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A Neuroimaging Study of Impulsivity in Risky and Safe Drivers

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1.1 Declaration

I declare that this thesis has not been submitted as an exercise for a degree at this or any other university and is entirely my own work.

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1.2 Acknowledgements

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1.3 Summary

Males and individuals under the age of 25 are injured or killed while driving more than other demographic groups. While this overrepresentation is said to be the result of a greater number of aberrant driving behaviours in these groups, it is also noted that not all younger individuals and males engage in risk-taking while driving. Research has demonstrated greater levels of both trait and behavioural impulsiveness in drivers with a history of aberrant driving tendencies, suggesting that heightened impulsivity may be an individual difference factor that contributes to risky driving. The current study aimed to replicate and expand on these findings by including a neuroimaging component.

Chapter one puts the research into context by presenting official statistics of collision involvement by age and gender, as well as a review of the factors that are thought to contribute to such collisions. This chapter also explores the literature related to risk-taking and impulsivity, with a particular mention of the relationship between heightened impulsivity and aberrant driving behaviours.

Chapter two reviews the results of an online survey that was employed to recruit participants for the main study. Demographic information was obtained from survey respondents to identify participants who met the particular age and gender requirements to make up the experimental groups of the main study. These participants were also screened for magnetic resonance imaging (MRI) eligibility and assigned into 'risky' and 'safe' driver groups based on their self-reported driving history. Chapter two validated the criteria used to differentiate between risky and safe drivers by comparing the amount of aberrant driving behaviours these groups reported on the Manchester Driver Behaviour Questionnaire (DBQ).

Eligible participants were recruited to take part in a study that assessed impulsivity using psychometric and behavioural measures, in addition to an investigation of the functional and structural neural correlates of impulsivity using functional magnetic resonance imaging (fMRI) and MRI. Chapter three outlines the overall method and procedure of this study, while chapters four to six separate out a discussion of the results of this experiment by methodology used; psychometric and behavioural measurements of impulsivity (chapter four), fMRI (chapter five) and MRI (chapter six). Each of these subsequent chapters outline the literature pertaining to how these measures relate to the concept of impulsiveness, gender differences on these measures, changes in these measures with age and how they may relate to risk-taking while driving. They will also present and discuss the results of the individual analyses.

Finally, chapter seven summarises these findings under three main hypotheses; that impulsivity declines with age, there are gender differences in impulsiveness and impulsivity contributes to aberrant driving, relating each back to the relevant literature and theories mentioned in earlier chapters. As the results of the study provide preliminary evidence that differences in psychometric or behavioural impulsivity between drivers may relate to underlying differences in the structure and/or function of the brain, recommendations are made in this chapter for how future research may study the longitudinal efficacy of cognitive training programs for reducing the impact of impulsivity on aberrant driving.

1.4 List of Abbreviations

Anatomical terms:	
ACC	Anterior cingulate cortex
CC	Corpus callosum
CSF	Cerebral spinal fluid
DLPFC	Dorsolateral prefrontal cortex
GM	Grey matter
IFG	Inferior frontal gyrus
ILF	Inferior longitudinal fasciculus
MFG	Middle frontal gyrus
NAcc	Nucleus accumbens
OFC	Orbitofrontal cortex
PCC	Posterior cingulate cortex
PFC	Prefrontal cortex
rCBF	Regional cerebral blood flow
SFG	Superior frontal gyrus
SMA	Supplementary motor area
VLPFC	Ventrolateral prefrontal cortex
VMPFC	Ventromedial prefrontal cortex
VTA	Ventral Tegmental Area
WM	White matter

Other:	
AAL	Automatic Anatomical Labelling atlas
ADHD	Attention Deficit Hyperactive Disorder
AMIS	Adaptive and Maladaptive Impulsivity Scale
ANCOVA	Factorial Analyses of Covariance
ANOVA	Analysis of Variance
AR	Autoregressive
BAC	Blood alcohol limit
BART	Balloon Analogue Risk Task
BIS-11	Barratt's Impulsiveness Scale
BOLD	Blood oxygen level dependent
CMV	Common method variance
DARTEL	Diffeomorphic Anatomic Registration Through Exponentiated Lie algebra algorithm
DBQ	Driver Behaviour Questionnaire
DD	Delay discounting
DSM	Diagnostic Statistical Manual
DTI	Diffusion tensor imaging
ECMT	European Conference of Ministers of Transport
EEG	Electroencephalogram
EFA	Exploratory Factor Analysis
EFC	Executive Function Capacity scale
EHI	Edinburgh Handedness Inventory
FA	Fractional anisotropy

fMRI	Functional magnetic resonance imaging
FSL	FMRIB Software Library
FWE	Family Wise Error
FWHM	Full width half maximum
GDL	Graduated Driver Licence
GLM	General Linear Model
GP	General Practitioner
HC	Highway-code
HDR	Hemodynamic response
HPF	High pass filter
HRF	Hemodynamic response function
ICA	Independent Components Analysis
IGT	Iowa Gambling Task
Kirby's DDQ	Kirby's Delay Discounting Questionnaire
KMO	Kaiser-Meyer-Olkin
LD	Loss deck
LDR	Larger delayed reward
MANOVA	Multivariate Analysis of Variance
MarsBaR	MARSeille Boîte À Région d'Intérêt
MDD	Major depressive disorder
MFFT	Matching Familiar Faces test
MID	Monetary Incentive Delay task
MNI	Montreal Neurological Institute
MR	Magnetic resonance

MRI	Magnetic resonance imaging
NEO-PI-R	Revised NEO Personality Inventory
NPHS	National Population Health Survey
OCD	Obsessive-compulsive disorder
ROI	Region of interest
RPIS	Resistance to Peer Influence Scale
RSA	Road Safety Authority
SD	Standard deviation
SENSE	Sensitivity encoding
SES	Socioeconomic status
SIR	Smaller immediate reward
SPM	Statistical Parametric Mapping
SPSI-R	Social Problem Solving Inventory– Revised
SPSS	Statistical Package for the Social Sciences
SSRT	Stop Signal Reaction Time task
SVC	Small volume correction
TCD	Trinity College Dublin
TCI-R	Temperament and Character Inventory, revised
TCIN	Trinity College Institute of Neuroscience
tDCS	Transcranial direct current stimulation
TPMs	Tissue probability maps
TR	Time resolution parameter
TRAID	Traffic Accident Information Database
UCD	University College Dublin

UL	University of Limerick
UPPS	Urgency Premeditation Perseverance and Sensation seeking
VBM	Voxel-based morphometry
VGA	Video graphics adapter
WFU	Wake Forest University
WHO	World Health Organisation
ZKA-PQ	Zuckerman-Kuhlman-Aluja Personality Questionnaire
ZKPQ	Zuckerman–Kuhlman Personality Questionnaire
ZSSS	Zuckerman's Sensation Seeking Scale

1.5 List of Presentations

The following are presentations arising from this work:

O'Callaghan, G., Kelly, A. M. C. & Gormley, M. *An fMRI study of impulsivity in 'risky' and 'safe' male drivers.* Oral presentation at the Annual meeting for the Society for Neuroscience, McCormick Place, Chicago, US; October 2015

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1 Chapter One: General Introduction

Adolescence is marked by heightened impulsivity and risk-taking behaviours (Blakemore & Robbins, 2012), such as experimentation with drugs or alcohol, smoking, unprotected sex and dangerous driving (Johnson, McCaul, & Klein, 2002). Decisions made during this period can have serious consequences, such as illness, injury, or death (Viner et al., 2012). Traffic collisions are the leading cause of death in people between the age of 15 and 24 globally, with an average of one thousand fatalities each day, approximately 400,000 annually (WHO, 2012a). In addition to inexperience, deliberate risk-taking is thought to contribute to these figures (Clarke, Ward, & Truman, 2005).

This may be due to the on-going maturation of regions responsible for exerting control over impulses and reward seeking behaviours (Casey, Getz, & Galvan, 2008; Casey et al., 1997; Giedd et al., 1999; Glendon, 2006, 2011; Gogtay et al., 2004; Shaw et al., 2008). However, while increased risk-taking is cited as the 'typical' teenage experience, a limited number of studies acknowledge that many younger individuals do not take risks (Dryfoos, 1991; Samanez-Larkin, Kuhnen, Yoo, & Knutson, 2010; Sercombe, 2014). Theory posits that individuals who engage in one type of problem behaviour are more likely to engage in others, such as risky driving (Bingham & Shope, 2004a, 2004b; Jessor, 1987; Møller & Sigurdardóttir, 2009). A slower decline in psychometric impulsivity from adolescence to adulthood in some individuals has been shown in longitudinal data to be a marker for increased use of alcohol and drugs (Littlefield, Sher, & Steinley, 2010; Quinn & Harden, 2013). Moreover, heightened impulsivity is considered by some to be an individual difference factor that may predispose

some younger drivers to take risks (Brandau, Daghofer, Hofmann, & Spitzer, 2011; Loo, 1979; O'Brien & Gormley, 2013; M. S. Stanford, Greve, Boudreaux, Mathias, & Brumbelow, 1996; Wickens, Toplak, & Wiesenthal, 2008).

The current chapter will explore the literature related to risk-taking and impulsivity in younger individuals, with a focus on aberrant driving behaviours. Official collision statistics and a review of the factors that contribute to such collisions will be used to put this study into context. Subsequent chapters will investigate the relationship between a history of risky driving and impulsivity, as measured psychometrically and behaviourally. Furthermore, functional magnetic resonance imaging (fMRI) will be used to compare the neural correlates of impulsivity between groups and magnetic resonance imaging (MRI) to probe structural differences. The present introduction will, therefore, also include an examination of the research into the neural correlates of driving in order to facilitate this thesis to draw together and relate its findings to the neural mechanisms of driving.

1.1 Prevalence of Road Fatalities

The Road Safety Authority (RSA) is an official statutory body in Ireland, charged with improving road safety, reducing the number of deaths and injuries on Irish roads (RSA, 2015a). The RSA also produces annual reports on Irish collisions. The most recent 'Road Collision Fact-book' was released in 2013 and details collisions from 2012. Brief summary reports for the years 2013 and 2014 have been produced, however, these do not give detailed age and gender breakdowns of the distribution of collisions. Therefore, this section will focus on the statistics made available by the 2013 report of the collisions in Ireland in 2012.

In 2012 Ireland ranked twenty third in Europe for the number of collisions per million population (RSA, 2013). In that year, 26,171 traffic collisions were recorded in Ireland. These collisions resulted in 162 fatalities (64 car driver fatalities), 474 serious injuries and 7,468 minor injuries. Eighty six per cent of these fatalities were attributed to driver error, pedestrians were found to be at fault in 11% of cases and poor road and/or environmental factors only contributed to 3% of fatal collisions (RSA, 2013). Males and young drivers (under 25) are overrepresented in fatal collision statistics (WHO, 2015), thus, inexperience, age and gender are said to relate to the risk of being involved in a fatal collision while driving (OECD & ECMT, 2006). Males are involved in more fatal collisions per kilometre driven (OECD & ECMT, 2006), making up approximately 77% of fatal collisions globally (WHO, 2015). In Ireland, males represented 65% of all driver fatalities in 2011, 56% in 2012, 80% in 2013 and 70% in 2014 (RSA, 2012b, 2013, 2014, 2015c).

Also, despite comprising approximately 10% of the driving population (both in Ireland and globally), young drivers make up 27% of fatal collisions internationally (WHO, 2012b) and represented 30.8% of fatalities in Ireland in 2011, 32.8% in 2012 and 26% in both 2013 and 2014. This is compared to, for example, 35 to 44 year olds in Ireland, who made up 7.15% of driver fatalities in 2011, 9.4% in 2012, 9% in 2013 and 13% in 2014 (RSA, 2012b, 2013, 2014, 2015c). Driving a car is a complex behaviour, which involves the combination of actions such as steering, observation, changing gears, braking, navigating, appreciation of other road users and reacting appropriately to situations. Over time these behaviours become automated but learner and novice drivers find it

difficult to combine them in an efficient manor (OECD & ECMT, 2006). While the majority of leaner and novice drivers are also younger, age and inexperience have been found to be separate risk factors for collision involvement. For example, when countries are compared, fewer fatal collisions occur in novice drivers in countries with higher licencing ages (OECD & ECMT, 2006). Figure 1.1 illustrates the number of fatal collisions in Ireland, separated by age category, over a ten year period.

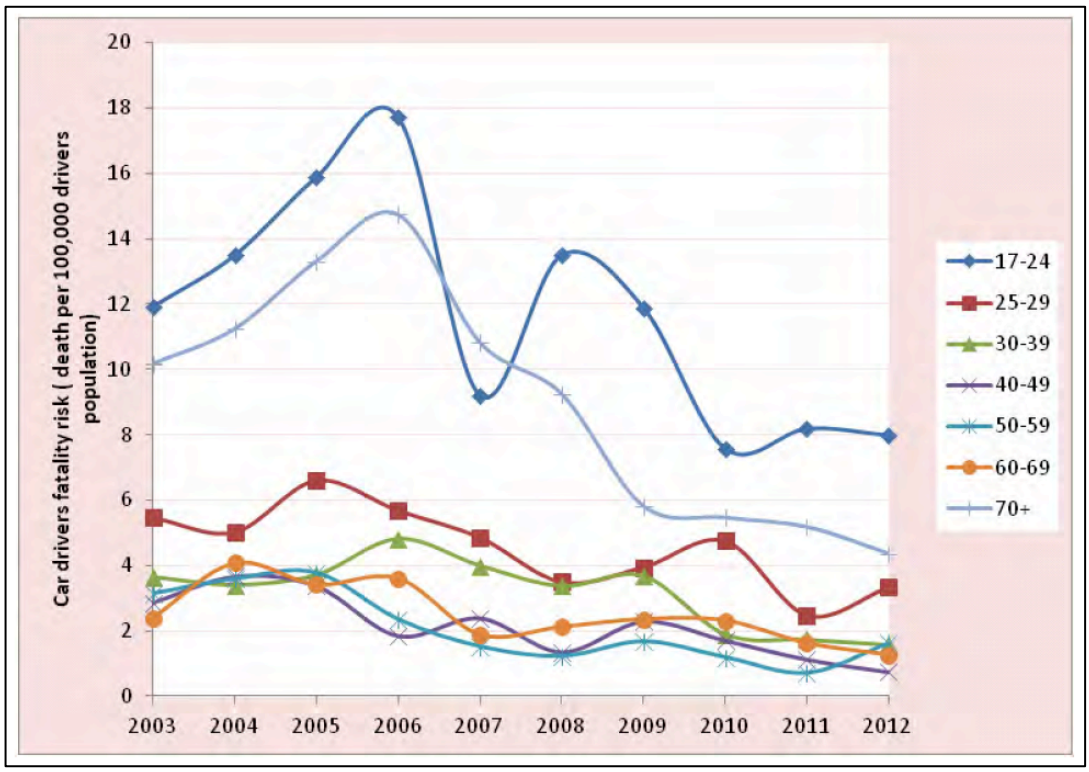

Figure 1.1: Fatal collisions per 100,000 drivers in Ireland, separated by age category

While younger drivers, overall, are more at risk, younger males are further overrepresented in collision statistics. For example, in 2012 in Ireland, males under the age of 25 were involved in almost twice as many collisions as younger females and over three times as many fatal collisions (RSA, 2013). Compared to the driving population in Ireland as a whole, younger males represented 25% of

driving fatalities that year (RSA, 2013). As males in this age category made up 5% of the driving population (leaner permit and full licence holders combined), they were five times overrepresented in fatal collisions that year, compared to other demographic groups. Although there is slight variation from year to year, this pattern generally holds, with the World Health Organisation reporting that males of this age are three times more likely to be involved in a fatal collision than age-matched females (WHO, 2015). Fatalities separated by age and gender for Ireland[1] are illustrated in figure 1.2, and for other OECD countries[2] in figure 1.3 (data from; OECD & ECMT, 2006; RSA, 2013).

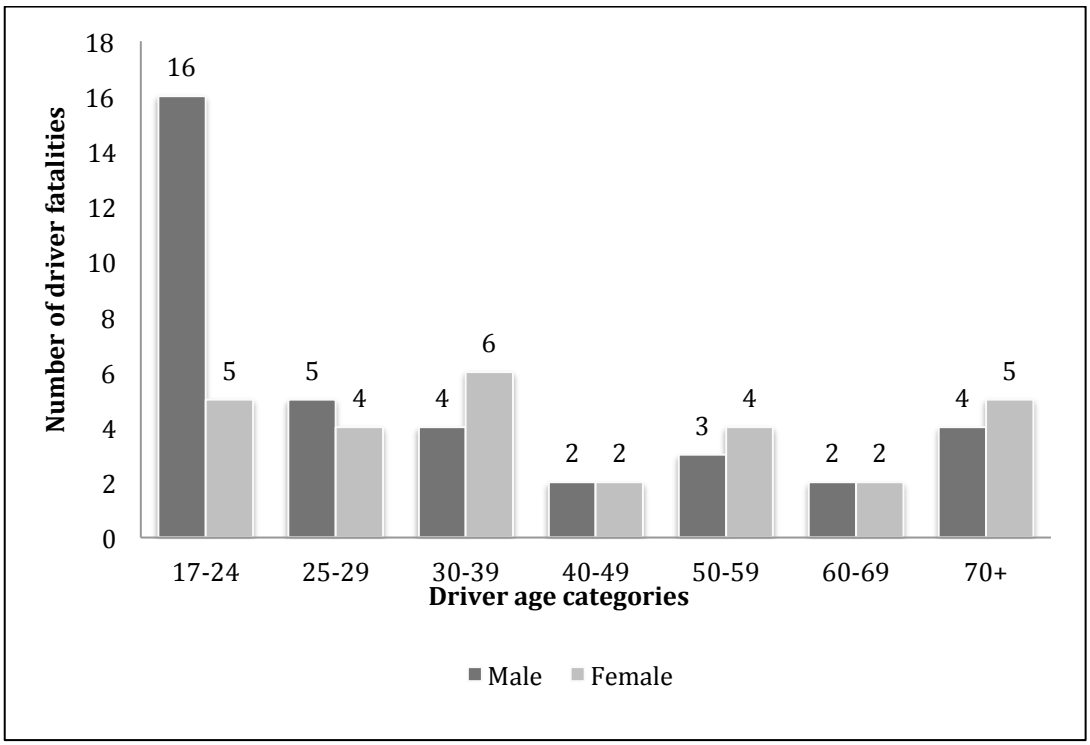

Figure 1.2: Age and gender breakdown of driver fatalities in Ireland in 2012

[1] Year 2012

[2] Year 2003 from the following OECD countries: Austria, Australia, Czech Republic, Denmark, Finland, France, Germany, Iceland, Japan, Korea, Netherlands, New Zealand, Norway, Poland, Portugal, Slovenia, Sweden, Switzerland, United States and United Kingdom.

1.2 Risks Associated with Driving

While the number of driving offences has been observed to decline by approximately eight per cent each year after licensure, and there is a six per cent decline in the number of collisions novice drivers have been culpable in, this decline appears to occur twice as quickly in female than in male novice drivers. This lead Clarke, Ward, Bartle, and Truman (2006) to conclude that young males are not only less skilled but also take more risks than other groups. Behaviours such as speeding, driving while intoxicated, not wearing a seatbelt and driving with peer passengers greatly increase the risk of being involved in a collision (Ferguson, 2003; Rice, Peek-Asa, & Kraus, 2003). These risk factors will be discussed in more detail below.

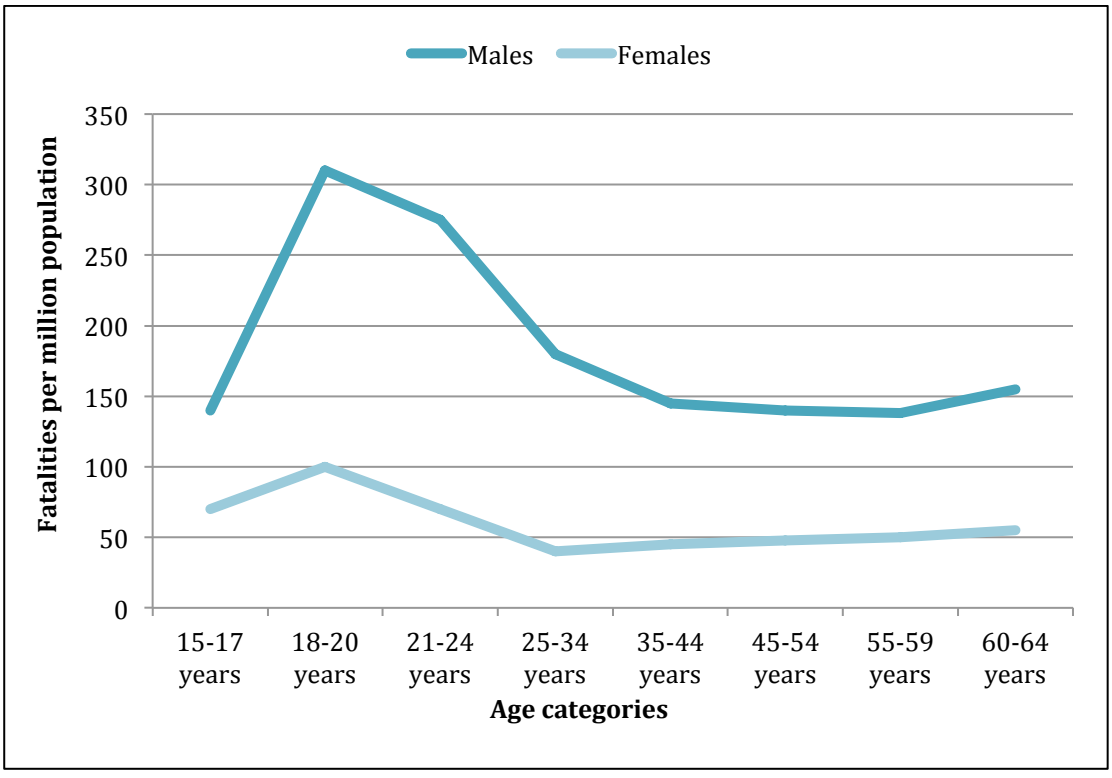

Figure 1.3: Age and gender breakdown of driver fatalities in OECD countries in 2003

1.2.1 Inexperience and age

Mayhew, Simpson, and Pak (2003) found that novices had a collision rate more than twice that of older drivers during their first month of unsupervised driving. The number of collisions novices were involved in declined by 70% from the second to the 24th month of licensure. Even after this decline, novices with two years experience were still found to be involved in 45% more collisions than older drivers. In general, novice drivers are most at risk of being involved in a collision during the first six months after receiving their licence (Mayhew et al., 2003). This is largely thought to be due to inexperience, as novices are driving unaccompanied for the first time. Despite the difficulty in separating the effects of age and inexperience on collision risk in drivers, some studies have demonstrated an interaction between the two factors that suggests that inexperience alone does not explain why younger drivers are overrepresented in collision statistics (McKnight & McKnight, 2003). For example, Mayhew et al. (2003) reported a significant interaction between age and inexperience during the first two years of licensure, where novice drivers between 16 and 19 had higher collision rates than novices over 20 years of age with the same amount of experience. Furthermore, as novices in the younger group had higher incidences of initial collision involvement, this group demonstrated a steeper decline in collision involvement with increasing experience (Mayhew et al., 2003). These findings are illustrated in figure 1.4.

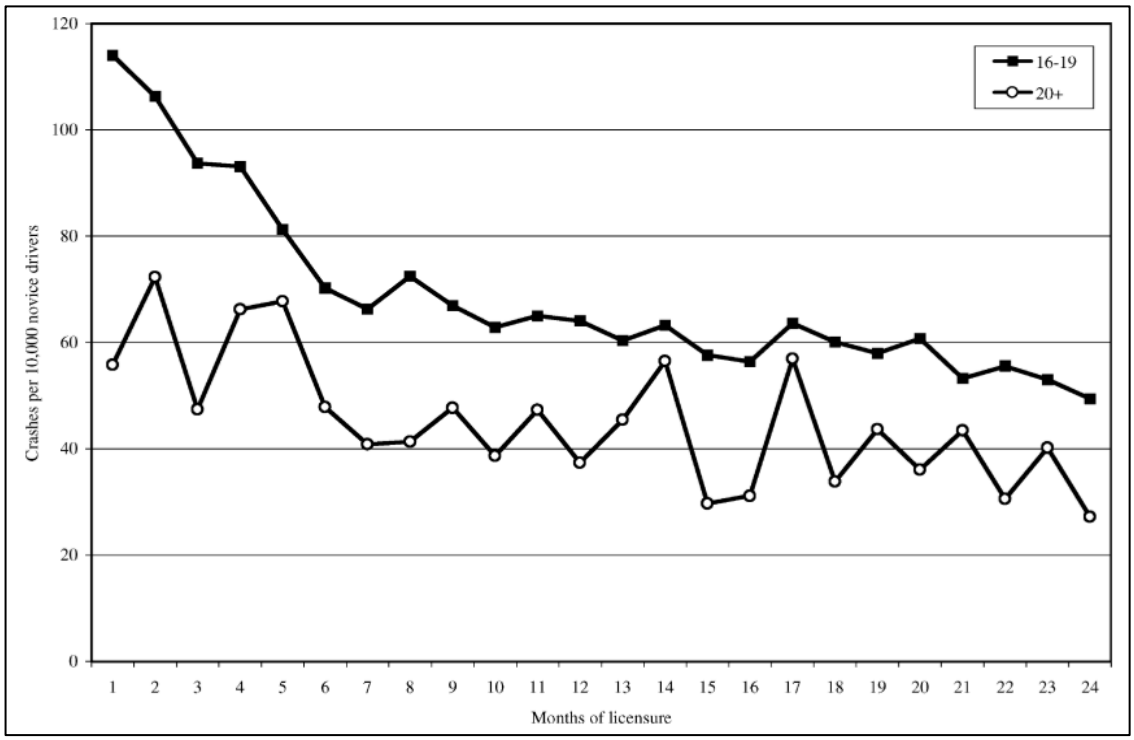

Figure 1.4: Illustration of the different slopes of collision decline by month of licensure in 16 to 19 year old novices and novices over the age of 20 (reproduced with permission from Mayhew et al., 2003, pg. 686).

1.2.2 Passengers

Research suggests that driving with peer passengers increases adolescent and young drivers' risk of being involved in a collision. Increases in the number of passengers (Ouimet et al., 2010; Tefft, Williams, & Grabowski, 2013) and carrying male passengers (Clarke, Ward, Bartle, & Truman, 2010) have been identified as particular risk factors. For example, in an observational study conducted by (Simons-Morton, Lerner, & Singer, 2005) it was noted that drivers in their late teens drove faster in the presence of a teenage male passenger. Furthermore, both young male and female drivers left a shorter following distance between them and the car in front while driving with a male passenger, relative to no passenger or female passenger. In contrast, while accompanied by

a female teenage passenger, young male drivers were observed to leave more space between their car and the car in front. In a review of collision statistics in the US, Ouimet et al. (2010) found that 15 to 20 year old males were most vulnerable when carrying 16 to 20 year old passengers. This study also suggested that peer aged passengers continue to be a risk factor in collision involvement throughout the twenties when both driver and passengers are male. Moreover, while carrying passengers young drivers are more likely to be judged as culpable in the collision (Lam, Norton, Woodward, Connor, & Ameratunga, 2003; Preusser, Ferguson, & Williams, 1998; Preusser, Williams, Lund, & Zador, 1990; Rice et al., 2003; Williams, 2003). The impact of peer passengers on young driver risk has been found to decrease with age (Gardner & Steinberg, 2005).

1.2.3 Alcohol

A blood alcohol concentration (BAC) of just 0.04g/dl is sufficient to impair performance while driving (Shinar, 2007), resulting in reduced vigilance, impaired perception, divided attention, slower reactions and longer time to make decisions. In Ireland, approximately 25% of drivers killed in collisions were over the legal BAC limit (RSA, 2012a). Clarke et al. (2010) found that, of all UK driving fatalities surveyed in their study, 20% of drivers were under the influence of alcohol and 4%[3] under the influence of drugs. Of the reported cases, cannabis was most commonly found, followed by amphetamines, MDMA (ecstasy), cocaine and heroin. These figures were more exaggerated between the

[3] The authors acknowledged that this figure is likely to be an underestimation of the contribution of drugs to fatal collisions, as toxicology screenings were not conducted in many cases.

hours of 8pm and 4am, with over 40% of collisions during this time attributed to drug and/or alcohol impairment. Males were found to be overrepresented in these statistics, as 23% of men culpable for a fatal collision were under the influence of drugs and/or alcohol, compared to 13% for women. Furthermore, the number of fatal collisions involving drugs or alcohol was found to decrease with age, with over a quarter of such fatalities occurring in drivers under the age of 30 (Clarke et al., 2010).

While drivers under the age of 20 drink and drive less often than older adults, they place themselves and others at greater risk when they do (Mayhew, Donelson, Beirness, & Simpson, 1986; Williams, 2003), as evidenced by a greater number of fatalities in younger drivers who were under the influence of alcohol. With increasing BAC the risk of being involved in a fatal collision increases at a much steeper rate in teenagers than is observed in older adults (Williams, 2003; Zador, Krawchuk, & Voas, 2000).

Males under the age of 25 with BAC levels above 0.05g/dl, compounded by inexperience, are two and a half times more likely to be involved in a collision than older, more experienced drivers with the same BAC levels (WHO, 2012a). The relationship between collision risk and alcohol in younger males is more pronounced when the number of fatal collisions are separated out by time of day. For example, in the UK, almost twice the proportion of fatal collisions due to alcohol consumption occurred in younger males between four and six in the morning compared to the day as a whole (Clarke et al., 2006). Clarke and colleagues confirmed that this figure meant that younger male drivers were

significantly more likely to be killed in a collision under the influence of alcohol between four and six am than any other demographic group.

1.2.4 Fatigue

As noted above, an increased number of fatal collisions occur during the hours of darkness. While alcohol contributes to a portion of these collisions, fatigue, known to impair driving performance, also plays a role (Shope & Bingham, 2008). This increased risk applies to drivers of all ages but especially to younger drivers (Rice et al., 2003). It is difficult to measure driver fatigue on scene after a collision (OECD & ECMT, 2006), nevertheless, fatigue has been implicated in the high instances of single car collisions observed in younger drivers at night, as fatigued younger individuals are particularly susceptible to distraction compared to older drivers (Williams, 2003). Evidence suggests that younger drivers are involved in 29% of all night-time collisions (Mayhew et al., 2003) and are three times more likely to be involved in a fatal collision at night than in the day time (Williams, 2003). Poor sleep hygiene in younger individuals has been implicated in this increased risk (Shope & Bingham, 2008). For example, self-reported sleep times have been observed to decrease between the ages of 13 and 20 and this has been found to relate to poorer performance on class tests (Wolfson & Carskadon, 1998). Lower levels of sleep in adolescents and younger adults has also been associated with increased risk-taking (O'Brien & Mindell, 2005).

1.2.5 Speeding

Excessive speed contributes to up to a third of all collisions (WHO, 2012a). It is also the most common driving violation committed by young male drivers (WHO, 2012a). W. A. Harrison, Triggs, and Pronk (1999) reported that exceeding the speed limit contributed to 30% of collisions in young males, compared to 21% of collisions involving young female drivers and 15% of collisions in older drivers. In a review of collisions in the UK, Clarke et al. (2006) found that, of the collisions young male drivers were culpable for, driving over the speed limit was found to have caused 19.4% of collisions overall, and 36.6% of collisions between the hours of four and six in the morning. Furthermore, Ouimet et al. (2010) observed that young male drivers were more likely to speed and leave a shorter distance between their car and the vehicle in front while doing so. While it has been reported that excessive speed contributed to 22% of fatal collisions and 19% of collisions resulting in serious injury between 1997 and 2011 in Ireland, the exact breakdown by age and gender has not yet been made available by the RSA (RSA, 2014).

1.2.6 Seatbelts

In the event of a collision, correct use of a seatbelt can reduce a driver's risk of fatality by up to 61% (WHO, 2012a). In the year 2012 in Ireland, 22% of all drivers killed in collisions were not wearing a seatbelt (RSA, 2014). While a breakdown by age and gender for these collisions has not yet been made available, other countries have reported lower seatbelt use (by approximately 10%) in young drivers (16 to 25) compared to adults and children (Everett et al., 2001; McCartt, Shabanova, & Leaf, 2003; Williams, McCartt, & Geary, 2003;

Williams, Rappold, Ferguson, & Wells, 1997; Williams & Shabanova, 2002), particularly in younger males (D. Begg & Langley, 2000). Such young drivers also self-report greater engagement in other risk-taking behaviours such as exceeding the speed limit and driving while intoxicated (D. Begg & Langley, 2000). In the US, up to two thirds of young drivers killed in a collision were not wearing a seatbelt (Shope & Bingham, 2008), compared to 34% in the UK (Clarke et al., 2010).

1.3 Risk-taking in Adolescence

Adolescence in humans is typically considered to fall between 12 and 18 years of age (Spear, 2000). During this time, adolescents undergo rapid physical growth and sexual maturation, as well as changes to self-confidence and social awareness (Dahl, 2008). Improved cognitive capacities with age result in increasing control over changing emotional processes, mediating arousal and goal-directed behaviours (Dahl, 2008). However, this is a protracted process, leaving adolescents vulnerable to heightened impulsive decision-making and behaviour for much of the second, and occasionally the third, decade of life (Stautz & Cooper, 2013). Hence, adolescence is often associated with novelty seeking, risk-taking, a lack of future thinking and consideration of the consequences of actions and decisions (Stautz & Cooper, 2013). While these characteristics are thought to have developed as an evolutionary mechanism to encourage autonomy, some adolescents have an exaggerated inclination towards risk-taking that increases the likelihood of life changing outcomes such as injury or death (Spear, 2000). A behaviour can be considered risky if it was performed without due consideration for future consequences (Panwar et al.,

2014), for example, adolescents are more likely than adults over the age of 25 to have casual sex partners, engage in unprotected sex, experiment with illicit substances, smoke cigarettes, be involved in a fatal collision, binge drink and engage in criminal behaviour (Johnson et al., 2002; O'Brien, Albert, Chein, & Steinberg, 2011; Steinberg, 2008).

Theories of risk-taking suggest that the higher the perceived risk of a particular behaviour, the less likely an individual will be to participate in the behaviour (Machin & Sankey, 2008; Reyna & Farley, 2006). Therefore, the ability to perceive risk has been deemed a protective factor (Mills, Reyna, & Estrada, 2008). Given the heightened levels of risk-taking observed in adolescence and young adulthood, research has examined whether adolescents appraise risks differently to adults (Johnson et al., 2002). While adolescents have been observed to understand the negative outcomes of certain risky behaviours (see Johnson et al., 2002 for examples), some younger individuals appear to perceive themselves to be, personally, less vulnerable to the negative outcomes associated with such behaviours (Cohn, Macfarlane, Yanez, & Imai, 1995). For example, Small, Silverberg, and Kerns (1993) found that adolescents who reported a history of risk-taking rated the consequences of such risks as less severe than their non-risky counterparts. An alternative explanation proffered is that, where the potential consequences are understood, some adolescents may consider the benefits of the behaviour to be worth the risk (Quadrel, Fischhoff, & Davis, 1993).

Other research by Reyna and Farley (2006) suggested that most adolescents and younger individuals do not expect or intend to take risks. Reyna and Farley

(2006) proposed that some participate in risk-taking while making decisions under the influence of emotion and/or impulsivity, while others may not deliberately avoid risks but intuitively grasp the trade-off between risk and benefit. The ability to control impulsive behaviours increases with age and coincides with the development of the prefrontal cortex (Paus, 2005). Individual variability in the decline of impulsivity may be related to the amount of risk-taking that younger individuals partake in (Arnett, Offer, & Fine, 1997; Finy, Bresin, Korol, & Verona, 2014). This is consistent with the observation that not all young individuals engage in risk-taking (Sercombe, 2014), and may implicate impulsive decision-making in risk-taking behaviours in adolescence (Panwar et al., 2014). Thus, the relationship between impulsivity and aberrant driving behaviours will be the focus of the current project.

1.3.1 Deliberate risk-taking while driving

Clarke et al. (2005) proposed that deliberate risk-taking contributes to many of the collisions that younger individuals are involved in, rather than lacking the skills to handle the situation. Younger drivers, and younger males in particular, report a more negative attitude towards, and less compliance with, the rules of the road (Bergdahl, 2005; Laapotti et al., 2006). For example young males have been found to disregard the speed limit more than other drivers (Williams, Kyrychenko, & Retting, 2006). This suggests that, in addition to inexperience, risk-taking resulting from an inability to regulate behavioural and emotional impulses while driving may play a role in collision involvement (Katila, Keskinen, Hatakka, & Laapotti, 2004; Keating, 2007). As not all individuals will take risks, certain individual difference factors are thought to relate to a driver's

likelihood to engage in deliberate risk–taking, such as higher levels of impulsivity and sensation seeking (Gregersen & Berg, 1994; Møller & Haustein, 2013; Rhodes & Pivik, 2011; Steinberg, 2004; Steinberg et al., 2008). Impulsivity, sensation seeking and sensitivity to reward have been observed to positively correlate with self-reported risky driving behaviours but not with driving outcome (such as the frequency of collision involvement) (Constantinou, Panayiotou, Konstantinou, Loutsiou-Ladd, & Kapardis, 2011). Specifically, these personality factors correlated with the number of reported driving violations and errors. Furthermore, motor impulsivity, disinhibition (a subscale of the sensation-seeking questionnaire) and sensitivity to reward corresponded with more reported aggressive violations in that study. While Constantinou et al. (2011) compared personality factors to self-reported driving tendencies, another study by West, Elander, and French (1993) compared groups based on a self-reported history of collision involvement. West et al. (1993) found that the number of collisions participants had accrued positively correlated with a measure of impulsive decision-making, with items such as 'how often do you ride on public transport without paying a fare?' and 'do you make decisions without considering all the implications?' The relationship between risky driving and impulsivity will be discussed in more detail in section 1.4.8.

1.4 Impulsivity

1.4.1 Definitions of impulsivity

Impulsivity is defined as predisposition to rapid, unplanned reactions to stimuli without consideration of the consequences (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001). Heightened impulsivity is seen to result from poor

self-control or restraint (Barratt, 1972, 1994) and a lack of forethought and planning in the decision-making process, which can increase the negative outcomes of actions (Sun, Cocker, Zeeb, & Winstanley, 2012). Impulsivity is also considered to be an inability to inhibit one's behaviour when the situation requires it or the inability to tolerate delays in earning rewards, referred to as 'delay of gratification' (Kirby & Maraković, 1996; Kirby & Petry, 2004). This theory has been supported by the effectiveness in which tasks designed to measure an individual's ability to tolerate delay and inhibit behaviours have been utilised in behavioural experiments of impulsivity (for example; Logue, 1995; Rachlin, 1995). Impulsivity is common but heightened impulsivity is associated with a range of pathologies (Kalenscher, Ohmann, & Güntürkün, 2006).

While most research quantifies trait impulsivity and sensation seeking separately, i.e. by using separate measures, there is debate in the literature as to whether these are two distinct constructs or whether sensation seeking is better described as a component of impulsivity (Lauriola, Panno, Levin, & Lejuez, 2013). Despite agreement that such measures produce highly correlated results, impulsivity and sensation seeking are often defined quite differently (Quinn & Harden, 2013). While impulsivity, as defined above, is considered acting in the moment without consideration of the consequences, sensation seeking, sometimes referred to as venturesomeness, is described as a preference for varied and exciting experiences (Dawe & Loxton, 2004; Duckworth & Kern, 2011; Eysenck, Easting, & Pearson, 1984; Eysenck & Eysenck, 1977, 1978; Gullo, Ward, Dawe, Powell, & Jackson, 2011; Magid & Colder, 2007; G. T. Smith, Fischer,

Cyders, Annus, & Spillane, 2007; Steinberg, 2007, 2008; Steinberg et al., 2008). Some longitudinal data suggests that these constructs develop along similar trajectories in adolescence (Harden & Tucker-Drob, 2011), while other cross-sectional research has observed distinct patterns of development during this period (Steinberg et al., 2008). This shall be discussed in greater detail in section 1.4.5.

Despite this, some research considers sensation seeking to be a component of impulsivity (Dir, Coskunpinar, & Cyders, 2014; S. Fischer, Smith, & Cyders, 2008). Zuckerman and Kuhlman (2000) expanded Zuckerman (1979)'s original model of sensation seeking to include impulsivity by positing that there is a synergistic relationship between these two traits, and that it is justifiable to combine them for certain types of analysis. This was based on evidence of a large portion of shared variance between the two constructs in correlational and factor analysis (as observed by; Joireman & Kuhlman, 2004; Lauriola et al., 2013). This adjustment to Zuckerman (1979)'s definition of sensation seeking was based on research by Glicksohn and Abulafia (1998), who proposed that there are two types of sensation seeking; impulsive and non-impulsive sensation seeking. In their model, Glicksohn and Abulafia (1998) referred to the adventure seeking behaviours commonly associated with sensation seeking as 'non-impulsive' sensation seeking behaviours (e.g. bungee jumping), while 'impulsive' sensation seeking behaviours were more likely to occur out of disinhibition. This latter type of sensation seeking was found to be more likely to relate to risky driving behaviours such as ignoring stop signals (Glicksohn & Abulafia, 1998). This suggests that the type of risky behaviour that the present study is

interested in, i.e. aberrant driving, is more likely to fall under the category of impulsive sensation seeking. Moreover, research has typically found a relationship between sensation seeking and recreational risk-taking (e.g. bungee jumping), with individuals who frequently engage in recreational risk-taking no more likely to take everyday risks, such as risky financial decisions (Hanoch, Johnson, & Wilke, 2006). Due to these observations, the current study excluded sensation seeking from its definition of impulsivity and, hence, did not include a psychometric measure of sensation seeking in the main experiment.

Further evidence to support the exclusion of a measure of sensation seeking was provided by a meta analysis of sensation seeking questionnaires by Cross, Cyrenne, and Brown (2013). Cross et al. (2013) observed a trend towards a floor effect in recent years on such measures, with a reduced ability of these to differentiate between groups, such as males and females. Cross et al. (2013) proposed that the items in these measures are becoming out-dated, with physical activities, such as skiing, no longer viewed as novel or intense. Cross et al. (2013) found that there is lower interest in engaging with activities that are no longer considered exciting, or which participants are likely to have tried. Based on their results Cross et al. (2013) recommended that sensation seeking questionnaires need to be redesigned to reflect more up-to-date extreme sports activities, otherwise alternative measures should be used.

1.4.2 Subtypes of impulsivity

In the study of impulsivity it is important to consider whether impulsivity is a single construct or comprised of many (Broos, Schmaal, et al., 2012). Moeller et al. (2001) defined impulsivity as a predisposition towards rapid, unplanned

reactions to stimuli without regard for negative consequences. Moeller and colleagues (2001) based this definition on what they considered to be the two main components of impulsivity; reactions to stimuli before all relevant information has been considered and a decreased sensitivity to negative consequences. This definition has established 'impulsive action' and 'impulsive choice' as two mutually exclusive components of impulsivity (Kalenscher et al., 2006). There are those who would support the theory of impulsivity as a three sided construct (reflexive impulsivity, impulsive choice and impulsive action) (Dalley, Everitt, & Robbins, 2011) or even five (inattention, impulsive reward-seeking, response slowing, impulsive responding and anxious responding) (Ersche et al., 2011), but the majority of research has conformed to this two faceted construct of impulsivity (for example; Broos, Schmaal, et al., 2012; Kalenscher et al., 2006; Kim & Lee, 2011; Paterson, Wetzler, Hackett, & Hanania, 2012; Sun et al., 2012; Winstanley, Eagle, & Robbins, 2006). Impulsive action and choice are assessed using different cognitive tasks and denoted by distinct neural networks, an inhibition system and one driven by reward (Casey et al., 2008), providing further evidence to support them as being independent constructs (Diergaarde et al., 2008).

1.4.3 Impulsive action

Impulsive action manifests in premature responding to stimuli, otherwise referred to as behavioural disinhibition. Kim and Lee (2011) said that impulsive actions are more common when a decision has to be made as quickly as possible. It is an issue of speed versus accuracy, with impulsive individuals typically responding to stimuli as fast as they can, often before all relevant information

has been considered. It can also be seen as a failure to suppress or inhibit inappropriate, reflexive actions once they have been initiated (Winstanley et al., 2006). It is considered to be due to a lack of planning and is linked to an increase in errors (Sun et al., 2012). Difficulty inhibiting responses can lead to undesirable consequences of action (Whelan et al., 2012) and is even associated with a pre-existing vulnerability to addiction (Diergaarde et al., 2008).

The increase in executive capacity and declining impulsive action observed to occur during adolescence has been attributed to the on-going maturation of the prefrontal cortex (Sowell, Delis, Stiles, & Jernigan, 2001). Regions such as the ventromedial prefrontal cortex (VMPFC; Kalenscher et al., 2006), the anterior cingulate (ACC; Braver, Barch, Gray, Molfese, & Snyder, 2001), the dorsolateral prefrontal cortex (DLPFC) and the right inferior frontal gyrus (rIFG; van Holst et al., 2012) are considered to be responsible for executive functions like cognitive control and behavioural inhibition. These regions, along with many others in the prefrontal cortex, have been found to be immature in adolescence and this may lead to the impairment of certain executive functions, such as behavioural inhibition (Bunge, Dudukovic, Thomason, Vaidya, & Gabrieli, 2002; Giedd et al., 1999; Rubia et al., 2000; Sowell, Thompson, Holmes, Jernigan, & Toga, 1999). A lot may be learnt about the construct of impulsive action and the prefrontal cortex's role in executive functions through the use of certain cognitive tasks. Impulsive action is often assessed using tasks that require participants to attend to information and respond as quickly as possible (Aichert et al., 2012). For the most part they come in the form of information sampling and response inhibition tasks, such as those utilising the Go/No Go paradigm (M. M. Mesulam,

1985). These tasks have been shown to recruit regions of the prefrontal cortex and so may be used to explore what effect an immature prefrontal cortex may have on certain cognitive abilities (Aichert et al., 2012; Bari & Robbins, 2013). This will be discussed in greater detail in chapter five.

1.4.4 Impulsive choice

All forms of impulsivity involve an impulsive action of some description but the main difference between impulsive action and choice is that the predominant or prevailing behaviour associated with the situation has to be forcibly inhibited to suppress impulsive choice (Winstanley et al., 2006). Diergaarde et al. (2008) said that impulsive choice reflects the decision-making process better than impulsive action. Impulsive choice is associated with poor decision-making with regard to the long and short-term consequences of actions, whether positive or negative (Diergaarde et al., 2008). It is typically considered to be an inability to delay immediate gratification with a disregard for negative consequences (Paterson et al., 2012) or a long-term disposition towards overestimating the value of immediate rewards and discounting the value of more distant ones. It has also been found that the longer a reward/punishment is delayed the greater its value/severity is discounted (Winstanley et al., 2006). This is also known as impulsive inter-temporal choice or delay discounting. Because of this, impulsive choice is typically measured with delay discounting questionnaires, such as Kirby's Delay Discounting Questionnaire (Kirby's DDQ; Kirby, Petry, & Bickel, 1999) and behavioural tasks about risk and reward, such as the Iowa Gambling Task (IGT; Bechara, Damasio, Damasio, & Anderson, 1994). In addition to these, tasks that are commonly employed by neuroimaging research to measure

reward processing in the brain may be useful in understanding the underlying mechanisms of impulsive choice in individuals, such as the Monetary Incentive Delay task (MID; Knutson, Fong, Adams, Varner, & Hommer, 2001). These measures, and the literature outlining how they relate to impulsivity and risky behaviour, will be discussed in chapter four.

Steinberg (2008) attributed greater impulsive choice tendencies during adolescence to an increased sensitivity to reward and a lack of executive control over reward seeking behaviours. This theory has been supported by the neuroimaging literature, which has posited a relationship between impulsive behaviours and both the reward processing and executive control systems of the brain (Casey et al., 2008). Fineberg et al. (2010) proposed that impulsive behaviours are driven by a striatal component (the ventral striatum and the nucleus accumbens shell) and inhibited by a prefrontal component (anterior cingulate and ventromedial prefrontal cortex, amongst others). During adolescence there is an imbalance in these systems, with the maturation of the limbic system in early adolescence relative to the continuing development of the prefrontal cortex into the mid-20s (Casey et al., 2008). This imbalance results in an increased sensitivity to reward and an underestimation of the potential negative consequences associated with that reward. This is proffered as an explanation for the increase in impulsivity and risk-taking behaviours during adolescence and young adulthood. It also may explain why impulsivity is seen to decline into adulthood as it coincides with the maturation of the prefrontal cortex. This theory, and the evidence that supports it, will be discussed in greater detail in chapters five and six.

1.4.5 Impulsivity and age

Contrary to the belief that personality is largely stable in adulthood (Costa Jr & McCrae, 1997; McCrae et al., 2000), evidence suggests that aspects of personality undergo changes throughout the lifespan, the most dramatic of which occur during adolescence (Caspi, Roberts, & Shiner, 2005; Collado, Felton, MacPherson, & Lejuez, 2014). For example, high levels of impulsivity in adolescence have been implicated in the noted increase in risk-taking propensities observed during this age (Steinberg, 2010; Steinberg et al., 2008). Compared to adults, adolescents demonstrate greater impulsivity (B. Fischer, Biscaldi, & Gezeck, 1997; Leshem & Glicksohn, 2007; Panwar et al., 2014; D. G. Smith, Xiao, & Bechara, 2012; M. S. Stanford et al., 1996), sensitivity to rewards (Cauffman et al., 2010) and discounting of future rewards and punishments (Reyna & Farley, 2006).

In a study by Steinberg et al. (2008), widely cited in the literature, psychometric impulsivity was observed to linearly decline between the ages of ten and 30, while sensation seeking followed a curvilinear pattern, peaking in their ten to 15 year old group before declining into adulthood. However, impulsivity is a multifaceted construct which is often measured in many different ways (Evenden, 1999). The methods that are employed to measure a construct, such as impulsivity or sensation seeking, can greatly influence the results obtained (Collado et al., 2014). Therefore, it should be noted here that both impulsivity and sensation seeking were measured using adapted versions of standardised questionnaires in the Steinberg et al. (2008) study. For example, impulsivity was measured using only three of the first order subscales of Barratt's Impulsiveness

Scale (only 18 of the original BIS-11 items were retained). The following subscales were omitted with little explanation; attention, cognitive complexity and self-control. Similarly, a reduced version of Zuckerman's Sensation Seeking Scale (ZSSS; Zuckerman, 1979) was employed to measure sensation seeking; only a selected six out of 19 items. Steinberg et al. (2008)'s justification was that they believed that many of the items of the ZSSS actually measure impulsiveness, rather than the discrete sensation seeking aspect of impulsivity.

Furthermore, Steinberg republished his 2008 article in 2010 with only minor edits, reiterating his original observations of impulsivity in adolescence but renaming his construct of sensation seeking to 'reward seeking' (Steinberg, 2010; Steinberg et al., 2008). Indeed, there is some inconsistency in how terms are used in Steinberg's research, with impulsivity, sensation seeking and reward seeking often referred to as the same constructs, for example, "these changes permit the individual to put the brakes on impulsive sensation-seeking behaviour and to resist the influence of peers, which, together, should diminish risk-taking" (Steinberg, 2008, p. 93). Therefore it is unclear what constructs were actually being measuring in these studies (Steinberg, 2010; Steinberg et al., 2008), or what effect altering the questionnaires could have made on the results they obtained. Despite these potential issues, other cross-sectional studies have replicated this linear decline in impulsivity from childhood to adulthood using other psychometric measures of impulsivity (for example; Galvan, Hare, Voss, Glover, & Casey, 2007).

Contrary to what is observed in cross-sectional research, longitudinal studies suggest that impulsivity may actually follow a curvilinear pattern of

development in adolescence, similar to sensation seeking. Longitudinal data collected as part of the National Longitudinal Study of Youth 1979 Child and Young Adult Survey revealed that 'cognitive control' (the name given to a psychometric construct that was generated from inverting the items on an impulsivity questionnaire) followed a quadratic pattern, falling to its lowest point at age 14-15 before linearly increasing (Shulman, Harden, Chein, & Steinberg, 2014). This study also reported an inverted-U shaped relationship between age and sensation seeking, peaking in mid adolescence (Shulman et al., 2014). Similarly, longitudinal data collected from 227 adolescents over five timepoints from the age of nine to 18 by Collado et al. (2014) demonstrated an inverted-U shaped relationship between impulsivity and age, with impulsivity increasing until approximately 13 to 17 years of age before declining. Furthermore, Collado et al. (2014) reported that sensation seeking increased at each timepoint in the study, while self-reported risk-taking increased along the same trajectory as impulsivity, before plateauing in late adolescence. Finally, a review of the data collected by the 'Children of the community' project, a longitudinal study of the outcomes of childhood psychopathology, observed that both impulsivity and sensation seeking followed a similar pattern of change in adolescence, increasing until approximately 15 years of age before declining (Kasen, Cohen, & Chen, 2011). However, it should be noted that this last example used their own, yet un-validated, impulsivity questionnaire and this may have influenced the results obtained by the study.

Regardless of whether impulsivity follows a linear or curvilinear pattern of development across adolescence, this construct is still implicated in the noted

increase in risk-taking associated with this developmental period. Different rates of change in impulsivity and sensation seeking between individuals in adolescence are considered by some researchers to be a vulnerability marker for increased risk-taking during this age (Littlefield et al., 2010). For example, Littlefield et al. (2010) found that individuals who declined in psychometric impulsivity more rapidly from age 18 to 25 also demonstrated a steeper decline in alcohol use during this time. Similarly, longitudinal data from Quinn and Harden (2013) suggested that individuals who show a slower decline in impulsivity with age are more susceptible to increased abuse of alcohol and recreational drugs, while those who were slower to decline in sensation seeking demonstrated increased alcohol use only.

The contradictory findings summarised above may be explained by the fact that these studies treated impulsivity as if it were one, homogeneous construct. However, as previously mentioned, the majority of the impulsivity literature does not view it as such. Cognitive and neuroimaging research commonly map the developmental trajectories of the subtypes of impulsivity separately. For example, cognitive control (the inverse of impulsive action) has been found to linearly increase with age, coinciding with the maturation of the prefrontal cortex (Giedd et al., 1999; Luna & Sweeney, 2004; Luna et al., 2001; Rubia et al., 2000; Sowell et al., 1999). However, the heightened sensitivity of the reward processing system relative to the immature 'control system' in adolescence is thought to be responsible for the increase, and subsequent decline, of reward seeking and risk-taking behaviours in adolescence, and may be more closely aligned to impulsive choice tendencies (Casey et al., 2008; Koob, Sanna, &

Bloom, 1998; Steinberg, 2008). Thus, the current study will employ different measures to assess these separate components of impulsivity in participants, psychometrically, behaviourally and with neuroimaging techniques. Furthermore, the literature of how each of these measures change with age will be discussed further in chapters four, five and six.

1.4.6 Impulsivity and gender

Women have been found to make fewer risky decisions than men, especially when a possible consequence of the decision is physical harm (Stoltenberg, Batien, & Birgenheir, 2008). Furthermore, it has been found that young males typically engage in more unsafe behaviours than young females (Gardner & Steinberg, 2005; Harré, 2000; Kelley, Schochet, & Landry, 2004). Young males, compared to young females, have been found to be more confident in their driving skills (DeJoy, 1992; Stoddart, 1987), to underestimate the risk associated with certain driving situations (Bragg & Finn, 1982; Matthews & Moran, 1986) and tend to predict that the consequences of collisions will be less serious than they would be in reality (DeJoy, 1992). These observed differences in attitudes are interesting given that young males are significantly, and consistently, overrepresented in fatal collision statistics. For example, 70% of all driver fatalities in Ireland in 2014 were male (RSA, 2015c), and younger males (under 25) were overrepresented in collision fatalities five fold in 2012 in Ireland (RSA, 2013). Therefore, gender differences in impulsivity may account for some of the variation in risk engagement between males and females (Constantinou et al., 2011; Côté, Tremblay, Nagin, Zoccolillo, & Vitaro, 2002; Dir et al., 2014).

As such, research tenuously suggests that males, in both adolescence and later adulthood, may be more impulsive than females (Côté et al., 2002; Lejuez et al., 2002; Pedersen, Molina, Belendiuk, & Donovan, 2012). For example, a large study by Chamorro et al. (2012), which surveyed 43,093 people in the United States, did find that the impulsivity of males was greater than females, however, other cross-sectional (Steinberg et al., 2008) and longitudinal studies (Collado et al., 2014; MacPherson, Magidson, Reynolds, Kahler, & Lejuez, 2010) have not replicated this finding.

In a meta-analysis of 227 studies conducted by Cross, Copping, and Campbell (2011), males were found to have higher overall impulsiveness (all measures combined) than woman, although the effect was small. To further investigate the effects of gender, impulsivity was separated into its subcomponents, i.e. examining each behavioural task and the subscales of the psychometric measures separately. When this was done, Cross et al. (2011) found that certain questionnaires did not demonstrate a reliable effect of gender, for example the Temperament and Character Inventory, Revised (TCI-R; Cloninger, 1999), and Eysenck's I_7 Impulsiveness Questionnaire (Eysenck et al., 1984). Despite this, males were found to score higher on the attention and non-planning second order subscales of the BIS-11 (Patton, Stanford, & Barratt, 1995), the dysfunctional impulsivity subscale of Dickman's Functional and Dysfunctional Impulsivity Scales (Dickman, 1990) and 'carelessness' in the Social Problem Solving Inventory–Revised (SPSI-R; D'Zurilla, Nezu, & Maydeu-Olivares, 1996). Together, these findings suggested that males consider the future to a lesser extent than females (Cross et al., 2011). A significant, but small effect was also

observed for the Urgency subscale of the UPPS (Whiteside & Lynam, 2001), where females reported greater regret for their impulsive actions.

For the behavioural tasks included in the meta-analysis, Cross et al. (2011) reported that males pursue rewards under increasing risk on the Balloon Analogue Risk Task (BART; Lejuez et al., 2002) for longer than females, perform significantly better (i.e. select more advantageous decks) on the IGT (Bechara, 2005) and there was no difference in performance on executive functioning tasks such as the Stroop tasks (Stroop, 1935), Stop Signal Reaction Time task (SSRT; Logan, 1994) and the Go/No Go task (Newman, Widom, & Nathan, 1985). Furthermore, Cross et al. (2011) noted that males' superior performance on the IGT reflected, to an extent, a strategy in females to avoid punishments, rather than a preference for high risk decks, which can result in poorer performance in the long run. Cross et al. (2011) concluded that there was a weak effect of gender on impulsiveness in the literature, and emphasised the importance of examining the subtypes of impulsivity separately. Hence, the potential gender differences of each measure utilised by the current study will be discussed in greater detail in chapter four.

1.4.7 Impulsivity, risk-taking and psychological disorders

Impulsivity results in a lack of forethought and planning in the decision-making process and can increase the negative outcomes of actions (Sun et al., 2012). Regardless of how it is defined, however, real world risk-taking is believed to be the behavioural expression of impulsivity (Enticott, Ogloff, & Bradshaw, 2006). Impulsivity is common but increased impulsivity is associated with a range of pathologies which involve some form of deficit in the frontal lobe region of the

brain, such as Attention Deficit Hyperactive Disorder (ADHD), pathological gambling and addiction (Kalenscher et al., 2006). In fact, combinations of extreme impulsiveness, novelty seeking, harm avoidance and reward dependence are thought to underlie many DSM (Diagnostic Statistical Manual) disorders (Cloninger, 1987), such as antisocial, narcissistic and borderline personality disorders (Van Den Eynde et al., 2008), bipolar disorder (Swann, Dougherty, Pazzaglia, Pham, & Moeller, 2004; Swann, Steinberg, Lijffijt, & Moeller, 2008), obsessive compulsive disorder (Kashyap et al., 2012), depression (Piko & Pinczés, 2014), mood disorder (Peluso et al., 2007) and posttraumatic stress disorder (Weiss, Tull, Viana, Anestis, & Gratz, 2012), as well as other behaviours such as suicide attempts (Dougherty et al., 2004; Quednow et al., 2006), driving violations and risky sexual behaviours (Chamorro et al., 2012).

High impulsivity has been linked to other behavioural traits such as aggression and irritability (Helfritz & Stanford, 2006) and can lead to maladaptive, unacceptable and harmful behaviours (Pietras, Cherek, Lane, Tcheremissine, & Steinberg, 2003). Impulsivity has demonstrated the ability to significantly predict delinquency behaviours, such that higher impulsivity indicated a greater likelihood to engage in problem behaviours (Colder & Stice, 1998). Similarly, violent offenders have been observed to have greater trait impulsivity than those convicted of non-violent offenses (P. Smith, Waterman, & Ward, 2006).

Addicted and ADHD populations have been the focus of much of the impulsivity literature (Arns, De Ridder, Strehl, Breteler, & Coenen, 2009; Bond, Verheyden, Wingrove, & Curran, 2004; Lane, Moeller, Steinberg, Buzby, & Kosten, 2007;

Markus & Jonkman, 2007; Rodriguez-Jimenez et al., 2006). ADHD is categorised by inattentive, hyperactive and impulsive behaviour and an inability to inhibit behavioural responses, the latter of which is thought to be one of the fundamental deficits which characterises ADHD (Winstanley et al., 2006). On a battery of behavioural tasks of impulsiveness, measuring response inhibition (Go/No Go and SSRT), cognitive inhibition (Stroop and Switching tasks), sustained attention and time discrimination, children with ADHD demonstrated premature responding across all six tasks compared to healthy controls in a study by Rubia, Smith, and Taylor (2007). Similar findings have also been observed in adults with ADHD compared to age matched controls (Malloy-Diniz, Fuentes, Leite, Correa, & Bechara, 2007). Furthermore, compared to healthy controls, individuals with a history of ADHD have scored significantly higher on the BIS-11 (Rodriguez-Jimenez et al., 2006) and on measures of delay discounting, choosing more immediate rewards over larger, delayed rewards (Solanto et al., 2001; Sonuga-Barke, 2003). The degree of preference for immediate rewards in individuals with ADHD has also been reported to relate to inattention, where individuals with poorer attention ratings demonstrated greater discounting of future rewards (Paloyelis, Asherson, & Kuntsi, 2009).

Maladaptive behaviours such as drug and alcohol addiction (Broos, Diergaarde, Schoffelmeer, Pattij, & De Vries, 2012) and pathological gambling (Petry, 2001; Steel & Blaszczynski, 1998) have also been found to have a strong link to impulsivity. Higher BIS-11 scores have been found for cocaine dependent adults (Lane et al., 2007) and ecstasy users (Bond et al., 2004) relative to controls. Greater delay discounting is also associated with addicted populations, where

the appeal of a reward appears to steeply decrease in value with increasing delays for individuals addicted to alcohol and drugs (Petry, Kirby, & Kranzler, 2002). A limited number of studies tentatively suggest that higher levels of impulsivity and poor behavioural inhibition in childhood may result in substance abuse problems in adolescence and adulthood (Nigg et al., 2006; Tarter et al., 2003; Wong et al., 2006). Despite this, further research is needed to establish whether impulsivity can really predispose someone to risky behaviour (such as addiction) or whether engaging in risk may itself increase impulsive behaviours. At the least, elevated levels of impulsivity in adolescence are associated with problematic gambling (Vitaro, Ferland, Jacques, & Ladouceur, 1998), experimentation with drugs and alcohol (Whelan et al., 2012) and even internet addiction (Cao, Su, Liu, & Gao, 2007). Also, as mentioned above, a slower decline in impulsivity from adolescence into early adulthood may be a vulnerability marker for increased abuse of alcohol and drugs (Littlefield et al., 2010; Panwar et al., 2014; Quinn & Harden, 2013; Soloff, Price, Mason, Becker, & Meltzer, 2010; Vitaro et al., 1998).

1.4.8 Impulsivity & driving

As official statistics would suggest, adolescents and younger adults (under 25) are more likely to be injured or killed while engaging in behaviours that involve risk, like driving (Glendon, 2006). Glendon (2011) stated that adolescence is the optimal time for learning new skills, such as driving, due to the developing brain's efficient ability to incorporate new information. However, due to a noted increase in impulsivity and risk-taking behaviours during adolescence this is also a dangerous period for both learner and novice drivers. Impulsivity has

been implicated by the research as a factor influencing aberrant driving (Brandau et al., 2011; Jakubczyk et al., 2013; Loo, 1979; Richer & Bergeron, 2009; M. S. Stanford et al., 1996; Wickens et al., 2008) and it may pay a particular role in crash involvement (Ulleberg, 2001). For example, it has been observed that drivers convicted of dangerous driving offences had higher impulsivity to those with no driving related convictions (Paaver, Eensoo, Pulver, & Harro, 2006).

In an effort to identify the characteristics of novice drivers who have previously been involved in a collision, Donovan, Umlauf, and Salzberg (1988) conducted a cluster analysis of participants' responses to several personality measures. Donovan et al. (1988) found that the most at risk group were high in impulsivity, sensation seeking and aggressive behaviours. Comparably, when Chamorro et al. (2012) divided participants into impulsive and non-impulsive groups, significantly more individuals in the impulsive group reported engaging in risky driving. Furthermore, by dividing participants into groups based on their responses to the Manchester Driver Behaviour Questionnaire (DBQ; Parker, Lajunen, & Stradling, 1998), Owsley, McGwin, and McNeal (2003) found that individuals who responded in the 75th percentile of self-reported deliberate driving violations were 2.8 times more likely than those in the 25th percentile to be highly impulsive.

The neural network thought to be responsible for successful behavioural inhibition is mainly comprised of regions of the prefrontal cortex, which continue to undergo maturational changes through to early adulthood (Casey et al., 1997). Glendon (2006) proposed that the immature state of this network in

adolescents and young adults may explain why younger individuals are overrepresented in collision statistics. Younger drivers may find it difficult to inhibit motor responses while driving, which may make some manoeuvres more difficult, such as stopping at traffic lights where a directional arrow is incongruent with the main light (Glendon, 2006, 2011). One study by O'Brien and Gormley (2013) examined impulsivity in both risky and non-risky young males, with a conviction of speeding serving as the criterion of risk. Impulsivity was measured psychometrically (with the BIS-11) and through performance on certain cognitive tasks. The study found that individuals convicted of speeding had higher trait impulsivity than non-offenders and made more commission errors (i.e. demonstrated poorer performance) on the Go/No Go task. This suggests that young drivers that speed may be high in impulsivity, which may impact on their ability to inhibit behaviours.

This discussion will be continued in chapter four, where the literature examining the relationship between aberrant driving and the specific psychometric and behavioural measures employed by the current study will be reviewed in greater detail.

1.5 Driving and The Brain

1.5.1 Activation during driving tasks

Driving is a complicated task that involves the integration of several behaviours such as selecting and dividing attention, visuomotor integration, visuospatial interpretation and decision-making (Calhoun et al., 2002). Certain cognitive models have been developed to explain how these processes are integrated

during driving (Ballard, Hayhoe, Salgian, & Shinoda, 2000; Groeger, 2000), based on early neuroimaging research with tasks selected to elicit activity in specific regions (Cabeza & Nyberg, 2000) or by using a subtractive method (Walter et al., 2001). However, since the publication of these earlier studies, Independent Components Analysis (ICA), an analytical technique that separates the mixed signals of global activation into their individual sources, has been the predominant method of analysis in the study of the neural correlates of driving (for examples see; Carvalho, Pearlson, Astur, & Calhoun, 2006; Graydon et al., 2004). Using ICA Calhoun et al. (2002) found increased activation in primary visual, higher order visual regions during a driving task and a decrease during rest (fixation). During the driving task, activation in the anterior cingulate cortex (ACC) and various frontal regions (including the left inferior frontal gyrus, IFG, and the orbitofrontal cortex, OFC) gradually decreased while a more dramatic decrease was observed in fronto-parietal and cerebellar (motor) regions. These findings are in agreement with an earlier study by Peres et al. (2000). Previous research has established that frontal and parietal areas are implicated in selected and divided attention (Corbetta et al., 1998; Corbetta, Miezin, Dobmeyer, Shulman, & Petersen, 1991), the anterior cingulate (along with the posterior cingulate cortex, PCC) with spatial attention, necessary for vigilance (Mesulam, Nobre, Kim, Parrish, & Gitelman, 2001), while activational changes in the OFC are seen to occur during breaches of expectation, error detection, inhibition and motivation (Nobre, Coull, Frith, & Mesulam, 1999).

The decrease in cerebellar activity observed may be the result of a passive task in which participants did not have to complete any motor action in response to

the virtual environment. Activation in the cerebellum is typically associated with tracking movements (Horne & Butler, 1995; X. Liu et al., 1997; Stein, 1986) and making motor responses, such as those necessary to steer and operate a car, as demonstrated by research where participants actively engaged in a simulated driving condition with the use of a joystick (Horikawa et al., 2005). It is important to note also that the decreased activation observed in regions associated with inhibition, vigilance, spatial attention and motivation may not reflect a decrease in neural activity. Although there is general consensus that positive change in the Blood oxygen level dependent (BOLD) reflects an increase in neural (synaptic) activation there is much debate about the true meaning of a negative change in BOLD, or 'deactivation' (Ernst et al., 2005). Originally believed to be the suppression or inhibition of synaptic firing (Raichle, 1998), deactivation may not actually represent a reduction in neural activity (N. Harel, Lee, Nagaoka, Kim, & Kim, 2002; Heeger & Ress, 2002). It may reflect a vascular phenomenon or reversed baseline state (Czisch et al., 2004), a metabolic process (Hampson, Driesen, Skudlarski, Gore, & Constable, 2006) or may even be related to increased activation in some circumstances (Reas, Gimbel, Hales, & Brewer, 2011, found that deactivation correlated with increased performance on a memory task). The decrease in activity observed in the Calhoun et al. (2002) study may have been due to a low demand task or caused by a decrease in default-mode activity during simulated driving but not during passive viewing a driving scene (Greicius & Menon, 2004). When taking the above into consideration, the findings of the Calhoun et al. (2002) study may be interpreted thus; during driving there is an increase in visual processing, fine and gross

motor control and visuomotor integration, alongside a recruitment of regions responsible for spatial navigation, inhibition, error monitoring and vigilance.

1.5.2 Passive versus active driving tasks

As mentioned previously, it may not be sufficient to study neural activation during passive viewing of a driving scenario. Some have suggested that there are no real differences between the results of passive and active driving tasks (Jeong et al., 2006), but there is a strong argument for the inclusion of both types of task when investigating the neural activity associated with driving. One study that used such a method, by Uchiyama, Ebe, Kozato, Okada, and Sadato (2003), compared BOLD signal during a passive following condition to an active one. Participants experienced a virtual reality while in an MRI scanner where they either passively viewed a driving scene in which their car followed a consistent distance behind another, or had to actively alter their own speed to maintain a distance of five meters between themselves and a car of varying momentum in front. They found increased activation in the bilateral cerebellum, basal ganglia, pulvinar nuclei of the thalamus, ventral and dorsal premotor cortex, inferior parietal lobule, left primary sensorimotor cortex, supplementary motor area (SMA) and ACC during the active task, compared to the passive one. They suggested that dorsal and parietal premotor regions are responsible for visuomotor integration (Johnson, Ferraina, Bianchi, & Caminiti, 1996), preparing and executing movements based on visual perception (Deiber, Ibanez, Sadato, & Hallett, 1996; Grafton, Fagg, & Arbib, 1998). However the basal ganglia may also play an important role in the selection of appropriate movements, visuomotor guidance and muscle activations (Jueptner & Weiller, 1998; X. Liu, Tubbesing,

Aziz, Miall, & Stein, 1999) and may even filter movements, optimising desired ones and inhibiting others (Mink & Thach, 1991). The simultaneous basal ganglia, thalamus and premotor cortex activations suggests a network in which the basal ganglia may monitor the spatial location of movement cues (Middleton & Strick, 2000). In addition to this, the ACC is said to be part of the 'error prevention network' and may be involved in error detection and correction (Carter et al., 1998; Mesulam et al., 2001). Recruitment of this region during driving may be crucial, although there has been conflicting evidence in the literature as to its involvement in the driving network, with studies like Uchiyama et al. (2003) finding activation in this region, while others like Walter et al. (2001) have not. Similarly, in another study by Horikawa et al. (2005), participants both passively viewed a driving condition and engaged in a simple, simulated driving task. During the task condition activation was observed in the cerebellum, middle temporal and occipital gyrus, cuneus, and precentral gyrus compared with the passive condition. The cerebellar activation, as discussed previously, may reflect the participant's motor response when steering the car, while most of the other regions are associated with the processing of visual information while driving. These findings support and validate the inclusion of both passive and active tasks when studying the neural correlates of driving.

