthwaite, P. H. (2008). On the reliability and standard errors of measurement of contrast measures from the D-KEFS. *Journal of the International Neuropsychological Society*, 14(6), 1069-1073.
- Crescentini, C., Seyed-Allaei, S., Vallesi, A., & Shallice, T. (2012). Two networks involved in producing and realizing plans. *Neuropsychologia*, 50(7), 1521-1535.
- Cripe, L.I. (1996). The ecological validity of executive function testing. In R. J. Sbordone & C. J. Long (Eds.), *Ecological validity of neuropsychological testing*. Delray Beach, FL: GR Press.
- Crosson, B., Barco, P., & Velozo, C.A. (1989). Awareness and compensation in post acute head injury rehabilitation. *Journal of Head Trauma Rehabilitation*, 4, 46-54.
- Crowe, L. M., Catroppa, C., Babl, F. E., & Anderson, V. (2012). Intellectual, behavioral, and social outcomes of accidental traumatic brain injury in early childhood. *Pediatrics*, 129(2), E262-E268.
- Cullen, N. K., Crescini, C., & Bayley, M. T. (2009). Rehabilitation outcomes after anoxic brain injury: a case-controlled comparison with traumatic brain injury. *Physical Medicine and Rehabilitation*, 1(12), 1069-1076.
- Davies, R. C., Williams, W. H., Hinder, D., Burgess, C. N., & Mounce, L. T. (2012). Self-reported traumatic brain injury and postconcussion symptoms in incarcerated youth. *Journal of Head Trauma Rehabilitation*, 27(3), E21-E27.

- Deb, S. (1999). ICD-10 codes detect only a proportion of all head injury admissions. *Brain Injury*, 13(5), 369-373.
- Deb, S., Lyons, I., & Koutzoukis, C. (1999). Neurobehavioural symptoms one year after a head injury. *British Journal of Psychiatry*, 174, 360-365.
- DelBello, M. P., Soutullo, C. A., Zimmerman, M. E., Sax, K. W., Williams, J. R., McElroy, S. L., & Strakowski, S. M. (1999). Traumatic brain injury in individuals convicted of sexual offenses with and without bipolar disorder. *Psychiatry Research*, 89(3), 281-286.
- Delis, D. C., Kaplan, E., & Kramer, J.H. (2001). *The Delis-Kaplan Executive Function System: Examiner's Manual*. San Antonio: The Psychological Corporation.
- Delucchi, K. L., & Bostrom, A. (2004). Methods for analysis of skewed data distributions in psychiatric clinical studies: working with many zero values. *American Journal of Psychiatry*, 161(7), 1159-1168.
- Denny, K.G., & Matthias, S. (2012). Trait aggression is related to anger-modulated deficits in response inhibition. *Journal of Research in Personality*, 46(4), 450-454.
- Department of Health. (2007). Mental Health Act. Retrieved 1 November 2012, from <http://www.legislation.gov.uk/ukpga/2007/12/contents>
- Dimoska-Di Marco, A., McDonald, S., Kelly, M., Tate, R., & Johnstone, S. (2011). A meta-analysis of response inhibition and Stroop interference control deficits in adults with traumatic brain injury (TBI). *Journal of Clinical and Experimental Neuropsychology*, 33(4), 471-485.
- Dolan, M., Millington, J., & Park, I. (2002). Personality and neuropsychological function in violent, sexual and arson offenders. *Medicine, Science and the Law*, 42(1), 34-43.

- Donders, J., & Warschausky, S. (2007). Neurobehavioral outcomes after early versus late childhood traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 22(5), 296-302.
- Dooley, J. J., Anderson, V., Hemphill, S. A., & Ohan, J. (2008). Aggression after paediatric traumatic brain injury: a theoretical approach. *Brain Injury*, 22(11), 836-846.
- Draper, K., Ponsford, J., & Schonberger, M. (2007). Psychosocial and emotional outcomes 10 years following traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 22(5), 278-287.
- Dudzinski, M.L., Norris, J.M., Chmura, J.T., & Edwards, C.B.H. (1975). Repeatability of principal components in samples: Normal and non-normal data sets compared. *Multivariate Behavioral Research*, 10(1), 109-117.
- Dunteman, G.H. (1989). *Principal Components Analysis*. London: Sage Publications Ltd.
- Dyall, L., Carter, K., Bonita, R., Anderson, C., Feigin, V., Kerse, N., & Brown, P. (2006). Incidence of stroke in women in Auckland, New Zealand. Ethnic trends over two decades: 1981-2003. *N Z Med J*, 119(1245), U2309.
- Dyer, K. F. W., Bell, R., McCann, J., & Rauch, R. (2006). Aggression after traumatic brain injury: Analysing socially desirable responses and the nature of aggressive traits. *Brain Injury*, 20(11), 1163-1173.
- Eames, P. (1990). Organic bases of behaviour disorders after traumatic brain injury. In R. L. Wood (Ed.), *Neurobehavioural sequelae of traumatic brain injury* (pp. 134-150). London: Taylor & Francis.

- Eastvold, A., Suchy, Y., & Strassberg, D. (2011). Executive function profiles of pedophilic and nonpedophilic child molesters. *Journal of the International Neuropsychological Society*, 17(2), 295-307.
- Ebrahim, S., & Harwood, R. (1999). *Stroke: epidemiology, evidence and clinical practice* (2nd ed.). New York: Oxford University Press.
- Eicher, V., Murphy, M. P., Murphy, T. F., & Malec, J. F. (2012). Progress assessed with the Mayo-Portland Adaptability Inventory in 604 participants in 4 types of post-inpatient rehabilitation brain injury programs. *Archives of Physical Medicine and Rehabilitation*, 93(1), 100-107.
- Ellis, M. L., Weiss, B., & Lochman, J. E. (2009). Executive functions in children: associations with aggressive behavior and appraisal processing. *J Abnorm Child Psychol*, 37(7), 945-956.
- Emory, L.E., Cole, C.M., & Meyer, W.J. (1995). Use of Depo-Provera to control sexual aggression in persons with traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 10(3), 47-58.
- Eslinger, P. J., & Damasio, A. R. (1985). Severe disturbance of higher cognition after bilateral frontal lobe ablation: patient EVR. *Neurology*, 35(12), 1731-1741.
- Feigin, V., Carter, K., Hackett, M., Barber, P. A., McNaughton, H., Dyall, L., Chen, M. H., & Anderson, C. (2006). Ethnic disparities in incidence of stroke subtypes: Auckland Regional Community Stroke Study, 2002-2003. *The Lancet Neurology*, 5(2), 130-139.
- Field, A. (2005). *Discovering statistics using SPSS* (2nd ed.). London: Sage Publications Ltd.

- Fine, E. M., Delis, D. C., & Holdnack, J. (2011). Normative adjustments to the D-KEFS trail making test: corrections for education and vocabulary level. *Clinical Neuropsychologist*, 25(8), 1331-1344.
- Finnerty, F., Glynn, L., Dineen, B., Colfer, F., & Macfarlane, A. (2009). A postal survey of data in general practice on the prevalence of Acquired Brain Injury (ABI) in patients aged 18-65 in one county in the west of Ireland. *BMC Family Practice*, 10, 36.
- Fleiss, J.L. (1971). Measuring nominal scale agreement among many raters. *Psychological Bulletin*, 76(5), 378-382.
- Fleminger, S., Greenwood, R. J., & Oliver, D. L. (2003). Pharmacological management for agitation and aggression in people with acquired brain injury. *The Cochrane Database of Systematic Reviews*, 1.
- Fleminger, S., Oliver, D. L., Williams, W. H., & Evans, J. (2003). The neuropsychiatry of depression after brain injury. *Neuropsychological Rehabilitation*, 13(1-2), 65-87.
- Fleminger, S., & Ponsford, J. (2005). Long term outcome after traumatic brain injury. *British Medical Journal*, 331(7530), 1419-1420.
- Fletcher, D., Mackenzie, D., & Villouta, E. (2005). Modelling skewed data with many zeros: A simple approach combining ordinary and logistic regression. *Environmental and Ecological Statistics*, 12, 45-54.
- Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res*, 12(3), 189-198.
- Fornazzari, L., Farcnik, K., Smith, I., Heasman, G. A., & Ichise, M. (1992). Violent visual hallucinations and aggression in frontal lobe dysfunction: clinical

manifestations of deep orbitofrontal foci. *Journal of Neuropsychiatry and Clinical Neurosciences*, 4(1), 42-44.

Fortuny, L. A., Briggs, M., Newcombe, F., Ratcliff, G., & Thomas, C. (1980).

Measuring the duration of post traumatic amnesia. *Journal of Neurology, Neurosurgery & Psychiatry*, 43(5), 377-379.

Fraas, M., & Bellerose, A. (2010). Mentoring programme for adolescent survivors of acquired brain injury. *Brain Injury*, 24(1), 50-61.

Freeland, J.C., Corker, T., Heritage, T., & James, A.I.W. (2012). Reliability and Validity of the BIRT Aggression Rating Scale (BARS). Manuscript submitted for publication.

Fyffe, C. E., Kahng, S., Fittro, E., & Russell, D. (2004). Functional analysis and treatment of inappropriate sexual behavior. *Journal of Applied Behavior Analysis*, 37(3), 401-404.

Gansler, D. A., McLaughlin, N. C., Iguchi, L., Jerram, M., Moore, D. W., Bhadelia, R., & Fulwiler, C. (2009). A multivariate approach to aggression and the orbital frontal cortex in psychiatric patients. *Psychiatry Research*, 171(3), 145-154.

Ghajar, J. (2000). Traumatic brain injury. *The Lancet*, 356(9233), 923-929.

Giancola, P. R. (2000). Executive functioning: a conceptual framework for alcohol-related aggression. *Exp Clin Psychopharmacol*, 8(4), 576-597.

Giancola, P. R. (2004). Executive functioning and alcohol-related aggression. *Journal of Abnormal Psychology*, 113(4), 541-555.

Giancola, P. R., Duke, A. A., & Ritz, K. Z. (2011). Alcohol, violence, and the Alcohol Myopia Model: preliminary findings and implications for prevention. *Addictive Behaviour*, 36(10), 1019-1022.

