Copyright Statement

The digital copy of this thesis is protected by the Copyright Act 1994 (New Zealand). This thesis may be consulted by you, provided you comply with the provisions of the Act and the following conditions of use:

- Any use you make of these documents or images must be for research or private study purposes only, and you may not make them available to any other person.
- Authors control the copyright of their thesis. You will recognise the author's right to be identified as the author of this thesis, and due acknowledgement will be made to the author where appropriate.
- You will obtain the author's permission before publishing any material from their thesis.

To request permissions please use the Feedback form on our webpage.
http://researchspace.auckland.ac.nz/feedback

General copyright and disclaimer

In addition to the above conditions, authors give their consent for the digital copy of their work to be used subject to the conditions specified on the Library Thesis Consent Form
The Neglected Offender: Exploring the Role of Executive Dysfunction in Violent Offending

Sheree A. Crump

A thesis submitted for the degree of
Doctor of Philosophy in Psychology
University of Auckland
January 2005
Executive dysfunction and aggression and violence have been consistently associated in the research literature over at least the last 50 years. Some literature documents profound behaviour changes and the development of antisocial traits after changes to the prefrontal cortex and its supporting neural networks. Other research makes tentative links using brain imaging and neuropsychological methods. Prefrontal dysfunction can lead to executive dysfunction, and executive dysfunction may constitute a pathway to violence. Robust demonstrations of the association between executive function and violence have been limited by poor methodology in previous studies. This study attempts to address methodological concerns and uses a neuropsychological battery to examine the role of the executive functions in the expression of violence through violent offending. It is hypothesised that problems with executive functioning as measured by a neuropsychological battery may be linked to violent offending through impulsivity and disinhibition.

Two studies were completed with sixty participants across the two groups. In the first study three groups of men were assessed on tests of executive dysfunction; 21 inmates convicted of violent crimes; 16 inmates convicted of non-violent crimes; and a matched control sample of 16 men in the community who are
conviction free. Results demonstrate a positive relationship between executive
dysfunction and violent offending. A similar association was demonstrated
between the length of formal education each participant across the sample
reported, with less years of education increasing the likelihood of participants
being in one of the incarcerated groups.

The second study examined neuropsychological differences between seven
murderers; three who killed impulsively and four who premeditated a murder.
There were no conclusive results from this study, which may be due to the small
sample size.

It is suggested that identifying pathways to violence can inform therapeutic
interventions. The indication of executive dysfunction as a pathway to violence
was used to develop an intervention specifically geared towards executive deficits
in violent offenders. The third study, Study Three (which was not completed due
to constraints imposed by the research setting beyond the control of the
researcher) includes the design of a study to assess the outcome of an intervention
for inmates with ED. The intervention itself is appended to this thesis.
Acknowledgements

My first thanks goes to all the participants that gave their time and energy to this thesis. Without your openness, your time, and your stories this project could not have gone forward. Thank you to all the staff at the Department of Corrections, both at Auckland Prison, and at Manukau Community Probation Services, and to Psychological Services at The Department of Corrections, and the staff at Department of Corrections Head Office. Thank-you to the staff at the community work and education institutions that helped in collection of participants.

My biggest thanks go to Associate Professor Jenni Ogden, my supervisor. It has been a long road that we have travelled; I thank you for your patience, your knowledge, and your belief in this work. Your help has been invaluable, and you reminded me many times of the forest when I was stuck in the trees. Thanks also to my second supervisor Dr Andrew Moskowitz. You provided me with some readings early on that fired me up, and to this day still motivate me.

Thank you so much Rob McNeil and Professor Mike Corballis for you help and advice on my results and statistical method. Daniel Shepard you also deserve thanks (and maybe some more shortbread) for all you advice as well.
To my family and friends who have stuck with me through times when my sanity was barely hanging on, I raise a glass to you now. Thank you mum for always boring your friends with how proud you are of me. Thank you nana for keeping the family up-to-date. Thank you Mary Grogan for your pep talks and regularly re-inflating my ego when it had been punctured. Thank you Karren Towgood, my room mate, who has had to put up with my unique filling system that has cluttered my half of the office, and for your invaluable help in formatting this thesis. Thank-you so much Mary, Karren, and Anna Clarkson for shining the touch in my darkest moments and providing the extra eyes required. Thank you Mary and Anna for your incredible help. The rest of you, Daniel Bassett, Michael Savage (who thought I’d never leave university), Natalie Flynn, my clinical classmates, all of you who are too many to list, thanks for all the drinks, the discussions, and the belief. Thank-you Caroline and Rebecca Leys for your keen eye in checking my grammar and flow in an earlier draft. And of course thanks to you my partner Tom Leys for your tremendous love and support, and I know you are as glad as I am that I have finally finished writing this thesis!
# Table of Contents

**ABSTRACT** II  
**ACKNOWLEDGEMENTS** IV  
**TABLE OF CONTENTS** VI  
**LIST OF TABLES** IX  
**LIST OF FIGURES** X  
**LIST OF ABBREVIATIONS** XI  
**1 INTRODUCTION** 1  
  1.1 AIMS OF THE THESIS 1  
  1.2 RATIONALE FOR STUDY 2  
  1.3 STRUCTURE OF THE THESIS 4  
**2 BACKGROUND** 5  
  2.1 NEUROANATOMY 5  
  2.1.1 The Frontal lobes 5  
  2.1.2 The Prefrontal Cortex 9  
  2.1.3 The Prefrontal/Subcortical Circuits 11  
  2.1.4 Summary of Neuroanatomy 18  
  2.2 CONCEPTUALISATIONS OF EXECUTIVE FUNCTION 19  
  2.2.1 The Cognitive Functions of the Prefrontal cortex 19  
  2.2.2 Executive Function in Practice 24  
  2.2.3 Limitations in Executive Testing 38  
  2.2.4 The Behavioural Assessment of the Dysexecutive Syndrome Battery 39  
  2.3 ISSUES PERTINENT TO THE CURRENT STUDY 42  
  2.3.1 The Prison Population in New Zealand 42  
  2.3.2 Cultural Issues 42  
  2.4 SUMMARY OF BACKGROUND 43  
**3 REVIEW OF THE LITERATURE** 45  
  3.1 VIOLENCE 47  
  3.1.1 A Note on Gender 49  
  3.2 NEUROBEHAVIOURAL FINDINGS IN VIOLENCE AND OFFENDING 51  
  3.2.1 Genetics 52  
  3.2.2 Neurotransmitters 56  
  3.2.3 Hormones 63  
  3.2.4 Animal Lesion Research 68
3.2.5 Human Lesion Research 70
3.2.6 EEG and Imaging Studies of the Brain-Violence Connection 78
3.2.7 Alcohol and Drug Use 89
3.2.8 Social and Developmental Factors 97
3.2.9 Executive Dysfunction Explanations of Offending and Violence 103
3.3 SUMMARY OF NEUROBEHAVIOURAL FINDINGS IN VIOLENCE AND OFFENDING 120
3.4 METHODOLOGICAL FLAWS OF EXISTING REVIEWED LITERATURE 123

4 STUDY ONE: VIOLENT AND NON-VIOLENT OFFENDERS, AND NON-VIOLENT COMMUNITY CONTROLS 137
4.1 INTRODUCTION 137
4.2 METHOD 138
4.2.1 Aims and Hypotheses 138
4.2.2 Recruitment 139
4.2.3 Selection Criteria 142
4.2.4 Explanation of Inclusion and Exclusion Criteria 143
4.2.5 Consent Procedures 144
4.1.6 Definition of Violence 146
4.2.7 Procedure 147
4.2.8 Measures 148
4.2.9 Test Order 164
4.2.10 Analysis 165
4.3 RESULTS 166
4.3.1 Demographic Data 167
4.3.2 Relationship of Variables 170
4.3.3 Examination of Means 172
4.3.4 Relationship between Executive Dysfunction and Participant Type 175
4.3.5 Substance Abuse 176
4.3.6 Traumatic Brain Injury 178
4.4 DISCUSSION 179
4.5 CONCLUSION 198

5 STUDY TWO: NEUROPSYCHOLOGICAL DIFFERENCES BETWEEN IMPULSIVE AND PREMEDITATED MURDERERS 199
5.1 INTRODUCTION 199
5.2 METHOD 202
5.2.1 Aims and Hypotheses 202
5.2.2 Procedure 202
5.2.3 Impulsive verses Premeditated 204
5.3 RESULTS 206
5.3.1 Demographic Characteristics 206
5.3.2 Neuropsychological results 207
5.3.3 Qualitative Results 208
5.3.4 Intoxication During Offending 209
5.3.5 Alcohol and Drug Use History 211
List of Tables

Table 1: Table describing some common broad deficits in behaviour noted when damaged sustained to the prefrontal cortex

Table 2: Neurobehavioural Evidence for Brain Dysfunction in Violent or Antisocial Individuals

Table 3: Demographic Characteristics of Study One Sample

Table 4: Summaries Of Selection and Data Collection Procedure for Study One

Table 5: Basis for ED Pattern Category

Table 6: Kruskal Wallis Test: Comparison of Ethnic Identity across Participant Type, test scores, and Years of Education

Table 7: Spearman’s rho Correlations

Table 8: Mean score (SD) of major tests and years of education for all groups

Table 9: Results of Kruskal-Wallis Tests across all the Participant Types for total BADS score, BDI-II and Years of Education

Table 10: Mean Scores (SD) for all Participant Types and Kruskall-Wallis Test on the BADS subtasks

Table 11: Results of Kruskal-Wallis Tests across all the Participant Types for total BADS score, BDI-II and Years of Education with participants who have reported drug use in the last 48 hours removed

Table 12: Summary of Selection and Data Collection Procedure for Study Two

Table 13: Demographic Characteristics of Murderer Sample

Table 14: Presentation of Neuropsychological Results of Murderer Sample

Table 15: Presentation of Qualitative Results

Table 16: Summaries of Selection and Data Collection Procedure for Study Three

Table 17: General Results for Study One

Table 18: Summary of Selection and Data Collection Procedure for Study Four

Table 19: Demographic Characteristics of Murderer Sample

Table 20: Results of Kruskal-Wallis Tests across all the Participant Types for total BADS score, BDI-II and Years of Education with participants who have reported drug use in the last 48 hours removed
List of Figures

Figure 2.1   View of the brain surface from above (Medline plus health information. www.nlm.nih.gov/medlineplus/ency/imagepage/1074.htm) ..... 6

Figure 2.2   Lateral view of left hemisphere of the brain (from Ogden, J.A. 1996; p. 7 with permission)................................................................. 7

Figure 2.3   Mesial view of left hemisphere of the brain (from Ogden, J.A. 1996; p. 7 with permission)................................................................. 7

Figure 2.4   Functional zones of the brain in a left lateral view (from Ogden, J.A. 1996; p. 10 with permission) ......................................................... 8

Figure 2.5. General structure of frontal subcortical circuits (adapted from (Tekin & Cummings, 2002))........................................................................ 12
List of Abbreviations

BADS - Behavioural Assessment of the Dysexecutive Syndrome

BDI-II - Beck Depression Inventory – II

COWA - Controlled Oral Word Association

COWA-M - Controlled Oral Word Association Maori Version

dlPFC - Dorsolateral Prefrontal Cortex

ED - Executive Dysfunction

OFC - Orbitofrontal Cortex

SCOLP - The Speed and Capacity of Language Processing Test

SD - Standard Deviation

STW - Spot the Word Vocabulary Test

TFR - The Test of Facial Recognition
1

Introduction

Experience is not what happens to a man; it is what a man does with what happens to him.

Aldous Huxley (1894 - 1963)

1.1 Aims of the Thesis

This thesis investigates whether executive dysfunction (ED) is related to categories of offending. The primary aim of this thesis is to assess whether deficits in executive functioning are more highly correlated with a history of violent offending than with a history of non-violent offending, or no offending. The assumption driving this investigation is that executive dysfunction will exacerbate or augment any tendency towards violent offending. Illuminating the role of executive dysfunction in relation to violence will inform rehabilitative interventions.

Three studies are presented. The first study examines the presence of executive dysfunction, as measured by a battery of neuropsychological tests in three samples of the population; violent and non-violent offenders, and a non-violent community control group. The second study explores neuropsychological differences between offenders who murder impulsively and offenders who plan their murders. The
implications of the studies in this thesis are largely applied; that is, it is anticipated that the results will add to current literature and knowledge about the cognition underlying violent behaviours, and will improve our ability to design and implement rehabilitation programmes that are effective for violent offenders with ED. The third study includes an outcome design to measure such a rehabilitation programme that takes into account the neuropsychological difficulties demonstrated by the sample.

In this research the emphasis is on impaired executive \textit{behaviours}. No imaging data pertaining to specific regions of the brain mediating executive function, or ‘hard signs’ of types of neuroanatomical dysfunction possibly present in individuals in the sample is available for examination. Aetiology regarding any ED that is present is also unavailable. However, consideration of the different geneses of ED (as measured by neuropsychological testing in the sample), and the types of brain damage that offenders are likely to encompass, is discussed.

Because of the strong link in the literature between compromised prefrontal cortex function leading to poor executive functioning and the expression of violence (Hawkins & Trobst, 2000), throughout the thesis references will be made to the prefrontal cortex as the neuroanatomical location of most interest. Other neuroanatomical structures that have been associated with aggressive behaviour and violence will be discussed briefly, predominantly with a reference to their interaction with prefrontal functioning and executive functioning.

\section*{1.2 Rationale for Study}

Dysfunction of the prefrontal cortex and the resulting association of this with aggression has been clinical lore for more than 50 years (Hawkins & Trobst, 2000). As executive dysfunction can impair an individual’s ability to predict the future consequences of an action, and decrease inhibition, it can impact on the
ability to make socially acceptable choices, sometimes leading to choices that break the law. Executive dysfunction may also impact on criminal recidivism due to the mix of executive deficits and environmental factors that can affect decision-making (Valliant, Freeston, Pottier, & Kosmyna, 2003), such as substance abuse, stressors (for example, poverty, peer influences), and situational factors (such as opportunity). Further, crimes may offer immediate gratification; they are often simple and easy to commit, requiring few formal skills or little academic education. Most crimes are spontaneous, and the obvious lack of planning is consistent with their being viewed as impulsive actions.

It is also interesting from a theoretical viewpoint to examine executive dysfunction in inmates, who are not routinely examined for such difficulties and therefore possibly not treated effectively while incarcerated. Information about brain-behaviour relations often come from populations that have acquired a brain injury, have experienced hospitalisation and medical follow-up, and are being cared for in rehabilitation programs. It is conjectured that many New Zealand inmates would fit a profile demonstrating executive impairments because of the high number of inmates who have experienced an assault to the head and face region, possibly with minimal medical follow-up. A study by Barnfield and Leathem (1998) conducted at a New Zealand prison found that “86.4% of the sample had sustained a TBI at some time in their lives, and more Māori subjects (91.4%) reported TBI than non-Māori subjects (79.4%),” (p.455). High rates of TBI amongst inmates, along with the evidence pointing to many crimes being impulsive or ‘opportunistic’ (Farrington, 1991b, 1994) with the offender ultimately being caught, make prisons potentially rich environments for research on ED and violence.

It may also be that inmates with poor executive functioning would be candidates for recidivistic offending. Functions that may assist recidivism include a lack of error utilisation, problems in accessing newly learnt information, and difficulty in following rules and understanding future consequences of current actions. Valliant
et al. (2003) found that the Wisconsin Card Sorting Test (WCST) significantly differentiated recidivist and non-recidivist offenders and related this to deficits in shifting set, poor prediction of consequences, planning, and goal-directed behaviour.

Consistent links have been noted throughout the literature between prefrontal damage (and/or developmental prefrontal problems), and executive deficits, and between these and increased aggression and acts of violence (Hawkins & Trobst, 2000). Some literature describes profound change and acquirement of ‘unsavoury’ and unsocial traits after ablation or damage to various parts of the prefrontal cortex, whilst other research makes more tentative links using imaging and neuropsychological methods, or the examination of retrospective information. The literature is not always clear in being able to link executive deficits to violence, nor is it able to explain entirely the aetiology of violent behaviour. Some of this is due to limitations in methodology, which this study has attempted to address, i.e., clear definition of offending types, the use of a matched control group, and the use of a comprehensive battery to assess ED.

Much work has been devoted to the examination of the prefrontal cortex over the last few decades, and more recently the functionality of the executive behaviours, and the impact they have on aggression and violence. Most of this work has been based in North America. A secondary aim of this current study is to see if similar patterns could also be demonstrated in a New Zealand population.

### 1.3 Structure of the Thesis

Following this introduction chapter, Chapter Two outlines the basic anatomy of the frontal lobes and their vast connections. This is kept brief, as it is not the purpose of this thesis to be an exhaustive account of the anterior regions of the brain, but simply to provide the language and framework of this thesis. Other neuroanatomical structures pertinent to aggression are also briefly reviewed. Next
the purported cognitive functions of the prefrontal cortex, or the executive functions, are presented to further extend the context for this thesis. This leads onto a presentation of some of the problems observed from a neuropsychological perspective in individuals who have compromised executive functioning.

In Chapter Three the literature on neurobehavioural accounts of violence and offending is reviewed, and a closer exploration of the links between violence and ED is presented, beginning with a general overview of violence. Chapter Four presents Study One and examines ED across the three groups. The specific hypotheses are presented in the methods of this chapter, and following the results there is an initial discussion of Study One. Chapter Five presents Study Two, which focuses on a murderer population using neuropsychological methods to examine for differences between premeditated murderers and impulsive murderers. Chapter Six details the methodology proposed to measure the outcome of a specialised intervention for inmates with ED. The intervention itself is appended and is based on indicators from Study One and Two. Finally, Chapter Seven provides a General Discussion of the entire thesis, and explores the findings from the studies in relation to each other and the existing literature, and the strengths and limitations of the present studies. A discussion of how problematic executive functioning may contribute to the development of violent behaviour is presented, along with suggestions for future research.
2 Background

The seat of the soul and the control of voluntary movement - in fact, of nervous functions in general, are to be sought in the heart. The brain is an organ of minor importance.

Aristotle (from De motu animalium, 4th century B.C.)

2.1 Neuroanatomy

The anterior cortex of the central nervous system will be described to set the context for the reader. The prefrontal cortex in particular is of importance and time will be spent exploring the links between locality and functionality, including how these functions are measured. This chapter provides the background that informs the theories of prefrontal function, and in particular, problematic executive functioning.

2.1.1 The Frontal lobes

Anatomically the frontal lobes include the large cerebral area anterior to the central sulcus and above the temporal gyrus and are roughly symmetrical. With their mesial border at the corpus-callosum, the frontal lobes comprise the largest area of cortical space. Various authors furnish different estimates, from 20%-30% (Kolb & Whishaw, 1990; Martin, 1998) of cortical area devoted to the frontal lobes, to as much as 50% (Damasio, 1991; Darling, Della Sala, Gray, & Trivelli,
From a functional perspective the frontal lobes are traditionally divided into the motor strip, the premotor strip, the limbic system, and the prefrontal cortex (see Figures 2.1, 2.2, and 2.3). The prefrontal cortex is located at the anterior pole of the brain, and is generally accepted as ending at the premotor strip. The blood supply to the frontal lobe is carried to the lateral surface by the middle cerebral artery (MCA), to the medial surface by the anterior cerebral artery (ACA) and to the inferior surface by both the MCA and ACA.

Figure 2.1 View of the brain surface from above (Medline plus health information. www.nlm.nih.gov/medlineplus/ency/imagepage/1074.htm)
The functions of the brain can be further understood through the division of the hemispheres into primary, secondary, and tertiary cortical zones (Figure 2.4). Luria conceptualised this way of understanding functional systems that can provide a useful aid to understanding even today. Luria hypothesised that the
primary zones are primary projection areas that contain sense-modality-specific neurons to which all incoming sensory information is projected (Luria, 1973). The secondary zones are also known as the association cortex. Modality-specific information is sent to the secondary zones from the adjacent primary zones, where it is integrated into meaningful wholes (Luria, 1973). The tertiary zones are where the integration of information across sense modalities occurs and is the area of the cortex central to this thesis. This zone is also known as the prefrontal cortex and is involved in executive functions, including the organisation of behaviour and thinking, planning, inhibition, and the ability to think in an abstract manner (Luria, 1973).

Figure 2.4  Functional zones of the brain in a left lateral view (from Ogden, J.A. 1996; p. 10 with permission)
2.1.2 The Prefrontal Cortex

In evolutionary terms the prefrontal cortex is the youngest region, both phylogenetically and cytogenetically, i.e., it was the last area of brain to develop in human evolution, and it is the last part of the brain to mature in a human. This region exhibits considerable growth during childhood and into adolescence (see Happaney, Zealazo, & Stuss, 2004 for a review). In view of what has been understood about the growth of the prefrontal cortex from middle childhood onwards, traditionally it was thought the prefrontal cortex was not functional until this time. However, current evidence indicates that some degree of prefrontal function emerges, and is required, as early as towards the end of the first year of life (Happaney et al., 2004).

When compared to other animals, particularly primates such as the macaque monkey and chimpanzee, the large size of the prefrontal area in humans is striking (Darling et al., 1998). It has been asserted that humans are also the only species in which the prefrontal cortex takes the largest portion of total cortical space (Darling et al., 1998). Benson (1994) remarks that because of the prefrontal cortex’s “massive” size, it has often been regarded as the key to human distinctiveness. Because of this factor many believe the prefrontal cortex to be the seat of our self-awareness and self-reflection.

However recent information from Semendeferi, Schenker, and Damasio (2002) suggests there is no difference in the overall size of the frontal cortices between humans and the great apes. They claim that most comparative work has been done on primates other than the great apes, such as the macaque monkey, which accounts for the dramatic differences seen. Semendeferi et al. (2002) suggest that the great differences observed between the behaviour of humans and primates lower on the evolutionary scale than the great apes, are most likely due to differences in size in other cortical areas, and a greater connectivity of the frontal area with other cortices in humans.
The prefrontal cortex can be divided into three gross areas: dorsolateral, orbitofrontal (or basilar-orbital), and anterior cingulate cortex (or mesial) (Fuster, 1997). These three areas, along with subserving circuitry and connections, are each alleged to be responsible not only for overall integration of all human behaviour but also to mediate separate functions leading to distinct behaviours. For example, the orbitofrontal region is the part of the prefrontal cortex posited as most concerned with inhibition of prepotent responding, insight, and expression of emotion due to its intimate links with the limbic system, whereas mesial damage will result in blunted affect, loss of initiative, and apathy (Stuss & Benson, 1983, 1984). High-level functions such as planning, cognitive flexibility, temporal ordering of recent events, regulating actions based on environmental stimuli, and learning from experience, are mediated primarily by the dorsolateral cortex and its subcortical connections (Heyder, Suchan, & Daum, 2004). Table 1 presents details of which behaviours are broadly present if one of the three areas is damaged.

Table 1: Table describing some common broad deficits in behaviour noted when damaged sustained to the prefrontal cortex

<table>
<thead>
<tr>
<th>Region</th>
<th>Behavioural Deficits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dorsolateral Cortex</td>
<td>cognitive disorganization</td>
</tr>
<tr>
<td></td>
<td>concrete thinking</td>
</tr>
<tr>
<td></td>
<td>perseveration</td>
</tr>
<tr>
<td></td>
<td>planning difficulties</td>
</tr>
<tr>
<td></td>
<td>loss of goal directed behaviour</td>
</tr>
<tr>
<td>Orbitofrontal Cortex</td>
<td>behavioural disinhibition</td>
</tr>
<tr>
<td></td>
<td>impulsivity</td>
</tr>
<tr>
<td></td>
<td>environmental dependency</td>
</tr>
<tr>
<td></td>
<td>inappropriate jocularity</td>
</tr>
<tr>
<td></td>
<td>lack of interpersonal sensitivity</td>
</tr>
<tr>
<td>Anterior Cingulate Cortex</td>
<td>apathy</td>
</tr>
<tr>
<td></td>
<td>appearance is depressed</td>
</tr>
<tr>
<td></td>
<td>lack of motivation</td>
</tr>
<tr>
<td></td>
<td>inhibited behaviour</td>
</tr>
<tr>
<td></td>
<td>poor planning</td>
</tr>
</tbody>
</table>
2.1.3 The Prefrontal/Subcortical Circuits

A large part of the knowledge of the representation of violence in the human brain is based on observations of individuals who commit violent acts (Filley et al., 2001). There has been no discovery of a ‘violence centre’ in the brain, but two functional systems, the prefrontal cortex and the limbic system, are thought to play significant roles (Filley et al., 2001). The limbic system is a group of structures widely believed to underlie many aspects of emotion and emotion based learning (Phan, Wager, Taylor, & Liberzon, 2004). Critical structures involved in emotion include the hypothalamus, hippocampal formation, parahippocampal gyrus, cingulate gyrus, septal region, and the amygdale (Phan, Wager, Taylor, & Liberzon, 2004).

Lesion studies examining the role of the hypothalamus and the amygdala identify these structures as those most implicated in limbic explanations of violence. For example, purposeful posterior hypothalamus lesions made in children with ‘uncontrollable violence’ reduced violent behaviour considerably and these changes remained constant during the 10-25 year follow-up, without compromising IQ, endocrine activities, and growth (Sano & Mayanagi, 1988). However, the strongest evidence implicates the involvement of the amygdala in violent behaviour. Amygdala related explanations of violence will be explored in this section after discussion of the broader prefrontal divisions.

Behavioural and neuroimaging evidence implicating circuits linking the prefrontal cortices to subcortical structures have been well described and accepted in the literature as the necessary neural underpinnings of executive functioning and emotional and social behaviour. Five generally accepted parallel basal ganglia thalamic-cortical circuits have been described in the literature (Alexander, DeLong, & Strick, 1986; Cummings, 1995; Tekin & Cummings, 2002). All five circuits share common features (Alexander et al., 1986), and these are represented in a figure adapted from Tekin & Cummings (2002; Figure 2.5).
Three of these five circuits are proposed to be involved in emotional and motivational processes. These processes can be viewed functionally as executive behaviour, and are described below with the other structures and regions involved. The other two circuits are primarily involved in motor activity and will not be described.

It is the dorsolateral prefrontal, orbitofrontal, and anterior cingulate cortex circuits that are thought to be dedicated to executive functions, emotional and motivational states, and social behaviour (Alexander et al., 1986; Cummings, 1995; Tekin & Cummings, 2002). Dysfunction in these three circuits will generally result in cognitive and behavioural impairments that are analogous to deficits realised after prefrontal cortex lesions (Heyder et al., 2004).
Dorsolateral Prefrontal Cortex

The dorsolateral prefrontal cortex (dIPFC) originating in Brodmann’s areas 9 and 10 (Tekin & Cummings, 2002) on the lateral surface of the anterior portion of the prefrontal cortex appears to have the greatest number of reciprocal cortico-cortico connections, including connection with the parietal association cortices, orbitofrontal cortex, and cingulate cortex. Subcortical connections comprise a circuit starting with projections from the dIPFC to the dorsolateral caudate nucleus, then from this site to the lateral mediodorsal globus pallidus interna and then to rostral portions of the substantia nigra. Next projections reach the ventral anterior thalamic nucleus, which in turn projects to regions of the prefrontal cortex, thus completing the circuit (Alexander et al., 1986; Cummings, 1995; Tekin & Cummings, 2002).

The dIPFC and its connections are involved in executive function and working memory (Andres & Van der Linden, 2002; Goldman-Rakic, 1996). The executive functions the dIPFC are involved in include the learning of new information, planning, self-regulation in response to environmental stimuli, temporal ordering of recent events, accessing remote memories, and shifting and monitoring appropriate behavioural sets (Duffy & Campbell, 1994); which includes multitasking (Burgess, 2000) and goal directed behaviour (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). Compromise in the dIPFC region and its circuitry is also thought to lead to reduced frustration (Giancola, 1995; Ridderinkhof et al., 2004; Seguin, 2004; Seguin, Pihl, Tremblay, & Boulerice, 1995). Therefore, individuals with damage to the dIPFC and/or the subcortical circuitry are often concrete and perseverative, have reduced mental flexibility and reasoning ability, and may have difficulty in accessing internalised rules for regulating behaviour. Due to a reliance on external cues, dIPFC damaged patients can display a lack of spontaneous behaviour and appear apathetic unless motivated by an environmental source. Alternatively this reliance can cause individuals to be reactive to environmental changes and cues with minimal
response regulation. Individuals with dIPFC damage can perform within expected normal limits on tests designed to access learned knowledge or sensory-motor skills, but deficits will be noted in those tasks that require working memory and problem-solving (Baddeley & Della Sala, 1996; Baddeley, Della Sala, Papago, & Spinnler, 1997; Heyder et al., 2004).

**Orbitofrontal Cortex**

The lateral orbitofrontal cortex (part of Brodmann’s area 10) and the orbitomedial frontal cortex (Brodmann’s area 11) begin a circuit by sending fibres to the ventromedial caudate nucleus (Tekin & Cummings, 2002). Neurons from here then project to the medial mediodorsal globus pallidus interna as well as to a rostromedial portion of the substantia nigra pars reticulata. Fibres from these areas then connect with ventral anterior and medial portions of the thalamus. These regions of the thalamus complete the loop with projections to the lateral orbitofrontal cortex (Tekin & Cummings, 2002).

Damage to the orbitofrontal cortex (OFC) and circuits can result in disturbances in complex social and emotional behaviour with a disruption in socially appropriate responding (Cicerone & Tanenbaum, 1997). Lesions in the OFC typically result in descriptions of individuals as disinhibited, impulsive, and irresponsible who constantly misinterpret others’ moods, have lack of regard for others and lack of concern for their own actions, poor insight, and reduced initiative (Blair, 2004; Dinn & Harris, 2000; La Pierre, Braun, & Hodgins, 1995; Seguin, 2004).

There are parallels between the effects of OFC lesions and antisocial personality disorder, and questions concerning the underpinning of antisocial disorders have been sought through a prefrontal cortex account (Seguin, 2004), and lately more specifically an OFC account (Blair, 2004). Impairment of the OFC in infants can result in an inability to acquire social and moral reasoning, and damage to the
OFC in adulthood can impair access to and implementation of previously acquired social and moral rules (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999).

**Anterior Cingulate Cortex**

The anterior cingulate cortex (ACC) and the general prefrontal cortex are functionally connected, amalgamating the ACC into the system of executive control (Heyder et al., 2004). The ACC circuit has its origins in Brodman’s rostral area 24 (Tekin & Cummings, 2002) and areas 25 and 33 (Devinsky, Morrell, & Vogt, 1995). Projections into the limbic striatum (which encompasses the ventral striatum, ventromedial caudate, ventral putamen, nucleus accumbens, and olfactory tubercle), reach into the mediodorsal and ventral anterior thalamus and continue back to the anterior cingulate cortex, thereby completing the loop.

Damage to the ACC can result in deficits similar to those seen in lateral prefrontal cortex lesions, such as inattention, reduced initiation, and decreased drive and motivation (Duffy & Campbell, 1994; Fuster, 1997). Although comprehensive research on the ACC is minimal relative to the dIPFC and the OFC, research to date suggests that ACC damage will result in reducing performance in developing plans and in the execution of plans (Carlin et al., 2000). Some patients with epilepsy originating in the ACC have been observed to exhibit sudden aberrant personality changes, sometimes violent, that appear during the interictal phase (Devinsky et al., 1995; Diaz, 1995; Filley et al., 2001). Increases in tics and obsessive-compulsive-type behaviours have also been noted in patients with elevated ACC activity (Devinsky et al., 1995; Yucel et al., 2003). Further, along with the amygdala, the ACC is involved in evaluating motivational content, expressing internal states, and assigning of emotional valence to stimuli, both internal and external (Devinsky et al., 1995; Yucel et al., 2003). Additionally the ACC implements a conflict-processing role that recruits cognitive control accordingly (Kerns et al., 2004). Reduced cingulate activity has been associated with akinetic mutism, diminished insight, depression, motor-neglect, and impaired motor initiation (Devinsky et al., 1995).
Overall, the ACC appears to play a crucial role in initiation, motivation, and goal-directed behaviours (Devinsky et al., 1995; Kerns et al., 2004; Yucel et al., 2003). Studies of the ACC and disorders associated with ACC dysfunction appear most often in a body of work that has been described as “affective neuroscience;” for examples see (Berthoz, Artiges et al., 2002; Berthoz, Blair, Le Clech, & Martinot, 2002; Charney, 2004; Eisenberger & Lieberman, 2004; Hofer, 2003; Konner, 2004; Muller et al., 2003; Phillips, Drevets, Rauch, & Lane, 2003).

Amygdala Connections

As well as the three neural circuits described above, a complex circuit consisting of the OFC, the amygdala, and the ACC has been implicated in various facets of emotional affective style and expression (Davidson, Putnam, & Larson, 2000). This circuit along with involvement from the dorsolateral prefrontal cortex, the hippocampus, hypothalamus, insular cortex, and other interconnected regions are involved in the amplification and maintenance of emotion. Emotional modulation is considered to result from complex neurochemical interactions of the hypothalamus, the amygdala and the prefrontal cortex (Fellous, 1999).

Both the amygdala and the orbitofrontal cortex (OFC) are implicated in the modulation of the circuitry that mediates reactive aggression (Blair, 2004), although their roles do differ. Deficits in mood and motivation are demonstrated following lesions or removal of sections of the amygdala (Isenberg et al., 1999; Phillips et al., 2003), traditionally demonstrated in animals, but with corresponding human literature. Studies examining lesions in the amygdala have implicated the amygdala in the roles of fear (Hamann, Ely, Hoffman, & Kilts, 2002; Veit et al., 2002), reward based learning (Hamann et al., 2002; Kringelbach, O'Doherty, Rolls, & Andrews, 2003), the startle response (Hamann et al., 2002; Phillips et al., 2003), as well as the evaluation of verbal information (Isenberg et al., 1999) and nonverbal body or facial expressions (Blair, 2003; Winston, O'Doherty, & Dolan, 2003).
Extreme bi-lateral amygdala damage may result in a phenomenon named Kluver-Bucy Syndrome. Six main symptoms have been associated with Kluver-Bucy Syndrome: visual agnosia; hyperorality; increased appetite; an irresistible urge to notice and then reject everything in sight; placidity and apathy; and altered (usually increased) sexual activity (Ozawa et al., 1997). What is notable in this syndrome is the extent of the placidity and lack of motivation, perhaps the antithesis of aggression.

The discovery that lesions in the medial amygdala in animals reduced fear in threatening situations (Gregg & Siegel, 2001), whereas electrical stimulation of medial regions of the amygdala produced aggressive reactions (Gregg & Siegel, 2001; Ursin & Kaada, 1960), resulted in the use of stereotaxic amygdalotomy for control of abnormal behaviour, including aggression, in humans (Lee et al., 1998; Narabayashi, Nago, & Saito, 1963). The resultant reduction in aggression after creation of lesions or removal of amygdala has been referred to as the ‘taming effect’ (Lee et al., 1998). Further descriptions of the taming effect are provided in section 3.2.5 Human Lesion Research.

In modern accounts of amygdala destruction and behavioural consequences in humans, the results are less convincing than those demonstrated in animal work, and a taming effect is thought to occur due to the inability of the amygdala to now process perceived threats that would normally activate the fight-or-flight response (Lee et al., 1998). This results in a reduction in automatic arousal levels, resulting in a decline in the number of aggressive outbursts, but does not change levels of actual control over aggression (Lee et al., 1998). Further, in more recent research in understanding the role of the amygdala in emotional regulation and affect, Anderson and Phelps (2002) propose from their results that the amygdala may be recruited for affective states, but is not necessary for their production. They suggest this will account for why there has historically been little evidence of measurable altered emotional experience following amygdalotomy in humans (Anderson & Phelps, 2002). This is similar to the conclusion drawn by Fellous...
(1999) who states that the amygdala is not an ‘emotional centre’ as traditionally viewed, but that instead it acts as a regulating filter, which actively searches for threatening stimuli to create a warning state about. This ‘amygdala filter’ sends information on for decisions regarding actions via projections directly with the OFC, and to the OFC and dlPFC via the temporal regions, the thalamus and the hypothalamus (Fellous, 1999).

The amygdala is thought to influence the development of morals. However, the neural organisation of moral emotions remains poorly understood (Moll et al., 2002), partially because the cognitive descriptors and values that we assign to them are culturally loaded. In an attempt to delineate the structures that serve basic and moral emotions, Moll et al. (2002) conducted an fMRI examination of seven normal subjects. The participants viewed emotionally charged material, and the fMRI analysis indicated activity in the thalamus, amygdala, and medial prefrontal cortex. Moral emotions also recruited portions of the ACC and temporal sulcus (Moll et al., 2002).

2.1.4 Summary of Neuroanatomy

The prefrontal cortex and its connections with deeper brain regions, particularly the limbic system, and with other cortices, play a particular role in mood and emotion, and in the development of and use of morals. The evaluation of relative risk for violence posed by limbic abnormalities alongside prefrontal cortex disinhibition is difficult to ascertain, but current evidence suggests that disturbance of the prefrontal cortex is the more powerful factor (Filley et al., 2001).

The prefrontal cortex is also intimately involved in the presentation of what is often conceptualised as ‘personality,’ and with a range of higher-order behaviours labelled executive functions. Executive functions include behaviours responsible
for functioning according strict societal rules, and in following and understanding rules, including the comprehension of complex subtle cues from other people.

The prefrontal cortex is understood to be at its most complex in humans. Its strong connections with the amygdala and other limbic structures result in this system having involvement in the modulation and expression of mood and emotion, as well as behaviours such as agitation, irritability, mania, depression, and aggression. Connections between the prefrontal cortex and the limbic system also enable the tertiary system to monitor and regulate impulses and information from the limbic system. The prefrontal cortex and the associated executive functions continue to develop throughout the life span, with the greatest maturation noted in adolescence. This gradual neurodevelopmental maturation reveals the significance of the interaction between genetic potential and environmental influences in forming viable structures in the brain that work at an optimum level.

Damage to the prefrontal lobes, or disruption of the pathways between the prefrontal cortex and the limbic system, may result in varying mood states or behaviours that were previously unusual for that individual. This disruption, and the behaviours that result, will be described in the following sections, along with an in-depth examination of what ‘executive function’ comprises and how executive functions are assessed.

### 2.2 Conceptualisations of Executive Function

#### 2.2.1 The Cognitive Functions of the Prefrontal cortex

The tertiary cortex, primarily the prefrontal cortex and its connections with subcortical and other cortical areas, mediate executive functions. Healthy executive functioning contributes to the ability to live an independent and satisfying life. Amongst other things, these functions guide our interactions with others and help us develop fulfilling relationships, assist in our learning of new
Background

material, and help us to simply get out of bed in the morning and go about our day.

While the terms ‘frontal lobe functions’ and ‘executive functions’ were synonymous in most of the literature of the previous century, neuroscientists currently view them as distinct, if overlapping, concepts (for examples of this debate see Baddeley et al., 1997; Della Sala & Gray, 1998). The terms executive function and executive control are used to represent this (Heyder et al., 2004). Even within these constructs, empirical evidence suggests that executive functions “fractionate” even further (Burgess, 2000). Additionally, it is noted that although executive function is linked with the prefrontal cortex, executive function is not exclusive to prefrontal cortical function. Some individuals with prefrontal cortex damage do not display any difficulties with executive function (Shallice & Burgess, 1991), whereas some patients with damage outside of this region do (Anderson, Damasio, Jones, & Tranel, 1991).

There are many definitions and models of executive function and control by the prefrontal cortex in the literature (Wood & Grafman, 2003), with corresponding overlap and differences, but there exists a general agreement of ‘executive function’ as an umbrella construct for the components that provide control or supervision of purposeful goal-directed behaviour and self-regulation (Gioia & Isquith, 2004; Morgan & Lilienfeld, 2000). Further, the prefrontal cortex is theorised as not being critical for the performance of simple and automatic behaviours, such as orientation to sound, but is considered to be vital in its function as a “top-down” processor (Miller & Cohen, 2001).

The most prominent theories of executive functioning have been described as either processing models, representational models, or a hybrid of these, with representation models thought to best fit with an evolutionary view of prefrontal cortex development (Wood & Grafman, 2003). Some of the models, with varying degrees of support, are, the working memory model or central executive (CE:
Baddeley & Della Sala, 1996; Baddeley, 1986; processing model), the attentional control or supervisory attentional system (SAS; Norman & Shallice, 1986; processing model), adaptive coding (Duncan, 2001; processing model) structured event complex model (Wood & Grafman, 2003; representational model), guided activation (Miller & Cohen, 2001; representational model), somatic marker hypothesis (Damasio, 1995, 1996; hybrid model), temporal organisation (Fuster, 1997; hybrid model), and working memory (Goldman-Rakic, 1996; hybrid model).

A model that is often evoked in attempting to explain aggressive dyscontrol is that of the somatic marker hypothesis (Damasio, 1995, 1996), which is a hybrid model, combining both processing and representational elements. The somatic marker hypothesis attributes defects in long-term advantageous decision-making to defects in emotion processing (Bechara & Damasio, In Press; Damasio, 1996). However this model only attempts to account for potential deficits in OFC functioning and is not addressing data relating to the dIPFC (Wood & Grafman, 2003), and is therefore not a comprehensive model of prefrontal or executive function. This is explored again in the General Discussion (Chapter Seven) when attempting to understand the results of the present study.

All of these models described above will not be discussed at length here but for a review see Wood & Grafman (2003). The arguments for a unitary approach versus a component approach will be described next, using examples from early major unitary approach constructs. The CE and SAS are discussed as they influenced the development of the major test battery used in this thesis (the BADS; Wilson, Evans, Emslie, Alderman, & Burgess, 1998).

One of the early prominent cognitive frameworks associated with the study of executive functioning is the multi-component model of working memory of Baddeley’s (1986). Incorporated into this model was a central frontal control structure for regulating cognitive functions; the Central Executive (CE). Baddeley
(1986) later also incorporated the influential *Supervisory Attentional System* (SAS) model, developed by Shallice (1982) and Norman and Shallice (1986). The SAS has a primary objective to regulate attention and control of behaviour, and is described by the authors as a cognitive model for control of behaviour that is necessary for effortful processing (Norman & Shallice, 1986). Both the CE and SAS can be viewed as unitary in design, advocating a single mechanism underlying executive function(s). An additional difficulty with these models apart from their unitary design is that they focus solely on processing and do not account for the purported representational ability of the prefrontal cortex (Wood & Grafman, 2003). However, even with these criticisms the CE and SAS are still viewed as a useful and informative construct for executive control, particularly in the selection and utilisation of executive components.

A source of controversy is whether executive functioning is a unitary or multidimensional construct. This was first reviewed by Teuber (1972) and revisited more recently by Duncan et al. (1997) and Miyake et al. (2000). More specifically, it is the degree to which different executive functions attributed to the prefrontal cortex, or the cognitive processing of the prefrontal cortex, can be considered unitary in that they project from the same anatomical underpinnings (Miyake et al., 2000), or the same high-level cognitive construct (i.e., the CE).

The CE and SAS constructs in original form do contain a unitary flavour and have been criticised for this. However, subsequent work appears to reinforce a certain common basis or unitary notion that typifies executive function (Duncan, Burgess, & Emslie, 1995; Duncan et al., 1997; Miyake et al., 2000), or at least a common processing ability of the prefrontal cortex (Duncan, 2001), even if individual components (and underlying circuitry) appear to be more heavily involved in differing functions. In contrast to arguments to retain a unitary approach, there is evidence for the non-unifying theory, or component theory, of executive function (Baddeley & Della Sala, 1996). A large body of evidence for a component theory stems from the demonstration of dissociation across tasks, for example, an
individual with ED scoring poorly on the Wisconsin Card Sorting Test (WCST) but not the Tower of London (TOL) (Godefroy, 2003). This is parallel to the neuroanatomical evidence, which demonstrates different functions are directed by differing circuits in the brain (Cabeza & Nyberg, 2000; Tekin & Cummings, 2002).

Miyake et al. (2000) reviewed the studies that have consistently demonstrated dissociation between common executive tests across diverse populations, reinforcing the notion that the functions of the prefrontal cortex cannot be unitary and require fractionation. Miyake et al. (2000) suggested that the evidence for abandoning a unitary construct as presented in the studies they reviewed should be viewed with caution, primarily because the authors of the studies reviewed arbitrarily dismissed tests demonstrating covariates under r = .40 (a covariate of 40 is not necessarily low in all opinions). Further, they suggested that even when concurrent validity is low it does not necessarily lead to discarding the concept of a unitary construct, as there could be explanations other than a pure component approach (Miyake et al., 2000). One explanation may be that of the ‘task-impurity’ problem (Burgess, 1997), as any executive task operates by using other cognitive processes (Miyake et al., 2000). This makes it very difficult to sensitively and precisely measure executive functions without tapping into other functions, which are typically served by other brain regions. This would suggest that there are currently no unambiguous or direct measures of executive function (Morgan & Lilienfeld, 2000), as measuring executive functions is fraught with difficulty due to the utilisation of other functions (language, spatial ability etc) to express executive behaviour. It may be that non-executive component functions utilised by executive function tests could be masking any possible unifying construct (Miyake et al., 2000). This is in line with the adaptive coding approach (Duncan, 2001), which states that the prefrontal neurons and networks impinge on almost all tasks.
Related to the task-impurity notion is that the tests themselves are flawed. A purported executive test could be implicating more components of other cognitive functions instead of executive functions. For example the WCST is criticised for relying heavily on a memory component, and for measuring components other than executive functions (Romine et al., 2004) as well as the continual finding that patients with posterior lesions may perform more poorly than patients with focal anterior lesions (Anderson et al., 1991). Even though the WCST is frequently used in examination of offenders and non-offenders it often fails to discern any differences (Bergeron & Valliant, 2001), although other executive function tests do. At best the WCST is considered to be sensitive to, but not exclusive to, executive deficits and prefrontal damage (Poole, Ober, Shenaut, & Vinogradov, 1999).

Despite criticisms of the fractionated approach there is still a move away from the unity view of executive functions and of prefrontal functioning. Current literature encourages a multidimensional approach to executive functioning (Channon, 2004; Miller & Cohen, 2001), even though some of these approaches appear to be heavily rooted in localisation theory and a return to older ideas, and run the risk of creating a frontal homunculus. However, even with the possibility of creating a homunculus, the encouraging of a component approach to account for the lack of sensitivity of individual tests due to ‘task-impurity’ may have benefits.

2.2.2 Executive Function in Practice

The common methods employed by neuropsychologists and those that are used in research are briefly presented next. A neuropsychological understanding of executive function and dysfunction is also presented.
Psychometric assessment is commonly based upon the *deficit measurement paradigm*. Tests are used to yield specific scores relating to a deficit or strength in a particular cognitive ability. One test score alone cannot be used to provide definitive information, and a test score has diagnostic significance only when combined with other test scores, the patient’s interview behaviour, and premorbid information such as academic or vocational accomplishments (Lezak, 1995). The main areas of function tested in a neuropsychological exam are *Memory, Attention and Concentration, Verbal Ability and Fluency, Visuospatial-Constructive Ability, Psychomotor Speed, Premorbid functioning*, and *Executive Functioning*. Other abilities and functions may also be examined according to the referral questions.

As well as this traditional form of neuropsychological testing, neuropsychology is being used more often today to inform knowledge about ‘real world’ difficulties for a client, and tests that are ecologically valid are being developed to assist this (Chaytor & Schmitter-Edgecombe, 2003). Ecological questions focus in part on what a client does rather than what a client can do.

An examiner can gain useful insights about the participant’s memory, attention, confidence, ability, behaviour, and more, by closely observing how the participant goes about the tests. Sometimes the most important information a test yields regards the *way* an individual completes the test, and this information may be particularly valuable when determining whether an individual has difficulty with executive functioning. Even when a test score is poor, indicating a possible deficit, the reasons why that test score is poor are equally important. An individual’s approach to a task will often reveal reasons why they ‘failed’ or ‘passed. This may be of particular importance in tests of executive ability, which struggle to gain ecological validity due to the complex nature of executive function and the somewhat artificial testing environment.
The information gained from an individual’s approach to tests is valuable in determining what behaviours that individual is having trouble with. As Walsh (1987) points out, the person’s failure will not be informative if you don’t know why they failed, due in part to the many ways in which an individual might fail. For example, a failure on a speeded performance task may lead to suspicions of a deficit in visuospatial ability, but when the same task is un-timed and the participant is left to conclude the task they may complete all items in the task successfully, so instead the failure could reflect an issue with psychomotor speed or mental processing speed.

Whilst neuropsychological deficits can rarely be implicated as the direct cause of a violent act, the dysfunction measured may play a role (Filley et al., 2001). Further, the contribution of brain dysfunction to violence may not only stem from structural damage but also from neurochemical or neurophysiologic disturbances not easily detectable by current conventional methods (Price, Daffner, Stowe, & Mesulam, 1990). Hence, neuropsychological examinations of individuals who commit violent acts, whether or not they have demonstrable lesions, adds much to the knowledge regarding brain correlates of behaviour (Filley et al., 2001).

**Case Studies**

Neuropsychology bases many of its assertions about brain-behaviour relations upon specific examination of brain-injured individuals, or individuals with neurological impairment. A case study approach where one patient (or a small group of patients) is thoroughly examined either experimentally or clinically, has contributed considerable valuable information to the neuropsychological literature. The assumption underlying a case study approach is that, given that the person was healthy before the brain damage occurred, the brain damage is related to any impaired function that results. The concept of syndromes grew out of clinical experience of repeatedly seeing clusters of symptoms together in people who had a similar neurological disorder or lesion.
Brain Imaging Techniques

The science and practice of neuropsychology is informed by structural brain imaging techniques including computed tomography (CT), magnetic resonance imaging (MRI), and single photon emission computerised tomography (SPECT); and functional techniques including functional magnetic resonance imaging (fMRI), electroencephalography (EEG), and positron emission tomography (PET). These functional imagery techniques provide information about the brain in action.

Executive Dysfunction

Executive dysfunction is typically examined through neuropsychological assessment and observation of difficulties the individual may be having in their life. Sometimes testing will reveal dysfunction in the absence of structural damage. As related earlier, executive function impairments are not always related to gross damage to the prefrontal cortex and may be associated with ‘invisible’ damage such as a break or diffuse axon shearing in the connections between a region of the prefrontal cortex and other cortices or subcortical areas.

Dysexecutive behaviours are part of a syndrome or syndromes often noted in people who have sustained various types of damage to the prefrontal region. The term ‘dysexecutive’ simply means that some (or all) executive behaviours are deficient or are severely reduced. When trying to define what exactly constitutes executive dysfunction, difficulties arise around the fact that the category is so large. The terms frontal lobe syndrome, executive dysfunction, and the dysexecutive syndrome, all refer to an amorphous, varied group of deficits, resulting from diverse aetiologies, different locations, and variable extents of abnormalities, with no two individuals having exactly the same pattern of difficulties. In addition, deficits in executive function are often, or have traditionally been, interpreted as a psychiatric problem (Elliott, 2003; Stuss &
Benson, 1984). Often executive dysfunction can be mistaken for depression, or depression mistaken for executive dysfunction, as they share many similar characteristics, in particular amotivation, lethargy, and changes in interest and appetites. Further complicating this picture is that executive deficits are commonly noted in psychiatric disorders such as schizophrenia, bipolar depression, and obsessive-compulsive disorder (Elliott, 2003).

The large and heterogeneous nature of the executive functions and dysfunctions can prove difficult for diagnosis and management as well as research. However, there is a general acceptance of how to recognise the central concepts of the dysexecutive syndrome in the literature and amongst clinicians (Benson, 1994; Burgess, 1997; Burgess, Alderman, Evans, Emmslie, & Wilson, 1998; Damasio, 1995; Duffy & Campbell, 1994; Hawkins & Trobst, 2000; Levin, Eisenberg, & Benton, 1991; Miyake et al., 2000; Ready, Stierman, & Paulsen, 2001; Stuss & Benson, 1984; Wilson, 1999). Acquired brain damage and disease are not the only cause of the dysexecutive syndrome. Problems in executive functioning may emerge due to developmental constraints, such as under-myelination, or when one of the structures (cortical or subcortical) that serve executive functioning does not reach potential.

Most of what we know about executive functions comes from the experimental literature, including research that associates localised damage with a particular behaviour via case studies, and functional imaging research examining which regions in the brain are activated during ‘executive’ tasks. When examining literature concerning frontal lobe lesions and resulting dysfunction, it must be realised that brain injury is rarely totally focal (Diaz, 1995; Foster, Hillbrand, & Silverstein, 1993; Stuss & Benson, 1984). A brain injury may result in more than one area of damage, and secondary and tertiary effects of the injury may lead to neighbouring localised areas of damage and diffuse effects. These resulting complications may contribute to the presentation of frontal deficits, and confound research and clinical diagnoses in this area. The literature is replete with
descriptions of prefrontal cortex activity, relating this region to the general executive functions categories of: directing and maintaining higher level attention; correlating internal and external information; generation of plans and intentions; and the inhibition, monitoring, and adapting of behaviour (Hawkins & Trobst, 2000).

Following are brief descriptions of the expected behaviours, memory, and other areas of cognition typically associated with executive dysfunction.

**Behaviours**

The behaviour changes noted in executive functioning include tactlessness, disinhibition, impulsivity, *witwelsucht* (childish joking), grandiose behaviour, apathy, changes in affect, verbal and physical outbursts, increases in irritability, inappropriate sexual behaviour, lability, euphoria, loss of spontaneity, lack of insight, diminished awareness of deficits, lack of self-awareness, lack of awareness of environment, confabulation, slowness in thinking, inertia, and egocentricity (see Stuss & Benson, 1983; 1984, for extended reviews and references).

Blumer and Benson (1975) coined the phrases *pseudodepression* and *pseudopsychopathy* to characterise the two typical broad categories of personality changes that take place after frontal impairment that remain useful today. The first, *pseudodepression*, includes the traits of apathy, indifference, inertia, lack of initiative, blunted emotional response, and reduced verbal output (Giancola, 1995). There is a suggestion from the authors that these types of behaviours are related to damage of the dIPFC (Blumer & Benson, 1975) and the ACC. The second term, *pseudopsychopathy*, includes the traits of irritability, impulsivity, lack of social graces including tact and restraint, increased profane language, promiscuity, and antisocial behaviour. This type of disturbance is thought to be
due to dysfunction in the OFC (Blumer & Benson, 1975; Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994).

Five common overlapping behavioural problems in people who have executive dysfunction have been identified and generally agreed upon in the literature (Hawkins & Trobst, 2000; Lezak, 1995; Mateer, 1999). These five behaviours include problems in volition, including the initiating of behaviour exhibited in decreased spontaneity, decreased productivity, and decreased rate at which behaviour is emitted, as well as decreased, or loss of, initiative. Next, are deficits in making mental or behavioural shifts, which can lead to perseveration or rigidity. Noted also are difficulties in stopping, which can often be observed alongside impulsive behaviour and disinhibition. Another common behavioural deficit is a reduction in self-awareness, which will result in an inability to perceive, and therefore learn from, performance errors. Lastly, a concrete attitude emerges in an inability to take things other than literally.

Social behaviour also depends somewhat on being able to predict the internal state of others. This may be particularly salient when attempting to perceive threat. The concept of Theory of Mind enables reasoning and prediction of other peoples’ state of mind and behaviour (Wood, 2003). The capacity to represent internal states of others comes from the prefrontal lobes (Frith & Frith, 1999) and the amygdala (Adolphs, 1999; Adolphs, Sears, & Priven, 2001; Adolphs, Tranel, & Damasio, 1998). Intact connectivity between the OFC and the amygdala allows for social and emotional appraisal of stimuli, be it threatening or otherwise, and the transfer of information to the dIPFC for action. However, it may be that over-learned familiar responses will bypass the dIPFC and action may occur immediately according to OFC appraisal (Adolphs et al., 2001; Wood, 2003). This may also occur during anaesthetisation of the dIPFC as well, for example while intoxicated. The bypassing of higher executive components, or the ‘top-down’ processor, and the structures that serve them, is in evidence when immediate
threat is perceived, activating the limbic system to flood the body with adrenalin and preparing the individual to fight or to flee.

**Memory**

Patients with frontal lobe damage typically do not seem to have a disorder of the memory system per se, but rather seem not to remember to remember (prospective memory; Baddeley & Della Sala, 1996; Burgess, 2000; Groot, Wilson, Evans, & Watson, 2002; Lezak, 1995; Shimamura, Janowsky, & Squire, 1991) A definition of prospective memory has been provided by Shimamura, Janowsky, and Squire (1991p. 191)

> “Prospective memory involves the ability to access, monitor, and manipulate associations within a temporal/spatial context as well as within a semantic context.”

Patients with executive dysfunction who have impaired memory are often unable to spontaneously search for the information that is to be remembered, and are unable to put in place a strategy to retrieve it. This prospective memory difficulty may interfere with cognitive processes such as acquiring new learning, or making and following plans. This ‘frontal amnesia’ can lead to confabulation in some people, perhaps due to the inability to remember some events combined with a need to ‘fill in the blanks.’

As well as retrieval difficulties, other memory disorders associated with frontal lobe damage involve impaired use of context for storage (Groot et al., 2002). This kind of memory loss impacts on learning because of a disorganisation of the stimuli needed in order to successfully encode and lay down a memory. Short-term memory or working memory problems appear, in part, to be due to the poor strategies in encoding information, the resulting extra processing time needed for consolidation, and the inability of individuals with executive difficulties to withstand interference (Burgess, 2000; Groot et al., 2002). Thus, problems with
memory after frontal lobe damage are often seen because the efficient programming of a ‘memory’ and subsequent efficient retrieval of the information relating to the memory, and the ‘metamemory’ aspects such as holding, monitoring, and manipulation of information are altered, not that the capacity to store a memory is lost.

Individuals with dIPFC damage can have difficulty in tasks that require working memory and problem-solving (Baddeley & Della Sala, 1996; Baddeley et al., 1997; Heyder et al., 2004), and the BADS has been designed to take this into account and sensitively measure the impact of this upon completing the battery (Wilson, Alderman, Burgess, Emslie, & Evans, 1996; Wilson et al., 1998).

**Daily Living Deficits**

Deficits in cognition and behaviour associated with frontal lobe damage tend to show up most clearly in the course of daily living, and may be difficult to examine formally. Deficits may be particularly apparent when observing the person attempting to approach novel situations outside the formal testing environment. Some of the functions pertinent to everyday living include planning, abstract reasoning, and attention.

The ability to ‘plan-ahead’ is often compromised in individuals with executive dysfunction (Wilson, 2000; Wilson, Emslie, Quirk, & Evans, 2001). This is an important ability in being able to live in everyday circumstances. As Ogden (1996) states in *Fractured Minds*:

> “Planning, preparing, and serving a relatively simple meal can become impossible. Shopping for groceries may be successful only if a list is constructed that groups different food types together in the order in which they appear along the aisles of the supermarket. Even then, if the individual does not cross off each item as he puts it in his cart, he might find that he has bought two of the same items and missed others completely.”

(Ogden, 1996, p. 146)
Executive dysfunction creates difficulties in new learning by impinging upon the ability to suppress response tendencies and through increasing impulsiveness. The incapacity to inhibit interfering cognitions and avoid distraction may negatively impact learning and interfere with the performance of tasks requiring recent memory (Burgess, 2000; Burgess, Veitch, de Lacy Costello, & Shallice, 2000; Milner, 1971).