A strength of the Horikawa et al. (2005) and Uchiyama et al. (2003) studies was that they, unlike many others, compared neural activation to performance on the active driving tasks. Uchiyama et al. (2003) found that performance in the active condition was negatively correlated with activation in the areas surrounding the ACC. The authors suggested that participants whose activation in the ACC was

less focused, i.e. spread to the surrounding areas, may have found the task more difficult due to less efficient error detection in this region. Horikawa et al. (2005) found regional cerebral blood flow (rCBF) in the primary visual cortex, right thalamus and mid-brain to be positively correlated with the time it took participants to complete the course in the simulated environment. Analysis on task performance revealed that the number of crashes correlated with rCBF in the PCC but that there was no apparent relationship between rCBF and other behaviours such as speed or lane changes. This may have been the result of an overly simple driving task in which participants were not allowed to select their own course, hence lacking an assessment of spatial orientation skill (Horikawa et al., 2005).

Graydon et al. (2004) posited that the observations of these studies may have limited applicability to driving, as driving in a virtual reality may not elicit the same network of brain activity typical of real world driving. In their study, Graydon et al. (2004) used a real driving scenario based on one developed by Angell, Young, Hankey, and Dingus (2002). Periods of real driving were filmed and simulated to predict on road performance. The task contained sections where participants had navigational control and could alter their speed, accelerating and decelerating, in response to a range of 'visual events' that occurred in the driving environment, such as brake lights, signals, overtaking and traffic signs. Like Calhoun et al. (2002), Graydon et al. (2004) found activation in high and low order visual processing regions (such as Brodmann's area (BA) 17–19, lingual and fusiform gyri and the pulvinar) and attention processing regions (anterior and posterior cingulate cortex, inferior and

superior parietal lobules, tempero-occipital junction and the area of the frontal cortex). These regions reflect both top-down and bottom-up systems of visual perception, attention and control, as proposed by Corbetta and Shulman (2002). According to their study top-down attention regulates perception and responses to sensory information and is thought to be controlled by a bilateral fronto-parietal network consisting of the intra-parietal sulcus and superior parietal lobule. The bottom-up attention system is recruited during the detection of behaviourally relevant sensory events. It consists of ventral fronto-parietal network, largely lateralised in the right hemisphere, containing the temporal-parietal junction, inferior parietal lobule, superior temporal gyrus and inferior/middle frontal gyri. Activation in these networks during the simulation demonstrates the complex visual and attentional demands of driving. Graydon et al. (2004) also found additional activation in cortical and sub-cortical regions implicated in motor coordination and control, vigilance and motor responses to visual events (the basal ganglia and cerebellum, similar to Horikawa et al., 2005) and motor preparation regions (Brodmann areas 6 and 8 and the thalamus). Their findings represent a network of visuomotor integration, attention, vigilance and motor response, all skills needed to drive in a safe and reliable manor.

1.5.3 Activational changes by complexity of the driving manoeuver

Michon (1989) proposed a three-tier hierarchical model of driving. The bottom, and simplest level, contains perceptual processing and action execution, the next covers the processes responsible for planning actions or adapting to the movements of other drivers and finally the top, and most complex, level

comprises of strategic processes such as route choice or consideration of road traffic rules. Schweizer et al. (2013) applied Michon (1989)'s model to the study of the neural correlates of driving in order to determine whether the demand of the driving task would impact on the driving network recruited. Schweizer et al. (2013) examined activation during a simulated driving task in which participants were faced with making decisions in situations of varying complexity. They found minimal activation during right turns (the simplest task) and some activation in visual-parietal and motor areas in the posterior part of the brain during left turns (without oncoming traffic). When making a left turn with oncoming traffic[4] larger activations of the posterior network were observed, along with additional activation of the cingulate cortex, an area important for cognitive-response selection and alertness (Vogt, Hof, & Vogt, 2004). In the presence of distractors, a significant shift in activation from posterior to anterior regions was observed. For instance, auditory distraction while driving straight elicited activation in prefrontal regions such as the ventrolateral prefrontal cortex (VLPFC) in addition to reduced activation in posterior auditory cortices. Frontal regions, primarily the dorsolateral prefrontal cortex (DLPFC), were additionally engaged by the network when participants were distracted during more complex manoeuvres. The VLPFC and the DLPFC are known to be involved in executive functions such as attention, behavioural inhibition, decision-making and multitasking behaviours (Christoff & Gabrieli, 2000) and activation in these regions during distracted driving illustrates the brain's capacity to compensate for increased task difficulty by

[4] This study was conducted in the United States where they drive on the right-hand side of the road. Left turns, in this instance, would mean crossing into oncoming traffic.

recruiting executive regions to modulate proliferated attentional needs. Calhoun et al. (2002) also demonstrated recruitment of both anterior and posterior regions, with attentional systems modulated by changes in speed. During speed conditions they noted a shift in activity from posterior to anterior regions (like in Schweizer et al., 2013), suggesting increased vigilance, but at the cost of error detection and disinhibition. The degree of the decline in error detection and disinhibition was found to be inversely related to speed, with increasing speed coinciding with a faster decline in activation in regions associated with these behaviours. These findings suggest that the network engaged during driving varies, depending on the complexity and demand of the task, tentatively supporting the model by Michon (1989).

As mentioned above, research has identified a complex network involving frontal, motor, parietal, occipital, cerebellar and visual regions while driving (Calhoun & Pearlson, 2012; Calhoun et al., 2002; Graydon et al., 2004; Uchiyama et al., 2003; Walter et al., 2001). However these studies say very little about how patterns of activation may vary by driving manoeuvre or how factors, such as traffic, may impact on the decision-making process while driving. Spiers and Maguire (2007) addressed this gap in the research by avoiding the 'race-track' like environments commonly used in such research in favour of an accurate virtual reality simulation of London city, complete with signs, signals and traffic. They discovered distinct patterns of activation for visual-spatial and visual-motor processes involved in driving manoeuvres such as turning, reversing, and stopping. Specifically they found increased activation in much of the dorsolateral precentral and postcentral gyri when starting from a stationary position, for

reversing they detected activation bilaterally in the lateral precentral gyrus, the anterior insula and VLPFC, stopping elicited localised anterior pre-SMA activation and manoeuvres to avoid collisions, such as swerving, activated SMA and lateral dorsal premotor areas along with the cerebellum, medial occipital and posterior middle temporal gyri, the middle and anterior cingulate cortex, bilateral VLPFC, left insula, precuneus and posterior parietal cortex. When turning at junctions they observed activation in the dorsolateral precentral gyrus in addition to activation extending dorsally from the occipital cortex to the superior parietal cortex in the left hemisphere, and laterally to the posterior middle temporal gyrus in the right hemisphere. They even detected diverging patterns of activation in the calcarine sulcus ipsilateral depending on the direction of the turn. Lastly, they found pre-SMA (more anterior than what was observed for stopping manoeuvres), cerebellar, lateral occipital, superior parietal cortices and precuneus activity for action planning behaviours. The above points suggest recruitment of visual, motor, spatial and decision-making processes, with the degree of complexity and integration of this network dependent on the demand of the driving task. The findings of Spiers and Maguire (2007) seem to indicate that the more challenging the driving manoeuvre the greater the number of regions recruited into the network.

1.5.4 Interference in the driving network: the effect of distractors

Driving, as a process, becomes sufficiently automated with experience, allowing drivers to perform other simultaneous tasks, like conversation (W. Schneider, 1999). There is some debate in the literature as to the effect of distractors on automated driving behaviours. Some have suggested that aspects of driving are

unaffected by a secondary task (Haigney, Taylor, & Westerman, 2000) and some have even found certain aspects to improve (Engström, Johansson, & Östlund, 2005), however there is general consensus within the literature that a secondary task results in a large overall decrement in driving (see a meta-analysis of the literature by Horrey & Wickens, 2006). For example, cell phone usage is considered to be distracting and is associated with increased risk while driving. Studies have proposed that it is the process of manually operating a phone that distracts drivers (Brookhuis, De Vries, & De Waard, 1991), but with hands-free devices becoming more common, is just attending to someone else's speech enough to distract people while driving (Ranney, Harbluk, & Noy, 2005; Shinar, Tractinsky, & Compton, 2005)? Just, Keller, and Cynkar (2008) found that neural activity while driving decreased during concurrent listening, suggesting that distractors may interfere with some of the processes involved in driving. Although there is little evidence on how this may impact real world driving it has potential implications for selecting and dividing attention. An individual's ability to shift attention between task-relevant goals and several sub-goals may reduce the effect of distractors. The ACC and DLPFC are thought to be the neural bases of dual-task performance (Bunge, Klingberg, Jacobsen, & Gabrieli, 2000; E. E. Smith et al., 2001), exerting top-down control over task-dependent regions during concurrent tasks (Kondo, Osaka, & Osaka, 2004, found dual-task performance correlated with mean signal change in the ACC and DLPFC). Cognitive training programs may reduce costs associated with distractors (Erickson et al., 2007). Over multiple sessions, feedback from these training programs has been shown to reliably reduce costs associated with performing multiple concurrent tasks. Furthermore, skills learnt during training

may be retained for several months and have even been observed to transfer to other tasks (Bherer et al., 2005; Kramer, Larish, Weber, & Bardell, 1999; Schumacher et al., 1999). These programs may have the potential to better equip drivers to deal with distractors.

1.6 Summary and Aims for the Current Study

In conclusion, adolescents have higher levels of impulsivity and engage in more risk-taking behaviours than adults (Blakemore & Robbins, 2012; Johnson et al., 2002; Steinberg, 2008). Impulsivity is common and does not necessarily result in risk-taking, but increased impulsivity has been found to lead to maladaptive behaviours (Kalenscher et al., 2006; Pietras et al., 2003). It is thought that impulsive action behaviours occur due to the immature prefrontal lobe's inability to consider all relevant information before reacting (Aron, 2007; Bari & Robbins, 2013). Furthermore, this executive system is unable to exert sufficient control over reward seeking behaviours. This imbalance results in an increased sensitivity to reward and an underestimation of the negative consequences associated with that reward (Casey et al., 2008). Because of this, adolescents tend to choose immediate rewards over long-term gains (i.e. have higher impulsive choice) (Reyna & Farley, 2006). The noted decrease in impulsivity from adolescence into adulthood coincides with the maturation of the prefrontal cortex (Giedd et al., 1999; Luna & Sweeney, 2004; Luna et al., 2001; Rubia et al., 2000; Sowell et al., 1999; Steinberg, 2008). Differing rates of these maturational processes might explain why younger individuals are significantly overrepresented in collision statistics (Glendon, 2006, 2011). Furthermore, greater impulsivity in males overall (regardless of age) could account for why

males are involved in proportionately more fatal collisions while driving than females (Cross et al., 2011).

The current study utilised tasks known to elicit activation from both the reward processing and behavioural inhibition systems of the brain in an effort to measure impulsive action and impulsive choice (the IGT, the MID task and the Go/No Go tasks). Younger (under 25) and older (over 29) male drivers, as well as young female drivers, were divided into 'risky' and 'safe' driver groups based on their self-reported history of risky driving behaviours. These groups were then compared on their performance on these tasks, self reported impulsivity (the BIS-11 and Kirby's DDQ) and driving tendencies (the Manchester DBQ). In addition, fMRI and MRI were employed to determine whether there were any structural or functional differences in the neural correlates of impulsivity between risky and safe drivers. Therefore, this research aimed to determine whether impulsivity may be a vulnerability marker for aberrant driving behaviours.

2 Chapter Two: Differentiating Between Risky and Safe Drivers

2.1 Introduction

The current thesis examined the relationship between risky driving and impulsivity. Male drivers and drivers under the age of 25 are overrepresented in fatal collision statistics (Sarma, Carey, Kervick, & Bimpeh, 2013), however, not all young and male drivers will take risks (Sercombe, 2014). It was necessary, therefore, for the current study to differentiate between drivers with and without a propensity towards risk-taking in order to investigate whether risky drivers have higher impulsivity than safe drivers.

The current chapter explores the relationship between self-report and objective measures of risky driving in an effort to provide criteria for differentiating between safe and risky drivers. The following sections outline the literature on the validity of self-reported collision involvement, the relationship between penalty points and driving risk and questionnaires for the measurement of risky driving tendencies.

Drivers were characterised as 'risky' in the current study if they reported being culpable in at least one collision and/or currently had penalty points on their driving licence. Subsequent chapters will use these criteria to compare 'risky' and 'safe' drivers on various measures of impulsiveness to determine whether increased impulsivity may be a vulnerability marker for engagement in aberrant or problematic driving.

2.1.1 Self-reported collision involvement

Self-reported collision involvement is subject to certain bias, such as memory loss, over-reporting and selective under reporting (af Wåhlberg, 2009; af Wåhlberg, Dorn, & Kline, 2011). For example, it is estimated that drivers forget approximately one third of minor collisions per year and 80% of minor near misses after as little as two weeks (Chapman & Underwood, 2000). However, collisions where the individual was culpable are least likely to be forgotten (Chapman & Underwood, 2000). While recall declines with time, studies have shown that reliable recall of collisions is retained for up to four years (Y. Harel et al., 1994; Warner, Schenker, Heinen, & Fingerhut, 2005).

Despite these concerns, agreement between self-reported collisions and official records is reasonably high (D. J. Begg, Langley, & Williams, 1999; McGwin, Owsley, & Ball, 1998). For example, Boufous et al. (2010) contacted 17 to 24 year old drivers a year after that had been involved in a collision and found that 85% of crashes were accurately recalled (matched with police records). Roberts, Vingilis, Wilk, and Seeley (2008) compared official collision statistics from Canada's Traffic Accident Information Database (TRAID) to data collected as part of a longitudinal, population-based survey, the National Population Health Survey (NPHS). The results of this study suggested that self-reported collision involvement was nationally representative of the prevalence of collisions in Canada during this time. This relationship has also been observed between self-report, police records, clinical data (hospital discharge files; D. J. Begg et al., 1999) and state records (Marottoli, 1997). These studies concluded that self-reported collision involvement may provide a reasonable alternative to official reports (D. J. Begg et al., 1999; Marottoli, 1997; Roberts et al., 2008).

It is acknowledged too that official reports of collisions, such as police reports, hospital data, insurance data, as well as fleet data from professional drivers, can be subject to bias (De Winter & Dodou, 2012) and may even have a higher degree of variance than self-reported data (af Wåhlberg & Dorn, 2012). There can be a high degree of inconsistency in registering practices over time and by region (De Winter & Dodou, 2012). Fatalities resulting from collisions are better operationally defined and, as such, fatality databases are more readily available and widely cited than records of injuries (Roberts et al., 2008). Injury data, on the other hand, has been found to be subject to both over and under reporting (Aronoff et al., 2007; and see meta-analysis by Elvik & Mysen, 1999). Injury data is typically based on notes made by police at the scene of collision or hospital records (Roberts et al., 2008). However, drivers may be motivated not to report such collisions to the police or to their insurance company, for example, to avoid accountability or to protect their no-claims bonus (O'Day, 1993; as cited in De Winter & Dodou, 2012, p. 87). Hence, it appears that all methods of recording collisions are subject to some level of bias.

As it was beyond the scope of the current study to recruit participants based on official reports, self-reported collision involvement was utilised as this has been recognised as a reasonable alternative (D. J. Begg et al., 1999; Marottoli, 1997; Roberts et al., 2008).

2.1.2 Penalty points

Certain countries implement a points system to reduce unsafe driving by punishing drivers for illegal offences, such as speeding and braking traffic lights (af Wåhlberg,

2011). Depending on the country, these points are referred to as 'penalty' points (Gras, Font-Mayolas, Planes, & Sullman, 2014) or 'demerit' points (Ball, Kirkpatrick, & Brenneman, 2005). Over 100,000 penalty points are awarded in Ireland each year, more than half of which are given for exceeding the speed limit (57%) (RSA, 2015b). Other common offences in Ireland are driving on/across a footpath (16.5%), using a mobile phone while driving (8%), not wearing a seatbelt (6%) and failure to obey traffic signals (3%) (RSA, 2015b). The accumulation of a certain number of points will lead to the revocation of a driving licence for a fixed period of time (af Wåhlberg, 2011; Gras et al., 2014). In Ireland, 12 points results in a driving ban of three years (RSA, 2015b).

The introduction of penalty points has been attributed to the reduction of collisions in certain countries, such as Spain, Ireland, France, Czech Republic, Germany and Italy (Izquierdo, Ramírez, McWilliams, & Ayuso, 2011). For example, a 9% decline in collisions, and 30% in fatal collisions, was observed between 2001 and 2005 in Italy after the introduction of a penalty point system (De Paola, Scoppa, & Falcone, 2013). Increased surveillance measures, sanctions and the publicity campaigns that typically go along with the introduction of penalty points are thought to contribute to these positive effects (Izquierdo et al., 2011).

Despite the apparent effectiveness of penalty points as a deterrent for engagement in unsafe driving on a national level, individuals who receive penalty points are thought to be at increased risk for future collision involvement (Izquierdo et al., 2011; Orsi, Marchetti, Montomoli, & Morandi, 2013) as the accumulation of such points are considered to be indicative of a pattern of high risk behaviour (Ball et al., 2005; Lansdown, 2012). In a large-scale, longitudinal study by Lawpoolsri, Li, and Braver

(2007), drivers who had received penalty points for speeding at the beginning of the study were almost twice as likely to have received further penalty points for speeding by the follow up. Moreover, these drivers were more likely to be either male or under the age of 25 (Lawpoolsri et al., 2007). Lawpoolsri et al. (2007), therefore, concluded that penalty points are not very effective at deterring individuals who are predisposed to speeding from doing so, suggesting that they may be a useful indicator of risky driving propensities. This, in addition to the observation that penalty points are a good predictor of future crashes (Izquierdo et al., 2011), and are more prevalent than collision involvement, has lead some to propose that they may be used in place of, or in addition to, collision involvement as a proxy variable for 'risky driving' in individual difference studies (af Wåhlberg, 2009).

2.1.3 Classification method: identifying risky drivers

Age and gender are consistently identified in the literature as key variables that contribute to both ordinary and fatal collision involvement (Sarma et al., 2013), with male drivers more likely to be involved in a collision than females and drivers of both genders under the age of 25 more likely to be in a collision than middle-aged drivers (Gwyther & Holland, 2012; Lonczak, Neighbors, & Donovan, 2007; Oltedal & Rundmo, 2006; Westerman & Haigney, 2000). Furthermore, research has observed that males (compared to females) and younger drivers (compared to older) self-report engaging in significantly more risky behaviours while driving, such as speeding, not wearing seatbelts and driving under the influence of alcohol (Sarma et al., 2013). Despite this, not all males and younger drivers engage in high levels of risk-taking (Sercombe, 2014), as such, the current study utilised certain criteria to differentiate between

those likely to be risk takers or not in order to investigate whether increased impulsiveness contributes to risky driving tendencies.

Different classification methods have been employed by the research. Non-offenders have been compared to; DWI (driving while intoxicated) offenders (Bouchard, Brown, & Nadeau, 2012), drivers convicted of speeding (O'Brien & Gormley, 2013) and drivers attending penalty courses that are offered instead of penalty points or fines for dangerous driving offences (Lev, Hershkovitz, & Yechiam, 2008). Risky and safe driver groups have even been devised based on their ratings of driving scenarios (Guppy, 1993) and scores on psychometric measures, such as driver behaviour questionnaires (Ba, Zhang, Peng, Salvendy, & Crundall, 2015; Lucidi et al., 2010).

However, the current study identified risky drivers as those who were culpable in a collision and/or currently had penalty points on their driving licence, while safe drivers had neither criterion. These criteria have previously been used in studies such as af Wåhlberg (2010), Cheng, Ng, and Lee (2012), af Wåhlberg (2011) and af Wåhlberg (2009), where drivers were not informed which group they belonged to. Penalty points are considered a useful indicator of past and future engagement in risky driving and are occasionally used in place of collision involvement in driver behaviour research (af Wåhlberg, 2009; Izquierdo et al., 2011; Lawpoolsri et al., 2007). Furthermore, while recall of collision involvement has been found to decrease with time (af Wåhlberg et al., 2011), research suggests that drivers are more likely to remember collisions for which they were culpable (Chapman & Underwood, 2000; Spitzenstetter & Lasman, 2009). As the current study was interested in intentional risky driving, rather than driving error, it was important to differentiate between participants who were actively involved in a collision from those who were not at

fault (af Wåhlberg, 2012; af Wåhlberg, 2009; Fernandes, Hatfield, & Soames Job, 2010; Parker, McDonald, Rabbitt, & Sutcliffe, 2000). Thus, only drivers who were culpable in a collision were included in the risky group of the current study. In addition, the number of collisions was not recorded, as previous research has suggested that collision involved versus not involved is a sufficient criteria for risk (af Wåhlberg, 2010, 2011; af Wåhlberg, 2009).

2.1.4 Measuring driving risk

2.1.4.1 Driving questionnaires

Driver behaviour questionnaires are very popular within traffic research as they are cheap, quick to administer and are the easiest was of gaining a snapshot of how drivers typically behave (af Wåhlberg et al., 2011). Several questionnaires have been developed to measure different behaviours related to driving, for example, perceived driving competence (Driving Skills Questionnaire; McKenna, Stanier, & Lewis, 1991), frequency of overt travel avoidance (Driving and Riding Avoidance Scale; Stewart & St. Peter, 2004), excessive precautions when travelling (Safety Behaviour Questionnaire; Ehring, Ehlers, & Glucksman, 2006), travel anxiety (Fear of Driving Inventory; Walshe, Lewis, Kim, O'Sullivan, & Wiederhold, 2003) and anger across various driving situations (Driving Anger Scale; Deffenbacher, Oetting, & Lynch, 1994).

Within the measures that specifically assess aberrant driving behaviours, af Wåhlberg et al. (2011) reported that most of these are yet to be validated as they are constructed for use on a single study or for exclusive use by a particular research group, such as the Driving Habits Questionnaire (Schwebel, Severson, Ball, & Rizzo,

2006), the Driving Behavior Rating Scale (Barkley, Murphy, Dupaul, & Bush, 2002), the Driving History Survey (Knouse, Bagwell, Barkley, & Murphy, 2005) and a number of others without specific names (Chou, Miyazaki, & Maruyama, 1994; Larson & Merritt, 1991; McGuire, 1972; D. I. Smith & Kirkham, 1982). However, the Manchester Driver Behaviour Questionnaire (DBQ; Reason, Manstead, Stradling, Baxter, & Campbell, 1990) has gained the most acceptance in the literature and has been the most widely used measure of aberrant driving to date (af Wåhlberg & Dorn, 2012; af Wåhlberg et al., 2011; Blockley & Hartley, 1995; De Winter & Dodou, 2010; Lajunen & Summala, 2003; Mattsson, O'Brien, Lajunen, Gormley, & Summala, 2015; Özkan, Lajunen, Chliaoutakis, Parker, & Summala, 2006; Parker, Reason, Manstead, & Stradling, 1995; Rimmö & Åberg, 1999; Sullman, Meadows, & Pajo, 2002; Westerman & Haigney, 2000). Hence, the current study employed the Manchester DBQ to aid in the validation of the criteria for risky drivers.

2.1.4.2 The Manchester Driver Behaviour Questionnaire (DBQ)

The original DBQ, developed by (Reason et al., 1990), contained items that measured errors (mistakes) and violations (deliberately breaking the law), both thought to impact on driver safety (Lajunen, Parker, & Summala, 2004). Since its introduction, additional items have been added to differentiate 'errors' into lapses and errors, and 'violations' into aggressive violations and highway-code (HC) violations (Lawton, Parker, Manstead, & Stradling, 1997; Parker et al., 1998). Lapses reflect behaviours associated with memory and attention problems, while errors include more serious mistakes such as failures of observation and misjudgements (Lajunen & Summala, 2003). Both aggressive and HC violations refer to a tendency to deliberately break the law, however HC violations are associated with behaviours that gain the driver some

kind of advantage, such as speeding or overtaking, while aggressive violations are more hostile in nature, such as forcing one's way on to a major from a minor road (Lawton et al., 1997).

The latest version of the DBQ, by Parker et al. (1998), has been utilised extensively in driver safety research in areas such as the genetics of driving behaviour (Bianchi & Summala, 2004), age differences in driving behaviour (Dobson, Brown, Ball, Powers, & McFadden, 1999) and associations with the likelihood of being involved in an accident (Dobson et al., 1999; Mesken, Lajunen, & Summala, 2002; Parker et al., 1995; Reason et al., 1990). The DBQ has been reported to have good reliability (Sundström, 2008) and the current, four factor structure of the DBQ has been replicated over multiple populations (Özkan et al., 2006), including cross-culturally (Bianchi & Summala, 2004; Dobson et al., 1999; Lajunen et al., 2004; Mesken et al., 2002; Parker et al., 2000; Sullman et al., 2002; Xie & Parker, 2002).

The validity of the DBQ has been questioned by some. af Wåhlberg and Dorn (2012) claimed that the DBQ lacks content validity as it does not contain items relating to medical and psychiatric disorders, for example, which have been observed to predict future accident involvement. However, De Winter and Dodou (2012) refuted this opinion, reiterating that it is the purpose of the DBQ to capture how a driver would typically behave, and that individuals who suffer from such disorders will be identified because they are likely to score higher on the lapse and error subscales of the DBQ. While the DBQ, like most psychometric measures, can be influenced by a degree of social desirability (af Wåhlberg, 2010), it has been found to correlate with objective measures of risky driving, such as speeding (De Winter & Dodou, 2012). For example, Åberg and Wallén Warner (2008) found that measurements of speed taken

from field tests positively correlated with the HC violations subscale of the DBQ. Subscales of the DBQ have also bee observed to predict collision involvement, however there is some debate over which subscales serve as the best predictor (De Winter & Dodou, 2010). The ability to significantly predict collisions has been found for the HC violations subscale by Özkan and Lajunen (2005), Davey, Wishart, Freeman, and Watson (2007) and Stradling, Parker, Lajunen, Meadows, and Xie (1998), and for the error subscale by DeLucia, Bleckley, Meyer, and Bush (2003), Freeman, Wishart, Davey, Rowland, and Williams (2009) and Sümer (2003), while others have found that both (Sümer, 2003) and neither (Blockley & Hartley, 1995; De Winter & Dodou, 2010) significantly predicted collision involvement. Furthermore, it is thought that the relationship between DBQ scores and collisions is independent of distance travelled (af Wåhlberg, 2009; De Winter & Dodou, 2012).

While the above studies used objective measures of collision involvement, such as police reports, the most common method employed by the literature is the comparison of DBQ scores and self-reported collision involvement. A meta-analysis by De Winter and Dodou (2010) revealed a consistent, strong relationship between self-reported collisions, both prospectively and retrospectively, and all subscales of the DBQ. Furthermore, De Winter and Dodou (2010) found that adding additional variables, such as miles travelled, to regression models does not significantly improve their predictive power. However, af Wåhlberg et al. (2011) warned that these results should be interpreted with caution, as the DBQ is likely to be influenced by Common Method Variance (CMV). CMV is the term used to describe a tendency to be consistent across related, self-report measures, which may artificially inflate correlations between such measures (Podsakoff, MacKenzie, Lee, & Podsakoff, 2003). A solution to

this issue was proposed by af Wåhlberg et al. (2011), who suggested that the DBQ will be less susceptible to CMV in drivers that report collisions in which they were culpable, as collision culpable drivers have been observed to score higher on the DBQ than collision non-culpable drivers (for example see Parker et al., 2000). Therefore, the current study included only drivers who were culpable in a collision in the risky driving group.

Finally, as younger individuals and males are overrepresented in damage only, injury and fatal collision statistics, it is of relevance to note that both violations and errors have been found to decline with age (De Winter & Dodou, 2010) and that males and females demonstrate diverging patterns of responding on the DBQ. Previous research has consistently observed that females report more errors and lapses on the DBQ than males, while males report a greater number of violations (for example; Berdoulat, Vavassori, & Sastre, 2013; De Winter & Dodou, 2010; Reason et al., 1990).

2.1.5 Summary and Hypotheses

2.1.5.1 Validating the criteria for driving risk

To summarise, risky drivers in the current study were those who had accrued penalty points and/or had been collision culpable, while safe drivers had no history of aberrant driving behaviour. To assess the appropriateness of these criteria for differentiating risky and safe drivers in the current study, respondents to an online survey were divided into groups based on their self-reported driver history and their scores on the subscales of the DBQ were compared. It was hypothesised that, independent of age and gender, risky drivers would report more errors, lapses and violations than safe drivers.

2.1.5.2 Other hypotheses

As age and gender are commonly identified by the literature as factors that effect collision involvement (Sarma et al., 2013), gender and age differences in responding on the DBQ were also investigated by the current study. Consistent with previous research, it was predicted that the number of aberrant behaviours reported on the DBQ would decline with age. Furthermore, it was expected that males would report significantly more violations than females, while females would report more errors and lapses.

2.2 Method

2.2.1 Design

An online survey was used as a screening tool for the main neuroimaging study. The survey measured several demographic factors including gender and age, as well as driving factors such as licence type, driving frequency, whether they had accrued penalty points, whether they had been collision involved and follow up questions on these. It contained an MRI screening questionnaire to determine participant's eligibility for the main experiment. The survey also collected details on nationality, education and handedness to ensure that there would be equal distributions of these across experimental groups. Finally, the survey contained the DBQ. The survey did not screen for other risk behaviours, such as alcohol or drug use.

In the main experiment participants were assigned into groups based on their driver history, gender and age. The following section assesses the validity of the criteria used to differentiate risky and safe drivers by comparing their responses to the DBQ.

2.2.2 Apparatus and materials

2.2.2.1 Online Hosts

A website dedicated to the study was created (see appendix B). This website was hosted by wix.com and contained information about the study, as well as a survey for interested parties to complete. The survey, hosted by kwiksurveys.com, in addition to screening participants for MRI eligibility, contained a number of demographic questions in order for the researcher to target participants that met the criteria for the experimental groups (see appendix C). Survey data were downloaded in Microsoft® Excel® (2011, Version 14.4.1) format from kwiksurveys.com. Responses were coded to match labelling in an SPSS 21.0 (Statistical Package for the Social Sciences by IBM-Corp, Released 2012) document and then transferred into this document.

2.2.2.2 Psychometrics: The Manchester Driver Behaviour Questionnaire (DBQ)

In an effort to validate the risk status of the participants in the main study, the online survey, in addition to screening for MRI eligibility, contained the 28-item version of the DBQ (see appendix D) by Parker et al. (1998).

Through principal components factor analysis, Parker et al. (1998) confirmed that the factor structure of the 28-item version resembled that of the original 50-item version by Lawton et al. (1997), in that it contained four subscales: error, lapse and violations; aggressive violations and HC violations. These subscales demonstrated good reliability, with Cronbach's alphas above .7.

The eight items within the 'error' subscale cover mistakes made while driving that are not in breach of the law (e.g. backing into something you had not seen). On the other hand, the eight 'lapse' questions deal with loss of concentration or forgetfulness (e.g. realising that you have no clear recollection of the road along which you have just been traveling). Violations were behaviours in which the driver breaking the law or deliberately driving dangerously; six items covering aggressive violations (e.g. getting involved in unofficial 'races' with other drivers) and six HC violations (e.g. disregarding the speed limit on a motorway).

2.2.2.3 Statistical analysis

Data preparation and analysis were conducted using Microsoft Excel® (2011) and SPSS.

2.2.3 Participant recruitment

The study's website was advertised through a variety of methods. The link was posted on online forums, including Dublin's craigslist, boards.ie, Reddit, thedublinforums.com and Gumtree (see appendix Ea). It was also featured on the Facebook pages of the Road Safety Authority (RSA) and Mondello Park Racecourse (see appendix Eb). An email campaign containing the link targeted Irish Universities, namely Trinity College Dublin (TCD), University College Dublin (UCD) and the University of Limerick (UL) was also used (see appendix Ec). Finally, it also appeared on posters throughout Trinity College and in the following NCT test centres: Arklow, Deansgrange, Fonthill, Greenhills, Naas and Northpoint (see appendix Ed).

2.2.4 Participants

774 car drivers completed the online survey in full: 449 Males and 325 females, 689 of which held a full driving licence. Participants ranged from 17 to 65 years of age (M=25.22, SD=9.0).

103 participants reported having penalty points ranging from one to 12 (M=2.91, SD=1.698). For a breakdown of the offences for which penalty points were awarded see table 2.1 below. 145 respondents reported being responsible for a collision, of which 33 had been collision culpable and had penalty points. For a breakdown of the types of collisions see table 2.2.

The highest level of education participants had reached was also recorded in the online survey (see appendix F). Highest level of education completed was controlled for in the main study by ensuring that there were equal proportions of each level across the experimental groups.

Table 2.1: Offences for which penalty points were awarded

Description:	**Number**
Speeding	79
Driving a vehicle while holding a mobile phone	9
Failure to obey traffic lights	5
Careless Driving	4
Failure to comply with traffic lane markings	3
Driving while intoxicated	2
No safety belt – driver	1
Failure to stop before a stop sign/stop line	1
Failure to yield right of way	1
Driving without a valid tax certificate	1

Table 2.2: Details of collisions participants were culpable for

Description:	**Number**
Collision between two or more cars	85
Single car collision	56
Collision resulting in minor property damage	55
Collision resulting in minor injury	25
Collision resulting in serious property damage	13
Collision resulting to injury to driver	11
Collision resulting in injury to passenger	4
Collision resulting in serious injury	3
Collision resulting in fatality	2
Collision resulting to injury to pedestrian/cyclist/other	1

2.2.5 Procedure

Interested participants were directed to visit the study's website. The website contained a link to a survey where individuals could sign up to participate in the main experiment. Participants were informed before beginning the survey that they would only be eligible for the €20 compensation if they participated in the second part of the experiment.

The online survey contained an information and consent page, a demographics questionnaire, the Manchester DBQ and a standardised MRI safety-screening questionnaire in order to assess whether the individual meets any of the exclusion criteria for the risks associated with the scanner. They were then debriefed and given the opportunity to submit their email address if they wished to be contacted about participating in the main experiment.

Participants were designated as risky drivers if they had one or more penalty points on their driving licence, and/or had been culpable for a collision. The validity of such criteria was determined by comparing risky and safe groups on their responses to the four subscales of the DBQ. Raw DBQ scores were entered into SPSS and values were summed to get totals for the subscales. The data was analysed using a Factorial Analyses of Covariance (ANCOVA) model, discussed in more detail in the results section.

2.3 Results

Factorial Analyses of Covariance (ANCOVA) were carried out on each of the four subscales of the DBQ (lapse, HC violations, aggressive violations and error) for all respondents to the online survey. The model contained 'risk status' (risky x safe) and gender (male x female) as between group variables and age as a covariate.

Table 2.3: Summary of the main effects of Risk Status on the four DBQ subscales

Subscale	**ANOVA**
Lapse	$F(1,689)=13.92$, $p<.05$, $\eta^2=.02$
	Risky (M=16.91, SD=5.25) **> Safe** (M=15.69, SD=4.68)
Violation HC[5]	$F(1,689)= 41.28$, $p<.05$, $\eta^2=.057$
	Risky (M=14.9, SD=5.09) **> Safe** (M=11.99, SD=4.21)
Violation Agg	$F(1,689)= 22.14$, $p<.05$, $\eta^2=.031$
	Risky (M=10.95, SD=3.78) **> Safe** (M=9.28, SD=2.98)
Error	$F(1,689)= 13.59$, $p<.05$, $\eta^2=.019$
	Risky (M=12.84, SD=4.06) **> Safe** (M=11.97, SD=3.39)

[5] Homogeneity of variance was not found for this subscale

Table 2.4: Summary of the main effects of gender on the four DBQ subscales

Subscale	**ANOVA**
Lapse	$F(1,689)=38.48$, $p<.05$, $\eta^2=.053$
	Females (M=17.46, SD=5.07) **> Males** (M=14.97, SD=4.43)
Violation HC[6]	$F(1,689)= 8.261$, $p<.05$, $\eta^2=.012$
	Males (M=13.43, SD=4.77) **> Females** (M=11.91, SD=4.33)
Violation Agg	$F(1,689)= 12.25$, $p<.05$, $\eta^2=.017$
	Males (M=10.18, SD=3.49) **> Females** (M=9.1, SD=2.91)
Error	$F(1,689)= 8.561$, $p<.05$, $\eta^2=.012$
	Females (M=12.71, SD=3.76) **> Males** (M=11.82, SD=3.45)

The analyses found main effects of risk status and gender for all subscales of the DBQ. The four analyses are summarised in table 2.3 and 2.4, with the main effects of risk status summarised in table 2.3, and the main effects of gender in table 2.4. A significant relationship between age and DBQ error scores[7] was also found, $F(1,689)= 8.561$, $p<.05$, $\eta^2=.012$, and no significant interactions between risk status and gender. Cronbach's alpha for the DBQ was found to be 0.869.

2.4 Discussion

2.4.1 Summary of findings

2.4.1.1 Validating the criteria for driving risk

To determine whether drivers who had been culpable in a collision and/or had accrued penalty points on their licence had a greater tendency to engage in aberrant

[6] Homogeneity of variance was not found for this subscale

[7] Follow up correlational analyses revealed that the strength of this relationship was as follows; r(698)= - .121, p<.001, two tailed

driving behaviours than those without, risky and safe driver groups were compared on their responses to the DBQ. In a large sample of drivers, the current study found this to be the case for all four subscales, with risky drivers reporting more errors, lapses, ordinary (HC) violations and aggressive violations than safe drivers. This was consistent with previous research which demonstrated higher scores on the DBQ are associated with penalty points (Davey et al., 2007) and collision involvement (Sümer, 2003). Thus, the criteria appeared useful in identifying individuals likely to engage in heightened risk-taking while driving and was retained for the main experiment, where risky and safe drivers were compared on various measures of impulsiveness.

2.4.1.2 Other hypotheses

Consistent with previous research, the current study found that females reported significantly more errors and lapses than males but males scored higher on both the violation subscales, aggressive and HC violations (Berdoulat et al., 2013; De Winter & Dodou, 2010; Reason et al., 1990). This illustrates different patterns of aberrant behaviours in males and females, which may explain why males are overrepresented in collision statistics (Sarma et al., 2013). As younger individuals are further overrepresented in collisions, the number of aberrant behaviours reported on the DBQ was expected to decline with age, in line with previous research (De Winter & Dodou, 2010). However, the current sample only demonstrated a decline in errors with age, as evidenced by a significant co-varying relationship between the two variables. This suggests that while errors, which are more likely to be affected by inexperience, decline with age, deliberate risk-taking, such as aggressive behaviour while driving, may be a trait that is independent of age.

2.4.2 Limitations and future directions

While af Wåhlberg et al. (2011) claimed that the DBQ should be less susceptible to common method variance (CMV) when drivers are reporting collisions that they have been culpable in, rather than just overall collision involvement, it is still possible that CMV could have produced an artificial effect, such that those who reported being collision culpable reported more aberrant driving behaviours on the DBQ out of a need to be consistent across measures. While CMV may have impacted responses to the DBQ in the current study, it is not thought to extend to measures of different constructs (Podsakoff et al., 2003) and, as such, the effects of CMV were not thought to have impacted on the results of the main study. Moreover, as subsequent measurements of impulsivity were obtained at a different time point to the survey, this could have protected against further potential effects of CMV.

Another issue with the current approach is that other types of risk-taking, e.g. misuse of drugs or alcohol, were not measured. As engagement in one type of risky behaviour is associated with an increased likelihood to engage in another (Dryfoos, 1991), it is unclear whether the risky drivers of the current study reflected those who would take risks across multiple domains, or whether their aberrant behaviours are limited to those while driving. Furthermore, the criteria used to differentiate between risky and safe drivers does not account for the possibility that some individuals may frequently engage in risk-taking while driving but may never have been involved in a collision. Such individuals may also not have received penalty points, as they had not been observed breaking the law while driving by the police. Furthermore, it is equally probable that an individual may have been involved in a collision due to lack of skill, rather than deliberate risk-taking. Therefore, a limitation of the current criteria was

that some drivers might have been classified as safe despite frequent engagement in risky driving behaviours. Future research could objectively measure driving behaviour over a fixed period of time to differentiate between risky and safe drivers. This could be achieved, for example, by using an in-car 'back box' device, such as those manufactured by ERM (2015), which record speed, harsh braking, fast lane changing, accident involvement and other types of reckless manoeuvres. As these observational techniques were beyond the scope of the current study, self-reported methods were used for classifying drivers. Due to this limitation, perhaps a more appropriate term for this group would be 'aberrant' or 'problematic' drivers. However, for the sake of convenience, and understanding the connotations of using this term, such drivers are referred to as 'risky' in the current study.

2.4.3 Conclusion

In conclusion, an evaluation of the appropriateness of the current criteria for identifying risky drivers passed reasonable scrutiny by comparing groups on their self-reported aberrant propensities. Therefore, the main study continued to classify risky and safe drivers based on these criteria in order to investigate whether such drivers may differ on various measures of impulsiveness, such as psychometric and behavioural measures, as well as on measurements of brain structure (using MRI) and function (using fMRI).

3 Chapter Three: General Method

3.1 Overall Design

The current study investigated the relationship between impulsivity, gender, age and risky driving behaviours. Several measures of impulsivity (psychometric, behavioural, as well as neuroimaging) were obtained and compared across six groups, characterised by unique combinations of age, gender and risk status, chosen to investigate why males, regardless of age, and younger individuals, regardless of gender, are overrepresented in collision statistics. This resulted in the following six experimental groups; young 'safe' females, young 'risky' females, young 'safe' males, young 'risky' males, older 'safe' males and older 'risky' males, where young individuals were aged between 18 – 24 and older males between 29 – 56.

An online survey was used to screen potential participants for Magnetic Resonance Imaging (MRI) eligibility. This survey also took a detailed driver history in order to determine whether individuals may be classified as 'safe' drivers or as those who take risks. Individuals meeting the study's criteria were recruited to partake in the main study, which employed Magnetic Resonance (MR) to image the brain while participants engaged with cognitive tasks of impulsivity.

As not of interest to the current hypotheses, older risky/safe females were not included in the study. As this meant that the study lacked a fully nested design, it necessitated the use of two analytical models; firstly that the DVs were compared in 2 x 2 Factorial ANOVAs for gender and 'risk status' (risky x safe drivers) across all younger individuals and, secondly, 2 x 2 Factorial ANOVAs for risk status and age (young vs. old) across all males.

3.2 Participants

The target number of participants for the current study was 120. Of the 774 respondents to the online survey, 499 did not meet any of the MRI exclusion criteria[8]. Eligible participants who also met the criteria for age, gender and risk status were contacted about participating in the main study. 316 individuals were contacted before the initial 120 scans could be collected. Testing sessions lasted an hour and forty minutes; an hour scan, with forty minutes of preparation beforehand. All participants were compensated €20 for their time.

3.2.1 Discarded and replaced participants:

After preprocessing[9] three participants were dropped from the study as their imaging data proved unsuitable for analysis. Participant number 100 (safe male driver, 45 years of age) was discarded due to the presence of an artifact in the frontal lobe that prevented this participant from being normalised to Montreal Neurological Institute (MNI) space (see appendix G). Two further female participants (both aged 20 and belonging to the risky and safe groups, respectively) were discarded because it was determined that there was excessive scan-to-scan motion in more than ten per cent of their scans across the three tasks (in accordance with best practice: see Lockwood et al., 2013; McCrory et al., 2011; Sebastian et al., 2012).

To ensure that there were 20 participants in each of the six experimental groups three new participants, matched for age, gender, risk status and the other demographic factors mentioned in the previous section, were recruited and tested to

[8] Which included screening for a history of neurological, psychiatric and psychological illness.

[9] For information on preprocessing steps see section 5.2.5.1

replace those that were discarded. These replacements passed all of the necessary quality checks during preprocessing, leaving the study with a total of 120 participants for analysis.

3.2.2 Breakdown of demographics for final 120 participants:

All volunteers had normal or corrected to normal vision, were residents of Ireland and spoke fluent English (92.5% had English as their first language). The majority of individuals were right handed (105), 13 were left-handed and two were ambidextrous. Overall, 58 participants had completed secondary level education, 21 had a certificate or ordinary level degree, 28 an honours undergraduate degree and 13 had completed a postgraduate course (either a Masters or PhD).

While there was large variability in the amount of driving participants did, 90.8% reported driving at least once a week with a mean distance driven annually of 9383.95 (SD=12484.717) kilometres. Approximately half of participants owned their own car (50.8%) and 20% of the cars driven were bought new. Table 3.1 lists the cars that participants drove. Categories A to F were devised as an indirect measure of SES (socioeconomic status), such that with each increasing category the associated cost of purchasing the vehicle first hand increased.

Drivers were characterised as either risky or safe, with participants assigned to the risky group if they met at least one of the following criteria: had at least two penalty points; were culpable in a collision that resulted in a minimum of minor damage to property. Based on these criteria 60 participants were assigned to the risky driver group and 60 to the safe driver group. These groups, in turn, were further subdivided into groups based on a combination of age and gender factors: young male drivers

(aged 18-24), young female drivers (aged 18-24) and older male drivers (aged 29-56). This division resulted in a total of six groups, each containing 20 participants. A more detailed breakdown of participants' ages can be found in table 3.2. Groups were matched for education, handedness, SES factors (determined by occupation, parent's occupations, parent's education and car driven) and the number of kilometres driven annually.

Table 3.1: Description of the cars participants drove

	Description	**Example**	**Engine size (L)**	**Cost (€) (in 1,000s)**	**Number**
A	Compact/Small/City car	Fiat 500	0.9-1.4	11 – 18	47
B	Medium/Midsize car	Ford Focus	1.4-2.0	17 – 30	36
C	Large car/Family car	Mazda 6	2.0 – 3.0	22 – 39	24
D	SUV/4 x 4	Jeep Wrangler	1.5 – 6.4	27 – 50	8
E	Executive car	BMW 3 Series	2.6 – 6.1	35 – 70	2
F	Luxury car/convertible	Jaguar XK	2.5 – 6.5	40 – 100	3

Twenty-nine individuals reported having penalty points on their driving licences, seven young females, nine young males and 13 older males. These young females had between two and three penalty points (M=2.29, SD=0.488), young males between two and four (M=2.78, SD=0.833) and older males between two and 12 (M=3.46, SD=2.727). A breakdown of the types of penalty points individuals had can be found in table 3.3.

Table 3.2: Breakdown of age by experimental group

Experimental group	Age range	Age mean	Age SD
Young safe males	18 – 24	20.65	2.159
Young risky males	19 – 24	20.35	1.348
Young safe females	18 – 23	20.45	1.395
Young risky females	18 – 23	20.55	1.356
Older safe males	29 – 52	38.65	7.761
Older risky males	30 – 56	38.30	8.591

Table 3.3: Details of penalty points risky participants had

Description:	Young Males	Young Females	Older Males
Speeding	5*	6	12**
Careless Driving	1*	0	0
Driving a vehicle while holding a mobile phone	2	0	0
Offence related to overtaking	1*	0	0
Driving while intoxicated	0	0	1
Failure to obey traffic lights	0	1*	0

*= 1 of whom was also collision culpable; **= 3 of whom were also collision culpable

Thirty-eight individuals admitted being culpable for at least one collision, 14 young females, 14 young males and ten older males. Descriptions of the collisions participants were culpable for can be found in table 3.4[10]. Of these, three of the younger males, one younger female and three older males were both collision culpable and had penalty points. Participants that met both criteria for risk are indicated in tables 3.3 and 3.4.

[10] In the online survey participants were encouraged to select as many of the answers to this question as necessary to describe the collision they were involved in and so the total of each column in table 3.4 will not equal to total number of participants who were collision culpable.

Table 3.4: Details of collisions risky participants were culpable for

Description:	**Young Males**	**Young Females**	**Older Males**
Collision resulting in minor injury	1	3	3**
Collision resulting in serious injury	0	0	0
Collision resulting to injury to driver	0	1	0
Collision resulting in injury to passenger	0	0	0
Collision resulting to injury to pedestrian/cyclist/other	0	0	0
Collision resulting in fatality	0	0	0
Collision resulting in minor property damage	4	8*	6**
Collision resulting in serious property damage	1	0	2*
Single car collision	5** +	7^	3*
Collision between two or more cars	8^^	6*	9***

Punctuation indicates where individuals also had penalty points for: * = 1 x speeding, ** = 2 x speeding, *** = 6 x speeding, + = 1 x dangerous overtaking, ^ = 1 x breaking a red light, ^^ = 1 x dangerous driving

3.3 Apparatus and Materials

Participants were recruited through an online survey, hosted by kwiksurveys.com. This survey facilitated the screening of participants for MRI eligibility, as well as containing a version of the Manchester Driver Behaviour Questionnaire (Parker et al., 1998).

In the main experiment, impulsivity was measured both psychometrically and through participants' performance on cognitive tasks. Psychometric impulsivity was measured with Barratt's Impulsiveness Scale (BIS-11; Patton et al., 1995) and Kirby's Delay Discounting Questionnaire (DDQ; Kirby & Maraković, 1996). Participants also completed three tasks while being scanned with functional MRI: the Iowa Gambling

Task (IGT; Bechara et al., 1994), a Monetary Incentive Delay task (MID; Nestor, Hester, & Garavan, 2010) and a Go/No Go task (Kaufman, Ross, Stein, & Garavan, 2003). These tasks assessed different facets of cognitive impulsivity and, therefore, facilitated the study of the neural correlates of impulsivity. For more specific details on the apparatus and materials used in this study, see chapters four to six.

3.4 Procedure

The researcher contacted eligible participants who had completed the online survey and arranged for them to come to Trinity College to participate in the main experiment. Individuals who met any of the MRI exclusion criteria (for example; if they had a history of stroke) were automatically excluded from the study. Risky and safe drivers were targeted based on the driving history they provided in the online demographic questionnaire.

When a scanning date was arranged a letter was sent to the participant's General Practitioner (GP) to notify them of the scan and of the possibility of being contacted by a Radiologist in the unlikely event that an abnormality is detected in the scan (see appendix H). This was per the policy in Trinity College's Institute of Neuroscience. Participants were notified of this policy in advance and their permission was obtained.

Testing took place in the Institute of Neuroscience, Lloyd Institute, Trinity College Dublin. Participants met the researcher in the Lloyd Institute and were taken to a testing room. They were fully briefed on the study's procedure, read the information sheet (see appendix I) and signed the consent form (see appendix J). They also read and signed a separate information/consent form about the potential risks associated

with entering the MRI scanner devised by Trinity's Institute of Neuroscience (see appendix K). This information/consent form also contained the list of exclusion criteria, which the researcher went through once again to ensure eligibility, and reminded them that their GP was informed that they were going for a scan. Once consent had been obtained, participants completed a further demographics questionnaire (see appendix L) and a pen and paper version of the BIS-11. Following these, a laptop was used to display the instructions and options for Kirby's DDQ, and participants recorded their selections on the answer sheet provided.

Upon completion of the psychometric measures, the researcher described, in detail, the scanning procedure. Participants were informed that the scan would last 55 minutes, with 15 minutes for structural scans, 35 minutes for the tasks and time set aside to set up each new task. Participants were warned that the scanner made a loud noise, and they would be provided with earplugs and headphones to minimise the effects of this noise. All participants were given the same instructions on how to complete the tasks and shown examples (see appendices M, N and O). All participants were compensated the same amount of money upon concluding the experiment (€20). Despite being a potential motivational factor, participants were not mislead to believe that their performance would determine the amount of money they received. Participants were made aware of this when they were instructed on the tasks. Next, they were shown an image of the response pad they would use while in the scanner and demonstrated which buttons they would need for each task (see figure 5.2 in chapter 5). Finally, it was impressed upon them the importance of maintaining a fixed position whilst in the scanner, and they were encouraged to make a particular effort to keep their head as still as possible for the entire session.

Participants were escorted down to the basement of the Lloyd Institute, where the MRI machine was located. They were met by the Radiographer in charge of the MRI machine, who went through all of the necessary safety checks before scanning. Sufficient time was allocated to make the participants comfortable in the scanner before beginning the experiment. They were shown the buttons they would be using for the tasks and an emergency bell was pinned to their clothing. At this point they were told of the intercom system linking the observation room to the headphones they would be wearing.

The clinical and anatomical images were collected in the first 15 minutes of each session. Next participants completed one run of the IGT (15 minutes) and two runs of each of the MID (4.83 minutes) and XY (4.4 minutes) tasks. Before attempting each task participants were reminded of the instructions and the buttons they would need over the intercom system. Participants' responses were monitored from the observation room to ensure that they were engaging with the task correctly. They were provided with positive encouragement over the intercom between runs in an effort to sustain engagement with the second run. On a rare number of occasions the scan was aborted and restarted. This only occurred if the researcher observed that a participant was responding in a way that suggested that they either forgot or did not understand the instructions to the task. The researcher would reinstruct the participant before recommencing the scan.

The tasks were presented in a pseudo-randomised order. Since the IGT was performed on an external laptop it had to occur either first or last in the task running order. Every effort was made to randomise whether the IGT came first or last and,

once its position had been decided upon, the order of the other two tasks was randomised. As a result, there were four possible run orders (see appendix P).

Upon completion of the experiment participants were debriefed (see appendix Q). They were compensated €20 and signed a receipt as proof of compensation (see appendix R). As the final component of their compensation, participants were emailed .jpeg images of their brain later that day and thanked again for their participation.

4 Chapter Four: Psychometric and Behavioural Impulsivity in Drivers

4.1 Introduction

Adolescents and young adults are injured or killed while driving more than older drivers (Harré, 2000). While not all adolescents are risk takers, evidence suggests that those who engage in one type of risk-taking are more likely to engage in others (Dryfoos, 1991). One explanation proffered for increased risk-taking in some younger individuals is that they may have heightened impulsivity. Impulsiveness generally declines with age from adolescence to adulthood (Steinberg et al., 2008), however, this decline may occur at different rates in some individuals, making them more prone to taking risks than others.

Individual differences in subtypes of impulsiveness are thought to contribute to behaviour and thought processes (Aichert et al., 2012) and even to relate to other personality traits, such as sensation seeking and aggression (M. S. Stanford et al., 2009). Highly impulsive individuals have been found to be more likely to engage in risky behaviours, such as fighting, shoplifting, drug use, risky driving and self-harm (Chamorro et al., 2012). The current chapter explores the relationship between driving behaviours and impulsivity, gender and age. Impulsivity was treated as a multi-faceted construct, with several psychometric and behavioural measures taken.

4.1.1 Measures of impulsivity

4.1.1.1 Psychometric measures of impulsivity

Questionnaires are often used to measure impulsivity. Some of the questionnaires specifically designed to measure impulsivity are Dickman's Functional and

Dysfunctional Impulsivity Scales (Dickman, 1990), the Personality Research Form Impulsivity Scale (PRF; Jackson, 1984; designed to measure personality traits related to areas of normal functioning) and the EASI-III Impulsivity Scales (Buss & Plomin, 1975; based on their four temperament theory of personality: emotionality, activity, sociability and impulsivity). In addition to these questionnaires several other psychometric measures contain an impulsivity subscale so that impulsive tendencies can be compared to other personality traits. Examples of these are the Karolinska Scales of Personality (Ortet, Ibáñez, Llerena, & Torrubia, 2002), the Revised NEO Personality Inventory (NEO-PI-R; Costa & McCrae, 1992), the Temperament and Character Inventory, Revised (TCI-R; Cloninger, 1999), the Multidimensional Personality Questionnaire Control Scale (MPQ; Tellegen, 1982) and the I_7 Impulsiveness Questionnaire within Eysenck's Personality Inventory (Eysenck et al., 1984; designed to measure impulsiveness, venturesomeness and empathy).

Despite the wide range of psychometric tools available for measuring impulsivity the most commonly used questionnaires in the literature are the UPPS (Whiteside & Lynam, 2001) and Barratt's Impulsiveness Scale (BIS; Barratt, 1994). The UPPS was developed by Whiteside and Lynam (2001) to measure aspects of impulsivity across the Five Factor Model of personality (FFM; McCrae & Costa, 1990). It combines elements from several of the questionnaires mentioned above, including Dickman's Functional and Dysfunctional Impulsivity Scales (Dickman, 1990), the NEO-PI-R (Costa & McCrae, 1992) and the TCI-R (Cloninger, 1999), as well as borrowing from other personality measures such as Zuckerman's Sensation Seeking Scale (ZSSS; Zuckerman, 1994). The UPPS got its name from the initials of its four subscales: urgency, premeditation, perseverance and sensation seeking. Despite the exhaustive

and comprehensive method used to develop this questionnaire, and its popular usage in the literature, a meta-analysis by Coskunpinar, Dir, and Cyders (2013) found that studies have reported unstable and inconsistent findings when using the UPPS to predict future problem behaviours, such as alcohol use. Coskunpinar et al. (2013) observed that the UPPS produced unreliable results across studies, as simple sampling variations, and the variables controlled for in multiple regression models, impacted on findings. Therefore the current study used the BIS (Barratt, 1994) to measure trait impulsivity.

4.1.1.1.1 Barratt's Impulsiveness Scale (BIS-11)

As a psychometric measure of impulsivity, the 11th revision of Barratt's (1959) Impulsiveness Scale, by Patton et al. (1995) was used in the current study. This section presents the evidence that suggests that this measure is able to differentiate between healthy controls and certain clinical populations for which heightened impulsivity is a symptom. This section also reviews the findings for how responses on the BIS may change with age and may differ between males and females, however, a discussion of how the BIS may relate to aberrant driving will be reserved for section 4.1.2.

The authors developed the original measure based on four diverse perspectives: the medical model, the behavioural model, the psychological model and the social model. The development of the BIS was based on research incorporating self-report, cognitive and behavioural tasks (Barratt, 1994; Barratt, Stanford, Kent, & Felthous, 1997; Patton et al., 1995). The BIS-11 (Patton et al., 1995), was designed to measure impulsivity and similar personality traits, such as extraversion and sensation seeking. The BIS-11 is a 30 item measure, with questions pertaining to a range of traits and

behaviours associated with impulsiveness, all rated on a four-point Likert scale from 1 = rarely/never, to 4 = almost always. It has been found to have internal consistency ratings between 0.79 and 0.83 for populations ranging from healthy to those with psychiatric disorders, prison inmates and substance abuse patients (Whiteside & Lynam, 2001).

It is arguably the most widely used measure of impulsivity, and has had notable influence on they way impulsivity is conceptualised in psychology and psychiatry (M. S. Stanford et al., 2009). Patton et al. (1995) identified six distinct first order and three second order subscales. The three second order subscales are each subdivided in two to make the six first order subscales; attentional impulsiveness (made up of 'attention' and 'cognitive instability'), motor impulsiveness ('motor' and 'perseverance') and non-planning impulsiveness ('self-control' and 'cognitive complexity'). These subscales are said to measure distinct cognitive processes and are described in more detail in section 4.2.3.1.1 (M. S. Stanford et al., 2009).

The BIS has been used to investigate how impulsivity changes with age and also to interrogate potential differences in trait impulsivity between the genders and between healthy controls and certain clinical populations. Cross-sectional research by Steinberg (2010) demonstrated a decline in overall BIS with age from adolescence to adulthood. There is little consensus in the literature, however, as to whether there are differences in impulsivity between males and females. While many have reported no gender differences in either total BIS or its subscales (for example; Patton et al., 1995), one study reported higher total BIS in females (Aichert et al., 2012) and another found that males scored higher on the perseverance subscale only (first order subscale) (M. S. Stanford et al., 2009).

The BIS has been found to successfully differentiate between healthy controls and clinical populations (Spinella, 2007). Higher total BIS scores have been observed in habitual substance abusers (for example ecstasy users; Bond et al., 2004; and cocaine users; Lane et al., 2007), borderline personality disorder (Van Den Eynde et al., 2008), ADHD (Malloy-Diniz et al., 2007) and pathological gamblers (Rodriguez-Jimenez et al., 2006), compared to healthy controls, to name a few. Similarly, some clinical groups score higher than healthy controls on some of the subscales of the BIS, for example adults with a history of suicide attempts have been found to score higher on both the motor (Dougherty et al., 2004) and attention subscales (Quednow et al., 2006). Furthermore, in patients with bipolar disorder the subscales of the BIS have been found to relate to the different affective states of the disorder; motor subscale to manic episodes, non-planning to depressive episodes, and the attentional subscale to both manic and depressive episodes (Swann et al., 2008). Significant positive correlations have also been found between BIS subscales and risky behaviours. Higher scores on the attention subscale has been found to strongly associate with longer drinking sessions (Balodis, Potenza, & Olmstead, 2009) and physical aggression (Harmon-Jones, Barratt, & Wigg, 1997), non-planning to a greater number of daily cigarettes (Dom, D'Haene, Hulstijn, & Sabbe, 2006) and motor impulsivity to binge drinking (Carlson, Johnson, & Jacobs, 2010).

4.1.1.1.2 Kirby's Delay Discounting Questionnaire (DDQ)

Kirby's DDQ was included in the present study to measure impulsive choice tendencies in drivers. While there is consistent evidence to suggest that clinical populations, such as substance abusers, will display greater delay discounting on this measure, the evidence of gender and age differences is not coherent. These findings

will be discussed in greater detail below. Research that has used delay discounting measures to study impulsive choice in drivers is discussed in section 4.1.2.

The delay-discounting model of impulsiveness (Ainslie, 1975, 1992; Rachlin, 1992) refers the choice of a smaller, 'sooner' reward over a larger, 'delayed' reward and is a component of 'impulsive choice'. Delay discounting pertains to the reduction in the present, subjective value of a future reward as the delay to that reward increases (Kirby et al., 1999). The greater the delay, the lower the perceived value of the reward and the less likely that this option would be chosen against an immediate alternative. The 'discount rate' determines the steepness of the reduction in present value with increases in delay. Kirby and Maraković (1996) derived a questionnaire based on the premise that individuals have different discount rates, with higher discounting associated with a preference for immediate rewards, as delayed rewards are greatly devalued to them.

Kirby and Maraković (1996) developed the original 'monetary-choice questionnaire' and validated it in a 621 strong sample. Kirby's Delay Discounting Questionnaire (DDQ), as it is more commonly known, contains a fixed set of 27 choices between smaller immediate rewards (SIRs) and larger, delayed rewards (LDRs), for example, "Would you prefer €11 now or €30 in seven days?" Kirby and Maraković (1996) devised the following formula for discounting, which assumes that the rate at which a reward is discounted is inversely related to the delay: $V = \frac{A}{1+kD}$, where V represents the value of the immediate reward, A is the amount of the delayed reward and D is the delay (in number of days) until receipt of the reward. The 'k' value is the discount rate at which the immediate and delayed rewards are of equal value. Delayed amounts and times for delay are calculated based on the premise that the delay

makes the immediate and delayed rewards proportionately equal, therefore individual choices between immediate and delayed rewards speaks to their individual discounting rate. A positive relationship between delay discounting and BIS scores has been reported, such that higher levels of the non-planning subscale of the BIS-11 are associated with steeper delay discounting (Swann, Bjork, Moeller, & Dougherty, 2002).

The discounting rates of experimental groups can be directly compared as a measure of their impulsive choice, where a group that chooses more immediate rewards, and hence have a higher discounting rate, could be interpreted as displaying greater impulsive choice tendencies (Kim & Lee, 2011). For example, Fields, Sabet, and Reynolds (2013) found that overweight and obese individuals discounted the value of future rewards to a greater extend to those of a healthy weight, while there were no differences in BIS-11 score between these groups. Furthermore, a meta-analysis conducted by MacKillop et al. (2011) found that three quarters of studies using delay discounting measures found that addicted participants had a higher discounting rate than healthy controls (MacKillop et al., 2011). Groups that reliably demonstrate steeper delay discounting than controls are; cigarette smokers, pathological gamblers and substance users, such as those addicted to stimulants, opiates, marijuana and alcohol. Furthermore, the meta-analysis revealed that greater discounting rates are associated with more severe clinical symptoms (MacKillop et al., 2011).

Changes in delay discounting with age have also been investigated by the literature. Some studies have found a linear decrease in delay discounting with age from pre-adolescence to adulthood (Green & Myerson, 2004; Green, Myerson, Lichtman, Rosen, & Fry, 1996; Green, Myerson, & Ostaszewski, 1999), and even from early (age 19) to

later adulthood (age 91) (da Matta, Gonçalves, & Bizarro, 2012; Löckenhoff, O'Donoghue, & Dunning, 2011), while others have proposed a curvilinear relationship between age and discounting, with greater discounting occurring in mid adolescence (G. W. Harrison, Lau, & Williams, 2002; Read & Read, 2004; Steinberg et al., 2009). Alternatively, an absence of a relationship between age and discounting has also been reported (L. W. Chao, Szrek, Pereira, & Pauly, 2009).

Similarly, there is a lack of consensus regarding gender differences in delay discounting. While a few studies suggest that women may discount to a greater degree than men (R. C. Beck & Triplett, 2009; C. L. Smith & Hantula, 2008; Weafer & de Wit, 2013), there is also evidence to the contrary (Petry et al., 2002). Some studies have also found no differences in delay discounting rates between males and females (Cross et al., 2011; Kirby & Maraković, 1995). In an effort to address whether this discrepancy was the result of differences in engagement with the measure between males and females, studies have informed participants that they would be given one of their selections at random at the end of the experiment. These attempts were unsuccessful in disambiguating this effect, however, as some have found that women discount more steeply as a result (Reynolds, Ortengren, Richards, & de Wit, 2006) and others have found greater discounting in males (Kirby & Maraković, 1996). The current chapter hopes to address some of these discrepancies by compared delay discounting rates by age, gender and driving risk factors.

4.1.1.2 Psychometric versus behavioural measures of impulsivity

As heightened trait impulsivity can be associated with impaired cognitive ability (see Markus & Jonkman, 2007), behavioural tasks are being increasingly used to investigate the construct of impulsivity. The use of concurrent self-report and

behavioural measures allows research to make inferences about the cognitive processes and underlying neural correlates of impulsivity (Cyders & Coskunpinar, 2011). Just like the self-report measures of impulsivity, different experimental paradigms tap into distinct facets of impulsiveness (Aichert et al., 2012). In a meta-analysis conducted by Cyders and Coskunpinar (2011), it was found that behavioural and psychometric measures are related but measure distinct aspects of impulsivity. Cyders and Coskunpinar (2012) followed on from this by administering ten of the most frequently used measures (as determined by their meta-analysis) to 77 participants. Using Exploratory Factor Analysis (EFA), Cyders and Coskunpinar (2012), revealed some moderate overlap between the measures but largely observed that psychometric and behavioural measures loaded on to different factors, suggesting that they represent distinct constructs. Therefore a combination of these approaches should be used in order to measure this multi-faceted construct in a comprehensive way. The inclusion of both psychometric and behavioural measures of impulsiveness may also help resolve some of the issues of contention in the impulsivity literature, such as whether or not there are differences between the levels of impulsivity in males and females.

4.1.1.3 Behavioural measures of impulsivity

Behavioural tasks are used to measure the specific components of impulsivity, eliciting behaviours like inhibition, behavioural control, behavioural cancellation, reward assessment and the consideration of future consequences. These tasks recruit aspects of the impulsivity network and therefore can be used in the study of the neural correlates of impulsivity (Pietras et al., 2003). These tasks can be separated into those that measure impulsive action and impulsive choice behaviours.

Tasks that measure impulsive action typically require participants to make rapid decisions and to inhibit or cancel behaviours. Commonly used examples of these tasks are the Stop Signal Reaction Time Task (SSRT; Logan, Schachar, & Tannock, 1997), Go/No Go tasks (for example Kaufman et al., 2003; based on the Go/No Go paradigm by M. M. Mesulam, 1985), Information Sampling tasks (for example the IST by Clark, Robbins, Ersche, & Sahakian, 2006) and the Wisconsin Card Sorting Test (WCST; Heaton, Chelune, Talley, Kay, & Curtiss, 1993). Tasks that are used to measure impulsive choice contain a greater decision-making component or assessment of responses to rewarding stimuli. Participants usually have to make decisions about reward or discover the nature of a task. Examples of these tasks are the Continuous Performance Test (CPT; Epstein et al., 2003), the Iowa Gambling Task (IGT; Bechara, 2005) and the Delay Discounting Choice Task (DDCT; Richards, Zhang, Mitchell, & De Wit, 1999). One task of impulsive action (the Go/No Go task) and one of impulsive choice (the IGT) will be discussed in greater detail below. The Monetary Incentive Delay task (MID; Knutson et al., 2001) is also discussed as a measure of neural response to rewarding stimuli.

4.1.1.3.1 The Iowa Gambling Task (IGT)

The IGT was employed by the present study to measure decision-making processes in drivers. The following section describes the IGT, outlines how it is analysed and contains an overview of the findings of previous literature pertaining to how performance on the IGT changes with age and gender differences in IGT performance. In addition, this section lists many of the clinical populations for which differences in IGT performance, compared to healthy controls, have been found, as well as a review of how performance on the IGT may relate to trait impulsivity. Studies that have used

the IGT to investigate differences between aberrant and safe drivers will be discussed in section 4.1.2.

The IGT is a measure of risky decision-making developed by Bechara et al. (1994). Participants are successful in the task if they can forgo short-term benefits for long-term gains. Buelow and Suhr (2009) conducted a large study in the reliability and validity of the IGT across various clinical and non-clinical samples. They reported that the IGT is a true measure of decision-making in a real world setting and reported good reliability, provided that the IGT is not administered to the same participant multiple times.

With the IGT participants must choose between four decks of cards; A, B, C, and D (Bechara, Tranel, & Damasio, 2000). All of the decks offer an immediate reward (of varying magnitude) followed by either an immediate or delayed punishment, with some decks punishing more frequently and with a greater magnitude than others. Deck A offers large rewards and large, consistent punishments. The rewards of deck B are similar to the rewards from deck A, however the punishments are less frequent and, as a result, are much greater in magnitude. Over a period of ten clicks selections from decks A and B result in an net loss of $250, earning them the name 'disadvantageous decks'. Individuals who select from these decks are seen to lack the ability to delay gratification, they are drawn to the large rewards of these decks and discount the punishments. Decks C and D give participants significantly smaller rewards than decks A and B, however the punishments are also smaller. Deck C is associated with small rewards and smaller, consistent punishments while deck D delivers small rewards and slightly larger, delayed punishments. Despite the low value of rewards given from decks C and D, ten selections from them will result in an

net profit of $250. These decks are considered to be the advantageous decks and participants who make more selections from these take long-term gains into account more so than immediate rewards. Therefore, participants must deduce which of the decks are advantageous and which are disadvantageous in order to be successful in the task. Individual scores on the IGT are calculated by subtracting the number of disadvantageous deck selections from the number of advantageous ones. Individuals perform poorly in the IGT if they are drawn to the large rewards given by the disadvantageous decks and do not consider the heavy losses associated with them. These tasks assess how an individual makes decisions about rewards and the degree to which an individual values immediate rewards over future ones.

The IGT has been used in the literature to investigate changes in risky decision-making with age and between healthy controls and certain clinical populations. Successful performance on the IGT has been found to increase linearly with age from age nine to 17 (Hooper, Luciana, Conklin, & Yarger, 2004). Similarly, adults demonstrate better performance on the task than adolescents, learning about the nature of the task faster than adolescents by selecting fewer of the disadvantageous decks (Cauffman et al., 2010). While this suggests a shift in risky decision-making between childhood and adulthood, research has found no difference between younger and older adults, i.e. between 18 and 32 years of age (Christakou, Brammer, Giampietro, & Rubia, 2009). Thus, performance on the IGT appears to reach adult levels by approximately 20 years of age in healthy controls. Typically, no gender differences are found for IGT performance when conventional methods of quantifying performance are used (Bayard, Raffard, & Gely-Nargeot, 2011; Bechara et al., 2000).

Studies have used the IGT to demonstrate deficits in decision-making in groups with high impulsivity and risk-taking (Busemeyer & Stout, 2002). Research has found that healthy individuals tend to perform significantly better on the task, choosing more of the advantageous decks than those with disorders linked to deficits in the prefrontal cortex. This typically results in healthy individuals obtaining a positive score on the task and those with certain disorders score negatively. For example, healthy controls were found to perform significantly better on the IGT than obese individuals (Brogan, Hevey, O'Callaghan, Yoder, & O'Shea, 2011), those with Huntington's disease (Stout, Rodawalt, & Siemers, 2001), Obsessive Compulsive Disorder (OCD; Cavedini et al., 2002) and antisocial personality disorder (Mazas, Finn, & Steinmetz, 2000). It has been used as a measure of risky decision-making in incarcerated criminal offenders (Yechiam et al., 2008), chronic drug and alcohol abusers (Bartzokis et al., 2000; Bechara et al., 2001; Grant, Contoreggi, & London, 2000; Petry, Bickel, & Arnett, 1998) and pathological gamblers (for example see Lucantonio, Stalnaker, Shaham, Niv, & Schoenbaum, 2012).