- Giancola, P. R., Godlaski, A. J., & Roth, R. M. (2012). Identifying component-processes of executive functioning that serve as risk factors for the alcohol-aggression relation. *Psychology of Addictive Behaviors*, 26(2), 201-211.
- Giancola, P. R., Josephs, R. A., DeWall, C. N., & Gunn, R. L. (2009). Applying the attention-allocation model to the explanation of alcohol-related aggression: implications for prevention. *Subst Use & Misuse*, 44(9-10), 1263-1279.
- Giancola, P. R., & Zeichner, A. (1994). Neuropsychological performance on tests of frontal-lobe functioning and aggressive behavior in men. *Journal of Abnormal Psychology*, 103(4), 832-835.
- Glenn, M. B., Rotman, M., Goldstein, R., & Selleck, E. A. (2005). Characteristics of residential community integration programs for adults with brain injury. *Journal of Head Trauma Rehabilitation*, 20(5), 393-401.
- Golden, C.J., Jackson, M.L., Peterson-Rohne, A., & Gontovsky, S.T. (1996). Neuropsychological correlates of violence and aggression: a review of the clinical literature. *Aggression and Violent Behavior*, 1(1), 3-25.
- Goverover, Y., Johnston, M. V., Toglia, J., & Deluca, J. (2007). Treatment to improve self-awareness in persons with acquired brain injury. *Brain Injury*, 21(9), 913-923.
- Grafman, J., Schwab, K., Warden, D., Pridgen, A., Brown, H. R., & Salazar, A. M. (1996). Frontal lobe injuries, violence, and aggression: a report of the Vietnam Head Injury Study. *Neurology*, 46(5), 1231-1238.
- Greve, K. W., Sherwin, E., Stanford, M. S., Mathias, C., Love, J., & Ramzinski, P. (2001). Personality and neurocognitive correlates of impulsive aggression in long-term survivors of severe traumatic brain injury. *Brain Injury*, 15(3), 255-262.

- Gunarathne, A., Patel, J. V., Gammon, B., Gill, P. S., Hughes, E. A., & Lip, G. Y. (2009). Ischemic stroke in South Asians: a review of the epidemiology, pathophysiology, and ethnicity-related clinical features. *Stroke*, 40(6), e415-423.
- Hancock, M., Tapscott, J. L., & Hoaken, P. N. (2010). Role of executive dysfunction in predicting frequency and severity of violence. *Aggressive Behaviour*, 36(5), 338-349.
- Hanlon, R. E., Coda, J.J., Cobia, D., & Rubin, L. H. (2012). Psychotic domestic murder: neuropsychological differences between homicidal and nonhomicidal schizophrenic men. *Journal of Family Violence*, 27(2), 105-113.
- Hanlon, R. E., Rubin, L. H., Jensen, M., & Daoust, S. (2010). Neuropsychological features of indigent murder defendants and death row inmates in relation to homicidal aspects of their crimes. *Archives of Clinical Neuropsychology*, 25(1), 1-13.
- Harris, A. W., Large, M. M., Redoblado-Hodge, A., Niesssen, O., Anderson, J., & Brennan, J. (2010). Clinical and cognitive associations with aggression in the first episode of psychosis. *Australian and New Zealand Journal of Psychiatry*, 44(1), 85-93.
- Harrison, J. E., Berry, J. G., & Jamieson, L. M. (2012). Head and traumatic brain injuries among Australian youth and young adults, July 2000-June 2006. *Brain Injury*, 26(7-8), 996-1004.
- Hart, T., Millis, S., Novack, T., Englander, J., Fidler-Sheppard, R., & Bell, K. R. (2003). The relationship between neuropsychologic function and level of caregiver supervision at 1 year after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 84(2), 221-230.

- Hart, T., Seignourel, P. J., & Sherer, M. (2009). A longitudinal study of awareness of deficit after moderate to severe traumatic brain injury. *Neuropsychological Rehabilitation*, 19(2), 161-176.
- Hawkins, K.A., & Trobst, K.K. (2000). Frontal lobe dysfunction and aggression: conceptual issues and research findings. *Aggression and Violent Behavior*, 5(2), 147-157.
- Heinz, A. J., Beck, A., Meyer-Lindenberg, A., Sterzer, P., & Heinz, A. (2011). Cognitive and neurobiological mechanisms of alcohol-related aggression. *Nature Reviews Neuroscience*, 12(7), 400-413.
- Henry, J. D., & Crawford, J. R. (2004a). A meta-analytic review of verbal fluency performance following focal cortical lesions. *Neuropsychology*, 18(2), 284-295.
- Henry, J. D., & Crawford, J. R. (2004b). A meta-analytic review of verbal fluency performance in patients with traumatic brain injury. *Neuropsychology*, 18(4), 621-628.
- Heuschmann, P. U., Grieve, A. P., Toschke, A. M., Rudd, A. G., & Wolfe, C. D. (2008). Ethnic group disparities in 10-year trends in stroke incidence and vascular risk factors: the South London Stroke Register (SLSR). *Stroke*, 39(8), 2204-2210.
- Hibbard, M. R., Gordon, W. A., Flanagan, S., Haddad, L., & Labinsky, E. (2000). Sexual dysfunction after traumatic brain injury. *NeuroRehabilitation*, 15(2), 107-120.
- Hillier, S. L., Hiller, J. E., & Metzer, J. (1997). Epidemiology of traumatic brain injury in South Australia. *Brain Injury*, 11(9), 649-659.

- Hoaken, P. N., Allaby, D. B., & Earle, J. (2007). Executive cognitive functioning and the recognition of facial expressions of emotion in incarcerated violent offenders, non-violent offenders, and controls. *Aggressive Behaviour*, 33(5), 412-421.
- Hoaken, P.N.S., Shaughnessy, V.K., & Pihl, R.O. (2003). Executive cognitive functioning and aggression: is it an issue of impulsivity? *Aggressive Behaviour*, 29(1), 15-30.
- Homack, S., Lee, D., & Riccio, C. A. (2005). Test review: Delis-Kaplan executive function system. *Journal of Clinical and Experimental Neuropsychology*, 27(5), 599-609.
- Hopkins, R. O., & Haaland, K. Y. (2004). Neuropsychological and neuropathological effects of anoxic or ischemic induced brain injury. *Journal of the International Neuropsychological Society*, 10(7), 957-961.
- Hopkins, R. O., Tate, D. F., & Bigler, E. D. (2005). Anoxic versus traumatic brain injury: amount of tissue loss, not etiology, alters cognitive and emotional function. *Neuropsychology*, 19(2), 233-242.
- Howard, C.J. (2012). Neurobiological correlates of partner abusive men: equifinality in perpetrators of intimate partner violence. *Psychological Trauma - Theory, Research, Practice nad Policy*, 4(3), 330-337.
- Huson, L.W. (2007). Performance of some correlation coefficients when applied to zero-clustered data. *Journal of Modern Applied Statistical Methods*, 6(2), 530-536.
- Hutcheson, G., & Sofroniou, N. (1999). *The multivariate social scientist*. London: Sage.

- Itoo, B. A., Al-Hawsawi, Z. M., & Khan, A. H. (2003). Hypoxic ischemic encephalopathy. Incidence and risk factors in North Western Saudi Arabia. *Saudi Medical Journal*, 24(2), 147-153.
- Iverson, G. L., & Hughes, R. (2000). Monitoring aggression and problem behaviors in inpatient neuropsychiatric units. *Psychiatric Services*, 51(8), 1040-1042.
- Jackson, J.E. (1991). *A user's guide to principal components*. New York: Wiley.
- Jacobs, R., & Anderson, V. (2002). Planning and problem solving skills following focal frontal brain lesions in childhood: analysis using the Tower of London. *Child Neuropsychology*, 8(2), 93-106.
- Jacobson, S. C., Blanchard, M., Connolly, C. C., Cannon, M., & Garavan, H. (2011). An fMRI investigation of a novel analogue to the Trail-Making Test. *Brain and Cognition*, 77(1), 60-70.
- Jacobsson, L. J., Westerberg, M., Malec, J. F., & Lexell, J. (2011). Sense of coherence and disability and the relationship with life satisfaction 6-15 years after traumatic brain injury in northern Sweden. *Neuropsychological Rehabilitation*, 21(3), 383-400.
- Jefferson, A. L., Paul, R. H., Ozonoff, A., & Cohen, R. A. (2006). Evaluating elements of executive functioning as predictors of instrumental activities of daily living (IADLs). *Archives of Clinical Neuropsychology*, 21(4), 311-320.
- Jefferson, A. L., Poppas, A., Paul, R. H., & Cohen, R. A. (2007). Systemic hypoperfusion is associated with executive dysfunction in geriatric cardiac patients. *Neurobiol Aging*, 28(3), 477-483.
- Johansson, S. H., Jamora, C. W., Ruff, R. M., & Pack, N. M. (2008). A biopsychosocial perspective of aggression in the context of traumatic brain injury. *Brain Injury*, 22(13-14), 999-1006.

- Johnson, C., Knight, C., & Alderman, N. (2006). Challenges associated with the definition and assessment of inappropriate sexual behaviour amongst individuals with an acquired neurological impairment. *Brain Injury*, 20(7), 687-693.
- Johnson, R., & Balleny, H. (1996). Behaviour problems after brain injury: incidence and need for treatment. *Clinical Rehabilitation*, 10(2), 173-180.
- Johnston, M. V., Nakajima, W., & Hagberg, H. (2002). Mechanisms of hypoxic neurodegeneration in the developing brain. *The Neuroscientist*, 8(3), 212-220.
- Jolliffe, I.T. (2002). *Principal Component Analysis* (2nd ed.). New York: Springer.
- Joyal, C. C., Black, D. N., & Dassylva, B. (2007). The neuropsychology and neurology of sexual deviance: a review and pilot study. *Sex Abuse*, 19(2), 155-173.
- Jurado, M. B., & Rosselli, M. (2007). The elusive nature of executive functions: a review of our current understanding. *Neuropsychological Review*, 17(3), 213-233.
- Kaiser, H.F. (1974). An index of factorial simplicity. *Psychometrika*, 39(31-36).
- Karver, C. L., Wade, S. L., Cassedy, A., Taylor, H. G., Stancin, T., Yeates, K. O., & Walz, N. C. (2012). Age at injury and long-term behavior problems after traumatic brain injury in young children. *Rehabilitation Psychology*, 57(3), 256-265.
- Kazdin, A.E. (2001). *Behavior modification in applied settings* (6th ed.). Belmont, CA: Wadsworth.
- Kean, J., Malec, J. F., Altman, I. M., & Swick, S. (2011). Rasch measurement analysis of the Mayo-Portland Adaptability Inventory (MPAI-4) in a

community-based rehabilitation sample. *Journal of Neurotrauma*, 28(5), 745-753.

Kelly, G., Brown, S., Todd, J., & Kremer, P. (2008). Challenging behaviour profiles of people with acquired brain injury living in community settings. *Brain Injury*, 22(6), 457-470.

Kelly, G., & Simpson, G. (2011). Remediating serious inappropriate sexual behaviour in a male with severe acquired brain injury. *Sexuality and Disability*, 29, 313-327.

Kelly, T., Richardson, G., Hunter, R., & Knapp, M. (2002). Attention and executive function deficits in adolescent sex offenders. *Child Neuropsychology*, 8(2), 138-143.

Kerr, K., Oram, J., Tinson, H., & Shum, D. (2011). The correlates of aggression in people with acquired brain injury: a preliminary retrospective study. *Brain Injury*, 25(7-8), 729-741.

Ketchum, J. M., Almaz Getachew, M., Krch, D., Banos, J. H., Kolakowsky-Hayner, S. A., Lequerica, A., Jamison, L., & Arango-Lasprilla, J. C. (2012). Early predictors of employment outcomes 1 year post traumatic brain injury in a population of Hispanic individuals. *NeuroRehabilitation*, 30(1), 13-22.