Defective abstract thinking and difficulties in shifting responses are other common consequences of impaired executive functioning, and can result in reduced mental efficiency (Burgess, 2000; Burgess et al., 2000; Stuss & Benson, 1984). After sustaining damage to the prefrontal cortex, people may also find it difficult to structure and organise responses. Structuring a response and preparing it for articulation is vital for human communication and independent living. This ability is thought to occur in the working memory, which is vulnerable to interference, especially in individuals unable to withstand distraction and interference from previous stimuli.

Imaging of the brain has highlighted activation in the dlPFC circuit (for examples see; Mouchia, Petrides, Petre, Worsley, & Dagher, 2001; Rezai et al., 1993) in problem solving using a common neuropsychological test that has been shown to be sensitive to (but not exclusive to) the dlPFC region; the Wisconsin Card Sorting Test (WCST). The WCST was introduced as a test of problem-solving and decision-making in 1948 (Berg, 1948; Grant & Berg, 1948). This test is commonly used to measure the use of external cues to guide behaviour, self-monitoring, and tendency to perseverate, as well as a measure of hypothesis generation and ability to shift response (Romine et al., 2004). However, the WCST is often criticised as being ‘task-impure’ and measuring components other than executive ones and not able to sensitively differentiate between areas of brain damage (Anderson et al., 1991; Poole et al., 1999; Romine et al., 2004).
A test that attempts to demonstrate some of the same qualities as the WCST while accounting for variables that contribute to task-impurity is the Rule Shift Cards Test from the executive function battery, the Behavioural Assessment of the Dysexecutive Syndrome (BADS). The Rule Shift Cards Test assesses inhibition of prepotent responding and perseveration, while removing the memory load. Another BADS subtask hypothesised to measure similar functions as the WCST, along with prospective memory and planning ability, is the Modified Six Elements Test (MSET). Healthy dIPFC functioning has been implicated in being able to complete this task successfully (Burgess et al., 2000).

Impairments in inhibition of prepotent responding, self-regulation, and decision-making are thought to be associated with poor OFC functioning in particular and can be assessed neuropsychologically with the Iowa Gambling Task (Bechara, Damasio, Damasio, & Anderson, 1994), and the different versions of the Go/No-Go Task (Dinn & Harris, 2000; La Pierre et al., 1995; Malloy, Bihrle, Duffy, & Cimino, 1993). The Stroop (Regard, 1981) is also a test considered to assess the mental flexibility and inhibition of responding that the OFC is purportedly heavily involved in.

A common neuropsychological test that is thought to capture aspects of goal orientated development and execution of a plan is the Tower of London (ToL) (Shallice, 1982). Studies involving the ToL suggest it assess abilities that are associated with inhibition (Mitchell & Poston, 2001) and planning, including response selection and evaluation (Baker, Rogers, Owen, & Firth, 1996). Imaging with positron emission tomography while successfully completing a version of the ToL (Baker et al., 1996) demonstrated activity in the ACC region and circuit that reached into the dIPFC. Similarly, support for activation of this region when performing on the TOL is demonstrated via magnetic resonance imaging (Levin, Mendelsohn, Lilly, & Fletcher, 1994). This type of novel ‘action planning’ can also be measured by the Action Program Test from the BADS (Wilson et al., 1996; Wilson et al., 1998). This test requires the participant to plan and mentally
arrange the order of responses to complete the task while not breaking any of the ‘rules.’ In an early version of this test by Klosowska (1976) patients (with localised ‘frontal’ lesions) who had difficulty on this test also reported vocational and daily activity action programming difficulties, suggesting that this BADS subtask has some ecological validity, and may be useful for informing prognoses.

Tests of verbal fluency have been used consistently over the last few decades to assess mental flexibility and ability to spontaneously generate and sort words to particular rules. Tests such as the Controlled Oral Word Fluency Test (COWA) are thought to be sensitive to general prefrontal cortex lesions, rather than associated with specific neuroanatomical structures (Henry & Crawford, 2004). In a recent large meta-analysis examining 31 studies with 1,791 participants, the presence of a prefrontal lesion was more reliably associated with poor performance on tests of verbal fluency than then in examinations with the WCST (Henry & Crawford, 2004). In examining for possible correlations between the profile score of the BADS and COWA using the letters F.A.S no concurrent validity was demonstrated (Norris & Tate, 2000; Wilson et al., 1998). However, the COWA was associated with daily living predictions of overall functioning and interacted with the BADS questionnaire measure of this (the DEX, described later in the method section, 4.1.8, of Study One).

Attention is an area that is often disrupted by frontal trauma. Attention encompasses several different capacities that enable one to be responsive to, and begin to process stimuli. Problems with attention and concentration are among the most common problems suffered by people who have sustained damage to the brain generally (Wilson, 1999), and in particular to the frontal lobes. If attention is compromised the impact on cognitive function will be demonstrated by the individual having trouble staying on task, learning new information, dividing attention between tasks and in shifting attention between stimuli.
Executive dysfunction and cognitive (or ‘intelligence’) dysfunction have traditionally been thought of as distinct. Difficulties in cognition for individuals with executive dysfunction are often not ‘true’ deficits in cognition, but rather difficulties in the performance of the cognitive functions. Executive dysfunction can directly impinge on cognition by impairing the person’s ability to approach, plan, or carry out cognitive tasks, and in defective monitoring of performance. Lezak (1995) describes it in *Neuropsychological Assessment 3rd Ed.* as:

“Questions about executive functions ask how or whether a person goes about doing something (e.g., Will you do it and, if so, how?); questions about cognitive functions are generally phrased in terms of what or how much (e.g., How much do you know? What can you do?)”  (Lezak, 1995, p. 43).

Much of our modern measures of intelligence and theories of intelligence are based on Spearman’s two-factor theory of intelligence (Spearman, 1927). Spearman’s theory of intelligence underscores a general factor, or ‘g,’ and one or more other factors ‘s.’ In Spearman’s theory, when we need to use a lot of mental energy to manipulate higher order concepts we call on ‘g.’ Some more recent authors conceptualise Spearman’s ‘g’ as a construct that unites, orchestrates, and commands all of the executive functions (for example see Duncan et al., 1997). In this vein, ‘g’ may also be seen as the unifying construct underlying executive function, and may be similar to the CE or SAS.

Later work by Cattell and Horn (1963; Horn & Cattell, 1966) based on Spearman’s two-factor analysis and Thurston’s later work (Thurstone, 1938; Thurstone & Thurstone, 1941), which viewed intelligence as multidimensional rather than a unity trait, developed the concepts of fluid and crystallised intelligence. Fluid intelligence is measured by tasks that require deductive and reasoning ability as well as tasks that require an interpretive understanding of a problem. Crystallised intelligence is measured by acculturative tasks, such as
those that require acquired general and vocabulary knowledge from the salient culture. Thus, tests that measure problem solving and manipulation of problems are considered tests of fluid ability, and tests of simple recall or recognition are considered crystallised measures.

Intelligence, if defined as ‘crystallized,’ which can be described as "the extent to which a person has absorbed the content of culture"(Belsky, 1990, p. 125) is generally left untouched after frontal assault. Early reviews on frontal ablation by Hebb (1939) concluded that the frontal lobes do not play a large role in this type of intelligence. Further, it is the crystallized type of intelligence that is measured in standard intelligence testing, creating uncertainty in the use of intelligence measures across individuals with varying backgrounds. For some individuals who have sustained a TBI the curious anomaly of the crystallized intelligence being left ‘untouched’ has been demonstrated, even while scoring poorly on tests of ‘fluid’ intelligence or executive function (Lezak, 1995).

Even though scholars have attempted to titrate intelligence and executive cognitions down into one or two factors, other authors claim this is a capricious assumption based on arbitrary theory (Andrewes, 2002). Andrewes (2002) asserts that the complexity of intelligence and executive functions cannot be unified by one simple measure and instead we must continue to develop the sensitivity of individual tasks that measure the components of executive functioning. However, research continues to be published that asserts that it is ‘g’ that is measured by traditional executive tasks, and that the concept of ‘g’ or fluid-intelligence, best explains the variance measured on these tests (Zook, Davalos, DeLosh, & Davis, 2004).
2.2.3 Limitations in Executive Testing

One of the conundrums clinicians typically encounter is the insensitivity of neuropsychological tests for measuring behavioural and cognitive difficulties. The measurement of executive functioning is considered to be particularly difficult, partly due to the reasons discussed in the sections above, including task-impurity and dissociation between tests.

Neuropsychological assessment following brain injury has considerable value in identifying deficits, but may have little value in measuring subtle residual problems that limit adaptive functioning post-recovery (Satish, Streufert, & Eslinger, 1999). For example, a brain-injured individual may be observed to have many functional difficulties in their daily living, but these are not examinable via traditional neuropsychological testing. These problems, while obvious to people observing the client behaviours, may not be reflected in quantitative test scores, although test behaviour may suggest a problem. To some extent this may be due to tests of executive functioning not incorporating measures of ‘test-taking behaviour.’ Another difficulty arises when trying to specify what it is the tests are actually measuring. Is it in fact the higher level functions they are purported to measure, such as planning, decision making, inhibition, and abstract thinking, or is it the underlying supporting functions, such as perception, visuospatial ability, and language which are utilised by the executive functions? Sound neuropsychological examination that makes use of many tests to examine all major functioning areas can address this question somewhat, but there will still be a margin of error in interpretation of test results.

Further problems in the neuropsychological assessment of executive functions may be the formality and structure provided by testing settings. Such a structured setting may not allow for the assessment of executive deficits seen under stress or when approaching novel tasks, or in daily living. Neuropsychological examinations are typically conducted in a quiet room with no social pressure,
distraction, or interference; the tests are given one at a time, one after the other, and with full instructions (Lezak, 1995). Such conditions are promoted in order to encourage the best performance of an individual, but they may limit the tests’ ecological validity. For example, an individual with executive problems who lacks internal monologue and/or the ability to self-regulate may respond well to externally applied structure in the test setting, thus masking the difficulties they have in ‘real life’ situations when forced to impose their own structure and participate in complex decision making processes. These arguments add to those in the literature calling for a reliable, ecological, and comprehensive battery approach to executive function testing (Chaytor & Schmitter-Edgecombe, 2003; Wilson et al., 1998) in an attempt to reflect the multidimensional aspects of executive function. The Behavioural Assessment of the Dysexecutive Syndrome BADS (Wilson et al., 1996) is an example of a battery developed to address ecological validity. This is the primary measure selected for the study and the rationale for its use is described next.

2.2.4 The Behavioural Assessment of the Dysexecutive Syndrome Battery

Contrasting with traditional executive tests, the BADS is considered to have ecological validity, in that the instrument aims to measure deficits in everyday life (Jelicic, Henquet, Derix, & Jolles, 2001). Individuals with a dysexecutive syndrome are difficult to assess for many reasons (some described above). Although the component skills of executive functioning may be intact, they may be unable to access or use these skills (Burgess & Alderman, 1990; Wilson et al., 1998). As most traditional tests of executive function rely on the individual components, rather than the integration of these skills into real-life tasks, individuals with executive dysfunction may perform adequately, even when observed to be impulsive or distractible (Wilson et al., 1998).

The BADS attempts to combine both unitary and multidimensional approaches to executive testing. Briefly, the BADS is a battery of six tests that result in an
overall profile score categorising the participant into levels of executive functioning. The BADS includes items that are purported as specifically sensitive to prefrontal cortex damage and to those functions traditionally viewed as executive, such as problem solving, planning, and organising behaviour over an extended period of time. The BADS attempts to assess capacities for everyday living that reflect the real life demands that are made when people have to solve problems, set priorities in the face of competing demands, and adapt behaviour to changing situations. The use of the profile score and understanding the scatter of the subtasks can help determine whether an individual has a general impairment of executive functioning or a specific kind of executive disorder. A summary score is derived for each of the six tests and these scores are summed to give an overall profile score for the battery. This can be compared with the normative data from the United Kingdom comprising 216 control subjects, ages 16-87 years; with estimated IQs ranging from 69-129 (further and more specific data on the BADS can be found in the Methods for Study One). The BADS correlates well with standard executive tests and is comparable with other tests in differentiating between neurological and non-brain-damaged participants (Norris & Tate, 2000) (concurrent validity with common tests is detailed in the Methods of Study One).

A major objective of ecologically valid tests is not to determine the brain locus of neurocognitive and functional deficits, but instead to predict behaviours in the open environment (Chaytor & Schmitter-Edgecombe, 2003; Moriyama et al., 2002; Ready et al., 2001; Silver, 2000), and use this information to predict outcome and plan treatment. In order to meet criteria as an ecologically valid test two general aspects of ecological validity are stressed. A measure must demonstrate verisimilitude; in that the data collection method is similar to real life tasks in an open environment. It must also demonstrate veridicality; that is the test results should reflect and predict phenomena in the real world (Chaytor & Schmitter-Edgecombe, 2003; Franzen & Wilhelm, 1996; Ready et al., 2001). The BADS has been developed with the purpose of reflecting these concerns (Burgess et al., 1998; Norris & Tate, 2000; Wilson et al., 1996; Wilson et al., 1998).
majority of participants can complete each of the BADS subtasks in 2-10 minutes. This further lends to the use of the BADS with individuals with ED, who may have difficulty with remaining on task. It also fits with an inmate sample as prison environments can be distracting places, and the nature of the BADS situation allows for regular off-task behaviour between tasks.

The BADS has been reported as useful in detecting executive dysfunction and predicting outcome for dysexecutive individuals with brain injury (Norris & Tate, 2000; Wilson et al., 1996), schizophrenia (Evans, Chua, McKenna, & Wilson, 1997; Ihara, Berrios, & McKenna, 2000, 2003; Krabbendam, de Vugt, Derix, & Jolles, 1999; Simon, Giacomini, Ferrero, & Mohr, 2003), bi-polar disorder (Cavanagh, Van Beck, Muir, & Blackwood, 2002; Jelicic et al., 2001; Surguladze, Keedwell, & Phillips, 2003) alcoholism (Ihara, Berrios, & London, 2000; Moriyama et al., 2002), and drug abuse (Jovanovski & Zakzanis, 2003; Zakzanis & Young, 2001). Further, due to its relatively stable test-retest reliability (Burgess et al., 1998; Jelicic et al., 2001; Wilson et al., 1998) it has also been used in evaluating treatment outcome. An example is a recent case study of a chronic drug user (predominately using drugs from the methampetamine family) who was experiencing chronic executive difficulties and was tested with the BADS prior to entering treatment with an anticholinesterase inhibitor (Donepezil), and again after three months of treatment, and then again three months after donepezil was discontinued. The total score on the BADS increased during the donepezil phase and then decreased again at washout (Jovanovski & Zakzanis, 2003), demonstrating the effectiveness of the BADS as a clinical tool in monitoring treatment.

A rationale for using the BADS apart from its ecological validity, and ability to provide both a profile of executive functioning and differentiate particular difficulties via subtasks, is that of the limited domain of testing in a prison. Access to inmates for testing in the current study was limited to only one session with each inmate for a short amount of time (no more than 2 and a half hours), so the
fact that the BADS had construct and concurrent validity with other commonly-used executive tests (Norris & Tate, 2000) supported the use of the BADS as the primary measure of ED. Of further value is the BADS’ superior role compared with other executive tests in predicting competency in everyday role-functioning (Moriyama et al., 2002; Norris & Tate, 2000). It is expected that the BADS results would inform the development of appropriate rehabilitation strategies for inmates with executive dysfunction.

2.3 Issues Pertinent to the Current Study

2.3.1 The Prison Population in New Zealand

According to the Census of New Zealand Prisons (Rich, 2000) the average sentenced male offender is Māori, aged 20-35 years and is convicted of a violent offence. He has also been in prison before, with 21% (both Māori and non-Māori) of the male prison population having had 6-10 previous convictions, and 10% having had 21-50 previous convictions. Of current inmates 62% have had a conviction before they were 20 years old. Violence is the biggest problem for offenders with 27% of male inmates having had a previous conviction for violence, and 62% of current inmates being incarcerated for a major offence involving violence. Inmates also have a poor formal academic background, with 69.1% leaving school early (before the compulsory minimum school leaving age of 15) or without any qualifications (Rich, 2000).

2.3.2 Cultural Issues

The prison population is not representative of the ethnicity of the general population in New Zealand. According to the 2001 Census, within the general population, 14.7% identify as having Māori ancestry, 80% as European, and 7% as Pacific Islands Peoples. In contrast, in the male prison population 51% of the population identify as Māori, 36% identify as Pakeha (Caucasian New Zealander),
and 10% as Pacific Islands Peoples according to the 1999 Prison Census (Rich, 2000). That the number of Māori in prison is disproportionate to the number of Māori living in New Zealand is a vital statistic that needs to be taken into consideration when working with such a population. This is a concern as most of the tests being used in the assessment procedure have not been normed for Māori or Pacific Islands Peoples. One test used in this study has been adapted and ‘normed’ on small groups of Māori, and one test has been approved for use with Māori.

The TBI figures for Māori in prison (Barnfield & Leathem, 1998) reflect the overall poor health of Māori in the general population. Māori are over-represented in many negative statistics for health and social indicators such as low income, unemployment, crime, illness (diabetes, heart disease, and obesity), alcohol and substance abuse, and injury. Māori also have a higher rate of injury from motor vehicle accidents, the ratio being 1.5:1 Māori: Non-Māori (Pomare et al., 1995), which may contribute to the high rate of TBI in Māori and the resulting consequences of it, such as unemployment. High Māori unemployment and other negative social indices reflect common findings amongst indigenous peoples with a colonisation history, such as the Aborigine in Australia, and the Native American populations in North America.

### 2.4 Summary of Background

The anatomical underpinning of the executive functions have been explored to provide the basis of understanding brain-behaviour relations regarding violence and offending, how they may inform our knowledge about violence, and how they may be measured in research.

Executive functions are the behaviours that enable us to operate ‘as ourselves’ every day, work methodically to perform the everyday chores that enable us to
progress smoothly through the day, and initiate new behaviour as required. Executive functioning also impacts upon the expression of mood and affect, and acts as a ‘gate-keeper’ to the ‘drives’ aspect of behaviour directed from deeper subcortical areas, and socially undesirable behaviours such as aggression and impulsivity. The dysexecutive syndrome is an umbrella term for a varied cluster of behaviours that can make it harder for an individual to comply with societal rules and customs, or go about their daily living with the same ease that an individual with intact executive functioning can, especially when under stress.

It is very difficult to present a definitive set of behaviours in executive and dysexecutive functioning, as the category is so large. An attempt has been made in this chapter to elucidate the key features of behaviour, memory, and other cognitions that are affected by poor executive functioning. The theoretical underpinning of executive function has also been discussed together with whether this can ever be a unitary construct or requires an understanding that comprises many components.

This section also introduced the topic of neuropsychology and how it examines executive functioning. The literature review that follows in Chapter Three largely relies on studies that have used neuropsychological approaches in exploring the connections between the expression of violence, offending, and executive functioning. One of the challenges for researchers and clinicians is to create valid and reliable ecological measures of executive functions, to enable us to form a clearer picture of a client's behaviour outside of the ‘laboratory,’ allowing professionals to provide more realistic and successful support.
The conclusion in the consensus statement of eminent researchers in the field of violence from the Aspen Neurobehavioral Conference stated there is evidence that violence can result from brain dysfunction along with social and evolutionary factors (Filley et al., 2001). Study of the neurobehavioral aspects of violence, particularly frontal lobe dysfunction, altered serotonin metabolism, and the influence of heredity generate promising leads in understanding violence causation (Filley et al., 2001).

The evidence associating brain dysfunction with aggressive dyscontrol is compelling (Hawkins & Trobst, 2000). Many lesion studies have associated frontal and temporal regions with antisocial behaviour (Hoptman, 2003). Numerous studies have focused particularly on the contribution of prefrontal dysfunction to aggression and violent behaviour. The relationship between prefrontal damage and aggression has been clinical lore for some time (Goldstein, 1944; Hawkins & Trobst, 2000; Hoptman, 2003; Krakowski, 1997; Silver, 2000; Silver & Yudofsky, 1987; Silver & Yudofsky, 1994), and as assessment techniques have improved, empirical evidence for this assertion has been
gathered. More specifically, it has been hypothesised that individuals with poor executive functioning are less able to inhibit impulsive behaviours (Blair, 2004; Lau, Pihl, & Peterson, 1995; Pihl & Lemarquand, 1998), monitor internal behaviour and carry out necessary intentions and planning and regulation of behaviour (Hawkins & Trobst, 2000), and these deficits may increase the likelihood of violence in vulnerable individuals.

This chapter presents an overview of the literature on violence, including neuroanatomical, neurochemical, and environmental factors thought to contribute to violent behaviour. The emphasis is on the neuropsychology of violence, particularly in relation to executive dysfunction or prefrontal pathology. A table is included that summarises some of the neuropsychological literature that uses neuropsychological assessments and/or neuro-imaging to examine the relationship of violence and executive functioning (Table 2). This table is restricted to neurobehavioural studies that focus on the relationship of executive function and/or prefrontal pathology to violence or antisocial behaviour. The table is not exhaustive, but does emphasise the consistent links that have been found in the literature between compromised executive/prefrontal functioning and increases in violent behaviour.

In line with the values of Western and many other cultures, in New Zealand acts of violence against individuals are viewed as criminal. In this thesis criminality is explored in the context of an impaired ability to inhibit inappropriate behaviours and poor general processing ability and how these deficits may lead to and maintain a violent criminal lifestyle. Developmental factors that may influence executive functioning and violence are also presented.
3.1 Violence

Violence is a pervasive feature of modern western society but lacks consistent definition and interpretation. The interpretation of violence is context dependent, and is influenced by the ‘type’ of violent act and where it happens, characteristics of the victim and their relationship with the perpetrator, and contemporary socio-political views. Violence is not a unitary construct, either in how it is perceived or measured or its expression. Violence may be physical or verbal, overt or passive. Violence, or more correctly the perception of what constitutes violence, is to some degree a product of the times. Social construction and the current zeitgeist are important in how acts of violence are perceived. Further, acts of violence do not always appear only in the context of angry affect (Scarpa & Raine, 1997). As well as being hostile and ‘on the offensive,’ violence and aggression can also be defensive. It is offensive (but not necessarily instrumental) violence that is expressed in an overt manner that this thesis examines, and particularly which neuropsychological functions (or lack thereof) underpin and mediate this expression.

Definitions of Violence

A clear definition of violence is essential in enabling further clarification of the factors contributing to violent behaviour as different paradigms have varying definitions of violence according to their focus. Definitions of what constitutes violence include behaviour that results in personal injury or destruction of property (Bandura, 1973), and the intentional infliction of some form of harm on others (Baron & Byrne, 2000). In paradigms for measuring violence, acts of aggression are defined and measured in many ways; including frequency, target, mode, intensity, and type (Barratt & Felthous, 2003). Violence in much of the reviewed research has been operationalised as physically attacking another human being, an animal, or property. Other definitions are more simplistic, defining violence as a behaviour intended to cause harm (Hoptman, 2003). Some research
includes only participants who have been convicted of a violent offence in order to ensure target and mode. To reduce ambiguity in what constitutes violence; this thesis defines violence as a physically aggressive act (mode and type) towards a person (target), which has resulted in a criminal conviction. In addition the offenders in this study require conviction at least twice (frequency).

Disagreement exists over whether violence and aggression are different concepts or are measuring the same construct. Some writers define violence as a clear and measurable act, and aggression as an underlying trait or propensity, or for some, an approach to life. In this thesis the terms ‘violence’ and ‘aggression’ are used interchangeably, in line with most of the literature examined, and conceptualised in the context of an individual being unable to utilise appropriate executive functioning to inhibit acts of violence or aggression. That is, aggression is viewed as violence for this study, and understood as an ‘act’ rather than a personality trait.

**Causes of Violence**

The causes of violence are multi-factorial and violence can be viewed as a result of a myriad of factors, with a large biological and environmental interplay (Filley et al., 2001; Jones, 1992; Stoff & Cairns, 1996). Suggested by this view, it is the composite of biology and environment that create and allow expressions of violence. Epidemiological data clarifies the frequency with which children and adolescents resort to physical aggression, with a negative association between increasing age and acts of violence observed (Fonagy, 2003). This could be suggestive of violence being unlearned rather than learned (Fonagy, 2003), and violence in adulthood that is not a result of acquired damage or disease may signal a failure of a normal developmental process.

Although there is some consensus among investigators on the influence of biological and environmental contributions to violence, the models and examinations offered for review cannot be reduced to a single paradigm for
understanding violence. However, the perspective of the current work attempts to highlight executive dysfunction explanations to account for some of the variance in violent and criminal behaviour. There are dynamic relationships between the myriad of contributing factors to violence. Brain-behaviour relations are just one segment and will be explored with an attempt to elucidate the key ingredients in the ‘development’ of violence and the role brain impairments and dysexecutive functioning play in this. Links between crime and executive functioning will also be examined.

3.1.1 A Note on Gender

Most of the studies presented in this review examine violence within male samples. This gender bias is representative of the literature on violence and aggression. The majority of research in the area of offending and/or aggression has concentrated on males, since offending by males is more frequent and serious than offending by females (Farrington, 1987, 1991b, 1994). Statistics in New Zealand point to higher rates for men than women involved in perpetrating violent acts. New Zealand has a population of four million, with the ratio of males to females reported as 97:100 (Statistics New Zealand: Te Tari Tatau, 2001). However, men are over-represented in prison, with 4713 males imprisoned at the time of the 1999 Prison Census compared to 204 females. The majority of men in prison are serving a sentence for a current violent offence (62%). In comparison, 40% of female inmates have a conviction naming violence as a major offence in their current sentence (Rich, 2000). Rates of previous convictions, which are a predictor for recidivism, are also higher for men, with 31% of men having 11 or more convictions compared to 18.5% of women (Rich, 2000).

It is clear in the literature that male violence and female violence are thought of differently, both with regard to expression and underlying motivation. Campbell and Muncer (1994) declare that the way men and women think about violence is fundamentally different. Supported by empirical research, they hypothesise that
men appear to obtain benefits, whether tangible or abstract, from acting violently, whereas women act violently as a consequence of emotional discharge. For women violence is often viewed as a sign of not coping (Campbell & Muncer, 1994). With regard to possible difference in the quantity and quality of violence perpetrated by men and women, it could be that women are equally ‘capable’ of violent acts and have equivalent levels of aggression as men, but due to social (or indeed executive) constraints this propensity is expressed in a different way. It must be noted that the statistic of 40% of convictions for women are violent in nature is considerable, and in fact some research reports no difference in the amount of physical violence inflicted by woman compared to men in heterosexual relationships (Archer, 2000). The violence or aggression generated by women may be expressed more passively, and in a manner that is more ‘acceptable’ to society, and viewed as ‘less violent’.

In social and developmental psychology there is a literature that supports females being more introverted and inward in their expression of aggression, as compared to the extroverted forms favoured by males (Dawson, 1994; Kovacs, Obrosky, & Sherrill, 2003). Assuming this is so, it would also mean there are fewer obvious social and economic consequences regarding female violence because it is more passive. That is, people are less likely to be physically hurt, meaning it is less of an overt problem and does not demand the immediate attention that male violence receives. Because it is seen as less of a problem, less research is carried out, and it almost becomes a ‘given’ that female violence when compared with male violence is a rarity. With most research focusing on discrete acts of violence, often in concert with conviction rates, violence by females is a less urgent issue. Where the literature does examine overt violence expression in women, it tends to focus on hormonal and social causation theories. Included are the pre-menstrual syndrome (PMS), hormonal surges during and after pregnancy, and reactions to abuse such as the ‘battered wife syndrome.’
In New Zealand violent offending amongst men is a significant problem that is costly in both social and economic terms. More men are in prison for violent crimes in New Zealand than women. As the population studied in this thesis is an all-male one, the literature presented in the next section concentrates almost entirely on males. The male gender will be used when discussing concepts of aggression and offending, and when female participants are involved in the studies reviewed this will be indicated.

### 3.2 Neurobehavioural Findings in Violence and Offending

The literature on aggression and approaches to treatment from the 1940s through to the early 1960s focused primarily on social and psychodynamic factors, almost to the complete exclusion of neurobiological factors (Otnow-Lewis et al., 1985). The discovery in the 1950s of anti-psychotic and antidepressant drugs capable of modifying psychiatric conditions that were previously resistant to psychotherapy alone stimulated the study of biological factors underlying behaviour (Otnow-Lewis, 1983). After the discovery of psychoactive agents, early biological research on aggression focused on hereditary, genetic, endocrine, and morphological abnormalities (Kreutz & Rose, 1972; Mednick, Brennan, & Kandel, 1988; Oliver, 1988). In parallel to these trends, behavioural scientists began to view aggressive behaviour as a pathological outcome of detrimental childhood experiences, poor attachment, or a faulty learning process (Moffitt & Henry, 1989; Raine, 1993; Spizberg & Cupach, 2003).

In linking biological theories to the relationship the prefrontal lobes have with the development and expression of violence, Golden et al. (1996) make an interesting point regarding the size of the prefrontal region. They indicate that the prefrontal lobes may be sensitive to biochemical disorders because of their great demand for oxygen and energy as the largest area of the cortex (Golden et al., 1996). The prefrontal cortex is involved in some of the most complex sets of behaviour, and
thus requires a higher level of general arousal to function properly. This factor makes the prefrontal region the area most likely to malfunction, even in normal individuals, when the person is fatigued, unwell, or influenced by neurotoxic substances. Certainly these patterns are magnified many times for the person who is already under-aroused due to existing organic factors.

In this thesis biological and environmental factors are addressed by means of arbitrary separations with brief accounts describing these factors and possible relationships with violent or criminal behaviour and how ED may further explain the relationships. This process provides clarity but belies the complex and holistic manner in which these factors work together to increase propensity for violence. Overall this thesis seeks to demonstrate an integrative approach in understanding the impact of ED on violence, and even while the components have been separated for discussion, the ‘whole’ of executive dysfunction as an explanation for violence is more than the sum of its parts. Studies of violent offenders are discussed in this review. Studies focussing solely on sexually violent offenders have not been reviewed due to the association of this type of offending with compromised temporal brain regions (Raine, 1993; Raine & Buchsbaum, 1996; Raine et al., 1994). Consequently, this thesis did not include any participants with sexual violence convictions. However, some studies reported do include sexual offenders along with violent assault offenders in their samples.

3.2.1 Genetics

A specific ‘crime-gene,’ ‘violence-gene,’ or gene sequence that encourages aggression has not been identified (Karr-Morse & Wiley, 1997). Indeed, given the complexity of crime development, including organic and social factors, and trends in crime statistics, crime type, differences across nations, and changes in law, it seems unlikely that such a specific crime-gene or sequence will ever be discovered. However, this daunting list of obstacles does not stand in the way of flourishing interest in this area. The role of genetics in aggression gains support
from within the field of cytogenetics, traditionally using evidence gained from family, adoption, and twin studies. Additionally, chromosomal work and examinations at the cellular level of the transfer from parent to child attempt to identify which genes influence which neurotransmitters.

Family studies attempt to distinguish the role of the environment in shaping violent or criminal behaviour and the role genetic factors play in predisposing someone to violent or criminal behaviour. Twin and adoption research has traditionally been the investigation method of choice in attempting to disentangle the influence of genes and environment. Common research includes examination of concordance rates on many behavioural variables of genetically identical or monozygotic (MZ) twins and fraternal or dizygotic (DZ) twins. Adoption studies have been used in further attempts to understand the biological impact on criminality and violence. Antisocial behaviour is behaviour that includes a pattern of pervasive disregard for and violation of, the rights of others and can include impulsivity and violence (Hare, 1999; Seguin, 2004). Antisocial difficulties appear to run in families and several adoption studies suggest a genetic component (Bartels et al., 2003; Cadoret, Troughton, & O'Gorman, 1987; Hudziak et al., 2003; Jary & Stewart, 1985; Turner, 1994; van Beijsterveldt, Bartels, Hudziak, & Boomsma, 2003). However opponents of adoption studies point out that those children adopted are often raised in similar environments to that of their birth parents (Turner, 1994). It may also be that by the time some children are adopted, their previous environment and stimuli (including in-utero teratogens such as alcohol, nicotine, abnormal hormone levels, and stress) may have already had a significant impact upon their brain and potential (Perry, 1997).

In an early large and comprehensive criminal twin investigation of over 3,000 pairs of twins born in Denmark between 1881 and 1910, 35% concordance in offending for male MZ twins and 13% for male DZ pairs was revealed (Christiansen, 1977; Cloninger, 1978). While this study did not have enough cases of violent actions and crimes to permit definitive conclusions, it does suggest the
importance of genetically transferred characteristics in increasing propensity for criminality. Later in examining violent crime and violence Raine (1993) averaged the concordance rates across various cohort studies of the genetics of adult crime using twins studies available at the time, revealing 51.5% of MZ twins were concordant for violence compared with 20.6% of DZ twins. However, these studies examined by Raine (1993) varied in terms of country of origin, age, gender, sample size, determination of zygosity, as well as definition of crime and violence. In a later investigation using separate parental ratings of the Child Behaviour Checklist for over a thousand sets of MZ and DZ twins (Bartels et al., 2003), the authors concluded that aggression is a highly heritable behaviour, passed through families both environmentally and genetically, with genetics (inter-rater agreement between separate parental accounts) accounting for the 69% proportion of influence among boys. However, using an inter-rater agreement as an estimate of genetic influence is dubious and underestimates the impact of the environment on the parental ratings. This rather large estimate of variance is yet to be replicated.

Some recent work has examined the role of a functional polymorphism in the enzyme monoamine oxidase A (MAOA), which mobilises the neurotransmitters of norepinephrine, serotonin, and dopamine. In attempting to understand why some maltreated children develop antisocial characteristics whereas others do not, a large cohort of males followed from birth to adulthood had their levels of MAOA checked and measured against various antisocial measures (Caspi et al., 2002). The study concluded that high levels of the MAOA genotype were associated with less antisocial behaviour. Members of the cohort who experienced both maltreatment and low activity of the MAOA genotype comprised only 12% of the sample but they accounted for 44% of the cohorts’ violent offending. This study has implications for the functions of the prefrontal cortex, as the neurotransmitters that MAOA are responsible for modulating, play an important role in prefrontal brain function. However, this study overlooks the contribution of social factors in moderating the effects of abuse, such as undergoing therapy to deal with past
abuse, or having access to at least one significant adult, who listened or helped, and the effect this has on measurable MAOA levels.

The Caspi et al., (2002) study has been replicated by Foley et al. (2004). In an examination of 514 male twins less than 18 years of age the authors found that low expression of the MAOA genotype increased risk for conduct disorder only in the presence of child abuse (Foley et al., 2004). Huang et al. (2004) examining 663 participants with psychiatric disorders and 104 healthy volunteers genotyped for MAOA, demonstrated that low expression of MAOA in males was associated with increased impulsivity when they had been physically abused before 15 years of age. In contrast, similar levels of MAOA in females who had been abused were not associated with changes in behaviour (Huang et al., 2004). More regarding the influence of this genotype on enzymes and neurotransmitters is explored in 3.2.2 Neurotransmitters.

In the search for genetic influences a large number of studies on genetic theories of aggressive behaviour have focussed on the examination of ‘super-males,’ that is, males born with the XYY karyotype. Piacente (1986) points out that XYY is found in 1.5% to 2.0% of the criminal population in North America, whereas only 0.14% of newborns in North America are born with the XYY karyotype (Karr-Morse & Wiley, 1997). However the interpretation of the studies that implicate involvement of XYY in increased levels of aggression is complicated by other factors, such as a high number of the criminal population coming from families of lower socio-economic class in which there is a history of substance abuse and criminality. Turner (1994) reports that although XYY males appear to be over-represented in prisons, their criminality is not particularly aggressive and appears to be more a function of low intelligence and poor school performance. There is also evidence in the literature that XYY males are no more likely to be aggressive in a conflict situation (Turner, 1994). Genetic abnormalities such as XYY are rare and therefore shed little light on the variation in criminal behaviour and acts of violence seen in the general population.
It may be that genes have an indirect influence on aggression by their effects on the peripheral and central nervous system through various means, such as varying the production of hormones and neurotransmitters (Turner, 1994). This could impact on the development of the prefrontal cortex and the executive function pathways. As genes also play a role in determining body size and appearance this could influence future interactions and feature in aggression development (Turner, 1994), with bigger children noted to be more dominant in the playground and often described as ‘bullies’ by their teachers. This may be particularly true in the case of XYY males, who as part of the disorder grow to be very tall.

3.2.2 Neurotransmitters

At the neurochemical level, several neurotransmitters have been implicated and associated with violence (Berman & Coccaro, 1998; Miczek, Fish, de Bold, & de Almeida, 2002; Piacente, 1986; Volavka, 1999), with the monoamine neurotransmitters of serotonin, norepineprine (NE), and dopamine (DA) gaining the most attention. The catecholamines (NE and DA) are associated with general arousal and irritability and working memory (Arnsten, 1998; Robbins, 2000), and can be considered to indirectly influence violent behaviour due to their activating effects on the nervous system (Jones, 1992; Krakowski, 2003) as well as contributing to a general lowering of frustration tolerance. Manipulations in animal studies have demonstrated that an increase in catecholamine compounds leads to an increase in aggressive behaviour (Piacente, 1986; Volavka, 1999). These animal studies have shown the increase in aggressiveness is specific, as the additional circulating catecholamine compounds have not been observed to increase other behaviours, such as grooming. The inhibitory or modulating neurotransmitters of serotonin or gamma-amniobutyric acid (GABA) are also involved in this process (Piacente, 1986) and are discussed next.

The frontal cortex produces and uses more serotonin than any other region of the brain, leading to extensive examinations in the literature of the role of serotonin in
behaviour, and particularly in respect to violence and impulsivity (Berman & Coccaro, 1998; Dolan, Deakin, Roberts, & Anderson, 2002). One of the functions serotonin provides for the anterior cortex is to operate as a regulator of glucose metabolism, providing the energy the brain needs to function. The frontal cortex is very sensitive to reductions in energy input or changes in neurotransmitter levels (Golden et al., 1996), and damage that causes the reduction of serotonin in either output or uptake may manifest as increased irritability and impulsiveness. Further, an intact serotonergic system is also purported to serve an inhibitory function, reducing the likelihood of such behaviours as self-mutilation, suicide, and aggression in response to stressors (Berman & Coccaro, 1998; Coccaro, 1992).

A demonstration of the complexity of the cortical and sub-cortical regions controlling behaviour is that excitotoxic lesions in the prefrontal cortex result in depletions of serotonin in the same way excitotoxic lesions in the anterior cingulate gyrus do, with both types resulting behaviourally in increased impulsivity (Muir, Everitt, & Robbins, 1996). This complexity is one of the difficulties in attempting to understand the aetiology of reduced serotonin and in developing treatment options. Drugs that increase levels or reduce re-uptake of serotonin are often prescribed to people with major depression as a mood stabiliser and/or to diminish anxiety. One of the functions serotonin appears to have in animals is that of inhibiting aggressive behaviour (Piacente, 1986; Krakowski, 2003; Miczek et al., 2002). Validating this assumption are procedures that dramatically reduce serotonin levels in animals such as drug administration, brain lesions, or thiamine-deficient diets, which can induce extreme aggressive behaviour (Miczek et al., 2002; Valzelli, 1984). The link between lowered serotonin levels and increases in aggressiveness has been documented in children as well as adults (Weisfeld, 1994). Serotonin levels can be assessed via analyses of the 5-HT metabolites (of which the major one is 5-Hydroxyindoleacetic acid; 5-HIAA), in the cerebrospinal fluid (CSF), blood, and urine. In particular 5-HIAA has been found to correlate well with actual levels of 5-HT in the frontal cortex (Berman & Coccaro, 1998).
Studies that implicate serotenergic dysfunction and violence are abundant (Pihl & Lemarquand, 1998) (Berman & Coccaro, 1998), and reduced 5-HT has been repeatedly implicated in the pathogenesis of impulsivity, aggressiveness, and suicide (Dolan et al., 2002). However, the relationship is complicated and there is no causal one-to-one relationship between serotonin and aggression (Krakowski, 2003). In early work, Asberg, Traskman, & Thoren (1976) reported significantly lower levels of 5-HIAA in the CSF of depressed patients who had committed violent suicide attempts, than those who had attempted non-violent suicides. In another study that examined 5-HIAA in the CSF, the levels were compared to scores on an aggressiveness questionnaire, with a significant and negative relationship found between the scores on the aggressiveness questionnaire and the levels of 5-HIAA in the CSF (Brown, Goodwin, Ballenger, Goyer, & Major, 1979). That is, the higher the aggression as scored on the questionnaire, the lower was the level of metabolite 5-HIAA. This group of authors proposed an association between lifetime propensity for aggression and chronically lowered CSF 5-HIAA (Brown et al., 1979).

Relationships between serotonergic dysfunction and violence were later replicated (Brown et al., 1982; Linnoila et al., 1983). In a large epidemiological population, analysis of serotonin concentrations in blood were taken from 781 21-year-old individuals (53% male) and measured against reports of violence and offending. Only amongst the men categorised as “violent offenders” were significantly lower levels of the serotonin metabolite 5-HT found when compared to the normative levels found in the general population (Moffitt et al., 1998). A similar effect was seen in non-suicidal but aggressive psychiatric inpatients (Stanley et al., 2000) when compared to non-aggressive psychiatric inpatients. However, in some of these studies there were no comparisons of levels of 5-HT, 5-HIAA, and corresponding independent impulsivity ratings across violent and non-violent offenders or controls, thereby limiting clarity of the connection between depleted 5-HT/5-HIAA and violent crime. Continual low CSF 5-HIAA has also been tracked over time in recidivist arsonists (Virkkunen, De Jong, Bartko, Goodwin,
& Linnoila, 1989), again implicating serotonergic dysfunction in the genesis of impulsivity disorders, as well as aggressive boys with conduct disorder who grow into aggressive adult criminals (Virkkunen et al., 2003).

In studies of men who murdered a domestic partner, Lindberg (1985) reported that these men had lower CSF 5-HIAA than a control group. This also held for men who attempted suicide (Lindberg et al., 1985). Interestingly in this study the 5-HIAA concentrations of men who murdered non-sexual partners were no different from non-violent controls. The authors conclude that violence will occur in the presence of serotonin dysfunction when associated with extreme emotional states (such as jealousy-inflicted rage, fear and frustration). This may explain why, in a later study that attempted to replicate these findings, only those men who had made repeated suicide attempts were reported to have lower levels of 5-HIAA, whereas the isolated single ‘stranger’- murderer experimental group were no different from controls (Lindberg, Belfrage, Bertilsson, Mattila Evenden, & Asberg, 2000).

Increases in impulsivity and violence and its association with measures of reduced 5-HT/5-HIAA have been repeatedly observed in the literature, but there is less agreement of the effect of depleted 5-HT on more integrative executive functions, for example the ability to plan. Park et al. (1994) produced decreases of 5-HT in healthy human volunteers with a drink of a lowering L – Trypotophan (an essential amino acid for the synthesis of serotonin) agent. This drink successfully reduces measures of 5-HIAA. Once 5-HT levels were assessed as low, participants were tested against controls on a range of neuropsychological measures. Although ingesting the drink resulted in poorer visual discrimination and new learning with Paired Associates, no effect was seen on typical tests of frontal function (including the Tower of London). This may be due to the proposed association the dlPFC has with planning. The dlPFC is not implicated in serotonergic dysfunction as heavily as the OFC, seen in the literature in various studies demonstrating impairment on impulsivity tasks (associated with OFC) but
minimal dysfunction on predominantly dlPFC tasks (Krakowski, 2003; Park et al., 1994), in samples with reduced 5-HIAA concentrations. When required to shift-sets of attention (not defined in the Park study as a prefrontal function or executive function test), there was a significant difficulty demonstrated by those who drank the L-Tryptophan-lowering blend. The ability to shift set can be marshalled into two main sets: the ability to inhibit a previously learned (or rewarded) stimulus, and the ability to discriminate the newly rewarding object. Animal studies have shown that specific OFC lesions in particular impair this ability (Robbins, 2000). Given the robust links the orbitofrontal cortex has with the limbic areas of the anterior cingulate and the amygdala, it makes sense that reward paired learning is compromised in OFC dysfunction.

Using the Iowa Gambling Task (Bechara, Damasio, Tranel, & Anderson, 1998) Rogers et al. (1999) demonstrated that the orbitofrontal region contributes to difficulty in decision making and learning while shifting set when under stress from neurodegenerative disease, damage, or in a laboratory reduction of L-trypotphan. In a later study, these results differed from the effect of methylphenidate, known to affect dlPFC functioning, which did not result in impairments on the gambling task (Robbins, 2000), demonstrating general divisions between functions subserved by the OFC and the dlPFC. A study inducing dietary depletion of tryptophan and utilising the Go/No-Go task was conducted longitudinally with 38 adolescent males (who had previously been neuropsychologically tested) who were categorised as aggressive and non-aggressive according to multiple teacher rating scales (LeMarquand et al., 1998; Seguin et al., 1995). They found that aggressive subjects made more commission errors. This result was significantly related to lower overall executive function over and above IQ and memory deficits from the previous neuropsychological testing.

In an attempt to clarify the role of 5-HT in impulsivity and aggression Dolan et al. (2002) examined 52 personality disordered offenders (predominately antisocial
type) and 24 controls with an assessment of 5-HT function, MRI scan, and neuropsychological testing of executive and memory functions as well as IQ. They suggest that impulsivity is a function of poor 5-HT function, impaired executive function and poor IQ. In contrast, aggression was related to impaired IQ and executive function only, with no association demonstrated with levels of serotonin or impulsivity. Thus, serotonergic dysfunction may increase impulsivity, but this does not necessarily cause aggression, or even covary with aggression. The generation of violence may involve the higher-order functions served by the dlPFC and sub-cortical underpinnings, which are not as vulnerable to fluctuations in serotonin.

Related to the relatively direct studies of serotonin and behaviour, are those that examine monoamine oxidase (MAO) activity. It is thought that MAO markers may reflect some aspect of the serotonergic system, such as size or functional capacity (Oreland & Shaskan, 1983; Stalenheim, 2004), and that lower levels of MAO turnover reflect serotonergic dysfunction (Daderman & Lidberg, 2002). Some authors have suggested that decreased MAO activity may serve as a marker for psychopathy (Stalenheim, 2004), and it is frequently associated with individuals with antisocial traits, including marked impulsivity (Daderman & Lidberg, 2002; Caspi, 2002 #310; Jaffee, Capsi, Moffitt, & Taylor, 2004). It has been used to successfully identify those offenders who are likely to offend again (Daderman & Lidberg, 2002). These markers may be able to be assessed even in adolescence (Caspi et al., 2002; Jaffee et al., 2004; Virkkunen et al., 2003).

Early work demonstrates that decreased MAO activity has been related to impulsiveness, sensation seeking and aggressiveness (Schalling, Asberg, Edman, & Oreland, 1987; von Knorring, Oreland, & Winblad, 1984; Zuckerman, 1984). Current work appears to replicate these findings and add further information, finding that MAO levels may remain as a constant predictor of future violent behaviour (Garpenstrand et al., 2002; Stalenheim, 2004). Hallman et al. (1999) reported an association between low MAO activity and continued violent crime in
a group of male forensic psychiatric patients. Later Stalenheim (2004) was able to replicate this by following a cohort of psychopathic criminals and normal controls 6-8 years after taking MAO measures, finding that reduced MAO activity significantly predicted continued criminal recidivism among the violent criminals over this time period.

Recent research into the impact of anticholinesterase inhibitors on the cholinergic system in improving levels of cognition may augment neurochemical and neuroanatomical explanations of executive functions and the structures serving them. Cholinergic stimulation has produced beneficial effects for overall cognitive performance in normals, particularly with working memory (Furey, Pietrini, Alexander, Schapiro, & Horwitz, 2000; Furey, Pietrini, & Haxby, 2000), as well as those with bipolar disorder (Sokolski & DeMet, 2000) and attention-deficit/hyperactivity disorder (Popper, 2000). Of particular interest is the finding by Jovanovski and Zakzanis (2003) of improvements in executive functioning, as measured by the BADS, upon the administration of the anticholinesterase inhibitor donepezil. Donepezil is typically used in early frontotemporal dementias with varying positive effect in temporarily relieving cognitive decline, with most effect demonstrated in improving the executive functions of organisation, planning, and multi-tasking (Jovanovski & Zakzanis, 2003). Treatment with donepezil has also proven useful in a single-case study design with a patient who had chronic schizophrenia and was suffering the effects of an earlier frontal lobotomy, including severe ED. Treatment with donepezil had a significant impact of relieving the negative effects of schizophrenia, and in particular increased his communication ability, reduced his environmental dependency, and resulted in a reduction in the amount of aggressive and behavioural incidents (Mazeh, Mirecki, Paleacu, & Barak, 2003).

In summary, it is apparent from the literature available that there is a correlation between impulsivity, aggressive behaviour and poor serotonin function, but it is not indicative of causality. However early and lifelong expression of aggression
and/or impulsivity appear to be associated with chronic serotonergic dysfunction (Soderstrom, Blennow, Sjodin, & Forsman, 2003). Repetitive acts of violence have been associated with continual poor 5-HT function, and this may be mediated by the violent acts measured in these studies being impulsive in nature, as it is impulsivity that has been associated most consistently with serotonergic dysfunction (Dolan et al., 2002; Krakowski, 2003), not violence per se. It is difficult to draw out from human research whether low serotonin induces aggressive behaviour through increasing impulsivity, or if low serotonin is a side effect of impulsive and aggressive behaviour. Further, it is problematic to interpret these results, as certain co-morbid factors are often not known, such as personality types, past history of mental illness, drug and alcohol use, and medical background. Research in the area that is easier to interpret is the experimental animal research where behaviours have been controlled through purposeful manipulations of serotonin. These results lend more confidence to a causal link between serotonin and aggressive behaviour.

### 3.2.3 Hormones

In attempting to clarify the role testosterone has in men, Gladue (1991) reported higher levels of overt aggression in both heterosexual and homosexual men when compared with women, and relates this to sex type rather than gender or sexuality differences; namely levels of testosterone. However, there have been few systematic and reliable investigations of hormones and aggression in humans (Ramirez, 2003). Interest in a possible link between violence and hormones stems from animal research indicating that testosterone can increase aggressive behaviour in some species (Blanchard & Blanchard, 2003). In the case of linking male hormone activity to acts of violence the literature is conflicting. An early review by Piacente (1986) found that research with inmates has failed to show a replicable correlation between violence and testosterone level. In investigations that do demonstrate an association, it is difficult to demonstrate a causal direction in which comes first; the violence or the elevated levels of testosterone? All
literature in this field is tempered by other research that shows androgen levels increase in competitive and combative activity in both humans and animals (for examples, see Piacente, 1986), as well as being linked to levels of social status (Ramirez, 2003). However there are some reports in the literature that stand out and are worth considering when examining links between aggressive acts and testosterone.

In particular it appears that testosterone may be linked to instrumental forms of violence (Mueller, 1998), and this may be mediated by the affect testosterone has on motivational processes. A reduction in punishment sensitivity and enhanced reward sensitivity has been demonstrated in animal research with the administration of testosterone (Boissy & Bouissou, 1994). Increases in testosterone levels have also been associated with lowered anxiety levels (Aikey, Nyby, Anmuth, & James, 2002). It is these characteristics (low anxiety, low punishment sensitivity, immediate reward sensitivity in the face of punishment) that are among the core underpinnings for the aetiology and maintenance of psychopathy, or antisocial personality disorder (Hare, 1984; Stalenheim, 2004; van Honk et al., 2004).

The association between increased testosterone and poor decision-making has recently been replicated with humans (van Honk et al., 2004). In a double-blind placebo controlled study of 12 healthy young women (20-25 years of age) using the gambling task, van Honk et al., (2004) demonstrated that one dose of testosterone was enough to significantly alter the participants’ decision making towards reward dependence, even in the face of disadvantageous odds and punishment. The study relied exclusively on women as the authors wanted to replicate the animal findings using similar paradigms and demonstrate as strictly as possible the effects of extra circulating testosterone. The study concentrated on women, as the effects of a single application of testosterone in men are unknown. Testosterone influencing antisocial motivational underpinnings is also implicated in threat perception. Using an emotional Stroop Task (pictures of facial affect)
van Honk et al., (1999) demonstrated that both male and female participants with high testosterone oriented more vigilantly to threatening faces. The authors suggested this is demonstrative of a readiness for aggression in a dominance encounter (van Honk et al., 2004; van Honk et al., 1999). The next step in understanding the impact of testosterone in decision making and its relationship to aggressive dyscontrol, would be to test across groups of offenders and matched community controls. Additionally, research with a group of individuals diagnosed with antisocial personality disorder may be useful.

A meta-analysis of 45 independent studies that examined the effects of testosterone in humans demonstrated a weak but positive correlation between testosterone and elevated levels of aggression (Book, Starzyk, & Quinsey, 2001). This review supported earlier work by Archer (1991) who also demonstrated a persistent but weak link. However, recently Archer, Graham-Kevan, and Davies (2004) in a re-analysis of the Book (2001) article, criticise the methodology of the review. They state that upon closer examination, the literature reported by Book and colleagues is too disordered across varying methods of collection and types of samples to make any clear positive associations between aggression and high levels of testosterone. However, the one association that does appear to remain constant when more rigorous methodological design is applied is that of offender category (Archer et al., 2004), namely, violent offenders are more likely than non-violent offenders to have raised testosterone levels.

In an analysis of studies relating to hormones and aggression in children and adolescents Ramirez (2003) concludes that there is an indirect relationship between testosterone and aggression. He presents evidence associating consistent relationships between aggression and testosterone; in initiating and mediating aggressive responding, and in raised testosterone levels as a consequence of perpetrating aggression. In other investigations with juveniles Chance, Brown, Dabbs and Casey (2000) assessed testosterone levels and behavioural measures in 45 boys with disruptive behaviours and 20 from a control group. They found that
high levels of testosterone were associated with withdrawal and aggression, especially amongst the older boys (nine to eleven years). Research indicates that repetitive violent acts that begin early in life are associated with slight, but consistently elevated levels of testosterone (Volavka, 1995; Ramirez, 2003). But once again, it is not clear whether it is the violent act causing the elevation of testosterone levels, or the reverse.

In an early offending study, Kreutz and Rose (1972) examined plasma testosterone levels of violent and non-violent inmates, with results demonstrating no significant measurable differences in levels. However, in a prison environment other factors may be influential in dictating levels of testosterone, including social success, limits on freedom and the stresses these place on an individual, as well as the environment being an all male and highly competitive one, consequently elevating testosterone levels across all inmates.

In a later inmate study, Dabbs et al., (1987) examined saliva testosterone in adolescent and young adult offenders and demonstrated a link between raised levels of testosterone in the young men who were consistently in trouble with prison authorities, involved in more fights, and who had committed more violent crimes. A larger study replicated this effect in adults when testosterone, criminal offence, and prison behaviour were examined in 692 adult male inmates (Dabbs, Carr, Frady, & Riad, 1995). A positive association was found between high levels of testosterone and those inmates that had committed violent crimes and who consistently violated prison rules, including fighting with other inmates. More recently, Dabbs et al. (2001) found in an examination of 230 male inmates, that a connection between testosterone and violence only existed in those men convicted of homicide when they knew the person they had killed, and that in most cases the killing was ruthlessly planned. The association did not hold for inmates convicted of robbery or sexual violence (Dabbs et al., 2001).
Another section of the hormone literature relevant to this thesis is the work examining the impact of excess hormone levels in-utero. Excessive levels of testosterone, measured in-utero, have been linked to impediment of cerebral development by some authors, who claim it may lead to latent behavioural, cognitive and executive difficulties. Geschwind and Behan (1982) make associations between increased in-utero testosterone, and dyslexia and other learning disabilities. Other researchers have found higher testosterone levels in learning-disabled children (Kirkpatrick, Campbell, Wharry, & Robinson, 1993) or children with limited cognitive ability (Chance et al., 2000), than children without these problems. Further, increased aggressiveness or delinquency is often associated with children struggling with learning difficulties (Moffitt & Henry, 1989; Otnow-Lewis, 1983; Otnow-Lewis et al., 1985; Weisfeld, 1994).

Many studies examining male violence have noted that 18-35 is the peak age range for perpetration of violence (Archer et al., 2004), and the age at which men are likely to be the victims of violence (Daly & Wilson, 1994). Daly and Wilson (1994) call this increased risk of harm to oneself and to others in young men the “Young Male Syndrome,” with the suggestion that puberty in males and the hormone surge that accompanies it signals an increase in dominance-promoting behaviour, aggression, and inter-male competition (Daly & Wilson, 1994). Moffitt (1993) has a similar construct, and terms this time of increased offending in young people (mainly men) “Adolescence-Limited Antisocial Behaviour”.

In contrast to examination of testosterone in aggression, there has been less examination of the effect of oestrogen in human aggression, particularly in men. In a large examination of a Finnish male population self-selected through newspaper advertisements, Eriksson et al. (2003) demonstrated an association between low levels of oestrogen and ‘lesser forms’ of aggression, such as verbal aggression and anger, as well as inflicting minor injury on another person, whereas testosterone was only associated with inflicting injury. Another interesting finding was that high levels of oestrogen in men were associated
reports of positive experiences in verbal negotiation with domestic partners as well as scoring highly on empathy measures.

In summary, although a conclusion cannot be reached on causality between hormones and aggression, there have been many studies over the last 20 years that make an association between levels of testosterone and levels of aggression and violence, as well as good or bad decision-making. Further, early indications of high testosterone in individuals appear associated with repetitive aggressive dyscontrol (Ramirez, 2003; Stalenheim, 2004). New research examining oestrogen in men along with anger and the ability to emotionally negotiate, provide yet more material for debate regarding the roles of the sex hormones in human behaviour. Further, the influence of androgens during critical periods of foetal development may create neurological vulnerabilities that will later influence cognitive and behavioural abilities.

3.2.4 Animal Lesion Research

Much of our knowledge about the neurological basis of violent behaviour comes from animal experiments. With animals, the stimulation or ablation of specific structures can affect aggressive behaviour. Comprehensive ablations of the prefrontal cortex generally result in the impoverishment of emotional life and the social isolation of the animal Fuster (1980; 1997). Certain prefrontal lesions involving the medial or basilar frontal cortex induce behavioural changes suggesting disinhibition of aggression and hunger drives (Fuster, 1980), causing hunger to be almost continuous and provoking an aggressive response to any situation perceived as even remotely threatening.

Fuster (1980; 1997) concluded in her seminal review of the literature that lesions of the frontal dorsolateral cortex, or large lobectomies that spare the posterior orbitofrontal area, increase the aggressivity of monkeys. She indicated that the frontal dorsolateral lesioned monkey attacks other monkeys, often without
apparent motive or the benefit of a warning, as if impelled by an uncontrolled hostility. This increase in aggression is accompanied by the blunting of emotional expression and communication. It is posited by Fuster that the monkey’s aggressive behaviour is not guided by the customary regulation of the limbic system by the lesioned dIPFC (Fuster, 1980; 1997). In contrast, OFC lesions in monkeys lead to a decrease in aggression and a tendency to avoid threatening situations. These early findings are somewhat at odds with later human findings and provide the crux of a dynamic argument taking place in the literature regarding which prefrontal structures (OFC or dIPFC) most inform explanations of antisocial behaviour.