The direct link between trait impulsivity and IGT performance is unclear. Studies have reported no significant relationship between IGT performance and either total BIS-11 or its subscales (Christakou, Brammer, et al., 2009; Gorlyn, Keilp, Oquendo, Burke, & John Mann, 2013) or the UPPS (Bayard et al., 2011). Using categorical variables for impulsiveness, some differences in performance have been reported within the literature. For example, by dividing participants into high and low impulsivity groups based on their responses to Eysenck's I_7 Impulsiveness Questionnaire, Franken, van Strien, Nijs, and Muris (2008) found that highly

impulsive individuals had impaired performance on the IGT, compared to the low impulsivity group.

In another study by Dretsch and Tipples (2011), impulsivity, sensation seeking and IGT performance were investigated using a reversed IGT and a combined impulsivity and sensation seeking measure. In the reversed IGT, 'win' and 'loss' were reversed while monetary amounts remained stable. This meant that participants lost a certain amount of money on each turn, equal in magnitude to the 'wins' of the original IGT. Thus, decks A and B had large losses on each turn, while C and D had smaller losses. Participants would win money from decks A and C every few trials but would only win from decks B and D after approximately ten selections. As decks A and B result in a net loss of $250 every ten selections in the original IGT, so they result in a net gain of $250 in the reversed IGT. Therefore, decks A and B are considered the advantageous decks in the reversed IGT. By dividing participants into high and low impulsivity/sensation seeking (ImpSS), Dretsch and Tipples (2011) found a significant interaction with gender such that high ImpSS males chose more advantageous decks than high ImpSS females, while there was no difference between low ImpSS males and females. The study concluded that males who are high in impulsivity and sensation seeking were drawn to the decks that were giving large, consistent losses, as they knew that they would occasionally receive a larger payoff. This is consistent with previous research that suggested that populations typically characterised as "poor decision makers", such as substance dependent individuals, perform better on the reversed IGT compared to healthy controls, even when demonstrating impaired performance on the standard IGT (Bechara & Damasio, 2002). Despite the ambiguity of these results they do suggest that individuals with

heightened impulsivity differ in their approach to the IGT than those with lower levels.

As mentioned above, IGT performance is typically weighted towards long-term outcome, i.e. profitable patterns of responding. This is quantified by subtracting the total number of selections from disadvantageous decks from the total number of selections from advantageous decks. An overall IGT score is usually calculated, as well as separate scores for each of the five blocks (set of 20 trials) to track performance over the course of the task (Bechara et al., 2000; Golub, Starks, Kowalczyk, Thompson, & Parsons, 2012; Gorlyn et al., 2013; X. Li, Lu, D'Argembeau, Ng, & Bechara, 2010). However, unconventional methods of analysing the IGT are becoming more common in the literature. Apart from the long-term outcomes of the decks (net profit versus net loss), each pair of advantageous and disadvantageous decks consists of one deck that delivers smaller, frequent losses and one that delivers larger, infrequent losses, relative to its pair. Recent studies are increasingly comparing the number of selections from frequent and infrequent deck selections in addition to the traditional 'IGT score' (Adida et al., 2011; Lin, Chiu, Lee, & Hsieh, 2007; Singh & Khan, 2009). The losses from infrequent punishment decks (decks B and D) can be considered 'delayed' and a preference for such decks may represent the discounting of future outcomes (Fridberg, Gerst, & Finn, 2013) or a preference for low frequency punishment (Shurman, Horan, & Nuechterlein, 2005).

Lin et al. (2007) separated frequent and infrequent punishment decks into two separate tasks (an AACC version and a BBDD version) to further investigate this preference for delayed punishments. In the AACC version of the task the basic principle of the IGT held, with participants making a notable shift in preference from

deck A to deck C over time. Interestingly, this shift occurred faster in this version than is observed in the traditional IGT, after one block, rather than two. However, in the BBDD version of the IGT, Lin et al. (2007) observed that participants demonstrated no such shift in preference, selecting equally from both decks over the course of the task. Thus, participants in this experiment appeared to be insensitive to future consequences, as decks B and D have different long-term outcomes, i.e. net loss and net gain, respectively.

In a meta-analysis of IGT studies, Dunn, Dalgleish, and Lawrence (2006) observed that both healthy participants and clinical populations preferred decks B and D to decks A and C in some studies. The degree of preference for such decks has also been observed to differ between healthy controls and certain groups. For example, studies have demonstrated a preference for deck B and/or D in schizophrenic samples, compared with control (Bark, Dieckmann, Bogerts, & Northoff, 2005; Ritter, Meador-Woodruff, & Dalack, 2004; Shurman et al., 2005). In contrast, controls have been found to select deck D more than addicted (Fishbein et al., 2005) and adolescent ADHD populations (Toplak, Jain, & Tannock, 2005), while the ADHD sample in the latter study preferred deck B more than controls. Furthermore, age effects have been reported by Hooper et al. (2004), such that both 11 to 13 and 14 to 17 year old adolescent groups selected more from infrequent punishment decks than nine to ten year olds. The current study will use the traditional method of analysing IGT performance, in addition to this alternative approach, in an effort to identify distinct patterns of decision-making in known risk takers.

4.1.1.3.2 Monetary Incentive Delay Task (MID)

The MID task was included in the current study to measure responses to monetary compared to neutral stimuli as a measure of reward processing in individuals. As mentioned below, behavioural differences on this task have not been previously reported in the literature, therefore the following section will focus on a review of previous research that has observed activational differences between healthy controls and clinical groups, as well as how the degree of activation in reward processing regions during this task may relate to trait impulsiveness.

Knutson, Westdorp, Kaiser, and Hommer (2000) piloted many variations of the MID task in an effort to develop a task that could differentiate between the neural processes of reward anticipation and outcome. Their final version of the MID contained three trial types, where one of three cues indicated whether money could be won or lost on that trial, or whether no money was at stake. Cue periods were followed by a target and then feedback as to whether participants had successfully responded within the target window ('hit') or not ('miss'). Hitting the target resulted in a monetary gain for 'win' trials, avoiding a loss on 'loss' trials and neutral positive feedback on 'no outcome' trials. Each trial, therefore, consisted of a cue period (anticipation), target and feedback (outcome), with fixation crosses separating each trial. This version, published by Knutson et al. (2001), has been widely used in the literature (for example; Filbey, Dunlop, & Myers, 2013; Scheres, Milham, Knutson, & Castellanos, 2007; A. D. Stanford et al., 2013), with many modifying the task to either include more or less conditions (for example; Bjork, Smith, Chen, & Hommer, 2011, included two win and loss conditions, each of $5 and 50c; Bjork, Smith, Chen, & Hommer, 2012, included two win magnitudes but no loss condition; and Jager, Block,

Luijten, & Ramsey, 2013, had only one win and one neutral trial type and no loss condition), replacing colour cues with words (Jia et al., 2011) or even replacing monetary rewards with points for use with children (Muller et al., 2013).

The MID has a low cognitive demand. There are no contingencies and, therefore there is no decision-making component to the task (Balodis & Potenza, 2015). Furthermore, studies often tailor task difficulty to population groups, reducing or expanding the target window based on reaction times during practice trials (see Balodis & Potenza, 2015 for a meta-analysis). Thus, differences in performance between groups on this task (such as male/female or clinical group/healthy control comparisons) are not reported in the literature. Instead, this task is implemented as means of quantifying differences in reward processing in the brain between groups. Activation in reward processing regions (such as the NAcc) during the MID has been consistently reported, leading to an inferred relationship between impulsivity and the degree of the BOLD response in the ventral and dorsal striatum (Andrews et al., 2011).

For example, not only have differences in striatal activation been observed between healthy controls and certain addicted populations but the degree of activation in these regions has been found to positively correlate with certain measures of impulsivity. Compared to controls, reduced activity in reward processing regions has been observed in current smokers, alcoholics and cannabis users for monetary gain compared to no outcome (i.e., 'win > neutral') feedback conditions (Balodis & Potenza, 2015). In contrast, when win hit feedback has been compared to fixation, cannabis users had greater striatal activity than controls (Nestor et al., 2010). Differences have also been found for former cocaine users at the various stages in their recovery. For cocaine users who only recently sought treatment for their

addiction, both greater and less activation in the striatum have been reported in response to the different conditions of the MID (Jia et al., 2011). However, cocaine users who had been sober for one to two years reliably demonstrated reduced activation, compared to controls (Bustamante et al., 2014). Similarly, for pathological gamblers, reduced activity in the striatum has been observed during both anticipation and outcome conditions compared to controls (Balodis et al., 2012) and compared to both controls and patients with OCD (Choi et al., 2012).

Furthermore, positive correlations between the degree of striatal activity during win > neutral conditions (both anticipation and feedback) and psychometric measures of impulsivity, such as the BIS (A. Beck et al., 2009) and the NEO-IF (Bjork, Smith, et al., 2012; Bjork, Smith, & Hommer, 2008), have also been observed. Age related changes in striatal activation during the MID will be discussed in greater detail in chapter five. The current chapter investigated whether differences in performance on this task by age, gender or 'risk status' emerge when task difficulty is consistent across all participants, i.e. identical versions of the task are used for each person.

4.1.1.3.3 The Go/No Go Task

The Go/No Go task is a task commonly used to measure impulsive action. The association between Go/No Go performance and trait impulsivity will be discussed below. This section also contains a review of previous findings showing changes in the number of commission errors (failed inhibitions) made on this task with age, gender differences in performance and differences in performance between healthy controls and gender. The discussion of how this task may relate to risky driving will be reserved for section 4.1.2.

Go/No Go paradigm tasks, such as the SSRT (Logan, 1994) and the Go/No Go task (Kaufman et al., 2003) require a participant to respond as quickly as possible to a target stimulus and inhibit responses to others. For example, in the Go/No Go task by Kaufman et al. (2003), participants press the space bar when an 'X' or a 'Y' appears on the screen and their reaction time is recorded. However they are instructed that if the same letter appears twice in succession they are not to respond to the repeated stimuli. Because of the rapid onset of stimuli it is difficult for some individuals to inhibit responses to the 'No Go' stimuli. Failure to suppress a no go response is thought to indicate a deficiency in behavioural inhibition or an inability to cancel behaviours.

The Go/No Go paradigm, originally proposed by M. M. Mesulam (1985), has been adopted by many investigators, who subsequently gave their tasks the same name. In a 1996 meta-analysis of such tasks, Corfitsen found that they consistently discriminate between healthy controls and patient groups with disorders for which behavioural disinhibition is a trait (e.g. those with ADHD), discussed in greater detail later. Corfitsen (1996) also reported that early imaging studies found Go/No Go tasks to reliably elicit activation in prefrontal regions associated with behavioural inhibition, providing further support for their use in assessing cognitive control. As the field of functional MRI has developed, studies have continued to support these findings (for example; Chikazoe, 2010; Menon, Adleman, White, Glover, & Reiss, 2001; Simmonds, Pekar, & Mostofsky, 2008), see chapter five for more detail.

Successful performance on the Go/No Go task, as quantified by fewer commission errors, has been found to increase with age from childhood through to adulthood (Bezdjian, Tuvblad, Wang, Raine, & Baker, 2014; Geier & Luna, 2009; Hooper et al.,

2004; Liston et al., 2006). Furthermore, there are fairly consistent reports indicating that males make more commission errors in Go/No Go tasks than females in samples of adults (Saunders et al., 2008), adolescents (Hooper et al., 2004) and children (T. Liu, Xiao, & Shi, 2013), however, studies have also reported no gender differences in university aged participants (Fernie, Cole, Goudie, & Field, 2010; Reynolds et al., 2006). What is more, while investigating the effects of alcohol consumption on inhibitory capacity, Fillmore and Weafer (2004) found that males demonstrated greater impairment on the Go/No Go task after consuming a moderate amount of alcohol.

A recent meta-analysis conducted by Wright, Lipszyc, Dupuis, Thayapararajah, and Schachar (2014) found that the following clinical populations have been observed to make more commission errors on the Go/No Go task, compared to healthy controls; ADHD, anxiety disorder, autism spectrum disorder (ASD), bipolar disorder (BD), depression, OCD and addicted samples. Furthermore, ADHD, ASD, BD, schizophrenia, personality disorder and addicted groups have also been observed to make significantly more omission errors (failing to respond to a go stimulus) than healthy controls. However, the meta-analysis also noted that there is a publication bias towards the investigation of inhibitory deficits in ADHD and addicted populations, and a neglect of disorders such as Tourette syndrome, reading disorder, oppositional defiant disorder and conduct disorder, despite behavioural disinhibition being a symptom of such disorders (Wright et al., 2014).

Some studies have proposed a link between trait impulsivity and executive functions, such as inhibitory control (Aichert et al., 2012), while some have not (Horn, Dolan, Elliott, Deakin, & Woodruff, 2003; Reynolds et al., 2006). For example, Keilp, Sackeim,

and Mann (2005) found the motor subscale of the BIS-11 to positively correlate with the number of commission errors made on a Go/No Go task. Thus, increasing levels of motor impulsiveness were associated with a greater number of failed inhibitions in that study. This association between BIS scores and inhibitory control has been replicated in the Go/No Go task by Reynolds et al. (2006; commission errors positively correlated with the first order subscale, cognitive complexity) and Aichert et al. (2012; overall BIS positively correlated with commission errors) and in the Stroop task by Enticott et al. (2006; total BIS and all three second order subscales positively correlated with Stroop errors).

4.1.2 Impulsivity and driving

Individual differences in impulsivity are thought to contribute to risk-taking while driving (Chamorro et al., 2012; Glendon, 2006; Paaver et al., 2006; Steinberg et al., 2008). While section 1.4.8 outlined evidence for the association between aberrant driving and impulsivity, the following section is a more detailed review of studies that have used the measures chosen for the current study, the Manchester Driver Behaviour Questionnaire (DBQ), BIS-11, Kirby's DDQ, the IGT, Go/No Go and MID tasks, to study impulsivity in drivers.

Psychometric measures have been used in drivers to demonstrate a link between trait impulsivity and aberrant or aggressive driving behaviours. The subscales of the DBQ have been reported to negatively correlate with the premeditation and perseverance subscales of the UPPS, and positively with the negative and positive urgency subscales (Pearson, Murphy, & Doane, 2013). Therefore, those who were higher in impulsive tendencies admitted to more mistakes and risk-taking while driving, while those who indicated a greater propensity towards slow and deliberate

thinking reported less driving errors, lapses and violations. These results are supported by Berdoulat et al. (2013), who found that the driving violations subscales of the DBQ (aggressive and highway-code violations) positively correlated with overall UPPS score (with the perseverance and premeditation subscales inversed). Similarly, the BIS-11 has shown to relate to risky driving behaviours. Dahlen, Martin, Ragan, and Kuhlman (2005) found that total BIS-11 score positively correlated with self-reported aggressive driving tendencies. Furthermore, the subscales of the BIS-11 and DBQ show differential relationships, such that motor impulsivity is positively associated with both aggressive and highway-code (HC) violations and non-planning impulsivity with just HC violations (Constantinou et al., 2011).

Delay discounting (DD) may also relate to an individual's tendency to take risks while driving. McCarthy, Niculete, Treloar, Morris, and Bartholow (2012) compared the DD of drivers who reported a tendency to drive within two hours of having three or more drinks, to those who claimed never to drive after drinking. Using the Two Choice Impulsivity Paradigm (by Dougherty, Mathias, Marsh, & Jagar, 2005), the study found no difference in DD between groups during a placebo condition. However, when administered alcohol, the 'drink driving' group chose more immediate rewards than 'non-drink drivers', and thus demonstrated steeper DD. This suggests that DD may be an indicator of an individual's likelihood to participate in risk-taking while under the influence of alcohol. Another study investigated the link between DD and drinking after driving in college students. Teeters and Murphy (2015) administered a drinking after driving (DAD) questionnaire and a pen and paper DD measure, which only differed from Kirby's DDQ by the number of items (60 compared to 27). Teeters and Murphy (2015) did not find a significant relationship between DD and DAD.

Comparing their results to McCarthy et al. (2012), who only observed DD differences in drivers during alcohol conditions, and MacKillop et al. (2011), who demonstrated that DD differences are easier to detect when comparing heavy drinkers or alcoholics to light drinkers, Teeters and Murphy (2015) concluded that their sample lacked the range of drinking habits needed to be able to detect a relationship between DD and DAD. Despite these tenuous links between DD and drink driving, a DD measure (Kirby's DDQ) was included in the present study to assess whether DD, as a measure of impulsive choice, may relate to a tendency to engage in everyday aberrant driving behaviours.

The Go/No Go and Iowa Gambling tasks have also been employed by the literature to interrogate the cognitive processes of those who take risks while driving. O'Brien and Gormley (2013) compared the impulsivity of young 'risky' and 'non-risky' drivers using the Go/No Go task, in conjunction with the BIS-11. Young drivers were categorised as risky if they had been convicted of a speeding offence. The study found that risky drivers performed worse than non-offenders in the Go/No Go task, committing more commission errors. This indicates that young drivers that speed may have poor behavioural inhibition. In another study by Fillmore, Blackburn, and Harrison (2008), drivers participated in driving simulations under both alcohol and placebo conditions. Participants also completed a Go/No Go task while sober. Fillmore et al. (2008) found that commission errors on the Go/No Go task positively correlated with speed in the simulator under the alcohol session. This provides further evidence to support a link between behavioural inhibition and risk-taking while driving. Jakubczyk et al. (2013) compared trait impulsivity (using the BIS-11) and a different measure of behavioural inhibition (the SSRT) to self-reported risk-

taking with questions such as, 'how many times during last 4 weeks did you have an accident while being under the influence of drugs or alcohol?" The authors reported that higher scores on the BIS were associated with greater self-reported risk-taking and that males reported more risk-taking overall, compared to females. However, no relationship between impulsivity and SSRT performance or between SSRT performance and self-reported risky driving were found. The current study will further investigate the link between behavioural inhibition and risky driving behaviours.

While no studies appear to have used the MID to investigate impulsivity in risky drivers, two studies have compared the performance of risky and safe drivers on the IGT. For example, Lev et al. (2008) found that 'traffic offenders', compared to safe drivers, selected more disadvantageous decks during each block of the IGT. The study also administered the International Personality Item Pool (IPIP) and observed that 'traffic offenders' had higher extraversion scores than non-offenders. Another study by Kasar, Gleichgerrcht, Keskinkilic, Tabo, and Manes (2010) examined decision-making in relapsed DUI offenders. Kasar et al. (2010) found that participants in the control group performed better than DUI reoffenders by the fifth block of the IGT. The IGT may, therefore, have some potential in demonstrating differences in decision-making between those who do and do not take risks while driving.

While most of the measures in the current study have been used to investigate how individual differences in impulsivity may contribute to risky driving, the author was unable to find an instance where all measures were combined in one experiment. As these measures have been commonly used to assess different facets of impulsivity (self-reported impulsivity with the BIS-11, delay discounting with Kirby's DDQ,

behavioural inhibition with the Go/No Go task, reward processing with the MID and decision-making with the IGT) they were included in the current study to achieve a comprehensive assessment of impulsiveness in risky and safe drivers.

4.1.3 Summary and Hypotheses

4.1.3.1 Hypothesis one: risky drivers engage in more aberrant driving behaviours than safe drivers – assessing the degree of aberrant driving within the current sample

To assess the degree of self-reported riskiness in the experimental groups of the current study, totals on the DBQ subscales were compared between 'risky' and 'safe' drivers. Unlike chapter two, however, the current study lacked a fully nested design as it did not include a sample of older risky and safe female drivers. Thus, two analytical models were employed to address this question; an analysis of 'risk status' in all males (younger and older) and in younger individuals (males and females). As was observed in chapter two, risky drivers were expected to have higher scores on all four of the subscales of the DBQ compared to safe drivers in both analytical models.

4.1.3.2 Hypothesis two: risky drivers are more impulsive than and safe drivers

While many studies have inferred a relationship between risk-taking while driving and impulsivity (for example, Casey et al., 2008), only a few studies have directly measured what aspects of impulsiveness may relate to risky driving tendencies. These studies have not yet built a coherent picture of how impulsivity may contribute to risky driving. For example, some have demonstrated a deficit in behavioural inhibition in offending drivers (O'Brien & Gormley, 2013) and others have only showed differences in DD between groups when participants were under the

influence of alcohol McCarthy et al. (2012). The current study used a combination of measures not previously reported in the literature to study potential differences between risky and safe drivers. It was hypothesised that participants who demonstrated a history of risk-taking while driving would have higher levels of both psychometric and behavioural impulsiveness, compared to safe drivers. As with hypothesis one, this question was addressed separately in all males and in all younger individuals. It was expected that some differences in impulsiveness would be independent of age and gender. As gender may impact on impulsiveness in risky drivers, interactions between driving risk group and gender were also investigated. Furthermore, positive correlations between self-reported driving risks on the DBQ and measures of impulsiveness were expected.

4.1.3.3 Hypothesis three: impulsivity declines between early and later adulthood

Differences in impulsivity between younger and older male drivers were investigated in the current study. A combination of psychometric and behavioural measures of impulsivity were utilised. Younger males were expected to make more commission errors on the Go/No Go task than older (Geier & Luna, 2009; Liston et al., 2006) and no difference between younger and older males was expected on the IGT (consistent with Christakou, Brammer, et al., 2009) or the MID (see Balodis & Potenza, 2015 for a review). Trait impulsivity was expected to be greater in the group of younger males, consistent with reports suggesting that impulsivity continues to decline until, at least, age 30 (Steinberg et al., 2008). Similarly, as a linear decline in DD has been observed from the age of 19 to 91 (da Matta et al., 2012; Löckenhoff et al., 2011), it was hypothesised that younger males would demonstrate steeper DD than older males in the current study. Finally, as young males are overrepresented in fatal collision

statistics (Glendon, 2006), the current study hypothesised that younger males would report more violations on the DBQ than older drivers.

4.1.3.4 Hypothesis four: there are gender differences in impulsivity

Failure to acknowledge the potential impact of gender on impulsiveness is cited as one of the reasons why conflicting results exist in the literature (Gay, Rochat, Billieux, d'Acremont, & Van der Linden, 2008). As such, the current study compared young male and female responses to psychometric and behavioural measures of impulsivity and driving risk. Based on the results reported in chapter two, it was expected that females would score higher than males on the error and lapse subscales of the DBQ, while males were expected to score higher on the two violations subscales; aggressive and HC violations.

In line with previous research, it was hypothesised that males and females would not differ in their responses to the BIS-11 (as seen in Patton et al., 1995) but that females would chose more immediate rewards than males men on Kirby's DDQ (previously reported by; R. C. Beck & Triplett, 2009; C. L. Smith & Hantula, 2008; Weafer & de Wit, 2013). It was also expected that young males would make more commission errors on the Go/No Go task than young females (based on the results of Saunders et al., 2008) and there would be no effect of gender on performance on the IGT (consistent with reports by; Bayard et al., 2011; Bechara et al., 2000) or the MID (Balodis & Potenza, 2015).

4.2 Method

4.2.1 Design

The current study investigated the relationship between age, gender, driving risk and impulsivity. Impulsivity was measured through the administration of psychometric measures, as well as through the analysis of participants' performance on certain cognitive tasks. A between groups design was utilised, with six experimental groups differentiated by a combination of these factors: young safe/risky males (aged between 18 and 24), young safe/risky females (between 18 and 24) and older safe/risky males (between 29 and 56).

4.2.2 Participants & Procedure

120 participants were included in this section. For details of these participants, see section 3.2. Participants completed the DBQ online prior to scanning and two pen and paper questionnaires in the pre-scanning session: the BIS-11 and Kirby's DDQ. Whilst in the scanner they completed one run of the IGT and two runs of each of the MID and Go/No Go tasks. For full details of the study's procedure see section 3.4. As the MID and Go/No Go tasks required participants to maintain a high level of arousal throughout, they were performed over two runs to give participants a break while in the scanner, while still sampling enough of each condition to calculate its Hemodynamic Response Function (HRF) for the functional analyses discussed in chapter five.

4.2.3 Apparatus and materials

Data preparation and analysis were conducted using Microsoft Excel® (2011) and SPSS 21.0 (Statistical Package for the Social Sciences by IBM-Corp, Released 2012). The IGT was presented using Psychtoolbox in conjunction with Matlab 2014a ("Matlab: MATLAB and Statistics Toolbox Release," 2014a) on an external laptop, a MacBook Pro, OS X 10.9.4, connected to the MR system using a Video graphics adapter (VGA). The MID and Go/No Go tasks were performed using Neurobehavioral Systems (NBS) Presentation® software (Version 0.70, www.neurobs.com) on the central MR system. Participants responded to the tasks using a 2 x 2 Fiber-Optic response pad. More details of the apparatus used in can be found in section 5.2.4.

4.2.3.1 Psychometric measures

4.2.3.1.1 Barratt's Impulsiveness Scale (BIS-11)

The 11th revision of Barratt's (1959) Impulsiveness Scale, by Patton et al. (1995) was used in the current study. The six first order subscales of the BIS-11 were used: attention (5 items), cognitive instability (3 items), motor (7 items), perseverance (4 items), self-control (6 items) and cognitive complexity (5 items). The attentional subscales ('attention' and 'cognitive instability') deal with loss of concentration/attention and distractibility, with 'attention' dealing exclusively with attention (for example, "I don't 'pay attention'") and 'cognitive instability' with distractibility (e.g. "I often have extraneous thoughts when thinking"). The items of the motor subscales ('motor' and 'perseverance') cover actions with short-term gains but potentially negative long-term outcomes. The first order 'motor' subscale contains general items related to acting on impulse, such as "I act in the spur of the

moment", and the 'perseverance' subscale contains specific examples of impulsive actions that may have long-term consequences, for example "I change jobs". Finally, the non-planning scales ('self-control' and 'cognitive complexity') contain mostly positively valenced questions, that are later reverse scored, about careful planning and good self-control (i.e. the 'self-control' subscale, with items such as "I plan tasks carefully") and about deliberate and slow thinking (i.e. the 'cognitive complexity' subscale, for example "I like to think about complex problems").

A copy of the BIS-11 and the exact breakdown of its scoring can be found in appendices S and T.

4.2.3.1.2 Kirby's Delay Discounting Questionnaire (DDQ)

Kirby's DDQ contains a fixed set of 27 choices between smaller immediate rewards (SIRs) and larger, delayed rewards (LDRs), for example, "Would you prefer €11 now or €30 in seven days?" (Kirby & Maraković, 1996). In the current study the 27 choices were displayed (A or B), one at a time, on a Microsoft® PowerPoint® (2011, Version 14.4.1) presentation slide while participants circled their preference on the answer sheet provided (see appendices U and V). This approach was taken in an effort to prevent earlier selections from interfering with future selections. On each slide, the 'A' option was displayed on the left hand side of the screen and the 'B' on the right. The SIRs and LDRs were randomly assigned to the 'A' and 'B' positions to prevent habitual responding. The choices were displayed in the same order as they appeared in the original DDQ, which had been contrived by the authors such that trial order did not correlate with the SIR or LDR amounts, their ratio, their difference, the delay to the LDR, or the discount rate corresponding to indifference between the two rewards.

4.2.3.1.3 The Manchester Driver Behaviour Questionnaire (DBQ)

Participants' responses on the DBQ (Parker et al., 1998), administered through the online survey, were also analysed in this section. For description of the DBQ see section 2.1.4.2.

4.2.3.2 Behavioural tasks

Participants completed the following tasks while being scanned with functional Magnetic Resonance Imaging (fMRI):

4.2.3.2.1 The Iowa Gambling Task (IGT)

The IGT is a measure of risky decision-making developed by Bechara et al. (1994). The task lasted 15 minutes. On each trial participants were asked to select from one of four decks of cards, A, B, C and D. The task is illustrated in figure 4.1. The exact instructions given to participants can be found in appendix M.

Decks A and B are considered to be disadvantageous, or 'bad' decks. Ten selections from either A or B resulted in a net loss of $250. However the nature of decks A and B were slightly different. Deck A was associated with large gains on each turn but consistent, large losses, or 'punishments'. Deck B also returned large gains on each turn but its punishments were more infrequent. After approximately ten selections from deck B participants would experience a very large loss. The first loss was fixed at $1,250 and this amount increased by $250 for each subsequent loss from deck B.

Figure 4.1: The Iowa Gambling Task

Decks C and D are considered to be advantageous, or 'good' decks. Ten selections from either C or D resulted in a net gain of $250. Similar to the disadvantageous decks, the nature of decks C and D were slightly different. Deck C was associated with moderate gains on each turn but consistent, moderate punishments. On half of loss trials from deck C the loss was less than the gain, resulting in a net gain. For the other half the loss was greater than the gain, resulting in a net loss. The gains from deck D were similar to those from deck C, however, similar to deck B, the punishments occurred infrequently. After approximately ten selections from deck D participants would experience a large loss, similar in magnitude to the losses of deck A, however these large punishments were not sufficient to result in a net loss.

4.2.3.2.2 Monetary Incentive Delay Task (MID)

Participants completed two runs of a modified version of the MID task by Nestor et al. (2010). Trials alternated between win and neutral, with win trials appearing both first and last in the task. Identical versions of the task were used for both runs. Each trial consisted of the following stages: cue (either win or neutral), target, feedback and fixation (rest). The task is illustrated in figure 4.2.

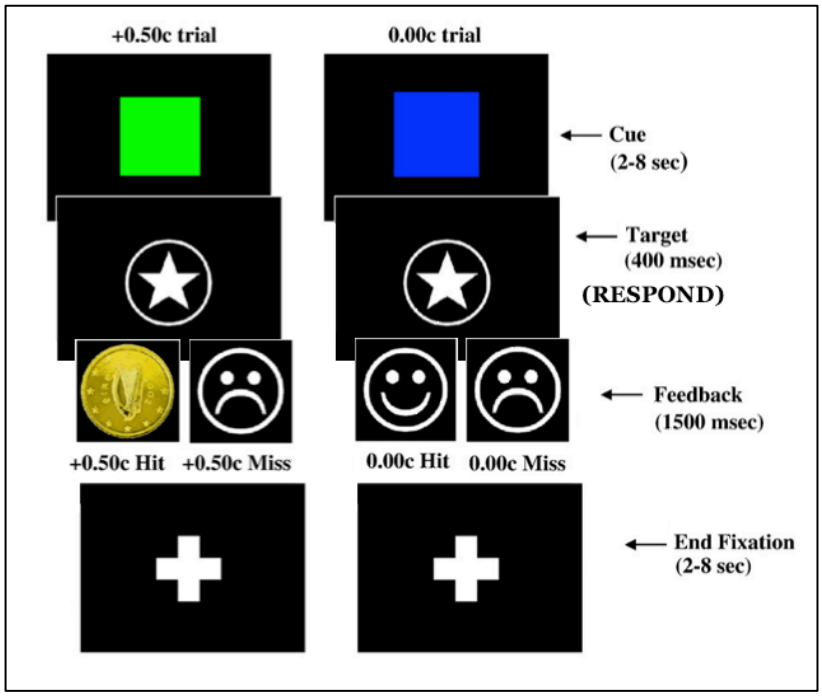

Figure 4.2: The Monetary Incentive Delay task

The colour of the cue reflected the nature of the trial, either win or neutral. Win trials were represented by a green box and indicated that money (50 cent) could be won on that trial. A blue box indicated that there was no money at stake for the trial (neutral trial). The target was a white star in a white circle. If participants responded while the target was on screen they received 'hit' feedback (a 50 cent coin for win trials and a 'happy face' for neutral trials), otherwise they were presented with a 'sad face' ('miss'

feedback). The exact instructions given to participants can be found in appendix N. Nestor et al. (2010) applied temporal jittering to this version of the MID, meaning that cue periods, target, feedback and fixation varied in duration, in order to prevent potential overlap of signals for later functional analyses.

4.2.3.2.3 The Go/No Go Task

Participants also completed two runs of a Go/No Go task by Kaufman et al. (2003). In the current version of the task, alternating 'X's and 'Y's appeared on screen. Letters appeared in white on a black background. The task is illustrated in figure 4.3.

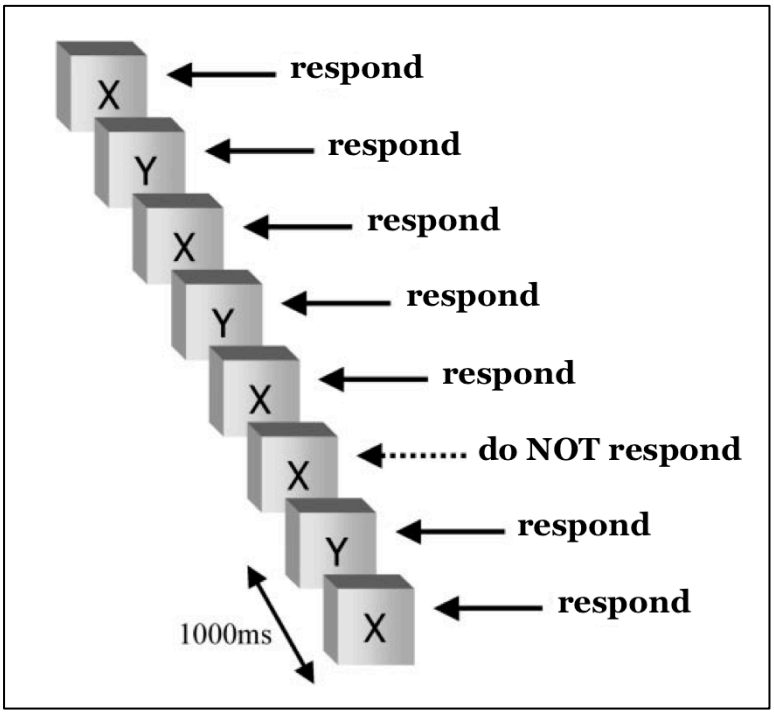

Figure 4.3: The Go/No Go task

Participants were instructed to respond to every letter they saw, unless the letter was a repeat of the previous one. Repeated letters, or 'lures' occurred in 10% of trials in this task, i.e. there were 25 no go stimuli in each run, 50 in total for the combined runs. The exact instructions given to participants can be found in appendix O.

4.2.4 Data preparation and analysis

4.2.4.1 Psychometric data

Pen and paper data were entered directly into SPSS. Some BIS-11 items were inverted before the total of each of the subscales was calculated (see appendix T). Kirby's DDQ performance was calculated in two ways: the number of times participants chose SIRs over LDRs (differentiating between small, medium and large delayed rewards) and their individual discounting rate ('k').

Kirby et al. (1999) provided a detailed description for how 'k' should be estimated from the participant's pattern of choices across the 27 questions on Kirby's DDQ. Individuals typically select the immediate reward when the discount rate is small and the delayed reward when the discount rate is large. As such, when the choices are sorted by rank order of discounting rate[11], as in appendix Wa, from smallest to largest, the point at which a participant demonstrates a shift in preference for immediate to delayed rewards reflects their individual discounting rate.

For example, if someone was to choose the immediate reward for question 19 (€33 now or €80 in 14 days) then their discounting rate would be greater than 0.10, but if they chose the delayed reward for question 11 (€11 now or €30 in seven days) then it would be less than 0.25. Therefore their discounting rate ('k') would be the midpoint between 0.10 and .25. Kirby et al. (1999) recommended using the geometric mean (the nth root of the product of n numbers) instead of the exact midpoint to avoid underweighting the smaller of the two rate parameters, yielding k=0.16 in this example ($\sqrt{0.1 * 0.25} = 0.1581$). In cases where no obvious shift in preference

[11] Calculated using the equation mentioned in section 4.1.1.1.2

occurs, participant's trend for each rank was used (as per the recommendation made by Kirby et al., 1999). For example if an individual chose two out of three SIRs for rank four it was determined that their discounting rate was greater than 0.0025. Similarly, if they chose two out of three LDRs for rank five then their discounting rate would be less than 0.006. Therefore their 'k' in this case would be 0.004 ($\sqrt{0.0025 * 0.006} = 0.0039$). Items sorted by delayed reward magnitude can be found in appendix Wb. Participants who did not demonstrate a consistent shift in preference from immediate to delayed rewards were excluded from the analysis of Kirby's discounting 'k'.

In addition to their overall discounting 'k', separate discounting parameters were estimated for small and large LDRs to further interrogate the relationship between discounting and delayed reward magnitude (as seen in R. T. Liu, Vassileva, Gonzalez, & Martin, 2012), as discounting rates have been observed to change with the magnitude of the delayed reward (Kirby, 1997).

4.2.4.2 Behavioural tasks

Individual IGT results were initially saved as Matlab workspaces and later transferred into individual SPSS documents. Preliminary analysis was conducted on individuals to summarise their results. This preliminary analysis identified the number of times deck selections were made, average reaction times, total money earned and total borrowed during the course of the game. These results were then summarised into a master IGT SPSS document.

Participant's overall IGT score was calculated by subtracting the total number of times they selected from the 'bad' decks (decks A and B) from the total number of

times they selected from the 'good' decks (decks C and D). A separate IGT score was also calculated for each of the five blocks of the task (trials 1 to 20, 21 to 40, 41 to 60, 61 to 80 and 81 to 100). Blocks one and two were treated as decision-making under uncertainty, as participants were not expected to have identified the disadvantageous decks during that period. Blocks four and five were analysed as decision-making under risk as there had been sufficient time for individuals to discern the nature of the decks. This approach is in line with previous research (Bayard et al., 2011; Ochoa et al., 2013) and supported by findings that observed that the performance of most healthy participants significantly improves during this time (Bechara & Damasio, 2002; Bechara et al., 2001). The number of times participants selected infrequent punishment decks (decks B and D) was also examined in the current study.

Log files containing behavioural data for the Go/No Go and MID tasks were automatically saved to the main experimental computer. These were later transferred to the researcher's computer and saved in Excel® format. The task start time was subtracted from each time point in each log file as well as a further ten seconds (to account for discarding the first five scans of each task, see section 5.2.5.1). Master MID and Go/No Go Excel® files were created, with separate tabs for each participant. For each participant, results for both run one and run two of each task were recorded in the same tab. Time points of interest were copied from the individual log files into these master documents.

For later use in constructing the design matrices for the functional analysis of the MID task (see section 5.2.5.4.2), time points of interest, including trial cues (win and neutral) and feedback conditions (hits or misses), were identified from the log files

and recorded in the master document. Hits[12] and misses to win and neutral cues were examined separately. This amounted to six conditions: win cues, neutral cues, win hits, win misses, neutral hits and neutral misses.

For behavioural analysis, the instances of win and neutral hits and misses were then counted and entered into a master MID SPSS document as well as the mean reaction time to win and neutral targets, which were automatically calculated and included in the raw log files produced by NBS Presentation. Performance in the MID was determined by the number of win and neutral hits participants secured over the course of the two runs of the task. There were 14 win and 13 neutral trials in each run, resulting in a maximum possible 28 hits to win and 26 hits to neutral stimuli over the two runs.

For the design matrices of the Go/No Go task (for use in section 5.2.5.4.3), the onset times of the stimuli for which successful and failed inhibitions (commission errors) occurred, as well as the onset of 'go' stimuli that participants failed to respond to (omission errors) were recorded in the master file.

Again, for behavioural analysis, the frequencies of each of these conditions were then calculated and entered into a master SPSS document. Separately, reaction times for each condition were entered into SPSS and means generated. These were then added to the master SPSS file. Better performance on the task was considered to be a greater frequency of successful responses to go stimuli and inhibitions to no go stimuli. The maximum number of successful inhibitions that could be made was 25 in each run, 50 overall. Only the number of successful inhibitions are reported in the results section of this chapter, as the number of commission errors (failed inhibitions) was,

[12] A 'hit' was defined as responding within the target window

necessarily, 25 minus the number of successful inhibitions for an individual run, and 50 minus the number of successful inhibitions for the two runs combined.

As mentioned in section 3.1, the psychometric data and performance on the behavioural tasks were analysed using 2 x 2 Factorial Analyses of Variance (ANOVA), comparing risk status (safe x risky) and gender effects in young individuals and risk status and age (younger x older) in males.

4.3 Results

4.3.1 Psychometric measures

4.3.1.1 Manchester Driver Behaviour Questionnaire (DBQ)

4.3.1.1.1 Young drivers

The DBQ can be divided into four subscales; lapse, HC violations, aggressive violations (Agg) and error. The means (M) and standard deviations (SDs) for these subscales in males (M), females (F), safe and risky individuals under the age of 25 can be found in table 4.1. The reliability of the DBQ for use in young individuals was assessed with Cronbach's alpha (α), which was found to be 0.835.

2 x 2 Factorial ANOVAs for 'gender' (males x females) and 'risk status' (safe x risky) were run on the four subscales of the DBQ in individuals under 25. Significant main effects of gender were found in the lapse and error subscales such that females scored significantly higher then males, lapse: $F(1,76)=9.223$, $p<.05$, $\eta^2=.108$, error: $F(1,76)=9.801$, $p<.05$, $\eta^2=.114$. In contrast, males scored significantly higher than females in the violation HC subscale, $F(1,76)=7.262$, $p<.05$, $\eta^2=.087$. The violation HC subscale was the only subscale for which a main effect of risk status was found. Risky

individuals scored significantly higher than safe participants for this subscale, $F(1,76)=6.235$, $p<.05$, $\eta^2=.076$. No other significant main effects and no significant interactions were found.

Table 4.1: Means and SDs for the DBQ subscales by gender and risk

		Safe			**Risky**			**M**	**F**
		M	**F**	**Tot**	**M**	**F**	**Tot**		
Lapse+	M	15.3	18.2	16.8	15.3	18.75	17.03	**15.3**	**18.5**
	SD	3.42	5.96	4.69	4.28	4.68	4.48	3.85	5.32
Violation HC*	M	12.8	10.9	**11.8**	16.1	12.6	**14.35**	**14.5**	**11.7**
	SD	4.28	4.77	4.53	4.82	4.19	4.51	4.55	4.48
Violation Agg	M	9	9.45	9.23	11.1	10.2	10.63	10	9.83
	SD	2.5	3.65	3.08	3.89	3.76	3.83	3.2	3.71
Error+	M	11.6	14.2	12.9	11.6	14.35	12.98	**11.6**	**14.3**
	SD	2.78	5.1	3.94	2.84	3.95	3.4	2.81	4.53

Relevant means are highlighted in bold, *= main effects of risk status and gender were found, += main effect of gender was found

4.3.1.1.2 Male drivers

The means and SDs for the four subscales of the DBQ in young (between 18 and 25) and older (over 29) risky and safe males can be found in table 4.2. Reliability of the DBQ in males (Cronbach's α) was 0.823.

2 x 2 Factorial ANOVAs for 'age' (younger x older) and risk status (safe x risky) were run on the four subscales of the DBQ in males. Risky male participants scored significantly higher than safe in both violation subscales; HC: $F(1,76)=15.038$, $p<.05$, $\eta^2=.165$ and Agg: $F(1,76)=5.86$, $p<.05$, $\eta^2=.072$. The relevant means are highlighted in bold in table 4.2. No other main effects of risk status were found, no main effects of age and no significant interactions.

Table 4.2: Means and SDs for the DBQ subscales by age and risk

		Safe			**Risky**			**Y.M.**	**O.M.**
		Y.M.	**O.M.**	**Tot**	**Y.M.**	**O.M.**	**Tot**		
Lapse	M	15.3	15.3	15.3	15.3	16.3	15.8	15.3	15.78
	SD	3.42	4.04	3.73	4.28	4.06	4.17	3.85	4.05
Violation HC*	M	12.8	11.5	**12.2**	16.1	15.7	**15.9**	14.45	13.58
	SD	4.28	3.07	3.68	4.82	4.78	4.8	4.55	3.93
Violation Agg	M	9	9.7	**9.35**	11.1	11.4	**11.2**	10.03	10.53
	SD	2.5	2.74	2.62	3.89	4.37	4.13	3.2	3.56
Error	M	11.7	11.5	11.6	11.6	12.2	11.9	11.63	11.85
	SD	2.78	2.46	2.62	2.84	4	3.42	2.81	3.23

Y.M. = young male, O.M. = older male. * = main effect of risk found

4.3.1.2 Barratt's Impulsiveness Scale (BIS-11)

4.3.1.2.1 Young drivers

The BIS-11 is comprised of six subscales; attention, cognitive instability, motor, perseverance, self-control and cognitive complexity. The means and standard deviations (SDs) for these subscales in males (M), females (F), safe and risky individuals under the age of 25 can be found in table 4.3. The reliability of the BIS for use in young individuals (Cronbach's alpha α) was found to be 0.833.

2 x 2 Factorial ANOVAs for gender (males x females) and risk status (safe x risky) were run on the six subscales of the BIS-11 in individuals under 25. Young risky participants scored significantly higher than safe in the self-control subscale of the BIS-11, $F(1,76)=5.743$, $p<.05$, $\eta^2=.07$. No significant main effects of gender and no further effects of risk were found.

However, within the cognitive instability subscale there was a significant interaction between gender and risk status, $F(1,76)=5.411$, $p<.05$, $\eta^2=.066$, such that there was no difference between risky and safe males (p=.194) but risky females scored

significantly higher than safe females, $t(38)= -2.042$, $p<.05$, two-tailed. A depiction of this interaction can be found in figure 4.4.

Table 4.3: Means and SDs for the BIS-11 subscales by gender and risk

		Safe			**Risky**			**M**	**F**
		M	**F**	**Tot**	**M**	**F**	**Tot**		
Attention	M	10.1	10.6	10.38	10.75	11.7	11.23	10.45	11.15
	SD	2.21	3.35	2.78	1.94	3.23	2.59	2.08	3.29
Cognitive Instability^	M	**7.15**	**6.25**	6.7	**6.3**	**7.35**	6.83	6.73	6.8
	SD	1.93	1.69	1.81	2.13	1.73	1.93	2.03	1.71
Motor	M	15.8	15.4	15.6	17.4	16.55	16.98	16.6	15.98
	SD	3.75	2.44	3.1	3.86	3.55	3.71	3.81	3
Perseverance	M	7.55	7.5	7.53	7.65	7.3	7.48	7.6	7.4
	SD	1.47	1.24	1.36	1.69	2.08	1.89	1.58	1.66
Self-control*	M	12.4	12.5	**12.45**	14.7	13.35	**14.03**	13.55	12.93
	SD	2.06	3.47	2.77	2.27	3.62	2.95	2.17	3.55
Cognitive Complexity	M	10.2	11.1	10.65	11.05	11.45	11.25	10.63	11.28
	SD	2.29	2.83	2.56	2.19	2.87	2.53	2.24	2.85

Relevant means are highlighted in bold. * = main effect of risk found, ^ = interaction between gender and risk status found

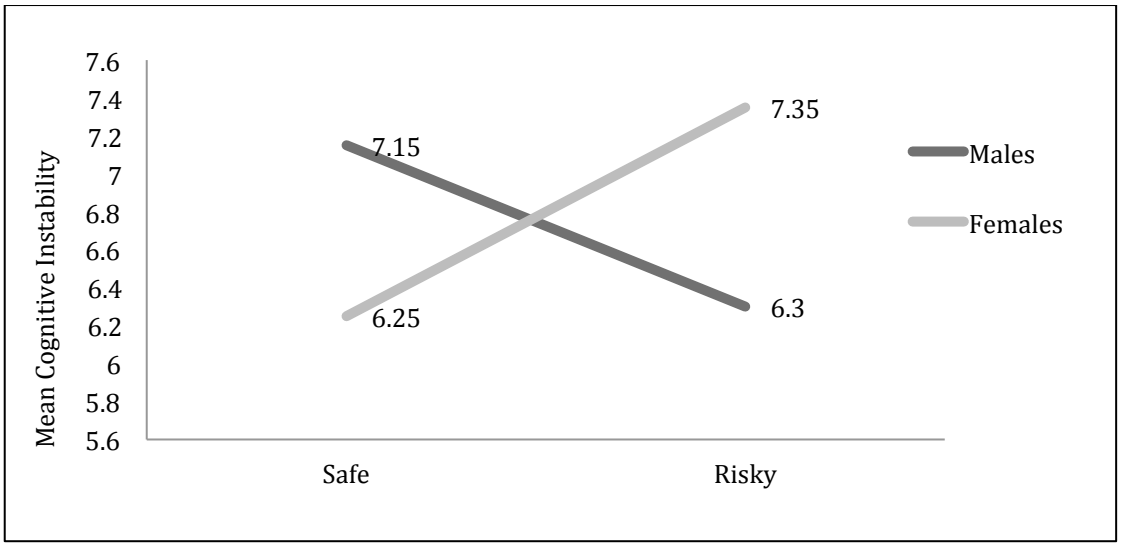

Figure 4.4: Graphical representation of the interaction between gender and risk status for young drivers on the Cognitive Instability subscale of the BIS-11

4.3.1.2.2 Male drivers

The means and SDs for the six subscales of the BIS-11 in young (between 18 and 25) and older (over 29) risky and safe males can be found in table 4.4. Reliability of the DBQ in males (Cronbach's α) was 0.805.

2 x 2 Factorial ANOVAs for age (younger x older) and risk status (safe x risky) were run on the six subscales of the BIS-11 in males. Younger males scored significantly higher than older males on the attention ($F(1,76)=7.114$, $p<.05$, $\eta^2=.086$), motor ($F(1,76)=7.412$, $p<.05$, $\eta^2=.089$) and self-control ($F(1,76)=14.209$, $p<.05$, $\eta^2=.158$) subscales of the BIS-11. Those in the risky driving category scored significantly higher than safe on the motor and self-control subscales of the BIS-11; motor: $F(1,76)=4.303$, $p<.05$, $\eta^2=.054$, self-control: $F(1,76)=13.556$, $p<.05$, $\eta^2=.151$. No other main effects of either risk status or age and no significant interactions were found.

Table 4.4: Means and SDs for the BIS-11 subscales by age and risk

		Safe			**Risky**			**Y.M.**	**O.M.**
		Y.M.	**O.M.**	**Tot**	**Y.M.**	**O.M.**	**Tot**		
Attention^	M	10.2	9.35	9.75	10.8	9.1	9.93	**10.45**	**9.23**
	SD	2.21	1.84	2.03	1.94	2.2	2.07	2.08	2.02
Cognitive Instability	M	7.15	6.7	6.93	6.3	6.4	6.35	6.73	6.55
	SD	1.93	1.38	1.66	2.13	2.01	2.07	2.03	1.7
Motor+	M	15.8	13.7	**14.8**	17.4	15.3	**16.4**	**16.6**	**14.5**
	SD	3.75	2.83	3.29	3.86	3.26	3.56	3.81	3.05
Perseverance	M	7.55	8.25	7.9	7.65	8.35	8	7.6	8.3
	SD	1.47	1.97	1.72	1.69	1.5	1.6	1.58	1.74
Self-control+	M	12.4	10.5	**11.4**	14.7	12.4	**13.5**	**13.55**	**11.4**
	SD	2.06	2.89	2.48	2.27	2.87	2.57	2.17	2.88
Cognitive Complexity	M	10.2	10.3	10.2	11.1	10.7	10.9	10.63	10.48
	SD	2.29	2.34	2.32	2.19	2.27	2.23	2.24	2.31

Relevant means are highlighted in bold. Y.M. = young male, O.M. = older male. ^ = main effect of age found, + = main effects of risk status and age found

4.3.1.3 Kirby's Delay Discounting Task (DDQ)

4.3.1.3.1 Young drivers

The mean number of small, medium and large SIRs chosen (smaller immediate rewards), in addition to Kirby's discounting 'k', are reported for males (M), females (F), safe and risky individuals under the age of 25 in table 4.5.

Table 4.5: Means and SDs for SIRs and Kirby's discounting 'k' by gender and risk

		Safe			**Risky**			**M**	**F**
		M	**F**	**Tot**	**M**	**F**	**Tot**		
Small SIRs+	M	5.55	5.2	**5.38**	6.25	5.6	**5.93**	**5.9**	**5.4**
	SD	1.15	1.24	1.2	1.25	1.23	1.24	1.2	1.24
Medium SIRs*	M	4.8	4.8	**4.8**	5.55	5.35	**5.45**	5.18	5.08
	SD	0.89	1.11	1	1.28	1.6	1.44	1.09	1.36
Large SIRs*	M	4.6	4.25	**4.43**	5.35	4.75	**5.05**	4.98	4.5
	SD	1.05	1.12	1.09	1.73	1.33	1.53	1.39	1.23
Discounting 'k'*	M	0.012	0.01	**0.011**	0.035	0.027	**0.031**	0.024	0.018
	SD	0.01	0.07	0.04	0.05	0.04	0.04	0.03	0.05
'k' for small LDRs*	M	0.028	0.025	**0.026**	0.056	0.037	**0.046**	0.042	0.031
	SD	0.03	0.02	0.02	0.05	0.04	0.04	0.04	0.03
'k' for large LDRs*	M	0.011	0.015	**0.013**	0.04	0.023	**0.031**	0.025	0.019
	SD	0.01	0.03	0.02	0.05	0.04	0.04	0.03	0.03

Relevant means are highlighted in bold. * = main effect of risk found, + = main effects of risk status and gender were found

2 x 2 Factorial ANOVAs for gender (males x females) and risk status (safe x risky) were run on Kirby measures in individuals under 25. For all SIR reward magnitudes, risky drivers chose more of the smaller, immediate rewards than larger, delayed rewards, when compared to safe drivers; small SIRs: $F(1,71)=4.95$, $p<.05$, $\eta^2=.065$, medium SIRs: $F(1,71)=8.121$, $p<.05$, $\eta^2=.103$, large SIRs: $F(1,71)=7.195$, $p<.05$, $\eta^2=.092$. There was a significant difference between males and females in their

preference for small SIRs, such that males chose more small SIRs than females: $F(1,71)=5.331$, $p<.05$, $\eta^2=.07$. This effect was not replicated in medium SIRs and was close to significant for large SIRs (p=0.051). No interactions between gender and risk status were found for any of the SIR reward magnitude types.

Kirby's discounting 'k' (overall and separated into small and large LDRs) did not meet the assumptions for normality (Kolmogorov-Smirnov test) and, hence, the data were transformed using a Log_{10} transformation. After transformation a significant difference was found between the discounting parameters of risky and safe participants, such that risky drivers discounted the delayed rewards more than the safe drivers; overall discounting 'k': $F(1,71)=7.516$, $p<.05$, $\eta^2=.096$; discounting for small LDRs: $F(1,75)=4.487$, $p<.05$, $\eta^2=.056$; discounting for large LDRs: $F(1,75)=6.915$, $p<.05$, $\eta^2=.084$. There was no effect of gender and no gender by risk status interaction for this variable. The relevant means are highlighted in bold in table 4.5.

Using a cut off of k=0.0523 individuals were assigned to 'impaired' and 'unimpaired' discounting groups, a value based on previous research (Kirby & Petry, 2004; Passetti, Clark, Mehta, Joyce, & King, 2008). A 2 x 2 Chi-square test for independence examining the proportion of impaired and unimpaired individuals in the risky and safe groups revealed that there were fewer impaired participants in the safe group and more impaired participants in the risky group than expected: $X^2= 7.634$ (1, N=75), $p<.05$, see table 4.6 below. Using this criterion, there was no difference in the number of younger males and females who demonstrated impaired and unimpaired responding on Kirby's DDQ.

Table 4.6: Observed and expected impaired discounting by risk status

		Unimpaired	**Impaired**
Safe	Count	37	1
	Expected	32.9	5.1
Risky	Count	28	9
	Expected	32.1	4.9

4.3.1.3.2 Male drivers

The means and SDs for the three SIR reward magnitudes and Kirby's discounting 'k' for young (between 18 and 25) and older (over 29) risky and safe males can be found in table 4.7. 2 x 2 Factorial ANOVAs for age (younger x older) and risk status (safe x risky) were run on SIR reward magnitudes and Kirby's discounting 'k' in males.

Table 4.7: Means and SDs for SIRs and Kirby's discounting 'k' by age and risk

		Safe			**Risky**			**Y.M.**	**O.M.**
		Y.M.	**O.M.**	**Tot**	**Y.M.**	**O.M.**	**Tot**		
Small SIRs^	M	5.55	4.8	**5.18**	6.25	5.8	**6.03**	**5.9**	**5.3**
	SD	1.15	1.82	1.49	1.25	1.58	1.42	1.2	1.7
Medium SIRs-	M	4.8	4.25	4.53	5.55	5.4	5.48	**5.18**	**4.83**
	SD	0.89	1.86	1.38	1.28	1.35	1.32	1.09	1.61
Large SIRs^	M	4.6	3.85	**4.23**	5.35	4.8	**5.08**	**4.98**	**4.33**
	SD	1.05	2.06	1.56	1.73	1.67	1.7	1.39	1.87
Discounting 'k'*	M	0.012	0.024	**0.018**	0.035	0.029	**0.032**	0.076	0.026
	SD	0.01	0.06	0.03	0.05	0.04	0.04	0.03	0.05
k' for small LDRs^	M	0.028	0.032	**0.03**	0.056	0.038	**0.047**	**0.042**	**0.035**
	SD	0.03	0.05	0.04	0.05	0.05	0.05	0.04	0.05
k' for large LDRs^	M	0.011	0.019	**0.015**	0.04	0.02	**0.03**	**0.025**	**0.02**
	SD	0.01	0.04	0.03	0.05	0.04	0.05	0.03	0.04

Relevant means are highlighted in bold. Y.M. = young male, O.M. = older male. * = main effect of risk found, ^ = main effects of risk status and age were found, - = main effect of age was found

Similar to the findings in young drivers, risky drivers demonstrated a greater preference for small and large SIRs than safe drivers but there was no difference for medium SIRs; small SIRs: $F(1,70)=7.097$, $p<.05$, $\eta^2=.092$, large SIRs: $F(1,70)=5,465$, $p<.05$, $\eta^2=.072$. After a Log_{10} transformation risky drivers had a significantly greater Kirby's discounting 'k' than safe drivers; overall discounting 'k': $F(1,70)=1.972$, $p<.05$, $\eta^2=.12$; discounting for small LDRs: $F(1,75)=4.062$, $p<.05$, $\eta^2=.051$; discounting for large LDRs: $F(1,75)=4.304$, $p<.05$, $\eta^2=.055$. There was a main effect of age for every SIR reward magnitude, such that younger males chose more SIRs than older males; small SIRs: $F(1,70)=4.096$, $p<.05$, $\eta^2=.055$, medium SIRs: $F(1,70)=9.323$, $p<.05$, $\eta^2=.118$, large SIRs: $F(1,70)=4.175$, $p<.05$, $\eta^2=.056$. This resulted in an effect of age for discounting 'k' for small LDRs ($F(1,75)=3.991$, $p<.05$, $\eta^2=.051$) and large LDRs ($F(1,75)=3.989$, $p<.05$, $\eta^2=.051$) but no effect of age for overall discounting 'k'. No significant interactions between age and risk status were found.

Using the cut-off mentioned above, a 2 x 2 Chi-square test for independence examining impaired discounting and risk status revealed no difference in the expected and observed proportions of impaired and unimpaired risky and safe drivers ($p=.091$).

4.3.2 Behavioural tasks

4.3.2.1 Iowa Gambling Task (IGT)

4.3.2.1.1 Young drivers

Means and SDs for the times to select from decks A, B, C and D (reaction times in seconds) are reported in table 4.8 below. Performance on the IGT was measured by 'IGT Score', which is calculated by the overall frequency of selections from decks C

and D ('good decks'), minus the frequency of selections from decks A and B ('bad decks'). As the task consisted of 100 trials, separate IGT Scores were also calculated for each of the five blocks of 20 trials in order to track performance across the task. Out of 100 selections the total number of selections from infrequent punishment (B and D) over frequent punishment (A and C) decks was also examined. The means and standard deviations (SDs) for these measures in males, females, safe and risky young individuals can be found in table 4.9.

Table 4.8: Means and SDs for IGT deck reaction times (RT) by gender and risk

		Safe			**Risky**			**M**	**F**
		M	**F**	**Tot**	**M**	**F**	**Tot**		
RT A^	M	**1.04**	**1.3**	**1.17**	**1.03**	**1.04**	**1.03**	**1.03**	**1.17**
	SD	0.24	0.32	0.28	0.24	0.21	0.23	0.24	0.27
RT B*	M	0.98	1.16	1.07	0.97	1.03	1	**0.98**	**1.1**
	SD	0.22	0.28	0.25	0.25	0.26	0.26	0.24	0.27
RT C	M	1.06	1.15	1.11	0.94	0.99	0.96	1	1.07
	SD	0.44	0.33	0.38	0.24	0.3	0.27	0.34	0.31
RT D*	M	0.97	1.12	1.04	0.89	0.99	0.94	**0.93**	**1.06**
	SD	0.27	0.38	0.32	0.18	0.22	0.2	0.23	0.3

Relevant means are highlighted in bold. M = male, F = female. * = main effect of gender, ^ = main effects of gender and risk status found, in addition to a significant interaction between the two

2 x 2 Factorial ANOVAs for gender (males x females) and risk status (safe x risky) were run on IGT Score, infrequent punishment deck selections (total B and D selections) and deck reaction times (RT) in individuals under 25. There was a significant main effect of gender for the mean time to select from deck A, $F(1,76)=5.561$, $p<.05$, $\eta^2=.068$, such that young females were slower (i.e. had greater RTs) than young males. There was also a main effect of risk status, with safe drivers having greater RTs than risky, $F(1,76)=5.594$, $p<.05$, $\eta^2=.069$.

However these effects should be interpreted in light of the significant interaction between risk status and gender, $F(1,76)=4.855$, $p<.05$, $\eta^2=.06$, where there was no difference between the RTs of risky and safe young males (p=.903) but safe young females were slower than risky young females, $t(38)= 3.045$, $p<.05$, two-tailed. A depiction of this interaction can be found in figure 4.5 below.

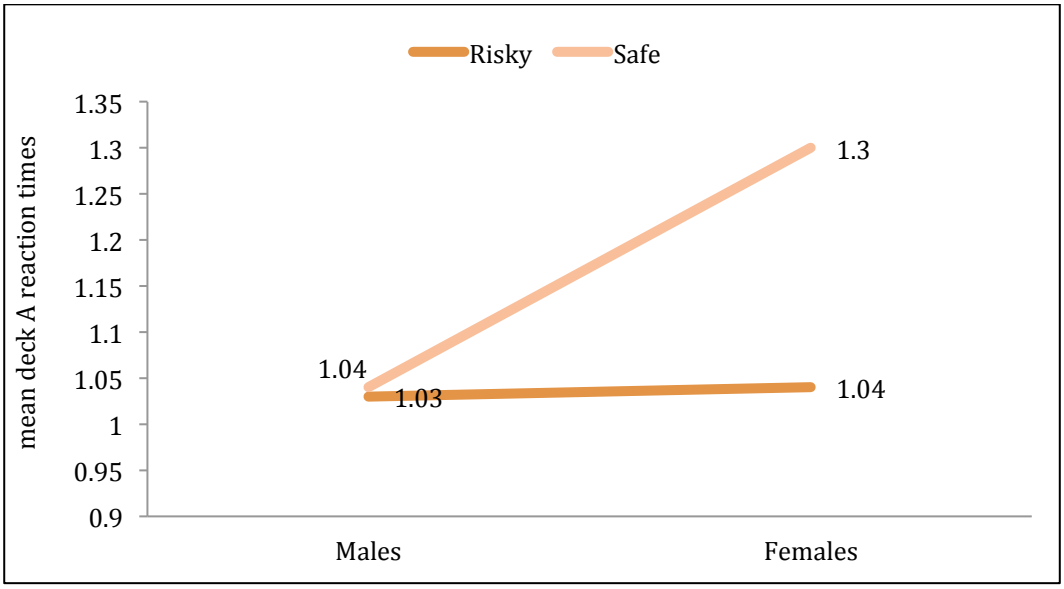

Figure 4.5: Graphical representation of the interaction between gender and risk status for young drivers for their reaction times to deck A

A main effect of gender was also observed for deck B RTs ($F(1,76)=4.315$, $p<.05$, $\eta^2=.054$) and deck D ($F(1,75)=4.266$, $p<.05$, $\eta^2=.054$) but not for deck C, resulting in an overall effect of gender for 'bad deck' RTs (A and B combined; $F(1,76)=5.019$, $p<.05$, $\eta^2=.062$) but not for 'good decks' (C and D combined). In all cases where a gender effect was observed, young females were slower than young males. There were no other significant effects of risk status and no other significant interactions for RTs.

There was a main effect of risk status for the number of deck B and D selections (infrequent punishment decks), $F(1,76)=8.11$, $p<.05$, $\eta^2=.096$, such that risky young

drivers demonstrated a preference for infrequent punishment decks and safe young drivers for frequent punishment decks (A and C). These means are highlighted in bold in table 4.9. There was no effect of either risk status or gender within IGT Score and no significant interactions between these two variables for IGT Score or total BD selections.

Table 4.9: Means and SDs for IGT performance measures by gender and risk

		Safe			**Risky**			**M**	**F**	**Tot**
		M	**F**	**Tot**	**M**	**F**	**Tot**			
IGT Score	M	14	20.2	17.1	17.6	10.4	14	15.8	15.3	15.5
	SD	31.1	28.1	29.6	30.3	27.9	29.1	30.7	28	29.3
Block 1^	M	-3	-4.4	-3.7	-3.2	-1.6	-2.2	-3.1	-3	**-3**
	SD	4.3	4	4.1	5.4	5.1	5.2	4.8	4.5	4.7
Block 2^	M	1.6	3.1	2.4	1	1.3	1.2	1.3	2.2	**1.8**
	SD	5.8	5.6	5.7	8.5	7.8	8.2	7.2	6.7	6.9
Block 3^	M	3.7	6.4	5.1	3.6	4	3.8	3.6	5.2	**4.4**
	SD	9.9	6.8	8.3	7.8	10.2	9	8.8	8.5	8.7
Block 4^	M	6.6	8.2	7.4	9.1	3.6	6.5	7.8	6	**6.9**
	SD	9.7	9.9	9.8	9.1	10.1	9.6	9.4	10	9.7
Block 5^	M	5.1	6.9	6	7.2	2.9	5	6.1	4.89	**5.5**
	SD	9.4	12.4	10.9	11	7.3	9.1	10.2	9.8	10
B+D Selections*[13]	M	46.1	48.6	**47.3**	58.5	55.5	**57**	52.3	52	52.1
	SD	15.6	15.7	15.7	15.2	14.1	14.6	15.4	14.9	15.1

Relevant means are highlighted in bold. M = male, F = female. * = main effect of risk, ^ = main effect of block was found (repeated measures)

A Mixed Factorial ANOVA with both gender and risk status as between groups factors and the IGT score over the five 'Blocks' as a repeated measurement revealed a significant main effect of block, $F(4,304)=28.309$, $p<.05$, $\eta^2=.271$, with no other

[13] Refers to the total number of combined selections from decks B and D out of a total 100 selections during the task. The number of A and C selections is this figure minus 100.

significant main effects or interactions. A line graph illustrating this change in IGT Score over the five blocks can be seen in figure 4.6.

Paired t-test post hoc suggested that the IGT Score of young individuals increased significantly on each block between one and four but there was no improvement between block four and block five; block one – block two: $t(79)= -4.663$, $p<.05$, two-tailed, block two to block three: $t(79)= -3.536$, $p<.05$, two-tailed, block three to block four: $t(79)= -3.347$, $p<.05$, two-tailed.

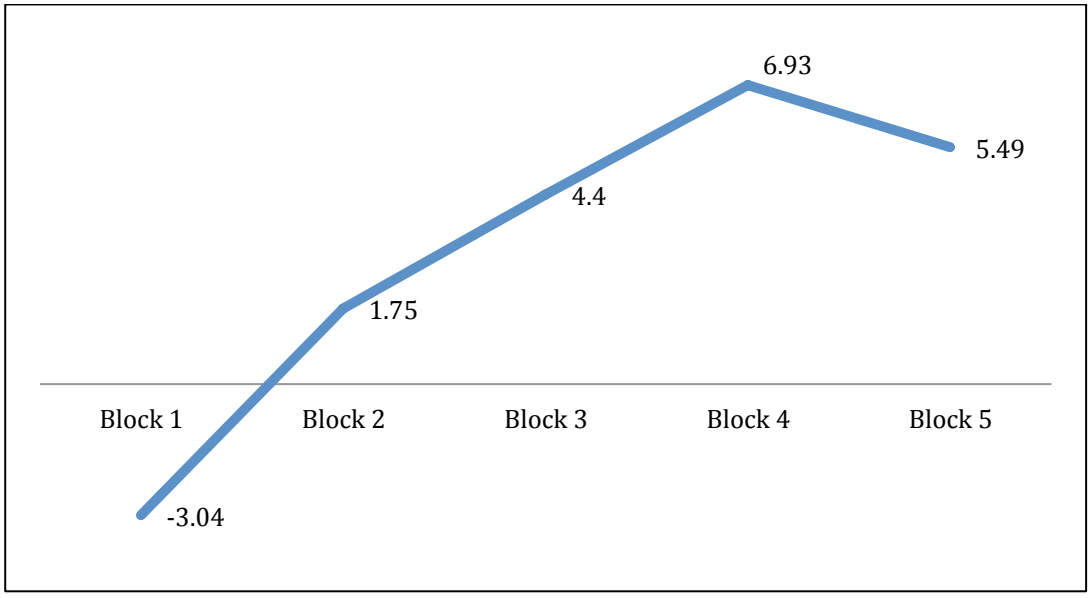

Figure 4.6: Line graph showing the change in IGT Score over the five blocks of the task in young individuals

4.3.2.1.2 Male drivers

The means and SDs for the reaction times, in seconds, of the four decks can be found in table 4.10, while the various measures of IGT performance of young (between 18 and 25) and older (over 29) male drivers can be found in table 4.11. 2 x 2 Factorial ANOVAs for age (younger x older) and risk status (safe x risky) were run on IGT Score, total BD selections and deck reaction times (RT) in males.

There were no main effects of risk status for reaction times for any of the four decks, however significant main effects of age were found for all decks, with the exception of deck C, with older males selecting from decks significantly slower than younger males; deck A: $F(1,76)= 9.177$, $p<.05$, $\eta^2=.108$, deck B: $F(1,76)= 8.156$, $p<.05$, $\eta^2=.097$, deck D: $F(1,76)= 10.045$, $p<.05$, $\eta^2=.117$. Similar to the findings in younger individuals, this resulted in an overall effect of age for 'bad decks' (A and B), $F(1,76)= 9.705$, $p<.05$, $\eta^2=.113$, but a non-significant difference between younger and older males for 'good decks' (C and D). No significant interactions between age and risk status were found.

Table 4.10: Means and SDs for IGT deck reaction times (RT) by age and risk

		Safe			**Risky**			**Y.M.**	**O.M.**
		Y.M.	**O.M.**	**Tot**	**Y.M.**	**O.M.**	**Tot**		
RT A*	M	1.04	1.2	1.12	1.03	1.2	1.11	**1.04**	**1.2**
	SD	0.24	0.25	0.24	0.24	0.25	0.25	0.24	0.25
RT B*	M	0.98	1.15	1.06	0.97	1.11	1.04	**0.98**	**1.13**
	SD	0.22	0.25	0.23	0.25	0.23	0.24	0.24	0.24
RT C	M	1.06	1.08	1.07	0.94	1.02	0.98	1	1.05
	SD	0.44	0.28	0.36	0.24	0.28	0.26	0.34	0.28
RT D*	M	0.97	1.08	1.02	0.89	1.12	1.01	**0.93**	**1.1**
	SD	0.27	0.24	0.26	0.18	0.24	0.21	0.23	0.24

Relevant means are highlighted in bold. Y.M. = young male, O.M. = older male. * = main effect of age was found

Despite the lack of main effects of either risk status or age for infrequent punishing decks (B and D), there was a significant interaction between risk status and age, $F(1,76)=4.51$, $p<.05$, $\eta^2=.056$. Further inspection of this interaction found that young risky male drivers demonstrated a greater preference for the infrequent rather than

frequent punishment decks than young safe male drivers ($t(38) = -2.547$, $p<.05$, two-tailed), but there was no difference between older risky and safe male drivers ($p=.76$). A graph of this interaction can be seen in figure 4.7. No significant main effects or interactions were observed for overall IGT Score.