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

Kim, J. S., Choi, S., Kwon, S. U., & Seo, Y. S. (2002). Inability to control anger or aggression after stroke. *Neurology*, 58(7), 1106-1108.

Kim, Y. J. (2011). A systematic review of factors contributing to outcomes in patients with traumatic brain injury. *Journal of Clinical Nursing*, 20(11-12), 1518-1532.

- Klatsky, A. L., Friedman, G. D., Sidney, S., Kipp, H., Kubo, A., & Armstrong, M. A. (2005). Risk of hemorrhagic stroke in Asian American ethnic groups. *Neuroepidemiology*, 25(1), 26-31.
- Knight, C., Alderman, N., Johnson, C., Green, S., Birkett-Swan, L., & Yorstan, G. (2008). The St Andrew's Sexual Behaviour Assessment (SASBA): development of a standardised recording instrument for the measurement and assessment of challenging sexual behaviour in people with progressive and acquired neurological impairment. *Neuropsychological Rehabilitation*, 18(2), 129-159.
- Kobak, K. A., Lipsitz, J. D., & Feiger, A. (2003). Development of a standardized training program for the Hamilton Depression Scale using internet-based technologies: results from a pilot study. *Journal of Psychiatric Research*, 37(6), 509-515.
- Kochanek, P.M., Clark, R.S.B., & Jenkins, L.W. (2007). TBI: pathobiology. In N. D. Zasler, D. I. Katz & R. D. Zafonte (Eds.), *Brain Injury Medicine: Principles and Practice*. New York: Demos.
- Kockler, T. R., & Stanford, M. S. (2008). Using a clinically aggressive sample to examine the association between impulsivity, executive functioning, and verbal learning and memory. *Archives of Clinical Neuropsychology*, 23(2), 165-173.
- Kolakowsky-Hayner, S. A., Miner, K. D., & Kreutzer, J. S. (2001). Long-term life quality and family needs after traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 16(4), 374-385.

- Kortte, K. B., Horner, M. D., & Windham, W. K. (2002). The trail making test, part B: cognitive flexibility or ability to maintain set? *Appl Neuropsychol*, 9(2), 106-109.
- Krakowski, M. I., & Czobor, P. (2012). Executive function predicts response to antiaggression treatment in schizophrenia: a randomized controlled trial. *Journal of Clinical Psychiatry*, 73(1), 74-80.
- Kramer, J. H., Reed, B. R., Mungas, D., Weiner, M. W., & Chui, H. C. (2002). Executive dysfunction in subcortical ischaemic vascular disease. *Journal of Neurology, Neurosurgery & Psychiatry*, 72(2), 217-220.
- Kramer, U. M., Kopyciok, R. P., Richter, S., Rodriguez-Fornells, A., & Munte, T. F. (2011). The role of executive functions in the control of aggressive behavior. *Front Psychol*, 2, 152.
- Kreutzer, J. S., Kolakowsky-Hayner, S. A., Ripley, D., Cifu, D. X., Rosenthal, M., Bushnik, T., Zafonte, R., Englander, J., & High, W. (2001). Charges and lengths of stay for acute and inpatient rehabilitation treatment of traumatic brain injury 1990-1996. *Brain Injury*, 15(9), 763-774.
- Kuroiwa, T., & Okeda, R. (1994). Neuropathology of cerebral ischemia and hypoxia: recent advances in experimental studies on its pathogenesis. *Pathology International*, 44(3), 171-181.
- Kusano, Y., Horiuchi, T., Tanaka, Y., Tsuji, T., & Hongo, K. (2012). Transient Kluver-Bucy syndrome caused by cerebral edema following aneurysmal subarachnoid hemorrhage. *Clinical Neurology and Neurosurgery*, 114(3), 294-296.
- Lachenbruch, P. A. (1992). Utility of logistic regression analysis in epidemiologic studies of the elderly. In R. B. Wallace & R. F. Woolson (Eds.), *Epidemiologic*

*Methods in the Study of Aging* (pp. 371-381). New York: Oxford University Press.

Lachenbruch, P. A. (2001a). Comparisons of two-part models with competitors. *Statistics in Medicine*, 20(8), 1215-1234.

Lachenbruch, P. A. (2001b). Power and sample size requirements for two-part models. *Statistics in Medicine*, 20(8), 1235-1238.

Lachenbruch, P. A. (2002). Analysis of data with excess zeros. *Statistical Methods in Medical Research*, 11(4), 297-302.

Lange, R. T., Iverson, G. L., Zakrzewski, M. J., Ethel-King, P. E., & Franzen, M. D. (2005). Interpreting the trail making test following traumatic brain injury: comparison of traditional time scores and derived indices. *Journal of Clinical and Experimental Neuropsychology*, 27(7), 897-906.

Langevin, R. (2006). Sexual offenses and traumatic brain injury. *Brain and Cognition*, 60(2), 206-207.

Lanoo, E., Brusselmans, W., Van Eynde, L., Van Laere, M., & Stevens, J. (2004). Epidemiology of acquired brain injury (ABI) in adults: prevalence of long-term disabilities and the resulting need for ongoing care in the region of Flanders, Belgium. *Brain Injury*, 18(2), 203-211.

Larochette, A. C., Benn, K., & Harrison, A. G. (2009). Executive functioning: a comparison of the Tower of London(DX) and the D-KEFS Tower Test. *Appl Neuropsychol*, 16(4), 275-280.

Latzman, R. D., & Markon, K. E. (2010). The factor structure and age-related factorial invariance of the Delis-Kaplan Executive Function System (D-KEFS). *Assessment*, 17(2), 172-184.

- Lau, M. A., Pihl, R. O., & Peterson, J. B. (1995). Provocation, acute alcohol intoxication, cognitive performance, and aggression. *Journal of Abnormal Psychology, 104*(1), 150-155.
- Lazeron, R. H., Rombouts, S. A., Machielsen, W. C., Scheltens, P., Witter, M. P., Uylings, H. B., & Barkhof, F. (2000). Visualizing brain activation during planning: the tower of London test adapted for functional MR imaging. *American Journal of Neuroradiology, 21*(8), 1407-1414.
- Leblanc, N., Chen, S., Swank, P. R., Levin, H., & Schachar, R. (2006). Impairment and recovery in inhibitory control after traumatic brain injury in children: effect of age at injury, injury severity and lesion location. *Brain and Cognition, 60*(2), 208-209.
- Lees-Haley, P.R., Smith, H.H., Williams, C.W., & Dunn, J.T. (1996). Forensic neuropsychological test usage: an empirical survey. *Archives of Clinical Neuropsychology, 11*(1), 45-51.
- Levin, H. S., Culhane, K. A., Mendelsohn, D., Lilly, M. A., Bruce, D., Fletcher, J. M., Chapman, S. B., Harward, H., & Eisenberg, H. M. (1993). Cognition in relation to magnetic resonance imaging in head-injured children and adolescents. *Archives of Neurology, 50*(9), 897-905.
- Levin, H. S., High, W. M., Goethe, K. E., Sisson, R. A., Overall, J. E., Rhoades, H. M., Eisenberg, H. M., Kalisky, Z., & Gary, H. E. (1987). The neurobehavioural rating scale: assessment of the behavioural sequelae of head injury by the clinician. *Journal of Neurology, Neurosurgery & Psychiatry, 50*(2), 183-193.
- Levin, H. S., O'Donnell, V. M., & Grossman, R. G. (1979). The Galveston Orientation and Amnesia Test. A practical scale to assess cognition after head injury. *The Journal of Nervous and Mental Disease, 167*(11), 675-684.

- Levine, B., Katz, D.I., Dade, L., & Black, S.E. (2002). Novel approaches to the assessment of frontal lobe damage and executive deficits in traumatic brain injury. In D. T. Stuss & R. T. Knight (Eds.), *Principles of frontal lobe function* (pp. 448-465). New York: Oxford.
- Lezak, M. D. (1983). *Neuropsychological assessment* (2nd ed.). New York: Oxford University Press.
- Lezak, M.D. (1987). Relationships between personality disorders, social disturbances, and physical disability following traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 2(1), 57-69.
- Lezak, M.D. (1995). *Neuropsychological Assessment* (3rd ed.). New York: Oxford University Press.
- Liddle, P. F., & Morris, D. L. (1991). Schizophrenic syndromes and frontal lobe performance. *British Journal of Psychiatry*, 158, 340-345.
- Lin, H. F., Yeh, Y. C., Chen, C. F., Chang, W. C., & Chen, C. S. (2011). Kluver-Bucy syndrome in one case with systemic lupus erythematosus. *Kaohsiung Journal of Medical Sciences*, 27(4), 159-162.
- Lippa, S. M., & Davis, R. N. (2010). Inhibition/switching is not necessarily harder than inhibition: an analysis of the D-KEFS color-word interference test. *Archives of Clinical Neuropsychology*, 25(2), 146-152.
- Lopez, B. R., Lincoln, A. J., Ozonoff, S., & Lai, Z. (2005). Examining the relationship between executive functions and restricted, repetitive symptoms of Autistic Disorder. *Journal of Autism & Developmental Disorders*, 35(4), 445-460.
- Luiselli, J.K., Sherak, D.L., Dunn, E.K., & Pace, G.M. (2005). Sexual behaviors among children and adolescents with acquired brain injury: an incidence

survey at a community-based neurorehabilitation center. *Behavioral Interventions*, 20, 17-25.

Lundqvist, A., Linnros, H., Orlenius, H., & Samuelsson, K. (2010). Improved self-awareness and coping strategies for patients with acquired brain injury--a group therapy programme. *Brain Injury*, 24(6), 823-832.

Luria, A.R. (1966). *Higher cortical functions in man*. New York: Basic Books.

Luria, A.R. (1973). *The working brain: an introduction to neuropsychology*. New York: Basic.

Lysaker, P. H., Whitney, K. A., & Davis, L. W. (2006). Awareness of illness in schizophrenia: associations with multiple assessments of executive function. *Journal of Neuropsychiatry and Clinical Neurosciences*, 18(4), 516-520.

MacDonald, B. K., Cockerell, O. C., Sander, J. W., & Shorvon, S. D. (2000). The incidence and lifetime prevalence of neurological disorders in a prospective community-based study in the UK. *Brain*, 123(4), 665-676.

Malec, J. F., Brown, A. W., Moessner, A. M., Stump, T. E., & Monahan, P. (2010). A preliminary model for posttraumatic brain injury depression. *Archives of Physical Medicine and Rehabilitation*, 91(7), 1087-1097.

Malec, J. F., Kragness, M., Evans, R. W., Finlay, K. L., Kent, A., & Lezak, M. D. (2003). Further psychometric evaluation and revision of the Mayo-Portland Adaptability Inventory in a national sample. *Journal of Head Trauma Rehabilitation*, 18(6), 479-492.

Malec, J. F., Mandrekar, J. N., Brown, A. W., & Moessner, A. M. (2009). Injury severity and disability in the selection of next level of care following acute medical treatment for traumatic brain injury. *Brain Injury*, 23(1), 22-29.