Fuster (1980; 1997) hypothesises that increases in aggression accompanying changes in dorsolateral lesioned monkeys may largely be the result of cognitive impairment, but as the research is dealing with non-human primates this opinion is difficult to substantiate. Nevertheless, early studies into dIPFC ablations in both humans and other animals tend to support this reasoning (Fuster, 1980; Miller, 1976; Milner, 1964). Brody and Rosovold (1952) interpret the abnormal social behaviour of monkeys with frontal dorsolateral lesions as the result of incapacity to use previously learned patterns of avoidance. Miller (1976) has a similar interpretation in commenting that monkeys with frontal dorsolateral lesions may be unable to sort out sensory information and cannot utilise the response tendency to flee.

In the late 1920s, Bard (1929) removed the entire cortex from cats and noticed that they responded to the slightest touch with aggressive responses that were not congruent with the situation. The responses were extremely severe and were also non-specific in their direction. Bard referred to this two-pronged reactivity as “sham rage.” Based on these observations and later electrical stimulation of deep-brain structures by other researchers, the areas of the hypothalamus and the amygdala were identified as being responsible for initiating this impulsive rage. The result of this study, based on the removal of the cortex, indicates its role as
the mediator of environmental stimuli, and in regulating messages from limbic structures.

When attempting to understand brain lesion research, it is important to recognise that even if a lesion is precise and clean, the putative functions of the prefrontal cortex are complex and involve a functional network extending widely over the various areas of the brain (Darling et al., 1998). For example, an individual or animal may become amotivated after a prefrontal lesion because of the break in the connections with the limbic system, not because of damage to a specific prefrontal region. Therefore it is often more useful to interpret lesion studies in terms of function rather than localisation. A description of deficits in executive functioning, rather than the location of damage to the prefrontal cortex, may have more utility. The implication that the connections between the prefrontal cortex and limbic structures impact powerfully on expression and perception of behaviour is again evidence for the powerful ‘top-down’ role of the executive functions (Giancola, 1995).

3.2.5 Human Lesion Research

It is important to note that no pure cause and effect relationship between damage to the prefrontal cortex (and its associated regions and sub-serving circuits) and increased aggression has yet been identified, but the literature is replete with examples of relationships between the two (Filley et al., 2001; Hawkins & Trobst, 2000). The literature based on human lesion studies does not yield the same definitive localisation and differentiation results as the animal literature, given that it is mostly based on accidental lesions and not purposeful experimental lesions and ablations. Thus it is more difficult to obtain clean results that can be relied upon for research purposes. The main problem with lesion studies in attempting to understand the different functions that different areas of the brain take part in is that damage to the anterior regions is rarely confined to the frontal cortex, let alone the prefrontal cortex (Darling et al., 1998).
Clinical populations that lend themselves to research in prefrontal cortex dysfunction include samples with: head injury, stroke, tumours, and surgery, including psychosurgery. Traumatic brain injury (TBI) often results in significant frontal injury, but also often produces diffuse pathology with localised damage to other areas. Other common areas of injury in TBI are the temporal lobes and the brain stem (Diaz, 1995). Penetrating TBI is somewhat more precise in its localising effects, particularly those produced by high velocity missiles or shrapnel (Stuss & Benson, 1984). Nevertheless, even in precise injuries there are complicating factors such as bleeding and swelling, which can cause secondary and tertiary effects.

Secondary and tertiary effects often result after a stroke as well. Strokes of the anterior cerebral artery (ACA) may result in clearly localised medial and inferior frontal pathology, but due to the rarity of ACA strokes, their value for localisation research is limited. Subarachnoid haemorrhagic strokes can result in ‘frontal amnesia’ and other executive deficits. Strokes of the middle cerebral artery (MCA) also affect the frontal area, specifically the lateral and inferior surfaces. However, the MCA also supplies large proportions of the rest of the cortex, particularly parts of the temporal and parietal lobes, thus making it difficult to distinguish the exact function of any frontal damage, although imaging may provide some clarity in defining areas of damage.

Frontal tumours are another potential source of information but provide a problem for precise diagnostic and prognostic research as they frequently go unrecognised until they become large and/or bilaterally situated (Hécaen & Albert, 1978). There will also be problems if the tumour is an astrocytoma, as these are infiltrative. Various functional problems may also occur due to pressure of the tumour on other brain structures, making it difficult to draw conclusions regarding localised structures involved in executive functioning.
Neurosurgery may result in disorders that can be localised to specific brain regions. Most experimental data in this area comes from surgical intervention carried out to reduce epileptic seizures or, historically, psychiatric disease. Such material, however, is subject to the criticism that the psychological and neuropsychological functions measured after surgeries are contaminated by premorbid pathology, i.e., the reason for the surgery. At present, while acknowledging caution is merited, and the need for appropriate control groups, research based on prefrontal surgery provides some useful localising information.

Milner (1964), in data derived from her pivotal work with a series of patients undergoing brain operations of varying loci and extent for relief of frontal seizures at the Montreal Neurological Institute, found that it is possible to demonstrate a deficit after frontal lobectomy in situations that require a constant shifting of response to meet changing environmental demands. Under such conditions, patients with dlPFC lesions seem unable to suppress their ongoing response tendencies to rid themselves of perseverative interference from previous sensory events. In addition, after a prefrontal lobectomy of the hemisphere dominant for speech, a deficit is seen in the production of spontaneous speech, which is measured as a reduction of word fluency. A reduction in word fluency is a useful measure of executive dysfunction, often demonstrating a loss in the ability to initiate verbal activity, and effectively use strategies that would promote fluency (Henry & Crawford, 2004).

The prefrontal cortex is important for social learning and anticipation of the consequences of behaviour. Certain prefrontal lesions may disrupt the mechanism responsible for understanding consequences and result in impulsive violence, with the individual being responsive to trivial stimuli (Krakowski, 1997). Prefrontal lesions may result in more persistent violence because of the difficulties in using environmental feedback to regulate actions. On the other hand, prefrontal lesions may also result in what is almost the complete opposite: apathy. Apathy and aspontaneity are most likely to be observed following bilateral lesions to the
medial areas of the prefrontal lobe, including the anterior cingulate cortex, which has been noted to play a role in alexithymia; which refers to impairments in the ability to access and express one’s emotions (Berthoz, Artiges et al., 2002).

As described earlier in section 2.1.3, when examining the neuroanatomical underpinnings of mood and motivation, lesions or removal of sections of the amygdala can result in remarkable changes in the expression or inhibition of mood and motivation (Blair, 2003; Hamann et al., 2002; Isenberg et al., 1999; Kringelbach et al., 2003; Phillips et al., 2003; Veit et al., 2002; Winston et al., 2003). The resultant reduction in aggression after lesions or removal of the amygdala has been referred to as the ‘taming effect’ (Lee et al., 1998). The ‘taming effect’ (Lee et al., 1998) was used purposefully first by Narabayashi and colleagues (Narabayashi, 1976; Narabayashi et al., 1963) using stereotaxic ablations of the amygdala in epilepsy and later uncontrollable aggressive behaviour in children (Narabayashi et al., 1963). Narabayashi and colleagues followed these cases throughout the child’s lifespan, and for a large number of the children noted that aggressive behaviour was not seen again (Narabayashi, 1976, 1980).

The success of these cases presented by Narabayashi resulted in others using stereotaxic amygdalotomy to alleviate aggression in psychiatric illness for example (Vaernet & Madsen, 1970) with varying success. Work by Narabayashi and colleagues is similar to that of Ramamurthi (1988) who performed 481 bilateral amygdalotomies for aggression in child patients of varying aetiologies who were untreated with more conservative methods, finding a follow-up success rate three years later of 60%. However, many of the improvements reported were in the small to moderate range, and developmental explanations may shed light on the cessation of aggression in children as they mature. The relatively large number of children with no significant change in aggression post-surgery is of concern considering the invasive and extreme nature of amygdalotomies as a solution for aggression.
More recently, other authors have commented that amygdalotomy as a treatment for behavioural disorders related to emotion and aggression is unsuccessful, especially when balanced against side-effects from the surgery. This result is possibly related to the varying aetiologies of the disorders and the methods of treatment used (Aggleton, 1992; Lee et al., 1998). Further, it may reflect the role of the amygdala in that the amygdala is recruited as a filter for affective states, but it is not responsible for them (Anderson & Phelps, 2002; Fellous, 1999). Both the amygdala and the orbitofrontal cortex (OFC) have been implicated in the modulation of the circuitry that mediates reactive aggression (Blair, 2004), although their roles differ. Explanations implicating the OFC-amygdala circuit alone are not comprehensive enough to explain all types of violence. Those that are not impulsive, reactive, or fear-mediated are not accounted for by this literature. Additionally it may be that the top-down processing of the dlPFC also contributes to what action is taken after monitoring information from the OFC-amygdala circuit.

As discussed in the background to this work, Blumer and Benson (1975) suggested that the dorsolateral prefrontal cortex was responsible for behaviours labelled pseudodepression and the orbitofrontal cortex for behaviours labelled pseudopsychopathy. Giancola (1995) argues that this view does not receive consistent support, and through his examination of the human research has posited that it is dorsolateral damage that “may lead to aggressive behavior via a deterioration of the executive cognitive functions” (Giancola, 1995, p. 445). In his view, OFC damage is thought to increase vulnerability to aggression in patients who already have a problem with dlPFC functioning and deficits in psychomotor and social processing, by disrupting emotion regulation (Giancola, 1995).

The conclusions drawn by Giancola (1995) and earlier by Milner (1964) correspond with the ones drawn by authors cited in the animal literature (for example Fuster, 1980), and later human works, (e.g., Blair, 2004; Giancola & Zeichner, 1997; Grafman et al., 1996; Seguin, 2004). Even accounts that favour
an OFC explanation for aggressivity (for example, Blair, 2004) still attempt to account for the influence of the dIPFC. It is considered that the dIPFC underlies a higher-order executive functioning process that orchestrates final observable behaviour. In particular, a compromised dIPFC may lower frustration thresholds, reduce prediction, decrease inhibition, and decrease social processing (Hoaken, Shaughnessy, & Pihl, 2003). This in turn would reduce the likelihood of an aggressive impulse or response from being inhibited and increasing the likelihood of risky or poor decision-making.

Many authors do still support the original terms (pseudodepression and pseudopsychopathy) suggested by Blumer and Benson (Blair & Cipolotti, 2000; Convit et al., 1996; Damasio, Tranel, & Damasio, 1990; Damasio, 1995, 1997), and have since extended them. Damasio, Tranel and Damasio (1990) for example introduced the term ‘acquired sociopathy’ after their case study of E.V.R (see Table 2). Prior to surgery for an orbitofrontal meningioma E.V.R was successful socially and professionally, but post-recovery he rapidly deteriorated into a state where he was unable to sustain relationships or work commitments due to impulsivity and reduced capacity for socially acceptable interactions, along with an inability to plan. The authors of this case attribute the resultant sociopathic changes to damage in the OFC. This is similar to the finding of increased violent behaviour and aggressive attitudes found in Vietnam War veterans with OFC damage, compared to controls and a group with lesions in other sites (Grafman et al., 1996).

Similar work demonstrating acquired sociopathy and the development of psychopathy is that of Anderson et al., (1999) who present case studies of two adults who sustained prefrontal cortex lesions before 16 months of age. In testing with the WAIS-R, RVALT, WRAT-R, COWAT, WCST, TOH and JOL (see table 2 for explanations of acronyms) the participants demonstrated normal performances on measures of general cognitive ability but had significant difficulty with the executive tasks, failing to demonstrate normal learning or rules
and use of feedback. When reasoning and decision-making was examined further with the Gambling Task they persisted in making risky decisions that provided high immediate rewards, but higher long-term losses. Congruently, skin conductance measures demonstrated minimal anticipatory anxiety when making risky decisions. Additionally, when given tests of moral reasoning their reasoning was estimated to be similar to that of a 10 year old. The authors compared these cases with patients who acquired similar prefrontal damage as adults and noted similar neuropsychological dysfunction, but the participants with adult-onset damage were able to reiterate rules and ‘facts’ of social knowledge even though they could not utilise them. In contrast, the adults tested who had sustained damage as infants, did not display any consideration of social, emotional and moral decisions (Anderson et al., 1999).

In attempting to account for the differences between the groups with similar prefrontal lesions, but who had sustained them in different developmental periods, Anderson et al. (1999) suggested that early prefrontal damage has resulted in a syndrome resembling psychopathy, and the adult-onset damaged participants have acquired sociopathy. A major difference for the consequences this may have is that the second group (adult-sustained brain damage) may be able to benefit from the use of compensatory strategies in order to make beneficial decisions for themselves and others, as the basic factual social knowledge has been internalised. However, the participants who sustained brain-damage as infants, thereby compromising the potential to acquire morals, appear to have been unable to internalise any social mores and so are more at risk and contain more risk for society. Indeed the behavioural and historical details provided noted repetitive aggressive and risky behaviour by both these participants (Anderson et al., 1999).

An attempt to relate Anderson et al.’s (1999) work to particular divisions in the prefrontal cortex is not possible as clear imaging of lesion information is not available for every patient, but the most consistently involved regions appear to be the OFC, although the lateral portions of the prefrontal cortex were lesioned to a
lesser extent. The researchers suggest this implicates involvement of the OFC and limbic structures in the neural underpinnings of antisocial behaviour (Anderson et al., 1999), and that the intactness of these structures is vital in the attainment of social behaviour and moral ability.

Blair and Cipolotti (2000) report on a case of acquired sociopathy in a patient with OFC damage, who became highly aggressive with little regard for others after his injury. They compared a 56 year old male (JS) with trauma sustained to the bilateral prefrontal cortex who had been diagnosed with antisocial personality disorder (APD) since injury, with a 46 year old male (CLA) with motor-neuron disease affecting his right prefrontal cortex and sparing the OFC. Complex assessment was carried out including interviews and tests of affect, social functioning and diffuse brain functioning, along with the WAIS-R, and a ‘frontal battery’ including the Cognitive Estimations Test, WCST, Stroop Test, Trail Making Test, and Verbal Fluency. The authors found that both patients demonstrated executive dysfunction consistent with lesion sites, but JS demonstrated ‘acquired sociopathy’ as well. They believe that the aberrant behaviour changes seen in JS but not in CLA are consistent with an OFC damage explanation of acquired sociopathy (Blair & Cipolotti, 2000).

A conundrum often observed with OFC damage, is that despite obvious changes in personality that can impair the individual’s daily living and social interactions, performance on neuropsychological tests can remain unimpaired (Damasio et al., 1990; Damasio, 1991). There may be several explanations for this; including that current neuropsychological tests may be insensitive, or particular tests used may be inappropriate for the area of damage. For example, many purported executive function tests are testing certain components (often not clearly identified) and these may not be the component of interest in a particular study or individual. Another explanation is that components of executive functioning may still be intact, but the ability to access them and use them may not. The ability to access and use executive components is largely dependent on the integrity of the dIPFC
(Dolan et al., 2002; Duncan et al., 1997). An explanation put forward by the authors who first coined the term ‘acquired sociopathy’ is the *Somatic Marker Hypothesis*. This suggests that OFC lesions impair the capacity to take emotions and emotional response into account when making a decision (Damasio et al., 1990; Damasio, 1996). This may lead to violation of social norms and self-defeating decision-making (Anderson et al., 1999), however these deficits are not readily testable in the laboratory. In paradigms that attempt to test the somatic marker hypothesis in influencing decision making, such as the Gambling Task, damage in the OFC has been demonstrated to reduce good decision-making that takes into account somatic cues (Bechara, 2004). In contrast other researchers have found that patients with discrete OFC lesions can perform within the norms (Manes et al., 2002) and that the dIPFC is also critical for good decision making on the Gambling Task (Fellows & Farah, 2004; Krawczyk, 2002; Manes et al., 2002).

In summary, although historically OFC lesions alone were thought to exacerbate the expression of aggression, it now appears both dIPFC and OFC lesions may increase aggressivity. OFC lesions appear to increase impulsivity and impinge on the acquisition of emotions and morals, while the dIPFC is required for general processing, perception of threat, and direction of behaviour. Lesions of the dIPFC contribute to lower frustration tolerance, reduce inhibitions, and decrease long-term goal directed behaviour.

### 3.2.6 EEG and Imaging Studies of the Brain-Violence Connection

Evidence for general brain dysfunction, and particularly prefrontal anomalies, associated with violent offending have been found in imaging and EEG studies (see Table 2). Caution must be taken however in interpretation due to the heterogeneity of individuals in many of the samples and across the samples, inconsistent operational definitions of violence, and differing methods and ‘strengths’ of imaging.
Electroencephalogram

In early electroencephalogram (EEG) examinations of criminals, general brain dysfunction has persistently been highlighted in criminal offenders. Stafford-Clark and Taylor (1949) evaluated 58 men and 6 women convicted of murder using EEG. This sample was divided into five groups: group 1 included offenders where the killing was incidental to a primary crime; group 2 included offenders considered to have a clear motive for killing; group 3 were individuals in which little motive could be found; group 4 were those who demonstrated a sexual motive; and group 5 included offenders who were found unfit to plead guilty due to insanity. Participants in group 5 (86%) had the most abnormal EEG findings, followed by group 3 (73%), group 4 (50%), group 2 (25%), and group 1 (less than 10%). The authors were unable to speculate on location or function of the abnormal activity, instead expressing concern regarding overall brain dysfunction correlating with motiveless crime.

Later, Hill and Pond (1952) examined inmates with recidivistic backgrounds and asserted that 70% of murderers and 65% of aggressive psychopaths had significant excesses of bilateral theta activity compared to non-violent inmates. Again this study did not demonstrate specific locations of theta activity nor relate this excess activity to particular functions. Williams (1969) examined 1250 inmates who had continuously committed violent crimes over the previous two decades. He found that 65% of the sample displayed various abnormal EEG recordings, particularly in the fronto-temporal areas. Williams (1969) interpreted this as consistent with temporal lobe epilepsy explanations of violence. Williams’ sample included many sexual offenders and operational definitions of violence were varied, limiting the conclusions that can be drawn from this study regarding violent offenders that do not sexually offend.

Contrary to some early positive findings of a correlation between antisocial behaviour and various abnormal EEG recordings, several other early studies
reported no link (Gibbs, Begchl, & Bloomberg, 1945; Silverman, 1943; Small, 1966). In general these earlier studies were plagued with methodological shortcomings including difficulties in recording the EEG, varying classifications of abnormality, varying definitions of violence, heterogeneous offender groups, and a lack of controls.

In a later study Volkow and Tancredi (1987) found an abnormal EEG pattern and cortical slowing in the anterior temporal lobes in four chronically violent schizophrenia sufferers when compared with non-violent individuals with schizophrenia. An excess of delta activity in the left frontal lobe was found to be present in the violent individuals that was not present in the non-violent schizophrenia patients. However, this population represents disturbed psychotic patients on polydrug medication, which could account for the patterns seen. Information given regarding medication regimes was not provided, but it is likely that the violent schizophrenia patients were on more medications or higher doses than the non-violent individuals.

Electroencephalogram evaluations in a study of 21 right-handed persistently violent male adults revealed a positive relationship between the level of violence and EEG changes in the frontal areas (Convit, Czobor, & Volavka, 1991). In this study more delta activity was related to a higher level of violence. Again this study used psychiatric patients and hence shared similar limitations with the study described above. This study also did not provide controls, further limiting conclusions regarding these abnormalities.

Wong, Lumsden, Fenton, and Fenwicket (1994) after retrospectively reviewing EEG records of 372 violent psychiatric patients, found a high proportion of anterior temporal lobe abnormality in the most repeatedly aggressive patients. This study is limited by its retrospective nature and the lack of report on medication and comprehensive psychiatric and TBI histories. However, of note is the temporal lobe abnormality, which could indicate involvement of parts of the
limbic system. Indeed, later studies by Wong and his team using MRI and PET on this population demonstrated left anterior temporal hypo-metabolism (Wong et al., 1997) in an examination of repeatedly violent forensic inpatients when compared with healthy controls.

Deckel, Hesselbrock and Bauer (1996) demonstrated decreased frontal lobe activation in a group of 89 young males (21-25 years of age) previously seen because of ‘juvenile delinquency,’ using EEG and neuropsychological testing. This study failed to use controls and consistent operational definitions, but is strengthened by corroborating EEG and neuropsychological measures to define ‘frontal’ activation. The resultant pattern on the neuropsychological tests will be explored later.

Using EEG with 20 male murderers and comparing readings to a normative data base, Evans and Park (1997) found that 17/20 men in the sample had significant abnormalities, and that the majority of these abnormalities were located in the prefrontal cortex or the right hemisphere. The authors suggest that abnormalities in these regions are in line with executive deficits (in the case of anterior EEG abnormalities) and emotional poverty (right hemisphere abnormalities) characterising violent offenders. Also using EEG in a murderer sample, Sakuta and Fukushima (1998) compared readings of ‘severe murderers’ against other violent criminals and demonstrated unspecified significant differences between the groups, with 79% of the murderers demonstrating abnormalities compared with 35% of the other criminals. However, the other violent criminal category included what the authors termed, ‘simple murderers,’ as well as offenders convicted of theft, arson or drug offences. This is a heterogeneous category to compare to the severe murderer cases that included aggravated murder and mass murder (killed two or more people at one time). CT and MRI scans were taken for some of the participants, and given the imprecise demarcation of the offenders it is perhaps unsurprising that morphological differences failed to significantly differentiate between the groups.
Reduced evoked potentials, specifically P300 responses, in 11 male inmate psychopaths during a visual oddball task were found by Kiehl et al. (1999) compared with 10 male inmate non-psychopaths. Further useful information could have been provided by descriptions of the nature of the convictions of the participants. This reduction in P300 response indicates processing difficulties for the psychopaths but diverges from Raine and Venables’ (1998) demonstration of enhanced P300 signals during a visual task in psychopaths. Methodological differences may explain these opposing findings as Raine and Venables (1998) employed a continuous visual task while Kiehl et al. (1999) employed a salient and novel single-application task. Despite the differences, both methods demonstrate a difference in processing between psychopaths and non-psychopaths.

Many studies implicate EEG abnormalities in violent recidivistic offenders (Raine, Venables, & Williams, 1990). This is thought to reflect the general under-arousal hypothesis purported to explain psychopathy. In a prospective study following 101 men, Raine, Venables, and Williams (1990) established a relationship between slow-frequency brain waves (along with electrodermal and cardiovascular under-arousal) as measured by EEG and criminality. Males assessed at 15 years old were followed-up almost 10 years later via the Criminal Records Office in London and re-assessed. Discriminant function analysis indicated that almost 75% of subjects could be correctly classified on the basis of arousal values, into either offender or non-offender groups (Raine et al., 1990). However, this variance cannot completely predict all criminal behaviour, with social and environmental variables potentially playing a role. Further, violent criminal behaviour was not operationally defined or separated from other types of offending or violent sexual offending.

It has been suggested that individuals who are impulsive and have difficulty in regulating their behaviour may be seeking stimulation to offset difficulties in receiving stimulation through normal means, and this may take the form of
criminality. Houston and Stanford (2001) compared 15 aggressive and impulsive participants with 15 controls cleared of any impulsive traits on initial screening, using EEG of evoked potentials. The authors concluded that the aggressive impulsive individuals demonstrated a pattern that is indicative of an ineffectual sensory gating mechanism. Additionally they concluded that the impulsive aggressive participants demonstrated patterns signifying a faster and more alert response to stimuli in order to compensate for a low resting level of arousal. This pattern could account for why these individuals are more likely to seek out extreme stimuli, be more responsive to trivial stimuli, be reward dependent and unable to delay gratification, and display increases in risky decision-making.

**Positron Emission Tomography and Single Photon Emission Computed Tomography**

Adrian Raine and his colleagues tested the notion that violent offenders may be characterised by frontal lobe dysfunction through assessing glucose metabolism using positron emission tomography (PET) in a population of murderers matched against a control group (Raine, Buchsbaum, & LaCasse, 1997; Raine et al., 1994). The researchers found that the murderers presented deficits specific to the prefrontal cortex that did not extend to temporal and parietal regions. The fact that glucose metabolism was reduced only in the prefrontal area is important, as prefrontal deficits have also been noted in people with schizophrenia. In the control population of individuals, including those with schizophrenia, there was also reduced metabolism of glucose in the right temporal and parietal lobes (Raine et al., 1994) along with frontal reductions. The participants with schizophrenia were not receiving anti-psychotic medication at the time of the PET procedure, but this does not discount the possible effect previous medication regimes may have had on the brain, therefore influencing the results of the scan. Drug histories of the participants, both prescription and abuse, were not reported by the researchers. Supporting Raine et al., (1994) is Goyer, Andreason, and Semple’s (1994) study using an auditory activation task and PET. In this study reduced
prefrontal glucose was associated with increased aggressive acts in a sample with antisocial personality disorder (Goyer et al., 1994).

Using PET with eight psychiatric patients with records of repetitive violence compared against eight healthy controls from the community Volkow et al. (1995) demonstrated poor glucose metabolism in both frontal and temporal regions in the psychiatric patients in comparison to controls. The mixed psychiatric histories of the experimental sample and the influence of medication limit the ability to draw definitive conclusions from this study.

Oder et al. (1992) used behavioural and personality questionnaires and single photon emission computed tomography (SPECT) with 36 participants that had sustained severe TBI within the previous 40 months, demonstrating relationships between high levels of disinhibition and aggression and frontal hypoperfusion. However, as the authors identify, the premorbid characteristics are influential in contributing to the brain injury as well as the resultant imaging, i.e., in this study violent individuals also scored as risk takers and as impulsive in the behavioural measures used and this appears to have been present pre-TBI.

Amen, Stubblefield, Carmichael, and Thisted (1996) used SPECT imaging with 40 medication-free adolescents and adults from a psychiatric population who were identified as aggressive, and 40 medication-free psychiatric patients who were categorised as non-aggressive. Each group consisted of 30 men and ten females. The individuals assigned to the aggressive group were identified as having either attacked another person or destroyed property within the six months prior to SPECT evaluation. An examiner external to the study read the SPECT results without knowledge of group membership. Sixty percent of the aggressive group had results demonstrating decreased activity in the prefrontal cortex as opposed to 35% of the non-aggressive group. Individuals with substance abuse or major head injury within the past year were excluded from the study, but exclusion did not reach beyond a one-year term.
**Computed Axon Tomography and Magnetic Resonance Imaging**

Using magnetic resonance imaging (MRI) and computed axon tomography (CT) with a forensic (but psychiatric) sample of 89 participants, Tonkonogy (1991) demonstrated that participants with frequent violent behaviour had lesions in the anterior-inferior area of the temporal lobe. The author speculated that this might result from tissue loss in the amygdala-hippocampal region of the temporal lobe. Again this study was uncontrolled and the varying diagnosis and aetiologies of individual psychiatric difficulties as well as inconsistent definitions of violence mean conclusive evidence for the brain violence link was not provided.

General but unspecified brain abnormalities were documented by Aigner et al. (2000) in a group of 82 (52 sexual offenders) forensic patients from a maximum-security prison. The men were separated into ‘high violence’ and ‘low violence’ groups depending on their offending and recent behaviour. Using MRI read independently by ‘blind’ neuroradiologists the authors demonstrated significant differences between the groups, with 65.5% of the high violent offenders demonstrating brain abnormalities as compared with 14.6% of the low violent group.

In an attempt to complete a comprehensive examination of relationships of brain damage and violence, brain imaging and neuropsychological measures were taken on 31 adult male murderers (Blake, Pincus, & Buckner, 1995), along with neurological and physical examinations. Nineteen inmates underwent imaging with MRI or CT, with 9 of 19 participants demonstrating significant changes or abnormalities, consisting primarily of anterior atrophy and white matter abnormalities. Twenty inmates had EEG and in contrast to other findings, minimal abnormalities were demonstrated in only eight men, whereas neurological examination revealed 20 of the 31 subjects displayed some form of neurological frontal dysfunction. Neuropsychological testing revealed wide-ranging abnormalities, including executive dysfunction in all subjects (Blake et
al., 1995). Difficulties with the Blake, Pincus and Buckner (1995) analysis include that not all men were convicted; three were awaiting trial, while others were appealing their conviction. Additionally, the types of murder were varied from serial murder, to murder while completing another crime (e.g., robbery), to ‘crimes of passion.’ It has been suggested by earlier literature that there may be fundamental differences between the types of individuals perpetrating differing types of murders (Tanay, 1969). Also, bias is induced in this sample as the murderers lawyers put forward their clients, with some attempting to find defences for clemency. However, even with these limitations and the relatively small sample, the authors state they were impressed by the large proportion of offenders demonstrating “frontal” signs of dysfunction 65%, compared to 29% of murderers showing temporal/limbic dysfunction, especially as they were expecting only temporal dysfunction due to their reading of the earlier literature (Blake et al., 1995).

Seidenwurm et al. (1997) used EEG, MRI, and PET with seven violent offenders and nine controls who presented at a neurology clinic for general and unspecified complaints (i.e., fatigue, headaches). They found hypoperfusion in the medial temporal lobes and hypothesised this to represent a dysfunction limbic system (Seidenwurm et al., 1997). Many of the crimes were extreme including serial killing and sexual violence against family members. An important difference in this study compared with others that have found frontal deficits, in murderers at least if not sexual offenders i.e., (Raine et al., 1998), is that no frontal lobe stimulation tasks were employed during examination.

**Other Imaging**

Using SPECT, MRI and regional cerebral blood flow (rCBF) in 21 (1 female) perpetrators of ‘violent impulsive crimes’ compared with 11 healthy controls Soderstrom et al (2000) demonstrated hypoperfusion and reductions in blood flow in anterior and temporal regions blood flow without corresponding structural
changes, indicated by normal MRI scans. This result held even when the authors removed experimental subjects from analysis who had a psychiatric illness, were on medication, or were substance abusers. The remaining seven subjects still demonstrated a pattern of frontal abnormality compared with controls (Soderstrom et al., 2000). The authors replicated these findings in a later study (Soderstrom et al., 2003), however each study included sexual assaulteders possibly accounting for the temporal abnormalities demonstrated.

Using magnetic resonance spectroscopy Critchley et al. (2000) found lower concentrations of N-acetyl aspartate, creatine phosphocreatine and choline-related compounds in the prefrontal cortex in repetitively violent inpatients compared to healthy controls. Controls came from both other patients and staff, with staff controls possibly reducing the effectiveness of the match.

A relatively large body of neuro-imaging evidence linking the frontal cortex to criminal behaviour comes from the work led by Adrian Raine. Over the last decade Raine and his team (Raine, Buchsbaum et al., 1997; Raine et al., 1994; Raine et al., 1998; Raine et al., 1990) have examined populations classed as antisocial and criminal and consistently found reduced glucose activity in the frontal region. Most recently in an examination of a sample with antisocial personality disorder (APD) they found attenuated cortex in the prefrontal lobes (Raine, Lencz, Bihrlle, LaCasse, & Colletti, 2000). In their work examining prefrontal grey and white matter volumes in APD individuals and controls, Raine et al. (2000) comment that prefrontal and autonomic deficits may predispose an individual to APD through limited social conditioning of the fear response. Individuals who are less responsive to social criticism would be less likely to respond to social punishment and disapproval, and therefore the authors assert, become predisposed to antisocial behaviour, including offending (Raine et al., 2000).
In an attempt to replicate and expand on the Raine et al. (2000) study Laakso et al. (2002) examined 24 male violent offenders and 33 control subjects. They concluded that in the violent offenders who met criteria for APD by scoring highly on the PCL-R checklist there was a measurable reduction in the left OFC, ACC and dIPFC regions compared to controls. However, upon closer analysis this difference was better accounted for by alcoholism and education differences. The controls were not evenly matched to the offenders’ education levels as a large number of controls sourced from the same university as the authors. This increased the education gap between the groups and limited the use of education as a variable accounting for variance in volume differences.

Kiehl et al. (2001) used fMRI to demonstrate that affective abnormalities in psychopathic subjects may be linked to dysfunctional input from limbic regions, including the amygdala, and that the poor activation of these areas during emotional tasks account for an inability of those diagnosed with APD to experience deep-seated emotions and process emotional material effectively and efficiently. Evidence for the roles played by the OFC, amygdala, and dIPFC in the integration and regulation of cognition, affect, and response inhibition have been put forward by Schneider et al. (2000). Schneider and colleagues examined 12 men with diagnoses of antisocial personality disorder (APD) and 12 matched healthy controls, using fMRI and two sets of conditioning stimuli. The authors found signal increases in the amygdala and dIPFC, compared to controls. They postulated that the process of accessing and learning from affective information is very difficult for those with APD, which places a heavy load on the regions responsible, whereas healthy normals can access this over-learned information easily and benefit from doing so (Schneider et al., 2000). This is similar to conclusion drawn by Intrator et al. (1997) who using SPECT demonstrated that in normals demands imposed by lexicon decision tasks are associated with less activation in frontal and temporal areas then in psychopathic subjects. Psychopath subjects used larger anterior areas for longer, processing emotional words in an effortful manner as if they were part of a second language (Hare, 1999).
Functional differences in processing between APD individuals and healthy normals has been recently corroborated with both imaging and behavioural measures (Gordon, Baird, & End, 2004; Kroner, Forth, & Mills, In Press), and even when psychopathic individuals are able to report on affective dimensions the processing remains dysfunctional (Kroner et al. In Press). In particular Gordon et al. (2004) using fMRI found a reliance on increased right dorsolateral prefrontal activation in participants that scored highly for psychopathy when performing an emotion recognition task compared to normals, indicating a heavy reliance on working memory, rather than the ability to access previously learned emotional lexicons.

Studies examining brain dysfunction and violence have varied in method as well as population and the sample sizes are generally small. However, the association of anterior deficits with aggression is suggestive of prefrontal dysfunction as a pathway to violence and antisocial behaviour. Anterior deficits are not always demonstrated in these populations, for example (Raine et al., 2001; Seidenwurm et al., 1997). Additionally, the prefrontal cortex is but one of a number of structures and circuits underpinning violence (Davidson et al., 2000; Raine, 2002).

3.2.7 Alcohol and Drug Use

Acute intoxication has been associated with crime and increases in violent behaviour for many years (Gerson & Preston, 1979). For example, higher proportions of violent offenders than non-violent offenders are intoxicated at the time of the offence (Lindqvist, 1986). However the nature of this association remains unclear (Volavka, Martell, & Convit, 1992). Steele and Southwick (1985) found that alcohol does not increase violent impulses or motives per se, but it reduces inhibitions against them, and so intoxicated people are more willing to do things they would like to do anyway but would normally override if they were sober. This section reviews studies that highlight the possible links between intoxication, executive dysfunction and aggression in both normals and offenders.
As a central nervous system depressant, alcohol may promote violent expression by reducing the inhibitory effects of the prefrontal cortex, but this dampening down may be expressed differently in different individuals. Volavka et al. (1992) point out that clinical observations suggest that alcohol has qualitatively different effects on behaviour in violent and non-violent individuals, with violent individuals becoming intoxicated at a faster rate, and becoming more aggressive than non-violent individuals. However, these observations came from studies where the alcohol was not dose controlled, so it may be that variation is explained by the violent individuals simply drinking more (Volavka et al., 1992). If this is so, it could be argued that the violent group drank more because of poor executive functioning, with the intoxication exacerbating established low baselines of functioning, leading to further reduced self-control and increased impulsivity, initiating aggressive behaviour.

Abram (1989) examined the effects of ingesting drugs and alcohol before offending and suggests that there is a strong probability that alcohol ingested immediately prior to a crime interacts with existing psychopathology and this directly influences the perpetration of violent crimes. However, his suggestions stem from his subjective observations and it cannot be assumed that without alcohol the crimes would not have been committed.

**Alcohol in Normals and the Link to ED and Violence**

Alcohol in paradigm use has been very useful for understanding the effect of reduced executive functioning in normals. In an examination of the literature on alcohol and cognition, Peterson, Rothfleisch, Zelazo, and Pihl (1990) concluded that the cognitive effects of intoxication are similar to the cognitive symptoms manifested by patients with executive dysfunction. Two important points support this conclusion. First, the prefrontal cortex is particularly vulnerable to the acute effects of alcohol consumption. Secondly, individuals with prefrontal lesions display similar behaviours to individuals who are drunk (e.g., reduced self-
control, impulsivity, and lack of regard for consequences of current actions). Hoaken, Giancola, and Pihl (1998) posit that alcohol disrupts the role played by the prefrontal cortex in completing ‘higher-order’ mental abilities. This is similar to work examining the impact of alcohol on serotonin levels (Pihl & Lemarquand, 1998), and is congruent with work by Langevin et al. (1987) who demonstrated that when violent offenders were injected with alcohol they experienced more behavioural disturbances and feelings of inferiority and paranoia than the control group. The experimental sample also had a higher rate of TBI suggesting an additive effect of alcohol abuse and existing brain dysfunction resulting in a propensity towards violence or an increased propensity for violence.

When the prefrontal cortex and associated circuitry is “anaesthetised,” the input of the limbic system will be more apparent, and may result in poorer decision-making and disinhibited behaviour. The restriction in integrative function caused by intoxication, can result in socially inappropriate behaviour. This is evidenced in poor impulse control, disinhibition, and lack of consideration about future consequences. Using controlled doses of alcohol and assessing functioning with neuropsychological testing, Peterson et al. (1990) demonstrated that high doses of alcohol resulted in reduced scores on tasks of prefrontal functioning, but had a lesser influence on ‘non-frontal’ test scores. When discussing the role of impaired executive functioning, Giancola (1995) has hypothesised that executive dysfunction disrupts the ability to interpret social cues, leading to misinterpretations of threats in conflict situations. Impairment of executive functioning via intoxication may additionally undermine the capacity of the prefrontal cortex to generate suitable alternative behaviours, and to build or call upon an established repertoire of adaptive responses that result in avoidance of aggressive situations and violent outcomes.

A study by Gantner and Taylor (1992) found that when comparing intoxicated and non-intoxicated subjects in threatening and non-threatening situations, intoxicated subjects acted more aggressively in both situations. Aggression was elicited and
measured using the Taylor Aggression Paradigm (TAP) (Taylor, 1967), which involves the monitoring of the level of ‘electric-shock’ administered by the subjects to their opponents. Shock was administered by pushing one of 10 buttons, graduated according to the level of electrical charge. The experiment also included threatening (shock-on) versus non-threatening (shock-off) parameters and provocation (told that the slower person on any given trial would receive the shock) versus non-provocation functions. The intoxicated subjects were the only participants who, in non-threatening situations but under increasing provocation, increased the shock levels for their opponents to very substantial levels (Gantner & Taylor, 1992). This, the authors elaborate, is support for the notion that it is how we perceive threats in the environment that determines our reactions, rather than what the actual threat is. This interpretation of perceived aversive stimuli may be combined with the inability to inhibit ‘knee-jerk’ reactions to it. The above example can assist us in imagining what it may be like for someone who has permanently under-aroused brain function, leading to frequent perception and over-reaction problems.

Research similar to that of Gantner (1992) and Peterson (1990) was conducted in 1995 by Lau and his colleagues. This time the effects of executive dysfunction and intoxication were combined. Lau, Pihl, and Peterson (1995) separated 48 men into groups of ‘high cognitive performance’ (those with no executive dysfunction) and 'low cognitive performance’ (those with some executive dysfunction) depending upon the results of two tests of frontal function: the Self-Ordered Pointing (SOP) task and the Spatial Conditional Associative Learning Test (SCALT). These two tests assess the ability of working memory to process and manipulate large amounts of information (Hoaken, Giancola et al., 1998). In each group half the participants were given a measured dose of alcohol. Aggression was then elicited and assessed using a modified version of the TAP (Taylor, 1967). Lau et al. (1995) found that the group with some executive dysfunction were more aggressive under high provocation than the group without executive dysfunction. When alcohol was involved, aggression as a function of provocation
increased for both groups. The conclusion drawn by the researchers was that alcohol intoxication and executive dysfunction are involved in the disinhibition of the behaviours that keep aggressive impulses in check (Lau et al., 1995). The inference that at some level alcohol intoxication mirrors executive dysfunction, at least during provocation situations, can also be drawn from these results. This may be further evidence of how our perceptions guide our actions, and if these perceptions are clouded by alcohol or executive deficits we may respond inappropriately to the reality of the environment.

It is important to recognise the input of the prefrontal cortex and the ‘intactness’ of our executive functions in our ability to perceive. Intoxication leads to an under-arousal of the brain, and especially the prefrontal lobes, leading to an inability of the executive functions to regulate perception, which in turn allows misinterpretation of the environment, increasing the propensity for violence. This is an assertion made by Leonard, Collins and Quigley (2003) who studied “bar-room brawls” in 190 healthy men who had recently been involved in them. The authors demonstrated that the men who had consumed the largest amount of alcohol and who were very intoxicated, had perceived themselves to be under more threat from others in their vicinity, which led them to become involved in more severe fighting, and in turn they perpetrated more incidences of severe and dangerous physical assaults.

Hoaken, Giancola, and Pihl (1998) review the literature that documents a relationship between reduced executive cognitive functioning (executive dysfunction) and aggression. They present a hypothetical model that suggests executive dysfunction or poor prefrontal function is an underlying mechanism for the link between acute alcohol consumption and intoxication and aggressive behaviour. They conclude that executive dysfunction appears to predict which individuals will or will not become aggressive, in both sober and intoxicated conditions.
**Drug and Alcohol Use and Offending and Violence**

Laboratory demonstrations of alcohol on executive function have been illuminating in understanding the impact of ED on violence. In attempting to find similar explanations in real-world drug and alcohol use on ED and violence and offending the evidence is less clear (Volavka et al., 1992; Fishbein, 2000). However, chronic and acute substance abuse is linked to changes in metabolism and cerebral blood flow and consequent changes in levels of arousal, attention, and functioning in the prefrontal and temporal cortices (Volkow et al., 1991).

Many studies that link crime with drug use show a covariance between addiction and crime: specifically, involvement in crime increases during periods of active addiction (Nurco, Hanlon, Kinlock, & Duszynski, 1989). What is less clear in these studies is the nature of the offences (Volavka et al., 1992). Violent behaviour has clearly been related to the use of amphetamine and cocaine (Volavka et al., 1992), but it is unclear if this association is an outcome of the substance type, or due to premorbid factors. For example, some studies of APD have shown that amphetamines, cocaine, and their derivatives are the drugs of choice among APD individuals, and APD has in turn been associated with drug abuse and violent acts (Volavka et al., 1992). It is suggested that a predilection for stimulants may be motivated by an APD individual seeking stimulation.

There are similarities noted in the literature between aggressive individuals and individuals who abuse drugs. Fishbein (2000) states that similar executive deficits characterise both aggressive individuals and drug abusers. Fishbein (2000) has developed a framework that links neuropsychological dysfunction (particularly poor executive functioning), drug abuse, and violence, and reports that the same biological substrates (the orbitofrontal area of the prefrontal cortex and its connections with the limbic system) appear to underpin all three. Functionally, Fishbein reports that impulsivity, poor decision-making, disinhibition, and an
inability to assess consequences are shared precursors for both drug abuse and violence.

In work linking the executive functions, violence, and the input of alcohol, Amen (1999) presents a case study of a young man who appeared to exhibit marked violent behaviour only when intoxicated. Between the ages of 18-20 years he had 10 arrests ranging from drunk and disorderly, to assault of his girlfriend, to armed robbery. When examined with SPECT in sober and intoxicated conditions, marked differences were noted in his brain functioning. Upon examination with SPECT when intoxicated he had reduced activity in the frontal cortex combined with increased cingulate gyrus activity. Amen (1999) hypothesised that this allowed a decrease in impulse control and judgement. This study seems to support the hypothesis of intimate connections between the prefrontal cortex and the deeper regions of the brain, and highlights what can happen if prefrontal functioning is dulled somewhat and subcortical regions are activated or allowed to dominate.

A further risk factor for the display of dysexecutive behaviours when intoxicated may be a vulnerability to the effects of alcohol. Vulnerability has been demonstrated in samples that have a neuronal loss in the basal orbitofrontal area, resulting in a measurable reduction of serotonin (Johansson, Bergvall, & Hansen, 1999). Vulnerability to alcohol may be due to an existing lowered tolerance for organic solvents, because of an already overall lowered general arousal level due to the effects of past head injury, mental illness, prescription medication, or previous alcohol and substance abuse.

Foetal Drug and Alcohol Exposure and Offending and Violence

Both animal and human studies have established a link between foetal nicotine exposure and altered brain development (Ajarem & Ahmad, 1998; Liu & Wuerker). Maternal smoking in humans has been associated with antisocial
behaviour in children (Fergusson, Horwood, & Lynskey, 1993; Orlebeke, Knol, & Verhulst, 1997; Weissman, Warner, Wickramaratne, & Kandel, 1999) and adults (Brennan, Grekin, & Mednick, 1999). Smoking during pregnancy is thought to affect later behaviour through both functional and structural routes (Liu & Wuerker, *In Press*; Raine, 2002). Animal research has demonstrated reductions in serotonin in forebrains of rats after prenatal nicotine exposure (Muneoka et al., 1997), and impairments in the basal ganglia and anterior regions of the brain as well as the cerebellum have been documented in humans after nicotine exposure in-utero, (see Raine, 2002 for a review).

In an examination of children, Weissman et al. (1999) found a fourfold increase in conduct disorder in prepubescent boys whose mothers smoked during pregnancy, compared to those that did not. Rasanen et al. (1999) in their Finnish cohort of 5636 males found a two-fold increase in violent offending in those who had been exposed to nicotine in-utero. In an examination of a Danish cohort of 4169 males Brennan et al. (1999) demonstrated that maternal smoking during pregnancy predicts criminal activity in offspring, even when socio-economic, parental, and perinatal variables were controlled. The foetal nicotine exposure was particularly related to recidivistic violent offending (Brennan et al., 1999). This is consistent with later replication by Rasanen et al. (1999) and Piquero et al. (2002). In contrast to these studies associating nicotine exposure in-utero to aggression, some studies of maternal smoking indicate factors are more strongly associated with later behavioural difficulties. For example, maternal smoking showed a strong dose-relationship between the amount of smoking while pregnant and the severity of conduct disorder among boys in Maughan et al.’s (2004) study, however upon closer examination, the mothers that smoked were remarkably different from the mothers who did not smoke and demonstrated more antisocial traits themselves as well as raising their children with less resources. When personality and environmental variables were added as covariates, the prenatal nicotine exposure did not factor significantly in association with the severity of conduct disorder.
Foetal alcohol exposure has long been associated with birth defects and severe behavioural changes. Roebuck et al. (1999) assessed 32 children exposed to alcohol (including 19 with foetal alcohol syndrome; FAS) in the womb and 32 children who had not been exposed, along with their parents in interviews and behavioural ratings. The groups were matched for age, gender, and socio-economic status. These authors demonstrated that mothers who drank alcohol heavily during pregnancy placed their offspring at increased risk for psychiatric and behavioural problems, including the exposed children without FAS (Roebuck et al., 1999). This study revealed that exposed children without FAS developed similar psychosocial difficulties to the children with FAS. These significant findings held even when living with an alcoholic parent was factored out.

3.2.8 Social and Developmental Factors

Controversy exists over the contribution of nature/nurture in terms of understanding violence and aggression. It is frightening for people to believe that ‘evil’ can be innate, yet it is equally disturbing to believe that humans and the environment can contribute to the violent characteristics of another person. A great deal of research has been published that attempts to clarify the variables in the environment that impact on personality development and expression of behaviour, including violence. The prefrontal cortex continues to develop long after critical periods have passed for other behaviours and structures, hence developmental research may assist clarification regarding how the prefrontal cortex underlies some behaviour and how these ultimately find expression.

The basis of behaviour, including the executive functions, begins at conception with the different genetic input from each parent, and continues to be shaped by the environment the foetus develops in. The developing brain in the womb is vulnerable to the environment and teratogens can selectively damage the developing foetus according to the time of gestation and the amount of the teratogen (Karr-Morse & Wiley, 1997). This is apparent in not only the foetus
being affected by what the mother may ingest (such as drugs, alcohol, and poisoned food), but in the emotional state of the mother. Stress that activates high blood circulation levels of corticosteriods in the mother has been found to affect the foetus and development of the brain, which in turn impinges on later function and behaviour (Dawson, Hessl, & Frey, 1994; Gitau, Cameron, Fisk, & Glover, 1998; Kaufman, Plotsky, Nemeroff, & Charney, 2000; van der Kolk & Fisler, 1994).

Significantly raised maternal cortisol levels appear to raise levels of cortisol in the foetus (Gitau et al., 1998). This can cause changes in the structure of the brain possibly leading to later developmental problems (Kaufman et al., 2000). Changes include certain brain structures not fully developing, or showing developmental delay. This may result in compromising abilities dependent upon the delayed structure. Which particular structure is compromised depends upon the developmental stages of the foetus. Once the baby is born the developing brain continues to be susceptible to interference. In cases of child abuse there is evidence of neurons and connections being utilised especially to cope with abuse in the child (Perry, 1997; van der Kolk & Fisler, 1994). This not only detracts from neuronal development of critical functions, but may also foster vulnerability in the child to development of later behavioural and psychiatric problems, such as hypervigilance and paranoia (Perry, 1997).

It is recognised that there are critical periods of development for certain connections and functions, and if the appropriate stimulation is not provided via the environment, the connections may never develop. Therefore the function reliant upon these pathways will not develop, or will be weak and vulnerable to later stressors (Miller, 1999). The critical period for the prefrontal cortex is traditionally viewed as adolescence, with major maturation occurring in late adolescence (Golden et al., 1996). However, the prefrontal cortex appears to experience continual input and ‘improvement’ over most of the lifespan, lacking the defined critical periods associated with some other neural functions, such as
the attainment of syntax and grammar. There is also evidence of intact executive or prefrontal function being employed by children under five, such as goal directed behaviour (Happaney et al., 2004; Heyder et al., 2004; Hofer, 2003). The quality of the input is important in an individual developing appropriate behaviour, and as Hofer (2003) states, early relationships and family are not only essential to the protection and feeding of an infant, but also vital in organising the functioning of the brain.

Environmentally induced executive deficits in early childhood may not be apparent until relevant neurodevelopmental stages are reached. Damage may have been sustained at a time when the structure or function was immature; hence as the child matures and it is time for certain structures to be utilised, that child may be noted to ‘grow into’ a deficit (Miller, 1999). For example, around the ages of five to eight years, the temporal, occipital, and parietal lobes begin to actively integrate information required for reading, writing, and arithmetic (Miller, 1999). A child who has sustained previous damage in one or more of these areas may be unable to integrate across these modalities and thus their ability to carry out the functions subserved by these structures is compromised, resulting in a learning difficulty. Similarly, prefrontal damage sustained early in life can affect a child in various developmental stages, but perhaps the impact will be most noticeable first in learning and expression, and then in social development during adolescence. Miller (1999) in a review of the literature reiterates research that demonstrates that damage to the tertiary zone will impact on a child’s new learning capacity by compromising the ability to simultaneously integrate information from two or more association cortices. Observable effects of early damage to the prefrontal cortex may not surface until the adolescent is required to conceptualise goals including long range planning, making decisions based on abstract information, shifting mind set, controlling impulses, self monitoring, and other behaviour we recognise as part of social maturity. Certainly these were the executive functions that patients who sustained prefrontal injuries as infants in the Anderson et al., (1999) study had difficulty with, with more difficulties encountered during their
adolescence and adulthood than their early childhoods. Adults require healthy executive functioning to allow them to meet societal needs. Adolescence is a time marked by turmoil and change, and an individual with limited prefrontal capacity will find it difficult to operate in this new world and enter adulthood without the ‘tertiary’ skills the prefrontal cortex is posited to be responsible for.

Prefrontal damage leading to executive deficits in children, whether present early or later, can lead to serious developmental consequences, which if severe, are likely to show significant impact during young adulthood. Moffitt and Henry (1989) describe executive dysfunction giving rise to early childhood behaviour problems that set the stage for later delinquent behaviour as the child ages in years but fails to mature socially. In fact, behavioural problems that look like truancy or purposeful rule-breaking may be linked, in some children, to compromised executive functioning due to difficulties in maintaining attention and mind-set, and problems with impulsivity and self-regulation. Moffitt (1993) proposes there is a difference between adolescence-limited and life-course persistent antisocial behaviour. Offenders who continue to rule-break beyond young adulthood display life-course persistent anti-social behaviour. These individuals demonstrate more severe and continuous executive dysfunction, particularly in the areas of impulsivity and interpretation of the environment. In contrast, adolescence-limited offenders are a group that appear ‘delinquent’ only during the teenage or young adult years. Moffitt hypothesises that this condition is mediated by lack of social maturity and slow developing executive functions, and once social maturity is gained the offending behaviour disappears.

*Plasticity* of function, or cerebral reorganisation, is often discussed in relation to paediatric brains. Plasticity is an important characteristic of normal development, but also allows functions lost as a result of neuronal damage to be taken over through cerebral reconstitution. It is important to note here that even if other systems are able to mediate the functions of the damaged structures, it may come at a high cost. The extra activity now required of undamaged parts of the brain
may compromise the functions originally mediated by the structures. This may lead to lowered ability in these functions as well as an obvious compromise in the ‘new’ function the brain is attempting to serve (Miller, 1999). In an individual whose brain is compensating for lost functions as well as a decrease in specific abilities, more insidious effects may be apparent such as lowered concentration, slowed processing speed and slower reaction times. These pervasive but non-specific effects will have a great impact on a developing child’s capacity to learn and interact in both a social and academic environment. Because these effects are so intangible, it makes them very hard to recognise as problems in their own right, and they will often be over-looked in the management of the child’s education and well-being. Miller (1999) makes the assertion that it is less the loss of previously acquired skills and more the effect upon the future capacity to learn that will have far reaching consequences in paediatric brain injury.

Early work by Bandura (1973) demonstrated the powerful principles of vicarious learning in children from their parents and the environment. In Bandura’s classic experiments with ‘Bobo-the-clown’ he demonstrated how it is possible to learn new behaviours, in this case violence, after witnessing it as a ‘norm,’ and then internalise this remote social reinforcement in order to continue to use it. Numerous real-world accounts have shown that children with behavioural and/or conduct problems often come from homes or schools that are disadvantaged or ‘deviant’ in some way (Rutter, 1985). In a longitudinal examination of more than a thousand infants followed from 1947 to 1980 in Newcastle (UK) (Kolvin, Miller, Fleeting, & Kolvin, 1988), the authors concluded that children in ‘deprived’ families were more at risk for offending during later childhood and beyond, and this relationship was stronger for males, as female offences only accounted for 6%.

Single-parent family research is finding increased delinquency in deprived families, especially amongst boys from fatherless families (Farrington, 1991a). Some researchers view this as a result of ‘stretching resources,’ as similar patterns
are also seen in very large families with two parents (Farrington, 1994). This stretching of resources may be a stressor in families that could impact upon how young minds are nurtured, both nutritionally and environmentally. Reduced stimulation and learning opportunities for these children, and possibly an increased likelihood of ‘falling in with the wrong crowd,’ are potential outcomes of impoverished resources.

In an examination of a Danish male cohort, Raine, Brennan, and Mednick (1997) found that when birth complications were combined with early maternal rejection, there was a predictable association with adult serious violent crime. This interaction remained significant even when the total number of crimes were added as a covariate. Thus, the effect could not be accounted for by the fact that violent offenders commit more crimes in general. A difference was noted between violence committed and threats of violence. Committed acts of violence had a significant relationship with the combination of maternal rejection and birth complications, whereas threats of violence were not associated with these variables. Another interesting finding from the study demonstrated that the interaction between birth complications and maternal rejection had a significant relationship with early-onset serious violence (first crime committed before 18 years). For those who committed their first violent crime after 18 years there were no significant differences between the factors of rejection only, birth complications only, and the combination of birth complications and maternal rejection. Raine et al. (1997) screened for maternal mental illness to examine whether this accounted for any of the variance in the birth complications/maternal rejection and violence outcome. All analyses suggested that maternal psychiatric illness did not account for the interaction.

Research has clearly established that the impact of child abuse is more devastating when it comes from the person entrusted with care for the child, such as a parent who is the main caregiver (van der Kolk & Fisler, 1994). The adverse effects of trauma on children can be mitigated by the mere presence of a supportive
caregiver even if the caregiver is actually unable to intervene and alter the outcome (Luthar & Zigler, 1991). The literature points to intervention being one of the most important factors in enabling an individual to ‘get back on track.’ Intervention or a lack of it may be explanatory regarding who will commit acts of violence and who will not, between individuals who appear to have similar backgrounds.

In research that examines the damaging influence of stress hormones on developing foetuses, some research demonstrates that the effect of stress and damage in-utero and in early development on both brain and social development are not irreversible. Kaufman et al. (2000) report on research that suggests subsequent supportive care-giving environments can moderate adverse effects of early stress. In their review of the effects of early adverse experiences on brain development they discuss animal studies that demonstrated marked differences in the way rat mothers handled pups that had been subjected to stress experiences (early handling and separation from the mother) when the pups were re-introduced to their mothers or foster-mothers. The main behaviour noticed was more licking and grooming of the pups showing greater corticosterone responses to acute stress. This simple ‘care-giving’ appeared to normalise the behaviour of the experimental pups to the point where no differences were observed in adulthood between rats subjected to early stress and rats left alone. The rats that were not fostered out to mothers that gave care continued to show high reactivity to stress as adults.

3.2.9 Executive Dysfunction Explanations of Offending and Violence

As already discussed, there is an established historical link between damage to the central nervous system (CNS), especially the prefrontal lobes, and violence (Blumer & Benson, 1975; Jones, 1992; Krakowski, 1997; Otnow-Lewis, 1983; Otnow-Lewis et al., 1985; Otnow-Lewis, Pincus, Feldman, Jackson, & Bard, 1986; Stuss & Benson, 1983). Although an association between CNS dysfunction
and aggression exists, the direction of the relationship is not clear. Is it because violent people are more likely to have sustained CNS damage, or vice-versa? Otnow-Lewis (1983) points out that whatever the mechanism involved in the association of CNS assault and aggression, evidence suggests that delinquent children suffer more head and face injuries than do non-delinquent children. She also notes that the more violent the children are, the more likely they are to have suffered such injuries. Otnow-Lewis et al. (1986) note that their entire sample of 15 inmates on death row for acts of violence that included murder, reported histories of severe head injury, and some were found to have major neurological impairment. Inmates who did not have major acquired impairments, still presented with ‘soft’ neurological problems, including ‘blackouts.’

Neuropsychological assessment of executive functions can describe how individual differences might result in the varying capacity for functions, such as a reduced potential to develop good executive functioning (Moffitt & Henry, 1989). Moffitt and Henry (1989) take the example of self-control and discuss how problems with this during development could lead to adult cases of APD. These authors stress that it is the interaction between the brain and environment that is the key in expression of behaviour. This neurodevelopmental interaction is particularly pertinent for the prefrontal cortex compared with other structures, as it requires more input from the environment over a longer period of time.