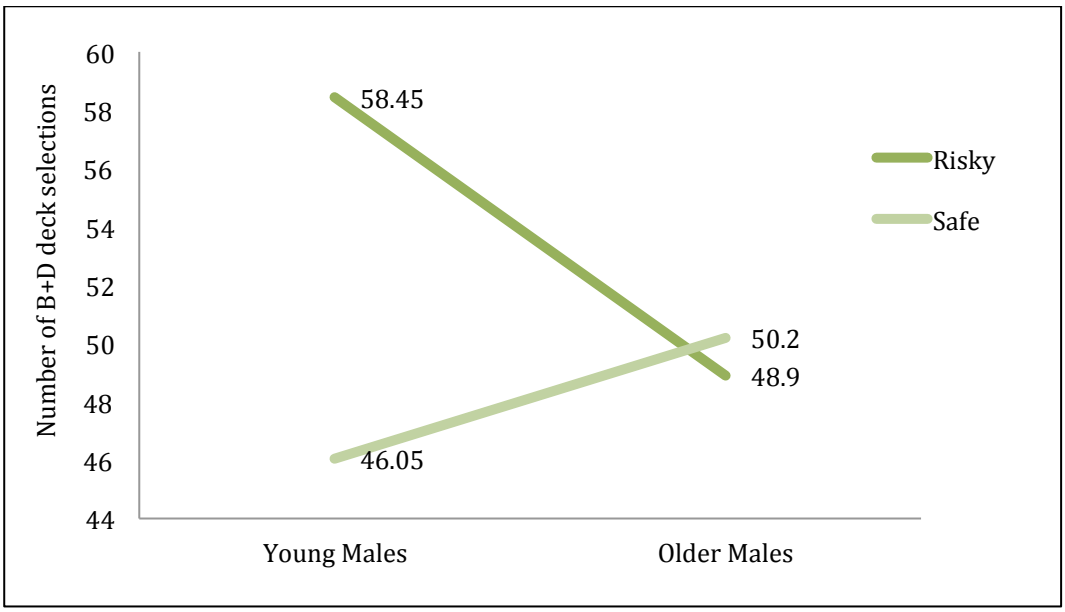

Figure 4.7: Graphical representation of the interaction between age and risk status for male drivers for BD deck selections

A Mixed Factorial ANOVAs with both age and risk status as between groups factors and the IGT Score over the five 'Blocks' as a repeated measurement revealed a significant main effect of block, $F(4,304)=30.25$, $p<.05$, $\eta^2=.285$, with no other significant main effects or interactions. A line graph illustrating this change in IGT Score over the five blocks can be seen in figure 4.8.

Paired t-test post hoc suggested that males improved between each block between one and four but there was no improvement between block four and block five; block one – block two: $t(79)= -4.88$, $p<.05$, two-tailed, block two to block three: $t(79)= -2.723$, $p<.05$, two-tailed, block three to block four: $t(79)= -4.133$, $p<.05$, two-tailed.

Table 4.11: Means and SDs for IGT performance measures by age and risk

		Safe			Risky			Y.M.	O.M.	Tot
		Y.M.	**O.M.**	**Tot**	**Y.M.**	**O.M.**	**Tot**			
IGT Score	M	14	25.2	19.6	17.6	20.1	18.8	15.8	22.6	19.2
	SD	31.1	32.9	32	30.3	25.9	28.1	30.7	29.4	30
Block 1^	M	-3	-1.6	-2.3	-3.2	-3.3	-3.2	-3.1	-2.5	**-2.8**
	SD	4.3	4.6	4.5	5.4	4.7	5	4.8	4.68	4.8
Block 2^	M	1.6	3.6	2.6	1	3.7	2.3	1.3	3.6	**2.5**
	SD	5.8	7.7	6.8	8.5	8.6	8.6	7.2	8.2	7.7
Block 3^	M	3.7	7.2	5.4	3.6	4.2	3.9	3.6	5.7	**4.7**
	SD	9.9	10.5	10.2	7.8	8.9	8.4	8.8	9.7	9.3
Block 4^	M	6.6	8.6	7.6	9.1	7.7	8.4	7.8	8.1	**8**
	SD	9.7	10.7	10.2	9.1	9.2	9.1	9.4	10	9.7
Block 5^	M	5.1	7.5	6.3	7.2	7.8	7.5	6.1	7.7	**6.9**
	SD	9.4	12.2	10.8	11	9.1	10	10.2	10.6	10.4
B+D Selections*[14]	M	**46.1**	**50.2**	48.1	**58.5**	**48.9**	53.7	52.3	49.6	50.9
	SD	15.6	13.8	14.7	15.2	13	14.1	15.4	13.4	14.4

Relevant means are highlighted in bold. Y.M. = young male, O.M. = older male. * = significant interaction between age and risk status was found, ^ = main effect of block was found (repeated measures)

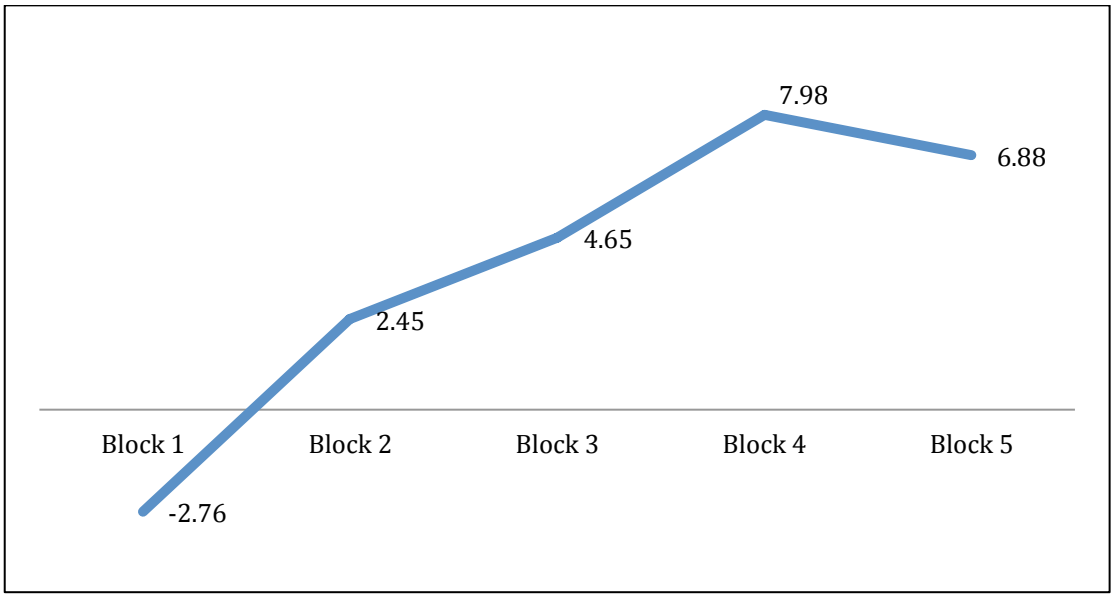

Figure 4.8: Line graph showing the change in IGT Score over the five blocks of the task in male drivers

[14] Refers to the total number of combined selections from decks B and D out of a total 100 selections during the task. The number of A and C selections is this figure minus 100

4.3.2.2 Monetary Incentive Delay Task (MID)

4.3.2.2.1 Young drivers

The means and standard deviations (SDs) for response times to targets that followed win and neutral cues (in milliseconds) in males (M), females (F), safe and risky individuals under the age of 25 can be found in table 4.12. Performance on the MID task was measured by the number of 'hits' obtained to either win or neutral stimuli. A hit was defined as a response within the target window and these resulted in the subsequent presentation of 'hit' feedback. Younger participants hit 67.61% of win stimuli (between 13 and 22 out of 28) and 70.38% of neutral (between 11 and 21 out of 26). The number of hits to win and neutral stimuli can also be found in table 4.12. 2 x 2 Factorial ANOVAs for gender (males x females) and risk status (safe x risky) were run on MID RTs and performance measures in individuals under 25. While no significant main effects of risk status were found for any of the variables in table 4.12, main effects of gender were found for all measures. Males hit more targets overall, regardless of cue type; frequency of win hits: $F(1,76)=16.271$, $p<.05$, $\eta^2=.176$, neutral hits: $F(1,76)=6.882$, $p<.05$, $\eta^2=.083$. Necessarily, it was also found that females responded to targets slower than males (i.e. had greater reaction times); win RT: $F(1,76)=14.072$, $p<.05$, $\eta^2=.156$, neutral RT: $F(1,76)=15.533$, $p<.05$, $\eta^2=.17$. No significant risk status by gender interactions were found.

A Mixed Factorial ANOVA with both gender and risk status as between group factors was used to examine the number of hits to both win and neutral stimuli between runs one and two in younger individuals. For win hit there was a significant effect of run, $F(1,76)=6.095$, $p<.05$, $\eta^2=.074$, where participants hit more in run one than run two.

Table 4.12: Means and SDs for MID RTs and performance measures by gender and risk

			Safe			**Risky**			**M**	**F**	**Tot**
			M	**F**	**Tot**	**M**	**F**	**Tot**			
Win Hit+	Run 1*	M	9.8	9.1	9.45	10.05	9.3	9.68	9.93	9.2	**9.56**
		SD	0.62	0.97	0.79	0.76	1.03	0.9	0.69	1	0.85
	Run 2	M	9.7	9	9.35	9.7	9.05	9.38	9.7	9.03	**9.36**
		SD	0.8	0.97	0.89	0.66	0.95	0.81	0.73	0.96	0.85
	Combined** £	M	19.5	18.1	18.8	19.75	18.35	19.05	**19.63**	**18.23**	18.93
		SD	1.24	1.77	1.51	1.21	1.87	1.54	1.23	1.82	1.52
Neutral Hit	Run 1^	M	9.65	9.25	9.45	9.5	8.8	9.15	9.58	9.03	9.3
		SD	0.81	0.91	0.86	1.05	1.4	1.23	0.93	1.16	1.04
	Run 2	M	9	8.75	8.88	9.5	8.75	9.13	9.25	8.75	9
		SD	1.26	1.21	1.24	0.95	1.55	1.25	1.11	1.38	1.24
	Combined^^ £	M	18.65	18	18.33	19	17.55	18.28	**18.83**	**17.78**	18.3
		SD	1.57	1.84	1.71	1.45	2.21	1.83	1.51	2.03	1.77
Win RT£		M	804.27	918.69	861.48	774.04	885.54	829.79	**789.16**	**902.11**	845.64
		SD	105.14	159.98	132.56	117.3	148.76	133.03	111.22	154.37	132.8
Neutral RT£		M	825.34	933.88	879.61	795.41	914.71	855.06	**810.37**	**924.29**	867.33
		SD	101.78	130.13	115.95	120.01	158.58	139.3	110.9	144.35	127.63

Relevant means are highlighted in bold. * = out of a possible 14, ** = out of a possible 28, ^ = out of a possible 13, ^^ = out of a possible 26. £ = main effect of gender found, + = main effect of run (repeated measure) found.

There was no difference between the number of neutral hits in runs one and two. For both neutral and win stimuli the between group effects of gender previously reported remained, and, as before, there were no effects of risk status. In addition there were no interactions with either of these variables and runs.

4.3.2.2.2 Male drivers

Overall, males hit 69.29% of win (between 16 and 22 out of 28) and 71.65% of neutral stimuli (between 15 and 22 out of 26). The means and SDs for MID RTs and performance measures in young (between 18 and 25) and older (over 29) risky and safe males can be found in table 4.13. 2 x 2 Factorial ANOVAs for age (younger x older) and risk status (safe x risky) revealed no main effects or interactions for performance measures of the MID. For RTs, the analysis found no differences between drivers of differing risk status, however, significant effects of age were found, such that older males were slower to respond to the stimuli than younger males: win RT: $F(1,76)= 5.731$, $p<.05$, $\eta^2=.07$, neutral RT: $F(1,76)= 5.267$, $p<.05$, $\eta^2=.07$. No interactions between age and risk status were found.

A Mixed Factorial ANOVA with both age and risk status as between group factors was used to examine the number of hits to both win and neutral stimuli between runs one and two in males. There were no differences between runs for either win or neutral hits and no main effects of age or risk status. Despite this, there was a significant interaction between risk status and runs for hits to win stimuli, $F(1,76)=4.11$, $p<.05$, $\eta^2=.051$, such that there was no difference between runs in young safe participants ($p=.555$) but risky participants made more hits to win stimuli in run one than run two; $t(39)= 2.317$, $p<.05$, two-tailed. No other significant interactions were found.

Table 4.13: Means and SDs for MID RTs and performance measures by age and risk

			Safe			**Risky**			**Y.M.**	**O.M.**	**Tot**
			Y.M.	**O.M.**	**Tot**	**Y.M.**	**O.M.**	**Tot**			
Win Hit^	Run 1	M	9.8	9.4	**9.6**	10.05	9.75	**9.9**	9.93	9.58	9.75
		SD	0.62	0.6	0.61	0.76	1.02	0.89	0.69	0.81	0.75
	Run 2	M	9.7	9.65	**9.68**	9.7	9.55	**9.63**	9.7	9.6	9.65
		SD	0.8	0.75	0.78	0.66	0.76	0.71	0.73	0.76	0.74
	Combined	M	19.5	19.05	19.28	19.75	19.3	19.53	19.63	19.18	19.4
		SD	1.24	1.05	1.15	1.21	1.63	1.42	1.23	1.34	1.28
Neutral Hit	Run 1	M	9.65	9.3	9.48	9.5	9.25	9.38	9.58	9.28	9.43
		SD	0.81	1.03	0.92	1.05	1.29	1.17	0.93	1.16	1.05
	Run 2	M	9	9.25	9.13	9.5	9.05	9.28	9.25	9.15	9.2
		SD	1.26	0.91	1.09	0.95	1.23	1.09	1.11	1.07	1.09
	Combined	M	18.65	18.55	18.6	19	18.3	18.65	18.83	18.43	18.63
		SD	1.57	1.57	1.57	1.45	1.95	1.7	1.51	1.76	1.64
Win RT*		M	804.27	874.83	839.55	774.04	832.64	803.34	**789.16**	**853.74**	821.45
		SD	105.14	102.1	103.62	117.3	151.59	134.44	111.22	126.84	119.03
Neutral RT*		M	825.34	890.14	857.74	795.41	859.87	827.64	**810.37**	**875.01**	842.69
		SD	101.78	102.39	102.08	120.01	167.96	143.99	110.9	135.18	123.04

Relevant means are highlighted in bold. Y.M. = young male, O.M. = older male. * = main effect of age found, ^ = interaction between risk status and run (repeated measure) found

4.3.2.3 Go/No Go Task

4.3.2.3.1 Young drivers

Performance on the Go/No Go task was measured by the number of successful no go (successful inhibition) responses made. The overall number of successful 'goes' made was also measured. Younger participants successfully inhibited their responses to 46.38% of lure stimuli[15] and responded to 90.79% of go stimuli. The means and standard deviations (SDs) for these in male (M) and female (F), risky and safe young drivers can be found in table 4.15. 'Successful Go' and 'Failed No Go' (commission error) response times (in milliseconds) in young individuals can be found in table 4.14 below.

Table 4.14: Means and SDs for Go/No Go RTs by gender and risk

		Safe			**Risky**			**M**	**F**
		M	**F**	**Tot**	**M**	**F**	**Tot**		
Go RT	M	884.3	888.4	886.4	848.2	888.4	868.3	866.3	888.4
	SD	81.3	87.2	84.3	74.6	61.4	68	77.9	74.3
Failed No Go RT	M	840.5	856.8	848.6	803.6	855.4	829.5	82	856.1
	SD	104.2	115.3	109.7	111.4	67.4	89.4	107.8	91.4

2 x 2 Factorial ANOVAs for gender (males x females) and risk status (safe x risky) were run on Go/No Go RTs and performance variables in individuals under 25. There were no main effects and no significant interactions for any of the performance measures or Go/No Go RTs.

[15] Range of successful inhibitions was between five and 39 out of 50

Table 4.15: Means and SDs for Go/No Go performance measures by gender and risk

			Safe			**Risky**			**M**	**F**	**Tot**
			M	**F**	**Tot**	**M**	**F**	**Tot**			
Correct Go	Run 1^	M	198.35	201.7	200.03	208.25	205	206.63	203.3	203.35	203.33
		SD	26.55	19.78	23.17	14.66	17.02	15.77	21.75	18.29	19.62
	Run 2	M	205	199.65	202.33	208.6	207.65	208.13	206.8	203.65	205.23
		SD	16.1	25.82	21.41	14.17	16.27	15.07	15.08	21.68	18.2
	Combined^^	M	403.35	401.35	402.35	416.85	412.65	414.75	410.1	407	408.55
		SD	37.99	43.72	40.44	27.6	27.82	27.44	33.48	36.62	34.39
Correct No Go	Run 1*	M	12.8	11.7	12.25	13.05	12	12.53	12.93	11.85	**12.39**
		SD	4.1	4.3	4.2	3.6	4.2	3.9	3.79	4.2	4.04
	Run 2	M	11.3	10.4	10.85	11.75	9.75	10.75	11.53	10.08	**10.8**
		SD	4.2	5.9	5.1	4.1	4.3	4.2	4.11	5.12	4.63
	Combined**	M	24.1	22.1	23.1	24.8	21.75	23.28	24.45	21.93	23.19
		SD	7.1	9.7	8.4	6.1	7.9	7	6.6	8.8	7.7

Relevant means are highlighted in bold. ^ = out of a possible 225, the remainder were omission errors, ^^ = out of a possible 450, the remainder were omission errors, * = out of a possible 25, the remainder were commission errors, ** = out of a possible 50, the remainder were commission errors, £ = main effect of run (repeated measure) found.

A Mixed Factorial ANOVA, with both gender and risk status as between group factors and the two runs of the Go/No Go task as a repeated measure, was used to examine the number of successful go's and successful no go responses between runs one and two. In young individuals there was no difference in the number of go responses made in the first and second runs, however there were fewer successful no go responses made in run two, compared to run one; $F(1,76)=12.864$, $p<.05$, $\eta^2=.145$.

4.3.2.3.2 Male drivers

The means and SDs for Go/No Go RTs in young (between 18 and 25) and older (over 29) risky and safe males can be found in table 4.16 below and Go/No Go performance measures in table 4.17. Male participants responded to 86.68% of go stimuli and successfully inhibited their responses to 49.26% of lure stimuli[16].

Table 4.16: Mean and SDs for Go/No Go RTs by age and risk

		Safe			**Risky**			**Y.M.**	**O.M.**
		Y.M.	**O.M.**	**Tot**	**Y.M.**	**O.M.**	**Tot**		
Go RT^	M	884.3	924.2	904.3	848.2	916.2	882.2	**866.3**	**920.2**
	SD	81.3	81.3	81.3	74.6	94.6	84.6	77.9	87.9
Failed No Go	M	840.5	867.3	853.9	803.6	884.1	843.8	**822**	**875.7**
	SD	104.2	79.8	92	111.4	110.7	111	107.8	95.2

Relevant means are highlighted in bold. Y.M. = young male, O.M. = older male. ^ = main effect of age found.

2 x 2 Factorial ANOVAs for age (younger x older) and risk status (safe x risky) were run on Go/No Go RTs and performance measures in males. An effect of age was

[16] Range of successful inhibitions was between nine and 44 out of 50

observed for the overall number of correct 'go' responses made ($F(1,76)= 13.634$, $p<.05$, $\eta^2=.152$), where older male drivers made fewer go responses than young male drivers. There were also significant effects of age for the RT measures, successful go ($F(1,76)= 8.382$, $p<.05$, $\eta^2=.099$) and failed no go ($F(1,76)= 5.405$, $p<.05$, $\eta^2=.067$), such that older males had slower reaction times than younger males. There were no main effects of risk and no significant interactions between risk status and age.

A Mixed Factorial ANOVA, with both age and risk status as between group factors and the two runs of the Go/No Go task as a repeated measure, was used to examine the number of successful go's and successful no go responses between runs one and two. There was a significant main effect of 'runs' for successful inhibitions (correct no go responses), $F(1,76)=6.989$, $p<.05$, $\eta^2=.084$, such that a greater number of successful inhibitions were made in run one, compared to run two. In addition to a significant main effect of age for correct go responses ($F(1,76)=13.634$, $p<.05$, $\eta^2=.152$), a significant effect of runs was observed, such that more go responses were made in run two: $F(1,76)=19.405$, $p<.05$, $\eta^2=.203$. However, these effects should be interpret in light of the significant interaction between age and runs, $F(1,76)=8.121$, $p<.05$, $\eta^2=.097$, where there was no difference between the number of go responses made across the runs in young males ($p=.194$), but older male drivers made more go responses in run two, compared to run one: $t(39)= -4.519$, $p<.05$, two-tailed. This interaction is illustrated in figure 4.9.

Table 4.17: Means and SDs for Go/No Go performance measures by age and risk

			Safe			**Risky**			**Y.M.**	**O.M.**	**Tot**
			Y.M.	**O.M.**	**Tot**	**Y.M.**	**O.M.**	**Tot**			
Correct Go^	Run 1	M	198.35	172	185.18	208.25	181.65	194.95	**203.3**	**176.83**	**190.06**
		SD	26.55	37.55	34.76	14.66	31.8	27.91	21.75	34.69	28.71
	Run 2	M	205	186.65	195.83	208.6	199.65	204.13	**206.8**	**193.15**	**199.98**
		SD	16.1	35.35	28.66	14.17	21.97	18.8	15.08	29.79	22.49
	Combined	M	403.35	358.65	381	416.85	381.3	399.08	**410.1**	**369.98**	390.04
		SD	37.99	68.27	59.04	27.6	50.81	44.19	33.48	60.5	47.74
Correct No Go*	Run 1	M	12.8	12.6	12.7	13.05	13.15	13.1	12.93	12.88	**12.9**
		SD	4.07	5.22	4.65	3.58	4.21	3.9	3.79	4.69	4.26
	Run 2	M	11.3	12.1	11.7	11.75	11.75	11.75	11.53	11.93	**11.73**
		SD	4.22	4.87	4.545	4.09	5.35	4.72	4.11	5.05	4.619
	Combined	M	24.1	24.7	24.4	24.8	24.9	24.85	24.46	24.8	24.63
		SD	7.13	9.51	8.32	6.12	8.96	7.54	6.63	9.24	7.93

Relevant means are highlighted in bold. ^ = main effects of age and run (repeated measure) and an interaction between the two were found, * = effect of run (repeated measure) was found

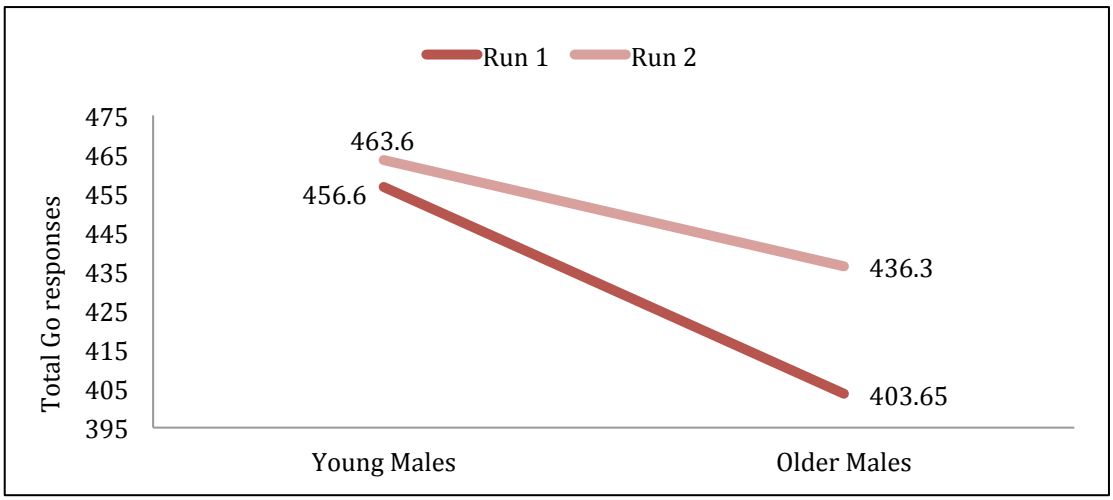

Figure 4.9: Graphical representation of the interaction between Runs and age in males

4.3.3 Exploratory Factor Analysis (EFA)

Eighteen variables across the five measures of impulsivity were chosen for inclusion in an EFA, the subscales of the psychometric measures (the BIS-11 and Kirby's DDQ) and the main outcome variables for the three behavioural tasks (the IGT, MID and Go/No Go tasks). These variables were subjected to principle components analysis using SPSS. To test the assumptions of EFA, the Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy and Bartlett's Test of Sphericity were calculated. The KMO was found to be was 0.723, indicating an adequate sample size for FA. Furthermore, Bartlett's Test was significant, suggesting the presence of meaningful correlations in the data.

Using the Kaiser–Guttman criterion the initial, un-rotated solution suggested the presence of six factors, explaining 70.71% of the variance. Upon inspection of the Scree plot, however, it was decided that a five factor solution would better reflect the data, see figure 4.10 below, and this explained 65.42% of the variance.

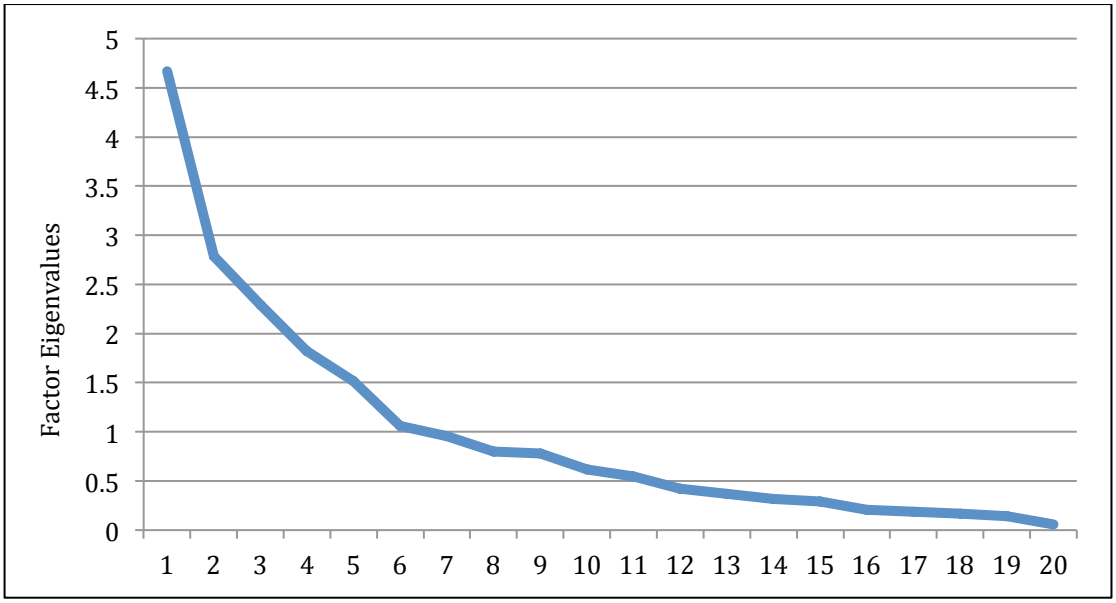

Figure 4.10: Scree plot of the factor Eigenvalues

Due to the nature of the items, the five factor solution was subjected to an oblique rotation, Promax, which first places the factors orthogonally in geometric space and then rotates them to allow for correlations between the factors. The factor correlation threshold, kappa (κ), was set at four, the default in SPSS (and as recommended by Pett, Lackey, & Sullivan, 2003). After rotation the five factors had the following Eigenvalues; 4.12, 3.17, 2.94, 2.07, 1.8, however, due to the correlations between factors, these values do not accurately represent the amount of variance explained by each factor.

The rotated solution revealed the presence of simple structure, with all factors showing a number of strong loadings, and all items loading substantially on only one factor. Table 4.18 below contains the structure loadings between the items and the factors, as these are unaffected by the correlations between the factors. The analysis revealed that each factor represented one of the five measures.

Table 4.18: Structure matrix showing the perpendicular loadings between the items and the five factors

Measure	Items	Factors				
		1	2	3	4	5
BIS-11	Attention subscale			.683		
BIS-11	Cognitive Instability subscale			.656		
BIS-11	Motor subscale			.742		
BIS-11	Perseverance subscale			.443		
BIS-11	Self-Control subscale			.792		
BIS-11	Cognitive Complexity subscale			.572		
Kirby's DDQ	Small SIR	.917				
Kirby's DDQ	Medium SIR	.929				
Kirby's DDQ	Large SIR	.915				
Kirby's DDQ	Kirby's 'k'	.948				
Go/No Go	Correct No Go					.655
MID	Total Win Hit				.864	
MID	Total Neutral Hit				.754	
IGT	Block 1 = trials 1 - 20[17]		-.386			
IGT	Block 2 = trials 21 - 40		.664			
IGT	Block 3 = trials 41 - 60		.853			
IGT	Block 4 = trials 61 - 80		.883			
IGT	Block 5 = trials 81 - 100		.786			

4.3.4 Relationships between measures

The relationships between the psychometric and behavioural measures were investigated using bivariate correlations. Due to the number of comparisons made, a Bonferroni correction was applied to adjust the alpha level to 0.0005 for each test (0.05/100).

With this adjusted alpha level a significant correlation was found between the attention subscale of the BIS and performance in the fifth block of the IGT

[17] IGT score across the five blocks was chosen instead of overall IGT score for Factor Analysis as this approach took into consideration how strategy may change over the course of the task

($r(120)= -.326$, $p<.0005$, two-tailed), such that higher levels of attentional impulsivity were associated with poorer performance during this final block of the task. Furthermore, a preference for smaller SIRs (smaller immediate reward) in Kirby's DDQ was associated with poorer performance on the IGT both overall ($r(120)= -.326$, $p<.0005$, two-tailed) and in the final block of the task ($r(120)= -.325$, $p<.0005$, two-tailed).

It was also observed that the frequency of hits to win stimuli in the MID task were positively related to the number of successful inhibitions made in the Go/No Go task ($r(120)= .349$, $p<.0005$, two-tailed). A significant relationship between reaction times to win stimuli (on the MID) and the time to select from deck B in the IGT ($r(120)= .37$, $p<.0005$, two-tailed) was also observed.

Finally, significant positive correlations between psychometric measures of impulsivity and the DBQ subscales were found. The lapse subscale of the DBQ was related to the cognitive instability subscale of the BIS-11 ($r(120)= .322$, $p<.0005$, two-tailed), such that greater cognitive instability was associated with more lapses in attention while driving. Furthermore, the HC violations subscale of the DBQ positively correlated with motor impulsiveness on the BIS-11 ($r(120)= .349$, $p<.0005$, two-tailed) and the number of SIRs chosen on Kirby's DDQ when the rewards were small ($r(120)= .321$, $p<.0005$, two-tailed) and medium ($r(120)= .339$, $p<.0005$, two-tailed) in magnitude.

At this corrected alpha level no other significant correlations were found between the measures.

4.4 Discussion

4.4.1 Summary of findings

4.4.1.1 Hypothesis one: aberrant driving tendencies in risky and safe drivers

To assess the relationship between driver history and self-reported risky driving tendencies in the current sample, scores on the four DBQ subscales were compared between risky and safe drivers. As the study lacked a fully nested design, younger males and females were compared on one Factorial ANOVA model (gender x risk status) and all males (older and younger) were compared in another (age x risk status). It was expected that risky drivers would report more aberrant driving behaviours than safe drivers.

In both models, risky drivers reported more HC violations than safe drivers. This would suggest that, in the current sample, drivers who have been collision culpable, and/or have accrued penalty points, intentionally break the law while driving (by, for example, breaking the speed limit) more than those who did not meet these criteria for risk. This finding was independent of both age and gender. A further difference between risky and safe drivers was observed when all males were compared. In this model, risky drivers also reported a higher frequency of aggressive driving violations. These males, therefore, engaged in more aggressive and hostile acts towards other drivers while on the road. Thus, the findings of chapter two were partially replicated in the current sample. This suggests that the criteria validated in chapter two were useful in differentiating between risky and safe drivers for the current study. However, more differences were detected between risk groups when all males were combined, i.e. two subscales

demonstrated a main effect of risk status as opposed to only one in the 'younger individuals' model. The fact that the distinctions between risky and safe drivers were not as numerous as those reported in chapter two would suggest that the drivers recruited to comprise the risky group of the current study were, perhaps, not very risky. This limitation will be discussed in greater detail in the limitations section.

4.4.1.2 Hypothesis two: risky drivers are more impulsive than and safe drivers

The relationship between impulsivity and driving risk were investigated in hypothesis two. It was expected that individuals with a history of risk-taking while driving would demonstrate higher levels of impulsiveness than safe drivers on the psychometric and behavioural measures employed by the current study; the BIS-11, Kirby's DDQ, IGT, MID and Go/No Go tasks.

In both models, risky drivers demonstrated steeper discounting on Kirby's DDQ and higher (lack of) self-control on the BIS-11, compared to safe drivers. Greater delay discounting (DD) in Kirby's DDQ was noted on almost all sub-measures in both models, with the only exception being medium LDRs in the analysis of all males. Therefore, independent of age and gender, risky drivers in the current study favoured immediate gains over long-term rewards and demonstrated lower levels of careful thinking and planning. Furthermore, according to the criterion set out by previous research (by; Kirby & Petry, 2004; Passetti et al., 2008), a greater proportion of risky younger drivers demonstrated impaired responding on Kirby's DDQ than safe. The criterion of impairment was devised based on research into the DD of addicted populations, such as those addicted to heroine or cocaine. This provides evidence to suggest that the extent to which some younger individuals

place a higher value on immediate rewards, and discount future ones, may result in poorer decisions while driving, which could relate to the overrepresentation of younger individuals in collision statistics. Thus, while DD was still an issue for older drivers, the reduction in DD with age (discussed under hypothesis three below) may explain why males continue to be overrepresented in collisions, but to a lesser extent than younger males.

Therefore, while previous research has only found risky drivers to display greater DD when they were under the influence of alcohol (McCarthy et al., 2012), the results of the current study provide more consistent evidence that greater impulsive choice, characterised by a preference for immediate rewards, may play a role in aberrant driving. Kirby's DDQ was particularly good at differentiating between drivers with and without a history of risky driving in the present study, regardless of the age or gender of the participants. This may relate to the extent to which speeding was a penalty point offence in the risky drivers of the current sample, approximately 40% of whom had penalty points for speeding. In contrast, the McCarthy et al. (2012) study used drivers who reported a tendency to drive after consuming alcohol, who may represent a subset of drivers whose DD tendencies are only affected whilst under the influence. The results of the current study would suggest that DD could play a greater role in aberrant driving tendencies than previously stated. The risky sample of the present study, which consisted predominantly of drivers who had been convicted of speeding offences, may have been more sensitive to differences in DD. For instance, drivers who are more likely to make decisions that have immediate benefits, but potentially

negative long-term outcomes, could decide to speed to reach a destination more quickly, rather than drive within the speed limit and arrive safely.

In addition to the self-control subscale of the BIS-11, a difference was observed in the motor subscale in males (dealing with a tendency to act in the moment) and an interaction between gender and risk status in younger individuals, where risky young female drivers scored higher than safe on the cognitive instability subscale of the BIS-11 (pertaining to distractibility), with no difference between risky and safe younger males. This additional difference could indicate that aspects of trait impulsivity may play more of a role in risk-taking in young females than in young males. Overall, these findings are similar to those reported by O'Brien and Gormley (2013), who found differences between young (17 to 21 year old) offender and non-offender drivers on the second order subscales for which cognitive instability and self-control belong, the attention and non-planning second order subscales, respectively. Furthermore, the effect sizes for these differences were similar to those of the present study. For example, for the non-planning subscale, O'Brien and Gormley (2013) found the effect to be $\eta^2=.08$, compared to $\eta^2=.07$ for self-control in the younger individuals of the current study. This suggests that individual differences in self-control are a good indicator of aberrant driving tendencies.

Moreover, while it is not clear why only male risky and safe drivers differed on the motor subscale, and female risky and safe drivers on the cognitive instability subscale, the significant correlations between these subscales and subscales of the DBQ, discussed in greater detail below, suggest that there is a meaningful relationship between aberrant driving and both motor impulsiveness and

cognitive instability. However, risky and safe groups did not differ on the following BIS-11 subscales; attention, perseverance and cognitive complexity, nor did these demonstrate a relationship with any of the subscales of the DBQ. There are two possible explanations for why this was the case; either the risky group of the current study was not *that impulsive*, or these subscales may not be relevant to aberrant driving. While the former explanation should be the subject of further investigation, an argument can be made for the latter, such that items pertaining to a dislike of staying still for long periods of time (i.e. the attention subscale, e.g. 'I "squirm" at plays or lectures'), specific examples of impulsive behaviours (i.e. the perseverance subscale, e.g. 'I change jobs') and about deliberate and slow thinking (i.e. the cognitive complexity subscale, e.g. 'I like to think about complex problems') may not be relevant to how an individual may behave while driving.

Overall, there is consistent evidence in the present study to suggest that self-control capabilities, the tendency to carefully plan behaviours and regulate thoughts, may be lower in risky drivers. This trait may lead to poor decision-making while driving or a lack of consideration of the consequences of aberrant behaviours. This is a novel finding of the current study, as previous research has not used first order subscales to study impulsivity in drivers. Such research typically calculates a total BIS-11 score (Eensoo, Paaver, & Harro, 2010, 2011; Fillmore et al., 2008; Jakubczyk et al., 2013) or has examined differences between driver groups based on the three second order subscales (Constantinou et al., 2011; Pearson et al., 2013; P. Smith et al., 2006). It is possible that the use of first order subscales allowed the present study to detect nuances between risky and safe drivers previously unreported by the literature.

When all males were compared in their performance on the behavioural tasks, no main effects of risk status were found. However, a significant interaction between age and risk status was observed for the number of infrequent punishment deck selections (decks B and D) on the IGT, such that young risky males demonstrated a greater preference for infrequent punishment decks than young safe males, while there was no difference between older risky and safe males. Similarly, when all younger individuals were analysed, risky young drivers selected more from the infrequent punishment decks in the IGT than safe younger participants, independent of gender. Together these findings suggest that younger individuals who take risks while driving may be more insensitive to infrequent punishments, be more punishment averse (Shurman et al., 2005) or may even discount future consequences (Fridberg et al., 2013) more than those without a history of risky driving. This may have particular relevance to the punishments/negative outcomes of aberrant driving behaviours, such as those in the form of being detected by the police or being involved in a collision, which are very infrequent. It is possible that drivers who continue to take risks may be ignoring such potential punishments.

When reaction times for the four decks of the IGT were compared between groups, an interaction between risk status and gender was also observed, such that safe females were slower to choose from deck A than risky females, while no difference between risky and safe younger males was found. Previous research has demonstrated that participants shift their preference away from deck A earlier in the task as it is relatively easy to ascertain that it is a disadvantageous deck, delivering frequent punishments larger in value than monetary gains (Lin et

al., 2007). As safe younger females were slower to select from this disadvantageous deck it suggests greater hesitation, possibly in anticipation of a large loss.

In younger individuals, more commission errors were made in run two of the Go/No Go task but there were no differences in the number of commission errors made between groups. Therefore, the behavioural findings of O'Brien and Gormley (2013) were not replicated. This may have been due to the difference in sampling method between this and the current study. The O'Brien and Gormley (2013) study recruiting only drivers who had been caught speeding, which is arguably a very risky offence and should be quite sensitive to a propensity towards risk-taking while driving. However the current study used combined criteria of collision culpable and/or the accumulation of penalty points when differentiating between risky and safe drivers, which may have been more susceptible to misclassification. This could mean that the drivers in the current study were not *as risky* as those in O'Brien and Gormley (2013).

No differences in performance on the MID were observed between risky and safe younger drivers. Overall, younger participants hit fewer win targets in the second run of the MID, compared to the first run. There was no difference in the number of neutral hits between the runs and no interactions between run and either gender or risk status. Poorer performance on the second runs of both the MID and Go/No Go tasks may indicate fatigue in the younger participants by the second run.

Finally, as previously mentioned, significant positive correlations between two DBQ subscales and some psychometric measures of impulsivity were observed,

such that higher trait impulsivity was associated with a greater number of self-reported risky driving behaviours. Specifically, higher scores on the lapse subscale of the DBQ was associated with higher cognitive instability on the BIS-11. This is not surprising as these two scales appear to measure similar constructs; loss of concentration and distractibility, respectively. Individuals who reported greater motor impulsiveness on the BIS-11, and demonstrated a greater preference for SIRs when the LDRs were small and medium in magnitude, scored higher on the HC violations subscale of the DBQ. The motor subscale of the BIS-11 refers to the general tendency to act in the moment (for example, 'I act in the spur of the moment' and 'I do things without thinking') and a preference for SIRs suggests steeper discounting of delayed rewards when such rewards are small to medium in size. As such, these findings suggest that a tendency towards acting in the present, without the consideration of future consequences, may be associated with deliberate driving violations, such as speeding, overtaking on the inside and not obeying traffic lights. While the relationship between DD and the DBQ has not been previously reported in the literature, the associations between the BIS-11 and the DBQ are consistent with a previous report that found motor impulsivity to relate to HC violations (Constantinou et al., 2011). No relationship was found between the DBQ subscales and indicators of performance on the behavioural tasks (the IGT, MID and Go/No Go tasks).

4.4.1.3 Hypothesis three: impulsivity declines between early and later adulthood

The trait and behavioural impulsivity of older and younger male drivers were compared. As younger males are involved in more fatal collisions than any other age group (Glendon, 2006), it was expected that younger males would self-report

more risky driving behaviours than older. However, no effects of age were observed for any of the subscales of the DBQ and no interactions between age and risk status. As a similar lack of decline in DBQ scores was observed with age in the larger, survey sample (with the exception of the error subscale), it may be that much larger sample sizes are needed to detect any decline. This is reflected in the results of a meta-analysis of the DBQ by De Winter and Dodou (2010), who found correlations very small in magnitude (approximately –.05) between age and the error and lapse subscales of the DBQ, and approximately –.2 in magnitude between age and the violation subscales, with a combined sample of approximately 25,000 drivers.

As trait impulsivity and DD have been observed to decline with age (da Matta et al., 2012; Löckenhoff et al., 2011; Steinberg et al., 2008), the current study hypothesised that younger males would score higher on BIS-11 subscales and choose more immediate rewards on Kirby's DDQ. This was supported by the data, with younger males scoring higher on the attention, motor and self-control subscales of the BIS-11 and choosing more immediate rewards than older drivers, regardless of LDR reward magnitude. This evidence suggests that males between 18 and 24 act and think more impulsively, demonstrating differences on subscales of the BIS-11 that tap into three elements of the measure, as well as demonstrate steeper DD tendencies than males over the age of 29. As no interactions between age and risk status were observed on these measures, the above differences may be considered to be entirely due to age effects. However, it should be noted that the cognitive instability, cognitive complexity and perseverance subscales did not demonstrate a decline between younger and older males. This suggests that

distractibility (i.e. 'cognitive instability'), deliberate thinking (i.e. 'cognitive complexity') and specific impulsive behaviours (i.e. 'perseverance') may be traits that remain stable during this age range. As some, but not all, aspects of impulsiveness were found to persist into later adulthood in males, this may explain why males continue to be overrepresented in collision statistics, despite an overall decline in collision involvement with age. The lack of clear distinction between the impulsiveness of the younger and older males of the current study may have made it more difficult to detect age related functional and structural changes in the brain.

Furthermore, some differences in behaviour on the behavioural measures were observed between older and younger males. In the IGT, older males took longer to select from disadvantageous decks than younger males, possibly indicative of a greater hesitation when choosing disadvantageously. Despite this difference in reaction times, no effects of age were observed on IGT performance, consistent with Christakou, Brammer, et al. (2009). Older males also had greater reaction times on the MID and Go/No Go tasks. No other differences in MID performance were detected, consistent with previous reports (Balodis & Potenza, 2015). While no difference in the number of commission errors on the Go/No Go task was observed between younger and older males (as was hypothesised, based on previous research by; Geier & Luna, 2009; Liston et al., 2006), young males were found to make more successful 'go' responses in the task overall. This, in conjunction with greater reaction times in the task, may indicate that older males were more hesitant to respond on each trial, possibly in anticipation of a 'lure' stimulus. This hesitation may have caused them to miss more go stimuli. These

behavioural differences are unlikely to relate to differences in impulsiveness and, instead, are just age related slowing of reaction times.

As mentioned previously, older males made fewer go responses overall in the Go/No Go task compared to younger males. However, it should be noted that when the two runs of the task were compared, older males made significantly more go responses in the second run of the task. The frequency of go responses remained stable between runs in younger males. Despite this difference, the number of successful and failed inhibitions on the Go/No Go task remained the same across runs in older males and there were no differences in the number of commission errors between younger and older males.

While males overall are overrepresented in both fatal and non-fatal collision statistics, younger males are even further overrepresented. As behavioural differences between younger and older drivers in the present study were mostly due to increasing reaction times with age, it is unlikely that these relate to this decrease in collision involvement in older males. However, trait impulsivity was largely observed to decline between early and later adulthood male participants.

If behavioural differences in impulsivity do exist between younger and older males, the tasks employed by the current study were unable to detect them. If this were the case then age differences in brain activation during these tasks should have been observed, as fMRI can be more sensitive to underlying neural processes not detectable in the outcome variables of behavioural tasks. As such, this was observed for risk status, as fMRI proved better able to detect differences between risky and safe drivers in the present study than the performance measures of these tasks. However, this was not found to be the case for age, for which only one

region of the brain demonstrated activational differences between younger and older males (the hippocampus). These results will be discussed in greater detail in chapter five. Therefore, despite a noted decline in trait impulsivity (as measured with the BIS-11 and Kirby's DDQ) with age in males, the current study provides no evidence to suggest that behavioural impulsiveness declines in males within the age range tested (18 to 56). Despite this, future research should continue to examine the relationship between declining trait impulsivity with age and a reduction in the rates of collision involvement.

4.4.1.4 Hypothesis four: there are gender differences in impulsivity

Gender differences in impulsivity and self-reported risky driving behaviours were investigated under hypothesis four. The majority of the gender differences reported in chapter two were replicated in the current sample. Young female drivers reported making more errors and lapses than young males and males reported a more HC violations.

As hypothesised, no effects of gender were observed on any of the subscales of the BIS-11, consistent with the findings of the original article that produced this version of the measure (Patton et al., 1995). As a number of studies have reported greater DD in females, the current study expected to replicate this effect (R. C. Beck & Triplett, 2009; C. L. Smith & Hantula, 2008; Weafer & de Wit, 2013). However, no difference in the discounting function 'k' was detected between groups. Instead, males demonstrated a greater preference than females for SIRs, but only when the LDR was small. This is consistent, in part, with findings by Petry et al. (2002), and suggests that males may have a greater preference for immediate rewards when the future reward is small. Thus, the current study

provides little evidence to suggest a difference in trait impulsivity between young males and females.

Some differences in reaction times were observed between the genders on the behavioural tasks. For example, females were slower to select from decks B and D in the IGT. This did not impact on their performance on the task, however, as no gender differences in IGT score or other performance variables were observed, consistent with previous research (Bayard et al., 2011; Bechara et al., 2000). Females were also slower to respond to both win and neutral targets on the MID task. This resulted in males hitting more win and neutral targets than females. This was an unexpected finding, as previous research utilising the MID has not reported behavioural differences on this task (Balodis & Potenza, 2015). This could indicate that young males were either better at or more engaged with the MID. This finding was stable across the two runs of the task. Finally, no differences in performance were observed on the Go/No Go task. Therefore, the current study was unable to replicate findings by Saunders et al. (2008) and Hooper et al. (2004) who observed more commission errors in males. These results are consistent, however, with Fernie et al. (2010), Reynolds et al. (2006) and a meta-analysis by Cross et al. (2011). In summary, with the exception of the MID, few differences between males and females were observed on the behavioural tasks. Females were slower than males in many instances, which may be indicative of a more cautious approach to the tasks. While this may be very modest evidence of reduced impulsive action in younger females, there is little to suggest from the current data that trait or behavioural impulsiveness accounts for the difference in collision involvement of younger male and female drivers. This was also reflected

in the findings of the comparison of the functional and structural properties of young male and female brains in the current study, discussed in chapters five and six, respectively.

4.4.1.5 Other; relationships between measures of impulsiveness

While many studies have reported no significant relationship between the BIS-11 and performance on the IGT (Christakou, Brammer, et al., 2009; Gorlyn et al., 2013), Gomide Vasconcelos, Sergeant, Corrêa, Mattos, and Malloy-Diniz (2014) found that higher overall score on the BIS-11 related to poorer performance in the forth and fifth blocks of the task. The current study partially replicated this finding, in that higher attentional impulsivity was associated with a greater number of disadvantageous deck selections (i.e., poorer performance) in the fifth block of the IGT. This suggests that individuals with higher attentional impulsiveness do not show as much improvement by the end of the IGT as those with lower levels. Furthermore, a greater preference for immediate over delayed rewards on Kirby's DDQ, when the delayed reward was small, was associated with poorer performance on the IGT overall (IGT score) and in the fifth block of the task. This provides support for the relationship between trait impulsivity and performance on the IGT.

No significant correlations between impulsivity (BIS-11 or Kirby's DDQ) and performance on the MID were observed. Thus, while individual differences in trait impulsivity may impact on the degree of striatal engagement during the MID (as seen in A. Beck et al., 2009), it does not appear to relate to performance on the task. Similarly, no relationship between trait impulsivity and performance on the Go/No Go task were found. Therefore the current study failed to replicate the

correlation between greater commission errors and higher scores on the motor subscale of the BIS-11 that was reported by Keilp et al. (2005).

Some overlap between the performance measures of the tasks was also observed. A greater number of successful inhibitions on the Go/No Go task was positively associated with the number of hits to win targets in the MID. This suggests that individuals who were skilled at one task were also good in the other. This could mean that the cognitive mechanisms necessary to be successful on the tasks are similar. Reaction time to win targets was also positively correlated with the time to select from deck B in the IGT. As deck B is associated with large gains and delayed punishments, and hits to win targets result in monetary feedback, this could indicate that some individuals are more motivated to achieve a monetary reward, which results in faster reaction times.

To investigate the extent to which psychometric and behavioural measures of impulsivity may overlap, Cyders and Coskunpinar (2012) conducted an EFA on several measures, administered to undergraduate students. Their analysis found some overlap between measures but ultimately concluded that they measure discrete aspects of impulsivity as each measure loaded onto separate factors. This was replicated in the current study. Despite the presence of some correlations between the measures, EFA found that the five measures of impulsiveness; the BIS-11, Kirby's DDQ, IGT, Go/No Go and MID tasks, loaded onto separate factors. This likely means that the different measures represent different underlying constructs.

4.4.2 Limitations and future directions

In their study, Cyders and Coskunpinar (2011) summarised several of the strengths and weaknesses with using self-report and behavioural measures to asses impulsivity. Psychometric measures are inexpensive and quick way to measure general traits and present states in individuals and larger groups. However, Cyders and Coskunpinar (2011) also acknowledged that it is relatively easy to manipulate responses to psychometric measures and, therefore, it is difficult to ascertain the extent to which self-reported behaviours actually relate to underlying cognitive constructs. Similarly, Ramírez and Andreu (2006) questioned the validity of the cognitive subscales of the BIS-11, as they do not believe that individuals are able to reliably, and objectively, self-report their own cognitive abilities.

Behavioural measures of impulsivity, on the other hand, are thought to capture a 'snapshot' of how an individual would actually respond in a certain situation, not how they think they would respond (Cyders & Coskunpinar, 2011). However, it is difficult to make predictions about how someone would act/react in a real world scenario, as many other variables are likely to influence action that are not included in the behavioural measure. Furthermore, it has been proposed that some individuals may have cognitive mechanisms that enable them to compensate for higher trait impulsivity, resulting in fewer observable differences in performance on these tasks (Kam, Dominelli, & Carlson, 2012). Consequently, Cyders and Coskunpinar (2011) said that laboratory based tasks too should be considered merely a 'trait' measure of behaviour. Cyders and Coskunpinar (2011) posited that studies might have an increased ability to make predictions if

laboratory measurements are taken across multiple timepoints or even if real world behaviour is measured over a variety of instances to determine whether task performance is limited to predicting outcomes from particular sequences of events. Alternatively, measurements of neural activity, such as electroencephalogram (EEG) or fMRI, could be obtained, as these may be more sensitive to differences in underlying cognitive processes between groups than behavioural measures (Kam et al., 2012). This is be explored in chapter five.

A further issue is the wide variety of psychometric and laboratory-based tools used to measure impulsivity. These measures cover a range of divergent processes associated with executive function, response inhibition, delay of gratification, decision-making and planning, just to name a few (Gomide Vasconcelos et al., 2014). Cyders and Coskunpinar (2011) assessed the validity of certain psychometric and laboratory measures of impulsivity, as well as the degree of overlap between them. 'Nomothetic span', the assessment of the validity of psychometric measures, typically focuses on criterion, content, convergent and divergent validity. Essentially, the extent to which a psychometric questionnaire measures the construct of interest, whether it is similar to other measures of the same construct and dissimilar to measures of unrelated constructs. The validity of behavioural measures is assessed using 'construct representation'. Construct representation is reported less often in the literature, yet it is arguably just as important as nomothetic span, as performance on such tasks is interpreted as an indicator of the individual's ability on a certain cognitive process (Strauss & Smith, 2009). Cyders and Coskunpinar (2011) assessed both nomothetic span and construct representation by measuring the degree of overlap between

psychometric and behavioural measures of the same construct, impulsivity, by means of a meta-analysis. Cyders and Coskunpinar (2011) concluded that, overall, self-report and lab measures of impulsivity are tapping into similar constructs, but weakly. They acknowledge that their study was limited by a potential bias in the literature to only publish statistically significant results, which could have resulted in the exclusion of research that found no relationship between measures. Cyders and Coskunpinar (2011) also found that more diverse samples, such as those with a higher percentage of males, or greater age ranges, showed stronger associations between self-report and laboratory measures. This could reflect genuine group differences in the relationships between measures, or it could mean that issues with sampling impacted on the results obtained.

While a meta-analysis approach is beneficial in determining patterns of findings in the literature, a potential limitation of this method is that these measures were administered across different samples. Applying various psychometric and behavioural measures of impulsivity to the same group of participants may give a more accurate representation of how these measures may relate to one another (Gomide Vasconcelos et al., 2014). Hence, this approach was adopted in a follow up study by Cyders and Coskunpinar (2012). They found only moderate overlap between measures and concluded that each largely assessed a discrete aspect of impulsivity. As such, there is a push in the literature to include both types of measures to ensure that the multidimensional construct of impulsivity is comprehensively assessed in individuals (Cyders & Coskunpinar, 2011, 2012; Gomide Vasconcelos et al., 2014).

While the current study did administer both psychometric questionnaires and computer based tasks of impulsivity to the same group of participants, different results may have been obtained if different measures were used, as demonstrated by meta-analyses of previous findings (Coskunpinar et al., 2013; Cross et al., 2011; MacKillop et al., 2011; Simmonds et al., 2008; Wright et al., 2014). Furthermore, it is problematic for studies to try to compare performance on different behavioural tasks as if they measure the same component of impulsivity (Cyders & Coskunpinar, 2011). Thus, if each subscale and performance measure quantifies a discrete component of impulsivity, then it may not be appropriate to claim that one group has greater overall 'impulsivity' than another, just that they have greater levels of this particular component (G. T. Smith, Fischer, & Fister, 2003). Therefore, while it is clear that future research should continue to include both psychometric and behavioural measures of impulsivity, it is also important to recognise the inappropriateness of generalising the findings from one measure to the overall construct of impulsivity. Moreover, without adopting a 'gold standard' of measures that should be used, inconsistent findings between studies are likely to continue to arise.

A final issue to note is that, while risky and safe drivers in the larger survey sample differed on all four subscales of the DBQ (see chapter two), in the subsample of drivers recruited for the main study only one of these subscales demonstrated a difference between young risky and safe drivers, and two between risky and safe males. It is reassuring that all drivers differed on the HC violations subscale, and males on the aggressive subscale, as this indicates that there was sufficient evidence of a difference in the amount of deliberate aberrant

driving behaviours between groups, rather than just a skill deficit (which would be indicated by differences on the error and lapse subscales). However, as groups did not differ across all four subscales, this suggests that the distinction between the risky and safe groups of the main experiment may not have been clear enough to detect some differences in trait and behavioural impulsiveness. This may have been due to sample size, as the effect sizes for differences in errors and lapses on the DBQ reported in chapter two were very small, indicating that a much larger sample than the present one may be needed to detect significant differences on these subscales. It is also possible, however, that some drivers were misclassified, for example that individuals who take risks while driving were classified as safe, as they had not yet been collision involved and had never been caught committing a penalty point offence by the police. Similarly, individuals may have been culpable in a collision due to lack of skill, rather than deliberate risk-taking, which meant they would have been incorrectly assigned to the risky group of the current study. Future research could utilise in car recording devices, i.e. 'black box' machines, to collect more objective measurements of participants' driving tendencies over a period of time to determine whether they regularly engage in aberrant driving behaviours, or not, when creating experimental groups.

4.4.3 Conclusion

In conclusion, certain aspects of impulsivity were found to relate to age and to a history of risk-taking while driving. Minimal gender effects were observed. The current study found that risky drivers demonstrated lower levels of careful thinking and planning and favoured immediate gains over long-term rewards. Younger risky drivers also appeared to discount future consequences, as

demonstrated by a greater preference for infrequent punishment decks in the IGT. Furthermore, significant associations were found between deliberate driving violations and both motor impulsiveness and a preference for immediate rewards in certain situations. This suggests that the degree to which an individual acts in the present may relate to risky driving behaviours such as speeding, overtaking on the inside and not obeying traffic lights.

This evidence provides justification for the continued investigation of how individual differences in impulsiveness may contribute to risky driving behaviours. As only modest relationships between measures were observed, and all loaded onto separate factors during EFA, each should be interpreted as relating to a discrete component of impulsivity. Future research should continue to include both psychometric and laboratory based measures of impulsivity but caution should be taken when generalising findings from one measure to the overall construct of impulsivity.

5 Chapter Five: A Functional Neuroimaging Study of Impulsivity in Drivers

5.1 Introduction

Decisions made in adolescence can have tremendous consequences for later life (Brand & Schiebener, 2013). Adolescence is characterised by increased impulsivity, risk-taking and reward seeking behaviours (Koob et al., 1998). Early maturation of the reward processing system relative to the immature 'control system' is thought to be responsible for this increase (Casey et al., 2008). The vulnerability this developmental stage brings is often proffered as an explanation for why adolescents and young adults are overrepresented in collision fatalities (e.g. Reyna & Farley, 2006). Despite frequent associations between the two in the developmental literature, this relationship is yet to be addressed directly by neuroimaging research.

Furthermore, while not every adolescent takes risks, research suggests that those that engage in one type of risk-taking are more likely to engage in others (Dryfoos, 1991). There may be neural processes, therefore, that make some younger individuals more vulnerable to risk-taking than others. Moreover, as males continue to be overrepresented in collision fatalities with increasing age, some aspects of impulsiveness may persist into later adulthood. To evaluate this theory, the current study will compare the blood oxygen level dependent (BOLD) responses of younger (18 to 24) male and female drivers and older male drivers (over 29) while they engage in tasks of inhibition, reward anticipation and decision-making under risk. In addition, the current chapter will examine whether individuals with and without a history of aberrant driving vary in their neural response to cognitive tasks examining aspects of impulsiveness. The following

sections explore the literature on the development of impulsivity in adolescence and the neural correlates of risky decision-making.

5.1.1 The neural correlates of impulsivity

Neuroimaging research has investigated the links between various measures of impulsivity and the developing brain in order to identify the neural correlates of impulsivity, which are thought to relate to risk-taking behaviours.

To date the research has suggested that there is a strong link between age-related declines in impulsivity and risk-taking and the structural and functional maturation of brain regions implicated in executive control and the processing of rewards (Casey et al., 2008; Paus, 2005). These brain systems, and what is known about their development, will be discussed in relation to impulsive action and impulsive choice in more detail below.

5.1.1.1 The neural correlates of 'impulsive action'

Impulsive action is associated with poor behavioural inhibition, leading to undesirable consequences of actions (Whelan et al., 2012). Successful inhibition requires cognitive control, which is the ability to read cues from the environment and shift behaviour accordingly (Botvinick, Carter, Braver, Barch, & Cohen, 2001). This requires selective attention and suppressing interference from distracting information (Levin et al., 1991). There is an increase in cognitive control and a decrease in impulsive action behaviours from childhood to adulthood (Casey, Thomas, Davidson, Kunz, & Franzen, 2002; Galvan et al., 2007).

Imaging studies have proposed that this coincides with the maturation of the prefrontal cortex in the mid 20s (Bunge, Dudukovic, et al., 2002; Casey, Galvan, & Hare, 2005; Casey et al., 1997; Durston et al., 2006; Giedd et al., 1999; Luna & Sweeney, 2004; Luna et al., 2001; Moses et al., 2002; Rubia et al., 2000; Sowell et al., 1999; Steinberg, 2004). While primary systems such as motor, somatosensory, visual cortices and other sensory systems develop first (Gogtay et al., 2004; Sowell et al., 2004), integration of these regions and more sophisticated networks develop later (Huttenlocher, 1979).

Successful inhibition is essential for the regulation of goal-directed behaviour as it involves the ability to consider all possible outcomes and the suppression of competing responses or ideas that would be less adaptive (Luna & Sweeney, 2004). The prefrontal cortex (PFC) is said to exert this top-down control over automatic or reward-driven responses in healthy adults (Aron, 2007). As the PFC is one of the last regions of the brain to mature (Bourgeois, Goldman-Rakic, & Rakic, 1994) it is unequipped to exert full top-down control over impulsive actions in adolescents and young adults (Christakou, Halari, et al., 2009). This is proffered as an explanation for the noted increase in impulsive action observed in adolescence.

Functional neuroimaging research has given insight into the regions involved in executive functioning, such as behavioural inhibition, using tasks such as the Go/No Go task. Neuroimaging research utilising this task implicated the following regions in cognitive control processes such as inhibition; the right inferior frontal gyrus (IFG), the dorsolateral prefrontal cortex (DLPFC), the ventromedial prefrontal cortex (VMPFC), the anterior cingulate cortex (ACC) and the pre-

supplementary motor area (SMA) (Behan, Connolly, et al., 2013; C. D. Chambers, Garavan, & Bellgrove, 2009; Garavan, Hester, Murphy, Fassbender, & Kelly, 2006; Kaufman et al., 2003). The right IFG is thought to be responsible for response inhibition where behaviours are initiated or cancelled based on an assessment of all relevant information (van Holst et al., 2012; Whelan et al., 2012). It has also been implicated, along with the ACC and DLPFC, in interference control (Bunge, Dudukovic, et al., 2002; Rubia et al., 2001). Activation of the ACC may be critical for attention (Carter, Mintun, Nichols, & Cohen, 1997), motor response behaviour (Paus et al., 1997) and error correction (Mesulam et al., 2001). The ACC is also thought to be involved in action/conflict monitoring (Carter et al., 1998; Carter et al., 2000), which is dissociable from inhibitory control (Garavan, Ross, Murphy, Roche, & Stein, 2002; Menon et al., 2001). Decreased activation in the ACC has been observed in cocaine users and increased activation in those with obsessive compulsive disorder (OCD) compared to healthy controls (Kaufman et al., 2003), linking evidence of cognitive control impairments to dysfunction of this region.

The VMPFC is involved in considering all of the relevant information in the environment (Bechara et al., 2000) and making a judgement based on preference for a chosen outcome (Paulus & Frank, 2003). Although not located in the PFC, the pre-SMA is also considered to be involved in inhibitory processes. While previously attributed directly to the preparation of actions (Ikeda et al., 1999) and inhibitory control (Floden & Stuss, 2006; Picton et al., 2007), the pre-SMA has recently been considered to be involved in determining the most appropriate course of action to achieve an anticipated reward (Whelan et al., 2012).

Finally, the DLPFC has been shown to regulate a variety of behavioural responses, suppressing those considered inappropriate (Wood & Grafman, 2003). It is involved in the inhibition of behaviours initiated by regions thought to be part of the 'impulse system' (Bechara, 2005), such as the brainstem and basal ganglia. Beeli, Casutt, Baumgartner, and Jäncke (2008) used transcranial direct current stimulation (tDCS), a non-invasive procedure which modulates excitability in regions of interest by altering neuronal membrane potentials (Liebetanz, Nitsche, Tergau, & Paulus, 2002), to modulate DLPFC activation while experiencing a virtual reality. Beeli et al. (2008) found that applying tDCS to the DLPFC modulated levels of impulsivity, as measured by the number of commission errors made during a Go/No Go task. The tDCS down regulated the DLPFC, reducing activation, and interfering with its capacity to exert top-down control over regions thought to be responsible for impulsive behaviours, resulting in an increase in impulsive responding to the Go/No Go task. Beeli et al. (2008) also noted that previous research that increased DLPFC activation with tDCS found a decrease in impulsiveness, providing further support for the function of the DLPFC.

Some studies have demonstrated that improved cognitive control with age is associated with increases in activation in task-relevant brain regions (Bunge, Dudukovic, et al., 2002; Rubia, Smith, Taylor, & Brammer, 2007). This finding, however, is not consistent across studies. Rubia et al. (2000) found that increased frontal activation with age did not significantly correlate with task performance on a stop task, concluding that the maturation of the PFC may involve improving the efficiency of already functional systems. This theory is supported by empirical research by Durston et al. (2006) and a review of the literature by Durston and

Casey (2006) who concluded that activation during tasks of cognitive control gradually moves from diffused to focused with increasing age. This may reflect regional specialisation and lateralisation (Lopez-Larson, Anderson, Ferguson, & Yurgelun-Todd, 2011; Uddin, Supekar, & Menon, 2010). These theories suggest that, even without differences in performance between adolescents and adults, the prefrontal cortex continues to increase in efficiency, resulting in a decline in impulsive action behaviours with age.

5.1.1.1.1 Gender differences in the neural correlates of 'impulsive action'

As males and females are represented differently in collision statistics, the current study was interested in investigating whether there were gender differences in activation between young males and females that would suggest that there is a neurological explanation for these differences. Relatively few fMRI studies have studied sex differences in cognitive functions (Rubia et al., 2013). Despite not finding differences in task performance between males and females, Christakou, Halari, et al. (2009) found differences in activation while participants engaged in a switching task and the Simon task (where participants respond congruously or incongruously to the position of a stimulus on screen). While exhibiting switching behaviour, females demonstrated greater activation in the IFG, caudate, inferior parietal lobe and ACC. As activation in these regions was also observed to increase linearly with age the authors interpreted these between groups differences as indicative of a more mature switching system in females. There is structural evidence to support this functional difference in the caudate, as it has been shown to develop earlier in females compared to males (Giedd, Castellanos, Rajapakse, Vaituzis, & Rapoport, 1997; Sowell, Trauner, Gamst, & Jernigan, 2002). During the

Simon task, activation in the left IFG, middle temporal gyrus and the posterior cingulate cortex (PCC) demonstrated greater activation in females and the right IFG, left premotor and right inferior parietal cortices showed greater activation in males. This study examined these effects over a large age range (13 to 38) but did not examine whether there were any interactions between age and gender. Therefore it is not clear from this research whether these divergences are stable across the lifespan or whether they change across the developmental stages.

Gender differences in the regions engaged in successful inhibitions in the Go/No Go task have been investigated by only a few studies but there is little consensus between their findings. Garavan et al. (2006) found greater activation in the middle frontal gyrus (MFG), inferior parietal lobe, right superior, middle and inferior temporal gyri, thalamus, lentiform and cerebellum in adult females (>18) when they successfully inhibited their responses to 'lure' stimuli in the task. In contrast, J. Liu, Zubieta, and Heitzeg (2012) observed increased activation in the ACC in adult males (>20), relative to females, when they made successful inhibitions compared to errors of commission. Greater activation in the MFG was only observed in females when individual differences in the Zuckerman–Kuhlman Personality Questionnaire (ZKPQ) were regressed from the model. Most recently, in a study of 13 to 38 year olds, Rubia et al. (2013) found that successful inhibitions elicited relatively greater activation of left ventrolateral and superior frontal gyrus (SFG), insula and putamen in females, and the right MFG, right inferior parietal lobe and PCC in males. Discrepancies between these studies may be the result of the various age ranges used. The current study aimed to clarify

these differences by focusing on males and females between the ages of 18 and 24 only.

5.1.1.2 The neural correlates of 'impulsive choice'

Impulsive action and choice are characterised by distinct neural networks (Diergaarde et al., 2008). Impulsive choice is said to relate to the decision-making process better than impulsive action (Winstanley et al., 2006). Winstanley et al. (2006) defined impulsive choice as a propensity towards making decisions that favour a smaller immediate reward, while discounting the value of more distant, larger rewards. This preference for smaller immediate rewards over larger delayed rewards is considered to be the opposite of 'self-control' (Kalenscher et al., 2006). Many adolescents are, characteristically, lacking in this 'self-control', putting a greater emphasis on reward seeking, through the pursuit of immediate rewards, and appearing less sensitive to punishments (Ernst, Pine, & Hardin, 2006).

Casey et al. (2008) argued that if an immature PFC was the sole cause for this preference, and resulting shift in behaviour, then these reward seeking tendencies would peak in childhood, rather than adolescence, and gradually decrease into adulthood. Therefore, while protracted PFC development alone may be an adequate explanation for why impulsive *action* steadily decreases from childhood to adulthood, this theory is unable to account for the increase in impulsive *choice* tendencies observed during adolescence, before a decline into later adulthood. Therefore, higher choice impulsivity in adolescence and young adulthood is thought to be caused by an imbalance between the early maturation of the reward processing systems of the brain relative to the continuing development of the PFC

into the mid 20s (Casey et al., 2008; R. A. Chambers, Taylor, & Potenza, 2003; J. R. Cohen et al., 2010; Steinberg, 2008). This theory is illustrated in figure 5.1 below.

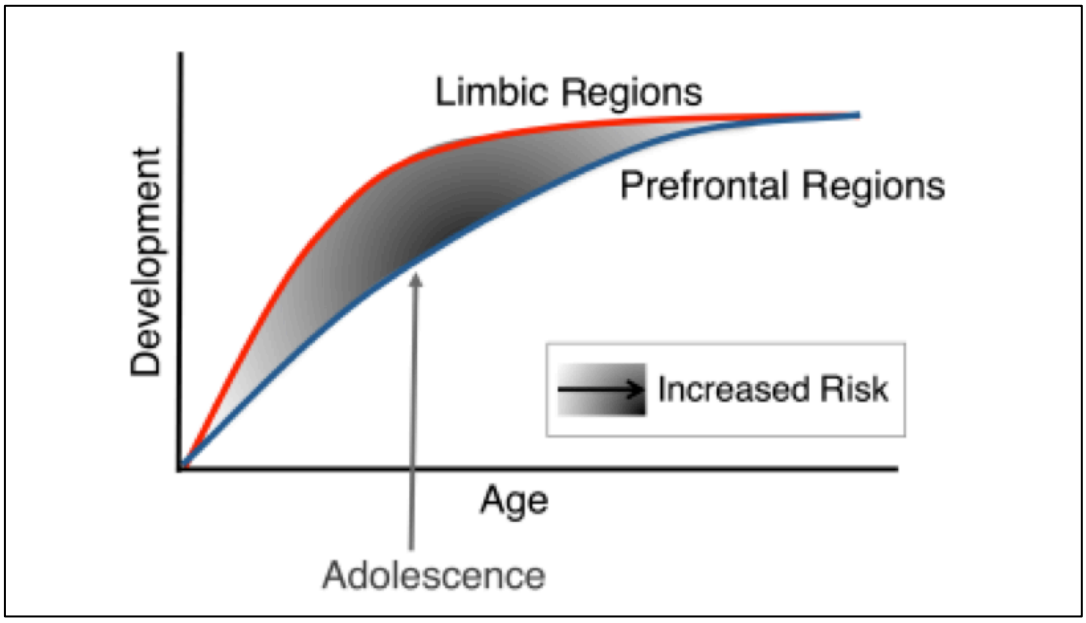

Figure 5.1: Neurodevelopmental model of increased impulsivity and risk-taking in adolescence as a function of the imbalance between the early development of the limbic system relative to the protracted maturation of the PFC (reproduced with permission from Somerville, Jones, & Casey, 2010, pg. 126)

This reward-sensitive dopamine system responds to a number of reward types, including monetary and social rewards (Galvan, 2010), and is implicated in the increase in risk-taking behaviours associated with that developmental stage. The two components of this theory; the reward system and control system, are discussed in further detail below and are typically studied using tasks known to recruit both reward processing and prefrontal regions, such as the Monetary Incentive Delay (MID) task and the Iowa Gambling Task (IGT).