- Malec, J. F., Moessner, A. M., Kragness, M., & Lezak, M. D. (2000). Refining a measure of brain injury sequelae to predict postacute rehabilitation outcome: rating scale analysis of the Mayo-Portland Adaptability Inventory. *Journal of Head Trauma Rehabilitation*, 15(1), 670-682.
- Malec, J. F., & Thompson, J.M. (1994). Relationship of the Mayo-Portland Adaptability Inventory to functional outcome and cognitive performance measures. *Journal of Head Trauma Rehabilitation*, 9(4), 1-15.
- Malec, J.F. (1999). Mild traumatic brain injury: scope of the problem. In N. R. Varney & R. J. Roberts (Eds.), *The Evaluation and Treatment of Mild Traumatic Brain Injury*. Mahwah, NJ: Lawrence Erlbaum Associates.
- Malec, J.F., & Lezak, M.D. (2003, April). Manual for the Mayo-Portland Adaptability Inventory (MPAI-4) 1st Edition. Retrieved 12 December 2009, from <http://www.tbims.org/combi/mpai/manual.pdf>
- Mattson, S. N., Goodman, A. M., Caine, C., Delis, D. C., & Riley, E. P. (1999). Executive functioning in children with heavy prenatal alcohol exposure. *Alcoholism: Clinical and Experimental Research*, 23(11), 1808-1815.
- McAllister, T. W., & Ferrell, R. B. (2002). Evaluation and treatment of psychosis after traumatic brain injury. *NeuroRehabilitation*, 17(4), 357-368.
- McDonald, C. R., Delis, D. C., Norman, M. A., Tecoma, E. S., & Iragui-Madozi, V. I. (2005). Is impairment in set-shifting specific to frontal-lobe dysfunction? Evidence from patients with frontal-lobe or temporal-lobe epilepsy. *Journal of the International Neuropsychological Society*, 11(4), 477-481.
- McDonald, C. R., Delis, D. C., Norman, M. A., Wetter, S. R., Tecoma, E. S., & Iragui, V. J. (2005). Response inhibition and set shifting in patients with frontal lobe epilepsy or temporal lobe epilepsy. *Epilepsy & Behaviour*, 7(3), 438-446.

- McKinlay, A., Grace, R. C., Horwood, L. J., Fergusson, D. M., & MacFarlane, M. R. (2010). Long-term behavioural outcomes of pre-school mild traumatic brain injury. *Child: Care, Health and Development*, 36(1), 22-30.
- McKinlay, A., Grace, R. C., Kaller, C. P., Dalrymple-Alford, J. C., Anderson, T. J., Fink, J., & Roger, D. (2009). Assessing cognitive impairment in Parkinson's disease: a comparison of two tower tasks. *Appl Neuropsychol*, 16(3), 177-185.
- McKinlay, A., Grace, R., Horwood, J., Fergusson, D., & MacFarlane, M. (2009). Adolescent psychiatric symptoms following preschool childhood mild traumatic brain injury: evidence from a birth cohort. *Journal of Head Trauma Rehabilitation*, 24(3), 221-227.
- McKinlay, A., Grace, R.C., Horwood, L.J., Fergusson, D.M., Ridder, E.M., & MacFarlane, M.R. (2008). Prevalence of traumatic brain injury among children, adolescents and young adults: prospective evidence from a birth cohort. *Brain Injury*, 22(2), 175-181.
- McKinlay, W. W., Brooks, D. N., Bond, M. R., Martinage, D. P., & Marshall, M. M. (1981). The short-term outcome of severe blunt head injury as reported by relatives of the injured persons. *Journal of Neurology, Neurosurgery and Psychiatry*, 44(6), 527-533.
- Medley, A. R., & Powell, T. (2010). Motivational Interviewing to promote self-awareness and engagement in rehabilitation following acquired brain injury: A conceptual review. *Neuropsychological Rehabilitation*, 20(4), 481-508.
- Mendez, M., & Shapira, J. S. (2011). Pedophilic behavior from brain disease. *Journal of Sexual Medicine*, 8(4), 1092-1100.

- Mesulam, M.M. (2002). The human frontal lobes: transcending the default mode through contingent encoding. In D. T. Stuss & R. T. Knight (Eds.), *Principles of frontal lobe function* (pp. 8-30). New York: Oxford University Press.
- Middleton, F.A., & Strick, P.L. (2001). Revised neuroanatomy of frontal-subcortical circuits. In D. G. Lichter & J. L. Cummings (Eds.), *Frontal-subcortical circuits in psychiatric and neurological disorders* (pp. 44-58). New York: Guilford.
- Miles, J., & Shevlin, M. (2001). *Applying regression and correlation: a guide for students and researchers*. London: Sage.
- Miller, B. L., Cummings, J. L., McIntyre, H., Ebers, G., & Grode, M. (1986). Hypersexuality or altered sexual preference following brain injury. *Journal of Neurology, Neurosurgery and Psychiatry*, 49(8), 867-873.
- Miller, L. A., Collins, R. L., & Kent, T. A. (2008). Language and the modulation of impulsive aggression. *Journal of Neuropsychiatry and Clinical Neuroscience*, 20(3), 261-273.
- Miller, N., & Campbell, D. T. (1959). Recency and primacy in persuasion as a function of the timing of speeches and measurements. *Journal of Abnormal Psychology*, 59(1), 1-9.
- Mitchell, M., & Miller, L. S. (2008). Prediction of functional status in older adults: the ecological validity of four Delis-Kaplan Executive Function System tests. *Journal of Clinical and Experimental Neuropsychology*, 30(6), 683-690.
- Miyake, A., Emerson, M. J., & Friedman, N. P. (2000). Assessment of executive functions in clinical settings: problems and recommendations. *Semin Speech Lang*, 21(2), 169-183.

- Moll, J., de Oliveira-Souza, R., Moll, F. T., Bramati, I. E., & Andreiuolo, P. A. (2002). The cerebral correlates of set-shifting: an fMRI study of the trail making test. *Arq Neuropsiquiatr*, 60(4), 900-905.
- Mooney, G. F., & Haas, L. J. (1993). Effect of methylphenidate on brain injury-related anger. *Archives of Physical Medicine and Rehabilitation*, 74(2), 153-160.
- Morgan, A. B., & Lilienfeld, S. O. (2000). A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. *Clin Psychol Rev*, 20(1), 113-136.
- Morris, R. G., Miotto, E. C., Feigenbaum, J. D., Bullock, P., & Polkey, C. E. (1997). The effect of goal-subgoal conflict on planning ability after frontal- and temporal-lobe lesions in humans. *Neuropsychologia*, 35(8), 1147-1157.
- Murray, G. D., Teasdale, G. M., Braakman, R., Cohadon, F., Dearden, M., Iannotti, F., Karimi, A., Lapierre, F., Maas, A., Ohman, J., Persson, L., Servadei, F., Stocchetti, N., Trojanowski, T., & Unterberg, A. (1999). The European Brain Injury Consortium survey of head injuries. *Acta Neurochirurgica*, 141(3), 223-236.
- Murrey, G. J., Hale, F. M., & Williams, J. D. (2005). Assessment of anosognosia in persons with frontal lobe damage: clinical utility of the Mayo-Portland Adaptability Inventory (MPAI). *Brain Injury*, 19(8), 599-603.
- Naudts, K., & Hodgins, S. (2006). Neurobiological correlates of violent behavior among persons with schizophrenia. *Schizophr Bull*, 32(3), 562-572.
- Norman, D.A., & Shallice, T. (1986). Attention to action: willed and automatic control of behaviour. In R. Davidson, G. Schwartz & D. Shapiro (Eds.), *Consciousness and self-regulation: advances in research* (Vol. 4, pp. 1-18). New York: Plenum.

- O'Leary, D. S., Flaum, M., Kesler, M. L., Flashman, L. A., Arndt, S., & Andreasen, N. C. (2000). Cognitive correlates of the negative, disorganized, and psychotic symptom dimensions of schizophrenia. *Journal of Neuropsychiatry and Clinical Neurosciences*, 12(1), 4-15.
- Oddson, B., Rumney, P., Johnson, P., & Thomas-Stonell, N. (2006). Clinical use of the Mayo-Portland Adaptability Inventory in rehabilitation after paediatric acquired brain injury. *Developmental Medicine and Child Neurology*, 48(11), 918-922.
- Oddy, M., & Herbert, C. (2003). Intervention with families following brain injury: Evidence-based practice. *Neuropsychological Rehabilitation*, 13(1/2), 259-273.
- Oddy, M., Moir, J. F., Fortescue, D., & Chadwick, S. (2012). The prevalence of traumatic brain injury in the homeless community in a UK city. *Brain Injury*, 26(9), 1058-1064.
- Odhuba, R. A., van den Broek, M. D., & Johns, L. C. (2005). Ecological validity of measures of executive functioning. *British Journal of Clinical Psychology*, 44(Pt 2), 269-278.
- Owen, A. M., Downes, J. J., Sahakian, B. J., Polkey, C. E., & Robbins, T. W. (1990). Planning and spatial working memory following frontal lobe lesions in man. *Neuropsychologia*, 28(10), 1021-1034.
- Owen, A. M., Doyon, J., Petrides, M., & Evans, A. C. (1996). Planning and spatial working memory: a positron emission tomography study in humans. *European Journal of Neuroscience*, 8(2), 353-364.
- Ownsworth, T., Fleming, J., Strong, J., Radel, M., Chan, W., & Clare, L. (2007). Awareness typologies, long-term emotional adjustment and psychosocial

outcomes following acquired brain injury. *Neuropsychological Rehabilitation*, 17(2), 129-150.

Ownsworth, T. L., Turpin, M., Andrew, B., & Fleming, J. (2008). Participant perspectives on an individualised self-awareness intervention following stroke: a qualitative case study. *Neuropsychological Rehabilitation*, 18(5-6), 692-712.

Paradiso, S., Robinson, R. G., & Arndt, S. (1996). Self-reported aggressive behavior in patients with stroke. *Journal of Nervous and Mental Diseases*, 184(12), 746-753.

Pardini, M., Krueger, F., Hodgkinson, C., Raymont, V., Ferrier, C., Goldman, D., Strenziok, M., Guida, S., & Grafman, J. (2011). Prefrontal cortex lesions and MAO-A modulate aggression in penetrating traumatic brain injury. *Neurology*, 76(12), 1038-1045.

Pathak, E. B., & Sloan, M. A. (2009). Recent racial/ethnic disparities in stroke hospitalizations and outcomes for young adults in Florida, 2001-2006. *Neuroepidemiology*, 32(4), 302-311.

Petrides, M., & Pandya, D.N. (2002). Association pathways of the prefrontal cortex and functional observations. In D. T. Stuss & R. T. Knight (Eds.), *Principles of frontal lobe function* (pp. 31-50). New York: Oxford University Press.

Phillips, L. A., Voaklander, D. C., Drul, C., & Kelly, K. D. (2009). The epidemiology of hospitalized head injury in British Columbia, Canada. *Canadian Journal of Neurological Sciences*, 36(5), 605-611.