Consequent to the continual association in the literature between various forms of brain damage and changes in behaviour, criminal, delinquent, and clinical populations have been examined neuropsychologically in order to uncover evidence of neurological dysfunction that may account for criminal behaviour and/or violent behaviour. Direct evidence regarding whether particular types of brain damage predate violent behaviour, or is the consequence of it, can only be addressed in humans via longitudinal studies. There is a small body of evidence accumulating suggesting that brain dysfunction as assessed by neuropsychological measures (Moffitt & Henry, 1989) or by slowing of EEG (Raine et al., 1990) may
predate criminal behaviour. Further, as explored earlier, neurological accounts of dysfunction, for example serotonergic dysfunction, may serve to explain the risk for criminality by impairing inhibition of violent responses or stimulating impulsivity.

Jones (1992) proposes that there are four ways in which impaired brain function may mediate violence: (1) increased arousal interfering with the ability to think; (2) decreased ability to inhibit impulses; (3) impairment of attention, concentration, memory, and higher mental processes; and (4) misinterpretation of external events and stimuli. This is considered a standard explanation by many authors, for example (Bergvall, Nilsson, & Hansen, 2003; Berman & Coccaro, 1998) and reflects the large input of compromised executive functions in mediating violence. Krakowski (1997) suggests that brain injury appears to be associated with persistent or recidivistic violence, whereas acute positive psychotic symptoms are more prominent in fleeting, transient violence that may occur in an isolated episode (Krakowski & Czobor, 1994). In later reports Krakowski (2003) states that aggressive individuals are likely to have co-morbid difficulties with impulse control and emotional regulation, and implicates serotonergic dysfunction in this.

A neurodevelopmental predisposition to crime and violence may be expressed as a disruption of the normal neural mechanisms that control behaviour (Scarpa & Raine, 1997). Raine and Buchsbaum (1996) propose that prefrontal dysfunction is best viewed as a risk factor for violence, rather than prefrontal dysfunction being the cause of the violence itself. They present a pathway model that proposes an additive interaction between the individual’s internal and external environment, suggesting there are a number of pathways by which frontal dysfunction can contribute to violence. These pathways include influences from the neurological, behavioural, personality, social, and cognitive levels. They go further to postulate that the greater the dysfunction, the greater the likelihood of these pathways being activated, and when more pathways are activated, the greater the risk of violence.
Using this pathway model a premorbid tendency towards aggression and violence in some individuals may find expression when executive function is compromised.

Brooks, Campsie, and Symington et al. (1986) reported in a five-year follow-up of 42 Scottish cases of traumatic brain injury, that threats and acts of violence at five-years post injury were well above the rates measured at one-year post-injury. Reported threats rose from 15% at one-year post-injury to 50% at the five-year follow-up, and acts of violence doubled from 10% at the one-year assessment to 20% by the fifth year. Also, ‘trouble with the law’ was up from 7% in the first year post-injury to 13% at five years. These data are interesting not only for the demonstration of a relationship between damage to the brain and violence, but the data also highlight that mediating factors between brain injury and offending do not necessarily lessen with time after the head injury. Although presumably some recovery or compensation occurs, offending behaviour may actually get worse, or occur for the first time. This underscores the need for appropriate recognition and management of the acquired behavioural difficulties some people demonstrate after a brain injury. As O'Leary (2000) states:

“in fact, if individuals with brain injuries do not receive proper rehabilitation services, they have potential to end up in psychiatric and legal systems due to their inappropriate behavior.” (O'Leary, 2000; Behavioral Interventions, p. 205-206)

The contribution neurological damage makes to violent behaviour must be understood in the context of the individual’s underlying personality and intelligence. The manner in which tension is expressed in behaviour depends upon the available repertoire of responses possessed by the individual, and the ability to access and use a behavioural set. If the repertoire of executive responses in people with frontal lobe damage is reduced in range or inaccessible, anger and frustration may be expressed in socially inappropriate and uncontrolled ways, due to an impaired ability to inhibit impulses from deeper brain structures, and to successfully monitor and self-regulate one’s behaviour. Individuals with reduced
executive function due to compromised prefrontal circuitry may also misinterpret signals from others and view them as aggression and make responses accordingly (Gantner & Taylor, 1992; Giancola, 1995; Hoaken, Giancola et al., 1998; Hoaken et al., 2003). This may be especially so if the person already had limited or ‘immature’ executive functioning before damage to the frontal cortex (Giancola, 1995).

Heinrichs (1989) noted in his research that the presence of a focal frontal lesion was the single biggest predictor of violence in a chronic neuro-psychiatric population. Heinrichs (1989) examined a chronic in-patient psychiatric population looking for variables that predict a higher likelihood of violence. Data was gathered for ten predictor variables: age, education, diagnoses of schizophrenia, diagnoses of affective disorder, history of seizures, presence of focal frontal lesions, presence of confirmed cerebral disease irrespective of locus, gender, length of in-patient stay, and alcohol abuse. Data was collected from over two years from patient reports and entered into a stepwise multiple-regression format to predict the number of violent incident reports recorded for each patient. All variables were found to contribute to a third of the variance in the frequency of incident reports. Of these, three variables were found to contribute independently to variance: focal frontal lesions, number of in-patient days, and presence of a seizure disorder. From these three, focal frontal lesions stood out as the single best predictor of violent incidents (Heinrichs, 1989), accounting for approximately 11% of variance on its own. Although not made clear in the study, it is assumed some participants would be on psychotropic medication. In addition, the study is limited in not making clear the exact neuroanatomical locations of the various frontal pathologies of the patients.

There appears to be many associations made in the literature regarding the impact of prefrontal lesions and aggressive behaviour (Hawkins & Trobst, 2000). However a review by Kandel and Freed (1989) of the available neuropsychological literature examining APD and prefrontal dysfunction
concluded that any link between aggression and frontal lobe functioning was weak at best. They state that the studies reviewed were methodologically flawed, citing in particular: inappropriate or lack of controls, inconsistent operational definitions, and the use of procedures that have little or no empirical validity in measuring frontal lobe dysfunction. However, their conclusions of limited findings are based on their opinions of what constitutes appropriate methodology (Hawkins & Trobst, 2000). Despite their concerns regarding methodology, Kandel and Freed (1989) do admit that there does appear to be a consistent link reported in the literature between executive dysfunction and violence.

Studies acknowledging Kandel and Freed’s (1989) criticisms are limited and many studies still continue to use assessment tools that are compromised in their ability to differentiate executive or prefrontal function, e.g. the continual use of the WCST, even though there is evidence to suggest that it is not a precise executive measure (Anderson et al., 1991; Romine et al., 2004). Many studies also fail to include a healthy control group, and instead supply two or more experimental groups. Additionally, a large amount of literature over the last decade has focussed on OFC explanations of impulsivity and related this specifically to APD, obscuring the input of other functions and brain regions (see Blair, 2004; Seguin, 2004 for reviews).

Offenders have been characterised by poor executive functioning in neuropsychological examinations (Bergeron & Valliant, 2001; Gorenstein, 1982; Lueger & Gill, 1990; Moffitt, Lyman, & Silva, 1994; Spellacy, 1978; Yeudall & Fromm-Auch, 1979). However, in most of these reports it has been difficult to understand what the offender’s background or conviction was, and not all controls used were samples from the general population, with psychiatric patients of various diagnoses often comprising control sets. Further, even when clarification is provided regarding offending, sexual offenders, general assault offenders, and property offenders may be grouped together in a ‘violent offender’ category. A criticism of this grouping is that differences have been reported between ‘general’
violent offenders (property and physical assault) and sexual offenders; namely sexual offenders have demonstrated more temporal compromise upon examination whereas ‘regular’ violent offenders have demonstrated more frontal deficits (Bergeron & Valliant, 2001; Brennan & Raine, 1997).

Even accounting for methodological inconsistencies, evidence for the association of executive dysfunction with violence comes from neuropsychological studies that have implicated general ‘frontal’ dysfunction in violent samples, typically using samples from the offender population. In an early study that compared 40 violent and 40 non-violent male inmates on a 31-variable neuropsychological test battery, Spellacy (1978) found that test subjects could be classified as violent or non-violent with 95% accuracy. It appears that the performance subtests of the Wechsler Adult Intelligence Scale (WAIS), Vocabulary from the WAIS, the Maze Test, and Word Fluency were the most sensitive to differences and better able to differentiate between groups. This outcome tends to support the hypothesis that the expression of violent behaviour is associated with compromised frontal-temporal functioning or poor executive functioning and poor verbal ability. However, these results are not explanatory for criminality as many of the inmates’ scores were in the low-average to average range, which are comparable to a large number of the general population who lead adaptive and non-criminal lives. In a similar study, but using the Halstead-Reitan Neuropsychological Test Battery (HRB) in a comparative study of 86 violent criminals (75 male) and 79 (69 male) normal controls, Yeudall and Fromm-Auch (1979) found significantly more prefrontal dysfunction in the violent group.

Gorenstein (1982) sought to demonstrate specific relationships between psychopathy and frontal lobe dysfunction using a sample of 20 male psychopaths, 23 psychiatric patients, and 18 healthy college students, and a neuropsychological battery consisting of the WCST, the sequential matching memory task (SMMT), and the Necker Cube. Across all these tests the psychopathy group made substantially more errors, and overall demonstrated a pattern of dysfunction
concerning these tasks in comparison to controls and psychiatric patients. However, the groups were not matched across age and education, and comparing offender and psychiatric individuals to college students is questionable. An understanding of offending background and diagnosis of psychopathy would also have been useful, and the test battery used to define frontal dysfunction is not comprehensive enough. Several attempts have been made to replicate this finding and failed (for example, Hart, Forth, & Hare, 1990; Smith, Arnett, & Newman, 1992).

Bryant and colleagues (1984) were able to demonstrate general brain dysfunction in their sample of 110 male inmates divided into violent and non-violent groups. Using the LNNB 73% of the violent participants had brain damage compared to 28% of those classified as non-violent. In particular the violent offender group demonstrated difficulties in planning, organising, and executing goal directed behaviour. Interestingly the non-violent group included inmates who had damaged property, which is now inconsistent with modern literature which operationalises this offence as violent behaviour (Barratt & Felthous, 2003).

In an attempt to be comprehensive and Langevin et al. (1987) tested 18 murderers, 21 violent inmates, and 16 non-violent male offenders with the HRNB, LLNB, WAIS-R, CT and EEG. No significant differences in CT or EEG were demonstrated across the sample, but significant differences were seen in neuropsychological test performance. Murderers and violent offenders demonstrated an overall impairment across the tests in contrast to non-violent offenders who performed in the normal range. The authors did not attempt to link impairment to specific functions.

Lueger and Gill (1990) compared the performance of 21 male adolescent offenders diagnosed with conduct disorder and 20 matched normal controls on measures of cognitive processes associated with executive functioning. The experimental group performed more poorly on measures of conceptual ability,
perseveration, sustained attention, and sequencing on memory and motor tasks, but not on non-executive tasks, suggesting executive dysfunction underlying conduct disorder.

In a large examination of 257 inmates examined with the Psychopathy Checklist – Revised (PCL-R) and divided into psychopaths and non-psychopaths then re-examined with standard neuropsychological tests (see table 2), no tests score differences of any significance between the groups were demonstrated (Hart et al., 1990). This may be due to the arbitrary dividing of psychopaths and non-psychopaths and using this as a comparison, whereas conviction history may have been a better division. Crime history of each inmate was not reported, and it is assumed because the PCL-R was the only definer in separating groups, that each group would have included a range of inmates with a range of violent and non-violent convictions. However, even with this limitation the overall impairment rate on all tests was low, which is unusual when compared to other studies.

Many studies have attempted to link psychopathy or APD with brain dysfunction, and this in turn with violence (Hare, 1984; Hare, 1999). In Deckel, Hesselbrock, and Bauer’s (1996) review, it was concluded that the development of APD might be linked to impaired frontal lobe functioning. In their own work they demonstrated an increased likelihood of APD membership in a group of 89 males with decreased frontal lobe activation, as measured by a battery of neuropsychological tests and EEG (Deckel et al., 1996). Antisocial personality disorder or psychopathy and antisocial behaviour studies typically define this behaviour by formal diagnosis of APD, using the PCL-R or through legal descriptions, i.e., inmate samples (Morgan & Lilienfeld, 2000). Using both these descriptions to describe antisocial behaviour Morgan and Lilienfeld (2000) conducted a meta-analytic review of 39 studies using neuropsychological methods, yielding 4589 participants from the years 1958-1994, and included both adult and adolescent populations. The authors concluded that there is a robust and statistically significant relationship between antisocial behaviour and executive
dysfunction (Morgan & Lilienfeld, 2000). However, the majority of the studies reviewed did not attempt to differentiate function or brain region, so antisocial individuals may demonstrate ED but whether they are also characterized by deficits in other domains remain unresolved.

Dinn and Harris (2000) completed a comprehensive neuropsychological battery along with autonomic arousal measures and personality questionnaires on 12 community volunteers diagnosed with APD using the PCL-R and 10 healthy controls. They demonstrated that participants with APD had significantly more trouble on some of the neuropsychological tests indicating compromise on the OFC, such as the Stroop and the Object Alternation Test, however expected differences on the Go/No-Go task were not demonstrated. This study would have been strengthened by the addition of robust dPFC measures in order for the authors to make a stronger argument for specific OFC deficits in APD individuals as compared to a more comprehensive ED syndrome. It may be that poor processing related to compromised dPFC impacted on the Go/No-Go task, however with the design in this study this remains speculation.

In a study that assessed 18 male violent offenders and 17 non-violent offenders and 17 non-offenders with a variety of behaviour and personality checklists along with the Porteus Maze (PM), the violent offenders demonstrated expected ratings on the scales in paranoia and deviant behaviour and had a significantly lower test age quotient in the PM than the other groups (Valliant, Gristey, Pottier, & Kosmyna, 1999). This study used lax criteria to define the violent-offender group including conviction of physical assault, pointing a firearm, murder, impaired driving causing death or injury, and sexual assault. Similarly, the non-violent offenders included those who had convictions of breaking and entering, fraud, drug possession, and traffic offences. Large and heterogeneous categorisation such as that described removes the ability to clearly identify which particular groups of offenders are more vulnerable to the deficits being measured. In addition, the non-offender group were male university students. A criticism in
using university student as a control is the vast differences in education and, often, socioeconomic background, would increase the likelihood of finding differences between offenders and non-offenders.

In a study that set out to examine executive differences between 55 adolescent and adult offenders, Bergeron and Valliant (2001) were surprised when all offender groups, regardless of age, were significantly impaired on executive functions compared to non-offender controls. The poorest executive functions for offenders in this study were judgement and adaptation ability (Bergeron & Valliant, 2001). Unfortunately this study failed to report offender type or provide control data.

Recidivism is a large problem, both for society as a whole and the prisons, and recidivist offenders usually commit violent offences (Rich, 2000). Valliant et al. (2003) examined 12 recidivist offenders (2 or more convictions) and 12 ‘one-time’ only offenders with personality inventories and the WCST. The WCST significantly differentiated the offenders, leading the authors to suggest recidivists lack flexibility in their thinking, and that they are less likely to adequately process social information and to act-before-thinking (Valliant et al., 2003). In this study the types of offences were not listed.

Stevens, Kaplan and Hesselbrock (2003) examined men who met criteria for APD (n =34), men who did not meet criteria for APD but had CD as children (n=25), and a healthy control group (n=32). The TMT, COWA, WCST, Porteus Maze, subtests from the WAIS-III, and Luria Motor Tasks were administered. All three groups displayed average or above-average general cognitive and verbal ability, with slightly decreased verbal-abstraction scores differentiating the APD group from the others. No other executive tests differentiated groups. This may be due to the APD group being sourced from the community, and hence functioning relatively well. This study may also present evidence for the remitting of behavioural deficits in children with CD as they mature.
Retrospective data enables us to establish a link between brain damage and violent behaviour; however, it is not possible to determine in retrospective data whether the brain damage caused the violence or was a result of it. Some prospective studies have shown that aggressive behaviour and irritability are common consequences of cerebral damage. Otnow-Lewis, Moy, Jackson, and Aaronson et al. (1985) documented the childhood neuropsychiatric and family characteristics of nine male subjects, clinically evaluated as adolescents, who later went on to murder as adults. These nine were compared with twenty-four incarcerated delinquents who did not go on to murder. The authors found that severe brain damage combined with paranoid psychotic thinking, created a tendency for the nine homicidal subjects to act swiftly and severely when they felt threatened, and that this was not the case for the 24 non-murderers. Foster, Hillbrand, and Silverstein (1993) examined 23 male inpatient participants of a forensic unit with a battery of neuropsychological tests, and then monitored their behaviour for 12 months. They concluded that a substantial relationship exists between neuropsychological impairment and the frequency of violent acts committed. In particular the authors suggest that executive deficits can be used to predict which individuals are likely to be aggressive in the following year. The functions most associated with repeat incidents of violence were deficits in switching set and self-monitoring (Foster et al., 1993). Moffitt et al (Moffitt et al., 1994) reported on a cohort from Dunedin New Zealand and demonstrated that poor overall neuropsychological functioning at 13 years of age in boys was predictive of later delinquency at 18 years of age. Limited verbal ability and executive function accounted for most of the variance.

In an examination attempting to clarify the functions that serve aggression, Hoaken, Shaughnessy, and Pihil (2003) divided men and women into high and low executive function groups and provoked aggressive reactions using the Taylor Aggression Paradigm. They tested response times with the Go/No-Go tasks, and found that impulsivity was not related to aggression and executive function when using set-times on decision making tasks as a measure. Instead the authors
suggested reduced processing of social cues and an inability to access or recall social explained the results and were associated with aggressive individuals. The authors speculate that poor social processing may mediate the executive function and aggression relationship rather than impulsivity per se (Hoaken et al., 2003).

Increased levels of aggression and/or antisocial behaviour, and acts of violence, are frequently noted in people who have suffered a frontal brain injury or disease both early and later in life (Blumer & Benson, 1975; Burgess & Wood, 1990; Grafman et al., 1996). Burgess and Wood (1990) report that after serious frontal brain injury “many patients find it difficult to tolerate frustration and react impulsively to minor forms of pressure or provocation” (p.122). In much of the research for the last 30 years it is primarily damage to the medial and orbitofrontal prefrontal cortex, and the associated social and emotional changes that have been associated with latent development of aggressive characteristics (Blair, 2004; Blair & Cipolotti, 2000; Blumer & Benson, 1975; Damasio et al., 1990; Damasio et al., 1994; Grafman et al., 1996; Volavka, 1995). In particular relating this to specific explanations of APD has been popular in last 10 years.

However, there is recognition that violence is an end product of a complicated combination of proceedings, including the interplay between different brain structures, with modern literature attempting to account for the influence of the dIPFC alongside the OFC (for example; Heyder et al., 2004; Miyake et al., 2000; Seguin, 2004). Although terms such as acquired sociopathy (Damasio et al., 1990; Damasio, 1996) have been used to describe APD type traits after prefrontal injury, the individuals involved rarely meet full criteria for APD as they lack the superficial charm and poise characteristics required (Hare, 1984; Morgan & Lilienfeld, 2000). Instead the major defining characteristic is a demonstration of an inability to delay reward and difficulty in understanding their impact on others (Damasio et al., 1990; Damasio, 1996; Morgan & Lilienfeld, 2000). Task impurity and task insensitivity also makes it difficult to assume that specific brain regions
are being measured, and then to use these to account for characteristics of the violent offender.

Broadly, psychologists assume that like other behaviours, criminal behaviour is a result of the interaction between the individual and the environment (Farrington, 1994). Individual differences may cause an individual to bring with them a certain degree of what may appear as an antisocial tendency, but they are also motivated by situational factors and life circumstances (Farrington, 1991b). Crimes offer immediate gratification; they are often simple and easy to commit requiring few formal skills or little academic education. Most crimes are spontaneous, and the obvious lack of planning is consistent with them being viewed as impulsive actions (Webster & Jackson, 1997). Robberies, often spontaneous, usually net less then $100, which is soon spent or lost; this is consistent with the emphasis on short-term goals and immediate gratification often sought by those with executive dysfunction (Hart & Dempster, 1997).

According to Farrington’s theory of offending (Farrington, 1991b, 1994) impulsivity is a central construct included in the theory, with the reasoning “in general, the more impulsive a person is, the more antisocial he will be” (Farrington, 1991b; p.13). Farrington goes on to describe that, when impulsivity is combined with a limited ability to manipulate abstract concepts, low self-esteem, low academic achievement, ego-centricity, and generally low inhibitions against anti-social behaviour, there is a stronger probability of an individual offending if the ‘appropriate’ environmental circumstances are in place. Examples of ‘appropriate circumstances’ that influence offending may be money worries, peer pressure, or simply a hard-to-resist opportunity to steal when standing alone at a shop counter with desirable goods on display. Crimes can also offer ‘thrills’ which some researchers see as an abnormal form of affect regulation, injecting some excitement into an otherwise dull life where most emotions - apart from the explosive kind - are experienced in a limited manner (Hart & Dempster, 1997; Steele & Southwick, 1985; Webster & Jackson, 1997). This notion is linked to the
‘general under-arousal’ hypothesis of brain function that purports individuals with under-arousal seek out catastrophic or ‘adrenalin-charged’ situations, and/or have an inability to react to confrontational situations in ‘normal’ or appropriate ways (Raine & Buchsbaum, 1996; Raine et al., 2000; Raine et al., 1990; Scarpa & Raine, 1997). Farrington (1994) discusses how individuals deficient in conscience may be people whom for various reasons, such as the reduced autonomic reactivity seen in some APD individuals, are resistant to classical conditioning. Due to these restrictions it is possible that these people may not internalise rules as easily as others. Farrington (1994) goes on to describe how even individuals with a strong conscience may still commit crimes as they may be people who have difficulty imagining the future consequences of present actions and due to impulsivity will discount the consequences that they can anticipate. These explanations by Farrington mirror those that may be expected of individuals whose compromised executive functioning impacts on self-control, inhibition, and the ability to anticipate future consequences of their actions.

Work led by Raine and his team in 1998 demonstrated that links between psychopathy and antisocial behaviour are not as clear-cut as they may first appear. The authors state that it is up for debate whether all ‘violent psychopathic criminals’ suffer from prefrontal dysfunction as measured through neuropsychological tests, as a group classified as ‘predatory’ in their study showed fewer links with prefrontal dysfunction compared to the group labelled ‘affective’ or convicted of impulsive-type murders (Raine et al., 1998). Farrington (1991b) makes the assertion that offenders are predominantly versatile rather than “specialist.” This is reflected in the examination of the recidivistic offender histories in this study and the discovery of a range of crimes that have been committed over their “career.” It is often seen that some people now incarcerated for violent crimes began their careers with convictions for non-violent offences such as shoplifting, car theft, and substance abuse/supply. In a London longitudinal study by Farrington (1991a) he reported that 86% of violent offender participants also had convictions for non-violent crimes.
The prevalence of TBI in incarceration settings appears to be at a higher rate than that of the general community population. This may be as a result of the ‘risky’ lifestyles led by offenders. For example, a New Zealand study carried out by Barnfield and Leathem (1998) in a Waikato Prison demonstrated a lifetime rate of TBI (severity not defined) of 86% across 118 inmates’ self-reports. For Māori the incidence was even higher, at 91%, possibly reflecting the increased risk for young Māori males. In a later American study these figures of prevalence were replicated and extended by examining for psychological and neuropsychological difficulties. In a random cross-sectional design of 69 inmates in a Washington County Jail the prevalence of TBI over a lifetime was documented at 87%, with 35.2% reporting TBI within the last year (Slaughter, Fann, & Ehde, 2003). In the inmates who had sustained brain injury in the prior year, their neuropsychological scores were found to be poorer (as may have been expected) and psychiatric difficulties were more likely when compared with the inmates who did not have a recent history of TBI. The anger and aggression scores were found to be significantly higher in those inmates who sustained a TBI in the past year (Slaughter et al., 2003). A logical question that may flow from these results is raised: does the high prevalence of TBI in inmates translate directly to increased risk of offending, and can it differentiate type of offending? An interesting extension to this study may have been to record whether or not the inmate who sustained the TBI in the last year acquired it in prison or prior to the incarcerating offence, and also if neuropsychological and psychological testing separated inmates along offending lines. Further, both of the prevalence examinations (Barnfield & Leathem, 1998; Slaughter et al., 2003) would have benefited from demographically matched controls, especially to account for the larger risk of TBI noted in poorer socio-economic demographics, which are also associated with increased criminality.

Evidence for the impact on offending, resulting from a change in brain and behaviour relations following a brain injury, comes from Sarapata and his colleagues (1998). They found that 50% of non-violent convicted felons reported
a prior history of TBI and current problems in cognitive and emotional functioning. In contrast only 5% of a college sample in this investigation reported a prior TBI. In the same study, Sarapata, Herrmann, Johnson, and Aycock. (1998) also reported that 83% of felons who had described a history of TBI also gave a date for their TBI that preceded the date of their first encounter with law enforcement. Some participants reported no trouble with the law until after experiencing a TBI that occurred in their late thirties. This provides interesting information regarding the importance of a healthy and fully operational CNS in inhibiting criminal behaviour. However, the use of a college sample as a control contains some bias, as individuals who enter and remain in college are usually at a high-functioning level and it can be hypothesised that those with recent brain injuries may not currently be studying.

A recent study attempts to use early experience of TBI as an explanatory factor for later offending (Leon-Carrion & Ramos, 2003). This study separated a group of inmates into violent offenders (36) and non-violent offenders (13) and held extensive interviews to ascertain brain injury, development, and school history. The authors concluded that those men with multiple brain injuries as a child were more likely to become delinquent later on in life, with an association between severity of TBI and violent offending. However, this study is limited in its applicability due to inclusion of only 13 non-violent men who had committed ‘white-collar’ crimes used for comparison against a violent group that included physical assault, sexual assault, and homicide offenders. More men in the non-violent group that had crimes similar to the violent offender group without the violence (e.g., break and enter) would have provided closer comparison in delineating neurobehavioural aetiologies of offending. Nevertheless, even with these limitations, the finding that an increase in TBIs sustained along with the severity of those TBIs is common in the violent offenders’ backgrounds adds to neurobehavioural explanations for violence. The authors go as far to conclude that avoiding TBI during childhood would be preventative for later crime development (Leon-Carrion & Ramos, 2003).
3.3 Summary of Neurobehavioural Findings in Violence and Offending

This section has reviewed literature exploring possible contributing factors to violence and offending, including neurological, biological, psychosocial, neuropsychological and environmental variables. Particular attention has been focused on executive dysfunction explanations of offending and violence. Broadly, this literature suggests that some types of brain dysfunction may play a role in violent and criminal behaviour. Neurological dysfunctions as an exclusive cause of offending and violence cannot be assigned, as such a conclusion is too simplistic and robust evidence for this does not exist.

Much of the literature suggests that violence emerges from the presence and interaction of a number of factors. These include biological factors, abusive rearing, modelling of ‘bad’ behaviour, and a lack of ameliorating or supportive factors. Particular regions and circuitry of the brain are implicated in the generation of ED and aggressive dyscontrol. Damage to the OFC and dPFC accompanied by possible compromise in the sub-serving anatomy, particularly the amygdala, appears to result in poor decision-making and self-regulation, and a tendency to respond more aggressively to a perceived threatening environment. Due to individual factors and various mitigating elements of each person’s environment, the ‘worst case scenario’ will not always occur. However, even if an individual raised with the developmental constraints of brain damage and aversive experiences never resorts to violence, if no moderating factors are present, it may be speculated that these factors impact at a less overt level.

The role of executive dysfunction in violence and offending is supported by evidence from alcohol studies. It is understood that intoxication lowers inhibitions by dampening down regulatory controls. The pervasive involvement of alcohol in violent crime suggests it facilitates aggression by weakening inhibitors; that is,
turning off the prefrontal cortex. It is suggested that the prefrontal area is anaesthetised by acute intoxication; thus the intoxicated person is experiencing something akin to executive dysfunction. The research from intoxication and substance abuse studies may aid in the understanding of appropriate inhibition and insight (two key behaviours often disrupted in executive dysfunction), and any links these may have to violence. They may also provide further evidence for the link between the prefrontal cortex and the limbic system, and especially the role the prefrontal cortex plays as a ‘gatekeeper.’ From the work that examines ingestion of alcohol (Amen, 1999; Giancola, 1995; Giancola & Zeichner, 1994, 1997; Hoaken, Assaad, & Pihl, 1998; Hoaken, Giancola et al., 1998; Peterson et al., 1990) it appears that depressing the actions of the prefrontal cortex reduces the range of self-checking behaviours and allows the more impulsive and aggressive behaviours associated with the limbic system to be expressed.

In the preceding section biological factors and environmental/psychosocial factors were examined separately. In reality these are closely linked due to the neurodevelopmental nature of the brain, and greatly influence each other in the day-to-day activation of the brain and consequent behaviour. After acquired brain damage the interaction of these factors is more evident, due in part to the additional difficulties imposed by an impaired brain, alongside possible changes in reactivity to the environment. The relationship between violence and brain injury is more complex, and probably more reciprocal, then simple cause or effect. In the face of organic, environmental, and social information regarding violence it seems that the rhetoric of chicken and eggs can be misleading. It can sometimes be that the egg (violence) comes first but at other times the chicken (brain dysfunction) takes precedence. As Herculano-Houzel (2003) stated in attempting to unravel explanations for complex brain-behaviour relationships, nature, the brain, and society cannot often be dissociated, as after all, the chicken and the egg are in fact one and the same animal.
The relationship between aggression and poor executive function has been documented throughout the literature (Grafman et al., 1996; Hawkins & Trobst, 2000; Krakowski, 1997; Stoff & Cairns, 1996), often through linking general brain dysfunction with aggression (Grafman et al., 1996; Krakowski, 1997; O'Leary, 2000; Otnow-Lewis, 1983; Otnow-Lewis et al., 1985; Otnow-Lewis et al., 1986), or implicating frontal components in imaging studies (Raine & Buchsbaum, 1996; Raine, Buchsbaum et al., 1997; Raine et al., 1994; Raine et al., 2000; Raine et al., 1998). Associations have also been demonstrated in ‘normal’ subjects in studies with alcohol (Giancola & Zeichner, 1997)), drug abuse (Fishbein, 2000), and self-harm or suicide (Lindberg et al., 2000; Lindberg et al., 1985), as well as interpersonal aggression and violent crime (for example Cherek, Moeller, Dougherty, & Rhoades, 1997; Cherek, Tcheremissine, Lane, & Pietras, 2003; Scarpa & Raine, 1997; Wang & Diamond, 1999).

Points of controversy in the literature are those explanations that rely solely on lesions in the OFC to describe aggressive dyscontrol and/or impulsivity. Explanations involving holistic accounts of the brain and functions, in particular the manner in which the OFC (and subserving structures) works with the dlPFC, in explaining aggressive dyscontrol are becoming more widely accepted. In attempting to make putative connections in what brain structures underlie aggression, the OFC has been implicated in impulsivity. Attempts have been made to link poor OFC to impulsivity and to increases in aggression or more generally, antisocial personality disorder (Blair, 2004; Rolls, 1996; Seguin, 2004). Executive functions related to dlPFC have also been indicated in underlying violence, particularly deficits in planning and monitoring of behaviour (Grafman et al., 1996; Hawkins & Trobst, 2000; Seguin, 2004). Converging empirical dlPFC and OFC evidence states that impulsivity resulting from reduced OFC function alone is not explanatory for violence (Krakowski, 2003), and that increased propensity for impulsivity is not empirically linked with violence if impulsivity is the only difficulty (Seguin, 2004). Instead it appears to be compromised OFC combined with poor dlPFC input (in particular the executive
Review of Literature

functions of adapting and regulating goals; planning behaviour; co-ordination of attention; mental flexibility; temporal sequencing; self-monitoring; working memory; and the ability to use feedback), that may engender vulnerability or exacerbate a propensity for aggressive dyscontrol. Rather than aggression being mediated through impulsivity resulting from dysregulation of the OFC, instead the mediator is poor overall executive functioning served by a dysfunctional dlPFC. This complements the explanatory mechanism of CE of SAS in controlling executive functions, and the use of the BADS in examination of ED in the sample of interest in this thesis.

3.4 Methodological Flaws of Existing Reviewed Literature

In all studies there will always be methodological issues and limitations. This may be particularly true in studying neurobehavioural aspects of violence and offending due to the varied nature of violence and the individuals that offend. The multifactorial nature of violence makes simple correlations between brain dysfunction and a violent act rarely possible (Filley et al., 2001). Further limiting methodology are the pragmatics involved in researching within a prison or forensic psychiatric environment.

Many of the studies reviewed use a small number of participants reducing the strength of conclusions drawn, and when a study used mixed offenders or psychiatric patients for within group comparisons, the results became even less robust. Part of the difficulty in generalising results or comparing across studies is that investigators of violence and offending can fail to consistently operationalise variables, and often vary in they way they measure these. Violence has been defined as including offences from property damage to rape to serial killing, sometimes all in one sample. The obvious differences that exist between damaging property and raping, for example, also make it difficult to suggest consistent neuroanatomical underpinnings of violence. Previous studies have
demonstrated that sexual offending is considered different classification-wise and is associated with different neuroanatomical deficits (Raine & Buchsbaum, 1996).

Another criticism is the lack of controls used in a vast number of studies, or the inappropriate use of clinical or psychiatric populations as controls, hindering the application of the experimental data. Also, when community controls have been used, they are often university students that haven’t been matched for background variables such as education. Frankly, it is assumed that university students have distinct characteristics be from prison inmates, who have typically left school early with minimal qualifications (Rich, 2000).

Studies that use APD as the defining characteristic may also be limited by inconsistent definitions of APD. Selection of participants may not be based on solid behavioural evidence and instead may reflect membership of a population, with no evidence of those individuals meeting strict operational definitions of violence beyond an arbitrary definition of APD adopted for that particular study.

Unreliable, problematic, invalid, and inconsistent diagnostic procedures have been used in many of the studies reviewed. Tests used to define executive dysfunction vary from study to study, and many studies do not attempt to differentiate functions and instead use an overall level of brain dysfunction as a covariate with violence and/or offending. Further, many studies lack crucial details for proper interpretations, such as the type of offending, and how violence was operationalised. Problematic diagnostic procedures is also a criticism of imaging studies, with the majority of studies using different methods for quantifying what it is they are measuring (Hoptman, 2003). The brain regions of interest in violence are currently amongst the hardest to image and imaging techniques that will usefully elucidate the diversity of the prefrontal cortex is currently not available (Hoptman, 2003).
Table 2: Neurobehavioural Evidence for Brain Dysfunction in Violent or Antisocial Individuals

<table>
<thead>
<tr>
<th>Reference</th>
<th>Participants</th>
<th>Methods</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stafford-Clark, B., &amp; Taylor, F. H. (1949)</td>
<td>64 murderers (6 females) classified into five groups according style of murder</td>
<td>EEG</td>
<td>Motiveless crime was correlated with the most EEG abnormality. Brain region not specified.</td>
</tr>
<tr>
<td>Hill, D., &amp; Pond, D. A. (1952)</td>
<td>100 mixed crime inmates (aggressive assault, murderer, robbery, and non-aggressive crimes)</td>
<td>EEG</td>
<td>70% of murderers and 65% of aggressive psychopaths had significant excesses of bilateral theta activity compared to non-violent inmate. Not specified to regions</td>
</tr>
<tr>
<td>Williams, D. (1969)</td>
<td>1250 recidivist violent inmates</td>
<td>EEG</td>
<td>65% of the sample displayed various abnormal EEG recordings, particularly in the fronto-temporal areas</td>
</tr>
<tr>
<td>Spellacy, F. (1978)</td>
<td>40 violent and 40 non-violent male prisoners</td>
<td>31-variable neuropsychological test battery and the MMPI</td>
<td>Test subjects classified as violent or non-violent with 95% accuracy with the neuropsychological battery alone, with executive tests showing the strongest differences. The MMPI alone classified 79%</td>
</tr>
<tr>
<td>Yeudall, L. T., &amp; Fromm-Auch, D. (1979)</td>
<td>86 violent criminals (75 male) and 79 (69 males) normal controls</td>
<td>Halstead-Reitan Neuropsychological Test Battery (HRB)</td>
<td>Significantly more anterior frontal dysfunction in the violent group</td>
</tr>
<tr>
<td>Author(s)</td>
<td>Subjects</td>
<td>Measures</td>
<td>Findings</td>
</tr>
<tr>
<td>---------------------------------</td>
<td>---------------------------------------------------------------------------</td>
<td>-----------------------</td>
<td>------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Gorenstein, E. A. (1982)</td>
<td>20 male psychopaths, 23 psychiatric patients, 18 healthy college students</td>
<td>WCST, SMMT, Necker Cube</td>
<td>Psychopaths demonstrated pattern relative to ‘frontal’ deficits compared to other groups</td>
</tr>
<tr>
<td>Volkow, N. D., &amp; Tancredi, L. R. (1987)</td>
<td>Four psychiatric patients with histories of irrational and repetitive violence</td>
<td>EEG, CT, PET</td>
<td>Decreased metabolism (PET) in left temporal lobe (TL) and cortical slowing (EEG) in anterior TL for all four participants; two patients had frontal atrophy (CT) and hypometabolism of the FL, these two also claimed “no remorse”</td>
</tr>
<tr>
<td>Hart, S. D., Forth, A. E., &amp; Hare, R. D. (1990)</td>
<td>90 psychopathic criminals and 167 non-psychopathic criminals</td>
<td>COWA, TMT, VRT, AVLT, VOT, WRAT-2, Block Design and Vocabulary from WAIS-R</td>
<td>Minimal impairment seen in test scores for majority of subjects and no difference in scores between groups</td>
</tr>
<tr>
<td>Peterson, J. B., Rothfleisch, J., Zelazo, P. D., &amp; Pihl, R. (1990)</td>
<td>72 male university students 18-34</td>
<td>Administered low, moderate, high does on alcohol and placebo; PMT, REY-Copy and Recall, TWFT, SOP, WCST, LM, PA, AQ, ASTVN, Y-PM, PR, RT</td>
<td>High doses of alcohol detrimentally affect a number of functions associated with the prefrontal cortex and the temporal lobe, especially planning, verbal fluency, memory and complex motor control</td>
</tr>
<tr>
<td>Raine, A., Venables, P. H., &amp; Williams, M (1990)</td>
<td>101 males tested at 15 years of age then again at 24</td>
<td>Arousal, skin conductance and EEG measures</td>
<td>Relationship between lowered central and autonomic measures of arousal at age 15 years and criminality at age 24 years in subjects with offending history compared with subjects without offending history</td>
</tr>
<tr>
<td>Author(s)</td>
<td>Study Details</td>
<td>Methodology</td>
<td>Findings</td>
</tr>
<tr>
<td>-----------</td>
<td>---------------</td>
<td>-------------</td>
<td>----------</td>
</tr>
<tr>
<td>Damasio, A., Tranel, D., &amp; Damasio, H. (1990)</td>
<td>Five adults with damage to ventromedial frontal cortices, including patient EVR matched against 5 ‘normal’ controls</td>
<td>Electrodermal recordings, all patients had previous neuropsychological assessments and scans</td>
<td>EVR is described prototypical of the ‘acquirement’ of sociopathic behaviour and failure to follow cues after damage to the ventromedial frontal cortex; somatic autonomic responses to social stimuli are abnormal, but unconditional ‘surprise’ elements (e.g., loud noise) produce normal autonomic responses</td>
</tr>
<tr>
<td>Tonkonogy, J. M. (1991)</td>
<td>89 forensic psychiatric patients</td>
<td>CT and MRI</td>
<td>Patients with frequent violent behaviour had lesions in the anterior-inferior temporal lobes</td>
</tr>
<tr>
<td>Convit, A., Czobor, P., &amp; Volavka, J. (1991)</td>
<td>21 right-handed males (21-53 years)</td>
<td>EEG compared against observations of violence, medication, and interventions required</td>
<td>Violence was significantly related to the hemispheric asymmetry in EEG for the frontotemporal derivations</td>
</tr>
<tr>
<td>Oder, W., Goldenburg, G., Spatt, J., Podreka, I., Binder, H., &amp; Deecke, L. (1992)</td>
<td>36 participants with severe TBI (5 female) examined an average of 40 months post-TBI</td>
<td>SPECT</td>
<td>Anterior hypoperfusion in aggressive subjects</td>
</tr>
<tr>
<td>Gantner, A. B., &amp; Taylor, S. P. (1992)</td>
<td>40 male undergraduates over 18 years old</td>
<td>Randomly assigned participants to alcohol/threat, alcohol/no-threat, placebo/threat, placebo/no-threat conditions; TAP; provocation/non-provocation</td>
<td>Under conditions of low provocation intoxicated participants behaved more aggressively than non-intoxicated in both the threatening and non-threatening conditions; only intoxicated participants increased aggression in the threatening and non-threatening conditions under increasing provocation</td>
</tr>
<tr>
<td>Study</td>
<td>Sample Characteristics</td>
<td>Assessment Methods</td>
<td>Results</td>
</tr>
<tr>
<td>-----------------------------------------------</td>
<td>----------------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Foster, H. G., Hillbrand, M., &amp; Silverstein, M. (1993).</td>
<td>23 male forensic psychiatric patients</td>
<td>Assessment with JOL, SCWT, SDMT, TONI, WCST, EPT, OAS prior to following aggression pattern for one year</td>
<td>Executive deficits predicted those individuals that would commit more violent acts in the following year</td>
</tr>
<tr>
<td>Giancola, P. R., &amp; Zeichner, A. (1994)</td>
<td>72 white-male community sample 18-32 years</td>
<td>Laboratory provoked aggression using modified version of TAP, SOP, CAT, abbreviated WAIS-R</td>
<td>Aggressive subjects significantly poorer performance on CAT; SOP showed no sensitivity</td>
</tr>
<tr>
<td>Raine, A., Buchsbaum, M. S., Stanley, J., Lottenburg, S., Abel, L., &amp; Stoddard, J. (1994)</td>
<td>22 murderers pleading not guilty by reason of insanity matched with 22 normal controls, each group contained 3 participants with schizophrenia</td>
<td>PET</td>
<td>When compared with controls significant reduction of prefrontal glucose in the left frontal cortex in murderers without schizophrenia was noted; participants with schizophrenia demonstrated bi-lateral frontal deficits</td>
</tr>
<tr>
<td>Goyer, P. F., Andreason, P. J., &amp; Semple, W. E. (1994)</td>
<td>17 patients with DSM – III personality disorders (PD) and 43 controls</td>
<td>Injection of fluorodeoxyglucose (FDG) PET, self-reported ‘impulsive aggression’ on modified aggression scale (MAS), CPT to assess frontal function</td>
<td>Significant inverse relationship demonstrated between scores on MAS and reduction in orbitofrontal glucose metabolism in PD participants; no differences on CPT between PD participants and controls</td>
</tr>
<tr>
<td>Authors</td>
<td>Study Description</td>
<td>Methods</td>
<td>Results</td>
</tr>
<tr>
<td>---------</td>
<td>-------------------</td>
<td>---------</td>
<td>---------</td>
</tr>
<tr>
<td>Wong, M. T. H., Lumsden, J., Fenton, G. W., &amp; Fenwick, P. B. C. (1994)</td>
<td>Retrospective exam of 372 forensic mental hospital patients</td>
<td>EEG, CT, WAIS, violence ratings of the institution</td>
<td>Increased frequency of temporal abnormalities noted in the patients rated as most violent</td>
</tr>
<tr>
<td>Lau, M. A., Pihl, R. O., &amp; Peterson, J. B. (1995)</td>
<td>48 male “social drinkers” community sample 18-40; 24 assessed as good frontal functioning, and 24 assessed with poor frontal functioning</td>
<td>Laboratory provoked aggression using TRT, SOP, CAT, abbreviated WAIS-R, sober condition and alcohol condition</td>
<td>Increased intoxication associated with increased aggression; increased provocation increased aggression, especially in intoxicated subjects; individuals in lower frontal functioning group significantly more aggressive when provoked</td>
</tr>
<tr>
<td>Blake, P. Y., Pincus, J. H., &amp; Buckner, C. (1995)</td>
<td>31 murderers</td>
<td>Neurological, neuropsychological, MRI and CT exams</td>
<td>Neurologic examination revealed evidence of &quot;frontal&quot; dysfunction in 20 participants. Temporal lobe abnormality indicated in nine murderers. Neuropsychological testing revealed abnormalities in all participants tested. There were EEG abnormalities in eight of the 20 participants tested, consisting mainly of bilateral sharp waves with slowing. There were MRI or CT abnormalities in nine of the 19 participants tested, consisting primarily of atrophy and white matter changes in the anterior regions.</td>
</tr>
<tr>
<td>Volkow, N. D., Tancredi, L. R., Grant, C., Gillespie, H., Valentine, A., &amp; Mullani, N. (1995)</td>
<td>Eight psychiatric patients with records of repetitive violence compared against eight healthy controls from the community</td>
<td>FDG and resting PET</td>
<td>Repeatedly violent patients demonstrated significant reduction in bi-lateral prefrontal and medial temporal regions</td>
</tr>
<tr>
<td>Authors</td>
<td>Study Details</td>
<td>Tasks and Methods</td>
<td>Results</td>
</tr>
<tr>
<td>---------</td>
<td>---------------</td>
<td>-------------------</td>
<td>---------</td>
</tr>
<tr>
<td>La Pierre, D., Braun, C. M. J., &amp; Hodgins, S. (1995)</td>
<td>30 male psychopathic inmates (diagnosed with PCL) and 30 male non-psychopathic inmates as controls</td>
<td>Visual go/no-go task, PMT, WCST, mental rotation task, modular smell identification task</td>
<td>Subjects with psychopathy significantly impaired on visual go/no-go, PMT errors and olfactory identification; WCST and mental rotation no different from controls</td>
</tr>
<tr>
<td>Amen, D. G., Stubblefield, M., Carmichael, B., &amp; Thisted, R. (1996)</td>
<td>40 (10 females) medication free aggressive psychiatric patients matched with 40 non-aggressive psychiatric patients</td>
<td>Resting SPECT</td>
<td>60% of aggressive group revealed decreased activity in prefrontal cortex opposed to only 35% of non-aggressive group; left-sided increase in activity in aggressive group particularly in basal ganglia and/or limbic system; no significant sex differences observed</td>
</tr>
<tr>
<td>Grafman, J., Schwab, K., Warden, D., Pridgen, A., Brown, H. R., &amp; Salazar, A. M. (1996)</td>
<td>57 normal controls and 279 Vietnam veterans who had suffered penetrating head injuries matched for age, education, and time in Vietnam; interviews with family</td>
<td>40 hours of clinical evaluation including EEG, BDI, NBRS, KAS, VHIS family questionnaire, and self report on violence, Violence Scales</td>
<td>Men with focal ventromedial FL lesions had significantly higher rates of aggressive and violent behaviour than non-head injured controls or veterans with lesions elsewhere in the brain</td>
</tr>
<tr>
<td>Deckel, A. W., Hesselbrock, V., &amp; Bauer, L. (1996)</td>
<td>89 men aged 21-25 from a community sample who were non-diagnosed but likely to have antisocial/adventurous traits</td>
<td>WCST, COWAT, PMT, TMT, LNMT, EEG, psychiatric interview based on DSM-III criteria</td>
<td>PMT VII Maze and LNMT added significantly to APD membership; Decrease in left vs. right frontal EEG activation in APD; WCST, COWAT, TMT did not significantly predict APD membership</td>
</tr>
<tr>
<td>Author(s)</td>
<td>Description</td>
<td>Methods/Imaging Techniques</td>
<td>Findings</td>
</tr>
<tr>
<td>------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Intrator, J., Hare, R., Stritzke, P., Brightswein, K., Dorfman, D., Harpur, T., Bernstein, D., Handelsman, L., Schaefer, C., &amp; Keilp, J. (1997)</td>
<td>Male patients in drug treatment divided into psychopath (8) and non-psychopath (9) compared with healthy controls (9)</td>
<td>PCL-R, SPECT, and a lexicon task</td>
<td>Psychopathic subjects exhibited increased rCBF in more anterior areas for longer when processing emotional words</td>
</tr>
<tr>
<td>Wong, M. T. H., Fenwick, P. B. C., Lumsden, J., Fenton, G. W., Maisey, M., Lewis, P., &amp; Badawi, R. (1997)</td>
<td>Male psychiatric violent offenders divided into repetitive violence (17) and non-repetitive violence (14) history compared with 6 healthy controls</td>
<td>PET</td>
<td>Reduced glucose uptake in both hemispheres in the anterior temporal lobes in the group with schizophrenia, with significantly less uptake in the left anterior temporal lobe in the repetitively violent patients</td>
</tr>
<tr>
<td>Seidenwurm, D., Pounds, T., Globus, A., &amp; Valk, P. (1997)</td>
<td>7 violent offenders 9 controls complaining of minor ‘vague’ neurological symptoms</td>
<td>EEG, MRI and FDG PET</td>
<td>Hypoperfusion in the medial temporal lobes in violent offenders compared with control subjects</td>
</tr>
<tr>
<td>Evans, J. R., &amp; Park, N.-S. (1997).</td>
<td>20 men convicted of murder compared with normative database</td>
<td>EEG</td>
<td>17/20 murderers had significantly abnormal scores when compared with norms. A high incidence of anterior abnormality and right hemisphere abnormality demonstrated</td>
</tr>
<tr>
<td>Study</td>
<td>Participants</td>
<td>Methods</td>
<td>Findings</td>
</tr>
<tr>
<td>----------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Raine, A., Buchsbaum, M. S., &amp; LaCasse, L. (1997) and follow up by</td>
<td>41 court referred individuals (2 females) charged with murder or manslaughter all pleaded “not guilty by reason of insanity” or as “not fit to stand trial”, sex matched to controls</td>
<td>FDG then scanning with PET following performing the Continuous Performance Task</td>
<td>“murderers” demonstrated significant bilateral metabolic decreases in prefrontal cortex and sub-cortical regions including superior parietal gyrus, left angular gyrus, and corpus callosum; prefrontal hypometabolism was more severe in ‘affective’ murderers</td>
</tr>
<tr>
<td>Raine, A., Meloy, J. R., Bihrlle, S., Stoddard, J., LaCasse, L., &amp;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sakuta, A., &amp; Fukushima, A. (1998)</td>
<td>30 ‘severe’ murderers (including mass murder and aggravated murder) compared with 39 ‘other violent’ offenders (including ‘simple murder,’ theft, arson, and drug offences)</td>
<td>EEG, MRI, CT</td>
<td>EEG significantly differentiated the groups, with 78% severe murderers showing abnormality compared with 26% in the other group. Although morphological abnormalities were demonstrated in 56% of the severe group compared with 27% of the other group these differences were not significant.</td>
</tr>
<tr>
<td>Kiehl, K. A., Hare, R. D., Liddle, P. F., &amp; McDonald, J. J. (1999)</td>
<td>11 psychopath inmates and 10 non-psychopath inmates</td>
<td>EEG and a visual oddball task</td>
<td>Reduced P300 responses in criminal psychopaths during a visual oddball task</td>
</tr>
<tr>
<td>Amen, D. G. (1999)</td>
<td>Case study on 20 year old male who became violent on may occasions only after becoming intoxicated</td>
<td>Resting SPECT when alcohol free and resting SPECT when intoxicated performed several days apart – the intoxication condition</td>
<td>Marked hyperactivity in right and left lateral FL, parietal lobes and cingulated gyrus when sober; Marked dampening effect whole brain but particular hypo-activity in frontal cortex, excessive activity in the anterior cingulated gyrus</td>
</tr>
<tr>
<td>Study</td>
<td>Group Description</td>
<td>Measures</td>
<td>Findings</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-----------------------------------------------------------------------------------</td>
<td>-----------------------------------------------</td>
<td>---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Anderson, S. W., Bechara, A., Damasio, H., Tranel, D., &amp; Damasio, A. R. (1999)</td>
<td>2 patients with prefrontal lesions acquired before 16 months of ages compared with 6 patients with adult acquired prefrontal lesions</td>
<td>WAIS-R, RVALT, WRAT-R, COWAT, WCST, TOH and JOL, Gambling Task, skin conductance, moral reasoning questionnaires</td>
<td>Similar neuropsychological profiles in both groups with both groups characterised by executive dysfunction. More difficulties with moral reasoning and judgement in patients with lesions acquired in infancy and more behavioural and social problems throughout the lifespan</td>
</tr>
<tr>
<td>Aigner, M., Eher, R., Fruehwald, S., Frottier, P., Gutierrez-Lobos, K., &amp; Dwyer, S. M. (2000)</td>
<td>82 (52 sex offenders) male forensic patients separated into high violence and low violence groups</td>
<td>MRI</td>
<td>Unspecified abnormalities demonstrated across the groups with 65.5% of the high violent offenders demonstrating abnormalities compared with 16.6% of the low violent offender group</td>
</tr>
<tr>
<td>Dinn, W. M., &amp; Harris, C. L. (2000)</td>
<td>12 APD community volunteers and 10 healthy community controls</td>
<td>Personality measures, skin conductance and heart rate measures, Stroop, COWA, Go/No-Go, Object Alternation Task</td>
<td>APD showed strong difference in the OAT which the authors claim is strong predictor of OFC dysfunction and parts of the Stroop, but no differences in another purported OFC task Go/No-Go</td>
</tr>
<tr>
<td>Blair, R. J. R., &amp; Cipolotti, L. (2000)</td>
<td>56 year old male (JS) with trauma sustained to bilateral FL and diagnosed (DSM-IV) with APD since injury compared with a 46 year old male (CLA) with a motor neuron disease attacking right FL who has severe dysexecutive troubles without Complex assessment including interviews and tests of affect, social functioning and diffuse brain functioning. Of interest to this thesis, administered were shortened WAIS-R, ‘frontal battery’ consisting of CET, WCST, HSC, Stroop</td>
<td>Following bilateral trauma to OFC JS presents with severe behavioural disturbance and profound impairment on a range of executive tests, he fulfils diagnosis for APD which the authors call ‘acquired sociopathy’ as aberrant behaviour not present before trauma; authors speculate that JS’s inability to generate expectations of others reactions in response to his own is due in part to damage to the orbitofrontal cortex; CLA has ED but not the aberrant behaviour who has right FL damage with sparing of the orbitofrontal cortex</td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Participants</td>
<td>Test</td>
<td>Imaging</td>
</tr>
<tr>
<td>-------</td>
<td>--------------</td>
<td>------</td>
<td>---------</td>
</tr>
<tr>
<td>Soderstrom, H., Tullberg, M., Wikkeslo, C., Ekholm, S., &amp; Forsman, A. (2000)</td>
<td>21 perpetrators of impulsive violent crimes (1 woman) aged 16 – 51, 11 healthy controls</td>
<td>Test, TMT, Verbal Fluency, WCFST</td>
<td>SPECT, rCBF and MRI taken pre-trial</td>
</tr>
<tr>
<td>Critchley, H. D., Simmons, A., Daly, E. M., Russell, A., van Amelsvoort, T., Robertson, D. M., Glover, A., &amp; Murphy, G. M. (2000)</td>
<td>13 mildly retarded and repetitively violent male patients and 14 (4 females) healthy controls</td>
<td>Magnetic resonance spectroscopy to measure concentrations of N-acetyl aspartate, creatine phosphocreatine and choline-related compounds</td>
<td>Increased activation in the amygdala and dIPFC in APD groups compared to controls when processing and accessing emotional lexicons</td>
</tr>
<tr>
<td>Schneider, F., Habel, U., Kessler, C., Posse, S., Grodd, W., &amp; Muller-Gartner, H. (2000)</td>
<td>12 men with APD and 12 matched healthy controls</td>
<td>fMRI</td>
<td>APD significant 11% reduction in prefrontal grey matter compared with healthy controls, 13.9% reduction when compared with substance abusers, and 14% reduction when compared with psychiatric controls; APD group also showed reduced autonomic activity under stress conditions</td>
</tr>
<tr>
<td>Raine, A., Lencz, T., Bihrle, S., LaCasse, L., &amp; Colletti, P. (2000)</td>
<td>21 community volunteers with APD, compared against 34 healthy community participants, 26 substance dependent men and 21 psychiatric controls</td>
<td>Prefrontal grey and white matter volumes assessed with MRI, autonomic activity monitored</td>
<td>Increased activation in the amygdala and dIPFC in APD groups compared to controls when processing and accessing emotional lexicons</td>
</tr>
<tr>
<td>Study Authors (Year)</td>
<td>Participants &amp; Conditions</td>
<td>Measures</td>
<td>Findings</td>
</tr>
<tr>
<td>----------------------</td>
<td>---------------------------</td>
<td>----------</td>
<td>----------</td>
</tr>
<tr>
<td>Houston, R. J., &amp; Stanford, M. S. (2001)</td>
<td>15 aggressive and impulsive participants with 15 controls</td>
<td>EEG and evoked potentials</td>
<td>Those with impulsive aggression exhibited inefficient EEG activity in gating behaviour and poor inhibition</td>
</tr>
<tr>
<td>Kiehl, K. A., Smith, A. M., Hare, R. D., Mendrek, A., Forster, B. B., Brink, J., &amp; Liddle, P. F. (2001)</td>
<td>8 criminal psychopaths and 8 non-criminal psychopaths alongside 8 matched healthy controls</td>
<td>fMRI of affective processing task</td>
<td>Limbic abnormalities in affective processing demonstrated in criminal psychopaths only</td>
</tr>
<tr>
<td>Laakso, M. P., Gunning-Dixon, F., Vaario, O., Repo-Tiikonen, E., Soininen, H., &amp; Tiikonen, J. (2002)</td>
<td>24 male violent offenders and 33 control subjects</td>
<td>PCL-R and fMRI</td>
<td>APD individuals demonstrate left frontal attenuation but authors believe this is accounted for by alcoholism</td>
</tr>
<tr>
<td>Stevens, M. C., Kaplan, R. F., &amp; Hesselbrock, V. M. (2003)</td>
<td>34 men with APD, 25 men who had CD as children but do not have current APD diagnoses, and a healthy control group (32 men)</td>
<td>TMT, COWAT, WCST, PM, subtests from the WAIS-III, and Luria Motor Tasks were administered.</td>
<td>Verbal-abstraction scores only differentiated the APD group from the others. No other executive tests differentiated groups</td>
</tr>
<tr>
<td>Stanford, M. S., Houston, R. J., Villemarette-Pittman, N. R., &amp; Greve, K. W. (2003)</td>
<td>14 males demonstrating premeditated violence and 14 non-violent healthy male controls</td>
<td>Personality and impulsivity measures, WCST, COWA, PPVT-R, TMT, RFF, EEG with auditory task</td>
<td>Differences on personality tests and failure to maintain set on the WCST between premeditated aggressors and controls. Some latency noted in responding demonstrated with EEG</td>
</tr>
<tr>
<td>Leon-Carrion, J., &amp; Ramos, F. J. C. (2003)</td>
<td>36 violent offenders (mixed physical assault and sexual assault) and 13 ‘white-collar’</td>
<td>Interviews and brain injury questionnaire</td>
<td>Violent offender had more juvenile TBI’s and more behaviour problems at school than ‘white-collar’ offenders</td>
</tr>
<tr>
<td>Gordon, H. L., Baird, A. A., &amp; End, A. (2004).</td>
<td>20 male college students</td>
<td>fMRI and a psychopathy questionnaire</td>
<td>Reliance on increased right dorsolateral prefrontal activation in participants that scored highly for psychopathy when performing an emotion recognition task compared to normals</td>
</tr>
<tr>
<td>Kroner, D. G., Forth, A. E., &amp; Mills, J. F (In Press)</td>
<td>110 adult offenders convicted of non-sexual offences</td>
<td>PCL-R, BPI, Strait Trait Anger Scale, Novaco Anger Scale, WRAT-R</td>
<td>Response latency measured and even when psychopathic individuals are able to report on affective dimensions the processing remains dysfunctional</td>
</tr>
</tbody>
</table>

APD = Anti Social Personality Disorder; AQ = Apraxia Questionnaire; ASTVN = Albert’s Simple Test of Visual Neglect; BDI = Beck Depression Inventory; CAT = conditional associative learning; CET = Cognitive Estimates Test; COWAT = Controlled Oral Word Association Test; CT = computed tomography; DSM-III = Diagnostic Statistical Manual; EEG = electroencephalographic testing; EPT = Emotion Perception Test; FL = frontal lobe; HSCT = Hayling Sentence Completion Test; JOL; Judgment of Line task; KAS = Katz Adjustment Scale; LM = Logical Memory; LNMT = Luria Nebraska Motor Test; MMPI = Minnesota Multiphasic Personality Inventory; MRI = magnetic resonance imaging; NBRS = Neurobehavioral Rating Scale; OAS = Overt Aggression Scale; PA = Paired Associates; PCL-R = Psychopathy Checklist Revised; PET = positron emission tomography; PM = Porteus Maze Test; PR = Pursuit Rotor; rCBF = regional cerebral blood flow; REY = Rey-Osterreith Complex Figure; RT = Reaction Time; RVALT; Rey Verbal Auditory Learning Test; SDMT = Single Digit Modalities Test; SOP = Self Ordered Pointing Task; SPECT = Single Photon Emission Computed Tomography; SCWT = Stroop Colour Word Test; TAP = Taylor Aggression Paradigm; TMT = Trial Making Test; TOH = Tower of Hanoi; TOL = Tower of London; TONI = Non-verbal Test of Intelligence; TWFT = Thurstone Word Fluency Test; VHS = Vietnam Head Injury Study; WAIS-R = Wechsler Adult Intelligence Scale – Revised; WCFST = Weigl Colour Form Sorting Test; WCST = Wisconsin Card Sorting Test; WRAT-R = Wide Range Achievement Test – Revised; Y-PM = Young-Phil Memory.
4

Study One: Violent and Non-Violent offenders, and Non-Violent Community Controls

The self is not something ready-made, but something in continuous formation through choice of action.