5.1.1.2.1 Reward Processing

In order to understand this theory it is necessary to have an appreciation for the regions of the brain responsible for processing reward and how they act together

in a functioning network. The mesolimbic and mesocortical systems play a major part in the processing of reward (T. A. Zhang, Maldve, & Morrisett, 2006). The mesolimbic system is a dopaminergic pathway associated with feelings of reward and desire. It begins in the Ventral Tegmental Area (VTA) and connects to the limbic system (via the nucleus accumbens, NAcc), the amygdala, the hippocampus and the medial PFC (Doremus-Fitzwater, Varlinskaya, & Spear, 2010).

The VTA is a group of neurons located in the middle of the midbrain and is the primary source of the release of dopamine in the brain (Kolb & Whishaw, 2006). These dopaminergic neurons (D_1 and D_2 receptors) communicate with the PFC whether to initiate or inhibit physiological responses to reward stimuli. They do so by either stimulating (D_1 receptors) or inhibiting (D_2 receptors) the production of cyclic adenosine monophosphate (cAMP; derived from adenosine triphosphate), which is responsible for eliciting a physiological response through intracellular signal transduction (Gazzaniga, Ivry, & Mangun, 2009).

Dopamine neurons fire when an individual receives an unpredicted reward and withdrawal of reward weakens the firing of dopaminergic neurons (Schultz, 2007). However dopaminergic neurons will still fire without receipt of reward if it is associated with a conditioned response. This results in a mismatch between expected and received rewards, known as 'prediction error signal' (Salamone & Correa, 2012). Better prediction error has been found to be associated with efficient cognitive processing. For example, Schönberg, Daw, Joel, and O'Doherty (2007) found that better performance on the Iowa Gambling Task (IGT) was found to correlate to stronger prediction error signals in both dorsal and ventral striatum.

It has been suggested that dopamine in the NAcc acts as a 'pleasure neurotransmitter' (Berridge, 2007, p. 393) and functions to reinforce rewards. The NAcc, located in the ventral striatum, receives input from the VTA. It is thought that the NAcc is responsible for predicting reward (Casey et al., 2008). The NAcc is further subdivided into limbic and motor regions known as the shell and core (Di Chiara, 2005). The NAcc shell regulates motivation and reward and has been implicated in addiction and behavioural inhibition behaviours (Pierce & Kumaresan, 2006) whilst the NAcc core is involved in sensory motor integration, reward processing, processing emotional cues and the regulation of goal-directed behaviour (T. A. Zhang et al., 2006). It has also been shown that Nafadotride (a dopamine antagonist) in the shell of the NAcc heightens impulsive behaviours while Nafadotride in the core reduced impulsivity (Besson et al., 2010).

The mesocortical pathway is thought to be responsible for motivation and emotional responses to reward (Pierce & Kumaresan, 2006). It is another neural pathway, connecting the VTA to the cerebral cortex (Overman et al., 2004). It has particular connections with the frontal lobe and is essential to the normative functioning of the DLPFC (Brodmann areas 9 and 46). Other parts of the network include the orbitofrontal cortex (OFC), thalamus, parts of the basal ganglia, hippocampus and the primary and secondary association areas of the neocortex (posterior temporal, parietal and occipital areas) (Doremus-Fitzwater et al., 2010).

The basal ganglia are particularly important in this network (Pierce & Kumaresan, 2006). They are a collection of forebrain structures, including parts of the ventral and dorsal striatum, that have strong connections to the cerebral cortex (Robbins,

Gillan, Smith, de Wit, & Ersche, 2012). The basal ganglia comprise six nuclei; the globus pallidus (also known as the pallidum), subthalamic nucleus, substantia nigra, nucleus accumbens (ventral striatum), caudate and putamen (dorsal striatum) (Di Chiara, 2005; Doremus-Fitzwater et al., 2010; Pierce & Kumaresan, 2006). The basal ganglia's role in dopamine production and reception is critical for the interpretation and regulation of reward (Gazzaniga et al., 2009). The regions contained in the mesocortical pathway act as an integrated network, which produces certain responses to reward over others. This system of reinforcement was evolutionarily beneficial as it motivates the satisfaction of basic needs that aid in survival (e.g. food to satisfy hunger). However, an imbalance in this system can result in maladaptive behaviours, such as risk-taking, and can be symptomatic of an underlying disorder (Fareri, Martin, & Delgado, 2008).

The MID task (Knutson et al., 2001) is commonly used to study reward processing through two stages; anticipation (also referred to as the 'appetitive' stage) and receipt of reward (also known as the 'consummatory' stage). It has been found to recruit both striatal and frontal regions (Balodis & Potenza, 2015). Activation in the NAcc is consistently observed during reward anticipation (Behan, Stone, & Garavan, 2013; Peters et al., 2011) as well as other aspects of the striatum, such as the caudate and putamen (Knutson et al., 2000; Nestor et al., 2010; Peters et al., 2011; S. Schneider et al., 2012). In contrast, activation in the SMA (Knutson et al., 2000) and in frontal regions such as the VMPFC, OFC, ACC, MFG and DLPFC have been observed upon receipt of monetary reward (Knutson et al., 2001; Knutson et al., 2000; Nestor et al., 2010). Activation during the MID has also been linked to

levels of impulsivity. For example, the degree of ACC activation during 'loss anticipation' was found to negatively correlate with an impulsivity score generated by Patel et al. (2013), which comprised of participants' performance on two cognitive tasks and their answers to five common psychometric measures of impulsivity (including Barratt's Impulsiveness Scale).

5.1.1.2.2 Control/regulation

Successful decision-making and control of reward seeking behaviours involves connections between prefrontal regions (the OFC, ACC, VMPFC and the DLPFC) and the reward systems described above (Fareri et al., 2008). The OFC is seen to be responsible for motivation, identifying salient information about a reward from the VTA and making an appropriate response (Volkow & Baler, 2012). OFC activation has been found to be larger (Miller & Cohen, 2001) and more diffuse in adolescents (Galvan et al., 2006) but becomes more localised and efficient with age. This is consistent with previous neuroanatomical studies (Gogtay et al., 2004; Sowell et al., 2003; Sowell et al., 1999) and functional neuroimaging studies (T. T. Brown et al., 2005; Casey et al., 2005; Casey, Thomas, et al., 2002; Casey et al., 1997; Durston et al., 2006) of prefrontal development.

As mentioned previously, the DLPFC is thought to be responsible for supressing inappropriate behaviour and the VMPFC for considering all of the relevant information in the environment. The VMPFC is also thought to be responsible for considering future outcomes of actions and suppressing the desire to choose immediate rewards (Bechara et al., 2000; Kalenscher et al., 2006). It has, therefore, been implicated in successful decision-making (Davis, Levitan, Muglia, Bewell, & Kennedy, 2004).

To investigate how these systems interact to form 'healthy' reward appraisal in adults, McClure, Laibson, Loewenstein, and Cohen (2004) carried out an fMRI study in which participants had to decide between rewards with varying delays before delivery. They proposed that, distinct from processing the actual value of a reward, there is a separate neural network responsible for adjusting the value of a reward based on the magnitude of the delay. They found that choices, regardless of the delay, recruited the lateral PFC and PCC. However, during choices of immediate rewards activation was observed in paralimbic areas associated with midbrain dopamine neurons (including the NAcc, medial OFC and medial PFC). McClure et al. (2004) concluded that a predisposition to choosing immediate rewards is driven by the limbic system, which is less sensitive to the value of future rewards, and an ability to delay gratification is mediated by the lateral prefrontal cortex and surrounding structures. This theory is consistent with the findings of previous research in that these regions are commonly implicated as responsible for impulsive decision-making (Biederman & Faraone, 2002).

As these prefrontal regions are immature in adolescence, impulsivity and risk-taking are thought to occur due to a reduced capacity for self-regulation (Steinberg, 2004), with a tendency for behaviours to be driven by reward (Galvan et al., 2006). An imbalance between the development of reward processing regions and the PFC, as illustrated in figure 5.1, results in a higher value being placed on immediate rewards and discounting of the value of more distant rewards (Steinberg, 2008). As the prefrontal cortex develops (e.g. through synaptic pruning and myelination, these processes will be discussed in greater detail in chapter six), executive function improves, allowing for greater control

over behaviours (Casey et al., 2005). This results in a decrease in impulsive choice behaviours with age (Hinson, Jameson, & Whitney, 2003), an increased ability to delay gratification (Mischel, Shoda, & Rodriguez, 1989) and a decreased sensitivity to rewards (Cauffman et al., 2010).

Adults, while sensitive to reward, have the capacity to control reward-related urges (J. R. Cohen et al., 2010), whilst adolescents often estimate rewards inappropriately (Ernst et al., 2005; X. Liu, Liu, & Teo, 2003). There is conflict in the literature as to whether this results in increased or reduced activation of reward regions in response to rewarding stimuli, possibly due to subtle differences in experimental and analytical methodologies (Galvan, 2010).

Some studies have found reduced activation in the ventral striatum in adolescents during anticipation of monetary rewards, when compared to adults (Bjork et al., 2004). This finding led the authors to conclude that the reward system in adolescence needs more intense stimulation to achieve adult level gratification, leading to engagement in risk-taking behaviours as a compensatory mechanism.

The prevailing theory, however, is that adolescents are more sensitive to reward, displaying enhanced response to large rewards due to insufficient regulation from the PFC (Casey et al., 2008; Davidson et al., 2004; Ernst et al., 2005; Galvan, 2010; Irwin, 1993; Somerville et al., 2010; Steinberg, 2008). It is suggested that this is not due to differences in motivation, as similar results have been shown in studies where the reward was not contingent on behaviour (Galvan et al., 2006; May et al., 2004). Moreover, as McClure and others have demonstrated, the striatum is implicated in making immediate decisions and the PFC with long-term decision-making (J. Li, McClure, King-Casas, & Montague, 2006; McClure et al., 2004), giving

further support for Casey et al. (2008)'s theory of neurobiological development as a viable explanation for the increase in preference for immediate rewards (increased impulsive choice behaviours) observed in adolescents and young adults.

5.1.1.2.3 Gender differences in the neural correlates of 'impulsive choice'

Functional neuroimaging studies have also demonstrated that gonadal steroid hormones in humans have a modulatory effect on the reward system. With a counter-balanced, repeated measures design, Dreher et al. (2007) found that adult women (>18) demonstrated greater activation of the OFC and amygdala in anticipation of a reward a few days after the end of their menstrual cycle, compared to 10 days afterwards. Furthermore, scans collected the day before ovulation revealed a greater response in the midbrain, striatum, and left fronto-polar cortex upon receipt of reward, compared to the day after. Dreher et al. (2007) also collected data from males in one session to make direct, between-groups comparison of reward anticipation and receipt. In anticipation of uncertain rewards males demonstrated greater activation of the putamen while women showed a greater response in the right hippocampus and left MFG and a greater response in the ACC and left amygdala upon receipt of reward. During reward feedback no regions showed greater activity in males.

In young individuals (19-23) males have been found to exhibit more reward related activation (in the ventral striatum, amygdala, OFC) compared to females during a videogame paradigm (Hoeft, Watson, Kesler, Bettinger, & Reiss, 2008). This may reflect genuine gender differences in reward processing during this age. These results should be interpreted with caution, however, as they may have been

confounded by differences in motivation and engagement in the paradigm. Finally, Diekhof et al. (2012) investigated whether males and females of undergraduate age differed in the neural mechanisms they employed to control impulsive choice behaviours (i.e. preference for immediate rewards). They found no effect of gender in the NAcc when successfully inhibiting preference for an immediate reward, but males demonstrated a significantly greater reduction in recruitment of the pallidum, putamen, temporal pole and ACC. These findings are supported by clinical studies showing differences in dopaminergic pathways from the VTA to the ventral striatum in males and females (e.g. Gillies, Virdee, McArthur, & Dalley, 2014; O'Connell & Hofmann, 2011). The potential differences in the neural correlates of impulsive choice between males and females (18-24) will be further investigated in the current study.

5.1.2 The neural correlates of 'risky decision-making'

The ability to make advantageous decisions is important to successful functioning (Brand & Schiebener, 2013). A variety of cognitive and emotional processes influence decision-making. Risk-taking, experiencing and evaluating immediate versus delayed wins and losses, and impulsivity have been found to constitute the multi-faceted concept of decision-making (Krawczyk, 2002).

The previous sections have dealt with the prevailing theory for why increased impulsivity and risk-taking are observed in adolescence. The link between impulsivity and risk-taking is not exclusive to adolescents, however. Heightened impulsivity, even in adulthood can lead to engagement in risk-taking behaviour (Bayard et al., 2011; Black, Serowik, & Rosen, 2009; Chamorro et al., 2012; S. Fischer et al., 2008; Verdejo-Garcia, Lawrence, & Clark, 2008). There is quite an

overlap between the neural correlates of impulsivity studied in adolescents and regions implicated in risky decision-making in adults.

When adults make risky verses non-risky decisions a network of activation that includes orbitofrontal, lateral frontal and parietal regions, along with the caudate, thalamus and cerebellum has been observed (Gilman, Smith, Ramchandani, Momenan, & Hommer, 2012; Krain, Wilson, Arbuckle, Castellanos, & Milham, 2006). When the outcome of such decisions has been measured, positive feedback (or wins) was associated with activation in the medial OFC, ACC (Jung et al., 2010) and the NAcc (Knutson & Cooper, 2005; Knutson et al., 2001), and negative feedback (or losses) with the insula (Mohr, Biele, & Heekeren, 2010), pre-SMA, DLPFC and lateral OFC (Jung et al., 2010). During decision-making these PFC regions are thought to integrate effort coding and risk costs (potential losses), associated with the SMA and insula, respectively (Burke, Brünger, Kahnt, Park, & Tobler, 2013).

Galvan et al. (2007) found that greater NAcc activation was correlated with a tendency to associate positive consequences with risky behaviours (as assessed by a modified version of the Benthin Risk Perception Measure by Gardner & Steinberg, 2005). Similarly, in this measure, greater ratings of the negative consequences of risk-taking were associated with reduced activation of the NAcc. Together this suggests that the NAcc may influence perception of the cost of risks, with hyperactivity of the NAcc leading to positive associations between risk and reward and hypoactivity leading to negative associations.

Most day-to-day decisions are made without certainty (Brand & Schiebener, 2013). The IGT is thought to mimic real life decision-making, containing periods of

uncertainty and risk, with reward and punishment feedback (Bechara et al., 1994; van Holst, van den Brink, Veltman, & Goudriaan, 2010). In order to be successful in the task participants must forgo the short-term benefits of the 'risky' decks, which deliver immediate, large rewards but result in long-term net losses. For naive participants, the first two blocks (40 trials) of this task are considered to reflect decision-making under ambiguity or uncertainty, where the probability of a loss is unknown and, therefore, the participant makes a decision without certain knowledge of the outcome. Selections made during the next three blocks (trials 41 to 100) demonstrate risky or safe decision-making, as it is likely that the probability of achieving a net gain on each deck should have been discerned. Selecting either of the two decks that have a high probability of a small, net gain is considered to be a 'safe' choice, while selections from the two decks that have a high probability of a large gain on each trial, but an overall likelihood of a net loss are deemed 'risky' selections (Krain et al., 2006). Across studies, the IGT has been found to recruit the following regions; amygdala, ACC, PCC, SFG, MFG, IFG, dorsal and ventral striatum, insula, and OFC (X. Li et al., 2010). Further evidence suggests that decisions made under risk (blocks three through five) may involve the 'affective' or 'motivational' system, representing a network of regions including the OFC, MFG, insula, ACC, caudate, middle temporal gyrus, inferior and superior parietal lobes (Kerr & Zelazo, 2004; Krain et al., 2006).

Although the IGT is commonly used to study the neural correlates of decision-making under ambiguity and risk, how performance on this task relates to the construct of impulsiveness is unclear. While several studies have found that increased impulsivity is associated with poorer performance on the task (Billieux,

Gay, Rochat, & Van der Linden, 2010; Franken et al., 2008; Kjome et al., 2010; Tomassini et al., 2012; Zermatten, Van Der Linden, D'Acremont, Jermann, & Bechara, 2005), several have not (Bayard et al., 2011; Gorlyn et al., 2013; Perales, Verdejo-Garcia, Moya, Lozano, & Pérez-Garcia, 2009; Young, Gudjonsson, Goodwin, Perkins, & Morris, 2013). This lack of consensus may be due to the varied measures of impulsivity employed by these tasks, ranging from cognitive measures like the Matching Familiar Faces test (MFFT), to psychometrics like the revised NEO Personality Inventory (NEO-PI-R), the Temperament and Character Inventory, revised (TCI-R), the urgency, premeditation, perseverance and sensation seeking scales (UPPS) and Barratt's Impulsiveness Scale (BIS-11). Of these measures, negative associations were more consistently found between IGT performance and the UPPS (Billieux et al., 2010; Zermatten et al., 2005) and the BIS-11 (Kjome et al., 2010; Tomassini et al., 2012). Despite this discrepancy, the usefulness of the IGT in assessing decision-making in drivers in the current study remains.

Limited neuroimaging research has attempted to differentiate between regions responsible for risky and safe decisions made while driving. For example, when resolving ambiguity in a driving task, Callan, Osu, Yamagishi, Callan, and Inoue (2009) found reduced activity the amygdala and the ACC. However, in a study of simulated driving in mid-adolescents (14 to 16 year olds), Kahn, Peake, Dishion, Stormshak, and Pfeifer (2015) found that ventral striatal activation preceded risky decisions (to 'go' when faced with an obstacle) and right IFG activation before safe decisions (to 'stop' when faced with an obstacle). In this study, the degree of activation in the right IFG was negatively correlated with scores on Zuckerman's

Sensation Seeking Scale (ZSSS; Zuckerman, 2007) but not the BIS-11. Although these regions represent only a small portion of what has been observed in the wider 'risky decision-making' literature mentioned above, they may be of particular relevance to the current study.

5.1.3 Summary and hypotheses

5.1.3.1 Hypothesis one: there are changes in activation with age

As previously mentioned, the PFC is said to continue to develop into the mid 20s. Increased impulsivity, reward seeking and risk-taking behaviours in adolescence are attributed to this immature prefrontal 'control' system relative to a more mature reward system (Casey et al., 2008). Young males are involved in more collisions than any other demographic group. While older males are also overrepresented when compared to age matched females, the rate of collision involvement declines with age in males. The current hypothesis investigated whether this decrease in collision involvement with age in males may be due to decreasing neurological impulsiveness, coinciding with the maturation of the PFC. It was hypothesised that older adolescent and young adult males (ages 18 to 24) would differ from older males (over 29) in the neural mechanisms by which they make decisions under risk, respond to reward stimuli and inhibit actions, but not to the same extent that has been previously reported in younger adolescent samples (12 to 17 years of age).

5.1.3.2 Hypotheses two to four: there are activational differences between risky and safe drivers

The impact of impulsiveness on driving risk was also investigated in the current study. The neural correlates of impulsivity, as measured through the Go/No Go, IGT and MID tasks, were compared between individuals with and without a history of risky driving to determine whether there may be neural mechanisms that are associated with individuals who engage in more aberrant driving behaviours. Specific conditions within the three tasks were examined using first level contrasts of primary interest in the fMRI analysis. These contrasts were chosen as it was hypothesised that they measured neural processes related to impulsive choice and action that, if compromised, may contribute to risk-taking while driving.

As discussed in chapter one, impulsive choice is defined as a tendency towards overestimating the value of immediate rewards and discounting the value of more distant rewards and punishments (Paterson et al., 2012). Research has observed that the longer a reward/punishment is delayed the greater its value/severity is discounted (Winstanley et al., 2006). Increased impulsive choice in drivers may manifest in the taking of risks to achieve gratification, such as speeding to reach a destination faster, without regard for the potentially large, negative consequences of such behaviour. Therefore, the current study employed first level contrasts to measure the neural responses of drivers for rewarding compared to non-rewarding stimuli ('win cue > neutral cue' and 'hit > miss' in the MID), their activity in response to monetary gains over losses/failure to receive a monetary gain ('win > loss' in the IGT and 'win hit > win miss' in the MID, respectively) and

responses to risky decisions without consideration of the consequences ('bad > good deck selections under risk', 'bad > good win feedback' and 'infrequent > frequent punishment deck loss feedback' from the IGT).

Impulsive action manifests in errors, such as premature responding to stimuli, otherwise referred to as behavioural disinhibition (Sun et al., 2012). It is also defined as a failure to suppress or inhibit actions (Winstanley et al., 2006). Under-recruitment of inhibitory and error monitoring circuitries in the frontal cortex may result in more errors while driving. To investigate whether participants with and without a history of aberrant driving demonstrated differences in recruitment of these circuitries, groups were compared on a linear contrast of successful inhibitions over commission errors ('successful no go > failed no go' in the Go/No Go task).

As a study of this nature had not been reported in the literature previously, certain secondary and exploratory contrasts were also generated to investigate further the potential relationship between driving risk and neural impulsivity. The results of these analyses are considered preliminary and should be interpreted with caution. All primary, secondary and exploratory contrasts are listed in section 5.2.5.5.

Three analytical models were employed in order to investigate whether such vulnerabilities, should they exist, were age or gender dependent. One model combined all of the young (18 to 24) male and female participants of the study, the second included only young males and the third combined younger and older male participants. The reasoning behind such models is outlined in further detail below.

5.1.3.2.1 Hypothesis two: there are activational differences between risky and safe younger drivers, independent of gender

The period of 18 to 24 years of age is associated with increased male and female driver fatalities. This age group is overrepresented in fatal collisions statistics compared to other demographic groups. For example, in a 2015 report of Irish collisions in 2014 it was found that 25% of driving fatalities occurred in this age category, despite only representing 10.6% of the driving population (RSA, 2014). It was therefore hypothesised that, independent of gender, 'risky' and 'safe' young drivers would differ in the intensity of their responses to the tasks employed by the current study on the contrasts indicated in section 5.1.3.2 above.

5.1.3.2.2 Hypothesis three: there are activational differences between risky and safe young male drivers

Young male drivers are particularly prevalent in collision statistics. A detailed report compiled in 2012 in Ireland found that more than three times as many young males were killed in collisions than young females (RSA, 2013). Therefore, younger males were of particular interested in the current study and were examined in isolation of young female drivers to increase the power of the fMRI analysis to detect differences between these groups. It was predicted that this approach would reveal additional differences in the contrasts highlighted in section 5.1.3.2 that may have been masked in the previous model.

5.1.3.2.3 Hypothesis four: there are activational differences between risky and safe male drivers, independent of age

While collision involvement in general appears to decline with age, older male drivers (30 to 54) were still involved in 2.5 times more fatal collision than females of the same age in 2011 in Ireland (RSA, 2012b). It is possible that aspects of impulsiveness persist into later adulthood in some males but not in others, manifesting in risk-taking behaviours. It was hypothesised, therefore, that some activation differences in primary contrasts (see section 5.1.3.2) between risk groups would be evident independent of age, i.e. they would be present when older and younger males were combined, and others would demonstrate an interaction between age and risk, such that age impacted on the differences between risky and safe male drivers.

5.1.3.3 Hypothesis five: there are gender differences in activation

Young males (18 to 24) were involved in 1.84 times as many collisions, and 3.2 times as many fatal collisions, as young females in Ireland in 2012 (RSA, 2013). If heightened impulsivity contributes to risky driving then there may be gender differences in activation that could explain this overrepresentation of young males. The current study attempted to address this by comparing activation between males and females between the ages of 18 and 24 during tasks assessing facets of impulsiveness. Little consensus has been reached in the literature to date about how the neural correlates of impulsivity differ between males and females. Therefore it was hypothesised that, within such a narrow age band, few differences would be detected.

5.1.3.4 Hypotheses six and seven: there are correlations between activation and other measures

The degree to which activation in regions associated with impulsiveness vary with increased impulsivity, as measured psychometrically (i.e. the BIS-11 and Kirby's Delay Discounting Questionnaire, DDQ) and through performance on the cognitive tasks (the MID, IGT and Go/No Go tasks) was also investigated. It was expected that higher levels of psychometric and behavioural impulsivity would be associated with increased 'neural' impulsiveness, i.e. greater recruitment of reward regions and reduced activity in prefrontal regions.

Finally, the relationship between activation in these regions and responses to the Manchester Driver Behaviour Questionnaire (DBQ) was examined as an alternative quantification of risky driving to the categories employed for between groups' analyses. It was expected that greater reporting of risk-taking on the driver behaviour questionnaire would be positively associated with increased 'neural' impulsiveness.

5.2 Method

5.2.1 Design

The main study used functional magnetic resonance imaging (fMRI) to investigate the relationship between age, gender, driving risk and impulsivity. It utilised a between groups design with the participants previously reported in chapters three and four. As before, groups were divided according to particular combinations of age, gender and driving risk. This resulted in the following six experimental groups: young safe/risky males (aged between 18 and 24), young

safe/risky females (between 18 and 24) and older safe/risky males (between 29 and 56).

Participants completed three tasks that measured different aspects of impulsivity while being scanned with fMRI. The BOLD response was measured as a proxy for brain activity and this was compared across groups using three models, outlined below.

5.2.2 Participants

For full details of the participants used in this chapter, see section 3.2. After further inspection, it was determined that two participants were inappropriate for inclusion in the analysis of one or more of the functional tasks. For the IGT, a risky young male (age 21) was found to have more than 10% motion and was therefore not included in the analysis of the IGT only. Furthermore, a safe young female (age 20) was found to have an artefact in the PFC in functional scans obtained during the runs of the MID and the Go/No Go task that unduly influenced the models produced during second level analysis. No such artefact was observed for this participant in the IGT. As data from at least one of the three tasks remained for these participants, they were not discarded from the analysis as a whole, only the affected tasks.

5.2.3 Procedure

Participants completed one run of the IGT and two runs of both the MID and Go/No Go tasks while being scanned with fMRI. As mentioned in chapter four, the MID and Go/No Go tasks were performed over two runs to allow participants to rest, while still sampling enough of each condition to calculate its hemodynamic

response function (HRF). Participants were not mislead to believe that their performance on the MID or IGT would determine the amount of money they received. Further details on the tasks and their scanning procedures can be found below. For a full account of the study's procedure see section 3.4.

5.2.4 Apparatus and materials

5.2.4.1 Functional MRI tasks

5.2.4.1.1 The Iowa Gambling Task (IGT)

The current, event related, version of the IGT was modified for use with the scanner by X. Li et al. (2010). The task was 15 minutes long (900 seconds, 450 scans, time resolution parameter (TR)=2 seconds), during which time participants made a total of 100 selections. On each trial participants were asked to select from one of four decks of cards, A, B, C and D, each containing 60 cards. This task is described in more detail in sections 4.1.1.3.1 and 4.2.3.2.1. The task is illustrated in figure 4.1 The IGT was presented in Matlab on an external laptop (for more details see section 5.2.4.2.2).

5.2.4.1.2 The Monetary Incentive Delay Task (MID)

Whilst in the scanner participants completed two runs of the MID, written in Neurobehavioral Systems (NBS) Presentation® (for more details see section 5.2.4.2.2) by Nestor et al. (2010). The task utilised an event related design. Nestor et al. (2010) proposed that the fixed, invariant delay between anticipatory cue and trial outcome in the original version by Knutson et al. (2001) may cause the activations associated with these unique conditions to overlap. Their modified

version applied extended temporal jittering between anticipatory cues, targets, feedback and the commencement of the next trial in order to avoid such a contamination of signals.

The current version, while still written by Nestor et al. (2010), had been modified since the original publication to exclude the loss avoidance condition. Previous studies that have not used a 'loss avoidance' condition in the MID have included a third condition, such as a non-response condition (Knutson et al., 2001) or a second win reward magnitude (Bjork, Smith, et al., 2012; Muller et al., 2013). However, as such studies subsequently combined monetary win trials for analysis, the current study chose to retain only two trial types, win (50c) and neutral (no reward), to have more instances of these conditions and, hence, have greater power to detect win and neutral effects.

The task was almost five minutes long (290 seconds, 145 scans, TR=2 seconds) and consisted of 27 trials, 14 win and 13 neutral trials. Trials alternated between win and neutral, with win trials appearing both first and last in the task. Identical versions of the task were used for both runs. Each trial consisted of the following stages: cue, target, feedback and rest. The task is illustrated in figure 4.2.

The colour of the cue reflected the nature of the trial, either win or neutral. Win trials were represented by a green box and indicated that money (50 cent) could be won on that trial. A blue box indicated that there was no money at stake for the trial (neutral trial). Cue stimuli were presented on screen for between two and eight seconds, with trials jittered to make target onset less predictable for the participant, as well as making later hemodynamic response (HDR) deconvolution easier.

Following the cue a target (a white star in a white circle) appeared for between 300 and 600ms. Participants were instructed to hit as many targets as possible by responding quickly when the target appeared on screen. Contrary to previous research (for example see Muller et al., 2013), this target window was not tailored to individual reaction times during a practice run. This meant that every participant completed an identical version of the task.

Participants then received feedback to say whether they had successfully responded within the target window or not ('hit' or 'miss'). If they successfully responded to a target that followed a win cue they saw an image of a 50-cent coin, this was referred to as 'win hit' feedback. The other feedback stimuli were of either happy or sad faces, a happy face followed a neutral hit and a sad face followed both win and neutral 'misses'. Feedback was presented for 1.5 seconds. Accumulative feedback, i.e. the total number of hits/misses, was not provided to participants. During the rest stage a crosshair was displayed on screen for between two to eight seconds before the onset of the next cue. All stimuli in this task were presented on a black background.

5.2.4.1.3 The Go/No Go Task

The present version of the Go/No Go task was written in NBS Presentation® (for more details see section 5.2.4.2.2) by Kaufman et al. (2003). All Participants completed two runs of the task (also known as the 'XY' task: Kaufman et al., 2003), each lasting just less than four and a half minutes (264 seconds, 132 scans, TR=2 seconds).

The Go/No Go task utilised an event related design. Alternating 'X's and 'Y's appeared on screen. Letters appeared in white on a black background. The task is illustrated in figure 4.3. Participants were instructed to respond to every letter they saw, unless the letter was a repeat of the previous one. Each run contained 250 trials, with 1000ms between stimuli. Trials in which a repetition occurred were referred to as lure or 'no go' trials and occurred in 10% of cases (225 'go' and 225 'no go' stimuli). The order in which the 'no go' stimuli were presented varied between run one and run two, to mitigate practice effects.

5.2.4.2 Hardware & Software

5.2.4.2.1 Magnetic Resonance (MR) Imaging procedure

Scanning was conducted on a Philips Achieva 3.0 Tesla MR system. A 32 channel Head coil with mounted mirror reflected a 1280x1024 pixel display that was projected onto a panel behind the participant's head outside the magnet. An initial reference scan allowed for the resolution of sensitivity variations. All imaging utilised a parallel sensitivity encoding (SENSE) approach with a reduction factor of 2.5 (Pruessmann, Weiger, Scheidegger, & Boesiger, 1999).

180 high-resolution T1-weighted anatomic MPRAGE transverse images (FOV 230mm, thickness 0.9mm, voxel size 0.9x0.9x0.9) were then acquired (total duration 343 s) to allow subsequent activation localization and spatial normalization. Functional images for each task consisted of 40 non-contiguous (0.3mm gap) 3mm transverse slices covering the entire brain. Images were acquired using a T2* weighted echo-planar imaging sequences (TR=2000ms, TE=25ms, FOV 240 mm, 80x80 matrix size in Fourier space).

5.2.4.2.2 Functional MRI tasks

The MID (Nestor et al., 2010) and Go/No Go (Kaufman et al., 2003) tasks were performed using NBS Presentation® software (Version 0.70, www.neurobs.com) on the 'float' computer of the MR system within Trinity College Institute of Neuroscience (TCIN), a Dell Precision T5500, running on a Windows 8 operating system. The IGT paradigm was written by X. Li et al. (2010) in Matlab ("Matlab: MATLAB and Statistics Toolbox Release," 2014a) based on the Psychtoolbox extensions (Brainard, 1997). This task was performed on a MacBook Pro, OS X 10.9.4, laptop, connected to the MR system and external monitors through an Apple Inc. Mini DisplayPort to Video graphics adapter (VGA).

Participants responded to the tasks using a 2 x 2 Fiber-Optic response pad, entirely made of plastic. The buttons in the left response pad were coloured blue (button one) and yellow (button two) and in the right they were green (button three) and red (button four), see figure 5.2 below.

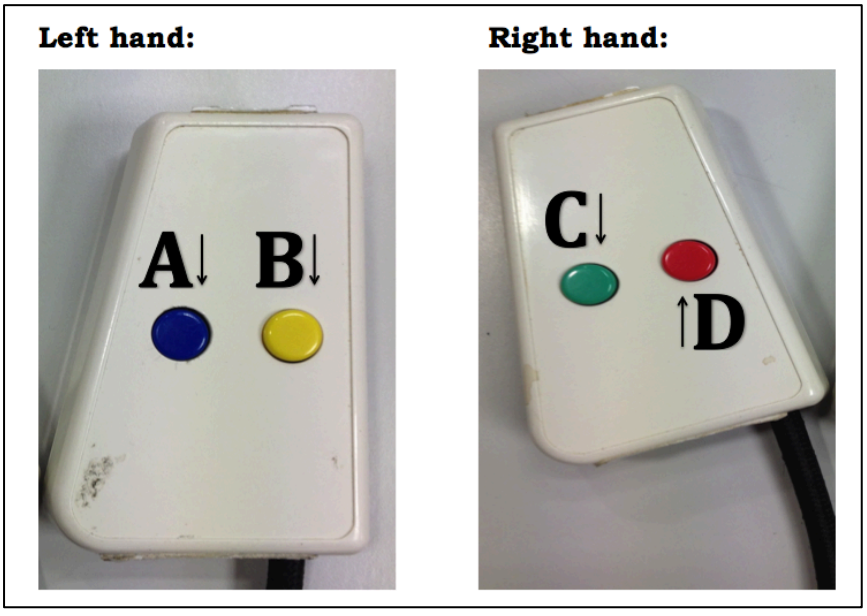

Figure 5.2: 2 x 2 response pad

Participants used all four buttons for the IGT, with blue = deck A, yellow = deck B, green = deck C and red = deck D. For both the MID and Go/No Go tasks participants only used button three, the green button, regardless of handedness.

5.2.5 Data preparation and analysis

5.2.5.1 Preprocessing

Analysis of fMRI data was conducted in Matlab 2014a ("Matlab: MATLAB and Statistics Toolbox Release," 2014a) using Statistical Parametric Mapping (SPM), version 12 (SPM12, 2014). Upon the recommendations made by SPM manuals (for example the SPM starter's guide by Hermans, 2011) and books on functional MRI data analysis techniques (see Frackowiak et al., 2003; Poldrack, Mumford, & Thomas, 2011) the imaging data underwent the following preprocessing steps: slice timing, realignment, coregistration, segmentation, spatial normalisation and smoothing.

Initial scans: The first five scans from each task were then discarded from later analysis to mitigate the effects of the scanner warming up.

Slice timing: the 40 continuous slices (acquired in ascending order) were corrected to account for acquisition timing. As it is recommended to correct to the middle slice (20 or 21 in this instance), slice 20 was chosen.

Realignment: as a certain degree of motion is to be expected in any scan, scans were then subjected to 'Realignment: Estimate & Reslice' to reorient images to the sixth scan (now the first scan), using SPM12's default settings. Motion parameters

were preserved for inclusion in the design matrices as regressors of no interest. This was done in an effort to negate motion related variance, post realignment.

Coregistration: as the first indirect step in normalisation, the data underwent two rounds of coregistration (using the default settings from 'Coregistration: Estimate' in SPM12), one to estimate the coregistration between the T1 images and the mean resliced T2* weighted images, and the second to estimate the coregistration between all resliced T2* weighted images, the T1 images and the MNI template located in the templates folder in SPM12.

Segmentation: the cerebral spinal fluid (CSF), white matter (WM) and grey matter (GM) of T1 images were segmented based on the Montreal Neurological Institute (MNI) template and the warp field calculations were stored for use in normalisation (second indirect step of normalisation). Segmentation was carried out using default settings in SPM12.

Normalisation: the normalization step warps each individual subject into standard space based on the MNI template. This was done separately for the structural and functional images using 'Normalise: Write'. Realigned and resliced functional images were normalised using default settings (voxel size = 3 x 3 x 3mm), while structural images were normalised using the 4^{th} Degree B-Spline interpolation method (1 x 1 x 1mm).

Smoothing: a smoothing filter was applied to the functional images using default settings. The smoothing step reduces noise and spatial differences between individuals, which is useful when making comparisons between groups.

5.2.5.2 Failed normalisation

Upon inspection of the data it was noted that normalisation failed for some participants, as realignment failed to relocate the crosshairs to the anterior commissures (Brett & Rorden, 2009), as defined by Talairach coordinates (Duvernoy, 1999; Talairach & Tournoux, 1988). This necessitated manual realignment in some participants, in which translation and rotation were manipulated using the 'Display' function in SPM12. Images were successfully realigned when the origin of the cross hairs (x, y, z coordinates 0, 0, 0) was located at the anterior commissures. Upon completion, coregistration and normalisation steps were repeated and the data inspected to confirm that normalisation had been successful.

5.2.5.3 Excessive motion

While the realignment technique of SPM is robust enough to handle slow, drift motion (Barch & Braver, 2013), the ArtRepair toolbox in SPM (by Mazaika, Hoeft, Glover, & Reiss, 2009) was used to identify rapid motion and bad volumes in the tasks. Scan to scan motion below 1mm/TR was considered acceptable, while scans in excess of this were flagged as outliers. Upon the advice of Mazaika, Whitfield-Gabrieli, and Reiss (2007), outlier volumes were repaired in smoothed scans (swra^.nii files) using an interpolation method.

These volumes could not be removed in SPM, as they can be with other programs such as FSL (FMRIB Software Library; Jenkinson, Beckmann, Behrens, Woolrich, & Smith, 2012), but instead were deweighted ('regressed out') in General Linear

Model (GLM) estimation to maintain unbiased estimates (Lockwood et al., 2013; Mazaika et al., 2007).

As previously mentioned, two young female participants had excessive motion in more than 10% of scans across all three tasks and so were discarded from the analysis and replaced with new participants, matched for age, gender and risk status. In addition, one young male participant was found to have more than 10% excessive motion in the IGT only, and so was only removed from the analysis of this task.

5.2.5.4 Design Matrices

Design matrix specification occurred on preprocessed scans. All designs were estimated using an autoregressive (AR(1) level autocorrelation) model and the HRF was determined using the canonical approach with no derivations. A high pass filter (HPF) of 128Hz, the default in SPM, was also applied. A detailed description for how the timepoints used in the design matrices were calculated can be found in section 4.2.4.2.

5.2.5.4.1 Iowa Gambling Task (IGT)

In addition to the motion parameters generated during realignment the columns outlined in table 5.1 were entered into the IGT design matrix. All durations for the above conditions were set to zero. If excessive scan-to-scan motion was present, an additional column containing a vector to deweight these problem scans was added to the design matrix. An example of the design matrix for the IGT can be seen in appendix X (participant 7).

5.2.5.4.2 Monetary Incentive Delay Task (MID)

Runs one and two of the MID were modelled as separate sessions in the design matrix. Each session contained the preprocessed scans for that run, a motion parameter file, six conditions with their respective onset times and, where applicable, their durations. Again, excessive scan-to-scan motion was accounted for using a deweighting vector. The six conditions modelled in each run of the MID can be found in table 5.2. The durations of win and neutral cues varied between two and eight seconds so these were also modelled in the design matrix. Feedback conditions were modelled as zero duration as these lasted for less than a second. An example of the design matrix for the MID can be seen in appendix Y (participant 7).

5.2.5.4.3 Go/No Go Task

Runs one and two of the Go/No Go task were also modelled as separate sessions. Similar to the MID task, each session contained the preprocessed scans, a motion parameter file and a deweight vector for excessive motion, where necessary. As Successful 'go' trials occurred only milliseconds apart these were treated as the baseline condition. This left three conditions to be included in design matrix modelling. When participants failed to respond to 'go' stimuli this was referred to as a 'Failed go' trial. No go trials were separated into whether participants successfully inhibited their response or made a commission error (responded to the lure stimulus). These conditions are summarised in table 5.3 below and were all defined as having durations of zero. An example of the design matrix for the Go/No Go task can be seen in appendix Z (participant 7).

Table 5.1: Description of the columns in the IGT design matrix

♯	**Column descriptions**
1	Deck A: Times participants selected from deck A
2	Deck B: Times participants selected from deck B
3	Deck C: Times participants selected from deck C
4	Deck D: Times participants selected from deck D
5	Win A: Onset times of win feedback from deck A
6	Win B: Onset times of win feedback from deck B
7	Win C: Onset times of win feedback from deck C
8	Win D: Onset times of win feedback from deck D
9	Loss A: Onset times of loss feedback from deck A
10	Loss B: Onset times of loss feedback from deck B
11	C(Win>Loss): Onset times of loss feedback from deck C, when the selection resulted in a net gain
12	C(Loss>Win): Onset times of loss feedback from deck C, when the selection resulted in a net loss
13	Loss D: Onset times of loss feedback from deck D

Table 5.2: Description of the columns in the MID design matrix

♯	**Column descriptions**
1	Win Cues: Onset times of win stimuli.
2	Neutral Cues: Onset times of neutral stimuli.
3	Win Hit: Onset times of hit feedback to win targets
4	Win Miss: Onset times of miss feedback to win targets
5	Neutral Hit: Onset times of hit feedback to neutral targets
6	Neutral Miss: Onset times of miss feedback to neutral targets

Table 5.3: Description of the columns in the Go/No Go task

♯	**Column descriptions**
1	Failed 'Go': Onset times of failed 'go' responses
2	Successful 'No Go': Onset times of successful inhibitions
3	Failed 'No Go': Onset times commission errors

5.2.5.5 First Level Analysis

First level analysis was used to produce summary data for contrasts of interest for each individual participant. The results of this analysis were used as raw data for second level analysis. The following tables contain a full list of the t-contrasts applied to each participant at first level for the three tasks; table 5.4 for the IGT (where 'LD' is used as an abbreviation for 'loss deck'), table 5.5 for the MID and table 5.6 for the Go/No Go task.

5.2.5.6 Group Specification & Second Level Analysis

Only the results of the second level analysis are reported in the results section of this chapter. Group level analysis was conducted in the latest version of SPM, SPM12 (SPM12, 2014). Similar to the approach utilised for the analysis of the psychometric and cognitive data, two analytical models were applied to the fMRI data for group level analysis using a GLM approach; a 2 x 2 Factorial Analysis of Variance (ANOVA) (gender x risk status) in younger individuals ("younger individuals" model), and a 2 x 2 Factorial ANOVA (age x risk status) in males ("males" model).

Table 5.4: IGT first level contrasts

♯	**Primary contrasts**	♯	**Secondary contrasts**
1	**Bad > Good (under risk)**	5	Bad > Good deck selections
2	**All win > All loss feedback**	6	Bad > Good deck loss feedback
3	**Bad > Good deck win feedback**	7	Infrequent LD > Frequent LD win feedback
		8	Infrequent LD > Frequent LD selections
4	**Infrequent LD > Frequent LD loss feedback**	♯	**Exploratory contrasts**
		9	Bad deck selections (A+B) > rest
		10	Good deck selections (C+D) > rest
		11	Frequent LD selections (A+C) > rest
		12	Infrequent LD selections (B+D) > rest
		13	Bad deck win feedback (A+B) > rest
		14	Good deck win feedback (C+D) > rest
		15	Frequent LD win feedback (A+C) > rest
		16	Infrequent LD win feedback (B+D) > rest
		17	Bad deck loss feedback (A+B) > rest
		18	Good deck loss feedback (C+D) > rest
		19	Frequent LD loss feedback (A+C) > rest
		20	Infrequent LD loss feedback (B+D) > rest
		21	All decks win feedback > rest
		22	All decks loss feedback > rest
		23	A feedback (win + loss) > rest
		24	B feedback (win + loss) > rest
		25	C feedback (win + loss) > rest
		26	D feedback (win + loss) > rest

Table 5.5: MID first level contrasts

♯	**Primary contrasts**	♯	**Secondary contrasts**
1	**Win Cue > Neutral Cue**	4	Win Cue > Win Hit
2	**Hit > Miss**	5	Win Cue > Win Miss
3	**Win Hit > Win Miss**	6	Neutral Cue > Neutral Hit
		7	Neutral Cue > Neutral Miss
		♯	**Exploratory contrasts**
		8	Win Cue > rest
		9	Neutral Cue > rest
		10	Hit > rest
		11	Miss > rest
		12	Win Feedback > Neutral Feedback
		13	Win Hit > rest
		14	Win Miss > rest
		15	Neutral Hit > rest
		16	Neutral Miss > rest

Table 5.6: Go/No Go first level contrasts

♯	**Primary contrasts**	♯	**Secondary contrasts**
1	**Successful No Go > Failed No Go**	2	Successful No Go > Failed Go
		3	Failed No Go > Failed Go
		♯	**Exploratory contrasts**
		4	Failed Go > Rest
		5	Successful No Go > Rest
		6	Failed No Go > Rest

These models were applied using the 'Full Factorial' approach through Factorial design specification and model estimation at the second level, which generates all main effects and interactions. Separately, a two-sample t-test was utilised to compare younger risky and safe male drivers directly, to remove any impact young females and older males may have had on the ability to detect differences between these groups. Independence and unequal variance were assumed in all models. For examples of second level design matrix see appendix AA.

To control for multiple comparisons, Family Wise Error (FWE) rate correction was applied at the peak-voxel level with a threshold of 0.05 and activations were required to exceed a cluster extend threshold of 50µl in magnitude. The FWHM was sufficient for this approach, ranging from 10 x 10 x 10mm to 14 x 14 x 14mm, which was more than three times greater than the voxel size (3 x 3 x 3mm).

5.2.5.7 Inference; Small Volume Correction (SVC) analysis

Due to the conservative nature of the correction for multiple comparisons, a Small Volume Correction (SVC) approach, a method commonly utilised in the analysis of fMRI data (e.g. Dimoka, 2012), was applied to these models. *A priori* regions of interest (ROI) were identified from the literature (for the IGT; Li et al., 2010; Lin, Chiu, Cheng & Hsieh, 2008; for the MID; Knutson et al., 2000; Nestor et al., 2010; and for the Go/No Go task; Garavan et al., 2006; Kaufman et al., 2003), a full list of which can be found in table 5.7. Anatomically defined ROI masks were created with the Wake Forest University (WFU) PickAtlas toolbox (version 3.0.5; Maldjian, Laurienti, Kraft, & Burdette, 2003) in SPM12 and resampled to voxel dimensions 3 x 3 x 3mm, in line with the dimensions of the current data. With the exception of the DLPFC, regions were defined using the Automatic Anatomical Labelling atlas

(AAL; Tzourio-Mazoyer et al., 2002). In order to be consistent with previous research, the DLPFC was defined as Brodmann areas nine and 46 (Callicott et al., 1999; Dalwani et al., 2011; Dulebohn, Conlon, Sarinopoulos, Davison, & McNamara, 2009; Garavan et al., 2002).

Table 5.7: Regions selected for SVC analysis

Task	**Region**	**Abbreviation**
IGT	Anterior Cingulate Cortex	ACC
(n=8)	Amygdala	N/A
	Hippocampus	N/A
	Inferior Frontal Gyrus	IFG
	Insula	N/A
	Orbitofrontal Cortex	OFC
	Posterior Cingulate Cortex	PCC
	Caudate, Putamen, Pallidum, Nucleus Accumbens	Striatum
MID	Anterior Cingulate Cortex	ACC
(n=7)	Dorsolateral Prefrontal Cortex	DLPFC
	Insula	N/A
	Caudate, Putamen, Pallidum, Nucleus Accumbens	Striatum
	Supplementary Motor Area	SMA
	Thalamus	N/A
	Ventromedial Prefrontal Cortex	VMPFC
Go/	Anterior Cingulate Cortex	ACC
No Go	Dorsolateral Prefrontal Cortex	DLPFC
task	Inferior Frontal Gyrus	IFG
(n=8)	Inferior Parietal Lobe	Inf Parietal
	Insula	N/A
	Putamen	N/A
	Supplementary Motor Area	SMA
	Ventromedial Prefrontal Cortex	VMPFC

As before, FWE rate corrected *p*-values for peak-voxels and a minimum cluster extend threshold of 50µl were employed. Furthermore, the peak-voxel threshold was adjusted for the number of ROIs employed, using a Bonferroni method. This gave an alpha threshold of 0.007 for the MID task (p= 0.05/7 = 0.00714) and 0.006 for both the IGT and the Go/No Go tasks (p= 0.05/8 = 0.00625). ROI masks were applied through the results viewer in SPM12.

5.2.5.8 Mean activation extraction and analysis

Where significant interactions were found in either of the two factorial models, mean activations were extracted and exported to SPSS 21.0 (Statistical Package for the Social Sciences by IBM-Corp, Released 2012) for follow up analysis. Mean activations were extracted through the eigenvariate function in the results viewer of SPM12. For each interaction an eight-millimetre sphere was placed around the peak-voxel in the cluster. Results were summarised to the Matlab workspace and then copied into an SPSS file. To confirm the presence of the interaction the full factorial model was investigated and post hoc analysis with Bonferroni corrections were applied where appropriate.

This same approach was utilised to extract mean activations for certain effects of interest in order to investigate the potential relationship between the degree of activation in said conditions, and other measures from the study. The mean activations were summarised in SPSS and correlations were run between these and selected psychometric and cognitive measures. The alpha level of 0.05 was adjusted for the number of comparisons made, using a Bonferroni approach, to 0.003 (20 comparisons, p=0.05/20 = 0.0025).

5.3 Results

5.3.1 Iowa Gambling Task (IGT)

5.3.1.1 Task activation

On each trial of the IGT, participants selected from one of the four decks and won some money. On some trials, however, participants would receive additional feedback to inform them that they had also lost some money. The magnitude of gains, losses and loss frequency varied across the four decks. All results reported are FWE corrected.

5.3.1.1.1 Deck selections

When selecting from each of the four decks, extensive activation was observed which included the *a priori* ROIs mentioned in table 5.7 details of which are summarised in table 5.8. In addition, activation was observed in regions, identified using AAL; the temporal gyri, pre and postcentral gyri, supramarginal gyri, cerebellum, occipital and parietal regions. The whole-brain activations during deck selections are illustrated in figure 5.3, FWE corrected.

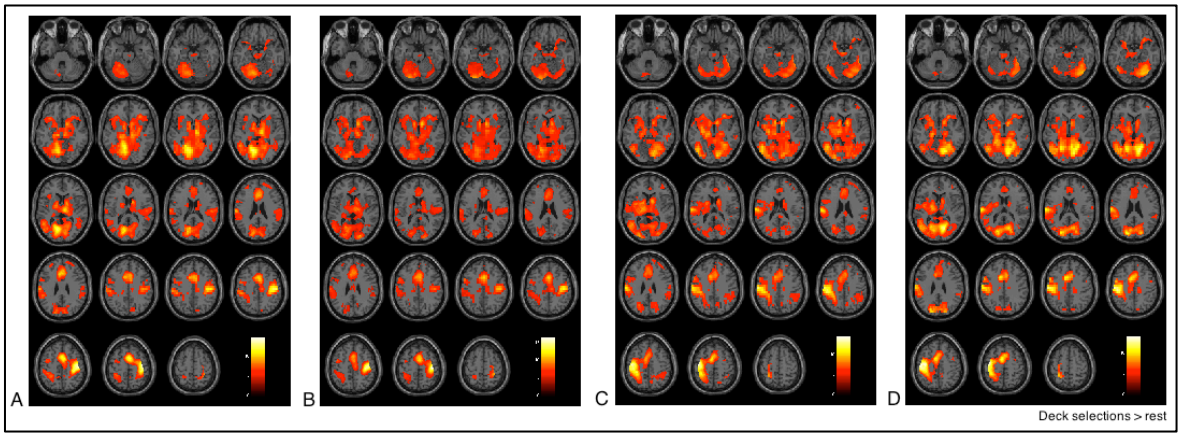

Figure 5.3: Activation associated with the selection of each of the four decks in the IGT, A) deck A selections, B) deck B selections, C) deck C selections, D) deck D selections

Table 5.8: Regions active when participants selected from the four decks of the IGT

ROI	Deck selections	Hemi.	Volume (µl)	F	Peak p (FWE)	Coordinates
Amygdala	A, B, C, D	Right	1,080	64.65	0.000	18, -1, -14
		Left	1,053	50.93	0.000	-18 -1, -14
ACC	A, B, C, D		13,176	108.48	0.000	3, 23, 28
				109.18	0.000	-6, 17, 31
Hippocampus	A, B, C, D	Right	3,753	85.67	0.000	18, -25, -11
		Left	4,428	88.24	0.000	-21, -31, -5
Insula	A, B, C, D	Right	7,965	69.9	0.000	33, 23, -5
		Left	8,991	71.28	0.000	-33, 17, -14
Striatum	A, B, C, D	Right	14,445	79.39	0.000	12, 5, 16[18]
				79.09	0.000	15, 5, -11[19]
		Left	8,613	71.43	0.000	-12, -1, -5[20]
				68.43	0.000	-12 8 -2[21]
SFG	A, B, C, D	Right	1,134	118.03	0.000	33, -10, 58
		Left	486	30.39	0.000	-24, -7, 55
OFC	A, B, C, D	Right	2,241	67.25	0.000	33, 26, -5
		Left	1,566	53.95	0.000	-33, 20, -14
MFG	A, B	Right	675	134.33	0.000	39, -7, 52
		Left	2,106	47.16	0.000	-30, 47, 25
MFG	C, D	Right	6,777	40.72	0.000	42, 44, 7
		Left	891	62.84	0.000	-30, -4, 52
PCC	A	Right	378	32.33	0.000	6, -43, 7
		Left	135	29.29	0.000	-9, -43, 7
PCC	B, C, D	Right	1,728	33.86	0.000	9, -40, 16
		Left	324	21.89	0.000	-6, -40, 7
IFG	A, B, C	Right	1,080	41.21	0.000	-36, 23, -2
		Left	621	39.41	0.000	36, 29, 1

[18] Peak located in right caudate

[19] Peak located in right NAcc

[20] Peak located in left pallidum

[21] Peak located in left putamen

5.3.1.1.2 Receipt of monetary win feedback

In this task, participants received a monetary gain as a result of their selection. Regions common to wins from all four decks were the inferior temporal gyri, precentral gyri, right SMA, MFG, cerebellum, occipital regions and the bilateral fusiform gyri. There was also some variation in response to the different reward magnitudes associated with the decks.

In response to monetary gains from decks A, B and C, activation was observed in the following regions; right (2,565µl; 6, 38, 28) and left ACC (1,728µl; -9, 47, -5), right (2,538µl; 21, -31, -5) and left hippocampus (1,836µl; -18, -31, -5), right (1782µl; 33, 23, -8) and left OFC (729µl; -30, 23, -11), right (4,023µl; 36, 20, -5) and left insula (3,186µl; -30, 20, -5) and the right PCC (2295µl; 0, -34, 28). When participants received feedback from deck D, however, there was no significant activation in the ACC, insula or OFC, and activation in the hippocampus and PCC was localised in the left hemisphere (hippocampus; 243µl, -12, -37, 7, PCC; 2,619µl, -9, -52, 28).

A similar pattern of activation in the right SFG was observed in decks A, B and D for wins (351µl; 27, 59, 1), however this was localised in the left hemisphere for wins from deck C (135µl, -12, 56, 13). Furthermore, a summary of the activation associated with monetary gains in the striatum and amygdala for each deck separately can be found in table 5.9. Patterns of activation for win feedback from the four decks are represented in figure 5.4, FWE corrected.

Table 5.9: Details of striatal and amygdala activation associated with monetary gain from each of the four decks of the IGT

Deck	ROI	Hemi.	Volume (µl)	F	Peak p (FWE)	Coordinates	Peak located
A	Amygdala	Right	216	17.62	0.006	30, 5, -20	
	Striatum	Right	702	48.89	0.000	27, 17, -8	Putamen
			3,726	48.37	0.000	9, 5, 7	Caudate
		Left	1,701	31.2	0.000	-12, 8, 7	Caudate
				27.51	0.002	-9, 2, -2	Pallidum
B	Amygdala	Right	540	24.47	0.000	18, -1, -17	
	Striatum	Right	6,426	75.86	0.000	12, 20, 4	Caudate
				56.1	0.000	27, 20, -2	Putamen
		Left	4,914	43.73	0.000	-9, 14, 4	Caudate
C	Amygdala				N.S.		
	Striatum	Right	270	45.8	0.000	27, 20, -2	Putamen
			594	30.71	0.001	9, 8, 4	Caudate
		Left	189	24.2	0.005	-18, -19, 22	Caudate
D	Amygdala				N.S.		
	Striatum	Right	3,078	43.42	0.000	27, 2, 1	Putamen
		Left	1,836	32.87	0.000	-24, 5, 1	Putamen

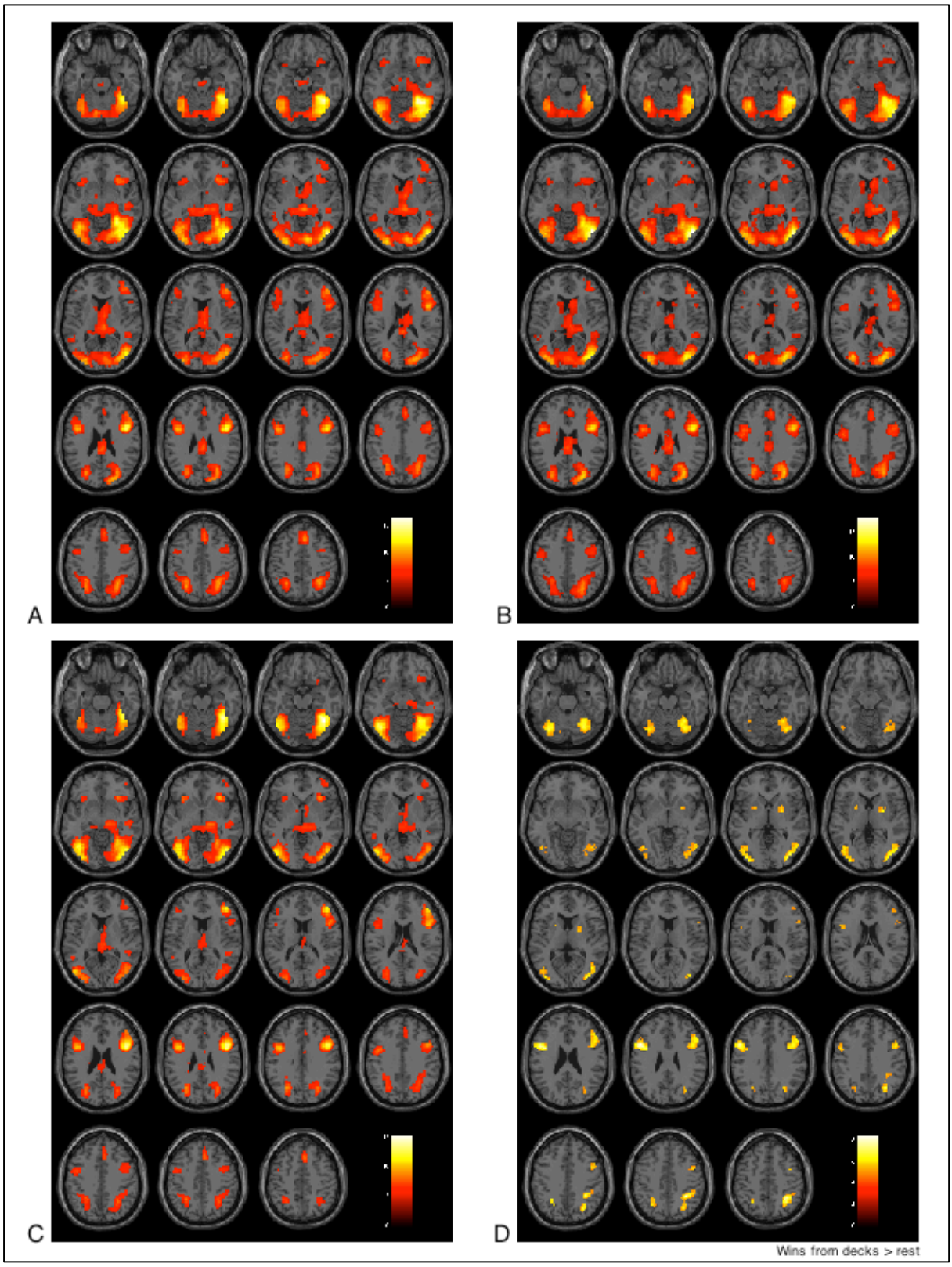

Figure 5.4: Activation associated with gains from the four decks in the IGT, A) deck A = large monetary gains, B) deck B = large monetary gains, C) deck C = small monetary gains, D) deck D = small monetary gains

5.3.1.1.3 Receipt of monetary loss feedback

On many trials participants would both win and lose money. Similar to win feedback, activity in certain regions was common to losses across all decks and some were not. During all loss conditions activation was observed in parts of the occipital cortex, cerebellum, inferior and superior temporal gyri, SMA, thalamus, right precentral gyrus and right fusiform gyrus.

Bilateral PCC activation was observed for losses associated with selections from decks A, B and C (right: 162μl, 12, -37, 13, left: 2,133μl, -6, -49, 31), whereas only left PCC activation was present for deck D losses (1,404μl, -3, -49, 28). When participants lost money from decks A, C and D they demonstrated significant responses in the left (351μl, -42, 11, 34) and right MFG (1,593μl, 42, 8, 37) and the left (1,917μl, -24, 32, 46) and right SFG (540μl, 27, 2, 55). For deck B, however, activity in the MFG was localised to the right hemisphere (2,214μl, 42, 8, 37) and SFG to the left hemisphere (162μl, -18, 26, 55).

The ACC was also active in response to loss feedback, with bilateral activation present in response to losses from decks B and C (9,963μl, left peak: -9, 41, 16, right peak: 6, 41, 13) and localised in the left hemisphere for losses from decks A (1,269μl, -3, 53, 1) and D (351μl, -3, 47, -5). Insular activation was present for losses from all decks, with bilateral activation observed for decks C and D (right: 3,186μl, 33, 20, -5, left: 2,538μl, -30, 20, -2), a greater magnitude of response for deck B (right: 5,076μl, 30, 17, -17, left: 4,860μl, -33, 17, -17) and activation in the right insula only for deck A (3,402μl, 33, 29, -2). Activation in the hippocampus, OFC, amygdala and striatum were also observed in varying magnitudes in

response to monetary loss. These are summarised in tables 5.10 and 5.11 and illustrated in figure 5.5, FWE corrected.

Table 5.10: Location and extent of activation in the amygdala, hippocampus and OFC in response to loss feedback from the four decks of the IGT

Deck	**ROI**	**Hemi.**	**Volume (µl)**	**F**	**Peak p (FWE)**	**Coordinates**
A	Amygdala				N.S.	
	Hippocampus	Right	675	62.13	0.000	15, -37, 10
		Left	729	54.5	0.000	-15, -37, 10
	OFC	Right	297	29.24	0.000	33, 26, -5
B	Amygdala	Right	1134	40.51	0.000	21, -4, -17
	Hippocampus	Right	2565	60.92	0.000	27, -37, 7
		Left	1107	55.75	0.000	-33, -37, -2
	OFC	Right	3483	86.48	0.000	30, 23, -11
		Left	1539	60.75	0.000	-39, 20, -5
C	Amygdala	Right	459	25.79	0.000	18, -1, -17
	Hippocampus	Right	243	37.24	0.000	30, -40, 4
		Left	459	23.75	0.002	-24, -40, 4
	OFC	Right	1809	54.12	0.000	45, 50, -5
		Left	297	30.22	0.003	-36, 23, -5
D	Amygdala	Left	351	21.5	0.000	-3, 47, -5
	Hippocampus	Left	351	25.34	0.001	-36, -34, -8
		Right	81	20.82	0.006	30, -40, 4
	OFC	Right	810	31.15	0.000	33, 23, -8

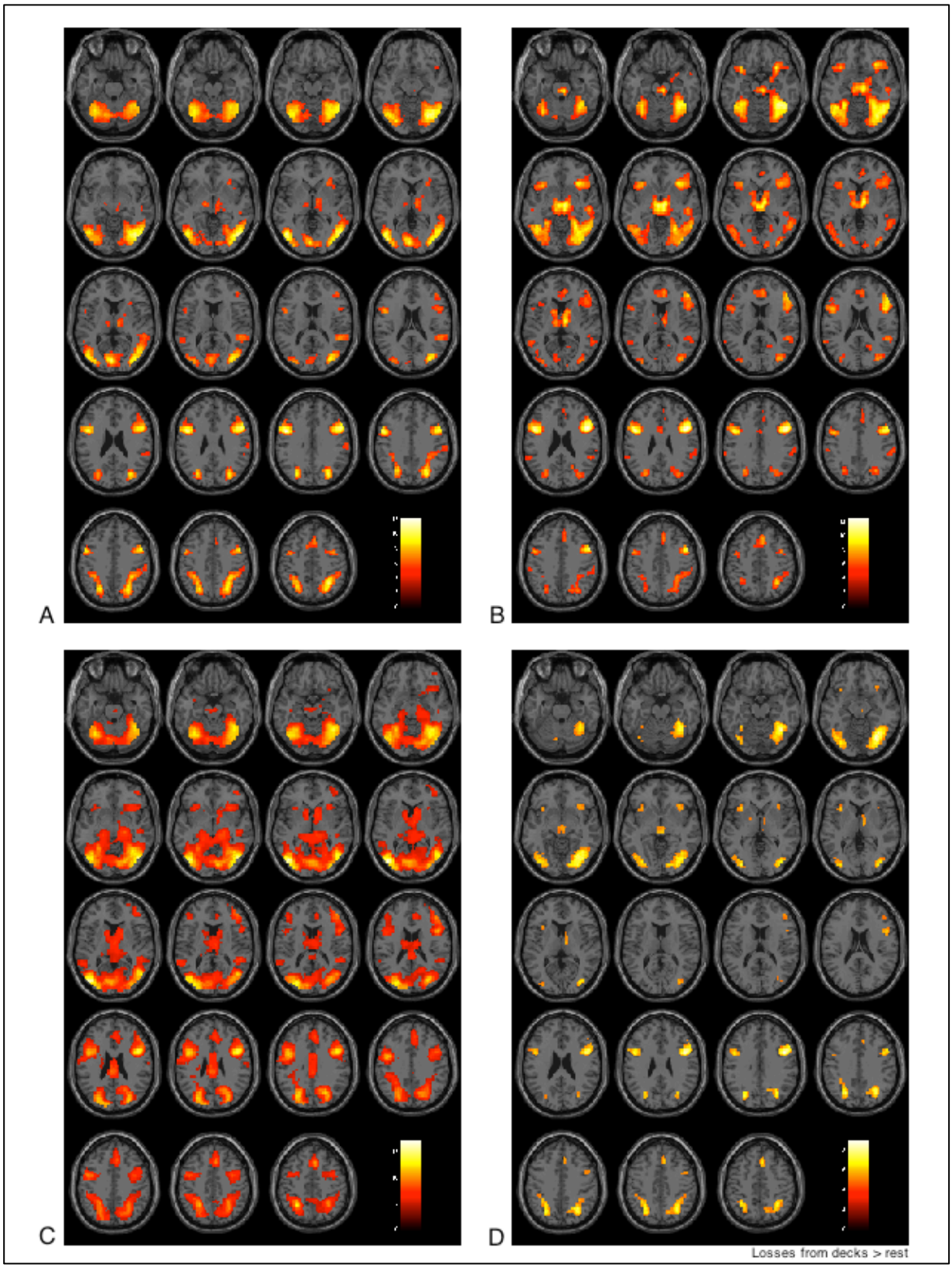

Figure 5.5: Activation associated with losses from the four decks in the IGT, A) deck A = large monetary losses, B) deck B = very large monetary losses, C) deck C = small monetary losses, D) deck D = moderate monetary losses

Table 5.11: Location and extent of activation in the striatum in response to loss feedback from the four decks of the IGT

Deck	Hemi.	Volume (µl)	F	Peak p (FWE)	Coordinates	Peak located
A	Right	378	44.77	0.000	18, -1, 25	Caudate
		243	37.08	0.000	27, 20, 4	Putamen
		486	26.84	0.002	18, -1, 4	Pallidum
	Left	837	49.56	0.000	-18, -10, 25	Caudate
B	Right	2916	77.5	0.000	18, 2, 25	Caudate
	Left	3564	68.16	0.000	-15, -1, 25	Caudate
C	Right	6669	68.43	0.000	9, 11, 4	Caudate
			65.49	0.000	27, 20, -2	Putamen
			64.17	0.000	15, 2, 1	Pallidum
	Left	5481	41.42	0.000	-9, 8, 4	Caudate
			39.13	0.000	-12, -1, 1	Pallidum
			38.98	0.000	-15, -13, 22	Caudate
D	Right	486	30.34	0.001	18, 8, 25	Caudate
	Left	621	31.8	0.000	-18, 2, 1	Pallidum
		432	22.7	0.006	-21, 2, 22	Caudate

Present for all win and loss conditions was bilateral IFG activation (right IFG: 13,797µl, left IFG: 10,584µl, with peak located at coordinates 42, 8, 25 and -42, 5, 28, respectively).

5.3.1.1.4 Win over loss feedback: 'Win > Loss'

When all win feedback conditions were combined and compared to all loss conditions extensive striatal activation (caudate, pallidum, NAcc and putamen) was observed, illustrated in figure 5.6, FWE corrected.

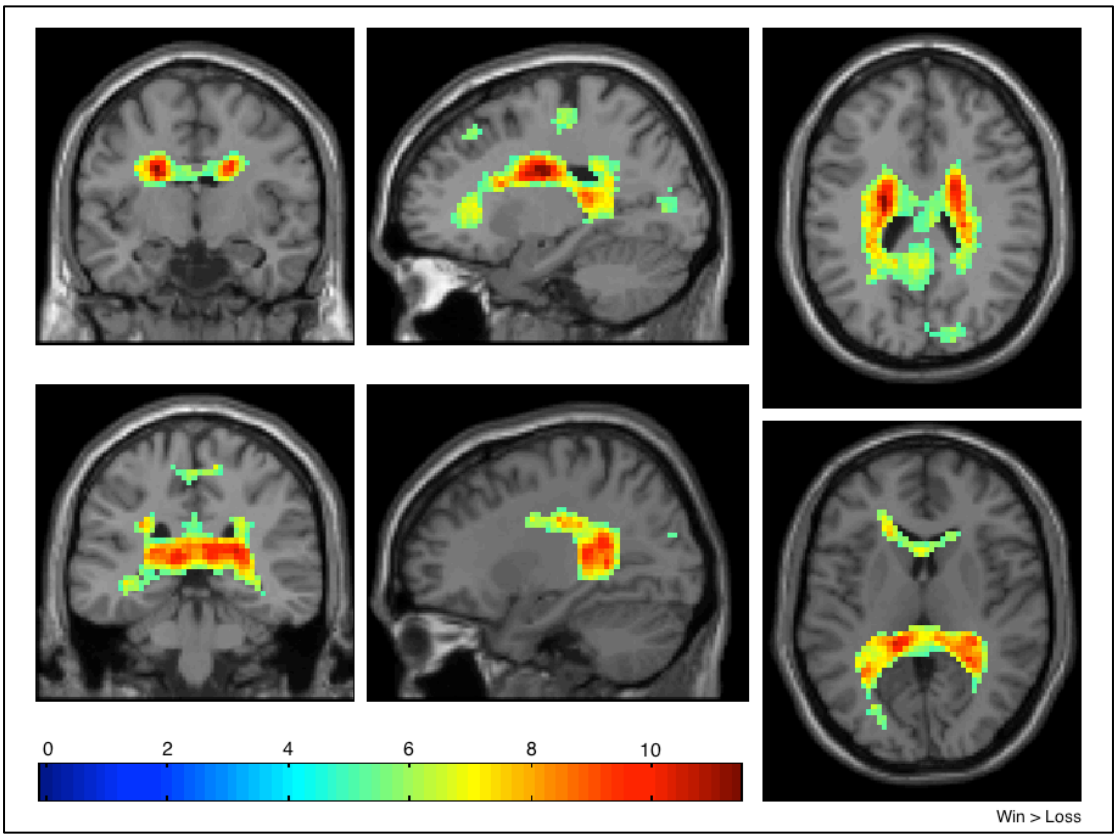

Figure 5.6: Regions demonstrating significantly greater activation upon receipt of monetary gain feedback in the IGT, compared to feedback of monetary loss.