Poletti, M., Lucetti, C., & Bonuccelli, U. (2010). Out-of-control sexual behavior in an orbitofrontal cortex-damaged elderly patient. *Journal of Neuropsychiatry and Clinical Neurosciences*, 22(2), E7.

- Ponsford, J., Sloan, S., & Snow, P. (1995). *Traumatic brain injury: rehabilitation for everyday adaptive living*. Hove, East Sussex: Psychology Press.
- Ponsford, J., Draper, K., & Schonberger, M. (2008). Functional outcome 10 years after traumatic brain injury: its relationship with demographic, injury severity, and cognitive and emotional status. *Journal of the International Neuropsychological Society*, 14(2), 233-242.
- Pontius, A.A., & LeMay, M.J. (2003). Aggression in temporal lobe epilepsy and limbic psychotic trigger reaction implicating vagus kindling of hippocampus/amygdala (in sinus abnormalities on MRIs). *Aggression and Violent Behavior*, 8, 245-257.
- Prigatano, G. P. (2005a). Disturbances of self-awareness and rehabilitation of patients with traumatic brain injury: a 20-year perspective. *Journal of Head Trauma Rehabilitation*, 20(1), 19-29.
- Prigatano, G. P. (2005b). Impaired self-awareness after moderately severe to severe traumatic brain injury. *Acta Neurochirurgica Supplement*, 93, 39-42.
- Prigatano, G. P., & Altman, I. M. (1990). Impaired awareness of behavioral limitations after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 71(13), 1058-1064.
- Raaijmakers, M. A., Smidts, D. P., Sergeant, J. A., Maassen, G. H., Posthumus, J. A., van Engeland, H., & Matthys, W. (2008). Executive functions in preschool children with aggressive behavior: impairments in inhibitory control. *J Abnorm Child Psychol*, 36(7), 1097-1107.
- Rabin, L. A., Barr, W. B., & Burton, L. A. (2005). Assessment practices of clinical neuropsychologists in the United States and Canada: a survey of INS, NAN,

and APA Division 40 members. *Archives of Clinical Neuropsychology*, 20(1), 33-65.

Rao, V., Rosenberg, P., Bertrand, M., Salehinia, S., Spiro, J., Vaishnavi, S., Rastogi, P., Noll, K., Schretlen, D. J., Brandt, J., Cornwell, E., Makley, M., & Miles, Q. S. (2009). Aggression after traumatic brain injury: prevalence and correlates. *Journal of Neuropsychiatry and Clinical Neurosciences*, 21(4), 420-429.

Rashid, R. M., Eder, K., Rosenow, J., Macken, M. P., & Schuele, S. U. (2010). Ictal kissing: a release phenomenon in non-dominant temporal lobe epilepsy. *Epileptic Disorders*, 12(4), 262-269.

Rees, P. M., Fowler, C. J., & Maas, C. P. (2007). Sexual function in men and women with neurological disorders. *Lancet*, 369(9560), 512-525.

Roberts, K. L., & Hall, D. A. (2008). Examining a supramodal network for conflict processing: a systematic review and novel functional magnetic resonance imaging data for related visual and auditory stroop tasks. *J Cogn Neurosci*, 20(6), 1063-1078.

Ropper, A.H., & Samuels, M.A. (2009). *Adams and Victor's Principles of Neurology* (9th ed.). New York: McGraw-Hill.

Ross, E.H., & Hoaken, P. N. (2011). Executive cognitive functioning abilities of male first time and return Canadian federal inmates. *Canadian Journal of Criminology and Criminal Justice*, 53(4), 377-403.

Saint-Cyr, J.A., Bronstein, Y.L., & Cummings, J.L. (2002). Neurobehavioural consequences of neurosurgical treatments and focal lesions of frontal-subcortical circuits. In D. T. Stuss & R. T. Knight (Eds.), *Principles of frontal lobe function* (pp. 408-427). New York: Oxford University Press.

- Salmon, D.P., Heindel, W.C., & Hamilton, J.M. (2001). Cognitive abilities mediated by frontal-subcortical circuits. In D. G. Licher & J. L. Cummings (Eds.), *Frontal-Subcortical Circuits in Psychiatric and Neurological Disorders*. New York: Guilford.
- Sanchez-Cubillo, I., Perianez, J. A., Adrover-Roig, D., Rodriguez-Sanchez, J. M., Rios-Lago, M., Tirapu, J., & Barcelo, F. (2009). Construct validity of the Trail Making Test: role of task-switching, working memory, inhibition/interference control, and visuomotor abilities. *Journal of the International Neuropsychological Society*, 15(3), 438-450.
- Schacter, D. L. (1990). Toward a cognitive neuropsychology of awareness: implicit knowledge and anosognosia. *Journal of Clinical and Experimental Neuropsychology*, 12(1), 155-178.
- Schaub, D., Brune, M., Bierhoff, H. W., & Juckel, G. (2012). Comparison of self- and clinician's ratings of Personal and Social Performance in patients with schizophrenia: the role of insight. *Psychopathology*, 45(2), 109-116.
- Schmid, M., Strand, M., Ardal, G., Lund, A., & Hammar, A. (2011). Prolonged impairment in inhibition and semantic fluency in a follow-up study of recurrent major depression. *Archives of Clinical Neuropsychology*, 26(7), 677-686.
- Schmidt, J., Lannin, N., Fleming, J., & Ownsworth, T. (2011). Feedback interventions for impaired self-awareness following brain injury: a systematic review. *Journal of Rehabilitation Medicine*, 43(8), 673-680.
- Schmidt, S. (2009). Shall we really do it again? The powerful concept of replication is neglected in the social sciences. *Review of General Psychology*, 13(2), 90-100.

- Schmitz, B. (2006). Effects of antiepileptic drugs on mood and behavior. *Epilepsia*, 47(Suppl. 2), 28-33.
- Schneider, D., Tahk, A., & Krosnick, J.A. (2007). Reconsidering the impact of behavior prediction questions on illegal drug use: The importance of using proper analytic methods. *Social Influence*, 2(3), 178-196.
- Schonberger, M., Ponsford, J., Olver, J., Ponsford, M., & Wirtz, M. (2011). Prediction of functional and employment outcome 1 year after traumatic brain injury: a structural equation modelling approach. *Journal of Neurology, Neurosurgery & Psychiatry*, 82(8), 936-941.
- Selassie, A. W., Zaloshnja, E., Langlois, J. A., Miller, T., Jones, P., & Steiner, C. (2008). Incidence of long-term disability following traumatic brain injury hospitalization, United States, 2003. *Journal of Head Trauma Rehabilitation*, 23(2), 123-131.
- Shah, M. K., Al-Adawi, S., Dorvlo, A. S., & Burke, D. T. (2004). Functional outcomes following anoxic brain injury: a comparison with traumatic brain injury. *Brain Injury*, 18(2), 111-117.
- Shallice, T. (1982). Specific impairments of planning. *Philosophical Transactions of the Royal Society of London*, 298(1089), 199-209.
- Shallice, T., & Burgess, P. W. (1991). Deficits in strategy application following frontal lobe damage in man. *Brain*, 114(2), 727-741.
- Sherer, M., Hart, T., Nick, T. G., Whyte, J., Thompson, R. N., & Yablon, S. A. (2003). Early impaired self-awareness after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 84(2), 168-176.

- Shiroma, E. J., Ferguson, P. L., & Pickelsimer, E. E. (2010). Prevalence of traumatic brain injury in an offender population: a meta-analysis. *Journal of Correctional Health Care, 16*(2), 147-159.
- Shiroma, E. J., Ferguson, P. L., & Pickelsimer, E. E. (2012). Prevalence of traumatic brain injury in an offender population: a meta-analysis. *Journal of Head Trauma Rehabilitation, 27*(3), E1-E10.
- Shores, E. A., Marosszky, J. E., Sandanam, J., & Batchelor, J. (1986). Preliminary validation of a clinical scale for measuring the duration of post-traumatic amnesia. *Medical Journal of Australia, 144*(11), 569-572.
- Shrout, P.E., & Fleiss, J.L. (1979). Intraclass correlations: Uses in assessing rater reliability. *Psychological Bulletin, 86*(2), 420-428.
- Siever, L. J. (2008). Neurobiology of aggression and violence. *American Journal of Psychiatry, 165*(4), 429-442.
- Sigurdardottir, S., Andelic, N., Roe, C., & Schanke, A. K. (2009). Cognitive recovery and predictors of functional outcome 1 year after traumatic brain injury. *Journal of the International Neuropsychological Society, 15*(5), 740-750.
- Sigurdardottir, S., Jerstad, T., Andelic, N., Roe, C., & Schanke, A. K. (2010). Olfactory dysfunction, gambling task performance and intracranial lesions after traumatic brain injury. *Neuropsychology, 24*(4), 504-513.
- Silva, J., Ownsworth, T., Shields, C., & Fleming, J. (2011). Enhanced appreciation of life following acquired brain injury: posttraumatic growth at 6 months postdischarge. *Brain Impairment, 12*(2), 93-104.
- Simon, H.A. (1975). The functional equivalence of problem solving skills. *Cognitive Psychology, 7*, 268-288.

- Simpson, G., Blaszcynski, A., & Hodgkinson, A. (1999). Sex offending as a psychosocial sequela of traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 14(6), 567-580.
- Simpson, G., Tate, R., Ferry, K., Hodgkinson, A., & Blaszcynski, A. (2001). Social, neuroradiologic, medical, and neuropsychologic correlates of sexually aberrant behavior after traumatic brain injury: a controlled study. *Journal of Head Trauma Rehabilitation*, 16(6), 556-572.
- Smith, J., Wells, L., & Dodd, K. (2000). The continuing fall in incidence of hypoxic-ischaemic encephalopathy in term infants. *British Journal of Obstetrics and Gynaecology*, 107(4), 461-466.
- Song, H., & Min, S. K. (2009). Aggressive behavior model in schizophrenic patients. *Psychiatry Research*, 167(1-2), 58-65.
- Sonnenberg, L. K., Dupuis, A., & Rumney, P. G. (2010). Pre-school traumatic brain injury and its impact on social development at 8 years of age. *Brain Injury*, 24(7-8), 1003-1007.
- Spinella, M. (2007). The role of prefrontal systems in sexual behavior. *International Journal of Neuroscience*, 117(3), 369-385.
- Spitz, G., Ponsford, J. L., Rudzki, D., & Maller, J. J. (2012). Association between cognitive performance and functional outcome following traumatic brain injury: A longitudinal multilevel examination. *Neuropsychology*, 26(5), 604-612.
- Sprague, J., Verona, E., Kalkhoff, W., & Kilmer, A. (2011). Moderators and mediators of the stress-aggression relationship: executive function and state anger. *Emotion*, 11(1), 61-73.
- Srinivasan, L., Roberts, B., Bushnik, T., Englander, J., Spain, D. A., Steinberg, G. K., Ren, L., Sandel, M. E., Al-Lawati, Z., Teraoka, J., Hoffman, A. R., &