John Dewey educator, pragmatist philosopher, & psychologist (1859 - 1952)

4.1 Introduction

Violent offending constitutes a critical societal problem. The development of a theoretical conceptualisation of violent offending is essential to enable the formation of informed and effective rehabilitative interventions. With the exception of a small number of studies, research attempting to link ED and violence has been compromised by inadequate methodology. This difficulty has reduced the robustness of the findings.

This study attempts to address several methodological limitations identified in the literature. Specifically, this study clearly defines violence and uses this definition for selection of participants into violent and non-violent categories, uses a comprehensive neuropsychological battery to assess executive function, and utilises a demographically matched control group of non-violent community subjects. This current study proposes that deficits in executive function, measured
primarily through testing with the Behavioural Assessment of the Dysexecutive Syndrome (BADS), contribute to and maintain a tendency toward violent offending.

The specific hypotheses of Study One are provided in the Methods that follow.

**4.2 Method**

**4.2.1 Aims and Hypotheses**

The aims of the study\(^1\) are:

1. To critically review the literature linking impaired executive functioning to increased expressed violence; this has been completed in the previous chapter.

2. To investigate whether or not executive dysfunction (ED) is prevalent in a prison sample.

3. To determine if ED is more frequent in inmates (or recent inmates serving probation) convicted of violent crimes than in inmates convicted of non-violent crimes and matched non-violent community control participants.

---

\(^1\) Ethics approval from The University of Auckland Human Subjects Ethics Committee was received for this research under the reference 00/67.
The hypotheses to be tested are:

I. A significantly higher proportion of inmates with a history of violent offending will display executive deficits as demonstrated by neuropsychological assessment, than inmates whose offending is non-violent, and non-violent community controls.

II. Qualitative observations of offenders’ behaviours will reflect their scores on their neuropsychological tests of executive functioning; that is, those who behave inappropriately during testing will have lower neuropsychological scores on executive function tests.

4.2.2 Recruitment

The two conviction groups recruited included men aged between 17 and 50, who were current inmates at Auckland Prison, or were clients of the Manukau Community Probation Service (CPS) who were either recently released from prison or serving a Home Detention sentence. The men involved in the study were those who met the inclusion criteria, and who voluntarily gave informed written consent to participation in the study. To ensure that I, as the investigator carrying out the assessment, remained blind to the violent or non-violent conviction status of the men, the inmates were selected by an independent liaison group (Auckland Prison staff or probation officers).

The liaison group were instructed to ensure that all men were chronic recidivistic offenders, and to select approximately 50% violent offenders and 50% non-violent offenders. The selection group was advised that in the inmates’ conviction history it was preferable for the violent offenders to have only ever committed primarily violent crimes, and similarly, the non-violent offenders to have committed only non-violent crimes. An example of the two different categories would be *aggravated robbery* for a violent offence and *robbery (non-aggravated)*.
for a non-violent offence. The problems these criteria created and reasons for their use in spite of these difficulties will be discussed later.

All inmates who participated in testing had their conviction history checked in a manual search of their records after the scoring of their results. This ‘un-blinding’ was important to ensure that the criteria were met for assignment to violent offender or non-violent offender groups, in case mistakes in categorisation had been made by the Liaison team.

The control group included men living in the community who had never been convicted of a crime and who reported that they were free from violent behaviour for at least the previous five years. These men were self-selected after attending a presentation about the study that stated the inclusion and exclusion criteria. These men were participants in community education and occupational training groups. Control participants were selected from these courses because they matched the inmate groups on the variables of ‘low education’ and difficulty maintaining employment. Additional controls were recruited from a pool of prison officers. Participants in the control group matched the inmates on all the general exclusion criteria, and also had not passed beyond sixth form education (school year 11).
The demographic characteristics of participants from all three groups are presented in Table 3.

**Table 3: Demographic Characteristics of Study One Sample**

<table>
<thead>
<tr>
<th>Baseline Characteristics</th>
<th>Violent Offender Participants (N=21)</th>
<th>Non-violent Offender Participants (N=16)</th>
<th>Community Participants (N=16)</th>
<th>P ≤ .01</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age in Years</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>29.7</td>
<td>30.4</td>
<td>37.4</td>
<td>ns++</td>
</tr>
<tr>
<td>Range</td>
<td>21 – 44</td>
<td>18 - 48</td>
<td>18 – 55</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>11.6</td>
<td>7.4</td>
<td>13.6</td>
<td></td>
</tr>
<tr>
<td><strong>Years of Education</strong></td>
<td></td>
<td></td>
<td></td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Mean</td>
<td>8.3</td>
<td>9.4</td>
<td>10.1</td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>4 – 10</td>
<td>7.5 - 12</td>
<td>9 - 12</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>3.1</td>
<td>1.3</td>
<td>0.9</td>
<td></td>
</tr>
<tr>
<td><strong>Spot the Word SS\textsuperscript{+}</strong></td>
<td></td>
<td></td>
<td></td>
<td>Ns</td>
</tr>
<tr>
<td>Mean</td>
<td>6.8</td>
<td>8.3</td>
<td>8.0</td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>2 – 12</td>
<td>5 - 14</td>
<td>3 – 14</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>5.0</td>
<td>2.7</td>
<td>2.6</td>
<td></td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>14 (66.67%)</td>
<td>8 (50.0%)</td>
<td>5 (31.25%)</td>
<td>Ns</td>
</tr>
<tr>
<td>NZ European</td>
<td>1 (4.76%)</td>
<td>7 (43.8%)</td>
<td>6 (37.5%)</td>
<td>Ns</td>
</tr>
<tr>
<td>Māori /non-Māori</td>
<td>2 (9.5%)</td>
<td>1 (6.25%)</td>
<td>2 (12.5%)</td>
<td>Ns</td>
</tr>
<tr>
<td>Pacific Island / Other</td>
<td>4 (19%)</td>
<td>0 (-%)</td>
<td>3 (18.8%)</td>
<td>Ns</td>
</tr>
</tbody>
</table>

\textsuperscript{+} The Spot the Word Test results in a standard scaled score with a range from 3-18, with the average = 10 (SD = 3).

\textsuperscript{+} ns indicates no significance demonstrated at or below the p = .01 level between the three participant types on variables measured.
4.2.3 Selection Criteria

‘Violent’ offender participants:
(i) all males 17-50 years of age
(ii) convicted of at least 2 violent offences
(iii) have no history or current experience of psychosis, epilepsy, or a major depressive episode
(iv) no sexual offenders (which is a violent offence under New Zealand law)

‘Non-Violent’ offender participants:
(i) all males 17-50 years of age
(ii) convicted of at least 2 non-violent offences
(iii) free of violent convictions, including sexual offences
(iv) have no history or current experience of psychosis, epilepsy, or a major depressive episode

Non-Violent Community Control participants:
(i) all males 17-50 years of age
(ii) never convicted of any crime
(iii) free from violence for at least the last five years based on self-report
(iv) have no history or current experience of psychosis, epilepsy, or a major depressive episode
(v) not educated past sixth form level
4.2.4 Explanation of Inclusion and Exclusion Criteria

(i) Men who have a history of neurological impairment because of traumatic brain injury (TBI) were not excluded. There are two main reasons for this: (1) the men who exhibit executive dysfunction may be doing so because of a TBI, and (2) the number of men in prison who have sustained a TBI is very high. Barnfield & Leathem (1998) report from their New Zealand sample of 118 men that 86.4% have sustained a head injury, with more Māori (indigenous New Zealander) participants (91.4%) reporting head injury than non-Māori participants (79.4%). Thus, to exclude them would result in extreme difficulties in recruitment.

(ii) Men who have a history of psychosis, past or present, have been excluded from the study because of possible associated confounding effects on the neuropsychological results. Men who were currently suffering from a major depressive episode were also excluded. This is for two reasons; (1) many of the symptoms of depression, such as apathy and withdrawal are similar to the presentation of executive dysfunction, and (2) significant depression may affect the participants’ test performances, due to factors such as amotivation and lethargy, and thus results may not be a true reflection of their abilities.

(iii) In order to reduce confounding variables in the data collected, participants with possible temporal lobe dysfunction because of temporal lobe epilepsy were excluded. Research has shown there may be a link between temporal lobe epilepsy and violence, particularly interictal violence, although rare and often un-directed (Devinsky & Bear, 1984; Piacente, 1986; Taylor, 1969). The temporal lobe, and in particular temporal lobe epilepsy, has also been associated with episodic dyscontrol, which is demonstrated by episodes of unprovoked

(iv) Men who have committed offences that include sexual assault as a component in their offending history have been excluded. The neuropsychological and neuroanatomical literature indicates that a dysfunction of the temporal lobe may play a part in the nature of this type of offending (Golden et al., 1996).

(v) All offenders in the non-violent offender sample consist of individuals convicted of breaking and entering without assault and are additionally required to demonstrate nil violent offences or sexual offences. Those with offences of only fraud or traffic infractions were not considered for the study. This definition provides closer matching with the violent offenders on all variables but violence.

4.2.5 Consent Procedures

Violent and Non-Violent Offender Participants

(i) Following referral by the independent screening group, potential participants read the Participant Information sheet and if interested returned a Registration of Interest form to the liaison screening group.

(ii) A meeting was arranged with each participant individually, during which I explained the research in more detail, and conducted a brief “screening” assessment to ensure they met inclusion criteria.

---

2 See appendix A for copies of Information Sheets, Registration of Interest Forms, Consent Forms, and the advertisement.
(iii) Interested participants had an opportunity to read information, hear explanations, and ask questions before providing written consent. Throughout the consent procedure and assessment I remained blind to the ‘violent’ or ‘non-violent’ status of the participants.

(iv) When all assessments were completed I checked the inmates against the key list compiled by the liaison screening group and the inmate’s file. This ensured that the inmates had been categorised correctly. Any men who had been placed in the wrong participant type by the liaison group were then sorted into the appropriate group.

**Non-Violent Community Control Participants**

(i) Participants from community occupational courses contacted me directly or through their course co-ordinator in response to an advertisement, or following a 30-minute presentation describing the research conducted at their course venue.

(ii) Other community participants approached me directly upon hearing about the research through other people, who had seen the advertisement, listened to the presentation, or had participated.

(iii) Some of the control group included prison officers (from Auckland Prison), who contacted me in response to a general email to all prison staff.

(iv) All potential control participants were given the *Participant Information* sheet, and were screened before giving written consent. Following this they participated in the neuropsychological assessment.
Table 4 provides a summary of the selection and data collection procedures across the sample in Study One.

### Table 4: Summaries Of Selection and Data Collection Procedure for Study One

<table>
<thead>
<tr>
<th>Study One</th>
</tr>
</thead>
<tbody>
<tr>
<td>Violent Offenders identified by prison officers</td>
</tr>
<tr>
<td>Non-Violent Offenders identified by prison officers</td>
</tr>
<tr>
<td>Community Controls self selected</td>
</tr>
<tr>
<td>Participant Information handouts distributed by Prison Officers</td>
</tr>
<tr>
<td>Participant Information handouts distributed by Prison Officers</td>
</tr>
<tr>
<td>Presentation of study delivered at work courses. Participant Information handouts distributed</td>
</tr>
<tr>
<td>Registration of interest by inmate</td>
</tr>
<tr>
<td>Registration of interest by inmate</td>
</tr>
<tr>
<td>Registration of interest by community participants</td>
</tr>
<tr>
<td>Chosen for study on inclusion/exclusion criteria by Prison Officer</td>
</tr>
<tr>
<td>Chosen for study on inclusion/exclusion criteria by Prison Officer</td>
</tr>
<tr>
<td>Chosen for study on inclusion/exclusion criteria by researcher</td>
</tr>
<tr>
<td>Testing</td>
</tr>
<tr>
<td>Testing</td>
</tr>
<tr>
<td>Testing</td>
</tr>
<tr>
<td>Categorisation</td>
</tr>
<tr>
<td>Categorisation</td>
</tr>
<tr>
<td>Categorisation</td>
</tr>
<tr>
<td>Un-blinding to offender type</td>
</tr>
<tr>
<td>Un-blinding to offender type</td>
</tr>
</tbody>
</table>

#### 4.1.6 Definition of Violence

As previously discussed, it is a challenging task to define violence for measurement and research purposes, partly because little agreement exists in the literature. In order to enable consistent and transparent participant selection, one of the most common measures used in the literature was adopted; that is, violence has been defined as convicted of physically harming another person. Additionally,
the physical assault must not include sexual violence. Furthermore, violent offender participants must be recidivist offenders, with more than two convictions for violence, therefore avoiding contention that their violent offending was a “one-off” or rare event, and furthermore suggesting a possible difficulty in learning from mistakes and changing behaviour.

In contrast, non-violent offenders are described as being convicted of 2 or more non-violent offences, but never convicted of a violent offence. The non-violent offences are of the breaking and entering and theft type. They are not fraud or traffic offences as these are considered too substantially different from the offences the violent inmates commit. This examination is an attempt to seek the subtle organic and behavioural variations between offenders who use and don’t use violence, who are in other ways very similar.

The non-violent community control subjects were required to have never been convicted of an offence and to self-report being violence-free for the last five years. The limitation of five years is important to remove the barrier to those participants who may have been aggressive only for an ‘adolescent-limited time’ (Moffitt, 1993).

Although every effort was made to check that men in the non-violent offender and control groups had not committed violent acts, it remains a possibility that some may have been violent but had not been convicted of violent crimes. The limitation this imposes on the study is examined in the General Discussion in Chapter Seven.

4.2.7 Procedure

All testing of inmates took place at Auckland Prison, a maximum and medium security prison 30km north of Auckland city. Testing of inmates transferred into a probation service was conducted at Manukau Community Probation Services,
which is situated in South Auckland. Testing of community control participants took place either at their home, their work, their education setting, or in an interview room at the University of Auckland. The control subjects were living in Auckland or Northland at the time of testing. There was no incentive or reimbursement scheme for participation. The participants were all able to read and write in English to at least the Intermediate School age level (i.e., 11 – 13 years of age), as assessed by the liaison group on the basis of existing literacy scores kept by the Department of Corrections.

Each testing session was conducted in a room with a table and two chairs, subject to no permanent observation by prison officers, probation officers or members of the community. It was hoped that this assessment environment would encourage the participant to be as candid as possible and assist concentration. The interview rooms themselves were across settings, including Auckland Prison, the University of Auckland, people’s homes, and places of work and education.

4.2.8 Measures

List of Measures:

Interview
The Beck Depression Inventory – II (BDI-II)
Likert Scale of Appropriateness
The Behavioural Assessment of the Dysexecutive Syndrome (BADS)
Controlled Oral Word Association Test - Māori version (COWA-M)
Benton Test of Facial Recognition (TFR)
Spot-the-Word-Vocabulary Test (STW)
During the initial interview before neuropsychological testing began, participants were asked a list of questions; this would take approximately 20 minutes. Some questions were applied only to incarcerated individuals. Participants were asked:

1. Have you ever had a head injury (an explanation and examples were given of what this is), and if so when did your first head injury occur? Was it before your first conviction?
2. Have you ever used drugs before, and if so, was this use regular? If so, are you still using them? Have you used within the last 48 hours (if so, what substance)?
3. Have you used alcohol before? If so, how often and how many units (with an explanation) in one sitting would you have? Have you drunk alcohol within the last 48 hours?
4. Were you intoxicated or high at the time of your offences?

The interview acted as a means of ‘breaking the ice’ and rapport building, as well as gaining important background information to aid in the estimate of general ability. Details of participants’ education and employment history were noted, as were observations pertaining to the way they performed the tests.

It was important to know if any of the participants were using substances within 48 hours of neuropsychological assessment, as this could affect their performance. The men were given repeated assurances that there would be no identifying information given in the thesis and that this information would remain confidential, and would not be revealed to the prison or probation authorities, or anybody in the community.
Likert Scale of Appropriateness

In order to understand and quantify each participant’s test approach and behaviour during testing, a Likert scale of Appropriateness was developed. A 5 point scale was used where: 1 = behaviour that was appropriate to the situation; 2 = behaviour that was mostly appropriate; 3 = some inappropriate behaviour; 4 = quite a lot of inappropriate behaviour; and 5 = behaviour that was extremely inappropriate. Immediately following the interview and neuropsychological assessment the Likert Scale was completed with reference to observations made in the session. At all times when the Likert Scale was applied I was blind to the convictions of the inmate participants.

An example of a rating of 1 (displaying behaviour that is appropriate) would be a participant who completed assessment in a conventional fashion, complying with instructions and conversing in a suitable manner. An example of a rating of 5 (displaying behaviour that was extremely inappropriate) would be a participant who continually discussed information he had been specifically instructed to keep to himself, or who may ask the examiner questions of an inappropriate manner or attempt to touch the examiner. Concrete examples of participants’ behaviours along with consequent ratings are provided in the Results section of this chapter.

The Behavioural Assessment of the Dysexecutive Syndrome (BADS)

The Behavioural Assessment of the Dysexecutive Syndrome (BADS; Wilson et al., 1996) is a test battery that examines the deficits in functional behaviour that may occur in someone with executive dysfunction. The tests have been specifically designed to be sensitive to deficits that may not ordinarily be visible in a well-structured testing environment. The BADS battery includes items sensitive to deficits in planning, problem solving, and organising behaviour over extended periods of time. These functions are important for everyday living, yet are difficult to assess accurately within the well-structured environment of a
neuropsychological exam. To this end, the BADS is an example of a battery designed to fulfil ecological validity.

Subtasks within the battery measure the different functions thought to represent the major components of executive function, such as temporal judgment, planning, cognitive flexibility, shifting-set, prospective memory, inhibition, task scheduling, self-monitoring, action planning and goal directed behaviour. The BADS separates out these components for ease of testing and to clarify areas of deficit. Additionally, the BADS takes account of the theoretical models of the Central Executive (CE) and the Supervisory Attentional System (SAS) by attempting to draw all the components together into a full understanding of the executing of the executive functions, by providing an overall ED profile score. This profile score also contains clinical utility, in that poor scores are associated with severe real-world difficulties and prognosis (Norris and Tate, 2000). Each of the subtasks of the BADS results in a score out of four. The six subtasks are summed together to provide an overall ED profile score out of a maximum of 24. Profile scores are given clinical ratings according to the age-norms developed. For the sampled age group in this thesis, the classifications for executive functioning as measured by the BADS are: Impaired 0-12; Borderline 13-14; Low Average 15-16; Average 17-20, High Average 21-22; and Superior 23-24.

In the development of the battery, the BADS was completed by a stratified sample of 216 healthy control subjects and 78 patients with a variety of neurological disorders (Wilson et al., 1996). The control sample had three bands from ‘below average’ IQ to ‘above average’ IQ, was gender balanced, and had equal numbers from four age groups: 16-31, 32-47, 48-63, and 64+. In the patient sample 59% of participants had sustained a TBI. (Note that no prison inmates were included in the ‘norming’ of the BADS). The authors of the BADS (Wilson et al., 1996) did not provide construct validity for the battery. However, overall construct evidence for the BADS as a full battery has been provided by one study. Norris and Tate (2000) demonstrated in their sample of 36 participants with neurological disorders
and 37 healthy controls, that the BADS could correctly classify participants 74% of the time. They state that the BADS is comparable to standard measures of executive functions in discriminating between neurological dysfunction and non-brain damaged participants. Norris and Tate (2000) also found that the ecological validity of the BADS is superior to standard executive tests.

Along with the six tests in the battery there are two 20-item questionnaires (DEX) that help to describe clinical measures, but are not needed for the overall BADS profile score. These questionnaires assess self and others’ reports of the individuals’ executive behaviour. The DEX were initially employed in the current research and were to be filled in by the participant and by a nominated person who knows the participant well (preferably one who lives with them), but were abandoned due to a very poor return rate. Participants completed the questionnaire as part of the testing situation, but these became redundant due to the lack of comparison ratings returned. In particular, the inmate participants found it very difficult to find someone they trusted enough to fill in the DEX, with some admitting during testing that they would not be able to follow through on giving the questionnaire to another person. Attempts to increase return were made within the limits of ethics (no incentives were allowed), by providing stamped and addressed envelopes. Control participants demonstrated a higher rate of return than experimental participants, but the total return and the distribution of return was not useful for analysis. The BADS authors recommend that the DEX be used clinically to help build up a solid picture to guide treatments, and this information may have had utility in guiding development of the rehabilitation programme (described in Study Three).
Descriptions of each subtask of the BADS follow:

**Rule Shift Cards Test**

This is a simple test broken into two parts in order to examine the participant’s ability to respond correctly to a rule, and to shift between rules. The test uses pictures of playing cards that are spiral-bound into a booklet. The first part of the test requires participants to flip through the cards and respond ‘Yes’ to a red card and ‘No’ to a black card. This simple yes-no rule is written on a card and kept in full view of the participant to reduce any memory constraints. The first part of the test establishes a response set. The second part of the test uses the same 21-page spiral-bound picture book of playing cards flipped as before, but this time the rule is different. The new rule requires saying ‘Yes’ if the card is the same colour as the last one, and ‘No’ if it is different. This next rule is now placed in front of the participant, and they are asked to read through it and to follow it. The challenge of the second part of the test is for the participant to disregard the first rule, and that ‘response set’, and instead follow the second rule. The second part of the test is the one used for scoring, and observations of perseverative errors are made. The measures taken are time to complete and number of errors. The Rule Shift Cards Test calls upon the ability of the participant to shift from one rule to another, to keep track of the colour of the previous card, and to utilise the current rule.

The Rule Shift Cards Test is considered to measure the executive functions of cognitive flexibility and inhibition, similar to the WCST and Trail Making Test (TMT). When concurrent validity of this subtask with WCST and TMT was examined, Norris and Tate (2000) demonstrated that it held for TMT only. However, it may be that the WCST is tapping into other abilities than planning and inhibition alone (Anderson et al., 1991; Romine et al., 2004), reducing the covariate between WCST and the Rule Shift Cards Test.
**Action Program Test**

The Action Program Test has been adapted from Klosowska (1976), and was designed to provide a novel and practical hands-on task for participants (Wilson et al., 1996). It is a test that requires the development of a plan of action in order to solve a problem. The test demands the physical manipulation of a beaker that is two-thirds full of water and has a lid with a small hole in the centre. This beaker is on a flat stand, and in the stand next to the beaker is a tube that has a cork in the bottom. Next to the stand are an L-shaped wire hook and a bottomless screw top container with its screw top lying next to it. The participant is told that they have to get the cork out of the bottom of the tube, without lifting the stand, the tube, the beaker, or touching the lid with their fingers.

For successful completion of the test the participant must first think of the solution. They must then keep the overall plan in their mind as they work back from it; i.e., they have to put their plan into action and attempt to do so successfully. Participants are given two minutes to advance through at least the first of five stages. If they have not completed the first stage once two minutes are up they are given a prompt, and this continues at each of the following four stages. Participants lose marks each time a prompt is needed. This test requires the five steps to be completed independently for the participant to gain full marks.

The Action Program test is comparable to the Porteus Maze (PM) and the copy of the Rey-Osterreith Complex Figure (RCF) measuring planning and problem solving. It has been demonstrated to be significantly associated with these tests, demonstrating concurrent validity (Norris & Tate, 2000). The Action Program subtask is also a robust predictor of brain dysfunction and role dysfunction (Norris & Tate, 2000).
Key Search Test

This test has been influenced in its development by a test originally described by Terman & Merrill (1937). In the Key Search Test participants are to imagine that a 100mm square on a piece of A4-sized paper is a field, and in this field they have lost their keys. Their task is to enter this field starting from a dot 50mm below the centre of the square and to search the field for their keys. They are to demonstrate, by using a pen to draw a line, where they would walk to search the field for their keys (Wilson et al., 1996). The participants are told to keep their pen on the paper the entire time. This task is timed. It is scored using time, the task components of entering and exiting the field, and the search pattern used including the amount of certainty that their search would yield the keys.

The Key Search Test is purported to measure planning in the same manner as the RCF and the PM, however when tested for concurrent validity the correlations were low (Norris & Tate, 2000). This may be because the Key Search Test attempts to be analogous to a real-life activity.

Temporal Judgment Test

Four short questions are presented in the Temporal Judgment Test. The questions concern events that take from several seconds to several years in time. For example, participants are asked to give estimates for time taken to blow up a party balloon and the lifespan of a dog. One point is given for each correct answer. For each question the correct answer can fall within a small range, for example the answers to the above questions respectively are ‘between 50 & 70 seconds’ and ‘between 9 & 15 years’ (Wilson et al., 1996).

This test was based on the Cognitive Estimates Test CET (Shallice & Evans, 1978), but the correlation between the two is very low ($r = .11$; Norris & Tate, 2000). This may due to the Temporal Judgement Test having only four items of measurement, making it less reliable than the other subtasks and less like the
original than intended. This subtask also has clear cultural constraints, possibly resulting in invalid results when used with a New Zealand population. These limitations will be discussed in the Discussion of this study.

Zoo Map Test

This test asks participants to plan a route around a zoo twice. The zoo is represented in two versions on A4-sized paper, and is essentially a maze with instructions and rules. The first version (Zoo Map One) asks participants to visit a series of locations while following the rules of starting at the entrance, finishing with a picnic, and using certain paths “only once.” The sequence in which the participants visit the locations is left up to them, but the inference is they should follow a certain sequence(s) in order to abide by the rules. In the second version (Zoo Map Two) participants are given the same locations to visit, but this time they must do so following a set order supplied with the zoo map. The same rules, such as where to start and finish, and which paths can be used, also apply in this version. Comparison of performance on both versions allow for evaluation of the participant’s spontaneous planning ability (Wilson et al., 1996). The Zoo Map Test is scored by time taken and number of errors made. Errors include the rules broken, visiting places that were not on the list, deviating from the pathways, and failing to make a continuous line.

The Zoo Map Test measures planning, monitoring, and regulation of behaviour in the same manner as the copy of the RCF and the PM in a comparison study (Norris & Tate, 2000), but correlations were low. The attempt at ecological validity and the complicated scoring method may obscure the similarities between the tests and it can be speculated that the Zoo Map Test measures executive functions of interest more thoroughly than the comparison tests chosen by Norris and Tate. The assumption of ecological validity is strengthened by the subtask being demonstrated as a robust predictor of brain dysfunction and role dysfunction (Norris & Tate, 2000).
Modified Six Elements Test

The participant is given instructions to complete three tasks in 10 minutes. The tasks are dictation, arithmetic, and picture naming. Each task is broken into two versions; A and B. Thus there are six elements to this test in total. To gain full marks the participant is required to attempt all the six elements in 10 minutes without spending more than 271 seconds on any one of them. They must also be mindful of the rule that they are not allowed to do two parts of the same task consecutively. Thus, if they do dictation-A, they cannot immediately do dictation-B, or if they do arithmetic-B, they cannot then go on and do arithmetic-A. A large digital clock counting off the seconds is placed in front of the participant so they can attempt to manage their time. The test is scored using the number of tasks completed, whether or not the rule was broken for any task, and the maximum amount of time spent on any one task (Wilson et al., 1996).

The Modified Six Elements is an adapted version of the original Shallice & Burgess (1991) test, and tests a participant’s prospective memory. Prospective memory enables us to hold a number of different things in mind in order to translate them into action. This subtask also measures the ability to plan for the future and carry out a plan successfully. It is also useful for demonstrating incidences of rule-breaking. The Modified Six Elements has demonstrated concurrent validity to the copy and strategy portion of the RCF as well as the PM (Norris & Tate, 2000). This subtask has also been proven to be a robust predictor of brain dysfunction and role dysfunction (Norris & Tate, 2000).

Controlled Oral Word Fluency

Word fluency tests are thought to show impairment when the dominant-for-speech hemisphere is damaged. Following a frontal lobectomy (sparing Broca’s area) of the dominant hemisphere, patients often show a reduction in spontaneous speech, and this reduction can be demonstrated and measured using tests of word fluency (Milner, 1964). In a review by Lezak (1995), PET studies showing bilateral
frontal involvement in fluency tests were described. Lezak also noted that patients with the lowest fluency scores had the most metabolic activity in the frontal region, suggesting that patients having the most trouble had to invest the most effort (Lezak, 1995). Word fluency tests are also affected by the ability to formulate or use advantageous strategies for generating words, such as using association as a cue, or sub-vocalisation (Lezak, 1995). The inability to utilise these types of strategies, and trouble in general word-finding ability, are problems noted in executive dysfunction.

The most common test used to measure word fluency is Controlled Oral Word Association (COWA), first described by Benton & Hamsher (1976). In this test participants are asked to orally generate as many words beginning with a certain letter of the alphabet as possible (in a one minute time frame). There are three trials with three different letters. In completing this test there are rules to follow: no proper nouns, no numbers, and no words that are the same except for their suffix, e.g., ‘wait’ and ‘waiting’. The most commonly administered version of COWA uses the letters F, A, and S. The letters used in COWA have been selected for their high frequency of occurrence in the English language.

A new version of the COWA has been used in the current research because of the high proportion of Māori. A version that is ‘Māori-friendly’ (COWA-M) is used to make some step towards being more culture fair, and to reflect the usage of English and Māori used interchangeably by some Māori and non-Māori living in New Zealand (Ogden, Cooper, & Dudley, 2003). This version has been used before in examining both the fluency of Māori and non-Māori participants (Cooper, 1997; Ogden et al., 2003). The letters used are T, P, and W, and were selected originally by Cooper on the basis of the frequency of both Māori and English words beginning with these letters (Borkowski, Benton, & Spreen, 1967; Cooper, 1997; Ogden et al., 2003).
In the test instructions participants are given examples of words they can use in both the Māori and English languages. An example given in the test instructions follows:

“I will tell you the letter and I will give you one minute to tell me as many words as you can with that letter. Try to tell me as quickly as you can. For example, if I say ‘K’, you might say to me ‘kitchen, korero, kick, kai’.” (Ogden et al., 2003) p. 133

In this instruction the fact that the participant can respond in both Māori and English is not directly told to them, but is implicit in the instructions. This reduces performance pressure on those participants who identify as Māori but do not speak Māori, and opens up the possibility for those participants who do know some Māori language to be comfortable in giving some Māori words. This applies to both Māori and non-Māori participants. In one study using these instructions, Pakeha participants responded favourably to the instruction and some even gave a few Māori words in response (Ogden et al., 2003).

In Cooper’s (1997) study the performances of the Māori participants and the non-Māori participants were compared with each other and against standardised normative data provided by Borkowski et al. (1967). The Borkowski et al. (1967) data provides norms for each of the three letters separately from a sample of ‘healthy’ American women (n = 66). The mean number of words generated for Māori men were 37.20, and for Pakeha men 43.20 (Cooper, 1997). When women were included in the sample and assessed with COWA-M, a total sample of 40 provided the norms of 39.6 for Māori and 38.9 for Pakeha (Ogden et al., 2003). These published norms were used for comparison with the samples in the current studies. In choosing the COWA-M for use in the current study, it is suggested that the letters T, P, and W and the ‘Māori-friendly’ instructions (Cooper, 1997; Ogden et al., 2003) will assist in reducing possible cultural bias in testing for Māori participants.
Test of Facial Recognition

The Test of Facial Recognition – Short-Form (TFR; Benton, Hamsher, Varney, & Spreen, 1983) was included as a test that should not be affected by ED. It requires patients to match photos of faces in three conditions of the test: A., matching of identical front-views, B., matching of a front-view with three-quarter views, and C., matching of a front-view under different lighting conditions. The participant in the first part of the test has to find only one match with the photo, and in the rest of the test they have to find three matches for the photo of the face. It is expected that individuals with right-sided parietal cortex damage perform poorly in the TFR (Benton, Hamsher, Varney, & Spreen, 1983). More importantly, in the context of localising brain function, a much larger number of patients with posterior lesions (usually right hemisphere) have defective performances than patients with anterior lesions (Schretlen, Pearlson, Anthony, & Yates, 2001).

Normative standards are based on the scores of 286 individuals between 16-74 years of age of both clinical and control participants. The norms demonstrated that participants with 12+ years of education performed significantly better, and this has been accounted for in the scoring. The short-form used is converted into long-form scores and any corrections for education are calculated resulting in scores that indicate performance thus: Normal 41-54; Borderline 39-40; Moderately Impaired 37-38; Severely Impaired <37.

Many investigations have demonstrated that lesions of the right cerebral hemisphere contribute to the disruption of facial recognition as measured by the TFR (see Schretlen et al., 2001 for a review). For example, when children with epilepsy were assessed before and after surgery, it was found that those with excisions in the posterior right hemisphere (usually parietal lobe) were significantly impaired in their ability to perform on the TFR compared with their previous scores and the scores of children who had surgery in the left hemisphere (Beardsworth & Zaidel, 1994). However, damage to anterior regions may also
impact on the TFR score, as reflected in a study demonstrating reduction in scores after frontal and temporal lobectomies (Braun, Denault, Cohen, & Rouleau, 1994).

Schretlen et al. (2001), interested in reports that both right posterior lesions and anterior lesions have been shown to affect TFR processing, examined 170 normal healthy participants aged from 20 up to 92 years of age. Using TFR and MRI, Schretlen et al. (2001) demonstrated that normal atrophy in posterior regions and frontal regions, combined with ventricles-to-brain ratio, accounted for variance in perception of faces, with age as a determinate. The authors hypothesised that age-related atrophy contributed to poorer processing speed and this predicted worse outcome on the TFR for older healthy participants. This result was only true for the older subjects. As the sample in that study differs significantly in age from the current sample tested in this study, previous studies demonstrating that lesions in the right posterior lobe diminish scores of the TFR compared to lesions in other areas are presumed satisfactory in establishing TFR as the test to differentiate from prefrontal or executive functioning.

The best evidence for frontal lobe dysfunction exists when frontal tests are administered together with ‘posterior-lobe’ tests (Kandel & Freed, 1989). It is important to have comparative data, so that when the frontal tests show greater impairment than posterior tests, it is more likely to be indicative of specific frontal pathology. If frontal tests are used alone, impairment may be merely a reflection of diffuse brain damage (Kandel & Freed, 1989). Associating tests with localised brain damage can be limiting, and although tests may demonstrate an association with a specific region of brain damage, that does not mean other regions or circuits are not also serving the functions the test measures. The lateral posterior cortices are posited to contribute most to the understanding of, and responses made to, spatial information. Due to time constraints, concerns of participant burden, and a reluctance to tire the participants to the detriment of their overall performance, only one ‘posterior’ test, the TFR, was administered.
The Spot the Word Vocabulary Test

The examiner needs to have an estimate of the ‘intelligence’ of the participant so all test results can be examined in this context. Poor test results may not necessarily be indicative of deficits in certain areas of functioning; they may simply be a reflection of the participant’s level of understanding, verbal ability, and/or previous education. The Speed and Capacity of Language-Processing Test (SCOLP) (Baddeley, Emslie, & Nimmo-Smith, 1992) is a two-test battery (the Spot-the-Word-Vocabulary Test; STW, and the Speed of Comprehension test; SCT). It allows the examiner to judge a participant’s level of vocabulary as an estimate of IQ, and to check for a slowed rate of thinking possibly due to brain damage. The STW only was administered in this study, and the test authors claim it is a reliable estimate of verbal intelligence, which is resistant to the effects of stress or brain injury (Baddeley et al., 1992).

In the STW the participant is shown a written list of word pairs, one word being a real word and one word a neologism (a made-up nonsense word). Upon being presented with the word pair the examinee is asked to mark which of the pair the real word is, and to guess if they do not know. The STW was chosen as it has an advantage over another common test of IQ estimation, the National Adult Reading Test (NART; Nelson & Wilson, 1991). Unlike the NART, the STW does not require the examinee to say the words out loud, and so there is less likelihood of embarrassment on the part of the participant and the associated anxiety this can produce, possibly limiting their test performance. Possible confounds of different accents and differing pronunciations of words due to cultural factors is also avoided.

Validity of the STW test in testing a similar construct to the NART was demonstrated by the test makers in demonstrating an association of $r = .87$ (Baddeley et al., 1992). A stratified sample of 224 individuals was tested to develop the norms. Raw scores taken from the person’s performance on the 60-
item test are converted into scaled scores from which percentiles were calculated. The resulting scaled scores are 3-18, with an *Average* score of 10, SD = 3. Although the authors claim the STW is ‘adaptable to any language’ (Baddeley et al., 1992) the version used contains only English words and was normed on a British population. It may underestimate Verbal IQ in New Zealanders, especially those who identify as Māori or Pacific Island Peoples. However, data collected in New Zealand examining differences between Māori and Pakeha on STW found that there were no significant differences between the two groups, suggesting that the test and the norms may be suitable for use with Māori (Cooper, 1997; Ogden et al., 2003).

**The Beck Depression Inventory – II**

The Beck Depression Inventory –II (BDI-II, Beck, Steer, & Brown, 1996), is a 21-item self-administered questionnaire that uses the DSM-IV criteria for major depression, requiring approximately 5 – 10 minutes to complete. Respondents are requested to read statements, and then circle the statement that most applies to them. Questions cover areas such as appetite changes, changes in interest, lethargy, motivation, irritability, sexual interest, and suicidal ideation. Items marked are added up and result in the clinical classifications for depression of: *Minimal* 0-13; *Mild* 14-19; *Moderate* 20-28; and *Severe* 29-63 (Beck, Steer, & Brown, 1996).

Depression and executive dysfunction can have symptoms in common, including apathy, problems with motivation, lethargy, and lack of interest in activities that they used to be interested in. If a participant had a ‘high’ depression score, he was questioned around the areas of concern identified, and a decision made regarding his continuation in the study. A concern in using the BDI-II is that it may not be an appropriate measure for an incarcerated New Zealand population, due both to population norm differences, and questions that ask about punishment and guilt.
Difficulties with using the BDI-II with an incarcerated population are highlighted in the *Discussion* of this chapter.

### 4.2.9 Test Order

Test order has not been shown to have any real effect on performance (Cassel, 1962), but to limit any possible order effects two different orders were used as follows:

- BDI-II
- BADS
- TFR
- COWA-M
- STW

and

- BDI-II
- TFR
- COWA-M
- BADS
- STW

The BDI-II was always presented first after the initial interview, as it was thought to provide an icebreaker and an easy introduction into what can be an anxiety-producing testing session. Also if any problems with depression were noted, then the participant could be interviewed further about the relevant items before making a decision as to whether it was appropriate to move on to the remainder of the testing. The first half of men enrolled in each of the three groups was given the first test order, and the second half received the second test order listed.
4.2.10 Analysis

The data collected in Study One was analysed predominantly with nonparametric tests due to the small sample collected, the violation of normality assumptions, and the type of measurements taken (e.g., BADS profile scores). Using nonparametric statistics creates more robust findings and increases the power efficiency when compared to analysis of small sample data with parametric methods. However, initially all data was run through an ANOVA to establish relationships of significance and reduce possibilities of Type II errors. Relationships of significance were hence identified and examined more closely with nonparametric methods. *Kruskal Wallis* tests were conducted to examine any significant differences between the means of tests for all the groups, for Māori only, and for participants who had not used substances within 48 hours of testing. Spearman’s rho and Partial correlations are presented to further examine relationships of interest. The lower p-values and the use of non-parametric methods is an attempt at reducing Type I errors. Throughout the results all statistical p values reported are 2-sided.

All men were categorised into one of two categories based on their quantitative test scores, and qualitative information from the interviews and test behaviour (as measured by the Likert Scale of Appropriateness). A *Clear ED* Pattern included men who demonstrated a clear executive dysfunction pattern (*Low Average* or poorer on the BADS combined with a 3, 4 or 5 on the Likert Scale). An *Unclear/No ED* Pattern included men who did not demonstrate a clear executive dysfunction pattern, that is, they had a BADS score of *Average* or better, or a *Low Average or Borderline* score with a Likert Scale rating of 1 or 2 (refer to table 5).
A cross-tabulation Chi Square ($\chi^2$) test was carried out to examine the relationship between group membership (violent participants, non-violent participants, and controls) and the ‘ED pattern’ group.

Chi square tests were also used to examine the reported incidence of traumatic brain injury (TBI), substance use and the relationship these variables have to participant type.

4.3 Results

Homogeneity of variance was tested with the Levene Statistic as well as examining Skewness and Kurtosis for all groups. As the spread measures indicated that the data collected on the BADS and Years of Education violated the assumptions of the ANOVA across the groups, the nonparametric version of the ANOVA (Kruskal-Wallis Test) was computed. The Kruskal-Wallis Test is reported in the following tables only for results that were found to demonstrate significance on the ANOVA.

An ANOVA set at $p = .05$ was computed first for all variables. This established significant differences across the participant groups for BADS ($p < .001$), BDI-II ($p = .023$), and Years of Education ($p < .001$). No significant differences were demonstrated between the participant groups for the other tests (STW, TFR,
COWA-M) or for Age and Ethnicity. Significant differences set at $p \leq .01$ between the participants (BADS; $p < .001$; Years of Education, $p < .001$) remained when more closely examined with a Kruskal-Wallis. The violent offender participants gained the poorest BADS scores and reported the lowest mean level of education.

Spearman’s rho rank-order statistic further demonstrated the close relationship of the BADS with Participant Type ($r = .58$) and Years of Education with Participant Type ($r = .54$). These can be seen in Table 6.

Crosstabulations run on the qualitative data indicated a high reporting of TBI and substance abuse across the entire sample. Although a high number of inmates reported marijuana use prior to testing, analysis with chi square, covariates, and Kruskal-Wallis demonstrated that this did not affect the direction and significance of the data.

A Kruskal-Wallis was carried out on the order in which the tests were given and demonstrated no impact on the data gained for any test.

4.3.1 Demographic Data

Table 3 in the Methods section of this study displays the demographics and demonstrates the uneven spread for some demographic variables across the participant types. The ‘unevenness’ results in difficulties drawing robust conclusions from the sample and will be discussed further in Chapter Seven (the General Discussion). One of the contributing factors to this were some incorrect classifications made by the liaison officers in assigning participants to the non-violent group (to which I was blind at the time of testing). Upon later examination some men were found to meet criteria for the violent offender group. After checking each offender’s conviction history, three Auckland Prison inmates were re-assigned as violent offender participants rather than non-violent offenders, and
two inmates were excluded from the study for violating criteria (both had sexual offending histories).

There is a predominance of Māori participants across the sample, with a high proportion of Māori in the offender groups, and particularly the violent offender group. However when ethnicity was examined within and across the groups with both ANOVA and a Kruskal-Wallis Test, no significant differences were detected. This was also the case when age was examined across the three groups with both an ANOVA and a Kruskal-Wallis Test.

The only demographic variable that reaches significance across the three Participant Types is Years of Education.

Table 6 demonstrates an effect of ethnicity, with Ethnic Identity (see Table 3 for the categories) impacting upon the test STW (p = .014) as well as on Years of Education (p = .002). Ethnicity did not differ significantly across participant types.

**Table 6: Kruskal Wallis Test: Comparison of Ethnic Identity across Participant Type, test scores, and Years of Education**

<table>
<thead>
<tr>
<th>Participant type</th>
<th>Bads Score</th>
<th>Spot the Word Test</th>
<th>Beck depression inventory</th>
<th>COWA-M</th>
<th>Years of Education</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chi-Square</td>
<td>8.73</td>
<td>9.69</td>
<td>12.85</td>
<td>4.87</td>
<td>11.06</td>
</tr>
<tr>
<td>df</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Asymp. Sig.</td>
<td>.068</td>
<td>.046</td>
<td>.014</td>
<td>.301</td>
<td>.026</td>
</tr>
</tbody>
</table>

Pacific Islands People demonstrated the most difficulty with the STW, scoring a mean scaled score of 5.6. Māori and Māori /non-Māori were the next with a scaled score of 7, which was the same as the only ‘other’ non-Pakeha participant
(Indian ethnicity). Pakeha gained the highest mean score of 9.9. Years of Education for the different ethnicity types were as follows: Pacific Islands People (8.9 years), Māori (8.7 years), Māori/non-Māori (8.2 years), the single Indian participant (11 years) and Pakeha (10.2 years).
4.3.2 Relationship of Variables

Spearman's rho is a rank-order correlation coefficient (r) that measures association at the ordinal level. This is a nonparametric version of the Pearson correlation coefficient and based on the ranks of the data rather than the actual values, yielding stricter but stronger values. Table 7 lists the covariants between pairs of variables.

Table 7: Spearman’s rho Correlations

<table>
<thead>
<tr>
<th>Bads Score</th>
<th>Spot the Word Test</th>
<th>COWA-M</th>
<th>Beck Depression Inventory II</th>
<th>Years of Education</th>
<th>Participant Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>r</td>
<td>1.00</td>
<td>.44**</td>
<td>-.48**</td>
<td>.51**</td>
<td>.58**</td>
</tr>
<tr>
<td>Asymp. Sig</td>
<td>.001</td>
<td>.023</td>
<td>.000</td>
<td>.000</td>
<td>.000</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Spot the Word Test</th>
<th>r</th>
<th>1.00</th>
<th>.40**</th>
<th>-.28</th>
<th>.53**</th>
<th>.22</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymp. Sig</td>
<td>.001</td>
<td>.003</td>
<td>.04</td>
<td>.000</td>
<td>.121</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>COWA-M</th>
<th>r</th>
<th>.31</th>
<th>.40**</th>
<th>1.00</th>
<th>-.11</th>
<th>.33</th>
<th>.31</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymp. Sig</td>
<td>.023</td>
<td>.003</td>
<td>.441</td>
<td>.016</td>
<td>.024</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Beck Depression Inventory</th>
<th>r</th>
<th>-.48**</th>
<th>-.28</th>
<th>-.11</th>
<th>1.00</th>
<th>-.33</th>
<th>-.40**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymp. Sig</td>
<td>.000</td>
<td>.039</td>
<td>.44</td>
<td>.017</td>
<td>.003</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Years of Education</th>
<th>r</th>
<th>.51**</th>
<th>.53**</th>
<th>.33</th>
<th>-.33</th>
<th>1.00</th>
<th>.54**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymp. Sig</td>
<td>.000</td>
<td>.000</td>
<td>.016</td>
<td>.017</td>
<td>.000</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Participant Type</th>
<th>r</th>
<th>.58**</th>
<th>.21</th>
<th>.31</th>
<th>-.40**</th>
<th>.54**</th>
<th>1.00</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymp. Sig</td>
<td>.000</td>
<td>.121</td>
<td>.024</td>
<td>.003</td>
<td>.000</td>
<td>.</td>
<td></td>
</tr>
</tbody>
</table>

NOTE: all analysis with N=53
** p ≤ .01 level

There are significant correlations between Participant Type and the BADS score, Years of Education, and BDI-II. Significant correlations also exist between the
BADS score and the STW, and Years of Education, and the BDI-II. The Spot the Word Test is also significantly correlated with COWA-M and Years of Education.

In order to further investigate the relationship between the BADS score and Years of Education upon Participant Type, partial correlation coefficients were run. A partial covariate between BADS and Participant Type controlling for Years of Education and BDI-II demonstrated a covariate of .36 (p = .010), whereas an examination of Years of Education and Participant Type controlling for BADS and BDI-II demonstrated a covariate of .34 (p = .014). The results of the partial correlations indicate that the relationships that both the BADS and Years of Education have with Participant Type remain significant when the effects of the BDI-II (and each others’ influence) has been controlled for.
4.3.3 Examination of Means

Table 8 presents the major tests in the neuropsychological examination and demonstrates a general trend for violent offender participants to have poor scores. Note that the Test for Facial Recognition (TFR) is presented in this table but not in the following analyses. This is because all participants except one (a control participant who missed out on a score of ‘normal’ by 1 mark) received a “normal” score. The TFR was included in the battery as a differentiation measure, and the ‘normal’ score gained by all but one of the participants supports a lack of generalised poor functioning in any of the participants. Thus, the difficulties measured as executive functioning on the BADS are more likely to be indicative of ‘true’ executive difficulties, usually associated with the prefrontal lobes, and not simply a result of low general ability.

<table>
<thead>
<tr>
<th>Participant type</th>
<th>BADS Score (^a)</th>
<th>Spot the Word Test (^b)</th>
<th>COWA-M</th>
<th>Beck Depression Inventory (^d)</th>
<th>Test of Facial Recognition (^f)</th>
<th>Years of Education (^g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Violent</td>
<td>13.48 (3.95)</td>
<td>6.76 (2.84)</td>
<td>32.05 (10.39)</td>
<td>14.95 (10.64)</td>
<td>100% (.00)</td>
<td>8.31 (1.38)</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>17.56 (2.94)</td>
<td>8.31 (2.70)</td>
<td>37.37 (9.06)</td>
<td>13.56 (7.90)</td>
<td>100% (.00)</td>
<td>9.34 (1.31)</td>
</tr>
<tr>
<td>Community</td>
<td>18.25 (1.95)</td>
<td>8.00 (2.63)</td>
<td>37.75 (9.44)</td>
<td>6.81 (7.29)</td>
<td>94% (.14)</td>
<td>9.96 (.74)</td>
</tr>
</tbody>
</table>

\(^a\) The BADS score ranges from 0 – 24, with lower scores indicating greater difficulties with executive functioning

\(^b\) The STW is a scaled score with a mean of 10, SD = 3, with lower scores representing poor verbal ability and lower IQ estimate

\(^c\) The COWA-M score is the total number of words scored as correct generated by each participant under time pressure

\(^d\) The BDI-II represents the overall score a participant received, with higher ratings representing higher levels of depression symptoms reported

\(^f\) The TFR score is represented as percentage of participants gaining a Normal score. Note that 94% in the Community Control Group represents 15/16 participants gaining a Normal score.

\(^g\) The Years of Education score represents the total length of time in years a participant has remained in a formal education institution
In order to examine test score differences more closely, Kruskal-Wallis Tests were performed on those tests that were significant at the $p = .05$ level on an ANOVA (BADS, BDI-II, and Years of Education). Table 9 details the results.

<table>
<thead>
<tr>
<th>Participant Type</th>
<th>BADS</th>
<th>BDI-II</th>
<th>Years of Education</th>
</tr>
</thead>
<tbody>
<tr>
<td>Violent<em>Non-Violent</em>Community Controls</td>
<td>18.19</td>
<td>9.21</td>
<td>15.31</td>
</tr>
<tr>
<td>Asymp. Sig</td>
<td>&lt;.001*</td>
<td>.015</td>
<td>&lt;.001*</td>
</tr>
<tr>
<td>Violent*Non-Violent</td>
<td>9.20</td>
<td>.001</td>
<td>3.92</td>
</tr>
<tr>
<td>Asymp. Sig</td>
<td>.002*</td>
<td>.975</td>
<td>.048</td>
</tr>
<tr>
<td>Non-Violent*Community Controls</td>
<td>1.12</td>
<td>6.23</td>
<td>2.67</td>
</tr>
<tr>
<td>Asymp. Sig</td>
<td>.292</td>
<td>.013*</td>
<td>.102</td>
</tr>
<tr>
<td>Violent*Community Controls</td>
<td>15.70</td>
<td>8.62</td>
<td>15.60</td>
</tr>
<tr>
<td>Asymp. Sig</td>
<td>&lt;.001*</td>
<td>.011*</td>
<td>&lt;.001*</td>
</tr>
</tbody>
</table>

* $p \leq .01$ level

Significant differences were found for BADS, and Years of Education across all three Participant Types, and these remained when pairwise comparisons were computed for the Violent Offenders and the Community Controls. In a pairwise comparison between the Violent Offenders and the Non-Violent Offenders, the BADS continued to demonstrate significance. The BDI-II appeared to differentiate incarcerated participants from community participants. Due to the sizeable representation of Māori in both offender types and particularly in the violent offender participant type, a Kruskall-Wallis set at $p = .01$ was computed comparing all participant types for Māori only across the variables of BADS,
STW, COWA-M, BDI-II, Age and Years of Education. Only the BADS remained significant across participant types (p = .005).

As can be seen in Table 10, the trend for significant differences between the groups during pairwise analysis of the profile score for the BADS held when the BADS subtasks were compared across Participant Types. The Modified Six Elements subtask and the Rule Shift Cards subtask demonstrated a significant difference across the entire sample and, in particular, between Violent Offenders and Community Controls.

<table>
<thead>
<tr>
<th>Participant Type</th>
<th>Rule Shift Cards subtask</th>
<th>Action Program subtask</th>
<th>Key Search subtask</th>
<th>Temporal Judgment subtask</th>
<th>Zoo Map subtask</th>
<th>Modified Six Elements subtask</th>
</tr>
</thead>
<tbody>
<tr>
<td>Violent</td>
<td>2.33 (1.68)</td>
<td>3.67 (0.58)</td>
<td>1.95 (1.36)</td>
<td>1.67 (1.15)</td>
<td>1.57 (1.21)</td>
<td>2.48 (1.21)</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>3.44 (0.81)</td>
<td>3.94 (0.25)</td>
<td>3.06 (1.18)</td>
<td>1.69 (1.08)</td>
<td>2.00 (1.15)</td>
<td>3.38 (0.89)</td>
</tr>
<tr>
<td>Community Control</td>
<td>3.63 (1.03)</td>
<td>3.81 (0.54)</td>
<td>2.94 (1.34)</td>
<td>1.56 (0.63)</td>
<td>2.63 (1.20)</td>
<td>3.69 (0.60)</td>
</tr>
</tbody>
</table>

Violent *Non-Violent *Community Control

| Asymp. Sig            | .014*                    | .186                   | .026               | .962                      | .046           | .002*                       |

Violent*Non-Violent

| Asymp. Sig            | .060                     | .087                   | .017               | .911                      | .269           | .018                        |

Non-Violent*Community Control

| Asymp. Sig            | .171                     | .526                   | .871               | .746                      | .155           | .261                        |

Violent*Community Control

| Asymp. Sig            | .008*                    | .285                   | .034               | .909                      | .016           | .001*                       |

* p ≤ .01 level
Of further interest is the better performance of the Non-Violent Offender group on the Key Search (primarily a planning task) subtask and the Temporal Judgement subtask, although these were not found to reach significance. The Temporal Judgement subtask was marked by poor performance by all groups; it is notable that there was a shared difficulty across the sample with this task.

4.3.4 Relationship between Executive Dysfunction and Participant Type

A chi-square cross-tabulation was conducted to examine the relationship between Participant Type and the combination of quantitative data (test score on BADS) and qualitative data (observations and 1 – 5 rating on the Likert Scale of Appropriateness). An illustration of ‘5’ (extremely inappropriate behaviour) on the Likert Scale was a violent offender who had difficulty staying in his seat, would walk around the room frequently, going through cupboards and examining items part way through testing, and would interrupt constantly to ask questions of a personal nature. He continued this behaviour even when warned that it was unacceptable. Examples of a ‘1’ are participants who remained focussed for the entire session, appeared proficient at following instructions and demonstrated an absence of inappropriate personal questioning. A cross-tabulation was run comparing Participant Type with the combination of the BADS score and the Likert score (resulting in a score of ‘patterns of ED,’ see Table 5 in the Methods).

A Pearson Chi-square test applied to the relationship between participant type and ED pattern across all three participant types reached significance, $\chi^2 (2, N = 53) = 16.75, (p < .001)$. Furthermore, in a Spearman’s rho rank correlation computation between scores gained on the BADS and scores gained on the Likert Scale of Appropriateness a strong negative correlation was found (-.59, p < .001), demonstrating that participants with poor BADS scores were likely to also gain higher Likert scores (3, 4, and 5) indicating inappropriate behaviour.

---

3 See Appendix One for Table 17 – General Results of Study One - that details each score received on the Likert Scale for all participants, along with their test scores.
More violent offenders demonstrated a clear ED pattern than did the other two participant types. There was a significant differentiation between violent offenders and non-violent offenders ($\chi^2 (1. N = 53) = 9.10, p = .002$). A comparison of violent offenders and community controls also resulted in a significant difference ($\chi^2 (1. N = 53) = 11.96, p \leq .001$). In contrast, no significant difference was found between the types of ED pattern for non-violent offenders and community controls $\chi^2 (1. N = 53) = .368, (p = .544)$. A Pearson Chi-square test applied to the relationship between Participant Type, Māori ethnic identity, and ED pattern did not reach significance, $\chi^2 (2. N = 27) = 4.500, (p = .105)$.

4.3.5 Substance Abuse

Forty-four of the participants across the sample reported actively using drugs recreationally in the past. Some described histories of using any ‘pill’ or ‘powder’ that came their way, and many reported using marijuana. Some participants reported using marijuana exclusively; often daily. One inmate reported regular heroin use before his incarceration. A cross-tabulation examining the historical use of drugs across the Participant Types indicated it was most common in incarcerated offenders when compared with community controls. A Pearson Chi-square value of $\chi^2 (1. N = 31) = 10.096, (p \leq .001)$ was found in a cross-tabulation of violent offenders and community controls, along with $\chi^2 (1. N = 31) = 10.109, (p \leq .001)$ for non-violent offender participants and community control participants. No difference was seen between violent and non-violent offenders.