5.3.1.2 Between group comparisons

5.3.1.2.1 Male drivers

5.3.1.2.1.1 Primary and secondary contrasts of interest

The effects of 'risk status' (risky vs. safe) and age (18-24 vs. 29+) in males on the BOLD response during the IGT were investigated utilising 2 x 2 Factorial ANVOAs at the group level for the primary and secondary first level contrasts outlined in section 5.2.5.5. No significant main effects or interactions were observed at either the whole-brain level or with the SVC approach.

5.3.1.2.1.2 Exploratory analysis

Exploratory analysis identified a significant main effect of risk status at the whole-brain level for feedback (a combination of win and loss) from deck C, such that risky male drivers had a significantly greater response than safe in the right pallidum (81µl, peak located at: 18, 2, 7 and identified through the AAL), $F(1,75)=27.99$, $p<.05$ (FWE corrected). No other effects were observed at the whole-brain level.

Several additional effects were detected with an SVC analysis using the regions outlined in section 5.2.5.7. The effects that survived additional corrections for multiple comparisons are summarised in table 5.12 below. The effects of risk status in the right putamen during 'C feedback > rest' and age differences in hippocampal activation during 'A feedback > rest' are represented in figures 5.7 and 5.8, respectively, FWE corrected.

Table 5.12: Summary of the main effects of risk status and age observed with SVC analysis

2nd level contrast	**1st level contrast**	**Volume (µl)**	**F[22]**	**Coordinates**	**Peak located**
Safe > Risky	Win A+B > rest	162	22.11	-24, 17, -14	Left OFC
	B feedback > rest	54	23.00	12, 11, 16	Right Caudate
	C feedback > rest	783	27.99	18, 2, 7	Right Putamen
Older > Younger	A feedback > rest	459	22.11	30, -28, -8	Right Hippocampus
	C feedback > rest	243	23.61	27, -25, -8	Right Hippocampus

[22] All with degrees of freedom 1,75 and significant at p<.006, FWE corrected

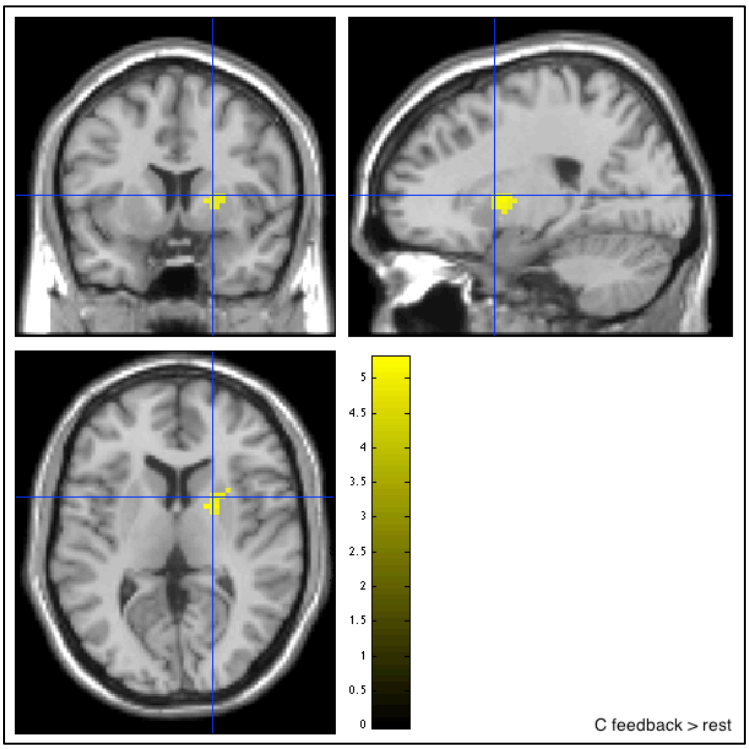

Figure 5.7: Area of the right putamen in which safe males demonstrated greater activation than risky during the 'C feedback > rest' condition of the IGT

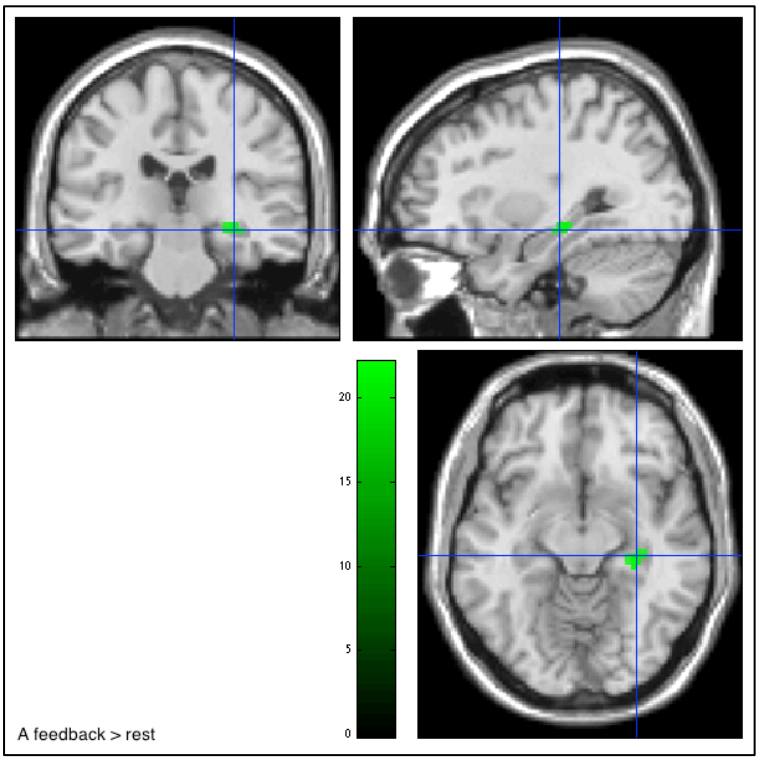

Figure 5.8: Area of the right hippocampus in which older males demonstrated greater activation than younger males during the 'A feedback > rest' condition of the IGT

Using the SVC approach, two significant interactions between risk status and age were detected. In the ACC a significant interaction, 135µl (0, 23, 16), was observed when participants selected from decks that delivered infrequent punishments (decks B and D), $F(1,75)=19.01$, $p<.006$ (FWE corrected). Effect sizes were extracted from the data and follow up analysis was conducted in SPSS. A significant, disordinal, interaction between age and risk status was found, $F(1,75)=20.45$, $p<.001$, $\eta^2=.214$, such that younger risky males demonstrated significantly more activation in the ACC for this condition (M=1.56, SD=1.6) than safe younger males (M=0.24, SD=1.21). Conversely, older safe males showed greater activation (M=1.41, SD=1.45) than older risky males (M=-0.05, SD=1.16). This interaction is illustrated in figure 5.9 below.

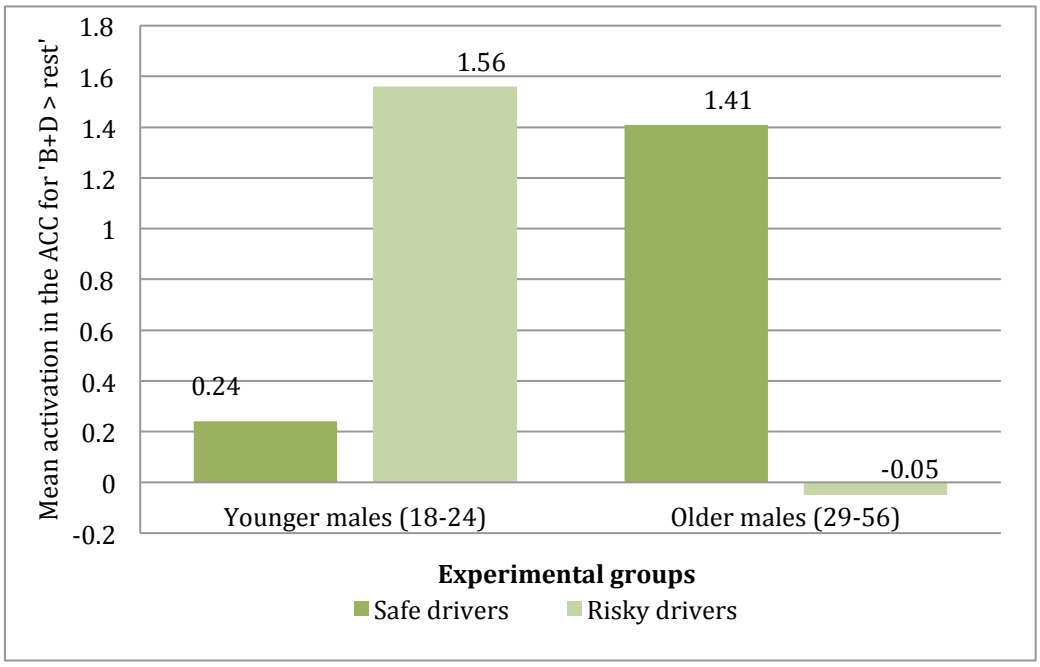

Figure 5.9: Graph illustrating the dis-ordinal interaction between age and risk status in the ACC in males when selecting from decks B and D in the IGT

A significant interaction was also found in the PCC, 81µl (9, -46, 28), when participants selected from advantageous decks (decks C and D), $F(1,75)=19.49$,

p<.006 (FWE corrected). Follow up analysis in SPSS confirmed the presence of an interaction for this condition, $F(1,75)=15.858$, $p<.001$, $\eta^2=.175$. Bonferroni post hoc revealed that older safe males had significantly greater activation in this condition (M=0.75, SD=0.9) than younger safe males (M=0.04, SD=0.78). No difference was found between younger and older risky males (younger: M=0.64, SD=0.45, older: M=0.04, SD=0.68). These differences are illustrated in figure 5.10 below.

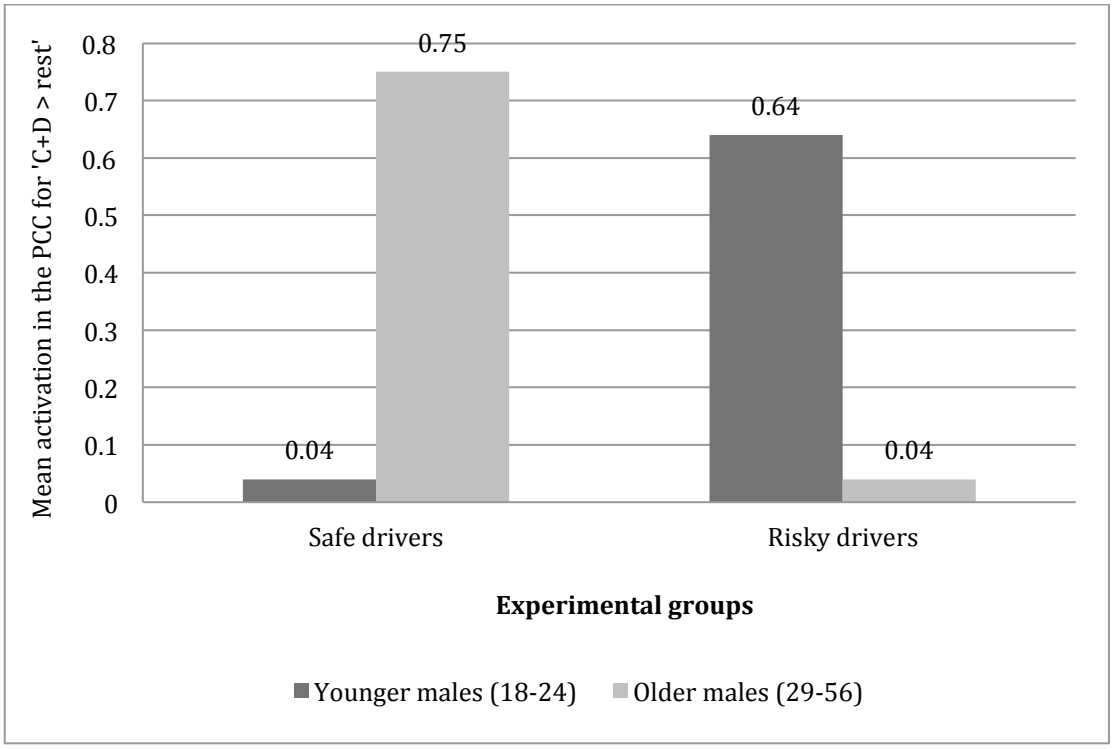

Figure 5.10: Graph illustrating the interaction between age and risk status in the PCC in males when selecting from advantageous decks (C and D) in the IGT

5.3.1.2.2 Young drivers

5.3.1.2.2.1 Primary and secondary contrasts of interest

The effects of risk status (risky vs. safe) and gender in younger drivers on the BOLD response were investigated utilising 2 x 2 Factorial ANVOAs at the group

level for the first level contrasts outline in section 5.2.5.5. At the whole-brain level, an effect of gender was observed in the right postcentral gyrus for disadvantageous over advantageous deck selections during the final two blocks of the task (bad > good deck selections under risk); $t(65)=6.13$, $p<.006$, FWE corrected (108µl, 30, -22, 43), where young females had a significantly greater response than young males. No other primary or secondary contrasts demonstrated significant main effects at whole-brain level or interactions between risk status and gender, and no further differences were observed with an SVC approach.

5.3.1.2.2.2 Exploratory analysis

Within the exploratory contrasts, no effects were found on a whole-brain level and no main effects were detected through the SVC analyses, once adjustments were made for multiple comparisons. However, SVC analyses revealed significant interactions between gender and risk status in the insula and amygdala for certain conditions.

In the amygdala (54µl, -30, -4, -20) a significant interaction was found, $F(1,75)=14.36$, $p<.006$ (FWE corrected), when younger individuals received monetary gain feedback from disadvantageous decks (decks A and B). Effect sizes were extracted to determine the source of the interaction in SPSS. A 2 x 2 Factorial ANOVA (gender x risk status) in SPSS confirmed the presence of an interaction in these data: $F(1,75)=14.438$, $p<.001$, $\eta^2=.161$, seen in figure 5.11.

Post hoc identified that young risky males had significantly greater activation for this condition (M=0.7, SD=0.62) than young safe males (M=-0.19, SD=1.03) while

there was no difference between young risky and safe females (risky: M=0.12, SD=0.82, safe: M=0.62, SD=0.72).

Figure 5.11: Graph illustrating the interaction between gender and risk status in the amygdala in younger drivers when they won from decks A and B in the IGT

An interaction was also present in the insula, 54µl (39, -1, 7), when younger individuals received monetary gain feedback from infrequent punishment decks (decks B and D), $F(1,75)=18.37$, $p<.006$ (FWE corrected). Follow up analysis revealed the presence of a disordinal interaction, $F(1,75)=20.595$, $p<.001$, $\eta^2=.215$. Figure 5.12 illustrates this interaction, where risky younger males showed a greater effect (M=0.84, SD=0.76) than safe (M=-0.1, SD=1) but the pattern was reversed in females, as safe females (M=0.45, SD=0.92) were greater than risky (M=-0.35, SD=0.68).

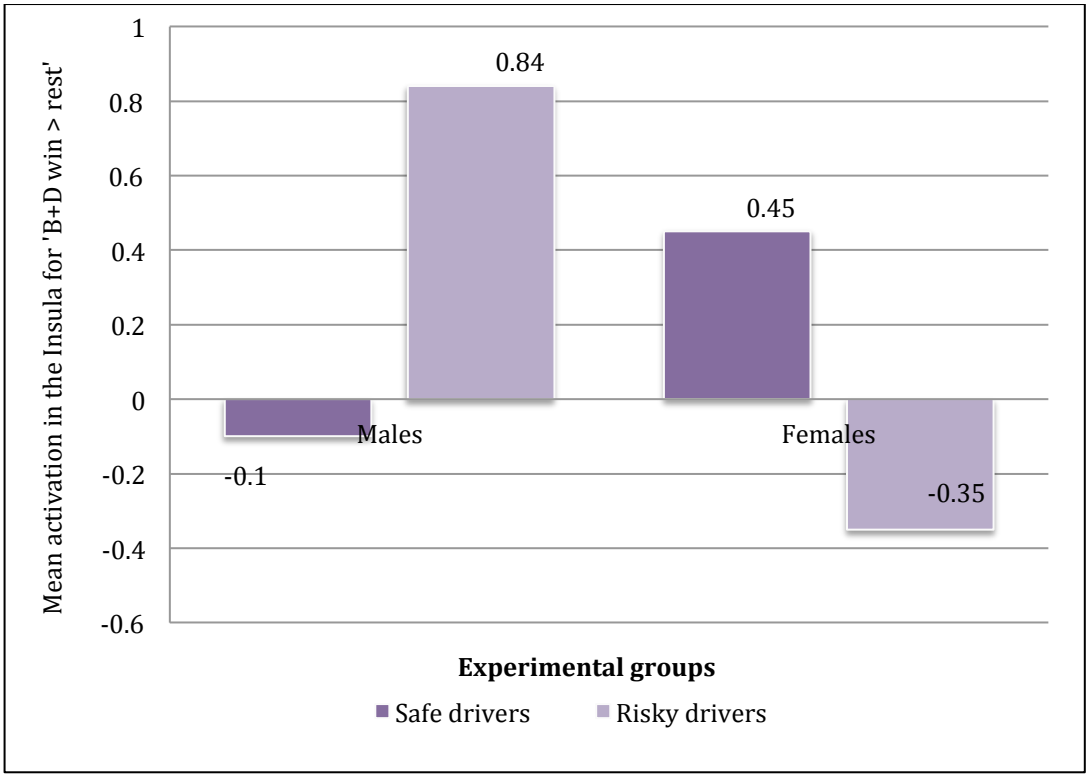

Figure 5.12: Graph illustrating the dis-ordinal interaction between gender and risk status in the insula in younger drivers when they won from decks B and D in the IGT

5.3.1.2.3 Young male drivers

5.3.1.2.3.1 Primary and secondary contrasts of interest

One-way ANOVAs of risk status in younger males on the primary and secondary first level contrasts mentioned in section 5.2.5.5 revealed no significant differences on a whole-brain level or within the regions of interest identified for the SVC analysis.

5.3.1.2.3.2 Exploratory analysis

No effects of risk were observed at the whole-brain level for exploratory contrasts, however, SVC analysis identified three instances where risky younger males demonstrated significantly greater activation when compared to safe. In the right

amygdala (81µl, 27, -1, -20) this difference was found when young males received feedback from deck A (t(37)=3.78, p<.006, FWE corrected). A significant effect was observed in the left IFG, deep within the frontal operculum (108µl, -39, 20, 19) when young males lost money from decks A and C (t(37)=4.91, p<.006, FWE corrected) Finally, risky young males displayed a greater response in the left insula (81µl, -45, 2, 1) when they received feedback from deck D (t(37)=4.48, p<.006, FWE corrected).

5.3.2 Monetary Incentive Delay Task (MID)

5.3.2.1 Task activation

In the MID, each trial consisted of cue, target and feedback periods. Win cues preceded a potential monetary gain (50c for hitting the target) and neutral cues were followed by neutral reinforcement (a happy face). On both trial types participants saw a sad face if they missed the target.

5.3.2.1.1 Cue periods

Similar patterns of activation were observed for neutral and win cue stimuli. During these cue periods significant activation was observed in occipital regions, the cerebellum, ACC, postcentral gyrus, DLPFC, thalamus, hippocampus, parahippocampus, SMA, putamen, inferior and superior parietal gyri, VMPFC, insula and aspects of the ventral and dorsal striatum (including the caudate, putamen, pallidum and NAcc), identified through AAL at a whole-brain level. These regions are illustrated in figure 5.13. *A priori* ROIs are summarised in more detail in table 5.13.

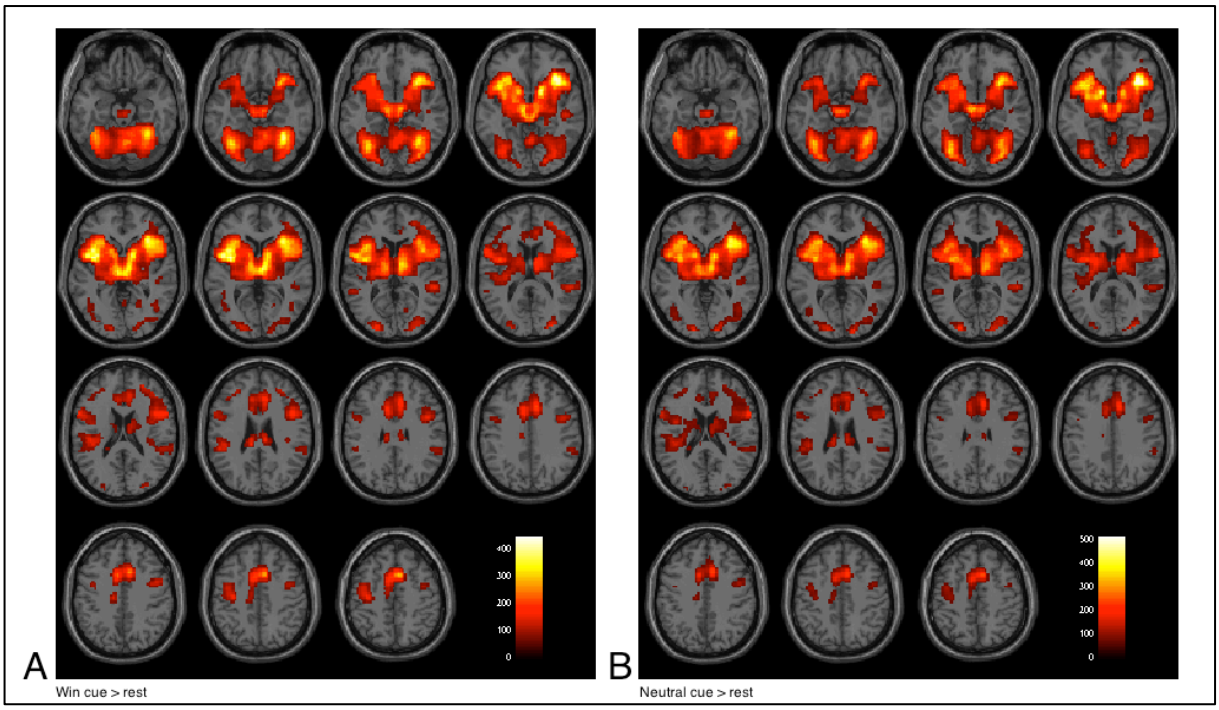

Figure 5.13: Axial slices showing activity in response to cue stimuli in the MID, A) activation in anticipation of a win target, B) activation in anticipation of a neutral target, FWE corrected.

Table 5.13: Regions active in anticipation of neutral and win target stimuli

ROI	Hemi.	Volume (µl)	F	Peak p (FWE)	Coordinates	Peak located
ACC	Right	15471	185.78	0.000	9, 29, 28	
	Left		166.84	0.000	-9, 17, 31	
DLPFC	Right	7938	135.97	0.000	42, 41, 10	
	Left		85.55	0.000	-42, 32, 19	
Insula	Right	13500	431.58	0.000	36, 23, -5	
	Left	13932	448.88	0.000	-36, 2, 4	
SMA	Right	15849	302.45	0.000	6, 11, 52	
	Left		244.68	0.000	-6, 2, 58	
Striatum	Right	18252	415.83	0.000	15, 5, -2	Pallidum
			302.11	0.000	30, 17, -2	Putamen
	Left	15228	313.89	0.000	-12, 8, -2	Pallidum
			290.14	0.000	-30, 5, 4	Putamen
Thalamu	Right	17280	400.3	0.000	6, -13, 4	
	Left		373.77	0.000	-6, -19, -2	
VMPFC	Right	7101	182.38	0.000	9, 20, 43	
	Left		194.83	0.000	-6, 14, 43	

5.3.2.1.2 Positive and negative feedback: hitting and missing the target stimulus

In response to hit feedback (a combination of both win and neutral hit feedback) activation in the angular gyrus, inferior and superior parietal gyri, pre and postcentral gyri, cerebellum and vermis was observed (identified using the AAL), in addition to the ROIs reported in table 5.7, the results of which are detailed in table 5.14.

These regions were also found to be elicited in response to miss feedback, although to a smaller extent than was observed during hit feedback, as illustrated in figure 5.14. Most notably, distinct trends in activation in the striatum for hit and miss feedback were observed. These differences are outlined in table 5.15.

Table 5.14: Regions active in response to hit feedback in the MID

ROI	**Hemi.**	**Volume (µl)**	**F**	**Peak p (FWE)**	**Coordinates**
ACC	Right	16821	156	0.000	3, 2, 28
	Left		167.94	0.000	-6, 2, 31
DLPFC	Right	10449	153.84	0.000	42, 2, 31
	Left		145.65	0.000	-42, 2, 31
Insula	Right	6777	92.44	0.000	39, 5, 16
	Left	7209	159.39	0.000	-36, -10, 10
SMA	Right	21465	294.88	0.000	6, 23, 52
	Left		275.05	0.000	0, -4, 55
Thalamus	Right	12501	112.88	0.000	9, -31, 4
	Left		108.6	0.000	-15, -31, 2
VMPFC	Right	12420	294.41	0.000	9, 26, 52

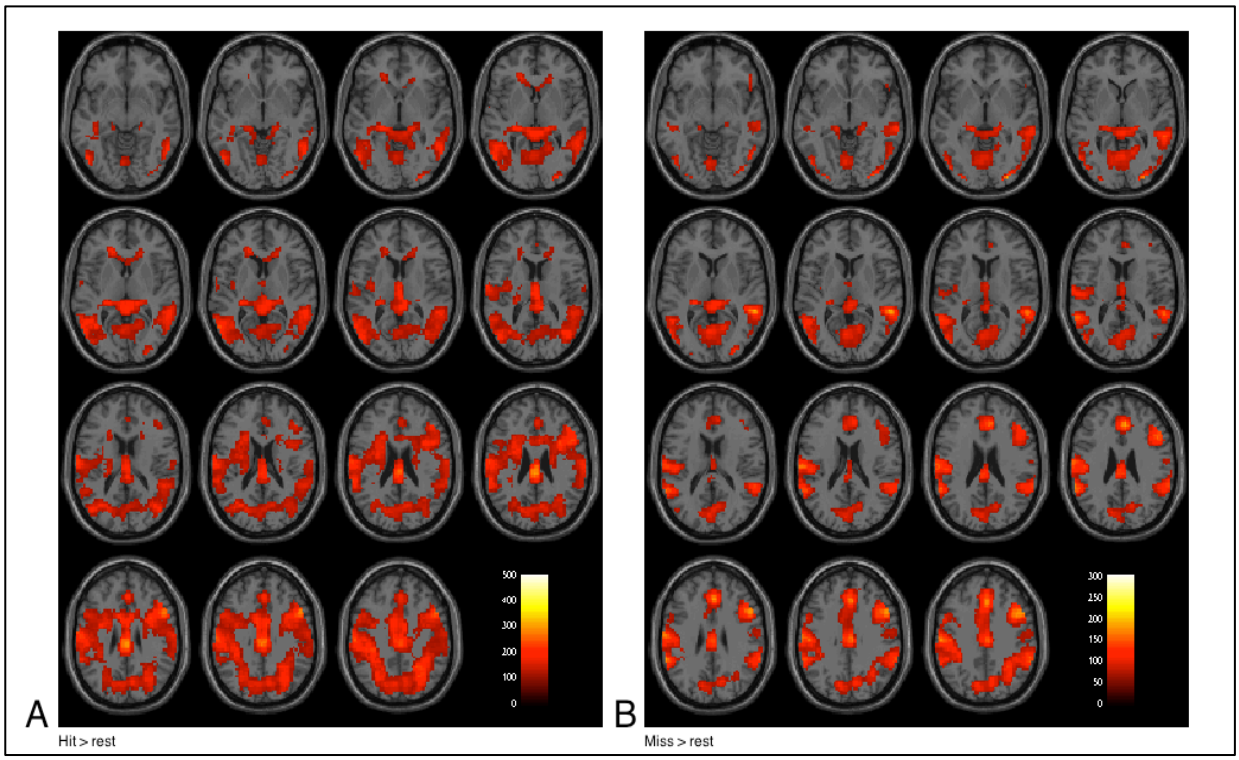

Figure 5.14: Axial slices showing the BOLD response to the feedback that followed target stimuli in the MID, A) hit feedback, B) miss feedback, FWE corrected.

Table 5.15: Striatal responses to hit and miss feedback in the MID

Contrast		Hemi.	Volume (µl)	F	Peak p (FWE)	Coordinates	Region
Hit	> Baseline	Right	9126	169.48	0.000	-21, -1, 22	Caudate
		Left	9909	147.83	0.000	18, 26, 4	Caudate
Miss	> Baseline	Right	1026	34.4	0.000	15, -7, 16	Caudate
		Left	1620	45.92	0.000	-18, 11, 22	Caudate
	< Baseline	Right	1242	54.74	0.000	15, 5, -5	Putamen
			270	70.37	0.000	12, 5, -18	NAcc
		Left	1269	83.13	0.000	-15, 5, -8	Putamen
			108	47.85	0.000	-15, 5, -11	NAcc

5.3.2.1.3 Anticipation vs. receipt: comparing cue and feedback conditions

Certain contrasts were also generated to identify processes unique to cue conditions when compared to feedback, for example 'win cue > win hit' and 'win cue > win miss'.

In the 'win cue > win hit' condition it was noted that there was greater activation in the insula (right 6,750µl, 36, 26, 4, left: 5,913µl, -30, 26, 1), thalamus (right 1,080µl, 6, -16, 2, left: 8,775µl, -3, -16, -2), right ACC (13,311µl, 3, 2, 28) and DLPFC (9,315µl, parts of Brodmann's area 46 only, right peak: 51, 14, 31, left peak: -51, 5, 40) for win cues and significant deactivation in the parietal cortex and regions such as the right SMA (1,971µl, 6, 23, 52), right VMPFC (9,585µl, 6, 29, 49), and the bilateral caudate (right 4,779µl, 21, -13, 25, left: 4779µl, -18, 14, 15) when compared to win hit feedback.

When win cue trials were compared to win miss feedback, largely similar regions to the above were found to be significantly active and deactive, with the addition of significant activation in the bilateral pallidum (right 4,077µl, 15, 5, -5, left: 3,807µl, -15, 5, -5) and deactivation in the left insula (2,322µl, -30, 23, 4) and bilateral thalamus (3,537µl, right peak: 6, -31, 4, left peak: -6, -31, 4).

The effects observed during 'win cue > win hit' and 'win cue > win miss' are illustrated in figure 5.15. Where regions coloured in red represent significant activation and blue represents deactivation.

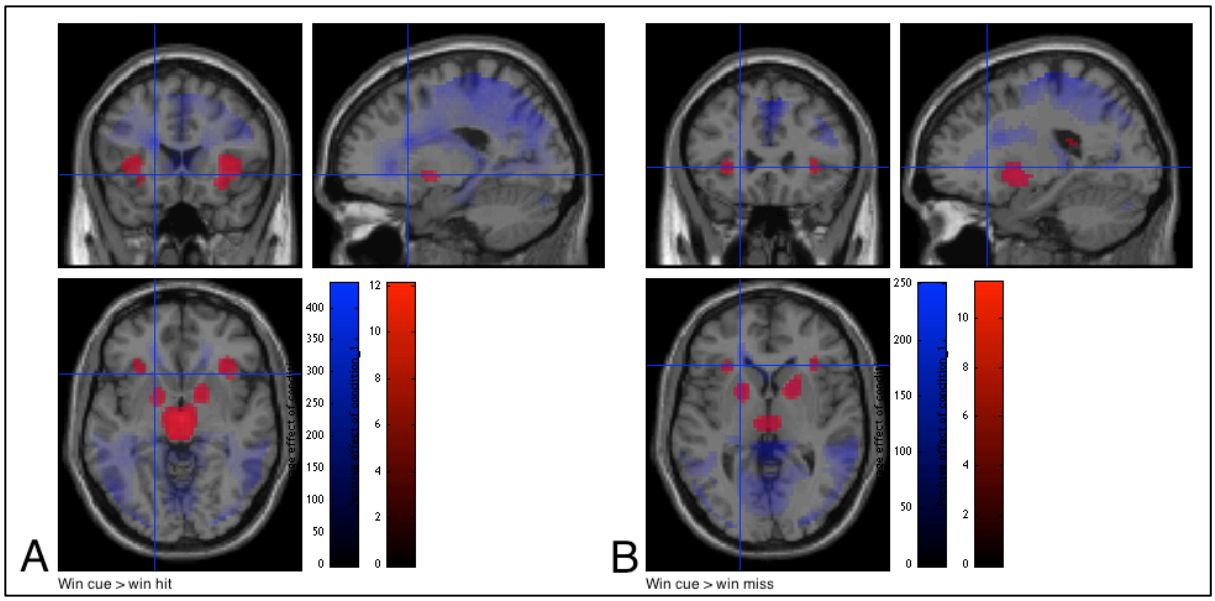

Figure 5.15: A) Win cue > win hit, B) Win cue > win miss, FWE corrected.

5.3.2.1.4 Hit vs. miss feedback

The differences between hit and miss conditions were assessed with a 'hit > miss' contrast. In this condition a positive response was observed in the ACC (right: 351µl, 3, 2, 28, left: 1,485µl, -9, 47, -5), right insula (2,133µl, 45, 23, -5) left thalamus (513µl, -3, -25, 10), VMPFC (567µl, right peak: 3, 56, 1, left peak: -9, 53, 1) and parts of the left and right striatum (right putamen: 1,377µl, 30, -7, 4, right caudate: 6,966µl, 18, -10, 25, left caudate: 9,963µl, -18, 11, -8). A significant, negative, response was found in the right IFG (1,053µl, 48, 20, -2) and left insula (1,296µl, -39, 17, 1). These effects can be seen in figure 5.16 below.

Similar results were observed when hit and miss conditions were compared in win trials only ('win hit > win miss'), see illustration in figure 5.17.

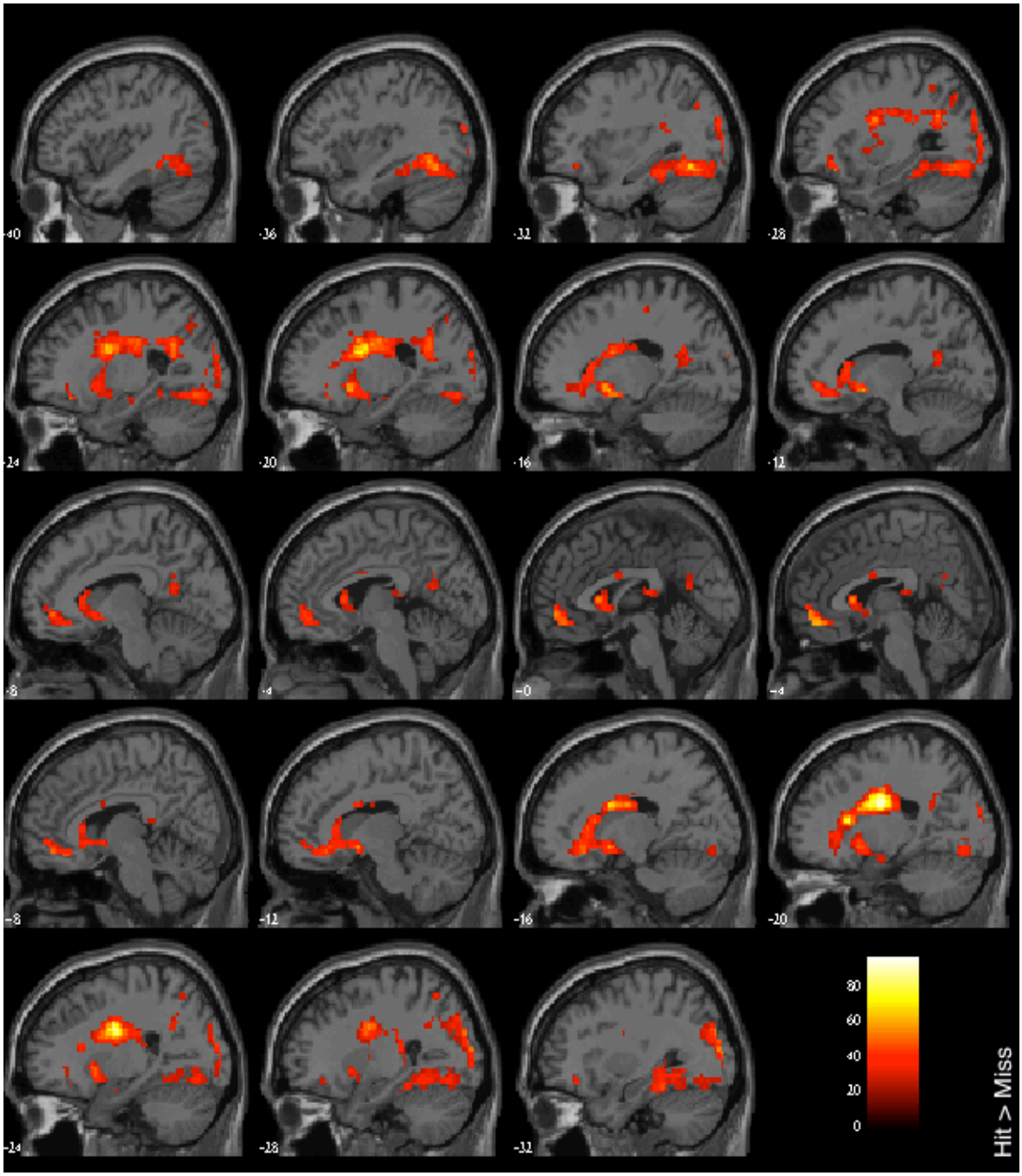

Figure 5.16: Sagittal view of participants' activation upon receipt of hit feedback, compared to miss feedback in the MID, FWE corrected.

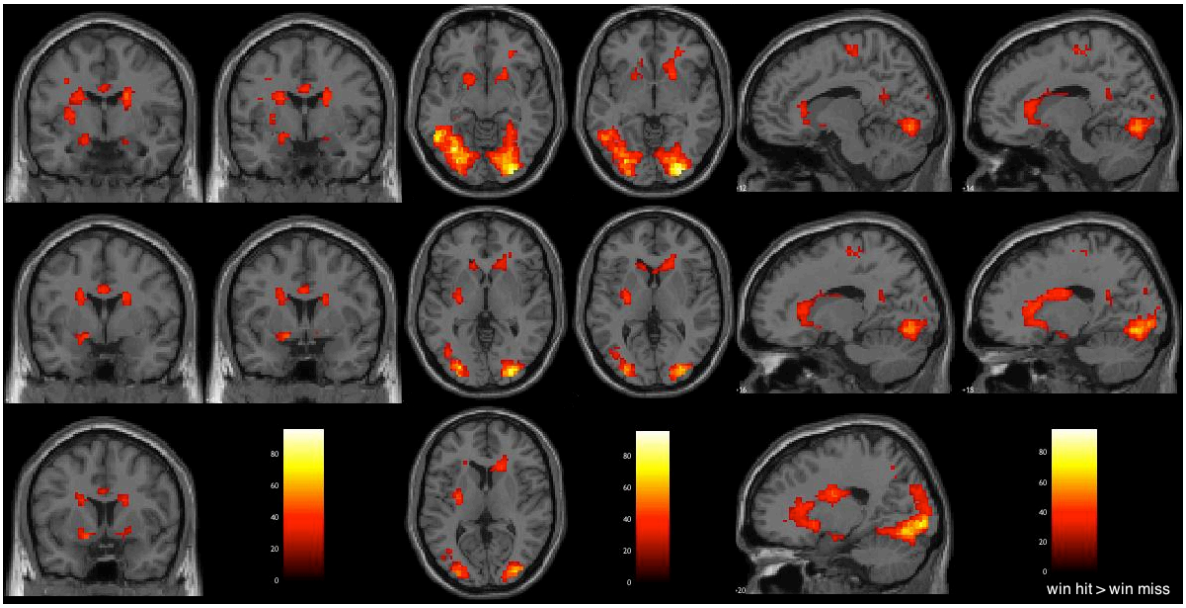

Figure 5.17: Coronal, axial and sagittal view of participants' activation upon receipt of hit feedback to win stimuli, compared to miss, in the MID, FWE corrected.

5.3.2.2 Between group comparisons

5.3.2.2.1 Male drivers

5.3.2.2.1.1 Primary and secondary contrasts of interest

Similar to the approach utilised for between groups analysis of the IGT data in males, 2 x 2 Factorial ANOVAs of age (18-24 vs. 29+) and risk status were applied at the group level to examine the MID contrasts generated at the first level (for list of these see section 5.2.5.5). No significant main effects or interactions were observed for the primary contrasts.

Whole-brain analysis of secondary contrasts identified a significant main effect of age in the left DLPFC (54µl, -21, -1, 40) for the 'win cue > win hit' condition of the MID. In this condition, younger males demonstrated greater activation in the DLPFC than older males, $t(76)=5$, $p<.05$ (FWE corrected). No other main effects or

interactions were observed at a whole-brain level, however, SVC analysis revealed two main effects of risk status in the right ACC, for 'win cue > win miss' ($F(1,76)=25.28$, $p<.006$, FWE corrected, 486µl, peak located at: 3, 35, -5) and 'neutral cue > neutral miss' ($F(1,76)=23.44$, $p<.006$, FWE corrected, 594µl, peak located at: 6, 35, 4), where risk drivers demonstrated a significantly greater response than safe. No other main effects and no interactions were observed for secondary contrasts.

5.3.2.2.1.2 Exploratory analysis

No effects of age were observed in exploratory contrasts on a whole-brain level, however, three effects of risk status were observed. These regions were identified through a probabilistic labelling atlas, the AAL. During win cue trial periods, risky male drivers displayed greater activation in the left paracentral lobule (108µl, -18, -25, 64) than safe males, $F(1,76)=26.99$, $p<.05$ (FWE corrected). Safe males demonstrated greater activation than risky in both neutral feedback conditions, hit ($t(76)=4.96$, $p<.05$, FWE corrected) and miss ($t(76)=4.87$, $p<.05$, FWE corrected). For neutral hit this effect was located in the right superior temporal gyrus (108µl, 57, -55, 22) and for neutral miss the effect was observed in the left ACC (81µl, 0, 29, 4). No other main effects and no significant interactions were found at the whole-brain level.

The SVC analysis revealed no significant effects of age and no interactions between age and risk status. The effects of risk status that survived additional corrections for multiple comparisons are listed in table 5.16.

Table 5.16: Summary of significant differences between risky and safe male drivers in certain conditions of the MID

1st level contrast	2nd level contrast	Volume in µl	Region	Coordinates (x, y, z)	F or t statistic*
Win miss > rest	S > R	594	Right ACC	3, 35, -5	*t*(76)=4.76
Neutral miss > rest	S > R	729	Right ACC	6, 35, 4	*t*(76)=4.46
Miss > rest	S > R	1,026	Right ACC	6, 35, 1	*t*(76)=5.49
Win cue > rest	R > S	864	Right SMA	6, -16, 64	*F*(1,76)=23.96
			Left SMA	-3, -22, 61	*F*(1,76)=20.26

* = All effects achieved a significance less that p=.006, FWE corrected. S = safe, R = risky

5.3.2.2.2 Young drivers

5.3.2.2.2.1 Primary and secondary contrasts of interest

The effects of gender and risk status (risky vs. safe) on brain activation in young drivers (<25) was investigated with 2 x 2 Factorial ANOVAs at the group level for the first level contrasts outline in section 5.2.5.5. No effects were found in primary contrasts. In secondary contrasts, SVC analysis identified a greater response in risky young drivers compared to safe in the right SMA (648, 12, -16, 64) for the 'win cue > win hit' condition; $F(1,75)=23.37$, $p<.006$, FWE corrected. No other significant effects were noted.

5.3.2.2.2.2 Exploratory analysis

Significant main effects of risk status for exploratory contrasts are summarised in table 5.17. The effects detected in the right SMA for 'win hit > rest' and 'hit > rest' are illustrated in figures 5.18 and 5.19, respectively, both FWE corrected.

Additional effects were observed in the left and right SMA and the right thalamus for certain conditions, however, these effects were no longer significant after adjusting the significance threshold to account for multiple comparisons. Similarly, in certain conditions, greater activation was observed in the right caudate in young females, compared to young males, for example 'hit > rest', however none of these effects survived additional corrections. This left no surviving main effects of gender. No other effects were observed.

Table 5.17: Summary of significant differences between risky and safe younger drivers in certain conditions of the MID

1st level contrast	2nd level contrast	Volume in µl	Region	Coordinates (x, y, z)	F or t statistic*
Win hit > rest	S > R	2,052	Right SMA	12, -16, 64	*t*(75)=4.98
Hit > miss	S > R	270	Left Thalamus	-21, -25, 13	*t*(75)=4.99
Miss > rest	S > R	216	Right ACC	9, 35, 1	*t*(75)=4.36
Hit > rest	S > R	1,755	Right SMA	9, -25, 61	*t*(75)=4.93
			Left SMA	-9, 16, 61	*t*(75)=4.48

* = All effects achieved a significance less that p=.006, FWE corrected. S = safe, R = risky

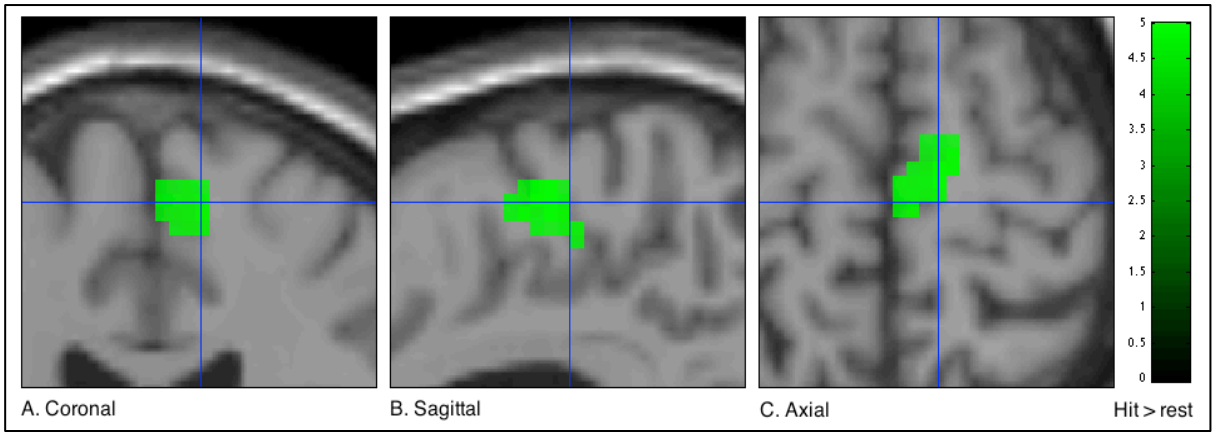

Figure 5.18: Coronal, sagittal and axial views of the part of the right SMA where safe young drivers demonstrated more activation than risky during the 'hit > rest' condition of the MID, 80 x 80 mm zoom

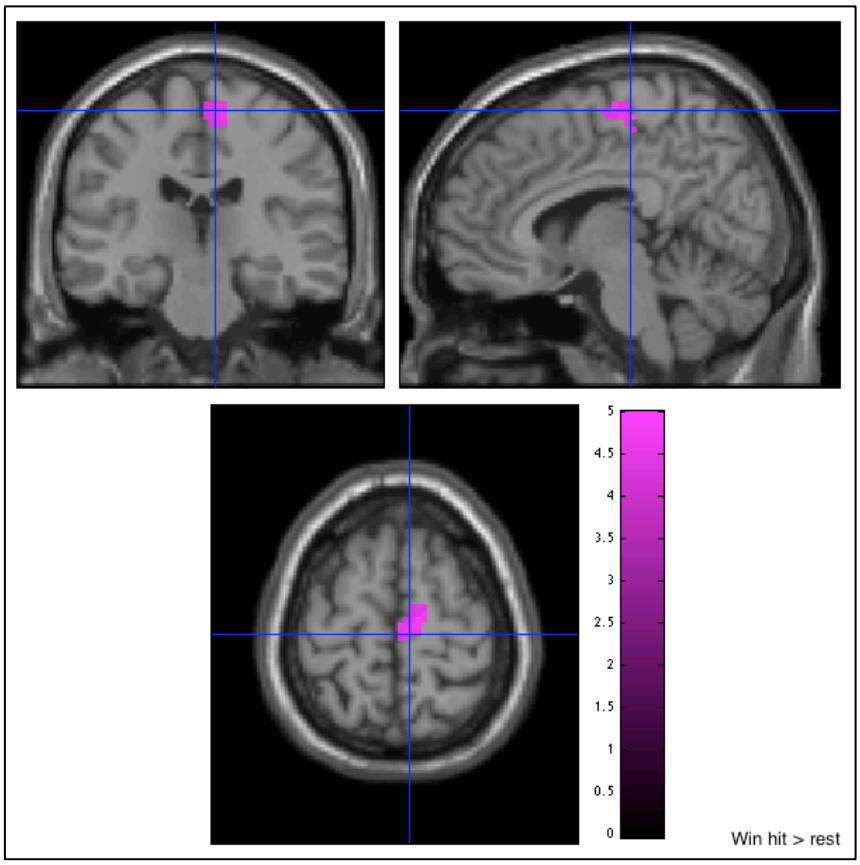

Figure 5.19: Young safe drivers demonstrated greater activation in the right SMA than young risky drivers when they received hit feedback following win stimuli in the MID, compared to rest

5.3.2.2.3 Young male drivers

5.3.2.2.3.1 Primary and secondary contrasts of interest

One-way ANOVAs of risk status in younger males on the first level contrasts mentioned in section 5.2.5.5 revealed no significant effects of risk for primary contrasts, either at whole-brain or SVC level. When comparing reward anticipation and receipt trials ('win cue > win hit', a contrast of secondary interest), safe young males had greater activity in the right DLPFC ($t(38)=5.68$, $p<.05$, FWE corrected, 81µl, located at: 24, 5, 61) and left precentral gyrus ($t(38)=5.59$, $p<.05$, FWE corrected, 81µl, located at: -36, -1, 46) than risky young males. These effects were evident at a whole-brain level and were identified

through the AAL in SPM12. A further effect of risk was observed with an SVC approach for this contrast ('win cue > win hit') in the left caudate tail (54μl, -18, 5, 19), where safe young males had a greater response than risky; $t(38)=4.35$, $p<.006$, one-tailed, FWE corrected.

5.3.2.2.3.2 Exploratory analysis

For the receipt of a monetary gain (hitting a win target), risky younger males demonstrated significantly more activation in the right DLPFC (81μl, 24, 2, 61) than safe, $t(38)=5.61$, $p<.05$ (FWE corrected), detected at a whole-brain level.

Furthermore, SVC analysis revealed the differences summarised in table 5.18. Aspects of the bilateral caudate that demonstrated a main effect of risk status for win hit feedback are illustrated in figure 5.20, FWE corrected.

Table 5.18: Summary of differences between risky and safe young male drivers in certain conditions of the MID

1st level contrast	**2nd level contrast**	**μl**	**Region**	**Coordinates (x, y, z)**	**t statistic***
Win hit > rest	R > S	108	Left DLPFC	-30, 38, 34	4.77
		135	Left SMA	-9, 2, 61	4.33
		81	Left Caudate tail	-18, 5, 19	4.45
		135	Right Caudate head	15, -7, 16	4.28
		81	Right Caudate tail	21, 20, 10	4.22
	S > R	54	Right Thalamus	18, -13, 16	3.97
Win miss > rest	R > S	54	Right Caudate head	18, 14, 16	4.26
Miss > rest	R > S	81	Right VMPFC	9, 26, 55	4.28
Hit > rest	R > S	81	Left SMA	-9, 2, 61	4.46
		108	Right Caudate head	21, 20, 10	4.3

* = All effects significant at less that p=.006, FWE corrected, with df = 38. S = safe, R = risky

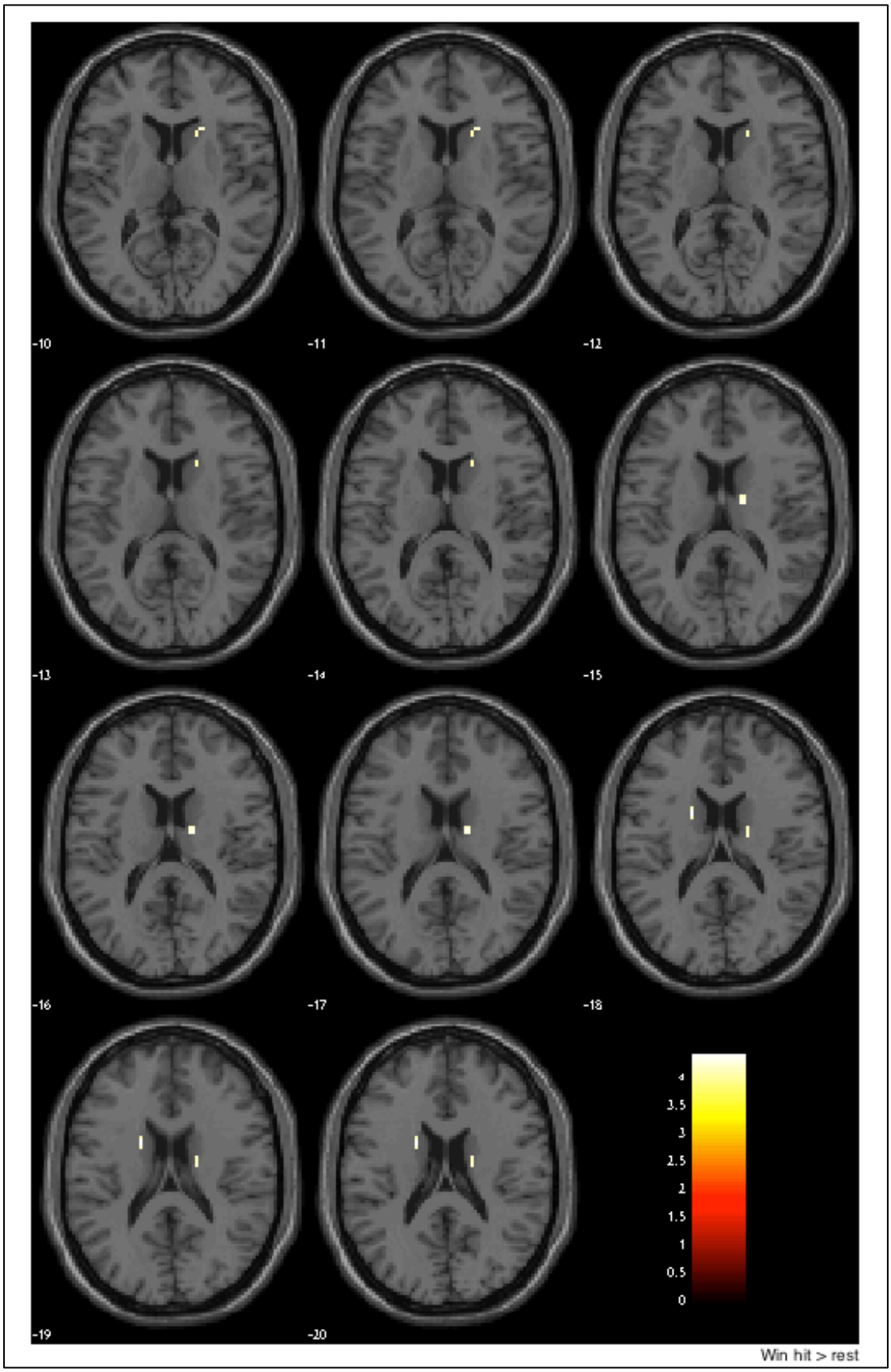

Figure 5.20: Slices showing the aspects of the left and right caudate where risky young males had significantly more activation to the 'win hit > rest' contrast of the MID than safe young males

5.3.3 Go/No Go Task

5.3.3.1 Task activation

In this task participants had to respond to alternating 'X's and 'Y's on screen and inhibit their response when a letter was repeated twice in a row. 'go' responses, i.e. responding to alternating stimuli, were treated as the baseline (rest) for this task.

5.3.3.1.1 Failure to respond to a go stimulus: 'Failed go > rest'

When participants made an omission error (failure to respond to 'go' stimuli) compared to 'baseline', activation was observed in the ACC (right: 2,916µl, 6, 29, 28, left: 1,134µl, -6, 38, -8), bilateral insula (right: 2,160µl, 33, 20, 7, left: 2,106µl, -33, 17, 10), bilateral SMA (9,045µl, peak on right side: 9, 17, 46) and part of the VMPFC (2,538µl, right peak: 6, 20, 43, left peak: -3, 14, 43). Figure 5.21 shows a sagittal view of these regions. All figures in show results using FWE correction.

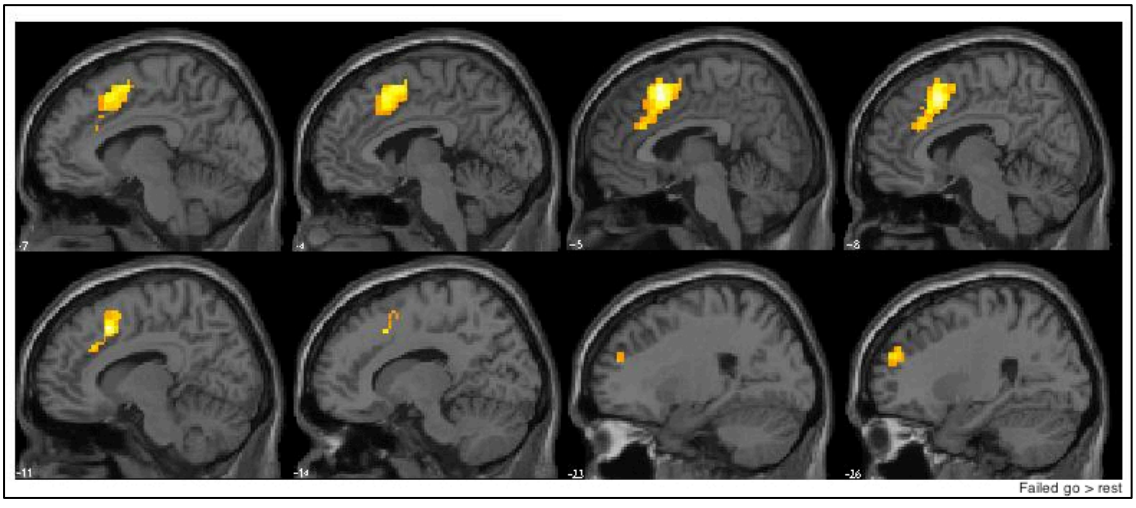

Figure 5.21: Sagittal view of the regions active for omission errors compared to baseline in the Go/No Go task.

5.3.3.1.2 Successful inhibition: 'Successful No Go > rest'

When participants made a successful inhibition there was involvement of the cingulum across posterior, middle and anterior regions, the inferior parietal cortex, right hippocampus, supramarginal gyrus, SMA, putamen, insula and frontal regions, detailed in table 5.19. Subcortical and cortical illustrations of these regions are displayed in figures 5.22 and 5.23, respectively.

Table 5.19: Regions active during successful inhibitions in the Go/No Go task

Region	**Hemi.**	**Volume (µl)**	**F**	**Peak p (FWE)**	**Coordinates**
ACC	Right	21870	225.84	0.000	9, 32, 22
	Left		166.24	0.000	-6, 35, 25
DLPFC	Right	7938	107.47	0.000	45, 44, 19
	Left	5265	49.05	0.000	-45, 41, 22
IFG	Right	10584	83.92	0.000	45, 38, 25
	Left	5211	63.52	0.000	-57, 8, 13
Inf Parietal	Right	8289	178.29	0.000	51, -40, 55
	Left	13608	131.33	0.000	-54, -37, 49
Insula	Right	6156	93.6	0.000	42, 20, -11
	Left	9207	101.55	0.000	-33, 8, 7
PCC	Right	243	32.32	0.000	6, -37, 7
Putamen	Right	6993	77.41	0.000	24, 2, 13
	Left	5373	60.63	0.000	-21, 5, 16
SMA	Right	17037	157.65	0.000	9, 14, 58
Thalamus	Right	5184	53.92	0.000	9, -31, 4
	Left		36.21	0.000	-6, -31, 4
VMPFC	Right	19062	157.31	0.000	3, 23, 40

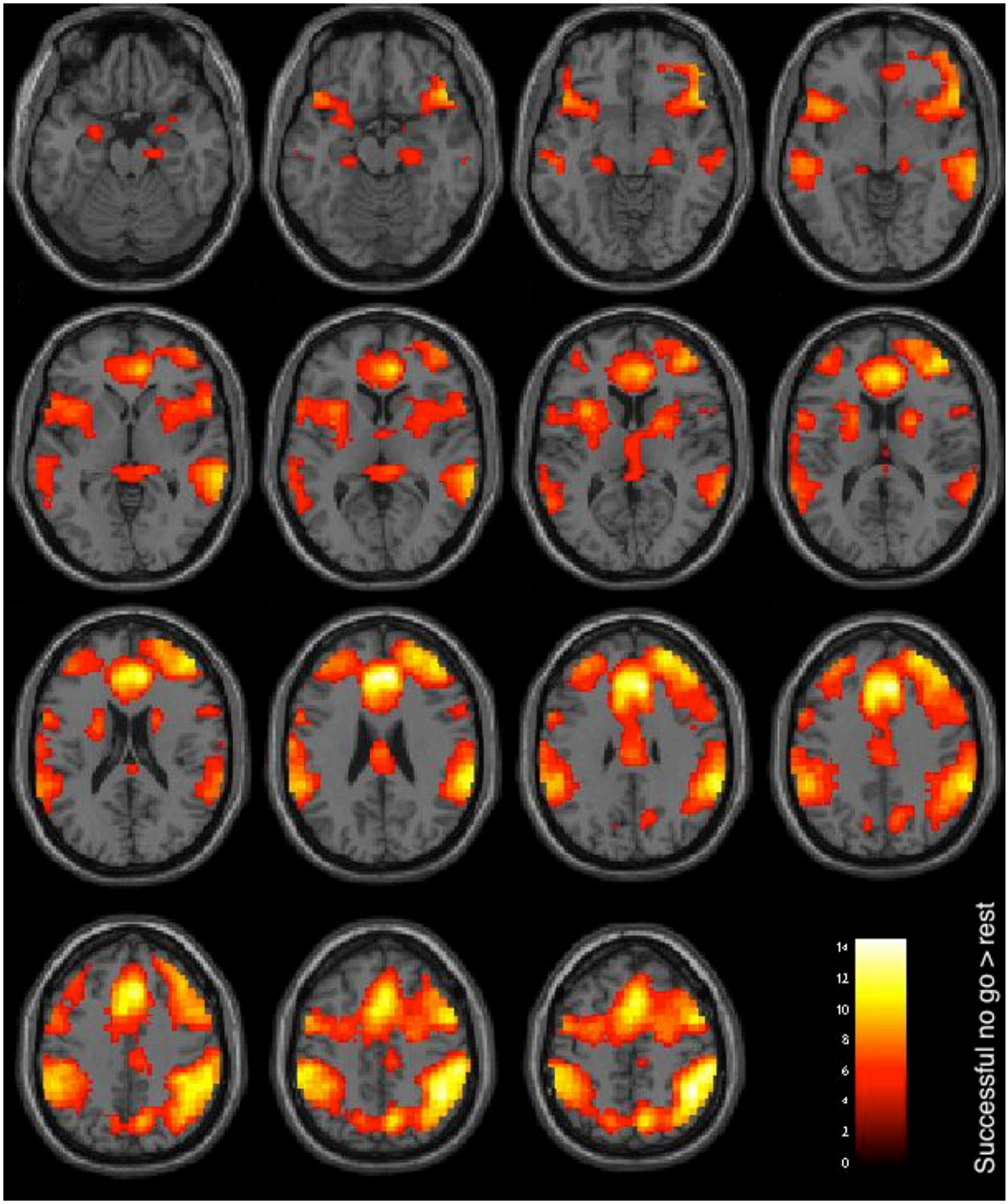

Figure 5.22: Axial view of the regions active when participants successfully inhibited their response to a no go stimulus, compared to baseline, on the Go/No Go task

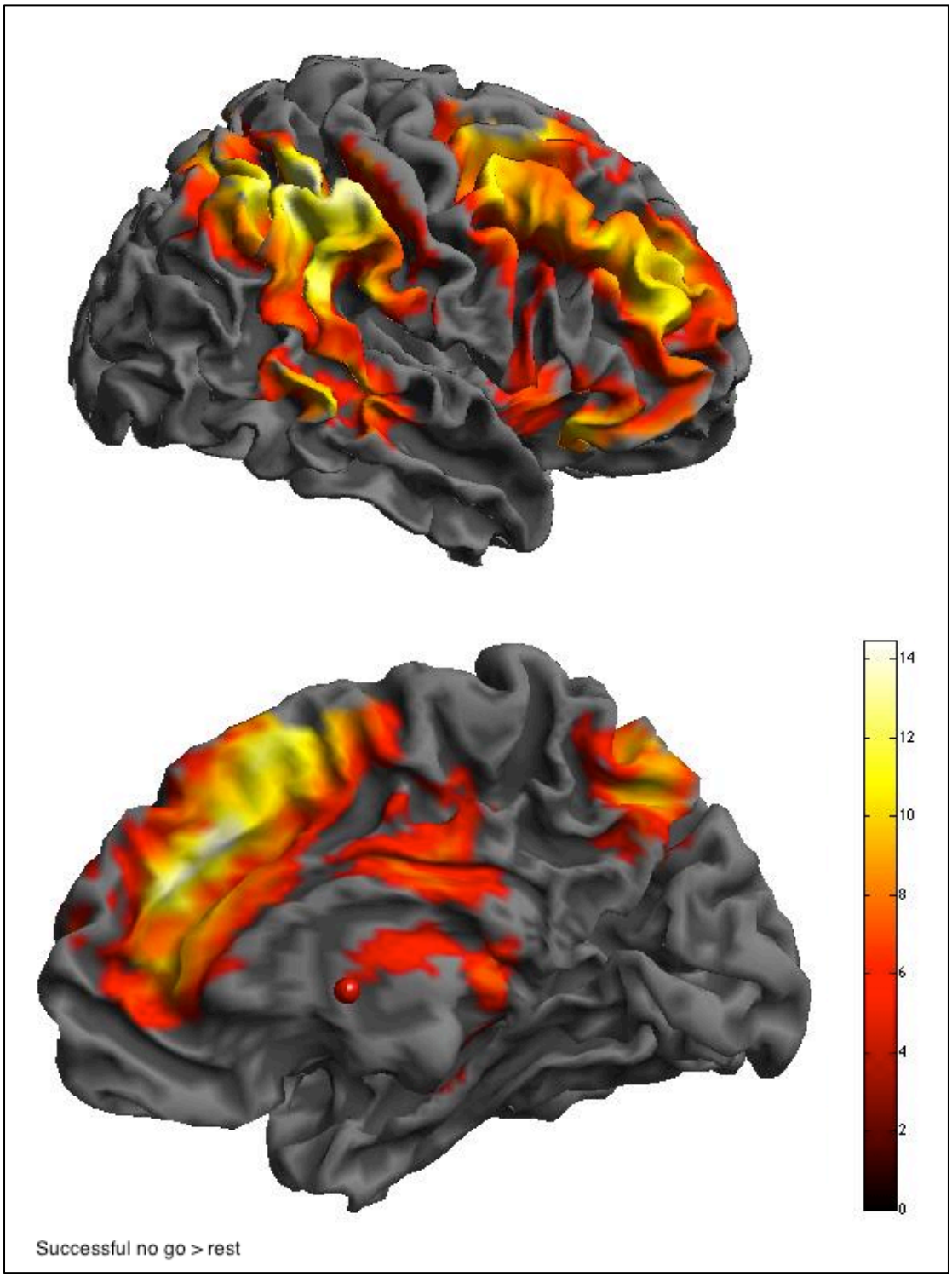

Figure 5.23: Rendering of the lateral and medial aspects of the right hemisphere that demonstrated significant activation when participants successfully inhibited their response to lure stimuli in the Go/No Go task

5.3.3.1.3 Successful inhibitions over errors of omission: 'Successful No Go > Failed go'

These regions were also active when successful inhibitions were compared to errors of omission, both of which do not involve a motor response. In this contrast, additional activation was also observed in the left and right caudate, thalamus and hippocampus. This is represented in figure 5.24 below.

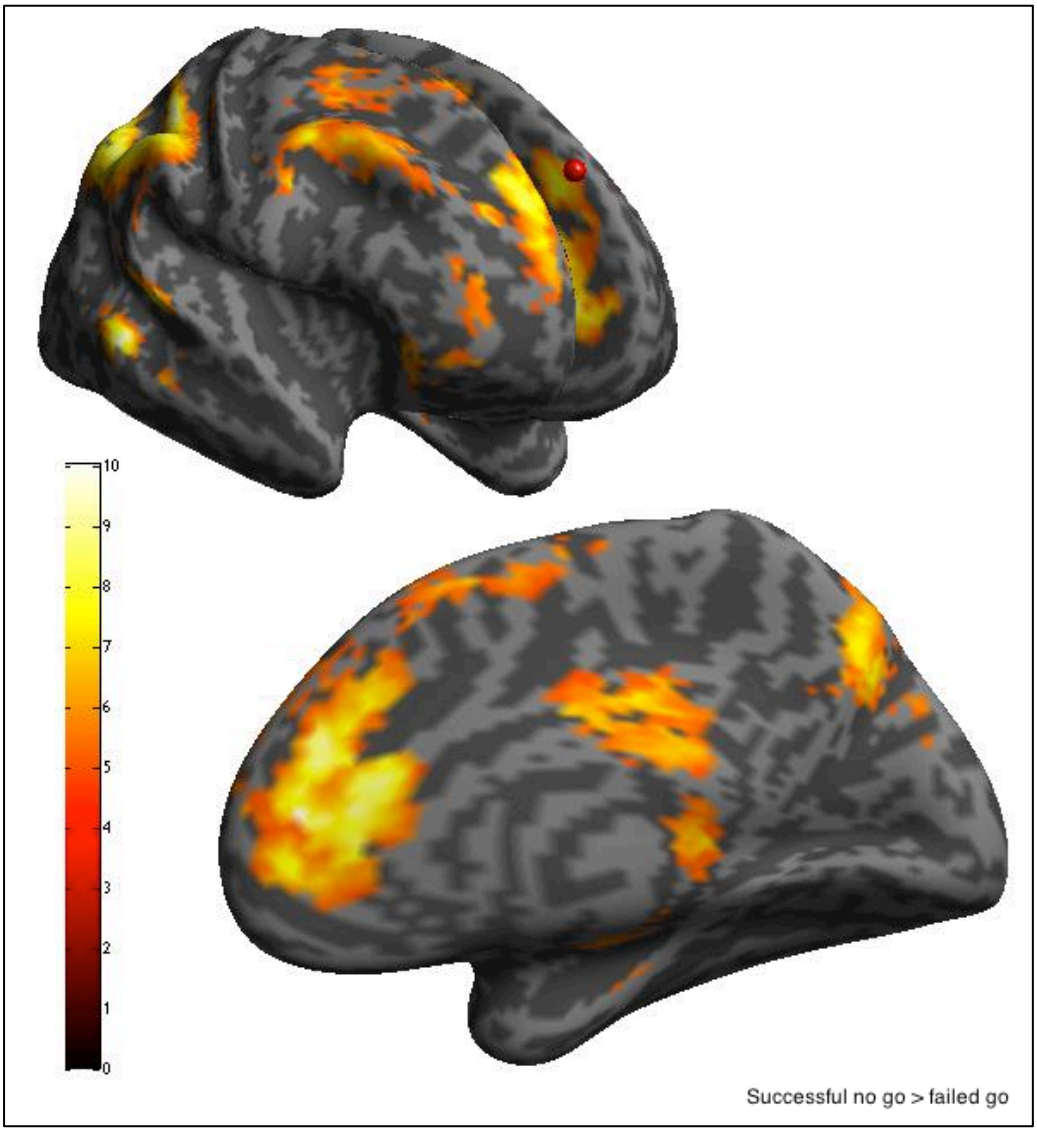

Figure 5.24: Inflated rendering of regions demonstrating greater activation during successful inhibition compared to errors of omission in the Go/No Go task. Medial aspect of the right hemisphere is shown at the bottom of the image.