- Katznelson, L. (2009). The impact of hypopituitarism on function and performance in subjects with recent history of traumatic brain injury and aneurysmal subarachnoid haemorrhage. *Brain Injury*, 23(7), 639-648.
- Starkstein, S.E., & Kremer, J. (2001). The disinhibition syndrome and frontal-subcortical circuits. In D. G. Licher & J. L. Cummings (Eds.), *Frontal-Subcortical Circuits in Psychiatric and Neurological Disorders*. New York: Guilford.
- Steudel, W. I., Cortbus, F., & Schwerdtfeger, K. (2005). Epidemiology and prevention of fatal head injuries in Germany--trends and the impact of the reunification. *Acta Neurochirurgica*, 147(3), 231-242.
- Stewart, I., Knight, C., Alderman, N., & Haywad, L. (2010). Inappropriate sexual behaviour observed within an older adult service: the use of the ST Andrew's Sexual Behaviour Assessment (SASBA) in formulation, intervention and outcome. *PSIGE Newsletter*, 110, 62-72.
- Strauss, E., Sherman, E.M.S., & Spreen, O. (2006). *A Compendium of Neuropsychological Tests* (Third ed.). New York: Oxford University Press.
- Strauss, E., Spreen, O., & Hunter, M. (2000). Implications of test revisions for research. *Psychological Assessment*, 12(3), 237-244.
- Strong, C. A., Tiesma, D., & Donders, J. (2011). Criterion validity of the Delis-Kaplan Executive Function System (D-KEFS) fluency subtests after traumatic brain injury. *Journal of the International Neuropsychological Society*, 17(2), 230-237.
- Stroop, J.R. (1935). Studies of interference in serial verbal reaction. *Journal of Experimental Psychology*, 18, 643-662.

- Stuss, D. T., & Anderson, V. (2004). The frontal lobes and theory of mind: developmental concepts from adult focal lesion research. *Brain and Cognition*, 55(1), 69-83.
- Stuss, D. T., Bisschop, S. M., Alexander, M. P., Levine, B., Katz, D., & Izukawa, D. (2001). The Trail Making Test: a study in focal lesion patients. *Psychological Assessment*, 13(2), 230-239.
- Stuss, D. T., & Levine, B. (2002). Adult clinical neuropsychology: lessons from studies of the frontal lobes. *Annu Rev Psychol*, 53, 401-433.
- Stuss, D.T., Alexander, M.P., Floden, D., Binns, M.A., McIntosh, A.R., Rajah, N., & Hevenor, S.J. (2002). Fractionation and localization of distinct frontal lobe processes: evidence from focal lesions in humans. In D. T. Stuss & R. T. Knight (Eds.), *Principles of frontal lobe function*. New York: Oxford University Press.
- Stuss, D.T., & Knight, R.T. (2002). Introduction. In D. T. Stuss & R. T. Knight (Eds.), *Principles of frontal lobe function* (pp. 1-7). New York: Oxford University Press.
- Suchy, Y., Whittaker, J. W., Strassberg, D. S., & Eastvold, A. (2009). Neurocognitive differences between pedophilic and nonpedophilic child molesters. *Journal of the International Neuropsychological Society*, 15(2), 248-257.
- Sullivan, K., & Bowden, S.C. (1997). Which tests do neuropsychologists use? *Journal of Clinical Psychology*, 53(7), 657-661.
- Summers, C. R., Ivins, B., & Schwab, K. A. (2009). Traumatic brain injury in the United States: an epidemiologic overview. *Mt Sinai Journal of Medicine*, 76(2), 105-110.

- Surius, A., Lind, L., Emmett, G., Borman, P.D., Kashner, M., & Barratt, E. S. (2004). Measures of aggressive behavior: overview of clinical and research instruments. *Aggression and Violent Behavior*, 9, 165-227.
- Szulc, A., Galinska-Skok, B., Tarasow, E., Konarzewska, B., Waszkiewicz, N., Hykiel, R., & Walecki, J. (2012). Clinical and cognitive correlates of the proton magnetic resonance spectroscopy measures in chronic schizophrenia. *Medical Science Monitor*, 18(6), CR390-398.
- Tacher, E.L., & Readdick, C.A. (2006). The relation between aggression and creativity among second graders. *Creativity Research Journal*, 18(3), 261-267.
- Tagliaferri, F., Compagnone, C., Korsic, M., Servadei, F., & Kraus, J. (2006). A systematic review of brain injury epidemiology in Europe. *Acta Neurochirurgica*, 148(3), 255-268.
- Tate, R. L. (2004). Assessing support needs for people with traumatic brain injury: the Care and Needs Scale (CANS). *Brain Injury*, 18(5), 445-460.
- Tate, R. L. (2010). *A Compendium of Tests, Scales and Questionnaires*. Hove, East Sussex: Psychology Press.
- Tate, R. L., Pfaff, A., Baguley, I. J., Marosszeky, J. E., Gurka, J. A., Hodgkinson, A. E., King, C., Lane-Brown, A. T., & Hanna, J. (2006). A multicentre, randomised trial examining the effect of test procedures measuring emergence from post-traumatic amnesia. *Journal of Neurology, Neurosurgery & Psychiatry*, 77(7), 841-849.
- Tate, R. L., Pfaff, A., & Jurjevic, L. (2000). Resolution of disorientation and amnesia during post-traumatic amnesia. *Journal of Neurology, Neurosurgery & Psychiatry*, 68(2), 178-185.

- Tateno, A., Jorge, R. E., & Robinson, R. G. (2003). Clinical correlates of aggressive behavior after traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neuroscience*, 15(2), 155-160.
- Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness. A practical scale. *Lancet*, 2(7872), 81-84.
- Telman, G., Kouperberg, E., Sprecher, E., & Yarnitsky, D. (2010). Ethnic differences in ischemic stroke of working age in northern Israel. *Journal of Stroke and Cerebrovascular Disease*, 19(5), 376-381.
- Tennant, A. (2005). Admission to hospital following head injury in England: incidence and socio-economic associations. *BMC Public Health*, 5, 21.
- Thornhill, S., Teasdale, G. M., Murray, G. D., McEwen, J., Roy, C. W., & Penny, K. I. (2000). Disability in young people and adults one year after head injury: prospective cohort study. *British Medical Journal*, 320(7250), 1631-1635.
- Thurman, D.J., Coronado, V., & Selassie, A. W. (2007). The epidemiology of TBI: implications for public health. In N. D. Zasler, D. I. Katz & R. D. Zafonte (Eds.), *Brain Injury Medicine: Principles and Practice* (pp. 45-55). New York: Demos.
- Tonkonogy, J. M. (1991). Violence and temporal lobe lesion: head CT and MRI data. *Journal of Neuropsychiatry and Clinical Neurosciences*, 3(2), 189-196.
- Tooth, L., McKenna, K., Strong, J., Ottenbacher, K., Connell, J., & Cleary, M. (2001). Rehabilitation outcomes for brain injured patients in Australia: functional status, length of stay and discharge destination. *Brain Injury*, 15(7), 613-631.
- Tranel, D. (2002). Emotion, decision making and the ventromedial prefrontal cortex. In D. T. Stuss & R. T. Knight (Eds.), *Principles of frontal lobe function*. New York: Oxford University Press.

- Treadwell, K., & Page, T. J. (1996). Functional analysis: identifying the environmental determinants of severe behavior disorders. *Journal of Head Trauma Rehabilitation*, 11(1), 62-74.
- Tsai, S. J., Hwang, J. P., Yang, C. H., Liu, K. M., & Lirng, J. F. (1999). Inappropriate sexual behaviors in dementia: a preliminary report. *Alzheimer Disease and Associated Disorders*, 13(1), 60-62.
- Tulsky, David S. (2003). *Clinical interpretation of the WAIS III and WMS III*. Amsterdam ; Boston: Academic Press.
- Tulsky, David S., & Price, Larry R. (2003). The joint WAIS-III and WMS-III factor structure: development and cross-validation of a six-factor model of cognitive functioning. *Psychological Assessment*, 15(2), 149-162.
- van den Heuvel, O. A., Groenewegen, H. J., Barkhof, F., Lazeron, R. H., van Dyck, R., & Veltman, D. J. (2003). Frontostriatal system in planning complexity: a parametric functional magnetic resonance version of Tower of London task. *Neuroimage*, 18(2), 367-374.
- Vendrame, M., & Azizi, S. A. (2007). Pyramidal and extrapyramidal dysfunction as a sequela of hypoxic injury: case report. *BMC Neurology*, 7, 18.
- Visscher, A. J., van Meijel, B., Stolker, J. J., Wiersma, J., & Nijman, H. (2011). Aggressive behaviour of inpatients with acquired brain injury. *Journal of Clinical Nursing*, 20(23-24), 3414-3422.
- Volavka, J. (2002). *Neurobiology of Violence* (2nd ed.). Washington, DC: American Psychiatric.
- Wagner, A. K., Fabio, T., Zafonte, R. D., Goldberg, G., Marion, D. W., & Peitzman, A. B. (2003). Physical medicine and rehabilitation consultation: relationships with acute functional outcome, length of stay, and discharge planning after

traumatic brain injury. *American Journal of Physical Medicine & Rehabilitation*, 82(7), 526-536.

Walling, S. M., Meehan, J. C., Marshall, A. D., Holtzworth-Munroe, A., & Taft, C. T. (2012). The relationship of intimate partner aggression to head injury, executive functioning, and intelligence. *Journal of Marital and Family Therapy*, 38(3), 471-485.

Warlow, C.P., Dennis, M.S., van Gijn, J., Hankey, G.J., Sandercock, P.A.G., Bamford, J.M., & Wardlaw, J.M. (2001). *Stroke: a practical guide to management* (2nd ed.). Oxford: Blackwell Science Ltd.

Wechsler, D. (1997a). *Wechsler Adult Intelligence Scale - 3rd Edition*. San Antonio, TX: The Psychological Corporation.

Wechsler, D. (1997b). *Wechsler Memory Scale - 3rd Edition*. New York: The Psychological Corporation.

Williams, W. H., Cordan, G., Mewse, A. J., Tonks, J., & Burgess, C. N. (2010). Self-reported traumatic brain injury in male young offenders: a risk factor for re-offending, poor mental health and violence? *Neuropsychological Rehabilitation*, 20(6), 801-812.

Williams, W. H., Mewse, A. J., Tonks, J., Mills, S., Burgess, C. N., & Cordan, G. (2010). Traumatic brain injury in a prison population: prevalence and risk for re-offending. *Brain Injury*, 24(10), 1184-1188.

Winqvist, S., Lehtilahti, M., Jokelainen, J., Luukinen, H., & Hillbom, M. (2007). Traumatic brain injuries in children and young adults: a birth cohort study from northern Finland. *Neuroepidemiology*, 29(1-2), 136-142.