Thirteen participants across the sample reported smoking marijuana in the 48 hours prior to the testing. Marijuana was the only substance reported by participants as having been consumed in the last 48 hours. Spearman correlations were run to establish if this had an effect on the neuropsychological scores gained. None of the covariates run demonstrated a relationship between current drug use and neuropsychological scores. Current drug use and conviction type established a
covariate of .31 (p=.026) and may be explained by more incarcerated men reporting using marijuana in the last 48 hours than men in the control group. However, differences between the participant types regarding use of marijuana in the previous 48 hours when examined by crosstabulation did not reach significance at $\chi^2 (2, N = 53) = 6.312, (p = .043)$.

In a closer examination of the means with the current drug users removed, the significant results for BADS, BDI-II and Years of Education in the violent population compared to community controls was maintained. The difference in BADS scores between the violent offenders and non-violent offenders also remained significant (refer to Table 11).

### Table 11: Results of Kruskal-Wallis Tests across all the Participant Types for total BADS score, BDI-II and Years of Education with participants who have reported drug use in the last 48 hours removed

<table>
<thead>
<tr>
<th>Participant Type</th>
<th>BADS</th>
<th>BDI-II</th>
<th>Years of Education</th>
</tr>
</thead>
<tbody>
<tr>
<td>Violent<em>Non-Violent</em>Community Controls</td>
<td>16.29</td>
<td>11.22</td>
<td>10.81</td>
</tr>
<tr>
<td>Asymp. Sig</td>
<td>&lt; .001*</td>
<td>.004*</td>
<td>.004*</td>
</tr>
<tr>
<td>Violent*Non-Violent</td>
<td>9.63</td>
<td>1.01</td>
<td>2.31</td>
</tr>
<tr>
<td>Asymp. Sig</td>
<td>.002*</td>
<td>.314</td>
<td>.13</td>
</tr>
<tr>
<td>Non-Violent*Community Controls</td>
<td>.79</td>
<td>6.21</td>
<td>2.65</td>
</tr>
<tr>
<td>Asymp. Sig</td>
<td>.376</td>
<td>.013</td>
<td>.103</td>
</tr>
<tr>
<td>Violent*Community Controls</td>
<td>14.34</td>
<td>9.40</td>
<td>11.44</td>
</tr>
<tr>
<td>Asymp. Sig</td>
<td>&lt; .001*</td>
<td>.002*</td>
<td>.001*</td>
</tr>
</tbody>
</table>

* $p \leq .01$ level
When the relationship between the categories (*Impaired*, *Borderline*, *Low Average*, *Average*, *High Average*) obtained on the BADS neuropsychological test (the primary measure of ED) were computed against use of drugs within the last 48 hours, no relationship of significance emerged, $\chi^2 (4. N = 53) = 2.846, (p = .584)$, further suggesting that the BADS results were not substantially influenced by recent marijuana use.

Fifty-one participants across the sample reported drinking heavily in the past, and a Pearson Chi-square value did not reach significance at $\chi^2 (2. N = 53) = 1.364, (p = .506)$ in differentiating Participant Type. No participants reported using alcohol 48 hours prior to participation.

Self-reported drug usage or intoxication during offending by incarcerated participants did not differ significantly between the violent and non-violent offenders. Twenty (95.2%) violent offenders and 14 (87.5%) non-violent offenders reported they were under the influence of substances when they offended. A Pearson Chi-square value of $\chi^2 (1. N = 37) = .730, (p = .393)$ indicated that intoxication through alcohol and/or drugs was more common when offending than offending whilst not under the influence.

### 4.3.6 Traumatic Brain Injury

The self-reported rate of TBI history was very high across all participants; 100% of violent offenders reported a TBI history, and 86% reported a TBI prior to their first conviction. Similarly, 94% of the non-violent offenders reported a TBI history; with 81% before first conviction. Sixty-three percent of community controls reported a TBI history. These reports of a history of TBI, although all high, were significantly different across the three participant types $\chi^2 (2. N = 53) = 12.108, (p = .002)$. When pairwise comparisons were made, the significant difference at $p = .01$ remained between the community controls and the violent
offenders $\chi^2(1. N = 37) = 9.399, \ (p < .002)$. Acquiring a brain injury before first conviction is common in the incarcerated groups and the proportion of violent offenders who sustained a TBI preceding conviction as compared to the non-violent offenders is not significantly different ($\chi^2(1. N = 37) = .133, \ (p = .715)$.

4.4 Discussion

A substantial number of inmates in Study One met criteria set by the BADS for executive dysfunction. Of the 21 violent offenders, 18 (86%) of these participants met criteria for a Low Average, Borderline, or Impaired performance. In contrast, 5 of the 16 (31.3%) non-violent offenders and only 2 of the 16 (12.5%) non-violent community controls fell into these ranges.

A greater number of violent offenders demonstrate executive impairment, as measured primarily by the BADS. Violent offenders had a mean score of 13.5 (Borderline), compared with the other participant types who both had mean scores indicating performance in the Average range. The results provide support for the first hypothesis in that there are significant neuropsychological differences between the three participant types. A higher proportion of the violent offender participants were found to have ED as measured by the BADS than both the non-violent offenders and the community controls. The significant differences on the BADS between the participant types remained when run for Māori only (the largest ethnicity group).

The finding that men who have been repeatedly convicted of crimes of physical violence are likely to demonstrate executive functioning deficits upon testing, supports the inmate and forensic psychiatric literature reviewed in this thesis (Williams, 1969; Spellacy, 1978; Yeudall & Fromm-Auch, 1979; Gorenstein, 1982; Convit et al., 1991; Foster et al., 1993; Raine et al., 1994; Goyer et al., 1994; Blake et al., 1995; Volkow et al., 1995; Amen et al., 1996; Intrator et al.,
It also supports literature from community samples of acquired brain injury and aggression, community antisocial personality disorder (APD) samples, and provoked aggression via a laboratory evoked reduction in executive function (Amen, 1999; Blair & Cipolotti, 2000; Damasio et al., 1990; Deckel et al., 1996; Gantner & Taylor, 1992; Giancola & Zeichner, 1994; Gordon et al., 2004; Grafman et al., 1996; Lau et al., 1995; Oder et al., 1992; Peterson et al., 1990; Raine et al., 2000; Raine et al., 1990). The results of this study are similar to previous research specifically comparing violent offending with non-violent offending has demonstrated more executive dysfunction in the violent offender group compared with non-violent offenders (Spellacy, 1978; Valliant et al., 1999). However, they are at odds with other studies with offenders have demonstrated minimal deficits in executive function or other neuropsychological functions, regardless of crime, or demonstrated minimal differences between offenders and controls (for example, Hart et al., 1990; La Pierre et al., 1995; Deckel et al., 1996). Even where differences have been demonstrated, the results are often not robust due to design factors. The improvements made to methodology in this study may account for finding more ED in violent offenders then in these previous studies.

Earlier research using non-violent groups has tended to use offenders with fraud and drug offences, who can be considered so different from violent offenders that they comprise an inappropriate comparison group, and significant differences demonstrated may not be useful. This may be particularly so when comparing a non-violent group comprised of traffic offences with a violent group compromised of murders and/or sexual offenders (for example Valliant et al., 1999). The non-violent offenders in the current study were better matched to the violent offenders on all variables other than violence than previous studies. Thus, to see such a large effect size between the BADS scores for these two groups, when so many
similarities exist, adds further support to hypothesis one. Explanations for why the BADS appears appropriate for the measurement of the constructs underpinning violence will be explored in the *General Discussion* in Chapter Seven.

The second hypothesis that inappropriate behaviour would be associated with poorer neuropsychological scores (on the BADS) was supported. Almost 62% of participants in the violent offender group demonstrated a pattern of clear ED (BADS score combined with Likert score), compared with 12.5% of the non-violent offenders and 6.3% of the community participants. However, it is noted that my clinical judgement of a participant’s behaviour and their quantitative results did not always match. For example, a pattern of average or better outcome on the BADS alongside inappropriate test behaviour was found for two participants, and four participants demonstrated appropriate behaviour while gaining poor BADS scores. This latter result however is not surprising, as the structure provided in a testing environment aids inhibition of inappropriate behaviour, thus control of behaviour is easier to maintain. Notably, a testing session lasted a maximum of two and a half hours, including breaks. Thirteen violent offenders and 2 non-violent offenders scored poorly on the BADS alongside inappropriate test behaviour as measured by the Likert Scale, resulting in meeting criteria set by this study of a ‘Clear ED Pattern.’

It could be argued that those individuals who demonstrated both a poor BADS score and a poor Likert score were merely reflecting off-task behaviour that interferes with performance on the tasks. It may be that participants scored poorly on the BADS due to an inability to stay on and complete a task, and this inability to maintain task was scored highly on the Likert Scale, forming a tautology. However, all participants were able to complete the BADS subtasks in the time given. All off-task behaviour came between the subtasks, supporting the use of the BADS with individual subtasks, each taking 2-10 minutes. Additionally, the overall testing situation was conducted in a manner that allowed many breaks to
minimise test anxiety, boredom, and restlessness, with the goal of maximising test performance on each task.

Drug use, alcohol use, and TBI were common across the sample, with a significant difference demonstrated in reported histories of drug use between the incarcerated participants and the community control participants. A history of TBI also differentiated violent offender participants and community control participants, with violent offenders more likely to report a previous TBI. Initially it was of concern that many men had used marijuana in the 48 hours prior to testing. Particularly concerning was that this might account for the poor scores on the BADS. However, reanalysis with those men who reported being drug free demonstrated strong significant differences remaining across the three Participant Types for the BADS, BDI-II and Years of Education. The influence of TBI, drugs, and alcohol will be examined more fully in the General Discussion (Chapter Seven) alongside results from Study Two.

Many relationships of interest were found in the computation of the Spearman rho correlations. As well as the predicted strong relationship between Participant Type and the BADS, Participant Type also had significant relationships with Years of Education and the BDI-II. These latter two relationships may reflect the significant difference demonstrated in the demographics of the spread of Years of Education across the sample, and the difficulty many of the incarcerated inmates had in answering the questions of the BDI-II (discussed further in the following sections). The BADS itself further demonstrated strong relationships with Years of Education, the estimate of Verbal ability (STW), and the BDI-II. The relationships between the BADS and STW and Years of Education are expected given that neuropsychological tests tap into academic knowledge and verbal ability. The strong correlation of the BADS with the BDI-II is of concern and will be discussed further in this discussion under the heading The Beck Depression Inventory - II.
The spread of the demographic data across the three Participant Types initially caused concern in that it may be a limiting factor of the results. However, statistical analysis of the demographic characteristics across the three Participant Types demonstrated that only Years of Education reached significance. This is likely to be an effect of the relatively high mean Years of Education for the control participants from a very small sample, combined with an outlier of 4 Years of Education for one of the violent offender participants. However, this significant finding needs to be recognised when interpreting the scores gained by the participants on neuropsychological tests and attempting to make associations between neuropsychological performance (which is influenced by educational experience) and group membership. For example, more men in the violent offender group had poor scores on the BADS, but similarly these men had the lowest mean Years of Education. Conceivably there may be some link between Years of Education and the BADS, which may have an additive effect that contributes to poor decision-making, or one may lead to the other, e.g., poor executive functioning might interfere with on-task behaviour and learning at school.

**Behavioural Assessment of the Dysexecutive Syndrome**

The relationship between the BADS score and participant type was the strongest of all the findings in Study One. The profile scores gained by violent inmates are in line with profile scores demonstrated by brain-injured populations, demonstrating the clinical utility of the BADS in measuring ED in a violent offender sample. For example, in this study violent offenders gained an average score of 13.5, compared with 14.03 for one brain injured sample (Wilson et al., 1998) and 15.6 for another mixed neurological dysfunction population (Norris & Tate, 2000). The control data is also similar, with 18.25 for non-violent community controls in this sample compared with 18.05 for healthy controls in Wilson et al.’s (1998) study and 18.05 in Norris and Tate’s (2000) study.
Additional evidence for the sensitivity of the BADS in measuring executive components in the non-clinical population of inmates and community controls is that the subtask scores gained by the violent offenders and the community controls are in line with those gathered by brain injured subjects and healthy normals (Norris & Tate, 2000; Wilson et al., 1998). For example, in the subtasks that were significantly different between violent offenders and community controls, the violent offenders scored a mean converted score of 2.33 (1.68) on Rule Shift Cards compared with a brain injured sample score of 3.31 (.92) from Norris & Tate (2000), and 2.48 (1.21) on Modified Six Elements in line with 2.83 (1.42) scored by Norris and Tate’s (2000) neurological dysfunction sample. The control data demonstrated similar parity.

When attempting to create a stronger gauge of ED by combining performance on tests alongside test behaviour, rather than the test scores alone, again the violent offender group had the largest number of participants in the sample that could be classified as having a ‘Clear ED Pattern.’ However, caution is needed in the interpretation of the Likert Scale scores plus the BADS, as although every precaution was taken to be objective in the ratings applied it is acknowledged that context (for example, interview room locations) may influence Likert Scale ratings. In testing of the inmates I was blind to their conviction histories. However some clues to Participant Type were available to me according to what part of the prison I was sent to in order to carry out the assessment (for example, maximum or minimum security). This may have biased my ratings on the Likert Scale. Additionally, I was aware when I was assessing a community control subject, and could deduce their participant history due to the participation criteria for control participants of nil criminal background. In future studies a selection of standard rooms where all testing is to be completed, with offender and community participants, allocated to rooms by an independent person may help to reduce this potential source of bias. Independent police checks on all participants would also be of benefit.
The executive functions measured by the BADS that the incarcerated participants had the most difficulty with were cognitive flexibility, inhibition of prepotent responding, perseveration, difficulty in maintaining goal directed behaviour, poor prospective memory, and poor self-monitoring. These represent those functions measured by the Rule Shift Cards, and the Modified Six Elements.

The Rule Shift Cards subtask differentiated violent offenders from community controls. This task has an attentional element, taps into cognitive flexibility, the ability to inhibit prepotent responding, and the ability to learn and apply new information in the face of previously learnt distracting stimuli. Problems in these areas may indicate difficulties with perseveration or being stimulus bound. In a prison environment this could have important consequences for those unaware of environmental triggers of negative behaviours.

The Modified Six Elements subtask differentiated between the violent offenders and the controls. This task required the participant to hold many variables in prospective memory in order to carry out an intention at a future time, while also making good use of a specified time interval. The poor scores on this task by violent offenders are consistent with Norris and Tate (2000), who found the Modified Six Elements a robust predictor of prefrontal brain dysfunction.

The Rule Shift Cards and Modified Six Elements are complex tasks designed to measure ED in an ecological manner and the results suggest that inmates convicted of violent offences would benefit from assessment for difficulties in self-monitoring and adaptation, prospective memory, and the ability to set and reach goals. This would help maximise any gain from rehabilitation, and to improve their chance of being integrated back into society with a reduced likelihood of re-offending.

The Zoo Map Test has a heavy planning component and relies in part on the ability to anticipate consequences of current decision-making. It was surprising
that this subtask failed to significantly differentiate between participants while other subtasks measuring similar components did so. When scoring this subtask some concerns emerged regarding the scoring criteria. For example, a quick performance combined with a long planning time is given a good score; however this style does not always indicate an optimal performance. When a participant does not succeed in visiting the required and appropriate places on the map, the long time taken to ‘plan’ may actually reflect very poor processing. This subtask contends that minimal planning time is a reflection of impulsivity and therefore a long period spent before starting demonstrates good planning. However, I often observed a lot of time taken before starting, but performance would prove that this time was not necessarily spent planning. For example, one participant scored ‘above average’ for performance on the Zoo Map even though in Zoo Map 1 he scored only 3 correct in sequence (out of a possible 8). In accordance with the scoring rules, he was rewarded for taking a long time to ‘plan,’ even though any pay-offs for planning were not demonstrated. The current scoring protocols in Zoo Map may mask any possible difficulties that this subtask purports to measure.

A similar example of scoring protocols masking dysfunction was described by Hoaken, Shaughnessy and Pihl (2003). Using the Go/No-Go test, a test of impulsivity, they found aggressive participants with poor executive functioning had longer response times than those participants with good executive functioning. This was contrary to previous research, which demonstrated that faster response time was linked with impulsiveness in aggressors. The Go/No-Go could not account for the longer response times, resulting in a measure of nil deficit. The authors concluded that in the aggressors participating in their study, it was not impulsivity that was linked with aggression, but poor general and social processing (Hoaken et al., 2003). Even though the Go/No-Go is employed as an executive test (usually with aggressive participants), Hoaken et al., (2003) argue that the test ineffectively accounts for ED and aggression that is associated with compromised processing ability. They emphasize that ‘good’ results on this test do not necessarily imply that no brain dysfunction, or deficits in behaviour, exist.
The poor social processing suggested by Hoaken et al. (2003) may be related to why some men in the current study took a long time at the planning-stage in the Zoo Map. Another explanation for this processing difficulty is social anxiety. It may be that the men having trouble benefiting from planning time did so because of general anxiety or social anxiety in the testing situation. This was also part of an explanation put forward when the observation of a long ‘planning’ time and subsequent poor planning was raised in personal communication with one of the authors of the BADS, who stated that:

“One should very much bear in mind that: (a) at the extreme it is doubtful that more time spent planning should be interpreted in a positive way; [and] (b) a long time spent planning followed by a subsequently poor performance suggests that the participants’ planning skills are not good, or might alert one to other variables to be considered (e.g. anxiety; factors which influence execution of the plan etc.), which may be complex to interpret.” (Personal Communication; Burgess, 28 July; 2004).

Future research could explore ways of discriminating good and poor use of planning time and design more valid scoring rules for the Zoo Map Test.

A subtask of the BADS considered to have demonstrated little utility was the Temporal Judgment Test. It is suggested that the difficulties seen with the Temporal Judgment subtask across all of the groups is a general flattening of effect due to the types of questions asked. For example, “How long does it take a window cleaner to clean the window of an average size house?” invariably gained responses ranging from an hour and up, at odd with the scoring rule: “Score 1 if between 15 and 25 minutes, otherwise score 0.” New Zealanders typically have large homes with many windows. Poor performance on this task may have artificially lowered the scores of all participants, but as failing on this test item was universal it did not change the rankings of the participants from best to worst score. However, it is clear that questions like this make sense culturally to United Kingdom residents, but not to New Zealanders, and the results suggest that in the future these questions should not be used with a New Zealand population. Socio-
economic status may also influence how participants answer the temporal questions. For example, a large proportion of the men when asked, “How long does it take to do a routine dental check up?” stated that they had never been to a dentist. However, the validity of temporal reasoning as a component of executive functioning remained with the questions, “How long do most dogs live for?” and “How long does it take to blow up a party balloon?” Over-estimates and under-estimates on these questions could be used validly with New Zealanders.

Norris & Tate (2000) also found difficulties with the Temporal Judgment subtask using an Australian population. However, they did not comment on possible cultural differences, instead they stated that the task was unreliable due to only four questions being asked. This assertion was supported by a poor correlation with the Cognitive Estimations Test (Shallice & Evans, 1978). Norris and Tate (2000) in their sample of 36 brain-injured and neurologically impaired participants demonstrated an average score of 1.47 on the Temporal Judgement task, and healthy controls averaged a score of 1.68. This is similar to scores gained by participants across Studies One and Two.

Overall the result of the BADS identifying ED more frequently in violent offenders is intriguing and requires further exploration. It is a possibility that the BADS may measure a function, that when compromised, is a pathway to violence or at least additive to existing pathways (or propensity) to violence in recidivist violent offenders. It is suggested that the ED measured by the BADS is likely to inform the underpinnings of violence rather than the recidivism. If the BADS were measuring behaviour underpinning the recidivism only, similar effect sizes should have been demonstrated in the non-violent group as well.
Years of Education

The number of formal years of education each participant had received was associated with Participant Type; on average, violent offenders had the least years of education while the non-violent community control group had the most. This measure was second only to the impact of the BADS score on Participant Type. It is an expected result as ‘academic ability’ is a well-known factor in performance on neuropsychological tests. Years of Education also correlated significantly with the BADS and STW.

Unfortunately, in spite of an attempt to match the control group with the offender groups for Years of Education, mean Years of Education was higher for the control participants than the mean for each of the two offender types. This may account for a proportion of the association between Years of Education and Participant Type. The lowest reported number of Years of Education was from a violent offender who stated “ending (my) education” in Standard Three due to the break-up of his family and entering into the first of many foster homes. This resulted in him attending school for only four years. In such a small sample this outlier may skew the spread of reported Years of Education. It may be that a larger sample would have smoothed this effect and it would be expected that the impact of Years of Education would reduce. However, there is a trend noted in the results between the men who gained a low BADS score and who also left school early. For example, the man with four years of education also gained a ‘Low Average’ range score on the BADS and scored in the 1st percentile for STW.

In attempting to account for the association of BADS with Years of Education and the effect of Years of Education on Participant Type, it may be that the participants with poor executive functioning found school lessons harder to attend to and benefit from, so left school earlier. Individuals struggling with executive functioning may well do poorly at school and leave school early, or are forced to leave because of inappropriate behaviour. Indeed, many men in the offender
groups reported being “expelled.” The decision to leave school early, or being expelled from school, could also be reflective of poor decision-making skills, lack of goal directed behaviour, and impulsivity associated with ED. Success in the New Zealand school system in senior classes depends to some extent on healthy executive functioning. Particularly important may be the functions of inhibition, self-regulation, self-monitoring, and rule following. Formal education also provides a context that reinforces executive functioning, so it is expected that those with more Years of Education would have better BADS scores, as they have had a longer exposure to a structured environment that promotes and supports maturation of executive functioning.

Higher cognitive ability may be a protective factor curtailing pathways to violence. The results indicated in Study One may reflect this, and the elucidation of ED as a pathway to violence may also stand for lack of exposure to education. Additionally, it is suggested that ED and Years of Education have an additive effect on strengthening pathways to violence. Worthy of further examination is the link between intelligence, particularly verbal intelligence as we currently measure it, and executive function. When Villemarette-Pittman, Stanford, and Greve (2002) examined a population that was regularly impulsively aggressive they were expecting to find deficits in verbal intelligence. Instead of finding language ability as a covariate they found that inefficient executive functioning impacted on competent completion of verbal tasks. The theory of unity can help explain this finding; that executive functioning impinges upon many tasks required of the brain (Duncan et al., 1997), or that the expression of executive functions relies on the competency of other domains. This explanation is similar to Spearman’s ‘g’ being regarded as performing a similar role as the CE or SAS in monitoring and executing the executive functions. Further, that when the ability to use executive function is measured one is measuring fluid-intelligence (Duncan et al., 1995; Zook et al., 2004).
Other authors disagree with an all-encompassing unity view of how executive function works, and indeed how it encroaches on intelligence or ‘g.’ Andrewes (2002) for example, suggests that this approach is too simplistic and is based on old and arbitrary statistical analysis. However, Zook et al. (2004) in their recent examination assert that it is ‘g’ that is measured by traditional frontal tasks, and that the concept of ‘g’ or fluid-intelligence, best explains the variance measured on these tests, while acknowledging that different executive tests do indeed measure different types and severities of executive functions. It is tentatively suggested that the BADS is a useful test battery in this research as it can encompass the global ‘g’ element of executive function as well as attempt to sensitively measure the components that are encompassed by executive function.

*The Beck Depression Inventory-II*

The BDI-II was significantly related to participant type across the sample and remained significant for the analysis between the offending participants and the community controls. A negative correlation demonstrated an inverse relationship between the BADS and the BDI-II. As BADS scores dropped (indicating ED), depression scores on the BDI-II rose (indicating low mood). This is a concern as it is documented in the literature that depression can impact negatively on neuropsychological results, and particularly upon executive functions (Grant, Thase, & Sweeney, 2001). Interestingly, Crew, Harrison, and Rhodes (1999) found no reliable differences when examining neuropsychological tests of executive function alongside measures of depression in depressed and non-depressed women. The authors state that their findings are an argument against “stereotypes” of depression negatively impacting on neuropsychological tasks. This was replicated by Rohling et al. (2002) in 420 patients with brain injury or neurological disease (54% male) assessed on both depression and neuropsychological measures. It was also replicated later by Lampe, Sitskoorn and Heeren (2004) in a group of women with depression matched to healthy
controls. Both groups of authors found no significant interactions or correlations between depression scores and executive function scores.

My study differs from these studies that have found a minimal association between ED scores and depression scores. Examination of scores across the sample reveals 50% (seven cases) of all participants with a Borderline or Impaired BADS score had a BDI-II score of Moderate or Severe depression. However, upon further questioning of the men using DSM-IV (American Psychiatric Association, 1994) criteria, all men who gained high scores on the BDI-II were found not to meet criteria for a Major Depressive Episode. It may be that the small sample size limits what can be concluded from finding these rates of depression according to the BDI-II. Moreover, another explanation for the high number of inmates demonstrating poor BDI-II scores may lie in the types of questions asked, and their appropriateness for a prison sample. Questions that may have been invalid as symptoms of depression for this population were those related to guilt, punishment, and loss of interest. Even when careful explanations were given about the context of answering almost all inmates would rate these questions relatively highly, and would discuss with me issues around being incarcerated compared with living in the community, as well as going to court and being convicted, rather than focusing on the last two-weeks of mood and behaviour. The inmates appeared to hold distinct ‘role’ and self-beliefs about ‘inside-of-prison’ and ‘outside-of-prison.’ Also, some inmates appeared to be telling me what they thought I wanted to hear, stating they felt guilty because they “supposed” they should. Moreover, the manner in which some participants answered questions may have added to high scores, such as reading quickly, answering impulsively not accounting for context, and difficulty comprehending the instructions. These response styles may reflect ED.
Other Variables of Interest

Given that poor BADS performance was seen significantly more in violent offender performances, any possible relationship between STW and Participant Type becomes important in attempts to clarify the reasons why performance on the BADS was poor. However, as demonstrated in analysis, the STW measure does not have a significant relationship with Participant Type, increasing the robustness of the association of BADS with Participant Type. Relationships for STW were demonstrated with Years of Education and COWA-M; perhaps demonstrating that these two tests both measure similar verbal abilities.

The lack of association between verbal tests and Participant Type is at odds with Halibrun (1982), who found a connection between low verbal ability and criminality. It also contrasts with later studies that have demonstrated impairments in verbal tasks by violent offenders (Nestor, Kimble, Berman, & Haycock, 2002). Halibrun (1982) has suggested that intelligence moderates violence or offending type in those inmates who score highly in psychopathy checklists, but it may simply be that the more intelligent are better at not getting caught. Some of the literature that has examined violence neuropsychologically has found low intelligence was a differentiating factor in aggressive individuals compared to non-aggressive individuals (Chance et al., 2000; Giancola & Zeichner, 1994; Turner, 1994). However some authors argue that this can be explained using a task-impurity assumption, stating that often what is measured as language ability is actually relying heavily on executive components (Brinkley, Bernstein, & Newman, 1999; Stanford, Greve, & Gerstle, 1997; Villemarette-Pittman, Stanford, & Greve, 2002) and therefore will possibly measure the same deficits as those measured by standard executive tests.

A task-impurity analysis may be pertinent to tasks that include elements of spontaneous organization, like the COWA for example. Either explanation however (verbal deficits or task-impurity in aggressors or psychopaths compared
to normals) does not account for why in this sample, no significant differences in performance on the STW and COWA-M are demonstrated across the participants. Performance on the STW in all groups was poor relative to the expected mean of 10, with the violent offenders scoring more than one SD below the mean, and the other groups within one SD, indicating a trend toward violent offenders performing more poorly on the STW.

It appears the COWA-M was measuring a similar ability to STW with these two tests correlating well. It is surprising that the COWA measure commonly used to assess both executive function and fluid verbal ability was not related in any significant way to Participant Type or to the BADS. Using the COWA-M, violent offenders averaged 32.05 words compared to 37.37 for non-violent participants, and 37.75 for community controls. Although the violent offenders generated fewer words they were not significantly different from the rest of the sample. It also appears that the COWA-M was equally appropriate for Māori (mean = 36.2 words generated) and Pākehā (mean = 40 words generated), with no significant difference detected between the different ethnic groups, and all groups within a standard deviation of published norms (Ogden et al., 2003). However, six participants identifying as Pacific Islands People scored a mean of 28 words generated across the task, which was probably a result of the majority of these participants having English as a second language.

The lack of interaction between the COWA-M and the BADS may cast doubt on using verbal fluency and spontaneity under time-pressure as a valid measure of executive functioning. That the COWA-M was not sensitive to any deficits, be they executive or language deficits, is an unusual finding. The COWA has been repeatedly demonstrated to tap into both these functions (Villemarete-Pittman et al., 2002), with poor scores in the phonemic task related to focal frontal lesions (Henry & Crawford, 2004).
Several explanations may account for this finding. Using the Māori version may have ultimately changed the nature of the test. This is unlikely however, as norms published for the COWA-M are in line with other norms for other letter combinations, such as F.A.S (Spreen & Strauss, 1991), previously published norms for T.P.W (Borkowski et al., 1967) and the norms developed for a New Zealand population (Ogden et al., 2003). Another explanation could be that the COWA-M is measuring components of executive function different from those the BADS measures, and that those components measured by the COWA-M are not impaired. It may be that the BADS is sensitive to minor dysfunctions that are more likely to be apparent in daily living. Earlier work supports that notion by demonstrating that the COWA and the BADS profile score are not related (Norris & Tate, 2000; Wilson et al., 1998), and that the COWA only demonstrates minimal correlation with the Action-Planning subtask (Norris & Tate, 2000). It could also be that the COWA-M is measuring a verbal ability similar to that of the STW, and that the scores simply reflect that this function isn’t impaired significantly within or across the groups. The finding of minimal impairment on the COWA-M requires further exploration and will benefit from being used in other studies in an attempt to clarify its role in measuring executive function(s).

**Ethnicity**

When an analysis of means was conducted with test scores and other variables, using Ethnic Identity as the grouping variable, STW and Years of Education reached significance across Participant Types. These results can reduce the significance of the main findings, as they may be influenced by ethnic/cultural differences. In particular there may be a ‘language effect,’ influencing the neuropsychological tests. One of the criteria for inclusion in the study was the ability to read and write in English at the level of Intermediate School. However, the study included men from different backgrounds not raised in New Zealand, primarily Pacific Islands participants. This group may have had more difficulty with the language and concepts of the testing. This could account for the
significant finding for STW, with the six participants identifying as Pacific Islands Peoples gaining an average scaled score of 5.7, the poorest mean score across the ethnic groups.

Years of Education demonstrated a similar divide, with Māori and Pacific Islands participants reporting fewer Years of Education than Pakeha. There are many reasons why people finish school early, and some have already been outlined above. It may also be that a New Zealand European based school system is difficult for children who do not identify as Pakeha or European. Leaving school early for Māori participating in this study is congruent with the New Zealand statistics (Statistics New Zealand, 1997; Statistics New Zealand: Te Tari Tatau, 2001).

Results for Māori were included in the overall analysis and also analysed separately due to the large percentage of Māori participants (50.9%) in the sample. Furthermore, a large proportion of Māori were represented in the violent offender group (66.7%), and non-violent offender group (50.0%). These figures do not reflect the proportion of Māori in the general population (14.7%; (Statistics New Zealand: Te Tari Tatau, 2001), but are similar to the New Zealand-wide representation of Māori in prison (51%; (Rich, 2000). A further explanation for the high representation of Māori in this study is that Auckland Prison’s catchment includes Auckland and Northland. Auckland plus Northland is an area of New Zealand that has a high proportion of Māori, with 43.2% of the 14.7% of population identifying as Māori in New Zealand residing in these areas (Statistics New Zealand: Te Tari Tatau, 2001). Note that the proportion of Māori in the control group (31.3%) is higher then the proportion of the New Zealand population identifying as Māori, but as fewer Māori are in the control group than in the incarcerated group there were a number of difficulties trying to match the groups in later analyses.
When the results were analysed for Māori participants only, differences emerged when compared with the total sample. Significant differences (p ≤ .01) for Māori were found for the BADS scores between the control participants and the violent offender participants. However no significant differences were found between violent offenders and non-violent offenders. That is, all Māori who were incarcerated had more similarities than differences, and as a group differed from Māori controls on the BADS.

In retrospect it is considered that more Māori should have been included in the community control sample in order to be able to make clearer comparisons with the incarcerated offenders who identified as Māori. The low proportion of Māori in the control group when compared to the offending groups is likely to have had a confounding effect on the statistical analyses. A larger sample size that is more representative of the ethnic make-up of New Zealand, but is large enough to include many Māori in the community control group is required to make firm conclusions about the impact of ethnicity on the results.

Another consideration in using neuropsychological test results to predict participant type is the cultural fairness of the tests. It may be that some tests measure abilities other than those they were designed to measure on the normative population (often from the United States of America or the United Kingdom). However, even with the cautions of limited sample size, the under-representation of Māori in the control sample for comparison purposes, and tests that may not be culturally fair, Māori as the largest ethnic group do demonstrate differences in BADS scores that remain relevant and significant between violent offenders and community controls. This reflects the findings of the total sample and supports the first hypothesis that more inmates with a history of violent offending will display executive deficits, than community controls.
4.5 Conclusion

The association of ED with violence has been proposed countless times by numerous authors. Unfortunately in the majority of studies investigating this association, methodological limitations have obscured the results and hence have limited application. Clear findings built on strong methodology that allow for the untangling of the many variables that feed into violence, criminality and executive function, are required in order to develop informed and effective treatments for individuals that offend violently. Methodological inconsistencies in the literature regarding the measurement of violence and the measurement of ED in aggressive individuals were addressed in this study. It is suggested that the methods used, including a clear and precise operational definition of violence, the psychometric tests selected, and the use of two defined experimental groups and a matched control group have strengthened the findings of Study One. Additionally, the strong design of this study, yielding robust and significant findings, improves the potential application of these results. The findings that a substantial number of recidivist violent offenders have ED can be used to inform future interventions for violent inmates with ED. The other large effect, Years of Education, may have application in informing preventative (for criminality) strategies. It is suggested that ED and poor education are additive to existing pathways to violence, in line with by Raine and Buchsbaum (1996).

This study is limited by the sample size, though the use of non-parametric methods helps to increase robustness. Nevertheless, even with the strong and significant effect sizes demonstrated, the small and confined sample of measurement limits the generalisability of these results to other settings and samples. Replication of the study and improvements on the design would be useful for future studies in addressing questions regarding the sensitivity of the tests utilised and the impact of an individual’s education on criminality. Limitations and indications for future studies are discussed more comprehensively in the General Discussion located in Chapter Seven.
5

Study Two: Neuropsychological Differences Between Impulsive and Premeditated Murderers

5.1 Introduction

In studying violence, the literature is beginning to highlight the importance of distinguishing between predatory or premeditated violence, and impulsive violence. Premeditated aggression or violence is considered as purposeful and controlled violence, which is usually instrumental in nature (Stanford et al., 2003). In contrast, impulsive violence has been described as reactive and emotionally charged and characterized by a loss of behavioural control (Barratt & Felthous, 2003; Raine et al., 1998; Stanford et al., 2003). Further, this distinction has proved useful in distinguishing appropriate treatment interventions according to the typology of aggressive behaviour (Barratt, Stanford, Felthous, & Kent, 1997).

A common finding in the literature is that there are minimal neuropsychological differences between premeditated and impulsive aggressors (Barratt, Stanford,
Kent, & Felthous, 1997; Stanford et al., 1997; Stanford et al., 2003). However, this sits alongside imaging literature that clearly identifies brain dysfunction differences between the two types of aggressors (Raine, Buchsbaum et al., 1997; Raine et al., 1994; Raine et al., 2000; Raine et al., 1998).

Murderers who are not guilty by reason of insanity and convicted murderers have been compared using neuropsychological testing. When differences are demonstrated, commonly it is poor verbal ability and a diagnosis of psychopathy in the convicted murderers that separates them from the murderers who killed when psychotic (Nestor, 1992; Nestor et al., 2002). Other neuropsychological functions have not typically demonstrated differences (Nestor, 1992; Nestor et al., 2002). For example, Nestor (1992) examined 40 violent forensic inpatients with the WAIS-R, the WRAT-R, and the TMT-B. The WAIS-R subtests Picture Arrangement and Similarities were used to assess executive function, along with the TMT-B. He found that the men who had been convicted of murder had more difficulties with language ability and general learning than they did on executive tasks, which all returned scores in the normal range. However, his sample included sexual murder as well, possibly accounting for the language and learning deficits if a temporal lobe dysfunction explanation is used to explain sexual violence.

A case study of an ‘out of character’ murder of his wife by a 65 year old man also demonstrated that he had verbal deficits only (Paradis, Horn, Lazar, & Schwartz, 1994). In particular, verbal new learning and memory scores were defective, whereas executive testing did not demonstrate any deficits compared to norms. This man had recently displayed aggressive and violent behaviour that was previously absent (Paradis, Horn, Lazar, & Schwartz, 1994). He was reported to demonstrate neuropsychological deficits in relation to left hemisphere dysfunction consistent with a large left-sided arachnoid cyst located in the middle cranial fossa (Paradis et al., 1994).
The relative normality of executive functioning in murderers in the literature is comparable to serotonergic dysfunction research that demonstrates no differences between murderers and non-murderers (Lindberg et al., 2000; Lindberg et al., 1985), at least in cases of stranger murder. However these findings are alongside research that demonstrates a higher proportion of executive deficits in ‘general’ samples of violent or aggressive individuals who have not murdered (for example; Blair & Cipolotti, 2000; Damasio et al., 1990; Gantner & Taylor, 1992; Giancola & Zeichner, 1994; Stanford et al., 2003).

An American study (Raine et al., 1998) compared murderers who committed premeditated murders with murderers who committed impulsive murders, and demonstrated some neuropsychological differences between the two. Raine and his colleagues suggested that premeditated or ‘predatory’ murderers had fewer difficulties with executive functioning than the impulsive murderers, or ‘affective murderers’ as they described them (Raine et al., 1998). This difference may explain why neuropsychological impairment on tests of prefrontal functioning are often not found when violent ‘psychopaths’ are assessed (Hare, 1984).

Positron emission tomography (PET) was used in the Raine et al. study to examine the hypothesis that the affective (impulsive) murderers would have lower activity in the prefrontal area and more in the sub-cortical region than the predatory (premeditated) murderers or controls. A form of the continuous performance task (CPT) was used as the authors believed that this task successfully demonstrated frontal activity in normal controls under the same imaging conditions. The Raine et al. study produced three main findings: (i) affective (impulsive) murderers demonstrated lower prefrontal activity and higher sub-cortical activity than the predatory (premeditated) murderers and the ‘normal’ controls; (ii) the predatory murderers had similar prefrontal activity as the controls, but higher sub-cortical activity than the controls; and (iii) the increased sub-cortical activity in both the murderer groups was restricted to the right hemisphere.
5.2 Method

5.2.1 Aims and Hypotheses

Study Two of this thesis is an attempt to replicate in part the findings from Raine et al. (1988) using more functional and ecological measures of neuropsychological functioning. Similarly, this study is an attempt to highlight any relationships between premeditated and impulsive offending and executive functioning. The same neuropsychological measures and methodology were used in this study as for Study One. Murderers were not included in Study One because of this possible difference related to executive or frontal-lobe dysfunction, and because of the extreme violence of the offences of the murderer group. This level of violence can be hypothesised as placing them in a different category from other violent offenders.

The hypothesis for Study Two is:

Men in the Impulsive Murderer offender group will demonstrate greater impairment of executive functions as measured neuropsychologically than men in the Premeditated Murder offender group.

5.2.2 Procedure

Although in the current study it was not possible to gain imaging data for any of the offenders, it was possible to access offenders whose crimes appeared to match descriptions of ‘impulsive’ or ‘premeditated’, and to test these inmates neuropsychologically.

Recruitment, consent procedures, and general method for the murderer study did not differ from Study One. The participants were recruited from male inmates at Auckland Prison (N = 40) in a ‘long-stay’ block with a current conviction of
murder. Further, from this pool of 40 inmates, potential participants were identified as ‘appropriate’ for the study (using criteria established in Study One) by prison officers, and were given the participant information sheet. The only additional criterion over and above those for Study One was that the last conviction had to be one of murder. Participants, after reading information about the study and agreeing to participate, gave written consent. Twelve inmates convicted of murder volunteered, with seven completing the assessments. The other five men who had consented to being involved in the research withdrew from the study before being assessed. Reasons for withdrawal are not known. Table 12 summarizes the selection and data collection procedure.

Table 12: Summary of Selection and Data Collection Procedure for Study Two

<table>
<thead>
<tr>
<th>Study Two</th>
</tr>
</thead>
<tbody>
<tr>
<td>Murderers</td>
</tr>
<tr>
<td>File review to determine inclusion/exclusion criteria. Participant information handouts given out</td>
</tr>
<tr>
<td>Registration of interest by inmate</td>
</tr>
<tr>
<td>Placement into Impulsive or Premeditated Murderer group categories</td>
</tr>
<tr>
<td>Testing</td>
</tr>
<tr>
<td>Categorisation according to test results</td>
</tr>
</tbody>
</table>
The men were tested with the same battery used in Study One. In Study Two, however, I was aware of the men’s conviction histories before I tested them, and this information was used to determine the definition of impulsive or premeditated murderer. This was required as there was no liaison group established for this study as in Study One due to constraints placed by Auckland Prison, so it was important to ascertain that inmates matched criteria before proceeding with testing. Further, under New Zealand law and types of convictions handed down, there are no clear codes for impulsive and premeditated murders. Instead insights into offence type are often found in the trial summary from the Judge.

All men were also assessed using the Likert Scale of Appropriateness during their interview and assessment.

5.2.3 Impulsive verses Premeditated

All participants had their most recent conviction as ‘Murder’ with no distinction made between impulsive and premeditated murder in the conviction code. The inmates’ file notes kept at Auckland Prison were used to determine whether the murder they had committed was impulsive or premeditated. None of the men who volunteered had been convicted of ‘Manslaughter,’ which is a less serious conviction under New Zealand law than ‘Murder,’ and may indicate that the crime is more likely to be impulsive or ‘accidental,’ rather than murder with intent. Further, as there was no clear statement in the inmates’ files regarding whether the murder was impulsive or premeditated, court notes detailing evidence and the Judge’s comments were relied upon in determining this. Although specific details of each of the murders cannot be given here in order to protect the confidentiality of the study participants, examples of each murderer category taken from actual details of the murder published in the public arena and with highly identifying details further disguised, are given below:
Premeditated Murder: evidence of planning, such as notes written, taped conversations, a confession stating an element of premeditation, a grave dug early for the dumping of a body, and Judge’s comments as to the fact of the murder being “executed in cold blood.”

Impulsive Murder: a violent physical fight while under the influence of substances, a ‘crime of passion’ followed by immediate regret and confession, and Judge’s comments as to the fact of the “impulsive and regretful nature” of the act.

Using this method four participants out of the seven were identified as having committed premeditated murder. However, in this group it may be that only one participant (Participant 6) could reliably be classed as a premeditated murderer under their own volition, as the other three (Participants 3, 4, and 5) that met the criteria were involved in the same ‘gang hit,’ and were reported to be “following orders.” In gang culture there are usually severe consequences when orders are not followed.
5.3 Results

Given the small sample size it is not possible to carry out statistical tests on the data, with only differences across subtasks of the BADS examined with statistics. Also, whilst it would be preferable to present the qualitative data in a case-by-case format, this was not possible, as this would break confidentiality. The results are thus presented in tabular form, followed by a summary of the ‘themes’ taken from the interviews and assessment of the men with quotes from the participants that highlight the findings.

5.3.1 Demographic Characteristics

From Table 13 it is clear that there are very few differences between premeditated murderers and impulsive murders. The main demographic difference of note is that the impulsive murderers are younger on average by 10 years (mean = 22.3 years of age) then the premeditated murderers (mean = 32.5 years of age).

<table>
<thead>
<tr>
<th>Identity</th>
<th>Age</th>
<th>Ethnicity</th>
<th>Premeditated/Impulsive</th>
<th>Spot The Word Scaled Score</th>
<th>Years of Education</th>
</tr>
</thead>
<tbody>
<tr>
<td>Participant 1</td>
<td>20</td>
<td>Māori</td>
<td>Impulsive</td>
<td>7.00</td>
<td>8.50</td>
</tr>
<tr>
<td>Participant 2</td>
<td>25</td>
<td>Pakeha</td>
<td>Impulsive</td>
<td>9.00</td>
<td>11.00</td>
</tr>
<tr>
<td>Participant 3</td>
<td>30</td>
<td>Māori</td>
<td>Premeditated</td>
<td>10.00</td>
<td>10.00</td>
</tr>
<tr>
<td>Participant 4</td>
<td>42</td>
<td>Māori</td>
<td>Premeditated</td>
<td>6.00</td>
<td>9.50</td>
</tr>
<tr>
<td>Participant 5</td>
<td>29</td>
<td>Māori</td>
<td>Premeditated</td>
<td>9.00</td>
<td>7.50</td>
</tr>
<tr>
<td>Participant 6</td>
<td>29</td>
<td>Pakeha</td>
<td>Premeditated</td>
<td>13.00</td>
<td>9.50</td>
</tr>
<tr>
<td>Participant 7</td>
<td>22</td>
<td>Māori/Pakeha</td>
<td>Impulsive</td>
<td>11.00</td>
<td>10.00</td>
</tr>
<tr>
<td>Mean</td>
<td>28.14</td>
<td>NA</td>
<td>NA</td>
<td>9.30</td>
<td>9.43</td>
</tr>
</tbody>
</table>
5.3.2 Neuropsychological results

As can be seen from Table 14, there appear to be no clear patterns of difference in neuropsychological outcome between the two murderer groups. Upon closer examination using Kruskall-Wallis, no significant differences were demonstrated in the means of any of the subtasks from the BADS.

<table>
<thead>
<tr>
<th>Identity</th>
<th>Premeditated/Impulsive</th>
<th>BDI-II(^a)</th>
<th>BADS(^b)</th>
<th>STW Scaled Score</th>
<th>COWA-M</th>
<th>Years of Education</th>
</tr>
</thead>
<tbody>
<tr>
<td>Participant 1</td>
<td>Impulsive</td>
<td>4 (Minimal)</td>
<td>16 (Low Average)</td>
<td>7.00</td>
<td>36.00</td>
<td>8.50</td>
</tr>
<tr>
<td>Participant 2</td>
<td>Impulsive</td>
<td>17 (Mild)</td>
<td>20 (Average)</td>
<td>9.00</td>
<td>46.00</td>
<td>11.00</td>
</tr>
<tr>
<td>Participant 3</td>
<td>Premeditated</td>
<td>7 (Minimal)</td>
<td>16 (Low Average)</td>
<td>10.00</td>
<td>41.00</td>
<td>10.00</td>
</tr>
<tr>
<td>Participant 4</td>
<td>Premeditated</td>
<td>13 (Minimal)</td>
<td>12 (Impaired)</td>
<td>6.00</td>
<td>56.00</td>
<td>9.50</td>
</tr>
<tr>
<td>Participant 5</td>
<td>Premeditated</td>
<td>0 (Severe)</td>
<td>19 (Average)</td>
<td>9.00</td>
<td>45.00</td>
<td>7.50</td>
</tr>
<tr>
<td>Participant 6</td>
<td>Premeditated</td>
<td>9 (Minimal)</td>
<td>18 (Average)</td>
<td>13.00</td>
<td>36.00</td>
<td>9.50</td>
</tr>
<tr>
<td>Participant 7</td>
<td>Impulsive</td>
<td>11 (Minimal)</td>
<td>17 (Average)</td>
<td>11.00</td>
<td>44.00</td>
<td>10.00</td>
</tr>
<tr>
<td>Mean</td>
<td>NA</td>
<td>9.14</td>
<td>16.85</td>
<td>9.30</td>
<td>43.42</td>
<td>9.43</td>
</tr>
</tbody>
</table>

\(^a\)BDI-II range classifications (along with raw scores) extracted from the raw data are presented, the definitions of which are given in the Methods for Study One

\(^b\)BADS are given as a profile score and executive functioning classification
5.3.3 Qualitative Results

Table 15 shows a striking pattern; every participant has a history of significant drug and alcohol use and a history of TBI, with at least one TBI occurring before his first offence. Another conspicuous result is that none of the premeditated murderers were intoxicated (according to their self-report and the trial notes) during their offence, whereas all of the impulsive murderers were.

Table 15: Presentation of Qualitative Results

<table>
<thead>
<tr>
<th>Identity</th>
<th>Premeditated / Impulsive</th>
<th>Drug History</th>
<th>Alcohol History</th>
<th>Intoxication During Offence</th>
<th>TBI History</th>
<th>TBI Before First Offence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Participant 1</td>
<td>Impulsive</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Participant 2</td>
<td>Impulsive</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Participant 3</td>
<td>Premeditated</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Participant 4</td>
<td>Premeditated</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Participant 5</td>
<td>Premeditated</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Participant 6</td>
<td>Premeditated</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Participant 7</td>
<td>Impulsive</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

All seven men in Study Two received a score on the Likert Scale of Appropriateness following assessment of 1 or 2 (1 = behaviour that is appropriate to the situation, 2 = behaviour that is mostly appropriate).

The following sections present, in the form of ‘themes,’ the qualitative data from the interview with the participants during their assessment. As for Study One, participants were asked questions about their alcohol and illegal drug use, and history of brain injury.
5.3.4 Intoxication During Offending

A clear difference between the impulsive murderer participants and the pre-mediated participants was that all of the impulsive group were intoxicated with alcohol and/or drugs at the time of their offending according to self-reports and records kept regarding the event. In contrast all of the premeditated group report they were not intoxicated or on drugs when they committed the offence of murder, or that they cannot recall if they were. Following are the descriptions from the participants reporting levels of intoxication, and non-identifying details about the murder.

**Participant 1 (impulsive):** When discussing the events leading up to the murder *Participant 1* reports how he had been on a solo drinking binge for at least three days that included a bottle of vodka a day. On the last day realising he had no food in the house, and wanting something to offset the emerging hangover, he set out in search of something to eat. In a drunken stupor he stumbled to his local shop and found it was closed, as was everything else in this small town. Motivated by his hunger and his plan to leave money on the counter for what he took, he broke in. After smashing a window to gain access the alarm started and panicked him, so he stepped up his pace, stuffed some sweets into his pockets, and ran.

It was while running away that circumstances leading up to the murder took place (and if reported here could identify the participant). *Participant 1* reports further, “I can’t really remember much (about the murder)...I had been drinking all day...when I got home (after the murder) I blacked out...the next morning I remembered some stuff...I turned myself in.”

**Participant 2 (impulsive):** Reports that he had not been sleeping for days leading up to the murder and had been drinking ‘some’ spirits (he was unclear in describing amounts). He denied taking any drugs or using medication in the lead up to the murder, but admits he often felt “spacey” as if he was “out of it” and he
felt he was always “losing track of time,” and thinks these occurrences may be related to times he drank alcohol. \textit{Participant 2} reports that he lost some time and then “woke up over the body,” with a spade in his hand and a body shaped hole. \textit{Participant 2} also remembers being out of breath and covered in dirt so surmised that he must have been digging. He goes on to say that he does not remember anything about the murder at all, or anything for some hours before he “woke up,” but takes responsibility, as “I was the only one who was there.”

\textit{Participant 3 (premeditated)} was clear that he was “straight” (not intoxicated or drugged) during his offence, which involved the planning of a murder as part of gang retaliation. He was the one who pulled the trigger, claiming that it was very difficult to do and he “almost lost (his) bottle,” but was ordered by another member who was with him to do it.

\textit{Participant 4 (premeditated)} was also part of the same gang murder as \textit{Participant 3} and was involved in the planning and driving the ‘getaway’ car. He also claims that he was straight during the offence as he had been drug free for at least a year building up to that murder, and also appeared ‘proud’ and without regret for his part in the murder. Participant 4 at the time of the murder was the president of his chapter of the gang and demanded respect and obedience from \textit{Participant 3} and \textit{Participant 5}.

\textit{Participant 5 (premeditated)} was the participant in the gang murder who shouted at \textit{Participant 3} to “pull the trigger!” According to his file notes and his reports he was the main organiser of this particular event and the one responsible for organising all the people required. Participant 5 claims he was drug free during the event as he was worried about the effects on his behaviour the pills he usually took were having (“uppers” he reported were making him violent).

In court documents for \textit{Participant 6 (premeditated)} it is detailed that the crime was cold and calculating and in the Judge’s summing up he describes it as “one of
the most passionless and deliberate killings New Zealand has ever seen.” Participant 6 was able to talk in detail about his planning and reasons for the crime and denies being intoxicated or drunk at the time, as he wanted “the operation” to run smoothly. On the day of the murder Participant 6 did not know who would be killed but knew that someone would get in his way (of robbing a warehouse), so had dug the grave one month earlier whilst also conducting surveillance of the targeted warehouse.

*Participant 7 (impulsive):* States that he “cannot remember the crime,” and goes on to state that this is not unusual for him when he has been drinking, as he was often “getting into trouble” after drinking and not being able to remember it. *Participant 7 recounts* that he was always being told he was doing things that he could not remember, which occurred during drinking or drug sessions. *Participant 7* states that the events surrounding the murder were much the same as usual for him, with lots of drinking going on all day and every day. He described drinking at least a bottle of spirits a day and many beers. He also admits to taking whatever drugs would come his way. *Participant 7* states all he remembers about the day of the murder is “getting out of it…fighting a bit with some guy at the pub…and what I’ve been told.”

### 5.3.5 Alcohol and Drug Use History

Another factor that clearly stood out is that all participants, both impulsive and premeditated, had histories of heavy alcohol and drug use, often starting in their early teens or younger. This theme is illustrated using the words of the participants.

*Participant 1 (impulsive):* When asked to state when he had first started drinking he replied “around 12 or 13 at family functions…just beer mainly.” He also reported that he began sniffing solvents at this age, including petrol every day for 2 years and reported that this made him “out of it,” and groggy. He also said “I
stunk as well, think it was the sweating out the toxins,” which caused him to give up solvents. Participant 1 also stated that he used to “do marijuana” but not any more as it makes him too paranoid.

Participant 2 (impulsive): States that he did not start drinking until 17 years of age and wouldn’t drink to get drunk “but I could drink a lot,” he said (a lot was defined as more than eight glasses of spirits and a dozen cans of beer). He also reported that he had tried “different drugs” but nothing long-term. He recalls his drinking happening “most weekends.”

Participant 3 (premeditated): Started drinking at 14 with “just beer at social occasions,” at first. He reports that his drinking steadily grew until he was drinking a lot in one session (for example, 24 cans of beer), but sessions were infrequent. At 17 years of age Participant 3 started smoking marijuana every day, and would supplement this with “the occasional pill or sniffing.” Participant 3 reported proudly that he gave up his daily marijuana habit eight months prior to this assessment.

Participant 4 (premeditated): Recalled that he first started drinking at parties when he was about 12-13 years of age, and went on to state that he would binge drink on purpose from about 14 years of age to force a “black out,” because life was easier when he wasn’t consciously participating. He started smoking marijuana at 14-15 years of age and was a regular smoker for a while but eventually it was a “once in a blue moon thing.” He also stated from the ages of 15 – 27 years he would regularly “do pills and LSD,” but has been drug-free since 1997.

Participant 5 (premeditated): At 12-13 years of age he began to experiment with alcohol and would steal it whenever possible. At 15 years of age this developed into a pattern of drinking beer and spirits twice a week “hard out to get drunk.” He would also have “just a puff or two” of marijuana “every now and then.” Between
the ages of 21-25 he also took a variety of pills (whatever was available) but came off them “as they made me violent.”

*Participant 6 (premeditated):* Remembers from eight years of age “drinking the leftovers” from his parents’ parties. From about 13 years of age his drinking became more regular and every weekend he would “drink to get drunk.” His drink of choice was whisky when he had money, otherwise it would be “purple death” (methylated spirits). This pattern continued until 18 years of age “when I started spewing blood.” At this point he reduced his drinking to “a few beers every couple of weeks.” From his early teens *Participant 6* has also smoked marijuana every day, including the 48 hours prior to testing. At this time he also experimented with “uppers and downers, opium and morphine,” and would “take a trip (LSD) once a year.”

*Participant 7 (impulsive):* Started drinking at the age of eight “whatever I could get my hands on.” At 12-13 the drinking became more frequent and he also started smoking marijuana and at 15 years of age would get drunk every day. At 17 he stopped drinking for six months “as it was getting me into trouble” and instead he said, tried various pills and opium “for a break.”

Although previous to their convictions substance use had been heavy for all of the participants, six of the seven participants were not ‘using’ in prison according to their self-report to me. Further, there was no report in their files that they had been caught through random urine testing for drugs. It may be that because all of these men were in the maximum-security wing of Auckland Prison it was harder for them to access contraband, as some inmates in other wings involved in the Study One reported relatively easy access to drugs (usually marijuana brought in by visitors or from patches in the fields they worked).

In maximum security there is a policy of regular spot urine testing and a positive finding on this can reduce privileges and increase time before probation. Several
participants commented on this fact. Some inmates went further to say that the reduced access and the spot-checking was positive as it helped them to give up drugs.

5.3.6 Traumatic Brain Injury History

All participants reported brain injuries prior to their first offence, with many participants reporting multiple brain injuries that began in childhood (often resulting from abuse by carers).

Participant 1 (impulsive): At 17 years of age Participant 1 (not long before the murder) reports that he was hit on the head by a crow bar in a fight with other youths and briefly lost consciousness. He did not seek medical attention and had the gash on his head stitched up by friends. He states he cannot remember much about this time as “I was pretty drunk that night.” Participant 1 denies any head injuries when younger even though he played rugby all through school, was regularly in fights, and reports a childhood peppered by “hidings” (beatings) from older family members.

Participant 2 (impulsive): Reports that at 15 years of age he fell 20 feet out of a tree. He states that he was “knocked out” briefly and taken to the hospital where he spent two weeks for a shoulder injury. Participant 2 does not think he was concussed and states he has no memory loss of the time. Just before his crime of murder he sustained a blow to the head that he reports left him “really vague,” and that he has no memory of anything around that time for at least four hours. He also states that when he “came to,” he could not remember his name and did not know where he was.

Participant 3 (premeditated): Sustained a hammer blow to the head when he was 12 years old and states he can’t remember anything about what care (if any) he received afterwards, but knows he wasn’t taken to the hospital.
At 17 years of age Participant 3 fell from a height and a long nail was embedded in his head. He reports no medical intervention and is unable to describe the effects of the injury. He also stated that he has had at least seven major fights where he has sustained blows to the head that usually left him feeling unwell, but he said, he was usually drunk at the time as well, so it was hard for him to decide the next day if the feelings were due to a head injury or a hangover.

Participant 4 (premeditated): At 12 years of age Participant 4 was “knocked out” after receiving “a hiding from my father.” He was taken to the hospital but does not remember anything about the visit. At 13 years of age Participant 4 fell from a moving car and hit his head on the road. He spent a night in hospital as a consequence of this. Participant 4 was intoxicated at the time of this accident and says this makes it hard for him to recall the details of the accident or it’s effects. He also states he recalls many “knocks to the head” playing rugby league and getting into fights, all through school and young adulthood.

Participant 5 (premeditated): States that he cannot really remember any obvious head injuries as a child, but there were times as a school boy playing rugby he would feel drowsy after being tackled. At 18 years of age he sustained a blow to the head during a fight that left him feeling “really sick and hung-over,” but it was hard for him to say if this was due to the blow or his drinking. At 24 years of age he was in a car accident where he smashed into a power-pole that left him with a deep gash under his right eye.