5.3.3.1.4 Failed inhibition: 'Failed No Go > rest'

When participants failed to inhibit their responses to 'no go' (lure) stimuli they demonstrated a significant, positive response in the supramarginal gyri, inferior parietal lobe, IFG, SMA, pre and postcentral gyri, medial temporal gyrus, ACC, DLPFC (medial aspect of Brodmann 9 only), VMPFC and insula. During these commission errors there was also significant deactivation in occipital regions, the putamen, thalamus, cerebellum. The effects that were observed in the ROIs specified in table 5.7 are summarised in table 5.20 below.

Table 5.20: Pattern of activation associated with errors of commission in the Go/No Go task

Direction	**Region**	**Hemi.**	**Volume (μl)**	**F**	**Peak p (FWE)**	**Coordinates**
> Baseline	ACC	Right	20223	294.03	0.000	6, 32, 25
		Left		281.45	0.000	-3, 29, 25
	DLPFC	Right	2700	52.93	0.000	6, 41, 19
		Left	2268	55.49	0.000	-3, 50, 19
	IFG	Right	1161	43.42	0.000	54, 23, -2
		Left	7128	48.46	0.000	-48, 26, 22
	Inf Parietal	Right	6669	144.13	0.000	45, -55, 52
		Left	5130	97.51	0.000	-57, -37, 46
	Insula	Right	4023	119.66	0.000	36, 20, -17
		Left	4050	106.26	0.000	-42, 11, -5
	SMA	Right	4752	128.57	0.000	12, 20, 58
	VMPFC	Right	14472	99.03	0.000	12, 23, 61
< Baseline	Putamen	Right	7452	171.79	0.000	18, 8, -8
		Left	7749	192.14	0.000	-15, 5, -8
	Thalamus	Left	54	26.89	0.001	-21, -13, 4

This pattern of activation can be seen in figure 5.25, where 'hot' tones (from red to yellow) represent significant activation and 'cool' tones (from dark to light blue) represent significant deactivation.

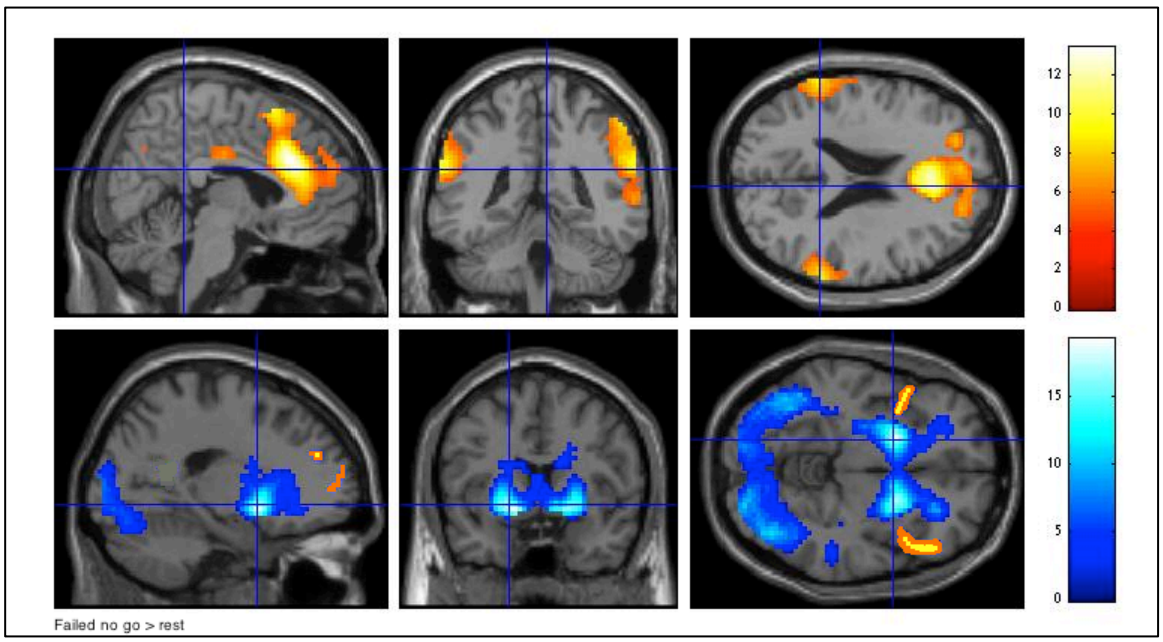

Figure 5.25: Activation pattern observed during errors of commission in the Go/No Go task.

5.3.3.1.5 Failed inhibition over failure to respond to go stimuli: 'Failed No Go > Failed go'

When comparing commission to omission errors, similar regions were found to be significantly active, with the exception of the insula, which was significantly deactivated in this condition (left: 1,431µl, -30, 26, 1, right: 810µl, 33, 29, -2). For this contrast, no significant signal was detected in the thalamus. Figure 5.26 illustrates these effects. Regions in red demonstrated greater activation during commission errors compared to omission. When participants failed to inhibit responses to lure stimuli they also demonstrated significant deactivation in blue regions.

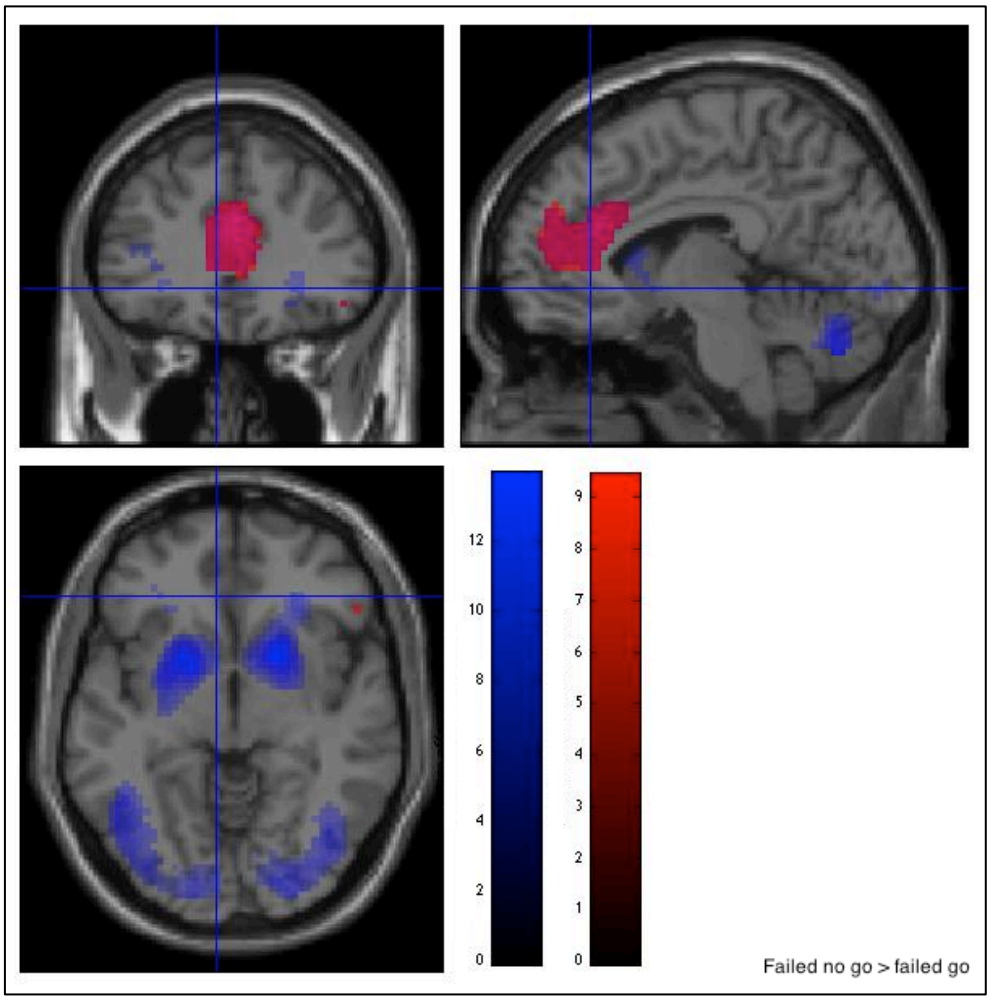

Figure 5.26: Illustration of the 'Failed No Go > Failed go' condition of the Go/No Go task

5.3.3.1.6 Successful inhibition over failed inhibition: 'Successful No Go > Failed No Go'

Finally, the location and extent of the ROIs that showed significantly more activation for successful inhibitions compared to errors of commission are summarised in table 5.21 and illustrated in figure 5.27. In addition to these regions significant effects were observed in the superior and medial temporal gyri, vermis, right hippocampus, right precuneus, occipital regions and parts of the cerebellum.

Table 5.21: Regions demonstrating greater activation for successful inhibition trials compared to errors of commission

Region	Hemisphere	Volume (µl)	F	Peak p (FWE)	Coordinates
ACC		999	28.2	0.001	0, 23, 19
DLPFC[23]	Right	5373	68.91	0.000	42, 35, 34
	Left	4509	60.76	0.000	-42, 29, 37
IFG	Right	9396	76.03	0.000	42, 32, 28
	Left	13716	67.9	0.000	-39, 44, 4
Inf Parietal	Right	6669	69.16	0.000	42, -40, 49
	Left	13797	84.73	0.000	-39, -40, 43
Insula	Right	783	41.74	0.000	33, -1, 13
	Left	3240	81.52	0.000	-27, 17, 4
PCC	Right	459	21.62	0.002	12, -40, 16
Putamen	Right	9126	337.34	0.000	24, 11, -2
	Left	7911	334.13	0.000	-24, 11, 1
SMA	Right	7722	50.67	0.000	12, 8, 49
	Left		59.9	0.000	-6, 2, 52
Thalamus	Right	13392	68.2	0.000	9, -7, 4
	Left		75.64	0.000	-21, -13, 7
VMPFC	Right	351	56.18	0.000	18, 50, 4
	Left	918	26.04	0.002	-9, 26, 43

[23] Brodmann 46 only

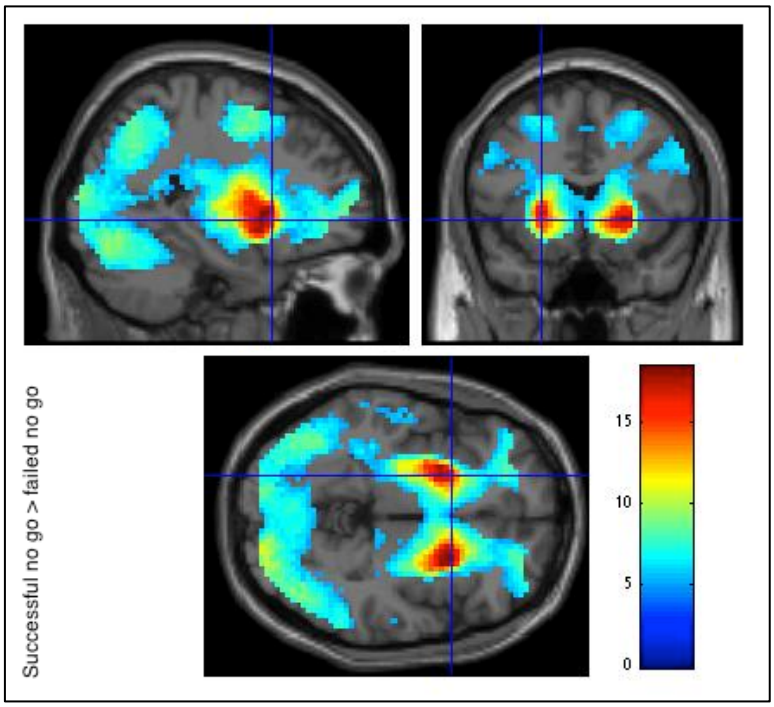

Figure 5.27: Regions of the brain that were active during successful inhibitions compared to commission errors in the Go/No Go task. This effect was largest in the bilateral putamen, coloured in red. Results corrected using FWE.

5.3.3.2 Between group comparisons

5.3.3.2.1 Male drivers

The effects of risk status (risky vs. safe) and age (18-24 vs. 29+) in males on brain activity during the Go/No Go were investigated utilising 2 x 2 Factorial ANVOAs at the group level for the first level contrasts outline in section 5.2.5.5. No main effects or interactions were observed at a whole brain level for primary contrasts. However, when successful inhibitions were compared to errors of commission with an SVC analysis ('Successful No Go > Failed No Go'), risky male drivers demonstrated greater activation than safe in the left ($F(1,76)=26.88$, $p<.006$, FWE corrected, 135µl, -12, -13, 61) and right SMA ($F(1,76)=19.45$, $p<.006$, FWE

corrected, 81µl, 12, 2, 52). No other effects survived corrections for multiple comparisons.

For secondary contrasts, a significant main effect of risk status was observed at the whole-brain level when commission errors were compared to omission errors ('failed no go > failed go'), such that safe males demonstrated greater activation in the right cuneus ($t(76)=5.51$, $p<.05$, FWE corrected, 81µl, located at: 15, -82, 25) and left postcentral gyrus ($t(76)=5.17$, $p<.05$, FWE corrected, 108µl, located at: -39, -31, 55), identified through the AAL probabilistic atlas. No significant effects of age and no interactions between age and risk status were observed at the whole-brain level.

When SVCs were applied, other differences between risky and safe male drivers were detected. When errors of commission were compared to errors of omission, safe male drivers were found to have a significantly greater response than risky in the left inferior parietal lobe, $t(76)=4.84$, $p<.006$ (FWE corrected), and the right SMA, $t(76)=4.77$, $p<.006$ (FWE corrected). These effects were located at coordinates -45, -28, 46 (108µl,) and 12, -28, 52 (216µl), respectively. The effect in the right SMA is illustrated in figure 5.28 below.

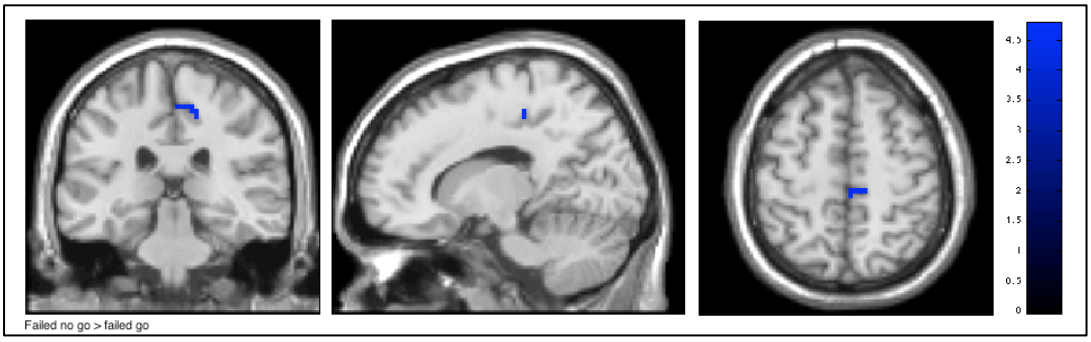

Figure 5.28: Coronal, sagittal and axial views of the right SMA where safe males demonstrated more activation than risky for 'Failed No Go > Failed Go' in the XY task

SVC analyses revealed no main effects of age, however a significant interaction between age and risk status in males was detected in the right DLPFC (162µl, 6, 41, 25) when they made successful inhibitions compared to errors of omission, $F(1,76)=22.3$, $p<.006$ (FWE corrected), illustrated in figure 5.29 below. When the effect sizes were exported to SPSS this interaction remained, $F(1,76)=23.52$, $p<.001$, $\eta^2=.237$, and was found to be the result of a significantly greater response in younger safe males (M=1.76, SD=1.21) compared to younger risky males (M=0.18, SD=1), where no such difference existed in older males (risky: M=1.3, SD=0.8, safe: M=0.56, SD=1.21). No other effects were observed and no significant main effects or interactions were found for exploratory contrasts.

Figure 5.29: Graph illustrating the interaction between age and risk status in the DLPFC in males for successful inhibitions over errors of omission in the Go/No Go task.

5.3.3.2.2 Young drivers

The effects of gender and risk status (risky vs. safe) on brain activation in young drivers (<25) during the Go/No Go task were investigated with 2 x 2 Factorial

ANOVAs at the group level for the first level contrasts outline in section 5.2.5.5. No significant main effects or interactions were found for primary, secondary or exploratory contrasts on either a whole-brain level or through SVC analyses, once the significance threshold had been adjusted for the number of comparisons made.

5.3.3.2.3 Young male drivers

One-way ANOVAs of risk status in younger males on Go/No Go task related activation during the first level contrasts mentioned in section 5.2.5.5 revealed no significant whole-brain effects and no group differences when a SVC approach was applied, in any of the contrast types: primary, secondary or exploratory.

5.3.4 Correlations with other measures

5.3.4.1 Male drivers

Mean activation for a selection of conditions across the three tasks were extracted to examine brain-behaviour relationships. Activation magnitude was then correlated with scores on subscales from the Manchester DBQ, the BIS-11 and Kirby's DDQ, certain behavioural measures from the IGT, MID and Go/No Go tasks and total number of penalty points accrued. To account for the number of comparisons made, the significance threshold was set to $p<.003$.

With this adjusted threshold there was a significant, positive, correlation between scores on the highway-code (HC) violations subscale of the DBQ and activation in the SMA during win and neutral cue periods; win: $r(80)=.346$, $p<.003$, two-tailed; neutral: $r(80)=.387$, $p<.003$, two-tailed. Activation in the SMA during win cue

periods also correlated positively with the aggressive violations subscale of the DBQ: SMA; $r(80)=.344$, $p<.003$, two-tailed. In all instances, higher scores on these subscales were associated with greater activation in the SMA.

In the self-control subscale of the BIS a significant, positive correlation was found with SMA activation for successful inhibitions over commission errors ('Successful no Go > Failed No Go'), $r(80)= .354$, $p<.003$, two-tailed. A scatter plot illustrating this relationship can be found in figure 5.30.

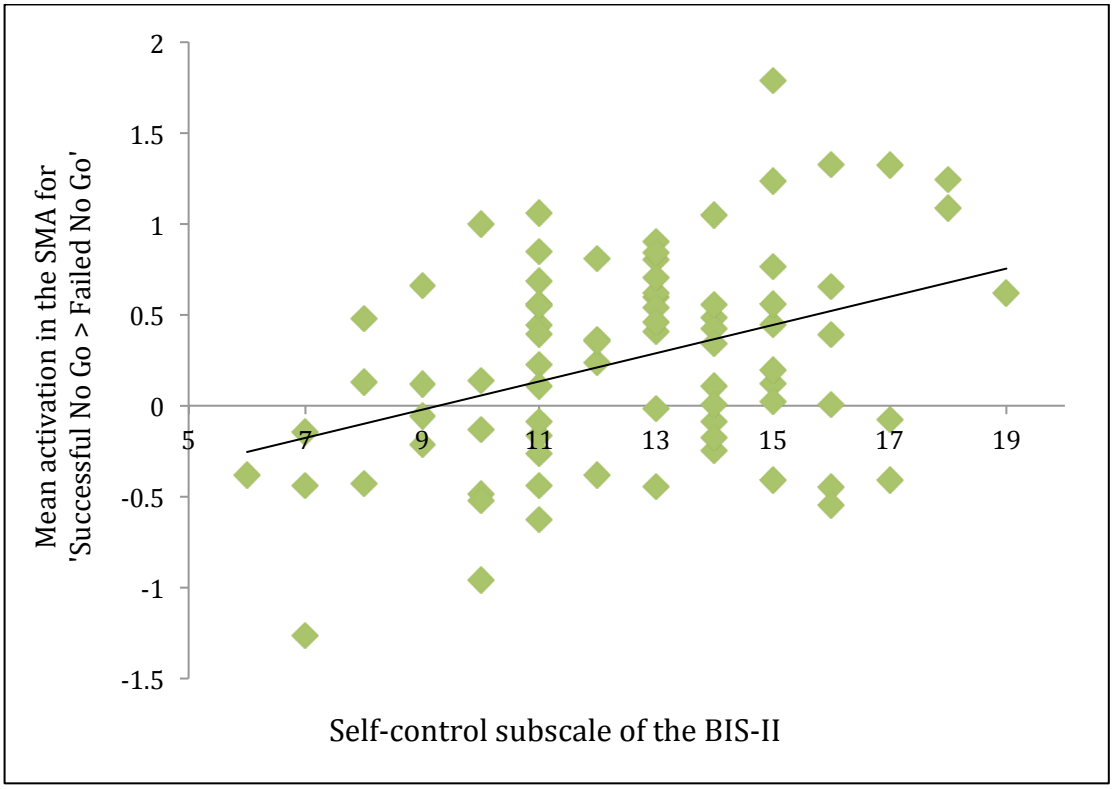

Figure 5.30: Scatter plot illustrating the relationship between the mean activation in the SMA for the 'Successful No Go > Failed No Go' contrast of the Go/No Go task and the self-control subscale of the BIS-11 in males

The self-control subscale of the BIS demonstrated a significant, negative correlation with hippocampal activation during feedback from deck A in the IGT, $r(79)= -.36$, $p<.003$, two-tailed, such that higher scores on this subscale were

associated with reduced activation in the hippocampus during this condition. No other significant correlations were found.

5.3.4.2 Young drivers

Mean activations for a selection of conditions were extracted across the three tasks in younger individuals. These were then correlated with the same measures used in the previous section and the significant threshold, again, was set to p=.003. Several significant relationships between activation observed during the MID task and behavioural measures from the MID were found. These are summarised in table 5.22 below.

Table 5.22: Correlations between average activation from the right caudate in certain conditions of the MID and behavioural measures from the MID

MID behavioural measure	**MID contrast**	**Pearson's r (p<.003, two tailed)**
No. of win hits	Win miss > rest	$r(79)$= **–.358**
	Neutral hit > rest	$r(79)$= **–.344**
No. of neutral hits	Neutral hit > rest	$r(79)$= **–.358**
Win RT	Win miss > rest	$r(79)$= **.332**
Neutral RT	Win miss > rest	$r(79)$= **.374**
	Neutral hit > rest	$r(79)$= **.379**
	Hit > rest	$r(79)$= **.351**

A significant correlation was also found between the number of penalty points accrued and the degree of activation observed in the SMA for the 'win cue > win hit' contrast of the MID, $r(79)= .338$, $p<.003$, two-tailed.

This condition of the MID also correlated positively with HC ($r(79)= .366$, $p<.003$, two-tailed) and aggressive violations ($r(79)= .345$, $p<.003$, two-tailed) in the DBQ. Furthermore, SMA activation when younger drivers received hit feedback for win targets negatively correlated with aggressive violations in the DBQ, $r(79)= -.341$, $p<.003$, two-tailed. In younger individuals, no other significant correlations were found between activation and psychometric or behavioural measures.

5.3.4.3 Young male drivers

Certain mean activations were extracted from younger males across the three tasks. These were correlated with the same select psychometric and behavioural measures and were subject to a stringent threshold of 0.003. Significant correlations between the self-control subscale of the BIS and certain conditions in the MID were found.

This subscale was found to be positively correlated with the degree of activation in the SMA during feedback from hitting win targets, $r(40)= .493$, $p<.003$, two-tailed, and during all hit feedback conditions, $r(40)= .53$, $p<.003$, two-tailed, the latter of which is illustrated in figure 5.31. Finally, this subscale also correlated with the magnitude of activation in the right caudate head, $r(40)= .472$, $p<.003$, two-tailed, during miss feedback for win targets in the MID. No other significant correlations were found.

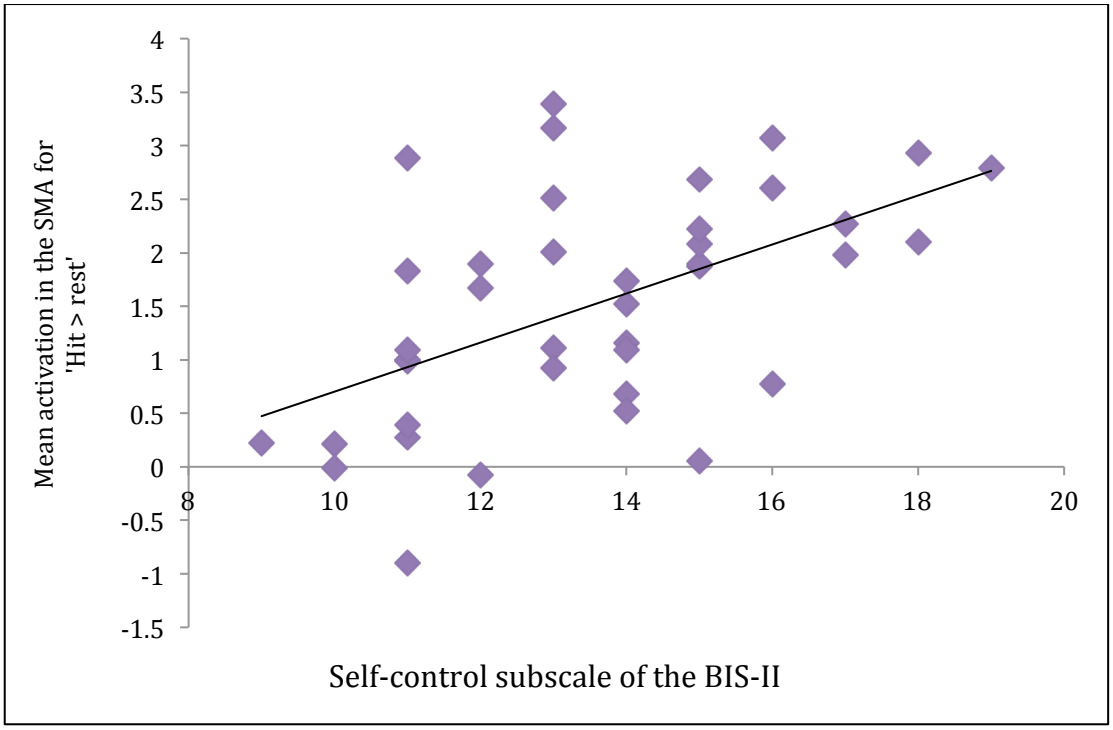

Figure 5.31: Scatter plot illustrating the relationship between the mean activation in the SMA for the 'Hit > rest' condition of the MID and the Self-control subscale of the BIS-11 in younger males

5.4 Discussion

5.4.1 Summary of findings

Firstly, while not of interest to the current hypotheses, it was useful to compare the activation observed during the various conditions of the three tasks to previous research in order to determine whether the current analytical models were appropriate. Such a comparison revealed that the regions found to be active during these tasks were consistent with the findings of the articles for which the ROIs were chosen for SVC analyses (for the IGT; Li et al., 2010; Lin et al., 2008; for the MID; Knutson et al., 2000; Nestor et al., 2010; and for the Go/No Go task; Garavan et al., 2006; Kaufman et al., 2003). Furthermore, inspection of activation maps included in articles that have used these tasks showed results similar to those observed in the current study (Garavan et al., 2006 for the Go/No Go task; X.

Li et al., 2010 for the IGT; and Nees et al., 2012 for the MID task). This suggests that the present study replicated the activation observed during task conditions that has been previously reported in the literature.

The main results of the between groups analyses are summarised below under the relevant hypotheses.

5.4.1.1 Hypothesis one: there are changes in activation with age

It was hypothesised that younger males (18 to 24) would demonstrate different patterns of activation in prefrontal 'control' systems and reward processing regions when compared to older adult males (over 29), but not to the same extent reported in previous research.

Such research has found that, during tasks of cognitive control, activation appears to become more regionally specific, or 'focused', with age (Durston & Casey, 2006; Durston et al., 2006). The current study found no differences in activation in prefrontal regions between younger (18 to 24) and older (29 to 56) males during the Go/No Go task. The most probable explanation for this lack of finding is that the age of the 'younger' group in the current study differed dramatically to those used in previous research. For example Durston et al. (2006) examined changes in neural response to a cognitive control task (similar to the Go/No Go paradigm) with age utilising a longitudinal design. Despite only comparing children at two timepoints (ages nine and 11), these findings have been widely, and perhaps inappropriately, generalised to the adolescent literature (e.g. Casey et al., 2008; Steinberg, 2008). It may be that, while the brain continues to develop structurally,

becoming more efficient with age, an already functioning system of inhibition exists in older adolescents.

Previous research also suggested that the magnitude of response in reward processing regions would differ between adolescents and adults. It was unclear, however, whether this would manifest in a greater (Ernst et al., 2005; Galvan et al., 2006) or a reduced response (Bjork et al., 2004) in adolescents, relative to adults. The current study failed to clarify these findings further, as no differences in reward processing were found between older and younger males. Again, the age ranges of the 'adolescent' samples in these studies were not directly comparable to that of the current study. In actuality the age range of the younger group in the current study was more similar to the 'adult' samples used in previous experiments. For example the Bjork et al. (2004) and Galvan et al. (2006) studies compared adolescents between 12 and 17 to adults from 22 to 28 and 23 to 29 years of age, respectively. Lopez-Larson et al. (2011) even included children in their 'adolescent' sample (9 to 17) and their adult sample spanned almost the entire range of that used in the current study; 20 to 40 years of age. The lack of differences in the activation of prefrontal and reward processing regions between younger and older males in the current study suggests that Casey et al. (2008)'s popular neurodevelopmental explanation for heightened impulsivity in adolescence (also adopted by Steinberg, 2008), may not apply to older adolescents and young adults (<25), despite the high frequency of engagement in risk-taking still observed during this age. It may also be the case, as will be discussed in greater detail below, that there are individual differences in the rate of maturation

of these systems, where protracted development in some individuals may manifest in increased risk-taking.

Although not directly related to this theory, one significant difference was found between younger and older males in the IGT. In order to be successful in the IGT a participant must have the ability to remember gains and losses from past selections in the task. X. Li et al. (2010) conducted a detailed fMRI study of the IGT and hypothesised that the hippocampus would form part of the task-activated network. However, they failed to find activation in the hippocampus in their sample of college age students (mean 23 years). In the current study, older adult males (29 to 56) demonstrated a significantly greater response in the hippocampus upon receipt of feedback from both frequent punishment decks (decks A and C, separately) when compared to younger males (18 to 24). This could suggest that older males employed a different system of encoding outcomes from the task into memory. The age range of the current study may explain why previous research has not observed hippocampal involvement in the IGT.

5.4.1.2 Hypothesis two: there are activational differences between risky and safe younger drivers, independent of gender

Due to the overrepresentation of younger individuals in collision statistics it was expected that some neural mechanisms that may make younger drivers more vulnerable to risk-taking would be independent of gender. This hypothesis was not supported in either the IGT or Go/No Go task. Therefore, when young males and females are combined, safe and risky drivers appear not to differ in the processes by which they make decisions under risk or inhibit actions.

In the MID, however, risky drivers demonstrated a greater response than safe in the SMA in anticipation of a monetary reward over receipt of that reward (win cue > win hit, a secondary contrast). This could reflect a greater motivation to obtain the monetary reward, as this region has been implicated in preparing to act in anticipation of a reward (Whelan et al., 2012). Further differences were observed in exploratory contrasts, for example, in response to feedback in the MID (compared to rest), safe drivers recruited the left thalamus and SMA during hit feedback and the right ACC during miss feedback to a greater extent than those with a history of risky driving behaviours.

Reduced activation in the thalamus in risky young individuals is similar to what has been observed in previous research into reward responses of clinical populations. In a meta-analysis of responses to positive and rewarding stimuli, W. N. Zhang, Chang, Guo, Zhang, and Wang (2013) found that patients with MDD (major depressive disorder) had reduced activation in the thalamus in response to monetary rewards, when compared to healthy controls. In the current study this reduction in response to hit feedback may be indicative of aberrant reward consumption in individuals vulnerable to risk-taking while driving.

As mentioned in the introduction, the ACC is implicated in action monitoring (Carter et al., 1998; Garavan, Ross, & Stein, 1999; Kaufman et al., 2003) and error detection (X. Li et al., 2010), while the SMA has been associated with preparation (Ikeda et al., 1999) and cancellation of actions (Picton et al., 2007), abilities necessary for efficient executive control (Botvinick et al., 2001). Reduced activation in the ACC and SMA has been observed in substance users (cocaine), compared to healthy controls, during both successful inhibitions and commission

errors on a Go/No Go task (Kaufman et al., 2003). As risky drivers demonstrated 'hypoactivation' of the ACC to 'misses' and the SMA to 'hits', this could be evidence for deficiencies of executive systems similar to what has previously been observed in substance abusers (Kaufman et al., 2003). Furthermore, previous research has demonstrated a significant, negative correlation between activation in the ACC and impulsiveness (Patel et al., 2013), such that increasing levels of impulsivity were associated with less activation in this region in anticipation of 'loss'. Since young risky drivers demonstrated a greater reduction in the ACC in response to miss feedback in the MID than safe younger drivers, this could indicate higher "neural" impulsiveness in risky drivers. As no differences were observed in primary contrasts, and only one secondary contrast demonstrated a significant effect of risk in younger individuals, these findings provide preliminary evidence to suggest that there may be neural mechanisms in younger individuals that put them at increased risk while driving. The implications of these findings are discussed at the end of hypothesis four.

5.4.1.3 Hypothesis three: there are activational differences between risky and safe young male drivers

The ability to detect differences between risky and safe individuals in the previous hypothesis may have been confounded by particularities of the young males. Males within this age range are involved in proportionately more fatal collisions than any other demographic group, and yet, not all young males participate in risky driving behaviours. To ascertain whether certain neural processes in young males were associated with risky driving, risky and safe younger male groups were compared directly. No significant differences were observed in the primary

first level contrasts identified in section 5.2.5.5 and only one difference in a secondary contrast was found, identified towards the end of this section. The following results were observed when younger risky and safe males were compared on exploratory contrasts and should therefore be interpreted as preliminary findings.

No differences in either performance or activation were found between these groups in the Go/No Go task. This suggests that young risky and safe males do not differ in the mechanisms by which they inhibit actions.

In the IGT, risky younger males demonstrated greater activation in the right amygdala in response to feedback from deck A, one of the two 'disadvantageous' decks. Healthy controls have been observed to switch preference away from this deck early in the task, suggesting that it is relatively easy to determine the disadvantageous nature of this deck (Lin et al., 2007). If selecting from this deck resulted in a greater response in the right amygdala to feedback in risky younger males, this could reflect greater anticipation of a negative outcome (as is proposed by Ernst et al., 2005) or even a greater negative association with A feedback in risky younger males, which may be part of the underlying reason why they selected from a combination of decks A and C less than safe young males.

As reported in chapter four, risky young male drivers demonstrated a greater preference for infrequent punishment decks (decks B and D) over frequent punishers (decks A and C) to safe. When risky younger males lost money from frequent punishment decks (A and C) they had a greater response than safe in the left IFG. While the IFG in the right hemisphere is most commonly associated with response inhibition (Aron, Robbins, & Poldrack, 2004; Garavan et al., 2006), the

left IFG has also been implicated in this process, as damage to this region has been linked to increased commission errors on a Go/No Go task (Swick, Ashley, & Turken, 2008). However this unlikely to apply in the current context as feedback conditions were passive in the IGT (i.e. did not require a response).

Alternative functions of the IFG have been proposed in the literature, for example it has been linked to orienting attention (H. H. Chao, Luo, Chang, & Li, 2009) and monitoring feedback (Walton, Devlin, & Rushworth, 2004). These functions may be of more relevance for interpreting the current finding as it applies to a feedback condition. As risky younger males demonstrated an aversion to these decks, by choosing decks B and D over A and C significantly more than safe younger males, a more intense response in the left IFG to punishment feedback from A and C may indicate greater monitoring of the frequency of punishments that led the risky young male drivers to select infrequent punishment decks (B and D) more often than young safe males.

Clarke et al. (2010) reported an overrepresentation of older drivers in junction collisions, where the driver disobeyed a traffic light or failed to give right of way when turning at an intersection. Younger drivers, on the other hand, were more likely to lose control on bends or curves due to excessive speed, recklessness or the involvement of alcohol. The distinction between the types of collisions that younger and older individuals tend to be involved in may relate to the behavioural and activational findings reported above, which suggest that risky young male drivers are punishment averse. A sensitivity, and resulting aversion, to immediate punishments, and a discounting of the severity of future ones, may relate to the underrepresentation of younger individuals in junction collisions. Disobeying

signals or right of way at junctions provides immediate negative feedback to drivers, i.e. the immediate danger of breaking a red light is known. Conversely, the punishments associated with speeding, for example, are less frequent, such as being caught by the police or a collision resulting from losing control on a bend. Young risky drivers' preference for infrequent punishments could suggest that they engage in aberrant behaviours such as speeding because the associated negative outcome is not reinforced regularly enough to affect behaviour.

In a final finding from the IGT, risky young males demonstrated a significantly greater response in the left insula when receiving feedback from deck D. Deck D has an infrequent schedule of punishment delivery, in that a large loss will only be made after every ten selections. Deck D is considered to be an advantageous deck, as, despite this loss, the participant will still have made a net profit of $250 over the ten trials. Despite a net gain, wins on a trial-to-trial basis are small in magnitude from deck D, making this a slow process. The insula may be involved in choosing between two competing responses (Bunge, Dudukovic, et al., 2002; Bunge, Hazeltine, Scanlon, Rosen, & Gabrieli, 2002), and making decisions under ambiguity (Krain et al., 2006). It may be that, while risky younger drivers seemed to show a preference for decks with delayed punishments (i.e. decks B and D), risky males demonstrate conflict when getting feedback from deck D, perhaps between the pleasure obtained from a gain but frustration from the small magnitude of the reward, relative to the large gains of deck B. This theory is supported by previous research that implicated the insula, along with the VMPFC, in processing frustration (Abler, Walter, & Erk, 2005).

Increased frustration may also explain why risky young males had greater activation in the right VMPFC when they received hit feedback in the MID (Abler et al., 2005), but not why they demonstrated a greater effect in the right caudate, relative to safe males, when they failed to obtain a monetary reward (failed to hit a win target). Previous research has associated activation in the caudate with the preparation of a motor response to obtain a reward (Schultz, Tremblay, & Hollerman, 1998). This could mean that, while both groups missed as many win targets, the risky young males were closer to making a response than safe males. However, as activation in this region has also been observed in response to monetary gain feedback (van Holst et al., 2010), an alternative explanation for this effect could be that risky younger males were demonstrating aberrant recruitment of reward processing regions compared to safe younger drivers.

Further evidence of the differences in reward processing between risky and safe younger males were observed in the caudate in the MID. Risky younger males had a greater response in the right caudate when they received monetary feedback (win hit feedback) and in the left and right caudate when they received hit feedback overall (a combination of monetary and non-monetary positive feedback). No differences were found when non-monetary positive feedback was examined on its own (neutral hit feedback). Safe younger males demonstrated a greater effect in the left caudate while anticipating a monetary reward, over receiving it, compared to risky younger males (secondary contrast: 'win cue > win hit'). This hyperactivation of the caudate in response to rewarding stimuli and hypoactivation in anticipation of a monetary reward may indicate that these risky

individuals are more sensitive to rewards upon delivery and, therefore, may be more motivated to engage in reward seeking behaviours.

From the results of the current study it appeared that risky and safe younger males may also differ in mechanisms of cognitive control. Risky young males showed a greater response than safe in the left SMA and left DLPFC when they obtained a monetary reward in the MID. As previously mentioned, the SMA is implicated in response selection (Whelan et al., 2012), preparation of actions (Ikeda et al., 1999) and inhibitory processes such as response cancellation (Aron, 2007; Floden & Stuss, 2006; Picton et al., 2007). Many functions have been associated with the DLPFC. It is thought to be responsible for suppressing inappropriate actions (Beeli et al., 2008; Wood & Grafman, 2003), making decisions that do not involve risk (Frith, Friston, Liddle, & Frackowiak, 1991; Hyder et al., 1997) and has also been noted to respond to the receipt of monetary rewards (Knutson et al., 2001; Knutson et al., 2000; Nestor et al., 2010). This last finding is in keeping with that of the current study, however it would appear that this DLPFC recruitment in response to a monetary reward was greater in risky younger males. This may be further evidence of a difference in reward assessment between risky and safe younger males. The implications of these findings are discussed at the end of hypothesis four. Furthermore, the presence of an excess of SMA activation in risky younger males, when no response was necessary (i.e. passive viewing of trial feedback), may reflect inappropriate response selection. The potential for a larger role of the SMA in heightened risk-taking will be discussed in further detail in chapter six.

5.4.1.4 *Hypothesis four: there are activational differences between risky and safe male drivers, independent of age*

Hypothesis four was addressed in two parts. To identify whether neural processes that make some males vulnerable to increased risk-taking are independent of age, all safe males were compared to all risky males, regardless of age.

Independent of age, risky and safe males demonstrated different patterns of activation in response to certain primary and secondary first level contrasts of the Go/No Go task. For successful inhibitions in this task, compared to errors of omission[24], safe males had greater activity in the right SMA and left inferior parietal lobe, implicated in action monitoring and response preparation (SMA; Ikeda et al., 1999; Whelan et al., 2012) and attention during cognitively demanding tasks (inferior parietal lobe; Chong, Williams, Cunnington, & Mattingley, 2008; Kihara et al., 2007; Krueger, Fischer, Heinecke, & Hagendorf, 2007). In contrast, when successful and unsuccessful inhibitions were compared in the Go/No Go task (i.e. 'Successful No Go > Failed No Go', a primary contrast of interest), risky males demonstrated greater activity in the SMA, bilaterally. This is contrary to previous findings by Kaufman et al. (2003) that found reduced SMA activity in cocaine users compared to healthy controls during this condition, suggesting that decreased SMA activation for behavioural inhibitions may be a marker for engagement in risk-taking behaviours such as substance abuse.

However, the Kaufman et al. (2003) study used tasks of varying difficulty between groups (i.e. the substance group attempted an easier version of the task than controls), and still found significantly more errors of commission in cocaine users.

[24] A secondary contrast

These results, therefore, may not be directly comparable to those of the current study, which found no difference in the number of successful inhibitions. As the SMA is implicated in executing appropriate and inhibiting inappropriate behavioural responses during Go/No Go tasks (Simmonds et al., 2008), it may be that, for the current sample, larger SMA activation in risky males during successful inhibitions reflects a greater effort to inhibit inappropriate responses.

Further effects of risk in males were limited to exploratory contrasts within the IGT and MID[25]. For instance, risky males were found to have greater activation in the right pallidum when participants received feedback from deck C and safe males showed a greater response in the left OFC for monetary gains from disadvantageous decks (decks A and B), in the right caudate for feedback from deck B and in the right putamen for feedback from deck C. While the striatum is implicated in the processing of reward, the OFC is thought to encode the expected value of a reward associated with a particular outcome (Hampton & O'Doherty, 2007; Knutson & Cooper, 2005; Knutson & Wimmer, 2007; Krawczyk, 2002). This hypoactivation of the reward network in risky males is similar to that observed in younger adolescents when compared to young adults (Bjork et al., 2004). Bjork et al. (2004) interpreted these findings to mean that adolescents engage in risk-taking behaviours to achieve the same level of gratification as adults. However, this may not be a complete explanation of this phenomenon. While the current study found reduced activation when all risky males were combined, an examination of risky younger males on their own showed a greater reward response in some contrasts when compared to safe younger males (see hypothesis

[25] With the exception of one secondary MID contrast, highlighted below

three above). Combined, these findings support the theory that risky males sometimes process rewards inappropriately. Some younger males may engage in risk-taking because of an over sensitivity to rewards but may also do so to compensate for a reduced reward response in certain situations. The latter vulnerability may persist into later adulthood, as this effect appears to be independent of age. Thus, aberrant reward processing may be situation dependent.

Greater activation of the SMA, bilaterally, was observed in risky compared to safe males in anticipation of a target that could bring about a monetary reward in the MID (i.e. win cue periods). Also in the MID, safe males had greater activation in the ACC in response to miss feedback. In contrast, when feedback conditions were subtracted from anticipation periods (e.g. 'win cue > win miss', a secondary contrast), risky males showed a greater activation of this region compared to safe males. These findings are very similar to what was observed in Hypothesis two when younger males and females were combined, suggesting that these features of risk-taking may be independent of both gender and age, i.e. that risky individuals may be more motivated to obtain a monetary reward (SMA in anticipation of a win target) and may demonstrate a deficit in error processing (ACC in response to miss feedback).

The second half of this hypothesis, which questions why some older males continue to take risks while driving, was addressed by examining interactions between age and driving risk in males. This analysis identified three significant interactions between age and risk, two exploratory contrasts in the IGT and one secondary contrast of interest in the Go/No Go task.

In the IGT, a disordinal interaction was found for activity in the ACC during selections from infrequent punishment decks (decks B and D), such that young risky males had significantly greater activation than young safe but the opposite pattern was found in older males. The ACC is said to be involved in action selection and error processing (J. W. Brown & Braver, 2007; Carter & Van Veen, 2007) but heightened activation in this region has also been linked to attentiveness to corrective behaviours (Carter et al., 1998). There was an interaction between age and driving risk for the number of selections made from infrequent punishment decks (decks B and D). Younger risky males showed a significant preference for infrequent punishers over frequent compared to younger safe males, but there was no difference between older risky and safe males. In light of this, the current finding may suggest that, driven by their preference for these decks, risky younger males attended to these stimuli more than safe younger males. This does not explain, however, why older safe males would have attended to these decks more than risky when there was no difference in the amount of infrequent deck selections made between these groups.

When selecting advantageous decks in the IGT, older safe males had a greater response in the PCC than younger safe males, however no difference was found between older and younger risky males. Previous research has suggested that the PCC conducts assessments of risk (Damasio, 1999; Damasio et al., 2000). Greater activation in this region in older safe, relative to younger safe, males could reflect the maturation of this region. Delayed development of the PCC, as evidenced by a lack of difference between older and younger risky males where a difference

exists for safe males, could be a vulnerability marker for risky decision-making in older adult males.

Finally, young risky males showed reduced activity in the DLPFC when successfully inhibiting their responses to lure stimuli (over errors of omission[26]), compared to younger safe male drivers. No such difference was found between older risky and safe male drivers. The DLPFC is thought have many functions, including response selection and interference control (Bunge, Dudukovic, et al., 2002; Rubia et al., 2001) and self-regulation of behaviours (Figner et al., 2010; Knoch & Fehr, 2007). Neither successful inhibitions, nor errors of omission, involve a motor response in the Go/No Go task. One requires participants to consciously inhibit a response to a no go stimulus and the other occurs when a participant fails to respond to a go stimulus. With the above proposed functions of the DLPFC taken into consideration, this finding suggests that safe younger males exerted greater control over the interference between the impulse to respond and behavioural inhibition on successful inhibition trials, when compared to errors of omission. Greater capacity for control over interfering thoughts and actions may make these young males more resilient towards driving risk. As this difference was not found between older groups it is proffered that this vulnerability may decline with age.

Therefore, while it appears that Casey et al. (2008)'s neurobiological theory itself may not explain why younger individuals are overrepresented in collision statistics, there is evidence to suggest that some younger individuals are vulnerable to engagement in risk-taking due to functional differences in brain

[26] A secondary contrast of interest

regions that are implicated in this theory. However, as the majority of these findings were based on exploratory analyses, the current findings only provide preliminary evidence to support this theory and should be interpreted with caution.

Despite this, the current observations may relate to previous reports that a slower decline in trait impulsivity from adolescence to adulthood in some individuals was a risk factor for later problematic behaviours, such as abuse of alcohol or drugs (Littlefield et al., 2010; Panwar et al., 2014; Quinn & Harden, 2013; Soloff et al., 2010; Vitaro et al., 1998). Some differences in activation were found to be independent of gender (see hypothesis two) and some were not (i.e. observed in younger males only, see hypothesis three). This may relate to the observation that, while younger individuals are overrepresented in collision statistics, younger males are even further overrepresented. Similarly, there were effects that were and were not independent of age (i.e. some instances of interactions between age and risk status, discussed under the current hypothesis). This is tentatively proffered as an explanation for why males continue to be overrepresented in collision statistics, despite a general decline with age. It may be that factors that make younger individuals more vulnerable to engaging in risky driving persist into later adulthood in some males. Attempts to replicate these findings should be made before further conclusions as to the generalizability of these findings can be drawn.

5.4.1.5 Hypothesis five: there are gender differences in activation

Due to their different representations in fatal collision statistics, it was expected that young males and females would demonstrate differences in activation in

response to tasks of impulsiveness. When all females were compared to all males only one instance of a significant difference in activation was found, thus this study does little to clarify whether there are gender differences in the neural correlates of impulsivity. This effect was observed in the right postcentral gyrus, where females demonstrated greater activation then males for selections from disadvantageous (A and B) over advantageous (C and D) decks during the phase of decision-making under risk in the IGT (final two blocks of the task).

Furthermore, in some conditions of the IGT interactions between gender and risk status were found, suggesting that some neural mechanisms associated with increased risk-taking vary between males and females. For example, when younger individuals won money from disadvantageous decks (decks A and B) an interaction was observed in the amygdala, such that young risky males demonstrated greater activation in this region than young safe males, while there was no difference between risky and safe females. As the amygdala is implicated in processing the emotional saliency and arousal of stimuli (Anticevic et al., 2012; Ernst et al., 2005), it could be said that risky young males had a greater arousal response to the large gains associated with these decks. This could indicate that younger risky males place a greater emotional salience on large, yet risky, rewards compared to safe males. This implicates the amygdala as a region that may contribute to increased risk-taking in males during this period of development.

A disordinal interaction was also observed in the insula for gains from infrequent punishment decks (decks B and D) in the IGT. During this condition, young risky male drivers showed greater activation in the insula than young safe males, while younger safe females had a significantly greater response than young risky

females. The insula has previously been implicated in the processing of the costs associated with making a risky decision (Burke et al., 2013). This could mean that risky and safe males and females encoded the risks associated with gains and losses from these decks differently. A link between insular activity and the processing of frustration has also been demonstrated in previous studies (e.g. Abler et al., 2005). An alternative explanation for the current effect is that these young individuals were experiencing varying levels of frustration when winning from these decks. Regardless of the precise explanation for the effect, this finding suggests that the development of the insula in young individuals may play a particular role in risky decision-making while driving.

Overall, the results of the current study do not support the hypothesis that greater neurological impulsiveness in younger males relates to their overrepresentation in collision statistics, compared to younger females.

5.4.1.6 Hypothesis six: there are correlations between activation and other measures of impulsivity

The direct link between psychometric, behavioural and 'neural' impulsiveness was investigated using a correlational analysis in the current study. It was hypothesised that measures of impulsiveness would positively correlate with activation in reward regions and negatively will 'control' regions during some conditions of the tasks. These relationships were investigated separately in all males, all younger participants and in younger males on their own.

In younger individuals, activation in the right caudate during certain conditions of the MID correlated with performance measures from the MID, such as number of

hits and reaction times. As these findings are very similar, only the following example will be discussed. It was observed that the number of times participants hit neutral targets was positively correlated with right caudate activity during neutral hit feedback conditions. This implies that individuals who are more sensitive to reward are better at the task, perhaps because they are more motivated to obtain said rewards.

Interestingly, a positive correlation was found between the self-control subscale of the BIS-11 and activation in the SMA and right caudate in younger male drivers. This subscale correlated with SMA activity in response to positive feedback (i.e. all hit feedback) and monetary feedback (i.e. win hits) and the right caudate when young males failed to obtain a monetary reward in the MID. Younger males who demonstrated lower levels of 'self-control' (as indicated by higher scores on this subscale) had a greater response in this reward processing region during non-rewarding feedback, possibly indicative of an inappropriate estimation of the value of such feedback.

Furthermore, low 'self-control' in younger males corresponded with greater SMA involvement in positive feedback conditions of the MID. As the SMA is implicated in action monitoring and response preparation (Ikeda et al., 1999; Whelan et al., 2012), this relationship could indicate that impulsive males were already preparing for the next target or, perhaps, that they were undergoing some mental effort to inhibit an inappropriate action in response to positive feedback from the MID. Interestingly, when all males were combined (younger and older), a positive relationship between the self-control subscale of the BIS-11 and activation in the SMA was found when making successful inhibitions over errors of commission in

the Go/No Go task. This could mean that males lacking in 'self-control' required a greater effort to inhibit their responses to lure stimuli in the task.

These findings suggest that, while differences in the neural correlates of impulsivity were not observed between older and younger males in the current study, there does appear to be a relationship between activation in such regions and levels of impulsiveness in both younger and older males.

5.4.1.7 Hypothesis seven: there are correlations between activation and other measures of aberrant driving

The link between self-reported risky driving behaviours, as measured with the Manchester DBQ, and brain activity during cognitive tasks of impulsivity was examined as an alternative to categories based on driving history. It was thought that, if impulsivity were a factor in risk-taking while driving, there would be a positive relationship between the number of risky behaviours reported and levels of activational impulsivity demonstrated in response to the stimuli of the three tasks.

In males a positive correlation was found between activation in the SMA during both cue periods (win and neutral) and the HC violations subscale of the DBQ. This was replicated with the aggressive violations subscale of the DBQ for win cue periods only. This suggests that drivers who take more deliberate risks, as opposed to errors or lapses in attention, recruit the SMA to a greater extent while preparing to make a behaviour response. With aggressive violations this relationship was only observed when preparing to make a response to obtain a monetary reward, which suggests that deliberately risky and aggressive drivers

are more motivated by monetary rewards than by neutral rewarding stimuli. As no differences in performance were observed during this task these findings may indicate that some males have a larger neural load when preparing actions. If this is the case then failures to compensate for this deficit may explain increased risk-taking behaviours in such an attentionaly demanding task as driving.

These findings were similar to what was observed in younger drivers. When all younger drivers were combined, positive correlations between activation in the SMA during cue periods for win stimuli, minus monetary feedback (i.e. the 'win cue > win hit' condition of the MID), were found with the HC and aggressive violation subscales of the DBQ. The interpretation of these findings is similar to the above, that young individuals who exhibit more deliberate and aggressive risky behaviours while driving require greater recruitment of the SMA when preparing to make a motor response. A novel and exploratory finding for the SMA in this condition was that it also positively correlated with the number of penalty points that young individuals had accrued, giving further support for the role of the SMA in risk-taking behaviours. Finally, activation in the SMA for win hit feedback negatively correlated with the aggressive violations subscale of the DBQ. This suggests a more dramatic decline of SMA activity when a monetary reward was obtained in individuals who demonstrated greater aggression while driving. The SMA may, therefore, have particular relevance to a propensity towards engagement in aberrant driving behaviours. This theory will be revisited in chapter six.

5.4.2 Limitations and future directions

Despite the magnitude of research to support their theory, Casey et al. (2008) observed that the majority of studies have examined the development of reward processing regions and the prefrontal cortex separately, rather than looking at their development in conjunction with one another and the development of the connections between them. Functional connectivity analyses are used as a means of determining which regions work in conjunction with one another to perform certain tasks. Such analyses have the potential to establish the extent to which reward processing regions are regulated by regions of the prefrontal cortex, how connections between these regions develop throughout adolescence and how they may relate to impulsive behaviours (Bjork, Lynne-Landsman, Sirocco, & Boyce, 2012). Future neurodevelopmental studies should utilise a functional connectivity approach to fMRI to investigate whether developing connections between these prefrontal regions and reward processing networks coincide with decreasing impulsivity from adolescence to adulthood.

As previously mentioned, the age range of the adolescent and young adult sample in the current study may not have been appropriate for investigating Casey and colleagues' (2008) theory of neurological development in adolescents. Differences in how adolescence is defined make direct comparisons of results across studies difficult (Galvan, 2010; Galván, Van Leijenhorst, & McGlennen, 2012). Adolescence is generally defined as the 'the gradual period of transition from childhood to adulthood' (Spear, 2000, pg. 417), however this definition is problematically vague. It appears that the majority of studies in this area have used samples within the approximate range of 12 to 17 years of age for adolescent samples and,

quite often, define 'adult' as anyone over the age of 18 (Galvan, 2010). Despite this relatively young categorisation of adulthood, a major component of Casey and colleagues' (2008) theory is that the PFC continues to develop into the mid 20s. This suggests that neuroimaging studies should continue to observe differences in reward and control processes between young adults up to the mid 20s and older adults. However, this was not the case in the current study. It may be that effect sizes for age differences in activation between older adolescent/younger adults and older adults are small, therefore, while the sample size of the overall study may not be considered a limitation, a much larger sample could be needed to detect differences between these groups. Future studies of the development of the neural correlates of impulsivity from adolescence to adulthood could utilise a much larger, cross-sectional sample to test this theory. Ideally, however, the development of these systems would be examined longitudinally (Bjork, Lynne-Landsman, et al., 2012) and linked to objective measures of risk-taking.

Furthermore, to address whether Casey and colleagues' (2008) theory applies to increased driving risk in adolescence, the current study could be replicated in a country that has a lower minimum driving age than Ireland. In Ireland, a full driving licence, which permits the individual to drive unaccompanied, cannot be obtained until 17.5 years of age. This limited the minimum age of the current study to 18. The methodologies utilised in the present study could be replicated in

the United States, for example, where the legal driving age is as young as 14 in some States[27].

It is important to note that, while the current study involved many tasks, groups, contrasts and comparisons, every effort was made to adequately account for this with corrections for multiple comparisons, both at a whole-brain level and when using SVCs. Because of these stringent corrections, some significant effects had to be excluded from this report. The results of this study may serve as a guide for future, more focused analyses, to restrict the number of comparisons and, therefore, retain enough power to detect significant effects.

Furthermore, power to sample the BOLD response may have been low in some participants, due to individual differences in the frequency of each trial type. A more accurate estimation of the hemodynamic response can be made when averaging from a greater number of trials. To promote similar levels of engagement in the tasks across participants, true incentives, such as performance-based rewards, could have been used. For instance, some participants may have had hit more win stimuli on the MID and demonstrated a different activational response to feedback with the knowledge that a hit on a win trial would result in a real monetary gain. Future research should consider utilising contingency based motivation, in addition to a more restricted number of contrasts at first level, to increase power to detect functional differences.

[27] Learning permits can be obtained from the age of 14 from the following States: Alaska, Arkansas, Idaho, Iowa, Kansas, Michigan, Montana, North Dakota, South Dakota and Wyoming (VDriveUSA, 2013)

Finally, while careful consideration was taken when categorizing drivers as either 'risky' or 'safe', some drivers may have been falsely categorized. With the current criteria it is possible that some of individuals in the safe group were risk takers but, at the time of testing, did not have any penalty points and had been culpable in a collision. Again, a longitudinal design could be utilized in the future to observe risk-taking across multiple time points throughout adolescence, young adulthood and into later adulthood.

5.4.3 Conclusion

Collectively, the above evidence from the exploratory analysis demonstrates that neural processes implicated in impulsive behaviours, such as attention, the processing of reward, risky decision-making and the preparation and cancellation of actions differ between some individuals. These findings indicate that there may be age-related as well as age-independent neural correlates of impulsivity that relate to risk-taking tendencies while driving that may be relevant for the development of preventative initiatives aimed at novice and more experienced drivers.

This provides some support for the role of impulsivity as an individual difference factor in risky driving. It is unclear, however, as to whether these differences make some drivers more vulnerable to increased risk-taking, or whether prolonged engagement in reward seeking and risk-taking behaviours elicits changes in the mechanisms by which some individuals make decisions. The limitation of such cross-sectional research may be addressed with future longitudinal studies of brain development and risk-taking propensities. The lack of significant differences between risk groups in the primary contrasts makes generalising these findings to

situations were impulsivity may contribute to problematic driving difficult. Therefore, the findings of the exploratory analysis should be considered to be preliminary and interpreted with caution.

6 Chapter Six: A Structural Neuroimaging Study of Drivers

6.1 Introduction

Suboptimal choices and high mortality rates during adolescence distinguish them form any other group (Casey, Jones, & Somerville, 2011). Increased risk-taking is attributed to heightened levels of impulsivity, sensation seeking and reward seeking behaviours in adolescents (Gardner & Steinberg, 2005; Scott, 1992; Steinberg, 2008). Understanding how the brain develops in adolescence, through both progressive (myelination) and regressive (synaptic pruning) processes, has provided potential explanations for this vulnerability (Silk & Wood, 2011). Modern imaging techniques, such as magnetic resonance imaging (MRI) and diffusion tensor imaging (DTI) allow for non-invasive examination of brain structure and volume *in vivo* (Kelly et al., 2009).

The research to date suggests that heightened impulsivity and risk-taking in adolescence may be the result of a miss-match between the developmental trajectories of reward processing systems, such as the striatum, and regulatory regions of the prefrontal cortex (PFC) (Casey et al., 2008; Paus, 2005; Somerville et al., 2010; Steinberg, 2008). The current chapter utilised MRI and voxel-based morphometry (VBM) analysis (Ashburner & Friston, 2001) to confirm previous observations of changes in grey and white matter volume with age. In addition, the current chapter served as an exploratory analysis of whether such volumes have any relation to impulsiveness, gender and risk-taking while driving.

6.1.1 Brain development

Both cross-sectional and longitudinal studies have used MRI to study the structural development of the brain. While studies have suggested that the brain reaches 95% (Giedd et al., 2006) or even 100% (Su, Kuan, Kaga, Sano, & Mima, 2008) of its total volume by the age of six, it continues to undergo dramatic changes throughout the lifespan (Giedd et al., 2006; Sowell, Thompson, Tessner, & Toga, 2001). Changes in brain structure and volume are examined in relation to three tissue types, cerebrospinal fluid (CSF), grey matter (GM) and white matter (WM). There is agreement across studies that overall CSF increases in volume with age (Coffey et al., 1998; G. Cohen et al., 1992; Good et al., 2001; D. L. Greenberg et al., 2008; Jernigan et al., 2001; Scahill et al., 2003; Sowell, Thompson, et al., 2001; Sowell et al., 2002; Walhovd et al., 2005; Walhovd et al., 2011), however, grey and white matter follow more complicated developmental trajectories. These are discussed in detail below.

6.1.1.1 Grey matter (GM) development with age

It has been observed that overall GM development follows an inverted U-shaped pattern (Giedd, 2004; Giedd et al., 2006; Gogtay et al., 2004; Sowell et al., 2003; Sowell et al., 2004). Although variability exists between the genders and between regions, generally GM has been found to peak in late childhood (Shaw et al., 2008; Silk & Wood, 2011) or early adolescence (Giedd et al., 1999) and, subsequently, show a protracted decline into late adulthood (Sowell et al., 2002). This decline has been found to occur most rapidly within the first five decades of life, reflecting maturational changes that improve cognitive functioning, such as increased myelination, rather than degeneration (Sowell et al., 2003; Sowell et al., 1999).

As previously stated, GM changes in brain regions occur at different rates. GM peaks first in sensory areas at approximately seven years of age (Huttenlocher & Dabholkar, 1997; Shaw et al., 2008). In a longitudinal study, Giedd et al. (1999) found that GM peaks in the parietal lobe at age 11, followed by the frontal lobe at age 12 and the temporal lobe at age 16. This latter finding is contradicted by Sowell et al. (2003), however, who found that GM in posterior temporal regions continued to increase until at least age 30 (the maximum age in their sample). These disparate findings may be explained by the different age ranges and methodologies (i.e. cross-sectional versus longitudinal) used by the two studies.

After this peak in GM it begins to decline, reflecting changes in the size and complexity of neurons (Giedd, 2004). GM decline occurs through processes such as axonal growth, increase in pyramidal cell somata, dendritic arborisation, synaptogenesis and synapse elimination (pruning). These processes are considered to be beneficial as they reflect the maturation and integration of structures (Huttenlocher, 1979; Huttenlocher & Dabholkar, 1997) and they coincide with the improvement of certain cognitive and behavioural capabilities (Sowell et al., 2003), such as higher-order executive functions (Casey, Giedd, & Thomas, 2000; Casey et al., 1997; Luna et al., 2001; Tamm, Menon, & Reiss, 2002), memory (Sowell, Delis, et al., 2001), and verbal intelligence (Sowell et al., 2004). Overall, brain regions have been observed to mature from inferior to superior and posterior to anterior in direction (Huttenlocher, 1979), i.e. starting with the primary and sensory areas and occurring latest in prefrontal regions and aspects of the parietal and temporal cortices (Gogtay et al., 2004; Sowell et al., 2003; Sowell et al., 1999; Sowell, Thompson, et al., 2001).

Tamnes et al. (2010) found that by the age of 30, GM has decreased non-linearly in the majority of regions, with the greatest change occurring in adolescence. This rapid decline in adolescence is followed by slower decline in adulthood (Shaw et al., 2008; Sowell et al., 2003). In a study of children and adolescents between seven and 16, Sowell et al. (2002) found that the most significant decreases in GM volume during this age were in the frontal lobe and mesial temporal lobes. Significant decline was also detected in the cingulate, but at a slower rate. A subsequent study by Sowell et al. (2003) found that the anterior cingulate cortex (ACC), specifically, in children was approximately 12% larger than older adults.

While Sowell et al. (2002) did not find significant GM decline in the parietal lobe during this age, other studies with larger age ranges have suggested that the parietal cortex actually demonstrates the most prominent GM decline during adolescence, followed closely by the frontal cortex (Sowell et al., 2004; Sowell, Thompson, et al., 2001). While the frontal cortex as a whole shows decline during adolescence, this process appears to commence in regions of the PFC at a much later stage when compared to other regions (Sowell, Thompson, et al., 2001). The dorsolateral prefrontal cortex (DLPFC), for example, has been show to be the last region to demonstrate GM decline, with this process not beginning until the end of adolescence (Gogtay et al., 2004).

There are two types of GM, cortical (outer surface of the brain) and subcortical (interior GM; e.g. the basal ganglia, amygdala and hippocampus). GM decline in cortical regions, such as those discussed above, has been found to occur more rapidly than in subcortical regions (Jernigan et al., 2001; Walhovd et al., 2005). GM decline in subcortical regions has been reported extensively in the literature,

however there is little consensus amongst these findings. A meta-analysis conducted by Walhovd et al. (2011) found that the literature is divided as to whether GM volume in the amygdala, thalamus and putamen, for example, decline with age. Walhovd et al. (2011) found that, out of fifteen studies surveyed, nine showed total hippocampal volume declined with age and five found no difference. Interestingly, one study found that hippocampus decreased in size in adult males only (Pruessner, Collins, Pruessner, & Evans, 2001), a finding that has also been replicated in the putamen (Nunnemann et al., 2009). Contrary to this finding, and not mentioned in Walhovd's review (2011), Giedd, Vaituzis, et al. (1996) found that the hippocampus decreased in volume in females only during adolescence. Of the regions showing concordance across studies, the pallidum has shown no significant decline with age, whilst the caudate consistently has (Giedd et al., 2006; Raz et al., 2005). While only two studies examined the effects of age on GM volume in the nucleus accumbens (NAcc), both found a significant decline with age (Walhovd et al., 2011).

6.1.1.2 White matter (WM) development with age

While GM volume both increases and decreases throughout the development of the brain, WM development follows a different pattern. WM increases in a roughly linear fashion with some variation by brain region (Giedd, 2004). There are three main types of WM fibres, those that connect the cortex with deep brain structures (projection fibres), those that connect regions within a hemisphere (association fibres), and those that are involved in inter-hemispheric communication (commissural fibres) (Wozniak & Lim, 2006). WM in an MRI scan indicates myelinated axons (Whelan et al., 2012). WM increases through the process of

myelination, where neuronal axons become wrapped in a protective, fatty sheath (Su et al., 2008). This increases the efficiency of neural communication by speeding up transmission between neurons, with transmission occurring approximately 100 times faster in myelinated neurons than unmyelinated neurons (Su et al., 2008). The velocity of this conduction is influenced by subtle factors such as axon diameter, myelin sheath thickness, internode length and the serial arrangement of internodes along the axon (Wang & Young, 2014).

Changes in white and grey matter volume have been found to relate to emotional, behavioural and cognitive performance. The pruning of unessential connections in local networks (as characterised by declining GM) and the strengthening of connections between regions (increasing WM) with age demonstrate the formation of more complex neural networks (Loos et al., 2009). Greater neural sophistication with age allows cognitive, behavioural, and emotional development to take place in an optimal fashion (Barnea-Goraly et al., 2005). Significant improvement of higher-order executive functions has been observed to coincide with these developments in adolescence (Casey et al., 2000; Casey et al., 1997; Liston et al., 2006; Luna et al., 2001; Sowell, Delis, et al., 2001; Tamm et al., 2002). For example, in a longitudinal study using DTI, Bava et al. (2010) found that increased WM correlated with better performance on complex attention, working memory and verbal fluency tasks.

While some evidence suggests that global WM increases most rapidly until the age of ten before a slower, linear increase into adulthood (Pfefferbaum et al., 1994), ground-breaking research by Giedd et al. (1999) that combined cross-sectional and longitudinal data found a steady, linear increase in WM volume from age four

to 22. The net increase in WM volume during this period was 12.4% and was reasonably even across the four lobes (Giedd et al., 1999). Subsequent cross-sectional research has corroborated and extended these findings to suggest that this pattern of WM growth increases into the late 30s (Grydeland, Walhovd, Tamnes, Westlye, & Fjell, 2013; Paus et al., 1999; Silk & Wood, 2011; Tamnes et al., 2010). These findings are, in turn, supported by post-mortem studies (Bartzokis et al., 2001; Benes, Turtle, Khan, & Farol, 1994; Yakovlev & Lecours, 1967). When linear and quadratic models have been applied to these data and directly compared, linear models have been found to be a better fit for the pattern of WM growth observed (Shafee, Buckner, & Fischl, 2015).

Despite a global increase in WM with age, and some evidence to suggest even rates of development across regions (Giedd et al., 1999), some studies have found moderate differences between regions. Age related regional changes in WM have been studied using both MRI and DTI approaches. Analyses of MRI data with VBM typically focus on global and regional WM volumes identified by a voxel-by-voxel analysis of signal intensity (Ashburner & Friston, 2001). Through the use of tissue probability maps (TPMs), each voxel is assigned the probability that it represents WM, GM or CSF tissue types. DTI, however, measures the diffusion of water within axons (Barnea-Goraly et al., 2005). Fractional anisotropy (FA) is an index of WM microstructure obtained from the analysis of DTI. Voxels are assigned an FA between zero (isotropic diffusion) and one (maximum anisotropic diffusion), where zero represents equal diffusion in all directions and one reflects the hypothetical instance where there is diffusion through a voxel only along one axis. Higher FA values, therefore, represent water moving along the principal diffusion

direction, rather than the transverse direction (Ladouceur, Peper, Crone, & Dahl, 2012). FA is sensitive to the diameter, density and myelination of WM fibres that connect brain areas and larger FA (Paus, 2005, 2010). FA values and WM volumes produced by VBM analyses have been found to be moderate to weakly related as they are both sensitive to different characteristics of WM (Fjell et al., 2008).

Using a voxel-by-voxel approach, Paus et al. (1999) found early evidence to suggest that not all regions show increase in WM during adolescence. Regions that did show increase were the internal capsule and arcuate fasciculus, corresponding to the speech regions, Wernicke's and Broca's area, respectively (Silk & Wood, 2011). A cross-sectional, DTI study by Barnea-Goraly et al. (2005) of WM development between the ages of six and 19 revealed increased FA in the internal capsule, in concordance with the findings of Paus et al. (1999). In addition, they observed increased FA in prefrontal regions and in pathways within the basal ganglia and between the basal ganglia and the thalamus during this age (Barnea-Goraly et al., 2005).

Giorgio et al. (2010) conducted a concurrent VBM and DTI study that followed adolescents longitudinally through to early adulthood. The overall age range spanned 13.5 to 22.1 years between the two timepoints. Using VBM they found extensive WM increases with age in aspects of the corpus callosum (CC) and extending symmetrically from retrosplenial WM to the WM underlying the medial parietal cortex and inferior to the temporal lobe portion of the cingulum. In addition, volume increase extended to WM in the left occipital cortex and the left inferior longitudinal fasciculus (ILF). FA analyses of the DTI data identified further WM increase in the bilateral arcuate fasciculus but failed to show an increase in

the internal capsule, as has been found in other studies (Barnea-Goraly et al., 2005; Paus et al., 1999). Giorgio et al. (2010) posited that this lack of finding was due to the different methodologies used, i.e. cross-sectional versus longitudinal, and their focus on the later stages of adolescence.