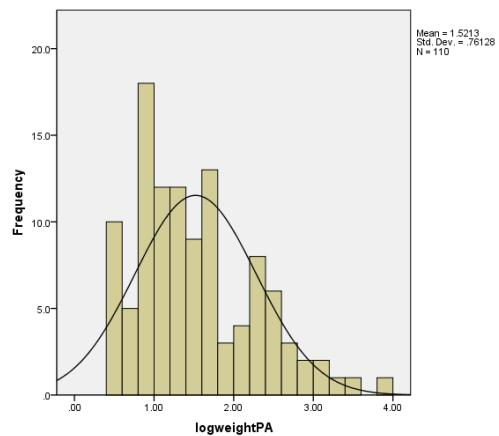
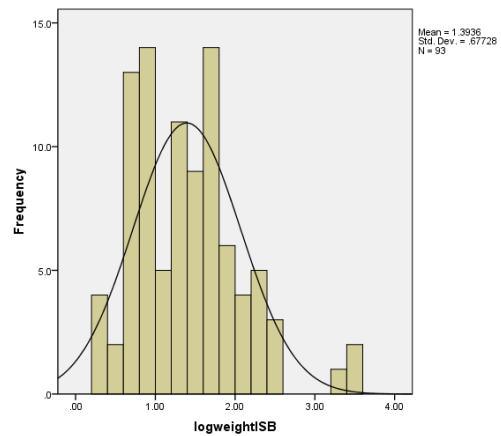
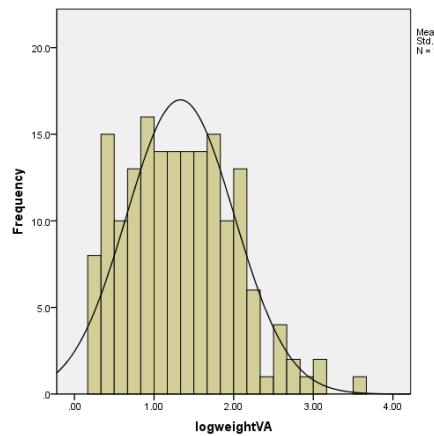
- Wittenberg, M.D., Sloan, J.P., & Barlow, I.F. (2004). Head injuries in Leeds: changes in epidemiology and survival over 12 years. *Emergency Medicine Journal*, 21, 429-432.
- Wood, R. L. (2001). Understanding neurobehavioral disability. In R. L. Wood & T. McMillan (Eds.), *Neurobehavioral disability and social handicap after traumatic brain injury*. East Sussex: Psychology Press.
- Wood, R. L., & Liossi, C. (2006a). The ecological validity of executive tests in a severely brain injured sample. *Archives of Clinical Neuropsychology*, 21(5), 429-437.
- Wood, R. L., & Liossi, C. (2006b). Neuropsychological and neurobehavioral correlates of aggression following traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neuroscience*, 18(3), 333-341.
- Wood, R. L., McCrea, J. D., Wood, L. M., & Merriman, R. N. (1999). Clinical and cost effectiveness of post-acute neurobehavioural rehabilitation. *Brain Injury*, 13(2), 69-88.
- Wood, R. L., & Rutherford, N. A. (2004). Relationships between measured cognitive ability and reported psychosocial activity after bilateral frontal lobe injury: An 18-year follow-up. *Neuropsychological Rehabilitation*, 14(3), 329-350.
- Wood, R. L., & Worthington, A.D. (2001). Neurobehavioural rehabilitation: a conceptual paradigm. In R. L. Wood & T. McMillan (Eds.), *Neurobehavioural disability and social handicap following brain injury*. East Sussex: Psychology Press.
- Woods, S.R., Sigford, B., & Lanham, R. (1998). Disinhibited hypersexuality and aggression in three male patients with traumatic brain injury. *Psychosomatics*, 39(2), 207-208.

- Woodward, T. S., Ruff, C. C., Thornton, A. E., Moritz, S., & Liddle, P. F. (2003). Methodological considerations regarding the association of Stroop and verbal fluency performance with the symptoms of schizophrenia. *Schizophr Res*, 61(2-3), 207-214.
- Worthington, A. D., Matthews, S., Melia, Y., & Oddy, M. (2006). Cost-benefits associated with social outcome from neurobehavioural rehabilitation. *Brain Injury*, 20(9), 947-957.
- Yates, P. J., Williams, W. H., Harris, A., Round, A., & Jenkins, R. (2006). An epidemiological study of head injuries in a UK population attending an emergency department. *Journal of Neurology, Neurosurgery and Psychiatry*, 77(5), 699-701.
- Yochim, B., Baldo, J., Nelson, A., & Delis, D. C. (2007). D-KEFS Trail Making Test performance in patients with lateral prefrontal cortex lesions. *Journal of the International Neuropsychological Society*, 13(4), 704-709.
- Yochim, B. P., Baldo, J. V., Kane, K. D., & Delis, D. C. (2009). D-KEFS Tower Test performance in patients with lateral prefrontal cortex lesions: the importance of error monitoring. *Journal of Clinical and Experimental Neuropsychology*, 31(6), 658-663.
- Yudofsky, S. C., Silver, J. M., Jackson, W., Endicott, J., & Williams, D. (1986). The Overt Aggression Scale for the objective rating of verbal and physical aggression. *American Journal of Psychiatry*, 143(1), 35-39.
- Zakzanis, K. K., Mraz, R., & Graham, S. J. (2005). An fMRI study of the Trail Making Test. *Neuropsychologia*, 43(13), 1878-1886.

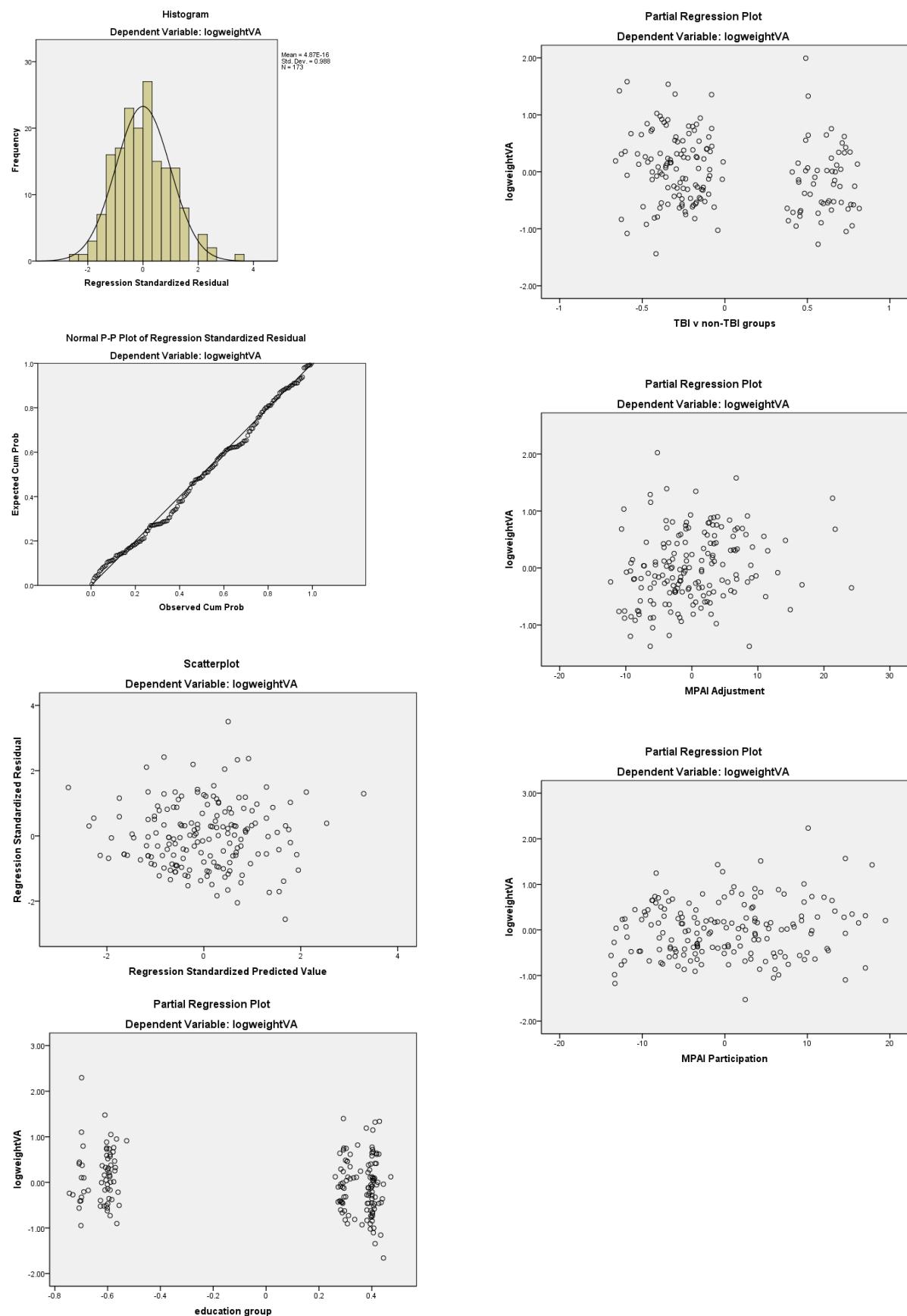
- Zald, D. H., & Kim, S. W. (1996). Anatomy and function of the orbital frontal cortex, II: Function and relevance to obsessive-compulsive disorder. *Journal of Neuropsychiatry and Clinical Neurosciences*, 8(3), 249-261.
- Zaloshnja, E., Miller, T., Langlois, J. A., & Selassie, A. W. (2008). Prevalence of long-term disability from traumatic brain injury in the civilian population of the United States, 2005. *Journal of Head Trauma Rehabilitation*, 23(6), 394-400.
- Zasler, N.D. (1994). Sexual dysfunction. In J. M. Silver, S. C. Yudofsky & R. E. Hales (Eds.), *Neuropsychiatry of traumatic brain injury* (pp. 443-469). Washington, DC: American Psychiatric Press.
- Zgaljardic, D. J., Yancy, S., Temple, R. O., Watford, M. F., & Miller, R. (2011). Ecological validity of the screening module and the Daily Living tests of the Neuropsychological Assessment Battery using the Mayo-Portland Adaptability Inventory-4 in postacute brain injury rehabilitation. *Rehabilitation Psychology*, 56(4), 359-365.