Participant 6 (premeditated): Reports many incidences of head injuries. The first major one he can remember is when he was eight years of age and he fell and hit his head on the doorframe. The front top right of his head was split open and taped-up with Sellotape by his mother. He received no other treatment. As a result of the fall he remembers, “blacking out briefly and feeling woozy” and he continued to feel “woozy” the next day as well. One year later during a play-fight with some friends he was whacked on the head with a broom handle that had a
nail sticking out of it. The nail embedded in the scar of the previous injury. *Participant 6* states he “blacked out on (his) feet…and walked home dizzy and out of it…seeing spots.” At 11 or 12 years of age he reports that he hit himself in the face with the back of an axe handle, which split the skin by his left eye. The gash was stitched up in hospital and he remembers feeling sick. At 15 years of age *Participant 6* reports that he “was set upon by 3 guys” and beaten about the head and stabbed shallowly in the chest. He did not go to hospital afterwards but recalls suffering headaches “for days afterwards.” He also reports a motorcycle accident in his early twenties where he ploughed into a ditch landing on his face and bending his neck and back. He did not seek hospital treatment after this but admits being bed ridden for a while and very stiff and sore and suffering from fatigue.

*Participant 7 (impulsive):* Reports many head injuries, with the first one he can remember occurring when he was seven years old. At this time he had to visit the hospital (after falling into the corner of the coffee table) to have “a big gaping hole in my head” stitched up. At 13 years of age while doing “stunt manoeuvres” on his BMX bike, he slammed face first into the handlebars after rising about six feet in the air. He described the immediate impact as feeling “like a sledgehammer hit me right between the eyes.” Immediately afterwards he was unable to walk or see and described feeling “really out of it...(and) had a wicked headache for ages.” *Participant 7* has also been “knocked out” at least three times while playing rugby, and recalls one time when he “woke up talking to my mates on the sidelines” with no recollection how he got there as his last memory was of himself playing on the field (similar stories are told by *Participant 7* regarding his experiences when drinking and losing large periods of time).

During his teenage years *Participant 7* was also involved in many fights, which included his head being targeted by punches, kicks, and weapons (such as planks of wood). Most of these fights would involve him blacking out and often coming to without any recollection of how he got there. At 15 years of age during one of these fights he was “knocked out with a cinderblock (and) came to with a dog
biting me.” Also at 15 years of age he was “thrown out of home” because he had come home with a Mohawk haircut. He described how his dad dragged him out to the front of the house and strangled him for a while then threw his bike at him, at which point Participant 7 passed out on the driveway. He regained consciousness sometime later when his older sister woke him and took him to her house.

5.4 Discussion

Study Two did not support the hypothesis that impulsive murderers would demonstrate impairment on measures of executive function compared to premeditated murderers.

It may be that there are differences between individuals who murder impulsively and those who are described as premeditated in a New Zealand population. However in this study no definitive conclusions can be drawn due to methodological concerns, primarily the very small size of the groups, and the difficulty in clearly demonstrating whether a participant committed an impulsive murder or a premeditated murder. Particularly problematic is that three participants included in the premeditated offender group were involved in a gang-related shooting, and the impetus for this crime, even though it was planned, may not reflect true and measurable individual differences in these participants who were “following orders.” Whilst the premeditated group may not be considered valid, the impulsive group does appear to be so. The interesting result of an absence of deficits of executive functioning in the impulsive murder group is in stark contrast to the high incidence of ED in the violent offenders from Study One. What is clear is that each participant in the study had similar stories that involved a historical heavy use of alcohol and other substances, and frequent brain injuries incurred through abuse, neglect, and poor decision-making.
The only obvious difference between the groups in Study Two was that all of the impulsive murderers reported being intoxicated and/or drugged at the time of the murder, and all of the premeditated murderers reported being ‘straight.’ The fact that three of the premeditated murderers were part of the same gang, who all separately denied intoxication, gives some credence to their reports; it seems likely that they would have all engaged in similar behaviour prior to the planned murder. This is comparable to Participant 6 as well who also planned a murder. He was very clear that he was not drinking during the murder, as he wanted to succeed at the robbery and murder and “get away with it.” With these facts and assumptions there is little reason to suspect they had alcohol or drug-induced transient executive dysfunction.

In understanding how test behaviour matches test scores, all but one participant (Participant 4) gained Likert Scale scores that were congruent with their Low Average to Average scores on the BADS. When examining the themes drawn from the interviews, it must be noted there can be many variables impacting on the account an offender gives of his crimes. Care was taken to try not to influence reporting; however other factors (for example, my gender, my ethnicity) and my presence may have contributed to the way some inmates reported, or chose not to disclose, events.

Given the histories that have been recounted it seems remarkable that the neuropsychological scores are not indicative of more deficits. What is particularly notable about the stories related by these men are the abuses inflicted upon them by others entrusted with their care and later continued through self-abuse, e.g., drinking alcohol excessively, drug taking, and physical fighting. The background of drug and alcohol abuse is not dissimilar from the sample in Study One; however the rates and severity of some of the TBIs reported and the abuse histories appear worse for the men in Study Two. The ages of the men in the sample of Study Two are similar to that of the men in the violent offender group in Study One. With these similarities in mind and the fact that some of the TBI
histories reported are more severe then in Study One, again it seems remarkable that more deficits were not noted across this sample, in line with findings from Study One.

If specific language abilities, including verbal memory were tested, perhaps differences demonstrated in previous studies may have been replicated (for example, Nestor, 1992; Nestor et al., 2002; Paradis et al., 1994). A future study could, for example, include the Californian Verbal Learning Test (CVLT) to inform the debate, along with general verbal ability tests. However, the COWA-M and the STW, both containing a verbal component, did not differentiating the groups or demonstrating dysfunction. Other tests that may have been useful in this study would be measures of impulsivity, especially since the study was attempting to differentiate between impulsive and premeditated murders.

What a client does in the testing environment does not always accurately reflect what they do in their natural life (Chaytor & Schmitter-Edgecombe, 2003). The ‘optimal performance’ test environment, free from major distractions with prompts provided and one-on-one attention and instruction, may mask any real-world deficits the participant struggles with. Striving for a participant’s best performance may actually reduce the ecological validity of tests that purport to measure this (Chaytor & Schmitter-Edgecombe, 2003). It may be that the distractions that I felt were present during the testing session, such as uncomfortable seating, occasional loudhailer announcements, and the walk-by of prison officers, were not actually distractions to the offenders participating. Participants may have actually benefited from the attempts at minimising distractions when compared to what they experience in their daily prison life. Hence, best performance was promoted, rather than gaining an understanding of the participants’ executive functioning under stress, which would typify that individual’s real world performance.
In Study Two all impulsive murderers were intoxicated while committing murder. This starkly contrasts with the finding that the premeditated murderers reported no intoxication while offending (which was noted in their court files). Although there was no difference between the two murderer groups on the BADS scores, and only one of the murderers scored in the Impaired range, the intoxication of the impulsive murderers may suggest that they were suffering from short-lived severe ED at the time they murdered. This is a hypothesis that would fit with work that simulates ED (using alcohol) in normals and measures resulting aggression (for example, Giancola, 1995; Giancola & Zeichner, 1994, 1997; Hoaken, Assaad et al., 1998; Lau et al., 1995), and alcohol intoxication proceeding violent offending (Abram, 1989; Leonard et al., 2003).

It is hypothesised that impulsive murderers would have had existing sub-clinical executive compromise (that testing used in this study did not establish) and have previously learnt to use a repertoire of aggressive behaviours. Further, they would have been highly vulnerable to extreme disinhibition and aggression when under stress, and this would have been particularly evident after consuming alcohol and/or drugs. The intoxication levels in the impulsive murderers would then lead to the temporary state of severe ED, and poor decision-making and risk-taking behaviour would increase, increasing the chances of severe assault, and in these cases, extreme assault leading to murder. This analysis fits with Leonard, Collins and Quigley’s (2003) finding with 190 healthy men who had been involved in “bar-room brawls.” These researchers demonstrated that the men who had consumed the largest amount of alcohol, and were very intoxicated, had perceived themselves to be under more threat, were involved in more severe fighting, and perpetrated more incidences of severe and dangerous physical assaults.

Nevertheless, the vast majority of people who are intoxicated and have a history of TBI do not commit murder. In explaining the result for the impulsive murders, at best, this substance-induced transient ED can only have resulted in an exacerbation of the offenders’ behaviour, but cannot fully account for it. This
suggestion still does not explain why no current difficulties with ED were measured by the BADS.

Further explorations of the issues raised by this study along with the limitations of the study are discussed alongside Study One in the General Discussion chapter.
6

Study Three: Development of an Intervention for Inmates with Executive Dysfunction

Punishment is the last and the least effective instrument in the hands of the legislator for the prevention of crime.

John Ruskin quotes (English writer and critic of art, architecture, and society, 1819-1900)

6.1 Introduction

The results of Study One indicate that executive dysfunction is common amongst violent inmates at Auckland Prison. Most prison officers, social workers, psychiatrists, nurses, and psychologists who work in a prison setting and who are involved in rehabilitation programmes for inmates have minimal, if any, knowledge of brain-behaviour relationships generally, and of executive dysfunction specifically. Thus, they are unlikely to structure rehabilitation programmes to maximise their effectiveness for inmates with ED.

Inmates with ED are likely to find it difficult to gain tangible and useable benefits from traditional interventions and rehabilitation techniques. It may be that they lack the ability to attend to new information for any length of time without distraction, or they may find it difficult to retrieve newly stored information and to
act on it appropriately and in a timely fashion. Some men may also have problems
inhibiting inappropriate responding, including acting or speaking without
thinking, which at times can lead to aggressive outbursts or provoke aggression in
others. Many of the men in Study One spontaneously reported they were
dissatisfied with their current rehabilitation offered by Auckland Prison, stating
that they felt they were being “lectured at,” or felt that what was being discussed
“goes over their heads,” or doesn’t concern them. Additionally, inmates may lack
the motivation necessary to participate fully in rehabilitation programmes whether
or not they have executive deficits. Motivation and a willingness to change
behaviour are key to a therapeutic process; if inmates are ‘forced’ to attend a
rehabilitation program, progress may be impeded, and any positive impact may be
limited.

Research is required that addresses the effectiveness of interventions for offenders
through the development and evaluation of rehabilitation programmes that are
informed by knowledge of how effective learning can occur in a group of inmates
whose abilities are constrained by ED. In this section an intervention for inmates
with ED is presented, as well as the methodology to test whether such a
rehabilitation programme would be effective.

The aim of this pilot intervention is to discover whether techniques that enhance
outcomes for people with ED, such as those used in traumatic brain injury clinics,
are transferable to prison inmates. These techniques include psycho-education and
repeated practice of skills that the individual has trouble with. The intervention is
designed around ‘building blocks,’ where skills are learnt and repeated until
consolidated, with each block building sequentially on the previous one. This is
hypothesised to be useful in circumventing prospective memory difficulties and to
utilise implicit memory. This approach will also assist the skills to become over-
learned and automatic. The building block format encourages errorless learning,
as learning in an errorless environment has been demonstrated to lead to better

This intervention addresses the executive behaviours that were most problematic for the violent inmates in Study One. These behaviours as measured by subtasks of the BADS are (i) the ability to plan and carry out a successful approach to a task that has many elements to remember and juggle, and the ability to divide attention, as measured by the Modified Six Elements; (ii) cognitive flexibility and the ability to inhibit responding and to concentrate in the face of distracting prior learnt information, as measured by the Rule Shift Cards; and (iii) the ability to plan and follow through a course of action and benefit from strategies as demonstrated by the Zoo Map.

Development of the intervention has drawn from literature reporting some success with similar clients in the TBI field in improving inappropriate behaviour and daily functioning difficulties (Evans, Emslie, & Wilson, 1998; Feindler & Ecton, 1987; Gajwski, Hirn, & Mayo, 1998; Goldstein & Glick, 1993; Levine et al., 2000; Mateer, 1999; O'Leary, 2000; Speilberger, 1999; Wilson, 2000). Additionally, this intervention has been informed by evaluation of the effectiveness of strategies applied within the forensic field (Antonowicz, 1994; McGuire, 1995; Morgan, Winterowd, & Fuqua, 1999; Reid & Gacono, 2000; Ross, Fabiano, & Ewles, 1988).

However, even if a successful rehabilitation programme targeting the types of executive dysfunction that lead to violence could be developed for prisoners, if minimal follow-up is in place for the offenders once released from prison, maintenance of new behaviours is likely to be short-lived. This is especially the case for those offenders likely to be released back into the same environment from whence they came.
6.2 Method

The pilot intervention is a 16-session intervention package (Appendix 3) that runs over eight weeks, comprising two 1-hour sessions per week. The intervention has been designed in order to meet the needs of men who have ED. It follows a highly structured format and includes a large amount of repetition in order to circumvent learning difficulties due to ED, such as poor prospective memory and diminished ability to implement newly learnt strategies. It is also needs-based; each session builds sequentially on the content of the previous session.

6.2.1 Aims and Hypotheses

The aim of Study Three is to develop an intervention programme for inmates with ED which will assist them to control behaviours that can lead to violence. Assessment methods that examine the impact of the intervention are also included.

The hypothesis to be tested is:

Only men in the group receiving the study intervention programme will demonstrate significant changes in their understanding of ED, an increase in self-monitoring skills, and a reduction in impulsivity.
6.2.2 Recruitment and Selection Criteria

Two groups of six violent inmates will be selected by a staff member at the prison according to the following inclusion criteria:

(i) all males 17-50 years of age
(ii) convicted of at least 2 violent offences
(iii) have no history or current experience of psychosis, epilepsy, or a major depressive episode
(iv) no sexual offenders
(v) are assessed as having ED as indicated by the BADS

6.2.3 Measures

The BADS and STW, COWA-M and TFR previously described in Study One are administered at the beginning and end of the study. Impulsivity and self-monitoring will be measured using the Barratt Impulsivity Scale II (BIS-II; Barrett and Slaughter, 1998; used with permission from Ernest Barratt) and the Victoria version of the Stroop (Regard, 1981). A Structured Interview assessing impulsive choices developed for this intervention will be administered. Hassle logs will be completed by the inmates, and Behaviour logs by prison officers. The Study intervention facilitator will discuss the logs with the participants each session. Brief descriptions of those measures not described in previous chapters follows.

BIS-II

The BIS-II is a 30-item self-rating checklist. It represents a personality gauge that attempts to measure a construct of impulsivity that is independent from anxiety. The BIS-II has three subscales: attentional impulsiveness, motor impulsiveness, and non-planning impulsiveness.
**Stroop**

The Stroop (Victoria Version; Regard, 1981) in the first component requires the participant to name the ink colour of rows of coloured circles and read colour words. In the following more complex section, the participant is to name the ink of words that are colour-incongruent. The interference generated by this enables a measure of cognitive flexibility and control, and the ability to inhibit prepotent responding.

**Structured Interview**

A Structured Impulsivity Interview was developed using information from conversations with participants from Study One and Two. This was designed to measure impulsivity and ‘thinking things through’ in an incarcerated sample with ED. A copy of this interview is appended to the thesis in Appendix Three.

**Hassle Logs**

A ‘hassle’ log has been developed for the participants in the intervention for ED. This log is similar to a structured diary page. They will be encouraged to fill in this log when they have been involved in a ‘hassle.’ The log is to assist reflection where they may have been impulsive or made the wrong choices. Reflection will be done in a structured way during intervention sessions, and discussion will be encouraged regarding the ‘good’ and ‘bad’ decisions made during the incident. Similarly, the log serves to promote discussion in how they can use new skills learnt during the intervention in the future. The hassle log has been appended.

**Behaviour Logs**

The behaviour logs are for the prison officers that have been paired with the six men in the intervention. In designing the behaviour logs, prison officers were
asked about activities in prison around which they have noticed the most friction take place. This information along with a request to create specific logs, in order to make it quicker for officers to note behaviours, resulted in the development of three logs. These logs are appended to the thesis and comprise: an Exercise Incident Log, a Meals Incident Log and a General Incident Log.

6.2.4 Procedure

The design to assess the outcome of the pilot intervention is based on an ABA format. Table 16 summarises the selection and data collection procedures for Study Three.
Table 16: Summaries of Selection and Data Collection Procedure for Study Three

<table>
<thead>
<tr>
<th>Study Three</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>STAGE ONE</strong></td>
</tr>
<tr>
<td>Violent offenders identified by prison officers are assessed on the BADS, STW, COWA-M, and TFR. Those who demonstrate ED are enrolled in the study and assessed on the BIS-II, STROOP, and Structured Interview</td>
</tr>
<tr>
<td><strong>STAGE TWO</strong></td>
</tr>
<tr>
<td>8 weeks of no contact after which, administration of BIS-II, STROOP, Structured Interview</td>
</tr>
<tr>
<td><strong>STAGE THREE</strong></td>
</tr>
<tr>
<td>6 men randomly assigned to the intervention group and 6 men randomly assigned to control group</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>STUDY INTERVENTION GROUP</th>
<th>CONTROL GROUP</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 men are randomly assigned to 8-weeks of the study intervention for ED. Hassle logs are filled out by the inmates and behaviour logs by paired prison officers</td>
<td>6 men are randomly assigned to 8-weeks of usual rehabilitation (e.g., drug and alcohol program) Behaviour logs are filled out by paired prison officers</td>
</tr>
</tbody>
</table>

| **STAGE 4** |
| Testing with BIS-II, STROOP, Structured Interview |
| **STAGE 5** |
| 8-week waiting period |
| **STAGE SIX** |
| Testing with BADS, STW, COWA-M, and TFR. and with BIS-II, STROOP, Structured Interview |

Twelve inmates will be required for Study Three, with six men assigned to each group. Both groups will remain in the study for a 24-week period, which is separated into 8-week time periods.
STAGE ONE: Participation in the study requires completion of the two-hour assessment that includes the BADS and all other tests administered to those in Study One and Study Two. The BADS, STW, COWA-M, and TFR are administered together to provide a clearer picture of the inmate’s overall functioning prior to the intervention, and to assure that poor performance on the BADS is likely to be because of ED. A diagnosis of ED of Low Average, Borderline, or Impaired on the BADS must be reached to meet criteria to remain. Once ED criteria has been met, all participants will then be assessed with the impulsivity measures: BIS-II, Stroop, and the Structured Interview. This battery will take approximately 30-minutes to administer.

STAGE TWO: During the first 8 weeks after the initial ED assessment (and after the first assessment of impulsivity is completed) the participants will complete a ‘baseline’ condition, during which they continue with normal prison routine. There will be no contact from the researcher (who also acts as the study intervention facilitator) at this time and no participation in any interventions.

STAGE THREE: One group of six men will be assigned to attend the Intervention for Inmates with ED and the other six to a control group. The control group will attend an established rehabilitation programme in the prison (such as Anger Management or Drug and Alcohol; run by a different clinical psychologist at the Prison.).

The intervention program will target ED behaviours and run for two 1-hour sessions a week. This design is an attempt to increase the likelihood of learning and retaining new information. All inmates in the intervention are paired with a prison officer ‘buddy’ who observes the inmate’s behaviour and fills in logs regarding this. This will provide valuable information regarding the utilisation of new skills.
STAGE FOUR: When the ED intervention and the control intervention have been completed, post-testing with the Stroop, BIS-II, and Structured Interview takes place.

STAGE FIVE: Following the intervention and the post-testing, participants return to normal prison routine for an 8-week period. Behaviour logs continue to be filled out by paired prison officers during this stage. Inmates in the study intervention group may choose to continue to fill out their Hassle logs, as taught in the intervention group.

STAGE SIX: There is no contact with the researcher until the end of an 8 week period, when a re-administration of the full testing battery used in Study One and Two is conducted, along with the Stroop, BIS-II, and Structured Interview for the final time.

6.2.5 Analyses

The means of the psychometric tests administered will be compared within and across groups. Due to the small sample size, non-parametric methods will be employed, similar to the methods used in Study One. Similarly, differences in the BIS-II across groups, and changes in the BIS-II across time will be compared.

The hassle logs and the behaviour logs are intended primarily as materials for the intervention, and not for analyses. However, it is possible that themes may emerge from what is reported. It may be possible to track the way in which participants are applying their newly learnt skills. Likewise, the Structured Interview will be examined for themes and for the implementation of new information that was learnt in the intervention by those participants who attended. The participants that did not attend the intervention will provide interesting comparison, not only regarding what may or may not have been learnt in the pilot intervention for
inmates with ED, and the impact of any intervention and time spent with a facilitator, but also possible practice effects in repeating the assessments.

6.3 Results and Discussion

It is not possible to report any results for this study, as the rehabilitation programme did not go ahead as planned. This intervention study for inmates with ED as described was planned for inclusion in this thesis, but permission (previously given) to proceed was withdrawn one day before the study was to commence by the Department of Corrections and Auckland Prison. Their reason for this was their concern that it would interfere with staff adjustment to the newly implemented Integrated Offender Management System. Subsequent enquiries regarding the possibility of testing this rehabilitation intervention have not met with a positive response. This is disappointing as it is considered important to investigate rehabilitation strategies that are informed by research, and that are designed to address the specific needs of violent inmates with ED.

Rehabilitation of executive functioning is acknowledged as one of the most difficult areas to habilitate or compensate for, and this may be even more so in a prison environment. Measuring outcomes within the highly structured prison environment may constrain transferability of this intervention to unstructured real-world settings. Generalising the treatment from a highly structured environment to an un-structured real-world environment will be one of the major difficulties to overcome. The results of Study One indicate that specialist rehabilitation is required for different groups. In particular, inmates with ED require rehabilitation programmes that take into account their impulsiveness, poor ability to learn from errors, poor ability to think abstractly, and difficulties imposing internal structure and organisation on their thoughts. Difficulties with working memory amongst inmates with ED pose another challenge for rehabilitation.
Further understanding of the underpinnings of recidivism and the contribution of ED is crucial to progress in reducing future offending. This preventative stance is in line with current literature (Brown, 2002; Valliant et al., 2003). However, current society appears more concerned with punishment than rehabilitation. (Editorial, 2001). Retribution certainly appears to be the primary public concern in New Zealand as the media is saturated by images of violent crime, even despite statistical evidence that violent crime is less frequent now than in the previous decade (Rich, 2000). Entire lobby groups, such as the Sensible Sentencing Trust, have been set up to lobby for longer sentences, for harsher punishment, and for more retribution. Within this retribution-focused socio-political climate, development of rehabilitation programmes lack public support and funding. It appears that most attempts at rehabilitation in the current climate are viewed by many as an unnecessary moderating of the (required) retribution elements of the prison sentence.

Nevertheless, effective rehabilitation must be developed and offered. Even as sentences for convicted violent offenders are lengthened, the majority will still re-enter society within 10 years and will require appropriate social tools to live successfully within society. Rehabilitation is vital, as punishment itself does not provide rehabilitation or an education. Conviction and sentencing to prison provides the restriction of freedom (punishment element) and safety for society. But any safety is temporary if the violent offenders who are released have not been targeted with individually-geared rehabilitation programmes, as violent offenders are likely to re-offend (Rich, 2000). The question of whether rehabilitation for inmates with ED can be successful, especially in reducing violence and offending, is a vital and costly question for the future. Costly indeed if it is not even attempted.
General Discussion

Experience teaches only the teachable.
Aldous Huxley (1894 – 1963)

One aim of this thesis was to critically review the literature relating to ED and violence. This was carried out in Chapters Two and Three, and the literature reviewed will be referred to in this discussion where relevant to the results of Studies One and Two.

An additional aim was to see whether ED was common amongst prison inmates at Auckland Prison (or recently released from Auckland Prison). This was found to be the case amongst recidivist violent offenders, with the exception of the murderers studied. This has implications for the way rehabilitation programmes are structured, and potential to inform the development of new programmes that may be useful for inmates with ED. The final aim of the thesis was addressed in Study Three (Chapter Six), with the design of an intervention for inmates with ED presented.

In this General Discussion chapter, brief summaries of the previous results and discussions for studies One, Two and Three are presented together, followed by a discussion of the TBI and substance use variables that were found in both Studies.
One and Two. Following this, the implications for this work will be discussed including suggestions for how this information can be applied, followed by a review of the limitations of the studies. Next, ways in which future studies can improve on the design of these studies will be presented along with questions raised by the present work that can be expanded on in future studies. Lastly the final conclusions are presented.

7.1 Brief Summary of the Studies

Study One was designed to address some of the major criticisms made of previous neurobehavioural studies of violence. Violence was clearly operationalised, a comprehensive neuropsychological battery was used to assess executive function along side non-executive tests, and a control group of matched community subjects was included alongside clearly delineated violent and non-violent offenders. These methodological improvements strengthen the findings of Study One. The results of Study One demonstrated the sensitivity of the BADS in assessing ED in an offender population. The BADS significantly differentiated violent offenders from non-violent offenders and non-violent community controls. The high rate of ED measured in the violent offenders may underlie a pathway to violence, possibly through increasing an existing propensity for violence in these individuals.

In Study Two, no significant links between murder category and performance on the BADS, or any other neuropsychological test, were evident. The most likely explanation for this may be methodological limitations; namely the small sample size and the difficulty in categorising murderers into impulsive and premeditated categories. Additionally, the men in the impulsive offender group were relatively young, possibly representing a different sample, distinct from an older sample with a long offending history (Rich, 2000). Raine et al. (1998) demonstrated prefrontal differences between premeditated and impulsive murderers using measures of glucose uptake in the brain. The significant differences found by
these authors may have been facilitated by their use of a larger sample and clearly delineating the premeditated and impulsive murderers. Also, examining glucose uptake may show differences that do not necessarily relate to neuropsychological test performance, or at least to the tests used in the current study. Despite the methodological differences acknowledged, it is surprising that the men in the impulsive group in Study Two were so different from the violent offenders in performance on the BADS from Study One, as their backgrounds were similar.

Study Three was originally intended for implementation and evaluation; however it was not able to be carried out due to restraints placed by Auckland Prison. Nonetheless, the design is one that should be investigated in future studies. The impetus for such a study and the need for informed rehabilitation interventions remain.

7.2 Traumatic Brain Injury and Substance Abuse Variables Common

Almost all offenders reported a history of brain injury. When reporting numbers and types of brain injury, participants gave descriptions ranging from a “knock on the head” leaving them dizzy and nauseous (suggestive of concussion or mild TBI), to severe TBI resulting in a coma and a lengthy stay in hospital. The entire murderer sample in Study Two reported a history of TBI, with all participants reporting injuries occurring before their first conviction. Similarly, all 21 participants (100%) in the violent offender group of Study One reported a history of TBI with 85.7% of men in this group stating that their first TBI preceded their first conviction. The non-violent offender participants also reported a high incidence of TBI (93.8%), with 81.3% of this group describing one or more TBI’s before their first conviction. These figures are comparable with the Barnfield and Leathem study (1998) demonstrating high rates of TBI amongst inmates, as well as the figures provided by Slaughter et al. (2003). Sustaining a TBI before a first conviction is also comparable to the research conducted by Sarapata et al. (1998)
which found that 50% of their non-violent convicted felons sample reported a history of TBI preceding their first conviction.

A possible explanation for the high rates of TBI generally reported across the entire sample in both studies could be that the majority of the participants reported that they had played rugby and/or rugby league, which can increase the likelihood of sustaining a concussion, and experiencing cumulative brain injuries (Faillace, 2002; Gronwall, 1989, 1991; Gronwall & Wrightson, 1975). With violent offenders from Study One the reporting of TBI increases to the 100% level, possibly reflecting the high-risk situations these participants report being involved in. Another explanation for the high rate of TBI across this sample could also be that the self-selected sample may include a high number of risk-takers, given that they volunteered to take part in the study (Suris et al., 2004). This is a common difficulty identified by aggression researchers using non-randomised samples where informed consent is gathered prior to studies. Another difficulty faced by aggression researchers is social desirability (Suris et al., 2004), and it may be that participants in this sample, particularly in the inmate sample, exaggerated the number of aggressive incidents and subsequent TBIs they had sustained to fit with a self-concept of masculinity.

Other explanations for the frequency of TBI reported across the sample include the consequences of offending as a high-risk activity, as risk taking of this sort will increase opportunities for sustaining a TBI. Even before offending as a young adult or adolescent occurs, those at risk of this as children often meet the criteria for Conduct Disorder and/or ADHD (Carr, 1999) and are more likely to be actively risk taking. Children with these difficulties are also more likely to sustain physical abuse from their caregivers (Patterson, 1982), again increasing the likelihood of sustaining a TBI.

The fact that the prevalence rate in the community control group is high when compared to general epidemiological studies suggests that the control group was
well matched to the inmates on lifestyle factors, including those that have put them at risk for sustaining TBIs. However, that acquired brain injuries did not result in executive dysfunction for this group at the same level as the inmates may be due to other individual factors that underlie both ED and offending. Additionally, it is acknowledged that the location and severity of injury, together with the interaction of the developmental stage of the individual, and the environment, is likely to influence the outcome of any ED. A history of TBI may contribute to offending in already vulnerable individuals, but alone cannot sufficiently explain it.

In this study it is difficult to accurately assess self-reports of TBI for over or under-reporting, without medical records. Despite this caution, along with the limitations of retrospective recall and the social desirability for some inmates to appear as ‘tough,’ the TBI rates are consistent with those demonstrated in previous studies (Barnfield & Leathem, 1998; Sarapata et al., 1998; Slaughter et al., 2003).

Data examined across Participant Type demonstrated that alcohol use is prevalent across all groups in both studies. This may also partly explain the high rate of TBI reported by all participants, as the use of alcohol may engender vulnerability towards acquiring a brain injury through increasing risk of falls and risky decision-making. It is notable that although the high use of alcohol across all the groups could not be used to predict group membership (Participant Type), again the violent offenders reported the highest rate of prior heavy use (100%). All participants in the murderer groups also reported a history of heavy alcohol use, with the impulsive murderers heavily intoxicated at the time of their offences.

Exploration of drug history in Study One demonstrated that participants in the violent offender group were the most likely to report experimentation or heavy use of drugs, followed by the non-violent offender group, and then the control group. These differences were strong enough to be able to predict group
membership. There were no differences demonstrated between the murderer groups in Study Two as all of the impulsive murderers and all of the premeditated murderers reported a history of heavy drug use. No significant differences were demonstrated between participants in Study One in the violent and non-violent offender groups when examining intoxication and/or being high when committing offences. The high reporting rate of inmates stating they were intoxicated and/or high when offending is consistent with the literature on offending (Farrington, 1991b; Nurco et al., 1989; Volavka et al., 1992; Volkow et al., 1991).

Interestingly, when examining the rate of alcohol use, no incarcerated participant admitted to having been intoxicated in the last 48 hours prior to testing, or even consuming alcohol while in prison. This may be in part due to the difficulty in acquiring alcohol and in destroying the evidence (glass bottles etc). The finding that no community participants (who have easy access to alcohol) reported being intoxicated in the prior 48 hours may represent an under-reporting or minimising of actual alcohol use.

The finding from Study Two, of intoxication with both alcohol and drugs for all impulsive murderers whilst committing the murder, is of particular interest. This starkly contrasts with the finding that the premeditated murderers reported no intoxication while offending (and this was noted in their court files). Although there was no difference between the two murderer groups on the BADS scores, and only one of the murderers (premeditated) scored in the Impaired range, its possible that the intoxication of the impulsive murderers suggests that they may have been suffering from short-lived severe ED at the time they murdered. This hypothesis has been explored in the discussion of Study Two in Chapter Five, and is consistent with other literature (for example, Abram, 1989; Giancola, 1995; Giancola & Zeichner, 1994, 1997; Hoaken, Assaad et al., 1998; Lau et al., 1995; Leonard et al., 2003). However, any speculation is limited by the restricted sample size.
Marijuana was the only drug reported as used within 48 hours prior to testing by the men across the sample. Whilst drug use within 48 hours of assessment is potentially problematic for optimum neuropsychological performance, and may have influenced the scores obtained by those men who report this, this regular use of marijuana is ‘normal’ for these men, in and outside of prison, and in that sense is no different than assessing men with a history of TBIs or possible permanent brain damage from alcohol abuse. All of these factors can increase cognitive dysfunction and probably executive dysfunction. It is likely that men who take drugs in prison had also taken drugs at least 48 hours before their offending, given their reported habits. Thus, in these cases, drug intoxication is an accurate reflection of their ‘normal’ state and hence reflective of their state when offending, and in rehabilitation programmes. Understanding that inmates may be regularly smoking marijuana, with minimal intent to reduce or stop, informs the development of rehabilitation programs accommodating the possible differences in brain function and cognition that may be evident in these inmates.

Nevertheless, in an analysis of the impact on the BADS of substance use in the 48 hours prior to testing in the Study One sample, no association was demonstrated between those who had smoked marijuana and their BADS scores. When the men who reported smoking marijuana in the last 48 hours were removed from the sample and the tests rerun on only the participants who did not report recent marijuana use, the significant differences for the BADS, BDI-II and Years of Education across Participant Types remained at similar levels.

Only one participant in Study Two (participant six) admitted to having used marijuana in the last 48 hours before testing, and all his scores fell within the normal range. The rate of one participant reporting marijuana use may be an underestimation of the drug use by inmates in Study Two compared to the rate of drug use revealed in Study One. Alternatively, it may accurately reflect the difficulty in accessing, hiding, and using marijuana in the maximum security wing of the prison, where random drug tests are carried out more often than in medium
and minimum security. A possible hypothesis is that the lack of tetrahydrocannabinol (THC) in Study Two participants might explain why little ED was found compared to the violent offenders in Study One. However, the analysis in Study One on the men who hadn’t smoked marijuana recently does not support this hypothesis.

When the men in prison who admitted to using marijuana recently were questioned about their reasons for smoking, many of them discussed how they “needed” to do it in order to “stop from going mad,” due to the boredom of prison life. Some also stated that smoking marijuana helped them to reduce their levels of aggression and “to chill.”

7.3 Implications of the Studies

There is much debate over whether a unifying construct or a component construct best informs knowledge on executive functions, or indeed provides the basis of executive function itself. In taking into account the way different executive deficits can impede performance in different individuals, task-impurity when measuring executive functions, and the manner in which the prefrontal cortex and executive functions play a role in top-down processing, the BADS used in this study is a useful compromise reflecting both unity and component concerns. The BADS was developed in order to reflect the real difficulties faced by those with ED that may not be measured in regular executive tests. The BADS is based on the premise that some of the building blocks of an individual’s executive function may be intact, but the ability to access and use these blocks (especially when under stress) may not be (Wilson et al., 1998). It may be that the utility of the BADS reflects the usefulness of hybrid models, incorporating both unity and component approaches, in understanding executive functioning.

The BADS demonstrated application in Study One, appearing to effectively access and assess underlying maladaptive functions that contribute to violence in
recidivist violent offenders, although it was less useful with the murderer sample in Study Two. As has previously been discussed, the inclusion of additional tests may have increased the likelihood of measuring any deficits. The inclusion of additional tests in future studies is discussed more fully in the Limitations section of this chapter.

The results of Study One indicate that executive dysfunction influences the expression of violence, especially when poor education histories and drug and/or alcohol abuse are present. How existing executive deficits, poor education, and substance abuse interact is not clear, but the prefrontal cortical areas that are typically deficient in ED are the same as those that are implicated in substance users with violent behaviour (Fishbein, 2000). These cortical underpinnings of ED and substance abuse described by Fishbein (2000) may be the same as those underlying the behaviour demonstrated by violent offenders from Study One. Acute alcohol intoxication can result in an inability to inhibit inappropriate behaviours, sometimes leading to aggression (Gantner & Taylor, 1992; Gerson & Preston, 1979; Giancola & Zeichner, 1994; Lau et al., 1995). Heavy alcohol use is a known exacerbating factor for ED and violence, at least in individuals who already have compromised executive functioning for other reasons, or a propensity for violence (Giancola & Zeichner, 1994, 1997). It may also be the case that low education or low intelligence contributes to an individual’s difficulty in utilising appropriate executive functioning to inhibit acts of violence or aggression. This utilisation of the executive functions in a useful and timely manner is a core aspect of what the BADS measures (Wilson et al., 1998).

The result of an interaction between TBI, ED, poor education, and substance abuse may culminate in chronic or acute ED of varying severity. The behaviours demonstrated may depend upon the varying strength of the different variables, the individual’s existing moral code and repertoire of behavioural responses, as well as the environment presently influencing behaviour. When an individual with vulnerability to ED is under further stress, risky and inappropriate decisions may
be made, possibly creating opportunities for offending. This type of ‘cumulative effect’ is consistent with the ‘pathway’ model proposed by Raine and Buchsbaum (1996), which states that many pathways lead to violence, and the more pathways ‘activated’ for a particular vulnerable individual, the greater the likelihood of violence. This too was the conclusion reached by the Aspen Neurobehavioural working group (Filley et al., 2001). Neurobehavioural susceptibility may reduce thresholds for violence but does not necessarily lead to violence on its own (Filley et al., 2001).

Clearly, avoiding excessive consumption of alcohol and recreational drugs is protective against making poor decisions and avoiding risk, and in reducing ED. In attempting to understand the protection a longer period spent in formal education provides, explanations are less immediately apparent. An initial explanation is simply that the extra time in school provides further exposure to learning, which will improve future decision-making. It may also be that remaining in school for a longer period of time improves the opportunities to socialise with peers, to witness appropriate modelling from teachers, and to develop a stronger moral code. More time spent in class may even reduce opportunities to engage in risky behaviours. From a neurodevelopmental perspective, this extra time may allow the brain to gain the necessary exposure required to aid maturation of the prefrontal cortex, and to put into practice executive skills.

An impoverished childhood could also increase the propensity for violence. In individuals who have been raised in households where violence is a frequent and ‘normal’ way to deal with demanding situations, a repertoire of maladaptive responses will be learnt, and possibly ‘hardwired’ (van der Kolk & Fisler, 1994; Perry, 1997). Delayed or compromised maturation of executive functioning may limit the possibilities of learning more adaptive ways of dealing with a stressful environment. Poor executive functioning may result in a failure in overall regulation of behaviour, and this could lead to poor decision-making, possibly
increasing the likelihood of violence and offending. Further, children and adolescents who are already demonstrating ED and/or antisocial behaviour are likely to behave in ways that result in increasing their risk of violent abuse from caregivers (Carr, 1999; Farrington, 1991a; Patterson, 1982) consequently increasing their risk of TBI and consequent ED.

In Study Two, themes that are similar to those apparent in Study One are observed. These include a TBI history, child physical abuse, and alcohol and substance abuse. At the very least, these themes appear as a common background in offenders’ histories, regardless of the final type and level of offending. Appraisal of this information only may lead to an assumption that similar conclusions could be drawn for the murderer sample in Study Two; i.e., the cumulative effect of poor developmental background combined with drug and alcohol abuse creates a pathway to offending through impacting on ED. However, the surprising finding that none of this appeared to influence BADS scores in the murderer sample, even given similar reports regarding background, may reduce the usefulness of the BADS as a comprehensive and sensitive battery for ED; at least in assessing those who have murdered. It may be that ED is not the construct underpinning a propensity for murder. Similarly, murder may be substantially different from other types of offending, and different even from assaultative offending that does not lead to murder. Specific impulsivity and affectivity tests may be required with this group to test the existence of any possible differences between premeditated and impulsive murderers. However, the largest factor influencing the lack of findings in Study Two may be that the sample size was too small.

In Study One it appears that the BADS may measure the way in which ED contributes to the propensity for violent offending in a particular individual. The sensitivity of the BADS in differentiating between the three participant groups in Study One has been aided by the methodological design. This highlights the importance of defining and separating the participant types being assessed.
Further, studies that include all types of offending in one group are likely to get mixed or non-significant results. The conclusion that the presence and severity of ED differentiates violent offenders from other groups is similar to literature finding differences between violent and non-violent groups using executive tests (for example, Valliant et al., 1999) as well as the general neuropsychological literature with aggressors or offenders (for example, Deckel et al., 1996; Giancola & Zeichner, 1994; Gorenstein, 1982; Stanford et al., 2003; Yeudall & Fromm-Auch, 1979), and imaging literature (for example, Raine et al., 1997).

Neuropsychological differences demonstrated in violent offenders compared to the other participants could be accounted for by the somatic marker hypothesis. In particular, inhibition and risky decision making without learning from mistakes is commonly demonstrated in violent individuals (Anderson et al., 1999; Blair & Cipolotti, 2000; Damasio et al., 1990; Damasio, 1996, 1998). Although somatic markers were not specifically examined in the current study, response styles reflecting poor inhibition and risky decision making were demonstrated on the two subtasks from the BADS that differentiated significantly between participants. The somatic marker hypothesis would describe violent offenders as having more difficulty in accessing their emotions, experiencing somatic markers during emotion, and using bio-feedback to influence decisions. For example, finding empathy for their victim and using this information to inform their future behaviour, or using emotional cues to delay gratification for a greater good, or interpreting a pounding heart and sweaty palms as ‘bad’ anxiety rather than a ‘good’ rush. Indeed some research demonstrates that some violent individuals, particularly those meeting criteria for psychopathy, do not experience the same somatic responses as the majority of people (Raine, 2002).

However, the impact of ED on decision-making can also be viewed as a processing difficulty without extending to a somatic marker hypothesis (Hoaken et al., 2003; Maia & McClelland, 2004). A processing difficulty is consistent with the poor overall BADS profiles of the violent offenders, as the overall BADS
profile score generated from a composite of all six subtasks attempts to estimate overall processing of executive function and the way in which the appropriate executive components are accessed and used (Chaytor & Schmitter-Edgecombe, 2003; Wilson et al., 1996; Wilson et al., 1998). The poor overall BADS performances by violent offenders in Study One are suggestive of processing difficulties. It is proposed that processing difficulties place a heavy load on working memory and will add to stress and fatigue, especially when under pressure, contributing to poor decision making. Further, the outcome of such a process may appear as impulsive behaviour to the observer, and if treated as such in an intervention, treatment may be ineffective. Demarcation of processing difficulties and impulsivity and the impact these have on violence were not undertaken in this study, but merit attention in future studies. Administering and comparing neuropsychological tests orientated towards distinct processing and impulsivity functioning may have utility.

Although the results of Study One indicate that ED is associated with violent offending, it should be viewed as an aggravating factor in violent criminal behaviour, consistent with the cumulative pathway model of Raine and Buchsbaum (1996). This limits its use as a mitigating factor for criminal responsibility. This is a position in line with the work on psychopathy (Hare, 1999). This would mean that defence counsel attempting to use the presence of brain dysfunction as an extenuating circumstance in offending would be under a burden of proof. They would be required to argue that the presence of brain dysfunction led to the offending and would have to eliminate all reasonable doubt (Diaz, 1995). Owing to the complex nature of violence, alongside the varied impact of TBI for different individuals, problematic research and differences of opinion in the area, using brain injury or the presence of executive deficits in a court of law to explain offending is very difficult. Certainly this is not commonly practiced in New Zealand. In North America where brain dysfunction explanations are attempted more often, they rarely stand up to the scrutiny required to provide proof beyond all reasonable doubt (Diaz, 1995).
A body of literature exists examining the impact of ED on psychopathy, and proposing ED as the construct underpinning psychopathy (for example, Hare, 1984; Raine et al., 2000; Stevens et al., 2003; Stanford et al., 2003). Using this approach, perhaps what has been measured by the BADS in this study is a neuropsychological component of psychopathy. Without corresponding diagnoses of the men in the sample or interviews with the PCL-R this is difficult to ascertain, but raises a question for future studies attempting to replicate the results.

One of the explanations for Psychopathy and violence is that they are underpinned by a compromised OFC (for example Blair, 2004). However, demonstration of the BADS sensitivity to ED in violent offenders throws into question singular OFC accounts of psychopathy, or at least singular OFC accounts of violence. A major subtask from the BADS that differentiated function was the Modified Six Elements Tests (MSET), and this relies more heavily on the dlPFC than on the OFC (Norris and Tate, 2000). The results in Study One may be evidence for the combination of OFC and dlPFC (and associated circuitry) working together to sustain appropriate social behaviour, rather than localising antisocial behaviour to one region alone. This suggestion supports current thinking; that both a dysfunctional dlPFC and OFC are implicated in the geneses of psychopathy, risky decision-making, and aggression (Maia & McClelland, 2004; Sanfey & Cohen, 2004; Seguin, 2004).

A caution to the suggestion that the BADS measures an underpinning of psychopathic behaviour is that deficits were not consistently demonstrated by the BADS in Study Two. If murderers are more likely to be psychopaths, the fact that the BADS did not demonstrate differences across the murderers, or indeed place them all in a deficit category, may reflect that the BADS is not measuring psychopathy. Obviously however murderers are not necessarily psychopaths, and being a psychopath is not a necessary requisite for being a murderer. But if a psychopathy explanation was to be used to explain the results amongst the violent
offenders in Study One, then perhaps the BADS failure to measure deficits in the murderers indicates it isn’t measuring the neuropsychological underpinnings of psychopathy, but something else specific to the particular sample of violent offenders in Study One. The limitation of the psychopathy explanation also stands for the non-violent offenders in Study One, who are breaking societal rules without physical violence, but may nevertheless meet criteria for antisocial personality disorder.

An interesting question that arises from these results is whether these findings can contribute to arguments regarding localising deficits or functions. There is a large body of recent literature hypothesising that the OFC is the primary structure for inhibition, appropriate decision making, and moral reasoning (Anderson et al., 1999; Blair, 2004; Damasio, 1996; Rolls, 1996). This angle implies that compromising the OFC will lead to deficits that will in turn lead to criminal and aggressive behaviour. Indeed poor OFC functioning has been consistently linked to participants with APD (Blair, 2004; Mitchell, Colledge, Leonard, & Blair, 2002; Rolls, 1996). Alternatively, a body of recent literature building on reviewed earlier research, states that the dIPFC impinges upon aggressive dyscontrol and criminality through the central function of integrating all of the executive functions from all areas. This theory asserts that the dIPFC principally contributes to frustration tolerance, planning, and directing behaviour towards a future goal whilst delaying reward (Giancola, 1995; Seguin et al., 1999; Manes et al., 2002; Hoaken et al., 2003; Maia & McClelland, 2004; Sanfey and Cohen, 2004; Seguin, 2004).

The dIPFC can be described as the ultimate executor of all the component functions belonging to executive functioning. This is consistent with both neural models (Fishbein, 2000) and cognitive models such as the CE and SAS and Duncan’s adaptive coding model (Duncan, 2001). In view of this, it is suggested that the dIPFC receives more attention in future research on understanding the genesis of aggressive behaviour and the rehabilitation of criminality and
aggression where ED is noted. Future investigations privileging the dlPFC will counterbalance the large amount of literature specific to the OFC as the structure of importance in neurobehavioural explanations of violence. Explanatory models using only an OFC account can be criticised for measuring impulsivity but not violence itself (Krakowski, 2003), and that being impulsive does not necessarily equate with being violent.

Much of the literature appears to converge in consensus that it is the combination of biological factors (including TBI), abusive rearing, bad modelling, and a lack of opportunity to ameliorate these factors that merge to create violence in our communities (Brooks et al., 1986; Dawson et al., 1994; Deckel et al., 1996; Jones, 1992; Karr-Morse & Wiley, 1997; Kaufman et al., 2000; O'Leary, 2000; Otnow-Lewis, 1983; Otnow-Lewis et al., 1985; Otnow-Lewis et al., 1986; Raine, 1993; Raine et al., 2000). The results of the current study suggest a history of substance abuse and poor educational opportunities may be additional contributing factors to violence. It is not inevitable that the presence and combination of these factors will lead to a life of crime or violence, but it is arguable that chances (pathways) are increased.

Numerous factors may coexist and interact, paving the pathway to violence. A developmental background of abuse and neglect together with damage to the brain has been associated with executive dysfunction (Perry, 1997), and may facilitate establishment of a repertoire of negative behaviours. Abuse and neglect combined with poor school attendance and leaving school early may impede the development of executive functions. Multiple TBIs may result from an associated increase in risk-taking or even the participation in encouraged sports. The addition of a drinking or substance abuse habit may damage and acutely anaesthetise the brain, particularly the prefrontal cortex, providing the ‘final straw’ for someone with limited executive skills and prefrontal function. Intoxication may further diminish an individual’s ability to regulate their behaviour and may prompt an increase in perceiving the environment as more threatening then it really is. Lack
of regulation by the prefrontal lobes increase the likelihood of reacting with little regard to any consequences. The impulse to act and the act itself are thus no longer sufficiently mediated by top-down processing, increasing the likelihood of egocentric or violent behaviour. The effect of intoxication on executive function and behaviour shares similarities with the sequelae manifested by an adult sustaining a significant head injury, if similar developmental and environmental factors are shared. If an individual already has overall low brain arousal combined with impoverished social skills, a series of TBIs, even if each is only mild or moderate, could reduce prefrontal function and have dire consequences, including problems with goal-directed behaviour, disinhibition, possibly leading to the expression of violence.

The bleakness of the description above is ameliorated somewhat by the recognition that not every child with a developmental background of abuse and organic damage to the prefrontal cortex will be forced onto a path of violence and crime. Not all children exposed to alcohol in-utero, or who are abused physically and emotionally, grow up to have learning and/or behavioural difficulties, or continue the cycle of violence, especially if there are moderating factors. These may include the presence of an adult in the child’s life who bolsters his self-esteem and offers ‘unconditional’ positive support, or a series of interventions that provides the young person with alternative choices, and an environment which supports these choices. Kapa Hakā groups (Māori performance groups) and Māori language nests in schools provide one positive example of an intervention with the potential of a range of positive outcomes (Robust, 2000; Smith, 2000). It is of relevance that Kapa Haka and Māori language courses within the prisons have also had a positive impact as reported by many of the Māori participants in the study. In fact they were the only interventions that incarcerated participants made positive comments about.

It is not the purpose of this thesis to propose that a ‘damaged’ prefrontal cortex or poor executive functioning alone causes violence, but rather, that ED reduces the
individual’s ability to inhibit ‘antisocial’ behaviour, to monitor, regulate and adapt behaviour, and to make ‘good’ decisions. Whether these behaviours arise from a developmental history, are acquired or are transient, is not the core issue in this study. Instead, it is that they are associated more often with violent offenders than non-violent offenders and community controls. That they exist in violent offenders deserves addressing, as violent offending is costly financially and socially for society. Their existence compels and informs models of forensic rehabilitation to adapt models or develop new ones that account for the needs of violent inmates with ED. Appropriate rehabilitation is required in order to make any serious headway in the recidivism of violent offenders.

For the inmates who participated in Study One and Two it could be argued that there have been limited moderating effects throughout their life, resulting in the strengthening of pathways towards violence and offending. The questions raised by this study include moral and legal considerations. If an inmate is unable to be rehabilitated, as they do not have the neural structures that allow new information to be laid down, strengthened, and accessed, should they remain behind bars indefinitely? Or can inmates with significant executive deficits be rehabilitated? As discussed in Study Three, it is advocated that inmates with ED need specialist rehabilitation rather than the current standard rehabilitation programmes that are currently offered in the prison service.
7.4 Limitations

The limitations of the studies need to be taken into account when interpreting the results. The limitations described below, constrain the generalisability of results to other incarcerated populations.

Small Population

The findings of Study One cannot be easily generalised to other settings or the general population, as the numbers involved are small. The small sample size limits the power of the analyses, and this needs to be considered when interpreting significant effects and findings of non-significance. It is not unusual to encounter difficulties of small sample size and non-normal data in psychology research that relies on special populations. However data from studies with smaller populations are often included in future meta-analyses in the research area, and are additionally strengthened by replication.

This study aimed to recruit a minimum of 60 participants (20 in each group) in order to meet minimum requirements for power. Having equal numbers across the participant groups would have been of benefit, and perhaps made the distribution more normal. Recruiting participants for this study was a very difficult task. To remain blind to the conviction history of the incarcerated inmates it was necessary to rely on liaison personal within the Department of Corrections at Auckland Prison to identify possible participants. This was an arduous task for an already overworked prison staff encountering continual management changes. Changing management often impacted upon my working relationships with the chosen liaison person, and I worked with four different members in the liaison team at different times. This considerably reduced the rate of participants coming through to the point of consent, as each new liaison person needed to be trained in the process, and a sufficient relationship developed so they would ‘go out of their way’ to aid in the research. At times the relationship with the Department of
Corrections would become somewhat strained, and data collection was impractical. Further, after assessment was completed, and participants had been categorised according to their BADS scores, it was noted upon three occasions when checking the master sheet prepared by the liaison persons that they had sent through participants they thought fitted the criteria for a non-violent offender, but in fact met the criteria for a violent offender. Another two participants were found to have sexual offending in their background, therefore not meeting the criteria to remain in the study. These difficulties account for some of the unevenness of numbers noted between the two offender groups.

**Non-Randomised Sample**

The non-random assignment of participants in the studies can be criticised, and categorises the studies as quasi-experimental, therefore lessening the validity and reliability of the results, and reducing their generalisation capacity. However, the nature of the variables in this study precludes random assignment, as the participants must give voluntary consent for the studies (for which there is no incentive scheme), and must meet certain criteria before beginning the assessment. It can be argued that most psychological studies involving the behaviour of human populations should be concerned about self-selection.

An additional limitation of the non-randomised sample was that I was able, consciously or unconsciously, to make assumptions about the inmate presenting to testing by the whereabouts of the interview. Hence, in the maximum-security wing I might assume that the participant is a violent offender. Similarly, in the minimum-security wing I might assume the inmate is incarcerated for non-violent offences. However, assumptions about inmates in minimum security may be incorrect as all inmates pass through minimum security towards the end of their sentence, and I had no access to information regarding where in their sentence an inmate was. These limitations are revisited later in the discussion.
Background Information

It may be that gaining further information about each of the participants (such as medical records and family situations) would have guided the interpretation of the results, particularly in trying to make tentative links of aetiology to any ED noted. However, it was never the intention of this study to be able to make claims of aetiology; instead the focus was on the current functioning of the men involved, and how this may have impacted on their group membership (for example, were men with ED more likely to be violent offenders?).

In this study it is recognised that the aetiology of ED is complex and varied, and to speculate the causes of ED for individuals in this study is not useful. Instead, understanding that a high proportion of inmates have ED, and the nature of that ED, can better inform the structure, style, and types of rehabilitation offered to inmates.

Definition of Violence

Conviction (at least twice) for violent offending that physically harms another (such as grievous bodily harm or assault) was used to operationally define violence. However, it is possible that incarcerated participants in the non-violent offender group have never been ‘caught’ and convicted for violent offending, and it may be that a number of men in this group have been violent in their everyday lives. Additionally, there is evidence that the longer someone has been offending, and the younger they started, the more likely their offending will become violent (Rich, 2000). It may be that some men in the non-violent offender group will in future be convicted of violent crimes. A valuable piece of information that could be included in subsequent research is the collection of ‘rap sheets.’ These record the number of offences (and when they occurred) an individual has been convicted of over their entire offending period. This would allow an estimate of any differences between the number of crimes in total for violent offenders and
non-violent offenders, and enable the hypothesis that ‘a high total number of offences predict violent offenders’ to be tested.

Unfortunately, the possible benefit of the collection of rap sheets to the design of the study was not apparent until the testing had been completed. However, at that point it was no longer feasible to gather official individual offending histories retrospectively. Accessing these records would require paying for the use of the vast Police and Corrections IOMS (Integrated Offender Management System) computers and returning to the participants to gain consent to access these records. New ethical approval would also be required. By the time testing was complete some of the men had already been released, and it would have been very difficult to locate them for consent purposes.

**Control Group**

Recruiting an appropriate control group will continue to be a significant challenge for research in this area. The high variability noted across the two incarcerated groups, which cannot be changed by the researcher, also makes it difficult to make assumptions about an incarcerated population as a whole, and makes finding a well-matched control group problematic.

The control group in this study did not match the incarcerated participants closely enough in levels of education. This disparity in Years of Education between the groups may have been responsible for some of the differences demonstrated in neuropsychological outcomes, and especially on the BADS score. A larger control group screened for Years of Education and estimate of Verbal IQ may have been an improvement. Also the inclusion of more men from job assistance courses and fewer prison officers used as controls would have resulted in a closer match. Future research should endeavour to make closer matches between ethnicity, age, education, work-history, and estimated IQ. However, in Study One, this would have required a case-by-case matching approach, or recruiting the control group
after the demographic analysis of the incarcerated groups was complete, with its own inherent difficulties

**Self-Report**

One of the methodological difficulties with self-report is social desirability and answers are often more related to ‘self-description,’ rather than an accurate portrayal of events and behaviours (Suris et al., 2004). All data collected on history of TBI and substance use were obtained via self-report from the men in the study, and interpretation is cautioned by this caveat. No corroboratory information was gathered, including medical reports for incidence of TBI. After discussions with the participants of the nature of the brain injuries reported and how they were acquired, it became clear that often there would be no medical information available. For example, many participants reported “just waking up” after falling, for example from a tree as a child or from their bike, and going home and not telling anyone and “sleeping for a few days.” Some participants acquired brain injuries during offending behaviour, such as crashing a stolen car, so were too scared to go to a hospital or general practitioner. Many participants also reported “knocks to the head” or “blackouts” during physical abuse from a parent or close relative when they were children, and it is unlikely that medical attention was sought in these circumstances.

Self-report was also relied upon for the control group when inquiring into ‘violence status.’ In order to be included in the control group, participants had to state that they had been ‘violence-free’ for at least five years. It is possible that some men participating as control participants may have been violent on a number of occasions in the past five years prior to the study, but were not willing to admit it, or indeed, did not even recognise it. However, control participants did freely offer (without incentive) to participate after being given information about the importance of not having acted violently in the past five years at least, and there is
no obvious reason to suspect that men who were aware of their own violent behaviours would volunteer.

**Gender**

These studies are restricted to an analysis of ED and its relationship to violence perpetrated by males. Future studies could and should involve female participants. Women are incarcerated less often in New Zealand than men, therefore providing less access. However, studies that compare both male and female behaviour may be able to further highlight the impact of certain neurodevelopmental issues, such as ED on violence, and also examine for gender effects, particularly related to hormonal differences and fluctuations, and the possible effects these may have on brain development and neuropsychological functioning.

**Ethnicity**

As was already discussed, the inclusion of more Pakeha in the incarcerated groups and Māori in the control groups would have led to conclusions that were more reliable when testing the general hypothesis that ED and violence are related, independently of ethnicity. However, the numbers of different ethnicities across the sample reflect those in the incarcerated New Zealand population (Rich, 2000). It may be that adjusting the numbers across the groups (increasing Māori in the control group and Pakeha in the offender groups) would artificially change the proportions to the extent of reducing the validity of a study examining rates of ED across a sample of New Zealand inmates.

**Cultural Fairness and Neuropsychological testing in a New Zealand Population**

Ideally in using neuropsychological tests, the data obtained should be compared to norms gained from a population that is representative of the sample. The use of the COWA-M norms adapted by Ogden et al. (2003) is an example of this, along
with the knowledge that the current STW norms appear fair for use with Māori and Pākehā New Zealanders (Ogden et al., 2003). However, all the other tests used in this study have American or United Kingdom norms only. However, even where the Ogden et al. (2003) norms have been used, it may be that incarcerated men are substantially different from men not incarcerated, and may require their own set of norms. This was especially apparent when questioning the incarcerated participants about their answers on the BDI-II, and on the questions comprising the Temporal Judgement subtask of the BADS.

Of course, as Ogden (1996) points out, using normative data alone does not entirely solve the problem of cultural fairness. Fair testing across cultures must take into account approaches to and understandings of the tests. Poor school performance and early leaving may impact on a formal neuropsychological testing session. This may be particularly pertinent in the multi-ethnic sample tested in these studies, as both incarcerated and non-incarcerated participants, and Māori, in particular, reported poor school attendance.

That said, when the results were examined in order to predict group membership, compared first for all ethnicities, and then for Māori, the finding that violent offenders were more likely to do poorly on tests of executive functions held.

Type I and II Errors

The small number of participants in Study One raises the potential for a Type I error; that significant effects may be found due to the influence of outliers on a small sample. Alleviating this concern in part, is that in general there were large effect sizes obtained in the expected direction at \( p \leq .01 \) between the variables of interest, even when using non-parametric methods.

However, the further the analysis gets from the raw data, and the stricter the levels of significance, the stronger the likelihood of making a Type II error. In this study
the use of .01 as the significance level of the rank examinations may have increased this risk, but the initial analyses using ANOVA set at $p = .05$ was an attempt to reduce this likelihood. Even with the strict parameters set, variables associated with Participant Type continued to demonstrate a strong and significant relationship.

Again, as discussed under *Small Population*, increasing the sample size would aid in reducing errors and the influence of outliers, strengthen the significant findings, and extend the applicability of the findings to similar populations.

**Limited Testing Battery**

The BADS was chosen to address issues of ecological validity, pragmatic-testing constraints, informing rehabilitation structure, and to attempt to account for both unity and component constructs in executive functioning. The BADS fulfilled these criteria well and differentiated executive functioning across participant types. However, other executive tests would provide evidence for or against the concurrent validity of the BADS subtasks with standard executive tests. In addition, other components may be assessed by other tests that the BADS does not measure. A broader selection of executive tests may result in a clearer and stronger argument for or against the hypothesis that violent offenders have poorer executive functioning than other groups. In order to explore questions of impulsivity versus poor processing in explaining the variance in aggression, inclusion of tests purported to test these functions would be useful.

Suggestions for other useful tests include the Go/No-Go test and the Iowa Gambling Task (Bechara et al., 1994), both of which have been used with APD individuals, normals showing aggression, and offender populations; with varying degrees of deficit or no-deficit noted (Dinn & Harris, 2000; La Pierre et al., 1995; Maia & McClelland, 2004; Malloy et al., 1993; Stevens et al., 2003). Adding these tasks may be particularly useful in addressing issues of impulsivity and
decision-making in relation to offending, in a manner that is more comprehensive than the BADS alone. Further, use of these measures would add to the literature that is attempting to establish them as reliable executive tests in offender populations.

However, the addition of the Go/No-Go task may be contentious as it is considered by some to be a test of general impulsivity only, rather than a useful test localising it as a component of executive functioning (Hoaken et al., 2003). Other authors have also found that it was not sensitive to differences between APD participants (assumed similar to offenders) and controls (Dinn & Harris, 2000; Stevens et al., 2003).

The Iowa Gambling task is alleged to rely on a paradigm too complex for participants to follow using cognition alone, so that participants must rely on emotion-based learning systems (somatic markers) thought to be the domain of the OFC (Bechara et al., 1994; Damasio, 1996). However recent research claims that localising the task to OFC and emotional learning only is contentious and that the dlPFC is involved, as demonstrated by participants demonstrating high-level cognitive knowledge of the task and the use of complex cognitive strategies to complete the task (Maia & McClelland, 2004). Further, other studies have demonstrated that patients with localised dlPFC lesions can perform very poorly on the Gambling task, and OFC patients can perform in line with controls (Fellows & Farah, 2004; Manes et al., 2002). Many authors now hypothesise that the relationship between emotion and decision-making in executive functioning is more likely to reflect interplay, rather than a heavy reliance on one or the other (Sanfey & Cohen, 2004; Maia and McClelland, 2004).
7.5 Future Directions

Study One was strengthened by the use of healthy controls, but as the matching was only approximate, future studies would benefit from increasing the size of healthy control groups. Additionally, replication with larger samples across all groups would allow separate comparisons for Māori, Pacific Islands People, and Pakeha as well as strengthen the general findings across the sample. Larger numbers would address issues of power and may be able to address issues of distribution. Even though a clear definition of type of conviction was used to define the violence measured in Study One, difficulties arose when relying on prison staff to clarify offenders. Clearer protocols in future studies are required for clarifying offenders as violent and non-violent, and the definitions used here provide a useful example for future studies. If at all possible, random selections of inmates from a large enough pool and later delineation to offender type will further strengthen any findings of replication studies.

Interesting questions raised by the current study that warrant closer examination in future studies examining ED in violence are the effect of education, both on executive functioning and on violence and offending and the impact of processing difficulties and impulsivity. ‘Years of Education’ was demonstrated to have an effect on group membership that was almost as large as the impact of the BADS score. More research is needed about the impact of education on performance in formal testing situations, on an individual’s ‘intelligence’ and ability as measured by psychometrics, and on executive functioning. It could also be that years of education reflect an individual’s environment via reflection of the familial value of education, or the familial education and socio-economic status. It may be that increased exposure to education is associated with reduced exposure to drug and alcohol use, unemployment, and risk-taking. Examining these questions using longitudinal methods is the next important and ambitious step, and may clarify any physiological impact socialisation and education has on the brain, especially in promoting executive function and moral development. Structural and functional
brain imaging carried out alongside neuropsychological testing would also be useful, to highlight brain areas damaged or dysfunctional across individuals and participant types, and would add to the literature on brain function and location. However, executive functioning represents a vast category and imaging may not yet be sophisticated enough to provide useful information about ED.

Specific tests purported to reliably measure OFC function may contribute to addressing questions regarding the part OFC and impulsivity play in regulating aggression. Further, ‘planning’ tests that reliably measure purported dIPFC and ACC function, like the Tower of London (ToL), may also have been useful, both to check rating reliability between the ToL and the BADS, particularly the subtask Modified Six Elements, and in providing further information on the importance of planning and directing behaviour. General processing speed can often be assessed alongside tests examining other executive function, and it can also be measured using subtests from the WAIS-III as well as other specific tests. Other tests that are asserted to test function mediated by other brain regions, such as the temporal region and the parietal cortex, may also be useful in describing or discounting overall brain dysfunction in offenders. The absence of deficits on tests that examine posterior brain-behaviour relations could strengthen any findings on anterior or executive function tests.

A future battery for a similar study with inmates and community controls might include the BADS, ToL, Go-No/Go, the Gambling Task, STW or NART, and spatial function test(s) of choice along with processing tests of choice. As well as these tests, certain questionnaires may have been useful to provide information about participants’ thinking styles, morality, and level of psychopathy; in order to explore whether any of these variables were related to participant type or associated with certain neuropsychological tests. For example, an impulsivity questionnaire like the Barratt Impulsivity Scale – II (BIS-II), or an aggression survey, or the very complex Psychopathy Checklist – Revised (PCL-R) would have been informative and allowed comparison to similar research. In particular,
using the PCL-R would provide comparison with the vast psychopathy literature that is beginning to examine prefrontal deficits as an explanation for APD. In this study the use of the PCL-R would not have been feasible, as the examiner had not had the thorough training required to administer the PCL-R competently in order to gain valid results.

The most promising and fruitful approaches to violence in society take a preventive stance and recognise that violence cannot be addressed by a one-dimensional approach. Research bringing together the multifaceted contributing factors to violence would better inform prevention approaches. A useful prevention strategy would need to involve action by educational, legal, health, social, and community agencies. In a clinical context, treatment of violence in those with known brain dysfunction or cognitive dysfunction can play a role in future prevention of violence. This may include pharmaceutical intervention as well as behavioural rehabilitation, and appropriate rehabilitative strategies for inmates with ED require promotion.

7.6 Final Conclusions

Poor executive functioning is common in the prison population examined, and is seen more often in inmates who repeatedly offend violently (without causing death). In the 53 men who participated in Study One, significant differences were found across participant types on their neuropsychological functioning, particularly in executive functioning as measured by the BADS, and on the variable Years of Education. This relationship is not significantly influenced by differences between the two main ethnic groups involved in the study (Māori and Pākehā), as a significant effect remained for the BADS scores across the three groups when Māori data only were analysed.

There were also significant effects noticed for substance abuse, with those with a strong drug use history being more likely to be incarcerated, and those with the
strongest drug history, combined with an alcohol history and a TBI history, belonging to the violent offender group. All these variables may interact to lower an individual’s executive function as measured by the BADS. The variables highlighted as contributing to violence, or at least associated with the violent offender sample, can be applied to prevention and rehabilitation interventions with at risk (for criminality and violence) individuals and recidivist violent offenders.

In Study Two, which examined neuropsychological differences in men who were convicted of premeditated and impulsive murders, marked differences were not apparent. This may be due to the small number in the sample and the difficulty in demonstrating group membership, (i.e., premeditated or impulsive murder). Notable however was the use of alcohol and other substances by the impulsive murderers at time of the murder, who all claim poor memory of the actual murder due to the degree of intoxication. Although clearly intoxication only rarely results in murder, perhaps in these men the ED resulting from heavy intoxication was sufficient to ‘permit’ them to commit murder. If applying a paradigm that includes ED as a pathway to violence, why these men had the potential to murder without chronic ED, is not clear, unless explained by substance induced temporary ED. However, such a statement can only remain speculative. In a future study, other factors examined via other psychometrics may provide further explanation of any differences that exist, as may a larger sample size. However, it may be that no strong neuropsychological differences exist between premeditated and impulsive murders, or at least differences that can be assessed with current neuropsychological tests.

Study Three detailed the methodology of a study designed to measure the outcome of an intervention for inmates with ED. It is unfortunate that this intervention, and resulting study of the outcome, was not able to go ahead as planned. Nevertheless, the development of the intervention draws attention to the imperative of designing rehabilitation strategies for offenders that account for
their individual differences. The high association of ED in violent offenders in Study One suggests that if inmates with ED are not offered specialised rehabilitation, there may be little expectation of actually helping these individuals make long-term behavioural changes and impact on recidivism.

This thesis has focused on violence and the neuropsychological pathways that lead to violence. The aim of identifying pathways to violence is that describing them may assist the development of strategies to effectively diminish the strength of these pathways. Identification of the factors contributing to a propensity for violent offending may enable an individual to compensate for deficits that impact on behaviour leading to violence. It appears that ED is one of those pathways, and a poor academic background and a history of substance abuse further strengthen this pathway. Although the rehabilitation of individuals with ED is difficult, there is hope with future research that remedies can be found. An intervention specific to each offender type, tailored towards each individual’s neuropsychological profile and strengths and weakness is essential. For violent offenders with ED, use of repetition and a sequential ‘building-block’ format in rehabilitative interventions may prove to have utility. Difficulties may arise in generalising skills gained within the intervention, beyond the prison setting and hence follow-up and reinforcement of these skills upon release of the offender back into the community will be imperative.


References


References


References


References


treatment of violent, aggressive behaviour. *Acta Neurochirurgica, 44*, 145-
151.

injury in cognitive functioning, emotional adjustment and criminal
behaviour. *Brain Injury, 12*(10), 821-842.

orbitofrontal damage: neuropsychological and strategic management

Zealand.


vulnerability to psychopathology: temperament traits associated with

Schneider, F., Habel, U., Kessler, C., Posse, S., Grodd, W., & Muller-Gartner, H.
(2000). Functional imaging of conditioned aversive emotional responses in
antisocial personality disorder. *Neuropsychobiology, 42*, 192-201.

Determinants of Benton Facial Recognition Test performance in normal

Seguin, J. R. (2004). Neurocognitive elements of antisocial behavior: relevance of

neuropsychological characteristics of physically aggressive boys. *Journal

lobe metabolism in violent subjects: correlation of imaging and
neuropsychiatric findings. *American Journal of Neuroradiology, 18*, 625-
631.

apes share a large frontal cortex. *Nature neuroscience*.

of the Royal Society of London. Series B-Biological Sciences*, 298, 199-
209.

Shallice, T., & Burgess, P. (1991). Deficits in strategy application following

Shallice, T., & Evans, M. E. (1978). The involvement of the frontal lobes in

lobe damage in memory disorders. In H. S. Levin & H. M. Eisenberg & A.
L. Benton (Eds.), *Frontal Lobe Function and Dysfunction* (pp. 173 - 195).
New York: Oxford University Press.


offenders and impulsive fire-setters. *Archives of General Psychiatry, 46*, 600-603.


Appendices

Appendix One:
Result information for Study One

Appendix Two:
Forms used in the studies

Appendix Three:
Rehabilitation Information
Appendix One

General Results Table for Study One
<table>
<thead>
<tr>
<th>Participant Type</th>
<th>Ethnicity</th>
<th>Age</th>
<th>BADS&lt;sup&gt;a&lt;/sup&gt;</th>
<th>STW&lt;sup&gt;b&lt;/sup&gt;</th>
<th>COW A-M&lt;sup&gt;c&lt;/sup&gt;</th>
<th>Years of Education&lt;sup&gt;d&lt;/sup&gt;</th>
<th>BDI-II&lt;sup&gt;e&lt;/sup&gt;</th>
<th>LIKERT&lt;sup&gt;f&lt;/sup&gt;</th>
<th>QQ&lt;sup&gt;g&lt;/sup&gt;</th>
<th>Drug History</th>
<th>Alcohol History</th>
<th>Drug Use</th>
<th>Intoxication during offence</th>
<th>TBI History</th>
<th>TBI Before First Conviction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Violent</td>
<td>Māori</td>
<td>24.00</td>
<td>14.00</td>
<td>4.00</td>
<td>49.00</td>
<td>9.00</td>
<td>26.00</td>
<td>2</td>
<td>2</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Violent</td>
<td>Māori</td>
<td>25.00</td>
<td>13.00</td>
<td>4.00</td>
<td>30.00</td>
<td>7.50</td>
<td>33.00</td>
<td>5</td>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Māori</td>
<td>24.00</td>
<td>11.00</td>
<td>11.00</td>
<td>36.00</td>
<td>8.00</td>
<td>8.00</td>
<td>4</td>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Māori</td>
<td>40.00</td>
<td>16.00</td>
<td>5.00</td>
<td>32.00</td>
<td>8.00</td>
<td>27.00</td>
<td>2</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Māori/non-Māori</td>
<td>33.00</td>
<td>16.00</td>
<td>3.00</td>
<td>26.00</td>
<td>4.00</td>
<td>12.00</td>
<td>3</td>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Violent</td>
<td>Pacific Island</td>
<td>22.00</td>
<td>15.00</td>
<td>6.00</td>
<td>37.00</td>
<td>7.00</td>
<td>14.00</td>
<td>2</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Pacific Island</td>
<td>32.00</td>
<td>11.00</td>
<td>7.00</td>
<td>14.00</td>
<td>10.00</td>
<td>29.00</td>
<td>4</td>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Māori</td>
<td>36.00</td>
<td>9.00</td>
<td>7.00</td>
<td>46.00</td>
<td>8.00</td>
<td>7.00</td>
<td>2</td>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Māori</td>
<td>32.00</td>
<td>17.00</td>
<td>10.00</td>
<td>60.00</td>
<td>10.00</td>
<td>5.00</td>
<td>3</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Māori/non-Māori</td>
<td>30.00</td>
<td>9.00</td>
<td>8.00</td>
<td>22.00</td>
<td>8.00</td>
<td>4.00</td>
<td>3</td>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Pakeha</td>
<td>44.00</td>
<td>15.00</td>
<td>10.00</td>
<td>28.00</td>
<td>10.00</td>
<td>24.00</td>
<td>3</td>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Participant Type</td>
<td>Ethnicity</td>
<td>Age</td>
<td>BADS</td>
<td>STW</td>
<td>COW A-M</td>
<td>Years of Education</td>
<td>BADS</td>
<td>STW</td>
<td>COW A-M</td>
<td>Years of Education</td>
<td>LIKERT</td>
<td>QQ</td>
<td>Drug History</td>
<td>Alcohol History</td>
<td>Drug Use</td>
</tr>
<tr>
<td>------------------</td>
<td>----------------</td>
<td>-----</td>
<td>------</td>
<td>-----</td>
<td>---------</td>
<td>-------------------</td>
<td>------</td>
<td>-----</td>
<td>---------</td>
<td>-------------------</td>
<td>--------</td>
<td>----</td>
<td>--------------</td>
<td>-----------------</td>
<td>----------</td>
</tr>
<tr>
<td>Violent</td>
<td>Māori</td>
<td>31.00</td>
<td>21.00</td>
<td>12.00</td>
<td>30.00</td>
<td>9.00 .00</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Māori</td>
<td>22.00</td>
<td>15.00</td>
<td>5.00</td>
<td>32.00</td>
<td>9.00 4.00</td>
<td>4</td>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Māori</td>
<td>26.00</td>
<td>15.00</td>
<td>11.00</td>
<td>27.00</td>
<td>8.00 13.00</td>
<td>4</td>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Māori</td>
<td>30.00</td>
<td>16.00</td>
<td>9.00</td>
<td>36.00</td>
<td>9.50 10.00</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Māori</td>
<td>38.00</td>
<td>17.00</td>
<td>7.00</td>
<td>29.00</td>
<td>9.00 8.00</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Pacific Island</td>
<td>21.00</td>
<td>7.00</td>
<td>6.00</td>
<td>36.00</td>
<td>8.50 16.00</td>
<td>2</td>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Māori</td>
<td>21.00</td>
<td>16.00</td>
<td>5.00</td>
<td>36.00</td>
<td>9.00 2.00</td>
<td>4</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Māori</td>
<td>34.00</td>
<td>4.00</td>
<td>4.00</td>
<td>22.00</td>
<td>7.00 31.00</td>
<td>4</td>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Pacific Island</td>
<td>38.00</td>
<td>14.00</td>
<td>2.00</td>
<td>24.00</td>
<td>7.00 29.00</td>
<td>3</td>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Violent</td>
<td>Māori</td>
<td>21.00</td>
<td>11.00</td>
<td>6.00</td>
<td>21.00</td>
<td>9.00 12.00</td>
<td>2</td>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Pakeha</td>
<td>38.00</td>
<td>17.00</td>
<td>11.00</td>
<td>39.00</td>
<td>11.00 5.00</td>
<td>2</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Māori</td>
<td>21.00</td>
<td>15.00</td>
<td>5.00</td>
<td>34.00</td>
<td>9.00 22.00</td>
<td>2</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Participant Type</td>
<td>Ethnicity</td>
<td>Age</td>
<td>BADS</td>
<td>STW</td>
<td>COW A-M</td>
<td>Years of Education</td>
<td>BDI-II</td>
<td>LIKERT</td>
<td>QQ</td>
<td>Drug History</td>
<td>Alcohol History</td>
<td>Drug Use</td>
<td>Intoxication during offence</td>
<td>TBI History</td>
<td>TBI Before First Conviction</td>
</tr>
<tr>
<td>------------------</td>
<td>-----------</td>
<td>------</td>
<td>------</td>
<td>-----</td>
<td>---------</td>
<td>--------------------</td>
<td>--------</td>
<td>--------</td>
<td>----</td>
<td>--------------</td>
<td>------------------</td>
<td>---------</td>
<td>-----------------------------</td>
<td>-------------</td>
<td>-----------------------------</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Māori/non-Māori</td>
<td>28.00</td>
<td>14.00</td>
<td>9.00</td>
<td>34.00</td>
<td>10.00</td>
<td>9.00</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Māori</td>
<td>29.00</td>
<td>12.00</td>
<td>6.00</td>
<td>28.00</td>
<td>7.50</td>
<td>31.00</td>
<td>3</td>
<td>1</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Māori</td>
<td>21.00</td>
<td>14.00</td>
<td>5.00</td>
<td>31.00</td>
<td>8.50</td>
<td>12.00</td>
<td>3</td>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Pakeha</td>
<td>29.00</td>
<td>18.00</td>
<td>12.00</td>
<td>49.00</td>
<td>12.00</td>
<td>19.00</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Pakeha</td>
<td>36.00</td>
<td>19.00</td>
<td>10.00</td>
<td>35.00</td>
<td>11.00</td>
<td>12.00</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Māori</td>
<td>48.00</td>
<td>14.00</td>
<td>8.00</td>
<td>37.00</td>
<td>8.00</td>
<td>26.00</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Pakeha</td>
<td>29.00</td>
<td>18.00</td>
<td>9.00</td>
<td>38.00</td>
<td>10.00</td>
<td>15.00</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Pakeha</td>
<td>24.00</td>
<td>22.00</td>
<td>5.00</td>
<td>26.00</td>
<td>9.00</td>
<td>5.00</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Māori</td>
<td>34.00</td>
<td>22.00</td>
<td>9.00</td>
<td>47.00</td>
<td>10.00</td>
<td>1.00</td>
<td>2</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Māori</td>
<td>18.00</td>
<td>18.00</td>
<td>6.00</td>
<td>27.00</td>
<td>9.00</td>
<td>10</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Pakeha</td>
<td>31.00</td>
<td>20.00</td>
<td>14.00</td>
<td>37.00</td>
<td>9.00</td>
<td>14</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Pakeha</td>
<td>33.00</td>
<td>19.00</td>
<td>10.00</td>
<td>37.00</td>
<td>10.00</td>
<td>8</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Māori</td>
<td>35.00</td>
<td>18.00</td>
<td>8.00</td>
<td>62.00</td>
<td>8.00</td>
<td>14</td>
<td>2</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Participant Type</td>
<td>Ethnicity</td>
<td>Age</td>
<td>BADS</td>
<td>STW</td>
<td>COW A-M</td>
<td>Years of Education</td>
<td>BDI-II</td>
<td>LIKERT</td>
<td>QQ</td>
<td>Drug History</td>
<td>Alcohol History</td>
<td>Drug Use</td>
<td>Intoxication during offence</td>
<td>TBI History</td>
<td>TBI Before First Conviction</td>
</tr>
<tr>
<td>------------------</td>
<td>----------------</td>
<td>------</td>
<td>-------</td>
<td>------</td>
<td>---------</td>
<td>-------------------</td>
<td>--------</td>
<td>--------</td>
<td>----</td>
<td>--------------</td>
<td>-----------------</td>
<td>----------</td>
<td>-----------------------------</td>
<td>------------</td>
<td>---------------------------</td>
</tr>
<tr>
<td>Non-Violent</td>
<td>Māori</td>
<td>32.00</td>
<td>18.00</td>
<td>6.00</td>
<td>37.00</td>
<td>7.50</td>
<td>14</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Community</td>
<td>Māori</td>
<td>18.00</td>
<td>20.00</td>
<td>7.00</td>
<td>21.00</td>
<td>9.50</td>
<td>2.00</td>
<td>1</td>
<td>2</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>No</td>
<td>N/A</td>
</tr>
<tr>
<td>Community</td>
<td>Māori/non-Māori</td>
<td>21.00</td>
<td>19.00</td>
<td>8.00</td>
<td>30.00</td>
<td>9.00</td>
<td>7.00</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>N/A</td>
</tr>
<tr>
<td>Community</td>
<td>Pacific Island</td>
<td>51.00</td>
<td>13.00</td>
<td>3.00</td>
<td>25.00</td>
<td>10.00</td>
<td>20.00</td>
<td>1</td>
<td>1</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>Yes</td>
<td>N/A</td>
</tr>
<tr>
<td>Community</td>
<td>Other</td>
<td>33.00</td>
<td>19.00</td>
<td>7.00</td>
<td>30.00</td>
<td>11.00</td>
<td>11.00</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>Yes</td>
<td>N/A</td>
</tr>
<tr>
<td>Community</td>
<td>Māori/non-Māori</td>
<td>17.10</td>
<td>18.00</td>
<td>7.00</td>
<td>34.00</td>
<td>10.00</td>
<td>23.00</td>
<td>2</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>N/A</td>
<td>Yes</td>
<td>N/A</td>
</tr>
<tr>
<td>Community</td>
<td>Pakeha</td>
<td>43.00</td>
<td>17.00</td>
<td>11.00</td>
<td>45.00</td>
<td>10.00</td>
<td>1.00</td>
<td>2</td>
<td>2</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>Yes</td>
<td>N/A</td>
</tr>
<tr>
<td>Community</td>
<td>Pakeha</td>
<td>49.00</td>
<td>19.00</td>
<td>12.00</td>
<td>52.00</td>
<td>10.00</td>
<td>1.00</td>
<td>2</td>
<td>2</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>Yes</td>
<td>N/A</td>
</tr>
<tr>
<td>Community</td>
<td>Pakeha</td>
<td>53.00</td>
<td>17.00</td>
<td>7.00</td>
<td>45.00</td>
<td>10.00</td>
<td>3.00</td>
<td>1</td>
<td>2</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>Yes</td>
<td>N/A</td>
</tr>
<tr>
<td>Community</td>
<td>Pacific Island</td>
<td>55.00</td>
<td>20.00</td>
<td>10.00</td>
<td>30.00</td>
<td>11.00</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>No</td>
<td>N/A</td>
</tr>
<tr>
<td>Community</td>
<td>Māori</td>
<td>49.00</td>
<td>19.00</td>
<td>8.00</td>
<td>53.00</td>
<td>10.00</td>
<td>3.00</td>
<td>1</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>No</td>
<td>N/A</td>
</tr>
<tr>
<td>Participant Type</td>
<td>Ethnicity</td>
<td>Age</td>
<td>BADS</td>
<td>STW</td>
<td>COW A-M</td>
<td>Years of Education</td>
<td>BDI-II</td>
<td>LIKERT</td>
<td>QQ</td>
<td>Drug History</td>
<td>Alcohol History</td>
<td>Drug Use</td>
<td>Intoxication during offence</td>
<td>TBI History</td>
<td>TBI Before First Conviction</td>
</tr>
<tr>
<td>------------------</td>
<td>----------</td>
<td>-----</td>
<td>------</td>
<td>-----</td>
<td>--------</td>
<td>-------------------</td>
<td>-------</td>
<td>--------</td>
<td>----</td>
<td>-------------</td>
<td>---------------</td>
<td>---------</td>
<td>-----------------------------</td>
<td>-------------</td>
<td>---------------------------</td>
</tr>
<tr>
<td>Community</td>
<td>Pakeha</td>
<td>41.00</td>
<td>19.00</td>
<td>8.00</td>
<td>50.00</td>
<td>11.00</td>
<td>5.00</td>
<td>2</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>Yes</td>
<td>N/A</td>
</tr>
<tr>
<td>Community</td>
<td>Pakeha</td>
<td>51.00</td>
<td>20.00</td>
<td>14.00</td>
<td>38.00</td>
<td>11.00</td>
<td>1.00</td>
<td>1.00</td>
<td>2</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>Yes</td>
<td>N/A</td>
</tr>
<tr>
<td>Community</td>
<td>Māori</td>
<td>42.00</td>
<td>18.00</td>
<td>6.00</td>
<td>40.00</td>
<td>10.00</td>
<td>5.00</td>
<td>1.00</td>
<td>2</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>No</td>
<td>N/A</td>
</tr>
<tr>
<td>Community</td>
<td>Pakeha</td>
<td>24.00</td>
<td>15.00</td>
<td>6.00</td>
<td>37.00</td>
<td>9.00</td>
<td>0</td>
<td>1.00</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>Yes</td>
<td>N/A</td>
</tr>
<tr>
<td>Community</td>
<td>Māori</td>
<td>27.00</td>
<td>20.00</td>
<td>7.00</td>
<td>38.00</td>
<td>9.00</td>
<td>14.00</td>
<td>1.00</td>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>No</td>
<td>N/A</td>
</tr>
<tr>
<td>Community</td>
<td>Māori</td>
<td>24.00</td>
<td>19.00</td>
<td>7.00</td>
<td>36.00</td>
<td>9.00</td>
<td>13.00</td>
<td>1.00</td>
<td>2</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>N/A</td>
<td>No</td>
<td>N/A</td>
</tr>
</tbody>
</table>

1. General Results included in this table are only the ones of interest to the main findings in Study One
2. The BADS profile score ranges from 0 – 24, with lower scores indicating greater difficulties with executive functioning.
3. The STW is a scaled score with a mean of 10, SD = 3, with lower scores representing poorer verbal ability and lower IQ estimate.
4. The COWA-M score is the total number of words scored as correct generated by each participant under time pressure.
5. Years of Education represent the total number of years spent at School.
6. The BDI-II represents the overall score a participant received, with higher ratings representing higher levels of depression symptoms reported.
7. The LIKERT is the score ranging from 1-5 from the Likert Scale of Appropriateness, with lower scores indicating more appropriate test behaviour.
8. The QQ = final 'score' when quantitative and qualitative information from Study One participants were combined; 1 = 'Clear ED Pattern;' 2 = 'Unclear/No ED Pattern.'
9. N/A = non-applicable
Appendix Two

Participant Information Form for Inmate Participants

Registration of Interest Form for Inmate Participants

Participant Information Form for Community Participants

Registration of Interest Form for Community Participants

Consent Form

Advertisement for Community Participants
Executive Dysfunction and Its Possible Contribution to Offending

Participant Information Sheet for Inmates at Auckland Prison and Inmates on Home Detention

You are invited to participate in research being conducted by Sheree Crump, who is a doctorate student at The University of Auckland conducting research for a Doctor of Philosophy degree with the Department of Psychology. Sheree’s thesis explores the relationship between brain function and aggression. Your participation in this study does not necessarily mean you are considered aggressive.

Sheree would appreciate any assistance you can offer her. Part of her research includes testing of prison inmates from different conviction backgrounds. Sheree needs to talk to inmates who have been convicted of violent crimes AND inmates who have been convicted of non-violent crimes. At the time of testing Sheree will not know what crime(s) you have been convicted of.

The tests are quite fun and include such things as planning a trip around a zoo, and getting a cork out of a bottle. The tests are not pass or fail type tests like those from school, so you should feel no pressure to perform in a certain way. Sheree will also need to administer a questionnaire to you and a separate questionnaire to someone else (with your permission) who knows you well within the prison environment (this is someone like a prison officer, case manager, or psychologist). This questionnaire examines the way you do things and has 20 questions. You will also be asked to fill in another short questionnaire, which will assess your mood. The testing session, including an interview, should take no more than two hours, which will include time for breaks from testing. All information you provide through the questionnaires and interview, and all the results from testing are confidential, and will only be available to Sheree and her supervisors at the University of Auckland.

All the testing will take place either on site at Auckland Prison, most likely in a room close to where you stay, or where you are living on Home Detention. The test situation will be made as comfortable as possible. All testing will be during week-days for a period of up to two hours. You may request a break at any time during the testing.
About the study

The aim of the study is to find out if there is a population of people in the prison system who behave in a particular way called ‘executive dysfunction’ due to a certain brain functioning pattern. Research shows that people who have some prefrontal damage to the brain (this is the front part of your brain) may exhibit certain types of behaviour. Damage to this part of the brain is common in people who have been knocked out, as well as for many other reasons. You will be given some tests which provide an idea of the ways you organise, plan, read words, and so on.

The results of the study are first and foremost intended to be used for Sheree’s thesis. The thesis will be a large write up of the results and is not intended for publication (although after the thesis it is hoped useful information from it will be submitted to a scientific journal). It is also hoped the results will help neuropsychologists (psychologists with a special interest in the brain) and prison psychologists in the designing of rehabilitation programs that can be tailored to the individual inmates’ needs. No names or personal information that can be used to identify you will be used in the study. A write up of your individual test results and what they mean are available for you at your request.

Risks and benefits

The only risk or discomfort you may experience in your involvement in this study is that you may feel tired at the end of the neuropsychological tests (remember you can stop at any time for a break). If you think there are any problems resulting from the testing the investigator Sheree Crump is available to talk to you about your concerns.

Taking part in the study or withdrawing from the study will not in anyway affect your eligibility for parole, or your time in Auckland Prison, or your time on Home detention.

There will be no direct benefits to you personally, although we hope you find the tests interesting and maybe even fun. However you will be benefiting research in the area of prison inmate rehabilitation, and the effect of executive dysfunction on behaviour. If you wanted your case manager or psychologist to be given your results to assist them in planning an improved rehabilitation program for you, this would be possible. You will also be helping Sheree in completing the requirements for her degree for which you have her many thanks.

Participation

If you wish to participate please let the researchers know by returning the attached Registration of Interest Form saying you would like to participate in the addressed envelope. If you would like to discuss the project further with Sheree before deciding whether to participate, tick the box indicating this. Once we know you wish to participate we will arrange for you to sign a consent form. Please note that one of the tests that is given in the battery involves the use of a tape recorder for up to 10 minutes. The tapes are wiped within a day of recording. The audio-tape is simply part of that particular test.
Thank you very much for your time and help in making this study possible. If you have any queries or wish to know more please write to the researcher at:

Sheree Crump  
Department of Psychology  
The University of Auckland  
Private Bag 92019  
Auckland.

Her supervisors are:  
Associate Professor Jenni Ogden  
Department of Psychology  
The University of Auckland  
Private Bag 92019  
Auckland

Dr Andrew Moskowitz  
Department of Psychology  
The University of Auckland  
Private Bag 92019  
Auckland

The Head of Department is:  
Professor Dianne McCarthy  
Department of Psychology  
The University of Auckland  
Private Bag 92019  
Auckland
For any queries regarding ethical concerns please contact:

The Chair
The University of Auckland Human Subjects Ethics Committee
The University of Auckland
C/o Research Office
Private Bag 92019
Auckland.

APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN SUBJECTS ETHICS COMMITTEE

On 17/5/00 for a period of 3 years, from 17/5/2000 reference 00/0067....
Registration of Interest Form

Yes I wish to participate in the research being conducted by Sheree Crump. Please contact me to set up a time for testing of executive functions.

I wish to discuss further with the researcher this project…………………………..□
(Tick box if you would like to know more before considering whether to participate)

NAME: ………………………………………
(Print clearly)

CONTACT DETAILS:……………………………………………………………………………………………………

……………………………………………………………………………………………………

……………………………………………………………………………………………………

……………………………………………………………………………………………………

SIGNATURE:……………………………………

DATE:

Send this form in the addressed envelope through internal mail to XXXX, Client Services, Auckland Prison.
Executive Dysfunction and Its Possible Contribution to Offending

Participant Information Sheet for men in community education and work placement courses

You are invited to participate in research being conducted by Sheree Crump, who is a doctorate student at The University of Auckland conducting research for a Doctor of Philosophy degree with the Department of Psychology. Sheree’s thesis explores the relationship between brain function and aggression.

Sheree would appreciate any assistance you can offer her. Part of her research includes testing of men in community education and work placement courses. Sheree invites you to participate in her research if you meet certain criteria:

- your ethnicity is Māori, Pakeha, Pacific Island peoples, or a mix of these
- you understand and can read and write in English
- you did not go beyond 6th form in high school
- you DO NOT have epilepsy
- you DO NOT currently suffer from depression
- you DO NOT currently suffer from mental illness
- you have NEVER been convicted of a crime
- you have been free from violence (have not hurt another, threatened to hurt another, or damaged property) for the last 5 years

The tests are quite fun and include such things as planning a trip around a zoo, and getting a cork out of a bottle. The tests are not pass or fail type tests like those from school, so you should feel no pressure to perform in a certain way. Sheree will also need to administer a questionnaire to you and a separate questionnaire to someone else (with your permission) who knows you well within your day to day environment; this could be a friend or family member. This questionnaire examines the way you do things and has 20 questions. You will also be required to fill out another short questionnaire that will assess your mood. The testing session, including an interview, should take no more than two hours, which will include time for breaks from testing. All information you provide through the
questionnaires and interview, and all the results from testing are confidential, and will only be available to Sheree and her supervisors at the University of Auckland.

All the testing will take place either on site at your course, your home, or in an interview room at the University of Auckland. The test situation will be made as comfortable as possible. All testing will be during weekdays for a period of up to two hours. You may request a break at any time during the testing.

**About the study**

The aim of the study is to find out if there is a population of people in the prison system who behave in a particular way called ‘executive dysfunction’ due to a certain brain functioning pattern. The people in prison have already been tested men in the community are serving as a comparison group. Research shows that people who have some prefrontal damage to the brain (this is the front part of your brain) may exhibit certain types of behaviour. Damage to this part of the brain is common in people who have been knocked out, as well as for many other reasons. You will be given some tests which provide an idea of the ways you organise, plan, read words, and so on.

The results of the study are first and foremost intended to be used for Sheree’s thesis. The thesis will be a large write up of the results and is not intended for publication (although after the thesis it is hoped useful information from it will be submitted to a scientific journal). It is also hoped the results will help neuropsychologists (psychologists with a special interest in the brain) and prison psychologists in the designing of rehabilitation programs that can be tailored to the individual inmates’ needs. No names or personal information that can be used to identify you will be used in the study. A write up of your individual test results and what they mean are available for you at your request.

**Risks and benefits**

The only risk or discomfort you may experience in your involvement in this study is that you may feel tired at the end of the neuropsychological tests (remember you can stop at any time for a break). If you think there are any problems resulting from the testing the investigator Sheree Crump is available to talk to you about your concerns.

Taking part in the study or withdrawing from the study will not in anyway affect your community course or any benefits you may be receiving from WINZ.

There will be no direct benefits to you personally, although we hope you find the tests interesting and maybe even fun. However, you will be benefiting research in the area of prison inmate rehabilitation, which we hope will benefit society as a whole through reducing re-offending. You will also be contributing to the literature on the effect of executive dysfunction on behaviour. You will also be helping Sheree in completing the requirements for her degree for which you have her many thanks.
Participation

If you do wish to participate please let the researchers know by returning the attached Registration of Interest Form saying you would like to participate in the addressed envelope. If you would like to discuss the project further with Sheree before deciding whether to participate, tick the box indicating this. Once we know you wish to participate, we will arrange for you to sign a consent form. Please note that one of the tests involves the use of a tape recorder for up to 10 minutes. The tapes are wiped within a day of recording. The audiotape is simply part of that particular test, none of your interview will be taped.

Thank you very much for your time and help in making this study possible. If you have any queries or wish to know more please write to the researcher at:

Sheree Crump  
Department of Psychology  
The University of Auckland  
Private Bag 92019  
Auckland.

Her supervisors are:  
Associate Professor Jenni Ogden  
Department of Psychology  
The University of Auckland  
Private Bag 92019  
Auckland.

Dr Andrew Moskowitz  
Department of Psychology  
The University of Auckland  
Private Bag 92019  
Auckland

The Head of Department is: Professor Diane McCarthy  
Department of Psychology  
The University of Auckland  
Private Bag 92019  
Auckland.
For any queries regarding ethical concerns please contact:

The Chair
The University of Auckland Human Subjects Ethics Committee
The University of Auckland
C/o Research Office
Private Bag 92019
Auckland.
Ph 373-7599 ext.: 7830

APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN SUBJECTS ETHICS COMMITTEE

On .17/5/2000. for a period of …3… years, from .17./5./00… reference .00../..067.
Registration of Interest Form

Yes I wish to participate in the research being conducted by Sheree Crump. Please contact me to set up a time for testing of executive functions.

I wish to discuss further with the researcher this project.............................□
(Tick box if you would like to know more before considering whether to participate)

NAME: ....................................................
(Print clearly)

CONTACT DETAILS:.........................................................................................
...................................................................................................................................
...................................................................................................................................
...................................................................................................................................

SIGNATURE:....................................................

DATE:
CONSENT FORM:
THIS CONSENT FORM WILL BE HELD FOR A PERIOD OF SIX YEARS

Consent form for participants involved in the testing of executive functions

Title of Project: Executive dysfunction and the possible contributions this syndrome makes to violence expression, including indications for rehabilitation of convicted offenders.

Researcher: Principal researcher Sheree Crump, supervised by Associate Professor Jenni Ogden and Dr Andrew Moskowitz.

I have been given and have understood the explanation of this research project. I understand that I will be asked to perform a series of neuropsychological tests, including one where there may be an audio-tape recording for up to 10 minutes, and fill in two questionnaires. I understand that someone who knows me well will be asked to fill in a questionnaire about my behaviour. I have had an opportunity to ask questions and have them answered. I understand that I may withdraw myself, or any information I have given, at any time up until I have participated in the study without giving a reason.

I agree to take part in the research
I agree/do not agree that during one of the tests an audio-tape will be used (circle one) note: you can still take part in the research if you do not agree to the audio-taping

Signed:

Name: (Please print clearly)

Date:

APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN SUBJECTS ETHICS COMMITTEE On .17/5/00. for a period of .3. years, from .17./.5./.2000.. reference .00../.067.....
Would you like to be part of a research project that that is examining why some people commit violent crimes?

We would like to talk to people that **ARE NOT** violent to compare against people we have already tested who are in prison for violent crimes.

We would be interested in talking to you if you meet certain criteria:

- **Male**
- **Age 20-40 years old**
- **English is your first language**
- **Do not have any tertiary education (finished school at high school or earlier)**
- **Are free from criminal convictions (minor traffic offences are ok)**
- **Are free from any current mental disorder including severe depression**
- **Are free from epilepsy**

If this does not sound like you but someone you know and you think they would be interested please let them know.

If you are interested, contact the person below to find out more. Your time commitment will be no longer than 2-hours in which you will be asked some questions and be given some fun tests (*definitely* not school type ones!) to do. This will provide information about certain behaviours and strengths you have. As a thank-you, you will receive a full report outlining your abilities, and if there are any weaker areas what you can do about them will be included in the report.

This research is being conducted by the Psychology Department at the University of Auckland and has ethics approval.

Contact Sheree Crump on (09) 373-7599 xt: 8523 or email sa.crump@auckland.ac.nz. You can also write to Sheree Crump, Dept Psychology, The University of Auckland, Private Bag 92019, Auckland.

**APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN SUBJECTS ETHICS COMMITTEE On ..17/5/00.. for a period of .3. years, from .17./5./2000.. reference ..00../..067.....**
Appendix Three

Proposed Intervention for Inmates with ED

Behaviour Log (Exercise)

Behaviour Log (Incident)

Behaviour Log (Meals)

Hassle Log

Structured Interview

Barrett Impulsivity Interview - II
Proposed Intervention for Inmates with ED

Session 1

Powhiri and introductions.

Explanation of what the group is, what it means to me, and what it means to the men.

There will be a getting to know each other and settling in time.

Establish and agree and sign to group rules.

Discussions around what the men hope to get out of it, their commitment and motivation.

The beginnings of psycho-education around executive dysfunction (ED). This will use materials such as pictures of the brain and lists of behaviours, perhaps a video. The men will come to understand how people can develop this condition and what it means to live with it. This psycho-education aspect of the group will run over a few sessions, along side other exercises. Errorless learning will be used throughout, rather than ask men to ‘guess’ at answers when learning new material. Also in all the sections visual aids will be used (i.e., written examples of behaviours seen in ED and strategies used to compensate for them), and each man will be given a copy of these to add to his ‘Kia Kaha’ folder.

Feedback. A discussion of what has been covered today and what each man has learnt. As an errorless learning approach is being used there is no setting of ‘homework.’
Session 2

Review of last session and repetition of areas that appear not to be remembered, continue psycho-education but add more input from the men of their own experiences, including discussion of the different ways they have learned to handle it. Use this to start identifying triggers/cues that each individual needs to be aware of. Strengths may also start appearing in this session, build on these.

Introduce the ‘Hassle Log’ and train in its use.

Create goals for the week that are kept in their Kia Kaha folders. (An example of a goal in this case may be participants noting when they think they are aware of a trigger or cue they learnt about in the session).

Session 3

Check how the week has gone. Revision of anything they noticed was different, or if goals had been met. This to happen first as a group and then individually later when the men are engaged in tasks so there is opportunity for further positive feedback or assistance if needed.

Review of any Hassle Logs that were filled in by the participants. This is done individually and privately with each man in the group session to maintain privacy about what they have reported.

Review of the two previous sessions and repetition of areas that appear not to be remembered; continue with explanations of ED from both the literature (in a simplified form) and from the men themselves.

The last two sessions would have provided some information on the pace of the group and what needs to be presented next. The men themselves would have provided some ideas of what they believe would be useful. It is likely to be in the
area of understanding weakness and building on strengths. Sessions will be designed for the future with input from the men.

Set new goals and gather expectations of the next session. In this case goals may include being aware of any behaviour of their own (or others) that they recognise as a result of ED.

Session 4

Review of last session and repetition of areas that appear not to be remembered.

Review of any Hassle Logs that were filled in. This is done individually and privately with each man in the group session to maintain privacy about what they have reported.

Go over what cues and triggers (both internal and external) the men have identified as ‘setting them off,’ or making them feel as if they are losing control.

Go over what losing control feels like for the men. Get the participants to identify cognitive, physical, and emotional sensations. Make lists on what are the easiest to identify.

Discuss what each of the men currently uses for coping. Define coping for participants and the consequences of coping and not coping.

Review and role play; cues + triggers

Create some goals for the week that are written in the Kia Kaha folders.
Session 5

Check how the week has gone.

Review of any Hassle Logs that were filled in. This is done individually and privately with each man in the group session to maintain privacy about what they have reported.

Review of last session and repetition of areas that appear not to be remembered.

Develop lists of activities that enhance coping and curb impulsivity. These will centre on activities that make the participants relaxed/happy, and how to target sensations that lead to feeling out of control or impulsive.

Review and role-play; cues + triggers + impulse reducers.

Create some goals for the week that are written in the Kia Kaha folders.

Session 6

Review of any Hassle Logs that were filled in. This is done individually and privately with each man in the group session to maintain privacy about what they have reported.

Review of last session and repetition of areas that appear not to be remembered.

Discuss times “when I feel I am impulsive” and understanding the triggers both internal and external. Link this with how they usually cope and what may be more positive ways to cope (this comes from the last session).

Review and role play; cues + triggers + impulse reducers and strength of impulse control
Create some goals for the week that are written in the Kia Kaha folders.

**Session 7**

Check how the week has gone.

Review of any Hassle Logs that were filled in. This is done individually and privately with each man in the group session to maintain privacy about what they have reported.

Review of last session and repetition of areas that appear not to be remembered.

Introduce reminders of how to be aware of triggers and cues. Make a list of what participants can use as reminders. Model using reminders.

Review and role-play; cues + triggers + reminders + impulse reducers and strength of control.

Create some goals for the week that are written in the Kia Kaha folders.

**Session 8**

Review of last session and repetition of areas that appear not to be remembered.

Review of any Hassle Logs that were filled in. This is done individually and privately with each man in the group session to maintain privacy about what they have reported.

A review of progress to date, discuss half way point in the intervention, what needs are being/not being met.
Introduce self-evaluation and self-rewarding. Model how this is done. Role-play some scenarios.

Review and role play; cues + triggers + reminders + impulse reducers + self-evaluation and strength of impulse control
Create some goals for the week that are written in the Kia Kaha folders.

**Session 9**

Check how the week has gone.

Review of any Hassle Logs that were filled in. This is done individually and privately with each man in the group session to maintain privacy about what they have reported.

Review of last session and repetition of areas that appear not to be remembered.

Continue in training in how to self-evaluate and be aware of triggers and cues.

Make list of self-rewards and how to deliver them.

Review and role-play; cues + triggers + reminders + impulse reducers + self-evaluation + self-rewards and strength of impulse control.

Create some goals for the week that are written in the Kia Kaha folders.
Session 10

Review of last session and repetition of areas that appear not to be remembered.

Review of any Hassle Logs that were filled in. This is done individually and privately with each man in the group session to maintain privacy about what they have reported.

Introduce ‘thinking ahead.’ Discuss and list short and long-term consequences, most and least serious consequences, internal, external, and social consequences.


Review and role-play; cues + triggers + reminders + impulse reducers + self-evaluation + self-rewards and strength of impulse control.

Review thinking ahead.

Create some goals for the week that are written in the Kia Kaha folders.

Session 11

Check how the week as gone

Review of any Hassle Logs that were filled in. This is done individually and privately with each man in the group session to maintain privacy about what they have reported.

Review of last session and repetition of areas that appear not to be remembered.

Continue to discuss the importance of thinking ahead and being aware of future consequences. Use events the men raise themselves from the past or recently.
Discuss times participants have clearly used a thinking ahead strategy that resulted in a positive outcome. Discuss times participants have used a thinking ahead strategy that resulted in a negative outcome. Explore what was different in the situations that provided the different outcomes.

Review and role-play; thinking ahead + cues + triggers + reminders + impulse reducers + self-evaluation + self-rewards and strength of impulse control.

Create some goals for the week that are written in the Kia Kaha folders.

**Session 12**

Review of last session and repetition of areas that appear not to be remembered.

Review of any Hassle Logs that were filled in. This is done individually and privately with each man in the group session to maintain privacy about what they have reported.

Introduce the ‘Impulsive Behaviour Cycle.’ Model this and how it can make me lose control and how to use what we have learnt so far to maintain control of my impulses.

Discuss how to break the cycle.

Review and role-play; thinking ahead + cues + triggers + reminders + impulse reducers + self-evaluation + self-rewards and strength of impulse control.

Review ‘Impulsive Behaviour Cycle.’

Create some goals for the week that are written in the Kia Kaha folders.
Session 13

Check how the week has gone.

Review of any Hassle Logs that were filled in. This is done individually and privately with each man in the group session to maintain privacy about what they have reported.

Review of last session and repetition of areas that appear not to be remembered.

Discuss curbing impulsivity with the skills learnt thus far. See where this fits into the Impulsive Behaviour Cycle. Continue to practice the use of the new skills to reduce impulsivity.

Practice how to plan ahead when aware the environment is about to be filled with cues. Discuss when is it best to avoid places the participant knows is fill of cues. Model and role-play ways to do this.

Review and role-play; thinking ahead + cues + triggers + reminders + impulse reducers + self-evaluation + self-rewards and strength of impulse control.
Create some goals for the week that are written in the Kia Kaha folders..

Session 14

Review of last session and repetition of areas that appear not to be remembered.
Review of any Hassle Logs that were filled in. This is done individually and privately with each man in the group session to maintain privacy about what they have reported.

Review of the entire intervention and what has been learnt so far and role-play; thinking ahead + cues + triggers + reminders + impulse reducers + self-evaluation + self-rewards and strength of impulse control.
Introduce ways in which to generalise the skills learnt in enhancing thinking ahead, planning ability and impulsivity curbing to other behaviours the participants may be having trouble with.

Introduce that soon the intervention will be over, put processes for ending therapy in place for this.

Create some goals for the week that are written in the Kia Kaha folders.

**Session 15**

Check how the week has gone.

Review of any Hassle Logs that were filled in. This is done individually and privately with each man in the group session to maintain privacy about what they have reported.

Review of last session and repetition of areas that appear not to be remembered.

Review the entire course. Discuss with the inmates what they are unsure of and would like to spend a little more time understanding. Get feedback on what they do and do not understand.

Men to make independent lists of ‘IF – THEN’ scenarios with and without thinking ahead plans. This is then repeated in a group format.

Review and role-play; thinking ahead + cues + triggers + reminders + impulse reducers + self-evaluation + self-rewards and strength of impulse control.

Remind the participants that this is the last week of the intervention and the next session will be the last session. Continue winding-down process.
Create some goals for the week that are written in the Kia Kaha folders.

**Session 16**

Review of last session and repetition of areas that appear not to be remembered.

Review of any Hassle Logs that were filled in. This is done individually and privately with each man in the group session to maintain privacy about what they have reported.

Reinforce performance of the men and congratulate them for coming through the other end. Reinforce what they have learnt and encourage generalisation to other behaviours.

Recap impulse control techniques.

Questions and feedback time.

End the intervention in a manner that suits the style of the participants. Discuss what happens next.
Behaviour Log Exercise

Name_________________ Date_________________ Place__________

Time_________

Was the inmate? (tick as many that apply)

Exercising in the yard/gym          Exercising in his cell          Talking to another inmate

Talking to a prison officer      Alone      Other (write down the circumstances)

During the exercise was there anything notable about his behaviour? (e.g. did he display any rudeness, aggressiveness, or impulsiveness, or any other executive behaviour you are familiar with. If unsure note down what you witness making sure to be as explicit as possible)

______________________________________________________________________
______________________________________________________________________
______________________________________________________________________

If there was a display of inappropriate behaviour during Exercising did the inmate attempt to curb it alone? (e.g., what did the inmate do?, what did he say? Did he make attempts to reduce inappropriate behaviour by withdrawing or changing the subject? Did staff have to get involved?)

______________________________________________________________________
______________________________________________________________________
______________________________________________________________________

What happened after the exercise? (e.g., what did the inmate do?, what did he say?, where did he go? If there was inappropriate behaviour did you observe any further attempts to inhibit it or make reparation?)

______________________________________________________________________
______________________________________________________________________
______________________________________________________________________
**Behaviour Log Incident report** (use this when you witness the inmate involved in a situation where they appeared aggressive, rude, impulsive, or any other executive behaviour you are aware of. If unsure note it down anyway, being explicit regarding the behaviours you witness and the events surrounding it)

Name_________________ Date_________________ Place__________

Time_________

Was the inmate?

Eating a meal  [] in the yard/gym  [] in his cell  [] in the corridor  []

Talking to another inmate  [] Talking to a prison officer  [] Alone  []

Other  [] (write down the circumstances) ________________________________

What happened before the incident? (e.g. what was the situation like, the atmosphere, who was talking to who, what was going on, what was the inmate’s mood/behaviour like?)

____________________________________________________________________
____________________________________________________________________
____________________________________________________________________

What was the incident? Who else was involved? (e.g. what did the inmate do?, what did he say? Did he make attempts to reduce inappropriate behaviour)?

____________________________________________________________________
____________________________________________________________________
____________________________________________________________________

What happened after the incident? (e.g. what did the inmate do?, what did he say when it was over? Did you observe attempts to inhibit any inappropriate behaviour? Where did he go after the incident?)

____________________________________________________________________
____________________________________________________________________
____________________________________________________________________
Further comments
Behaviour Log Meals

Name_________________ Date_________________ Place__________

Time_______

Was the inmate? (tick as many that apply)

Eating a meal in the dining room  ☐  Eating a meal in the yard/gym  ☐  Eating a meal in his cell  ☐  Talking to another inmate  ☐  Talking to a prison officer  ☐

Alone  ☐

Other ☐ (write down the circumstances) _________________________________

During the meal was there anything notable about his behaviour? (e.g. did he display any rudeness, aggressiveness, or impulsiveness, or any other executive behaviour you are familiar with. If unsure note down what you witness making sure to be as explicit as possible)

______________________________________________________________________

______________________________________________________________________

______________________________________________________________________

If there was a display of inappropriate behaviour during mealtime did the inmate attempt to curb it alone? (e.g., what did the inmate do?, what did he say? Did he make attempts to reduce inappropriate behaviour by withdrawing or changing the subject? Did staff have to get involved?)

______________________________________________________________________

______________________________________________________________________

______________________________________________________________________

What happened after mealtime? (e.g., what did the inmate do?, what did he say?, where did he go? If there was inappropriate behaviour did you observe any further attempts to inhibit it or make reparation?)

______________________________________________________________________

______________________________________________________________________

______________________________________________________________________
Hassle Log

Name__________________________ Date____________________
morning_________ afternoon_________ night_________
(record time)

Where were you?
dining room_________ cell_________ recreation_________
work detail_________ corridor_________ other___________

What happened? (tick)
somebody told me to do something  somebody “started” with me
somebody said something I didn’t like  I did something wrong
somebody was doing something I didn’t like  other  (write what
happened)

Who was that somebody? (tick)
another inmate  staff  family  other  (write who it was)

What did you do? (tick)
had a verbal argument  ignored it  walked away
hit them  talked it over  told someone
broke something  used impulse control  felt really sad
threw something □ was contained by staff □ other □  
(write what you did)

______________________________________________________________________  
______________________________________________________________________  
______________________________________________________________________  
______________________________________________________________________  

How did you handle yourself? (tick)  
poorly □ not so well □ OK □ good □ great □

How angry were you? (tick)  
burning □ really angry □ moderate angry □ mild angry, but ok □ not angry at all □

How in control did you feel? (tick)  
totally out of control, and needed help to get it back □ quite out of control □  
a little out of control but I could contain it □ not out of control at all □

How aware were you about your feelings leading to your actions? (tick)  
not aware of feelings at all □ a little aware, but unable to do anything □  
mostly aware but did not act on awareness □ mostly aware and attempted to act on awareness □ totally aware but did not act on awareness □ totally aware □

Any further comments on the situation, your actions, and feelings and thoughts
______________________________________________________________________
______________________________________________________________________
______________________________________________________________________
______________________________________________________________________
______________________________________________________________________
Impulsivity Open-Ended Questions (Structured Interview)

If you were driving down the motorway and a car pulled in-front of you in the fast lane without indicating, and then slowed right down to 80kms an hour (you were doing 100kms) what would you do? Is this the same or different from what you think you should do?

If you were going on a weeklong trip outside of the city you lived in, how long would you spend planning the trip? What kind of things would you consider regarding the trip? Is this the same or different from what you think you should do?

If you won $10,000 with an instant kiwi scratch card what would you do with it? Is this the same or different from what you think you should do?

How do you plan for the future? What kind of things do you take into consideration?

How easy do you find it to concentrate on just one thing?

If you were having a few drinks at the pub and you ran out of money and a mate offered to lend you some, but you already owed him and some other mates heaps, would you take the money? Why/why not?

If you were going for a job interview the next day what type of things would you be thinking about? Is this the same or different from what you think you should do?

Do you find it easier to just do things without thinking about them first – “on the spur of the moment” – or do you spend longer making decisions. Can you give me some examples of what information you use to make up your mind?

Do you think you should just do stuff without thinking about it, or have a think about it first?
Personal Evaluation: BIS-11

Name: ___________________________ Date: ___________________________

**Directions:** People differ in the ways they act and think in different situations. This is a test to measure some of the ways in which you act and think. Read each statement carefully and **DARKEN THE APPROPRIATE CIRCLE** to the right of the statement. **Answer quickly and honestly.**

<table>
<thead>
<tr>
<th></th>
<th>Rarely/Never</th>
<th>Occasionally</th>
<th>Often</th>
<th>Almost Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>I plan tasks carefully</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>I do things without thinking</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>I make up my mind quickly</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>I am happy-go-lucky</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>I don’t “pay attention”</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>I have “racing” thoughts</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>I plan trips well ahead of time</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>I am self-controlled</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>I concentrate easily</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10.</td>
<td>I save regularly</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11.</td>
<td>“squirm” at plays or lectures</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12.</td>
<td>I am a careful thinker</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13.</td>
<td>I plan for job security</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14.</td>
<td>I say things without thinking</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15.</td>
<td>I like to think about complex problems</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16.</td>
<td>I change jobs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17.</td>
<td>I act “on impulse”</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18.</td>
<td>I get easily bored when solving thought problems</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19.</td>
<td>I act on the spur of the moment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20.</td>
<td>I am a steady thinker</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>21.</td>
<td>I change where I live</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>22.</td>
<td>I buy things on impulse</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>23.</td>
<td>I can only think about one problem at a time</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24.</td>
<td>I change hobbies</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25.</td>
<td>I spend or charge more than I earn</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>26.</td>
<td>I have outside thoughts when thinking</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>27.</td>
<td>I am more interested in the present than the future</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>28.</td>
<td>I am restless at lectures or talks</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>29.</td>
<td>I like puzzles</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30.</td>
<td>I plan for the future</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Copyrighted by E.S. Barratt and J. Patton