## **APPENDICES**

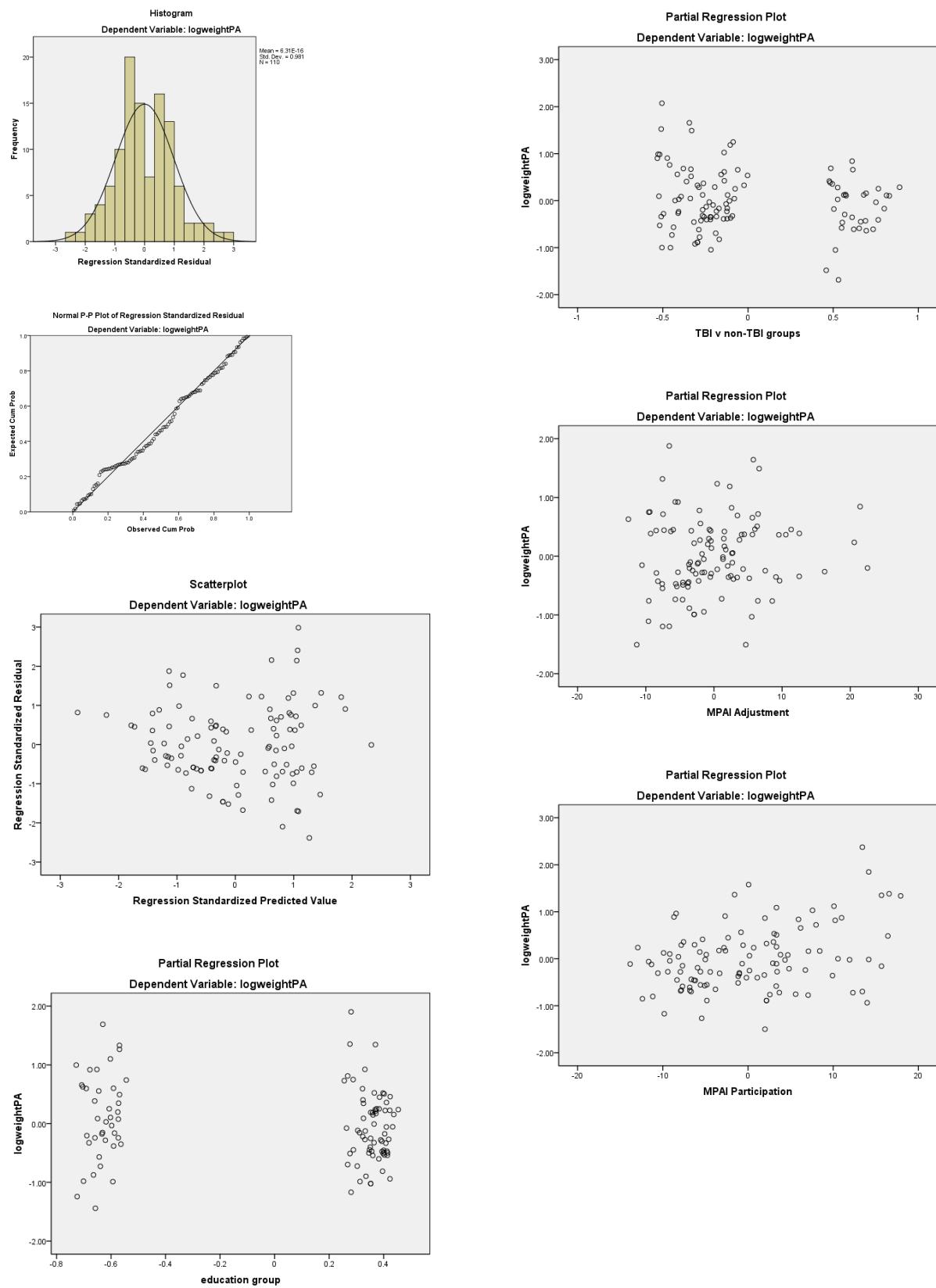
## APPENDIX 1: Histograms of non-zero weighted log-transformed values



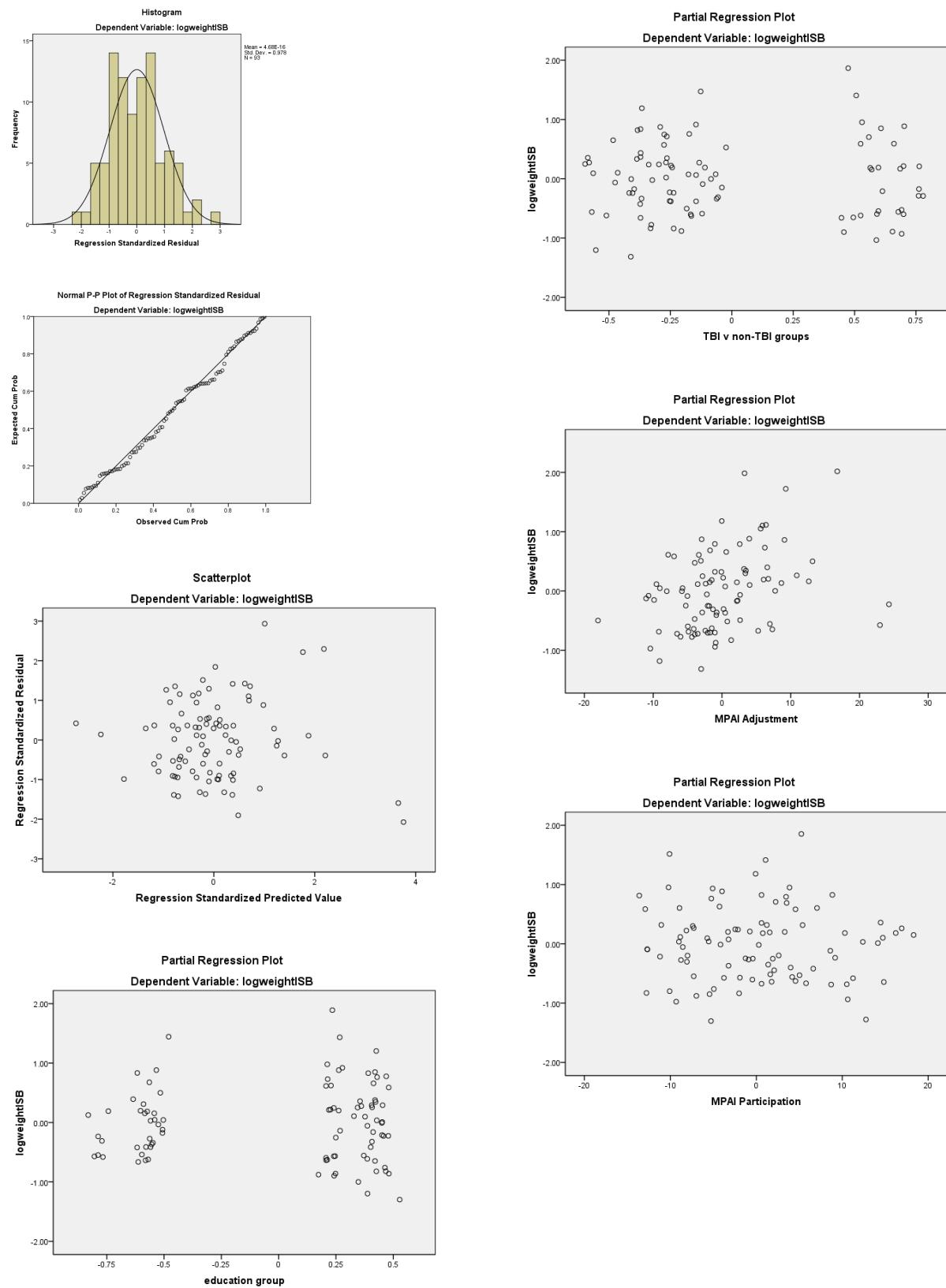
## APPENDIX 2: Plots for linear regression residuals – verbal aggression



## APPENDIX 3: Plots for residuals – physical aggression



## APPENDIX 4: Plots for residuals – inappropriate sexual behaviour



## APPENDIX 5: Correlations between MPAI-4 items and outcomes

MPAI-4 items	Verbal aggression		Physical aggression		Inappropriate sexual behaviour	
	n=301	n=173	n=301	n=110	n=301	n=93
Part A. Ability						
1. Mobility	.06	.10	.08	.37**	.02	.07
2. Use of hands	-.02	.14	.09	.30**	-.08	.01
3. Vision	.07	.01	.03	.11	-.05	-.10
4. Audition	.00	.12	.02	.08	.00	-.02
4. Audition rescore	-.03	.12	.01	.07	-.02	.02
5. Dizziness	.08	.10	.10	.19*	.03	.23*
6. Motor speech	.07	.12	.12*	.33**	.02	.08
7A. Verbal communication	.08	.14	.17**	.30**	-.01	.25*
7B. Nonverbal communication	.17**	.17*	.14*	.37**	.09	.17
7. Communication (higher score)	.15*	.16*	.16**	.33**	.04	.21*
8. Attention/concentration	.11	.16*	.07	.42**	.11	.11
9. Memory	.12*	.05	.11	.21*	.00	.13
10. Fund of information	.13*	.21**	.15*	.21*	.05	.14
11. Novel problem-solving	.17**	.15	.19**	.41**	.05	.17
12. Visuospatial abilities	-.01	.04	.05	.14	.01	-.07
Part B. Adjustment						
13. Anxiety	.09	.20**	.07	.26**	-.04	.24*
14. Depression	.11	.19*	.11	.11	.07	.33*
15. Irritability, anger, aggression	.53**	.52**	.53**	.41**	.31**	.26*
16. Pain and headache	.10	.13	.07	.18	.00	.19
16. Pain and headache rescore	.13*	.13	.08	.15	.04	.19
17. Fatigue	.04	.05	.07	.34**	.02	.25*
18. Sensitivity to mild symptoms	.10	.05	.11	.05	-.03	.25*
19. Inappropriate social interaction	.50**	.39**	.48**	.32**	.42**	.36**
20. Impaired self-awareness	.31**	.24**	.26**	.15	.11	.26*
21. Family/other relationships	.14*	.21**	.18*	.23*	.01	.16
Part C. Participation						
22. Initiation	.15*	.18*	.18*	.29**	.01	.01
23. Social contact	.08	.21**	.09	.30**	.08	.23*
24. Leisure and recreation	.05	.17*	.06	.22*	.03	.11
25. Self-care	.03	.25**	.10	.43**	-.02	.20*
26. Residence	.07	.17*	.11	.24*	.05	.14
27. Transportation	.00	.10	.04	.21*	.03	.11
27. Transportation rescore	.01	.17*	.08	.30**	.04	.15
28A/B. Employment	.07	.08	.06	.11	.04	-.01
29. Managing money and finances	.12*	.21*	.21**	.22*	.03	.33**
Part D. Pre-existing and associated conditions						
30. Alcohol use pre-injury	.12*	.28**	.14*	.05	.15*	-.08
30. Alcohol use post- injury	.17**	-.01	.08	-.11	.06	.12
31. Drug use pre- injury	.12*	.21**	.14*	.19*	.12*	-.01
31. Drug use post-injury	.15**	.00	.10	-.09	.14*	.14
32. Psychotic pre-injury	.03	-.04	.03	-.11	.07	-.26*
32. Psychotic post-injury	.23**	.29**	.19**	.12	.20**	.24*
33. Law violations pre-injury	.10	.17*	.06	.10	.09	.01
33. Law violations post-injury	.17**	.07	.13*	.12	.12*	.21*
34. Other physical pre-injury	-.04	-.07	-.07	.10	-.05	.11
34. Other physical post-injury	-.04	-.03	-.09	.02	-.06	.02
35. Other cognitive pre-injury	.03	.08	.13*	.13	.00	-.05
35. Other cognitive post-injury	.08	.08	.13*	.09	.03	-.03

\* p < .05. \*\* p < .01.