While studies have postulated that WM peaks in the 30s (Barnea-Goraly et al., 2005), 40s or even 50s (Benes, 1989; Benes et al., 1994; Grydeland et al., 2013), one finding that is common to all studies is that there is no *decrease* in WM in healthy adolescents and young adults (Giorgio et al., 2010; Liston et al., 2006). In fact, WM does not appear to decline in volume until the forth or fifth decade of life, at the earliest (Benes, 1989; Benes et al., 1994; Grydeland et al., 2013; Jernigan et al., 2001; Walhovd et al., 2005).

6.1.1.3 The relationship between grey and white matter development

It is important to determine whether the changes in grey and white matter volume with age reflect maturation or degenerative processes (Jernigan et al., 2001). It appears from the research that the various tissue types undergo different maturational processes simultaneously (Sowell, Thompson, et al., 2001), however these may not be entirely mutually exclusive. A certain amount of GM decline and increasing WM is attributed to the conversion of GM to WM through the process of myelination (Tamnes et al., 2010). Due to this simultaneous WM increase and GM decrease, Sowell and colleagues have posited that the changes that occur between adolescence and the late 40s are likely to reflect normal maturational processes (Sowell et al., 2004). Nonetheless, GM decline with age cannot be solely attributed to WM increase, as GM volume continues to decrease after WM volume has peaked (Sowell et al., 1999). Thus, Sowell et al. (2003) have suggested that any GM decline

that occurs after this peak in WM (i.e. in the late 40s to early 50s) likely reflects degeneration.

6.1.2 Gender differences in brain development

Many of the above studies have also examined whether brain development varies between males and females. Total cerebral volume peaks at an average of 10.5 to 11.5 years in females and 14.5 years in males (Giedd et al., 2006; Lenroot et al., 2007). GM peaks in the parietal lobe at age 10.2 on average for females and 11.8 in males, age 11 for females and 12.1 for males in the frontal cortex and in the temporal cortex at 16.7 years for females and 16.5 years for males (Giedd, Snell, et al., 1996). Furthermore, across studies it has been found that overall cortical complexity is greater in females, especially in frontal and parietal regions (see Cabeza & Nyberg, 2000 for a review). Owing to the different rates of development in males and females, global dimorphism of the human brain varies with age but, on average, male brains are approximately 9% larger than females, even when accounting for BMI (Giedd, Snell, et al., 1996).

Possibly due to the earlier peak in GM in females, it has been found that significant myelination in females also occurs at a younger age (Blanton et al., 2004; Leussis & Andersen, 2008; Paus, 2010). Despite this earlier onset of myelination, the general pattern of GM decline and WM increase occurs at a slower rate in females compared to males (De Bellis et al., 2001; Giedd, 2004; Giedd et al., 1999; Lenroot et al., 2007). While research has investigated gender differences in both the rate of WM development and global WM volumes, few studies have examined regional WM differences between the genders. One of the few regions to be examined is the CC. The research is divided about the subject, but some studies have found the CC

to be larger in females, while other studies have found no effect of gender in this region (for review see Ladouceur et al., 2012).

In a large, longitudinal study of brain development from the age of three to 27, Lenroot et al. (2007) found that, while GM followed an inverted U shaped trajectory in both genders, regional GM volumes peaked between one and two years earlier in females, compared to males. Research into the sexual dimorphism of subcortical regions has found the following regions to be proportionately larger in females; the caudate (Giedd et al., 1997; Paus et al., 1996), thalamus (Paus et al., 1996) and the DLPFC (Goldstein et al., 2001). In adult males the amygdala (Highley et al., 1999; Marcil, Bergeron, & Audet, 2001), pallidum (Giedd et al., 1997) and paracingulate gyrus (Swaab & Fliers, 1985) have been found to be larger than females when total brain volume is taken into account. Furthermore, significant increase in amygdala volume during adolescence has been observed in males only, and increases in the hippocampus only in females (Giedd et al., 1997; Giedd et al., 2006; Giedd, Snell, et al., 1996; Giedd, Vaituzis, et al., 1996). This pattern appears to be reversed in later adulthood, with research demonstrating a decrease in hippocampal volume in males only (Pruessner et al., 2001).

6.1.3 Brain development, impulsivity and risk-taking during adolescence

As previously mentioned, while primary systems, such as motor and sensory, develop early in adolescence (Gogtay et al., 2004; Sowell et al., 2004), both neuroimaging (Galvan et al., 2007; Giedd et al., 2006; Gogtay et al., 2004; Sowell et al., 2003) and post-mortem studies (Huttenlocher, 1979) have observed that PFC development, as measured by GM decline, does not occur until much later. The maturation of the PFC in later adolescence and early adulthood is associated with

a linear increase in executive functioning capacities and is implicated in the decrease in impulsive tendencies observed (Casey, Thomas, et al., 2002; Galvan et al., 2007). The DLPFC, for example, which is thought to regulate behaviour (Mesulam et al., 2001; van Holst et al., 2012; Whelan et al., 2012) and control 'impulses' (Wood & Grafman, 2003) is one of the last regions to show GM decline in adolescence (Gogtay et al., 2004).

As discussed in earlier chapters, while on-going maturation of the PFC may explain declining impulsive action from childhood to adulthood, this does not account for the increase in risk-taking propensities observed in mid adolescence. If prefrontal development alone was linked to risk-taking, then children would exhibit the same level of risk-taking, if not more, than adolescents, due to their PFC cortex being relatively more immature (Casey et al., 2011). Research has combined the study of the development of cortical and subcortical regions, such as the striatum, in an effort to better understand this phenomenon. The prevailing theory in the literature is that increased impulsive choice and risk-taking in adolescence occurs as the result of the imbalance between the early maturation of the limbic system relative to the protracted development of the PFC (Casey et al., 2008; Casey et al., 2011; R. A. Chambers et al., 2003; J. R. Cohen et al., 2010; Steinberg, 2008). This theory is illustrated in figure 5.1 in of chapter five.

Adolescent responses to reward have been documented extensively in the functional neuroimaging literature (e.g. Galvan, 2010; Galvan et al., 2007), however accounts of the structural development of these systems in adolescence are varied. While Casey and colleagues' (2008) neurobiological theory postulates that the limbic system is mature in early adolescence, relative to the PFC,

neuroimaging evidence suggests that components of this system, such as the basal ganglia, are undergoing developmental changes throughout adolescence. Measures of subcortical regional GM volume have found that aspects of the basal ganglia (e.g. the pallidum, caudate and putamen) are larger in younger adolescents relative to adults (Jernigan et al., 2001) and show a significant decline in volume with age into adulthood (Sowell et al., 1999). Furthermore, Using DTI, Liston et al. (2006) found that the strength of frontostriatal fibre tracts (WM tracts connecting the striatum to PFC regions) increased with age.

Neurochemical studies have also investigated the quantity and expression of dopaminergic receptors in the striatum and PFC in adolescence (see the following for detailed reviews; Bava & Tapert, 2010; R. A. Chambers et al., 2003; Spear, 2000). Because of the invasive nature of this research it is mostly conducted in rodents and non-human primates. Evidence from these studies suggests that dopamine (D_1) receptors are over produced in adolescence. There are more D_1 receptors in the NAcc, caudate and putamen in adolescents compared to younger and older ages and approximately one third of these receptors are pruned by adulthood. The density of dopaminergic connections between the striatum and the PFC has also been found to increase in adolescence. With the exception of one study that did not find D_1 receptor pruning in the NAcc by adulthood (Teicher, Andersen, & Hostetter Jr, 1995), these findings have been consistent across studies of rats (Brenhouse, Sonntag, & Andersen, 2008; Cunningham, Bhattacharyya, & Benes, 2002; Kalsbeek, Voorn, Buys, Pool, & Uylings, 1988; Kelley et al., 2004; Tarazi & Baldessarini, 2000; Tarazi, Tomasini, & Baldessarini, 1998), non-human primates (Lambe, Krimer, & Goldman-Rakic, 2000; Rosenberg

& Lewis, 1994, 1995) and a post-mortem study in humans (Tunbridge et al., 2007).

Combined, findings from structural and neurochemical studies suggest that reward processing regions are still undergoing maturational changes in adolescence, contrary to what is suggested by Casey and colleagues' (2008) theory of risk-taking during this age. Instead, increased reward seeking behaviours may be the result of an excess in dopamine receptors in the striatum, relative to the PFC in adolescence (Steinberg, 2008). However, as it has also been observed that connections between prefrontal regions and the striatum strengthen between adolescence and adulthood, the aspect of Casey and colleagues' (2008) theory regarding the PFC's inability to exert sufficient top-down control over reward seeking behaviours in adolescence may still hold. Exerting top-down control over reward seeking may be made even more difficult during adolescence due to an abundance of dopamine receptors (Spear, 2000; Steinberg, 2008). Nevertheless, due to the difficulty in conducting neurochemical studies in human subjects to validate these findings, the generalisation of such findings in rats and monkeys should be done with caution. The current study will re-examine this hypothesis in males by identifying whether striatal regions, in addition to prefrontal regions, show GM decline with age using VBM analysis.

6.1.3.1 GM volume and psychometric measures of impulsivity and risk-taking

As some adolescents are more prone to risk-taking than others, the inclusion of measures of individual differences, such as impulsiveness, in this research may be important (Casey et al., 2011). Using data from one timepoint in the IMAGEN project (age 14), a large longitudinal study of brain development throughout

adolescence, Schilling et al. (2013) found that impulsiveness in the TCI-R (Revised Temperament and Character Inventory) inversely related to GM volume in the orbitofrontal cortex (OFC) at this age, which, in turn, was positively correlated with performance on a perceptual reasoning task. This is the first evidence of structural correlates of both cognitive functioning and impulsiveness in healthy adolescent subjects. These findings, however, were not replicated by a later, smaller study in older adolescent males (ages 18 and 19). Using questionnaires, Kwon, Vorobyev, Moe, Parkkola, and Hämäläinen (2014) generated two groups of young males, one with high and one with low impulsivity[28] and susceptibility to peer influence[29]. They did not find any differences in regional GM volume between these groups using VBM, nor did they find a relationship between volume and the number of risky decisions made in a driving task.

Psychometric measures have also been used to identify whether regional GM volumes may be associated with risky driving behaviours in older adults. In a sample of elderly (65 to 76) Japanese drivers, Sakai et al. (2012) compared GM volume to self-reported risk-taking and executive function. Driving risk was measured using a driver behaviour questionnaire (DBQ) and executive function through the Executive Function Capacity scale (EFC), which contains items like, ''it's often hard for me to alternate between two different tasks''. Sakai et al. (2012) found that, independent of gender, those with lower executive function reported more risk-taking on the DBQ and had reduced GM in the SMA (supplementary motor area).

[28] Assessed using the Zuckerman-Kuhlman-Aluja Personality Questionnaire (ZKA-PQ)

[29] Resistance to Peer Influence Scale (RPIS)

To further these findings, the current study investigated the relationship between brain volume, impulsivity and risk-taking. GM density was compared to behavioural and individual difference measures of impulsivity and driving behaviour, in addition to an exploratory analysis of potential differences in regional GM volumes between 'risky' and 'safe' drivers.

6.1.4 Summary and Hypotheses

6.1.4.1 Hypothesis one: the PFC will demonstrate steeper GM decline than the striatum

The current study used VBM to investigate the extent to which Casey and colleagues' (2008) theory applies to the structural development of the brain from late adolescence to later adulthood in males. Changes in grey and white matter volume were measured in males from the age of 18 to 56, with a particular focus on changes to cortical prefrontal regions and subcortical regions like the striatum. Based on Casey and colleagues' (2008) neurobiological theory it was hypothesised that continued development of the PFC would be observed in younger males when compared to older. Furthermore, it was expected that any decline observed in striatal regions would be to a much lesser extent or at a slower rate than in the PFC.

As much of the research suggests that the PFC continues to develop into the mid 20s, it was also anticipated that a decline in GM density in the PFC would be observed in the other two analytical models employed by the current study: younger males (18 to 24) and all younger individuals (males and females within this age range).

6.1.4.2 Hypotheses two, parts A, B and C: there are differences in brain volume between risky and safe drivers

There may be certain structural vulnerability markers to increased risk-taking. Research by Schilling et al. (2013) and Sakai et al. (2012) have identified structural differences between groups, young and old respectively, that could potentially underlie risk-taking behaviours. To build on these findings, the current study conducted an exploratory analysis of regional GM volumes in certain regions of interest (ROI), comparing them between known risk takers and presumed safe drivers. It was hypothesised that the density of some of the regions implicated in reward processing and in exerting executive control over behaviours, such as regions of the PFC and the SMA/pre-SMA, would differ between risky and safe drivers.

The three analytical models previously mentioned were employed in order to investigate whether such vulnerabilities, should they exist, are age or gender dependent. Thus, hypothesis two was addressed in three parts. Part A dealt with whether such differences are independent of gender by comparing all risky and safe younger drivers, a combination of males and females. Part B sought to identify any structural differences that may be unique to younger risky and safe male drivers. Finally, part C combined all younger and older, safe and risky male drivers, in an effort to identify any differences that may be independent of age.

6.1.4.3 Hypothesis three: there are gender differences in brain tissue volumes

The current study aimed to add to the body of literature that have demonstrated differences in tissue volumes between adolescent males and females. As much of

the research into the sexual dimorphism of the brain has demonstrated different rates of development of GM in males and females in early adolescence, the current study, which utilised a much older 'adolescent' sample by comparison, did not expect to detect differences in the rate of development of regions. As studies have shown differences in regional GM volumes between adult males and females (for example that the caudate is larger in females; Giedd et al., 1997), the current study expected to replicate some of these findings.

6.1.4.4 Hypotheses four: there are correlations between GM volume and psychometric and behavioural measures of impulsiveness

The degree to which GM volume in regions associated with increased impulsiveness in adolescence may vary with self reported impulsivity (as measured with Barratt's Impulsiveness Scale, BIS-11 and Kirby's delay discounting questionnaire, DDQ) and behavioural impulsivity (as measured by performance on the Go/No Go task, Monetary Incentive Delay task, MID, and Iowa Gambling Task, IGT) was also investigated. It was hypothesised that negative correlations between regional GM volumes and measurements of impulsivity would be observed, in accordance with previous findings on the relationship between GM volume and psychometric impulsivity (Sakai et al., 2012; Schilling et al., 2013).

6.1.4.5 Hypothesis five: there are correlations between GM volume and self-reported aberrant driving behaviours

GM volume in these regions was also compared to an alternative measure of driving risk, the Manchester DBQ. It was expected that if any regions differed in

GM volume between risky and safe drivers, this volume would be correlated to DBQ scores.

6.2 Method

6.2.1 Design

In this section, voxel-based morphometry (VBM) analysis was used to compare the volumes of grey and white matter in risky and safe drivers, and in young males and females. Participants were divided into the same groups that were used for the analysis of the functional data: young safe/risky males (aged between 18 and 24), young safe/risky females (between 18 and 24) and older safe/risky males (between 29 and 56), and a similar analytical approach was adopted.

In addition to between group comparisons of brain tissue volumes, the relationship between grey matter volume, age and measures of impulsivity were also investigated.

6.2.2 Participants & procedure

From the original 120 participants mentioned in section 3.2, one participant of Asian decent (older safe male driver, age 34) was not included in the VBM analysis due to their potential influence on the DARTEL[30] template. This left 119 participants for analysis. T1-weighted images were obtained from these participants during scanning. For full details of the study's procedure see section 3.4.

[30] Diffeomorphic Anatomic Registration Through Exponentiated Lie algebra algorithm

6.2.3 Apparatus and materials

6.2.3.1 The Edinburgh Handedness Inventory (EHI)

A 12-item, pen and paper version of the Edinburgh Handedness Inventory (EHI; Oldfield, 1971) was included in the standard MRI information and consent booklet given to all participants prior to scanning in Trinity College Dublin. The data from this questionnaire was used in the current section to control for the potential confounding effects of handedness.

Since its development by Oldfield in 1971, the EHI has been the most commonly used measure of handedness in the cognition, behaviour and neuroscience literature (Edlin et al., 2015). The current version of the measure used 12 of the original 20 items, a list of which can be found in appendix AC. Participants indicated either a right or left hand preference for completing each of the actions by entering a cross into the appropriate column. One cross indicated a preference, two crosses that they exclusively use that hand for the action and a cross in both columns indicated no preference or use of both hands.

6.2.3.2 Hardware and software

As mentioned in section 5.2.4.2.1, scanning was conducted on a Philips Achieva 3.0 Tesla MR system with a 32 channel Head coil. T1-weighted anatomic MPRAGE transverse images (180 high-resolution, FOV 230mm, thickness 0.9mm, voxel size 0.9 x 0.9 x 0.9) were acquired for each participant in the same session as the functional scans. These images took 343 seconds to acquire.

All preprocessing and analyses were conducted in SPM12 (SPM12, 2014) with Matlab 2014a ("Matlab: MATLAB and Statistics Toolbox Release," 2014a). White matter labels were derived from the diffusion tensor imaging atlas developed by the International Consortium of Brain Mapping, (ICBM-DTI-81; Mori et al., 2008; Oishi et al., 2008) in FSL (FMRIB Software Library; Jenkinson et al., 2012). Further statistical analyses were conducted in SPSS 21.0 (Statistical Package for the Social Sciences by IBM-Corp, Released 2012).

6.2.4 Data preparation and analysis

6.2.4.1 Preprocessing

Preprocessing and analysis of the MRI data was conducted in Matlab 2014a ("Matlab: MATLAB and Statistics Toolbox Release," 2014a) using SPM12 (SPM12, 2014). Preprocessing steps: segmentation, creation of a DARTEL template and normalisation to Montreal Neurological Institute (MNI) space, were taken to prepare the T1 weighted images for statistical analysis based on the recommendations made in the SPM12 manual (Ashburner et al., 2014). In addition to these steps, overall tissue volumes were calculated.

Segmentation: the images were segmented into the six standard tissue types using TPMs. A light regularisation and a 60mm full width half maximum (FWHM) cut off were applied. Grey matter (GM), white matter (WM) and cerebral spinal fluid (CSF) volumes in native space were generated for the calculation of total inter-cranial volume. For GM and WM, files in DARTEL space were also generated for use creating DARTEL templates. The following other settings were employed for segmentation; an Markov Random Field (MRF) clean-up procedure of one, light

clean, zero smoothness, a sampling distance of three, no deformation and the default settings for warping regularisation and affine registration (ICBM space template – European brains).

DARTEL template: the saved, DARTEL imported files (rc1^ and rc2^.nii files) created during segmentation were used to generate the DARTEL templates. The default settings in SPM12 were retained for this stage of preprocessing. There were 18 iterations within this phase. With each iteration the individuals within the separate models were brought closer to the template and new, more refined template was generated after every three iterations. This resulted in six templates, with the sixth template being the most refined. The actual amount of tissue in each region was preserved and flow field estimates of the degree of warping needed to fit each participant to the template were recorded. Separate templates for males, younger individuals and younger males only were generated.

Normalization to MNI template: the DARTEL templates for each of the three analytical models were then, separately, normalized to MNI space. The final (sixth) template for each model and the saved flow fields were used to normalize WM and GM to the MNI template. The data were resampled to 2 x 2 x 2mm voxel dimensions and a 6 x 6 x 6mm Gaussian smoothing kernel was applied to the data.

Calculating tissue volumes: a dependency was set in the preprocessing batch to calculate the volumes of GM, WM and CSF generated during segmentation for use in subsequent analysis.

6.2.4.2 Covariates of no interest

Certain variables were included in the model specification phase of the analysis in order to control for their potentially confounding effects. The reasoning behind, and calculation of, two of these (tissue volumes and handedness) will be discussed below.

Total inter-cranial volume was calculated by summing the volumes of GM, WM and CSF that were generated during preprocessing. In addition to this, a between groups analysis of the three separate tissue types was conducted in SPSS. If a volumetric difference was found between groups for an individual tissue type, independent of total inter-cranial volume, this tissue type was also entered as a covariate of no interest in the model estimation phase. The results of this analysis are discussed in section 6.3.1 below. In summary, it necessitated the inclusion of a regressor of CSF volume in the factorial model of gender and 'risk status' in younger individuals, and in the analysis of risk status in younger males only.

The EHI was employed in the current study to account for the effects of handedness from the analysis of brain volumes. Oldfield (1971) proposed that one's handedness index should be calculated using the following formula;

$$H = 100.\frac{\sum_{i=1} X(i, R) - \sum_{i=1} X(i, L)}{\sum_{i=1} X(i, R) + \sum_{i=1} X(i, L)}$$

For each participant, the sum of the crosses in the left hand column is subtracted from the sum of the crosses in the right hand column. This number is divided by the total number of crosses and multiplied by 100. Scores range from -100 to

+100, with positive scores indicating a right handed preference and greater magnitudes corresponding to a greater degree of preference for that hand. These figures were included in the design matrices, despite a lack of significant difference in the EHI between groups.

6.2.4.3 Group Specification & Second Level Analysis

Group level analysis was conducted in SPM12 (SPM12, 2014). Gender and risk status were compared in individuals under the age of 25 with a full 2 x 2 Factorial analysis of variance (ANOVA) model. Age, handedness (as measured using the EHI), CSF volume and total inter-cranial volume were entered into the model as covariates. A one-way analysis of variance was utilised for the analysis of risk status in older and younger male drivers. In this analysis, age was entered as a covariate of interested and the effects of handedness and total inter-cranial volume were regressed out. CSF was not included as it did not differ significantly between risky and safe males, was not seen as a potential confound. Finally, a two-sample t-test approach was utilised to compare younger risky and safe male drivers directly. The variance contributed by age, handedness and total inter-cranial volume were regressed out.

Independence and unequal variance were assumed in all models. For examples of design matrices see appendices AB. These models were applied to GM and WM separately. To control for multiple comparisons, Family Wise Error (FWE) rate correction was applied at the peak-voxel level with a threshold of 0.05 and activations were required to exceed a cluster extend threshold of 50µl in magnitude.

6.2.4.4 Inference; Small Volume Correction (SVC) analysis

It has been proposed that impulsive behaviours are driven by the ventral and dorsal striatum and inhibited by prefrontal cortical regions (Fineberg et al., 2010). According to a leading theory by Casey and colleagues over a series of studies (e.g. Casey et al., 2005; Casey et al., 2008; Casey et al., 1997), an imbalance between these driving and inhibitory processes exists in adolescents and young adults, resulting in reduced cognitive control and increased sensitivity to reward. The prefrontal cortex continues to develop into the 20s (Casey et al., 2005), and its development may play an important role in the maturation of higher cognitive abilities and declining levels of impulsivity (Casey, Tottenham, & Fossella, 2002; Casey et al., 1997).

In the current section, a Small Volume Correction (SVC) approach was applied to further investigate this hypothesis. GM volume in the striatum, insula and certain frontal structures were compared between groups using regions of interest identified in the literature above. In the SVC analysis of age and risk status in males the hippocampus replaced the insula, as this region was of more interest to that analysis.

A full list of these regions can be found in table 6.1. Anatomically defined ROI masks were created with the Wake Forest University (WFU) PickAtlas toolbox (version 3.0.5; Maldjian et al., 2003) in SPM12 and resampled to voxel dimensions 2 x 2 x 2mm, in line with the dimensions of the current data. With the exception of the DLPFC, SMA and pre-SMA, regions were defined using the Automatic Anatomical Labelling atlas (AAL; Tzourio-Mazoyer et al., 2002). As the AAL did not contain definitions for the pre-SMA and DLPFC an alternative approach was taken

for these regions. As mentioned in chapter five, and in keeping with previous research, the DLPFC was defined as Brodmann areas nine and 46 (e.g. Garavan et al., 2002). The pre-SMA was separated from the SMA using guidelines set out on page 229 of Simmonds et al. (2008), based on a meta-analysis of anatomical studies of this region.

All ROI masks were applied through the results viewer in SPM12. As before, *p*-values for peak-voxels were corrected using the FWE rate approach and a minimum cluster extend threshold of 50µl were employed. The alpha threshold was adjusted for the ten regions used in the SVC analysis to 0.005 (p=0.05/10 = 0.005).

6.2.4.5 MarsBaR extraction and correlational analysis

To calculate the magnitude of the relationship between GM volume, age and other measures of impulsivity and driving risk, the GM volumes for particular regions were calculated. Only regions where the GM signal passed the initial FWE corrected threshold were used.

This meant that GM signal was extracted from one region (the SMA) in younger individuals and in younger males, and all ten regions, mentioned in table 6.1, in males. Where an eigenvariate value for each participant was extracted from within a sphere in the analysis of the fMRI data, the current data necessitated the extraction of the mean value for the GM signal within the whole, anatomically defined ROI. This was achieved using the MarsBaR toolbox (MARSeille Boîte À Région d'Intérêt, version 0.44; Brett, Anton, Valabregue, & Poline, 2002) in SPM12.

Structural ROIs generated using the WFU PickAtlas toolbox were imported into MarsBaR and converted into a compatible format for analysis. MarsBaR ROI analysis was carried out on the results files produced during the VBM group level analysis, outlined in the previous section.

Table 6.1: Regions selected for SVC analysis

Task	**Region**	**Abbreviation**
"All Males" **n=10**	Anterior cingulate cortex	ACC
	Amygdala	N/A
	Dorsolateral prefrontal cortex	DLPFC
	Hippocampus	N/A
	Inferior frontal gyrus	IFG
	Orbitofrontal cortex	OFC
	Caudate, putamen, pallidum, nucleus accumbens	Striatum
	Supplementary motor area	SMA
	Pre-supplementary motor area	Pre-SMA
	Ventromedial prefrontal cortex	VMPFC
"Younger individuals" **and** "Young males" models **n=10**	Anterior cingulate cortex	ACC
	Amygdala	N/A
	Dorsolateral prefrontal cortex	DLPFC
	Inferior frontal gyrus	IFG
	Insula	N/A
	Orbitofrontal cortex	OFC
	Caudate, putamen, pallidum, nucleus accumbens	Striatum
	Supplementary motor area	SMA
	Pre-supplementary motor area	Pre-SMA
	Ventromedial prefrontal cortex	VMPFC

For each ROI imported, MarsBaR calculated a mean time course for every voxel in the ROI and summarised these further into a grand mean value for each participant. These values were exported to Excel and, subsequently, transferred to SPSS. Correlations between GM volumes and selected psychometric and cognitive measures were conducted. As before, the alpha level of 0.05 was adjusted for the number of comparisons made to 0.003 (20 comparisons, p=0.05/20 = 0.0025).

6.3 Results

6.3.1 Tissue volumes

Volumes of white matter (WM), grey matter (GM) and cerebral spinal fluid (CSF), as well as total intercranial volume (the sum of these three tissue types) were compared between experimental groups in order to identify potential confounds for the VBM analysis. The means and standard deviations (SDs) for these volumes in older males, younger males and younger females can be found in table 6.2.

For consistency, these volumes were compared using the same analytical models that were used for the between groups VBM analysis: all males were compared (one-way ANOVA of risk status), then younger individuals (gender x risk status) and, finally, younger males (one-way ANOVA of risk status).

All risky and safe males were compared using one-way ANOVAs. These analyses found that there were no significant differences between risky and safe male drivers in their total intercranial volume or any of the individual tissue volumes (F<1). The tissue volumes of individuals under the age of 25 were compared with a 2 x 2 Factorial ANOVA of gender and risk status (risky vs. safe drivers). There was a significant main effect of gender for total intercranial volume and each of

the three individual tissue types, such that males had greater volume than females, the means and standard deviations of which can be found in table 6.2 above (total intercranial volume; $F(1,78)=65.14$, $p<.001$, $\eta^2=.46$, GM; $F(1,78)=51.21$, $p<.001$, $\eta^2=.396$, WM; $F(1,78)=46.21$, $p<.001$, $\eta^2=.37$, CSF; $F(1,78)=14.4$, $p<.001$, $\eta^2=.16$). In young individuals there was a significant main effect of risk status for CSF volume, $F(1,78)=5.62$, $p<.05$, $\eta^2=.07$, where safe drivers had more CSF (M=0.2, SD=0.05) than risky drivers (M=0.18, SD=0.03). There were no other main effects of risk status and no significant interactions with gender. Risk status and tissue volumes were also examined in younger males with one-way ANOVAs. Safe younger males had significantly more CSF (M=0.22, SD=0.06) than risky younger males (M=0.19, SD=0.03), $F(1,38)=4.89$, $p<.05$, $\eta^2=.114$. There were no other significant differences in tissue volumes between younger risky and safe males.

Table 6.2: Means and SDs for three tissue types; GM, WM and CSF

		WM^	**GM^**	**CSF***	**Total^**
Older males	Risky	0.49 (SD=04)	0.81 (SD=.08)	0.26 (SD=.07)	1.56 (SD=.12)
	Safe	0.48 (SD=.06)	0.8 (SD=.08)	0.27 (SD=.05)	1.55 (SD=.13)
	Overall	0.49 (SD=.05)	0.8 (SD=.08)	0.27 (SD=.06)	1.56 (SD=.12)
Younger Males	Risky	0.47 (SD =.04)	0.86 (SD =.06)	**0.19** (SD =.03)	1.5 (SD =.08)
	Safe	0.47 (SD =.04)	0.87 (SD =.05)	**0.22** (SD =.06)	1.56 (SD =.12)
	Overall	**0.47** (SD=.04)	**0.86** (SD=.05)	**0.2** (SD=.05)	**1.54** (SD=.1)
Younger females	Risky	0.41 (SD=.03)	0.78 (SD=.04)	**0.17** (SD=.02)	1.36 (SD=.07)
	Safe	0.41 (SD=.0.4)	0.78 (SD=.06)	**0.18** (SD=.02)	1.37 (SD=.1)
	Overall	**0.41** (SD=.04)	**0.78** (SD=.05)	**0.17** (SD.02)	**1.37** (SD=.9)

Relevant means are highlighted in bold. Total refers to total intercranial volume. ^ = main effect of gender was found, * = main effects of gender and risk status were found.

6.3.2 White matter (WM)

Regional analysis of WM volumes in both younger and older males was conducted using a one-way ANOVA of risk status, with age included in the model as a covariate of interest. The variance contributed by total intercranial volume was regressed out of the model, as well as each participant's EHI score.

No significant, global differences were found between the regional WM volumes of risky and safe males. There was also no relationship found between regional WM volumes and declining age, however, a significant, positive relationship was found between age and WM volume in the following regions; bilateral external capsule, bilateral uncinate, and the hippocampal aspects of the left and right cingulum bundle. Details of the precise location and volume of these regions can be found in table 6.3 and they are illustrated in figure 6.1.

Table 6.3: Regions that demonstrated a significant positive correlation between WM and age in males

Region		**Volume**	**Peak p**	**Peak**	**Peak**
External capsule	Left	3024	0.000	6.58	-30, -4, 10
	Right	904	0.002	5.88	28, 2, 9
Uncinate	Left	328	0.000	6.57	-32, 2, -24
	Right	664	0.001	6.28	30, 3, -26
Cingulum bundle, hippocampal aspect	Left	536	0.004	5.77	-21, -21, -27
	Right	664	0.001	6.13	21, -20, -27

Global analysis of regional WM volumes in younger individuals was conducted using a 2 x 2 Factorial ANOVA model of gender and risk status, with age entered as

a covariate of interest and CSF volume, total intercranial volume and EHI score entered as covariates of no interest. In this model no regional WM differences were found between risky and safe young drivers, no significant effects of gender and there were no interactions between gender and risk status. In addition, no significant relationship was found, either positively or negatively, between regional WM volumes and age.

Regional WM was also examined in younger males alone, using a one-way ANOVA of risk status. Again, CSF volume, total intercranial volume and EHI score were regressed out of the analysis but age was entered as a covariate of interest. No significant differences were found between risky and safe young male drivers and there was no significant relationship between regional WM volumes and age, in either direction.

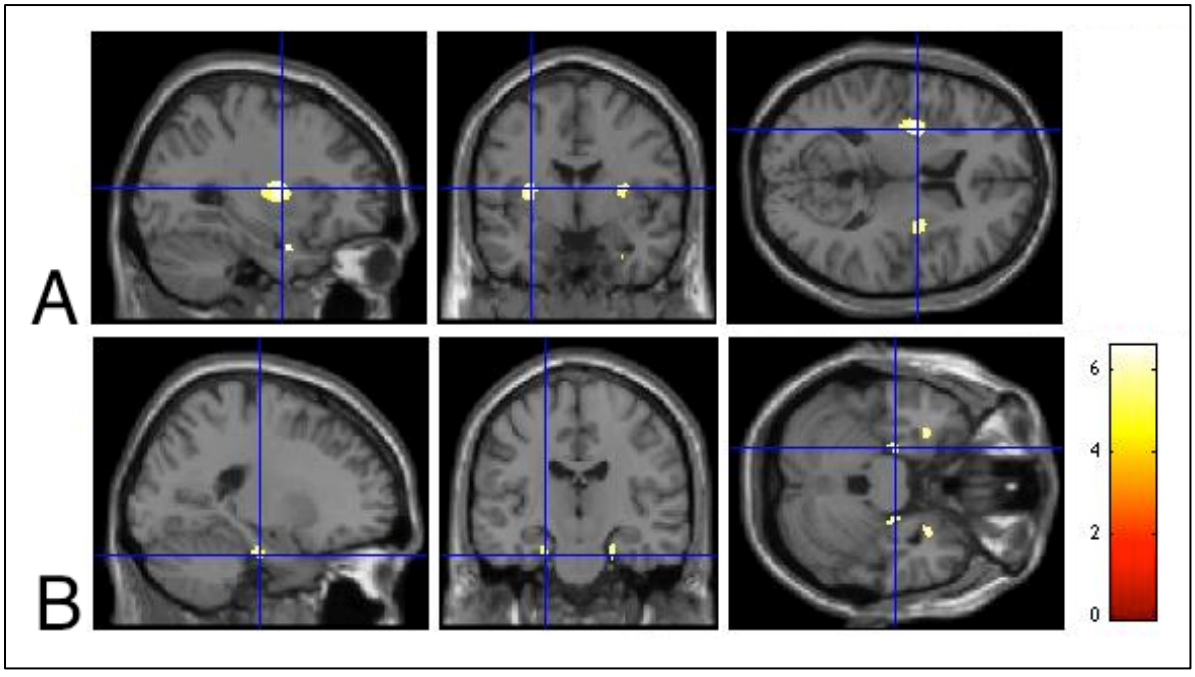

Figure 6.1: A) Aspects of the bilateral external capsule that showed WM increase with age in males, B) Illustration of WM in the hippocampal aspect of the left and right cingulum bundle that showed a positive correlation with age in males.

6.3.3 Grey matter (GM)

6.3.3.1 Male drivers

6.3.3.1.1 GM volume and age in males

While there were no significant increases in GM with age, extensive decline in regional GM volume was observed. This decline extended to the bilateral precuneus, middle and anterior cingulum, bilateral thalamus, left caudate, aspects of the bilateral hippocampus, bilateral angular gyri, temporal gyri, occipital gyri, fusiform gyri, pre and postcentral sulci, superior parietal gyri, aspects of the cerebellum, right lingual gyrus, and frontal regions including the superior, frontal, middle and inferior gyri, as well as the OFC. These regions are illustrated in the following three figures, where figure 6.2 demonstrates subcortical GM regions that decreased in volume with age and figures 6.3 and 6.4 show declining cortical GM regions on the exterior and interior aspects of the right and left hemispheres, respectively.

Average GM volumes were extracted from the same ROIs used in the SVC analysis of risk status in males. Table 6.4 summarises the correlations between GM in these regions and age. With the exception of the SMA, pre-SMA and the DLPFC, separate Pearson's correlations were calculated for the left and right hemispheres. Despite being identified in SPM as a region showing significant GM decline with age, the correlation between overall GM volume in the hippocampus and age was not significant (p=.064).

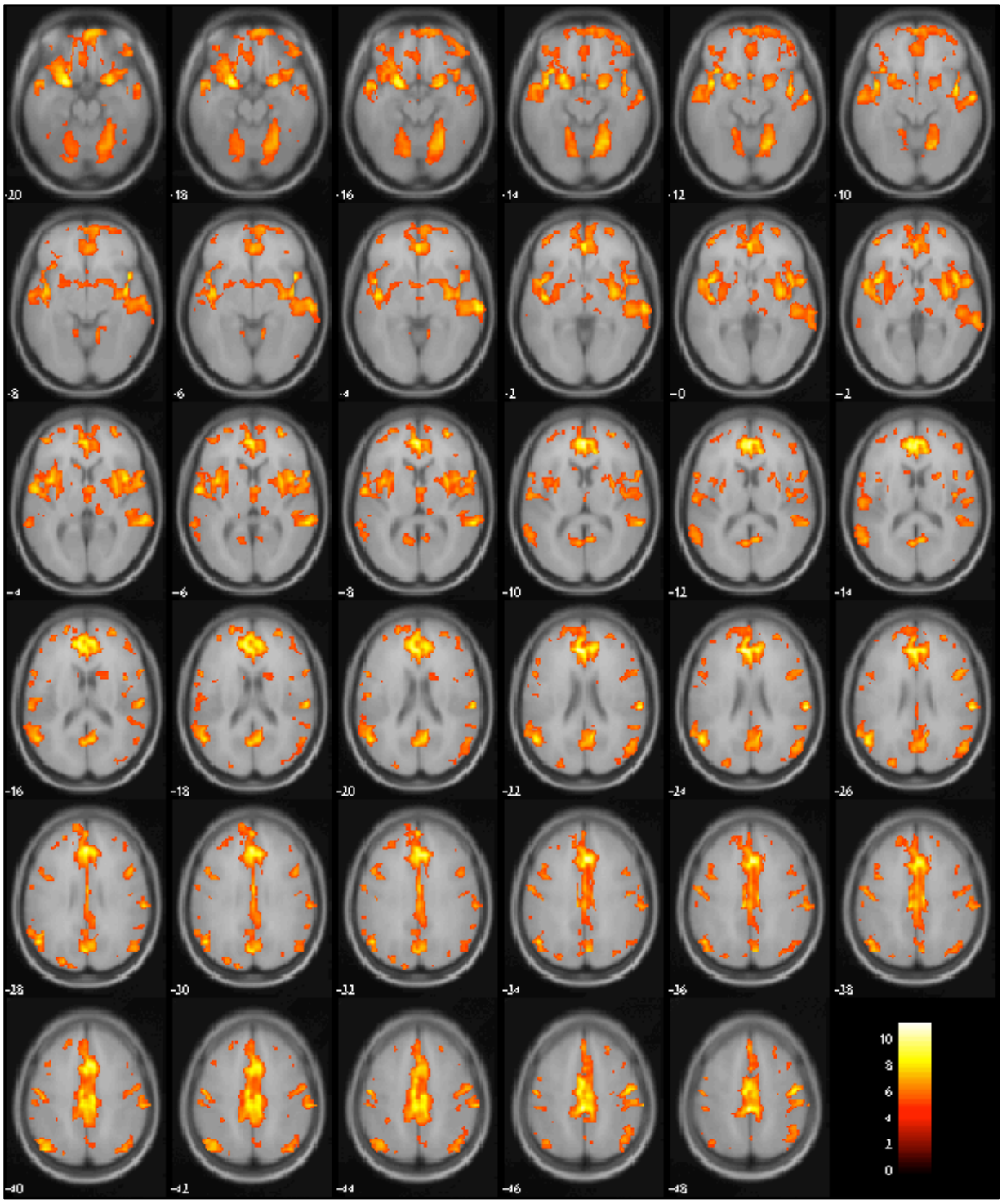

Figure 6.2: Axial slices showing GM decline with age

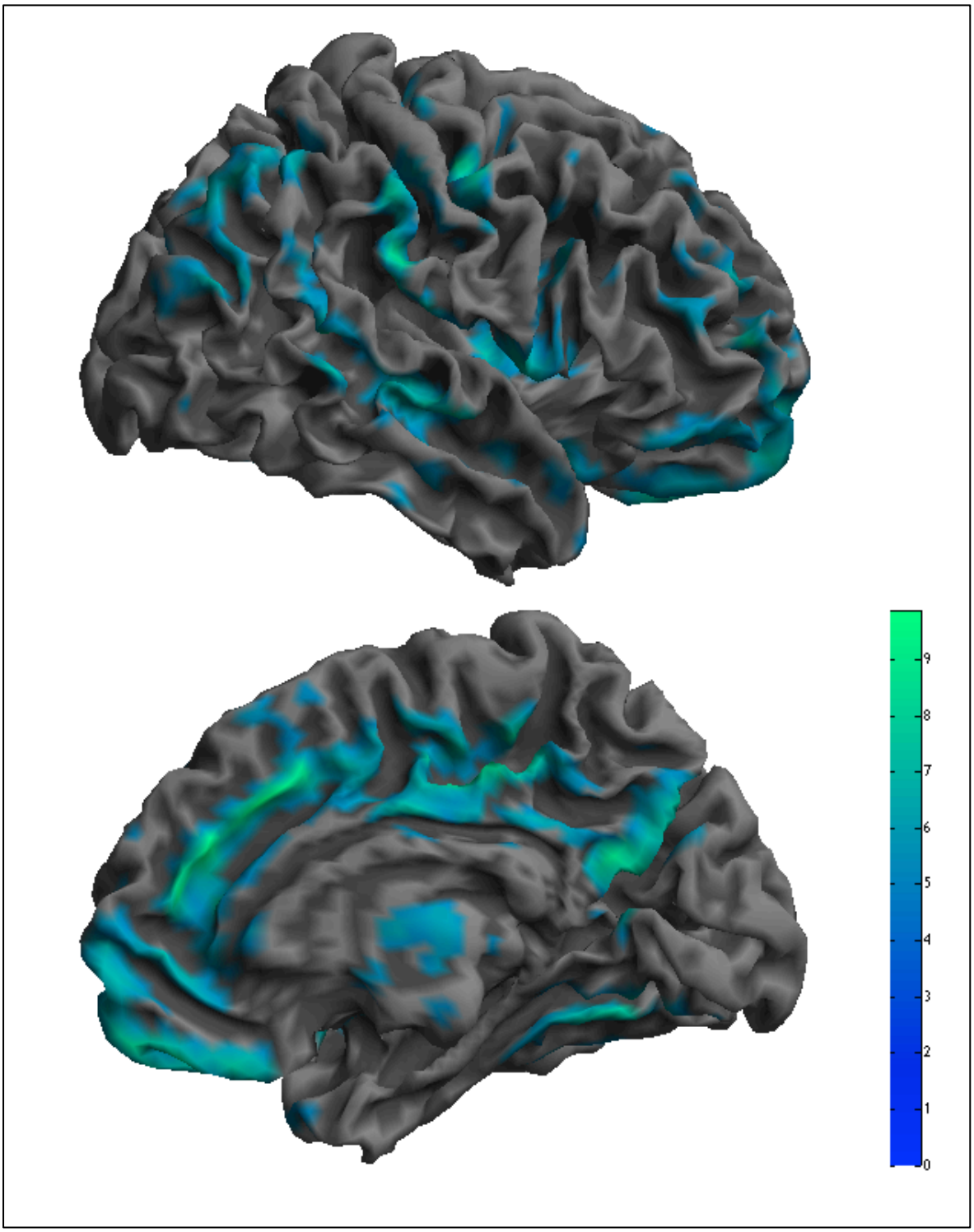

Figure 6.3: Rendering of GM decline with age in the right hemisphere, both lateral and medial surface

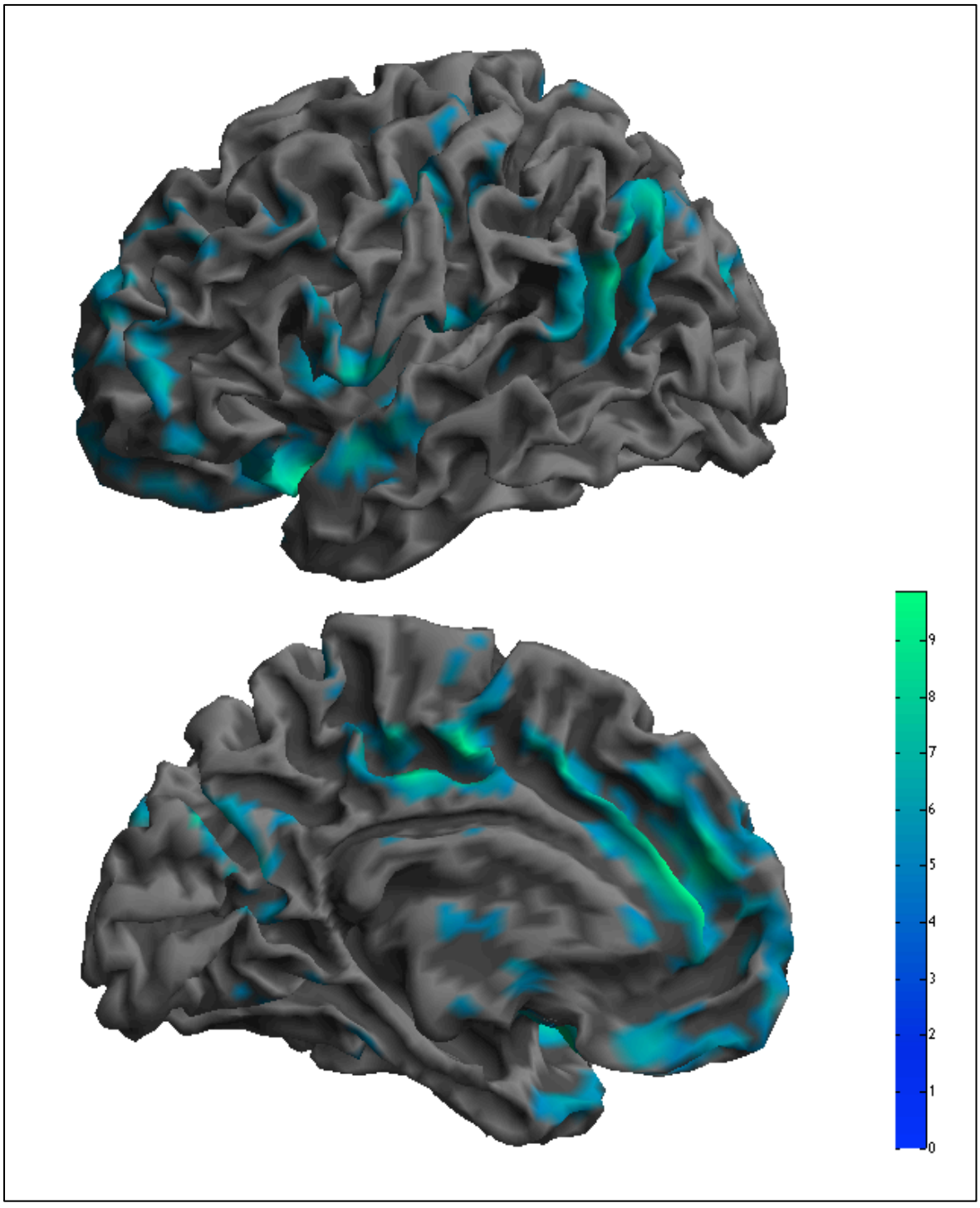

Figure 6.4: Rendering of GM decline with age in the left hemisphere, both lateral and medial surfaces

Table 6.4: Correlations between GM volume in certain ROIs and age in males

Region		**Volume in µl**[31]	**Peak p (FWE corrected)**	**Peak T**	**Peak Coordinates (x, y, z)**	**Pearson's r**[32]
ACC	Left	44912	0.000	11.44	-2, 45, 9	**–.608**
	Right		0.002	5.02	2, 18, -9	**–.587**
Amygdala	Left	2464	0.000	9.52	-21, 3, -16	**–.400**
	Right	1872	0.000	8.81	20, 2, -15	**–.346**
DLPFC		21760	0.000	9.91	-8, 39, 22	**–.564**
IFG	Left	17672	0.000	7.13	-56, 10, 20	**–.549**
	Right	18768	0.000	7.75	57, 10, 6	**–.586**
OFC	Left	38704	0.000	9.75	-22, 8, -21	**–.541**
	Right	36160	0.000	7.97	8, 57, -22	**–.479**
Striatum	Left	22928	0.000	7.88	-18, 6, -15[33]	**–.500**
	Right	27368	0.000	8.23	33, -6, 2[34]	**–.529**
Pre-SMA		26400	0.000	9.72	4, 20, 39	**–.591**
SMA		10280	0.000	6.25	-6, -6, 58	**–.568**
VMPFC	Left	53880	0.000	9.79	-9, 42, 18	**–.532**
	Right		0.000	9.7	2, 30, 32	**–.446**

[31] In certain regions SPM gave only one value for the volume of the whole region across the left and right hemispheres.

[32] Each with 79 participants and $p<.001$, two tailed

[33] Peak was located in the right putamen

[34] Peak was located in the left nucleus accumbens (NAcc)

6.3.3.1.2 Between groups analysis

Regional analysis of GM volumes in both younger and older males was conducted using a one-way ANOVA of risk status. The relationship between age and GM volume was also investigated. As with the analysis of WM volume, the effects of total intercranial volume and handedness (EHI score) were regressed out of the model.

The effect of risk status in males was investigated using a SVC approach, as outlined in section 6.2.4.4. Safe males were found to have more GM in the SMA than risky males, however, when the threshold was further adjusted for the number of comparisons made, this difference was no longer significant (p=.028). There were no other differences between risky and safe males.

6.3.3.2 Young drivers

Global analysis of regional GM volumes in individuals under the age of 25 was conducted using a 2 x 2 Factorial ANOVA model of risk status and gender. Age was included in the model but CSF volume, total intercranial volume and EHI score were included as nuisance covariates. An initial inspection revealed no significant, global, difference between risky and safe young drivers, no interaction between risk status and gender and no relationship between GM volume and age. Some differences in regional GM volume between males and females were detected at a global level, however. In males, a cluster of greater GM density was detected in the cerebellum, located at coordinates -12, -94 -26, $t(72)=5.59$, $p<.05$, 112µl. Two regions showed greater GM volume in females compared to males, the middle

cingulate cortex (MCC) (104µl, $t(72)=5.77$, $p<.05$, 3, -6, 21) and part of the OFC (56µl, $t(72)=5.68$, $p<.05$, 3, -27, 18).

An SVC was adopted using the ROIs mentioned in table 6.1. No relationship between GM volume and age was found in these regions, no further effects of gender and no interactions between gender and risk status. A significant main effect of risk status was found in the left SMA, such that young safe drivers had more GM in this region than young risky drivers, $F(1,72)=4.53$, $p<.005$, 344µl (x, y, z: -15, -4, 62). This effect can be seen in figure 6.5. No other regions demonstrated a main effect of risk status.

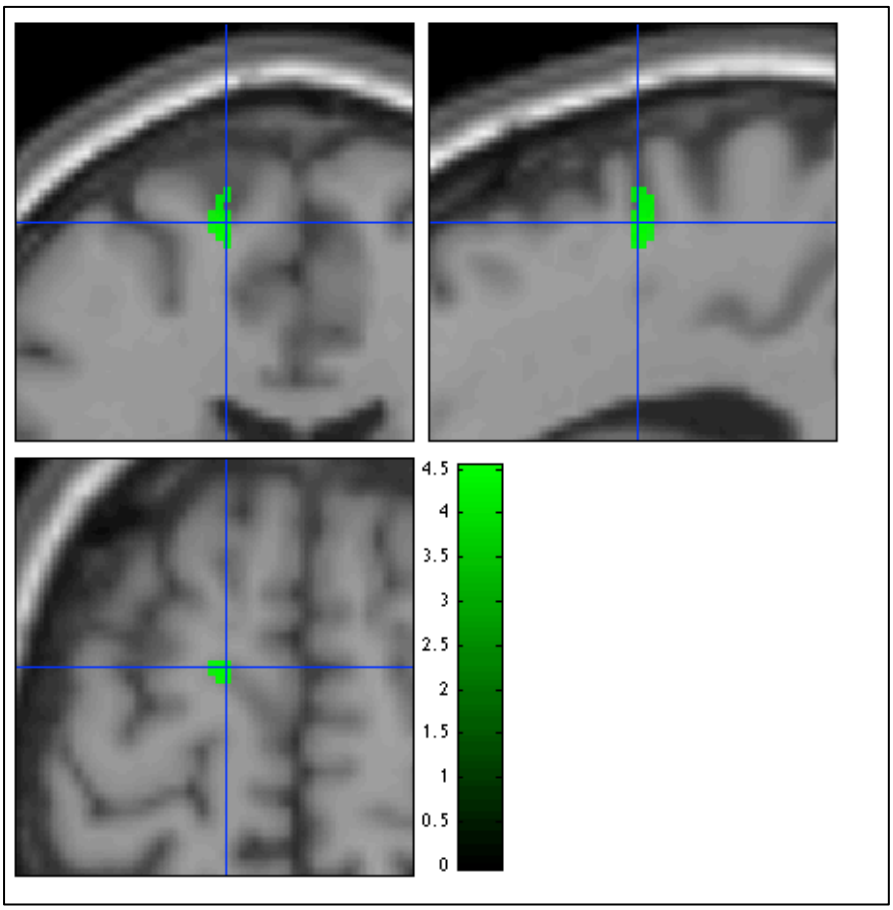

Figure 6.5: Region of the SMA where safe young drivers demonstrated greater GM volume in the than risky drivers, 80 x 80 mm zoom.

6.3.3.3 Young male drivers

Regional analysis of GM volumes in younger male drivers was conducted using a one-way ANOVA of risk status. Age was included as a covariate of interest and CSF volume, total intercranial volume and handedness (EHI score) were regressed out of the model. An initial inspection revealed no significant, global, difference between risky and safe young male drivers and no relationship between GM volume and age. An SVC analysis was adopted using the ROIs mentioned in table 6.1. No relationship between GM volume and age was found in these regions. Similar to the finding in all younger individuals, young safe males were found to have more GM volume in the SMA than young risky male drivers, $t(34)=4.38$, $p<.005$, 160µl (x, y, z: -15, -6, 63). This effect can be seen in figure 6.6. There were no other differences between risky and safe young males.

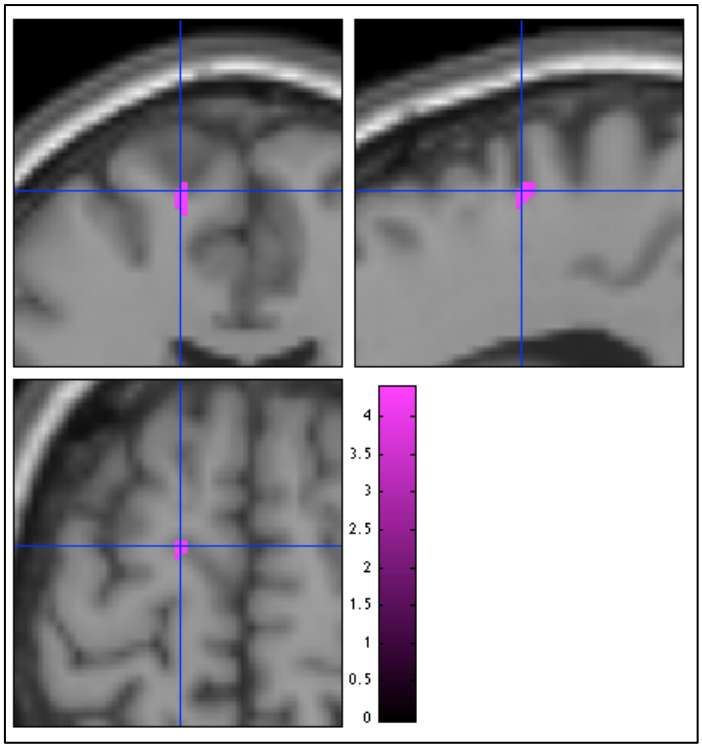

Figure 6.6: Region of the SMA where young safe male drivers demonstrated greater GM volume than young risky male drivers, 80 x 80 mm zoom.

6.3.3.4 Regional GM volume and its relation to other measures

6.3.3.4.1 Psychometric measures

In males, extracted GM volumes from the ROIs mentioned in section 6.2.4.5 were correlated with certain subscales from the Manchester DBQ, the BIS-11 and Kirby's DDQ. These psychometric scales were also correlated with average GM volume in the SMA in both younger individuals and in younger males only. After adjusting for the number of comparisons made, no significant correlations remained between GM volume and any of the psychometric scales.

6.3.3.4.2 Behavioural measures

These GM volumes were then correlated with certain behavioural measures from the IGT, MID and the XY tasks. After adjustment for multiple comparisons, no significant correlations were found between GM volumes and behavioural measures in younger individuals or in young males. In the analysis of all males, however, significant negative correlations were found between participants' combined reaction times to infrequent punishment decks (decks B and D) and GM volume in the left and right ACC ($r(79) = -.381$, $p<.003$, two-tailed; $r(79) = -.343$, $p<.003$, two-tailed), left IFG ($r(79) = -.349$, $p<.003$, two-tailed), right insula ($r(79) = -.337$, $p<.003$, two-tailed), SMA ($r(79) = -.362$, $p<.003$, two-tailed) and pre-SMA ($r(79) = -.34$, $p<.003$, two-tailed), such that decreases in GM volume in these regions were associated with slower responses to decks B and D (increased RT). Figure 6.7 shows the relationship between GM volume in the left ACC and infrequent punishment deck RTs.

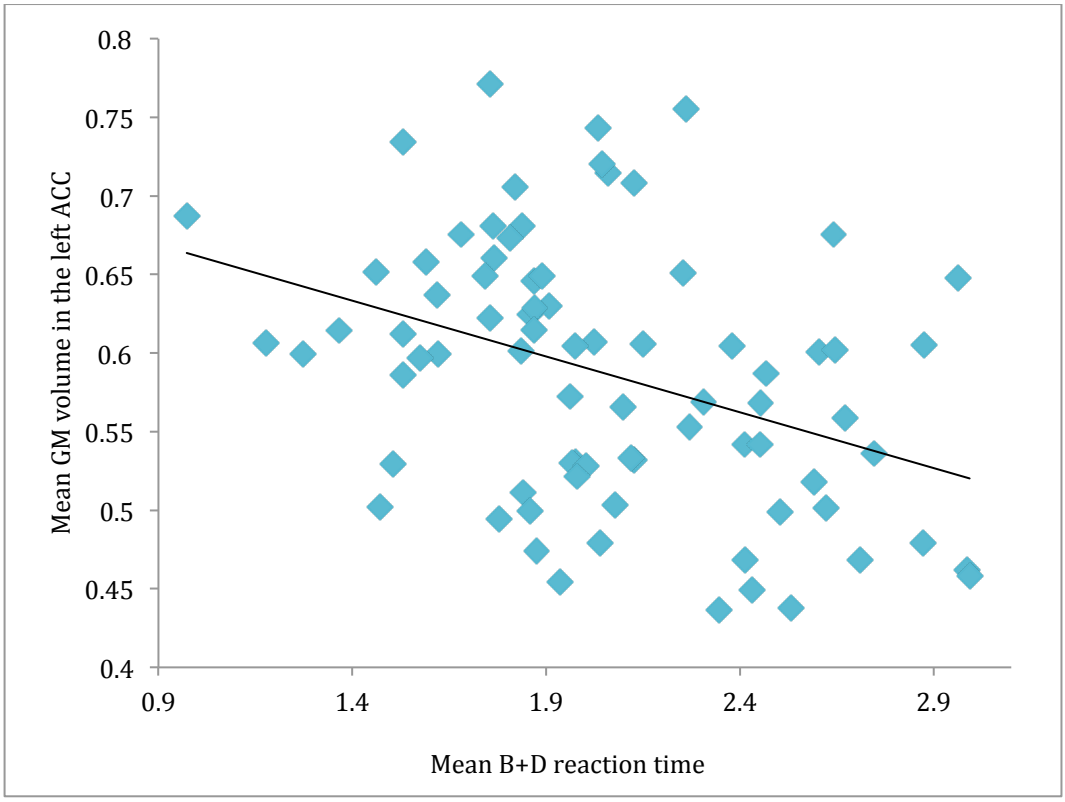

Figure 6.7: Scatter plot illustrating the relationship between mean GM volume in the left ACC in males and the average time to select from decks B and D (infrequent punishment decks)

6.4 Discussion

6.4.1 Summary of main findings

6.4.1.1 Hypothesis one: the PFC will demonstrate steeper GM decline than the striatum

The current study observed extensive decline in GM volume with age in males between the ages of 18 and 56. Decreasing GM density occurred across the four lobes of the brain, however the most extensive decline was noted in the cingulum, particularly the anterior aspect, and other prefrontal regions such as the OFC and the DLPFC. As simultaneous GM decline and WM increase were observed in males, these changes with age are likely to reflect maturation, rather than degenerative

processes (Sowell et al., 2004). It is important to note too that, while the age range of males in the current study was quite large (18 to 56), the 'older' group comprised mostly of males between the ages of 29 and 39, with an overall average age of 38.48 (8.08) years. Within the older male sample, 62.5% of participants were aged between 29 and 39, 20% were between 40 and 49 and 17.5% were between 50 and 56. Therefore the likelihood of these results being confounded by degeneration is slim.

Regional GM volumes were extracted for certain ROIs and Pearson's correlations were calculated to determine the magnitude of GM decline with age, using guidelines provided by J. Cohen (1988). The results suggest that there is a very strong relationship between age and GM decline in the right IFG, DLPFC, SMA, pre-SMA and ACC, a strong relationship with left IFG, OFC, striatum and left VMPFC and a moderate relationship with GM decline in the amygdala and right VMPFC. While a small cluster of GM decline was identified in the inferior aspect of the left hippocampus, the correlation between overall hippocampal GM volume and age did not reach significance. This is inconsistent with previous research that observed overall hippocampal volume decreased with age in adult males (Pruessner et al., 2001), but consistent with previous evidence of decreasing amygdala volume (Giedd et al., 1997; Giedd et al., 2006; Giedd, Snell, et al., 1996; Giedd, Vaituzis, et al., 1996). Also in accordance with the literature, extensive GM decline with age was observed in prefrontal regions, such as the DLPFC and OFC, reflecting the continued maturation of the PFC into the mid 20s (Giedd, 2004; Gogtay et al., 2004; Sowell et al., 2003).

Research has suggested that, while the pallidum may not decrease in volume with age, the caudate, putamen and NAcc appear to do so (Giedd et al., 2006; Nunnemann et al., 2009; Raz et al., 2005; Walhovd et al., 2011). The current study observed significant decline in GM in all of these regions, with peak voxels located in the right putamen and left NAcc, suggesting a strong relationship in these regions in particular. However, as the MarsBaR extraction method gives a single value for the GM signal across an entire ROI, correlation coefficients for the relationship between GM decline and age were only generated for the left and right striatum, without differentiating between the sub-regions within the mask. The current study hypothesised that GM decline in prefrontal regions would occur more rapidly than in striatal regions, as these are thought mature earlier in adolescence (Casey et al., 2008). This hypothesis was tested by comparing the magnitude of the relationships between age and GM volume in these regions. While some prefrontal regions, such as the ACC, demonstrated a greater decline with age than the striatum, the difference between these correlation coefficients was not sufficient in magnitude to support this hypothesis. Therefore it appears prefrontal and striatal regions mature at a similar rate from late adolescence into later adulthood in males.

Despite evidence of the continued development of the PFC, the indication that the striatum is undergoing a similar rate of change in younger males calls into question the applicability of the neurobiological theory of risk-taking put forward by Casey et al. (2008) in an older adolescent sample. From conclusions drawn in chapter five, the continued structural development of the brain during late adolescence and early adulthood may not have as dramatic an impact on reward-

seeking behaviour as expected, as there were very few differences in performance or neural activation found between older and younger males. Therefore, where increased risk-taking may be caused by an imbalance in the development of the reward processing and executive control systems of the brain in younger adolescents, this was not replicated in the current sample. As activational differences were observed between risky and safe drivers in chapter five, there may instead be differences in brain structure that could explain why some drivers are more vulnerable to increased risk-taking while driving. Hypothesis two, parts A, B and C, addresses whether any potential differences are independent of age and/or gender.

Furthermore, as many studies have demonstrated the continued development of the PFC into the mid 20s (Giedd, 2004; Gogtay et al., 2004; Sowell et al., 2003), a significant GM decline and WM increase with age was expected in younger individuals, i.e. in the analysis of all younger individuals and in the separate analysis of younger males. However, no significant correlations between age and these volumes in any region were detected in either of the younger analytical models. This may be due to the restricted age range utilised in the current study. This limitation is discussed in greater detail in section 6.4.2.

6.4.1.2 Hypotheses two, parts A, B and C: there are differences in brain volume between risky and safe drivers

As it was hypothesised that the development of some brain structures may underlie a propensity towards risk-taking in younger individuals, independent of gender, tissue volumes were compared between all risky and safe drivers between 18 and 24. Unexpectedly, safe young drivers were found to have significantly more

global CSF volume than risky. This was found in all younger drivers and also in younger males when they were examined separate from all other groups. No differences in global CSF volume were detected between risk groups when younger and older males were combined.

Traditionally, CSF is thought to be responsible for providing nutrients and removing waste from the brain, however some studies have also suggested that the composition CSF may influence behaviour (Veening, de Jong, & Barendregt, 2010). For example, animal studies have suggested that CSF may be important for regulating behavioural states and even levels of consciousness as it conveys peptide signals to the thalamus, hypothalamus and pineal gland (for review of the CSF literature see; Zappaterra & Lehtinen, 2012). Concentrations of neurotransmitters such as 5-hydroxyindoleacetic acid (5- HIAA; the main metabolite of serotonin) and oxytocin in human CSF have been linked to aggression, psychopathology and suicidal tendencies (Mann & Currier, 2007; Soderstrom, Blennow, Sjodin, & Forsman, 2003). In elderly adults, greater CSF volume has also been linked to poorer performance on cognitive tasks (Coffey et al., 2001). However, CSF increase and cognitive decline are more likely to be a consequence of the age of the participants in that study, rather than there being a causal link between the two. Thus, the relationship between CSF volume and behaviour is yet unclear. To prevent confounding effects, CSF volume was subsequently regressed out of the analyses of grey and white matter volumes in younger individuals and younger males.

As the SMA and pre-SMA have been reported to have distinct structural properties, with the former having greater connections with primary motor areas

and the latter with the DLPFC (Hoshi & Tanji, 2004), separate anatomical masks were defined for these regions in the current chapter. The results revealed that, while there was no difference in global GM volume between groups, safe younger drivers were found to have significantly greater GM density in the SMA than risky. This finding was replicated in younger males when they were examined separate to other groups. An effect in the same direction was also detected in the SMA when all males were combined, however this did not pass additional adjustments to the peak voxel threshold for the number of comparisons made.

Due to its structural connections with the primary motor cortex, the SMA is thought to be more directly related to motor output than the pre-SMA (Nachev, Kennard, & Husain, 2008). The SMA has been implicated in response initiation (Kawashima et al., 1996) and selection (Rowe, Hughes, & Nimmo-Smith, 2010). While it has been proposed that other regions are involved in more executive forms of stopping (Rubia et al., 2001), the SMA may be particularly important for mediating motor inhibition for voluntary action (Aron, 2007; Bari & Robbins, 2013; Simmonds et al., 2008). Patients with SMA lesions show prolonged reaction times and show a greater number of failed responses to 'go' stimuli in the Go/No Go task (errors of omission) (Fellows & Farah, 2005; Picton et al., 2007). Previous functional research has also identified the SMA as a potential marker for engagement in risk-taking behaviours, such as substance abuse (Kaufman et al., 2003).

Generally, cross-sectional VBM studies that have detected differences in SMA volume between groups appear to find that it is larger in healthy controls compared to certain disorders, such as Parkinsonian variant of multiple system

atrophy (MSA-P) (Tir et al., 2009). This pattern is not limited to the SMA, however. In the VBM literature in general, where volumetric differences are found between groups, smaller volume is typically associated with the clinical group when compared to healthy controls (e.g. Bloemers et al., 2014; Kosaka et al., 2010). Investigations of GM volume in the SMA have observed that reduced GM density in this region is associated with increased self-reported risk-taking and poorer demonstrations of executive functions. For example, Ersche et al. (2011) found that individuals who self-reported a greater number of years of cocaine use had reduced GM density in the SMA (i.e. a negative correlation between SMA GM density and years of cocaine abuse). Other studies have demonstrated negative correlations between SMA GM volume and psychometric measures, such as the (lack of) self-control subscale of the MMPI (Minnesota Multiphasic Personality Inventory) (Matsui et al., 2002) and the DBQ (Sakai et al., 2012), and with behavioural measures, such reaction times on the Stop Signal Reaction Time task (SSRT) (Hu, Chao, Zhang, Ide, & Li, 2014). Similarly, self-reported executive function capacity has been observed to positively correlate with SMA volume, indicating that less GM density in this region was associated with lower levels of executive function (Sakai et al., 2012). These studies provide evidence for the relationship between SMA structure, risk-taking and cognitive control abilities.

Some of the functional differences reported in chapter five may be relevant to the current finding. Namely, that greater SMA activity was observed in risky drivers during certain anticipation and feedback conditions, possibly reflecting a mechanism to compensate for this difference in volume. From this it would appear

that SMA volume might be a vulnerability marker of risk-taking while driving in younger individuals.

No differences in either global or regional WM volumes were observed between risky and safe drivers in any of the analytical models. Therefore, the current study provides no evidence to suggest that individuals who exhibit safe driving behaviours have stronger connections between reward processing and cognitive control regions. Future studies could employ DTI to better address this question.

To summarise, safe younger drivers were found to have more global CSF and more regional GM volume in the SMA than risky younger drivers. Thus, only part A of hypothesis two was supported, that volumetric vulnerability markers of risk-taking were independent of gender. Parts B and C of hypothesis two were rejected as no differences were found to be unique to younger males and no differences were independent of age. As functional and structural differences in the SMA were observed in younger individuals, this region, and its development into the mid 20s, may be a particularly important vulnerability marker for engagement in aberrant driving behaviours during this age. However, these results should be interpreted with caution. The sample size of the current study was small, and potentially insufficient to demonstrate true, between groups differences in brain volume. Furthermore, factors that have been found to influence brain volume, such as binge drinking (Coleman Jr, He, Lee, Styner, & Crews, 2011; Lisdahl, Thayer, Squeglia, McQueeny, & Tapert, 2013), were not controlled for and may have influenced the results.

6.4.1.3 Hypothesis three: there are gender differences in brain tissue volumes

In accordance with previous research, the overall brain size of males was larger (11.8% larger[35]) than females (Giedd, Snell, et al., 1996). This was also the case for all three tissue types (WM, GM and CSF) when analysed separately. As hypothesised, due to the age range of the current sample no differences in the rate of development of tissue types were observed between young males and females. However, it was also expected that some of the regions that have previously demonstrated sexual dimorphism in the literature would be replicated in the current study. Subcortical regions such as the caudate (Giedd et al., 1997; Paus et al., 1996) have been found to be larger in females and the amygdala, (Highley et al., 1999; Marcil et al., 2001) and pallidum (Giedd et al., 1997) have been found to be larger in males. The current study did not replicate any of the above subcortical findings but, instead, found that young males had more GM volume in the most posterior aspect of the cerebellum and females in the MCC and the OFC. This provides support to the theory that, while brain volume may be greater in males, certain frontal regions may show more cortical complexity in females (Cabeza & Nyberg, 2000). Greater prefrontal cortical complexity in younger females could be considered indicative of greater structural maturity. However, this is insufficient evidence of structural differences between younger males and females to generalise these findings to the overall difference in the instance of collision involvement between these groups.

[35] A combination of global WM and GM tissue volumes produced by the VBM analysis

6.4.1.4 Hypotheses four: there are correlations between GM volume and psychometric and behavioural measures of impulsiveness

GM volume was extracted from the ten ROIs mentioned in table 6.1 in males and the SMA in younger individuals and younger males. To investigate the extent to which individual differences in impulsiveness may be related to the structure of these regions, GM volumes were correlated against psychometric and behavioural measures of impulsivity. No relationship was found between GM volumes and responses to the BIS-11 or Kirby's DDQ in any of the analytical models. Similarly, no significant correlations between GM volume and behavioural measures were found in younger individuals or in younger males.

When all males were combined, however, moderate negative correlations were found between reaction times to infrequent punishment decks (decks B and D) in the IGT and GM volume in several regions, including the ACC, SMA and right insula, meaning that greater GM density in these regions was associated with faster selections from these decks. These regions have been implicated in inhibitory processes (SMA; Aron, 2007), decision-making under uncertainty (Insula; Krain et al., 2006), attention, conflict monitoring and error detection (ACC; Carter et al., 1998; Carter et al., 2000; Carter et al., 1997; Mesulam et al., 2001). While decks B and D both have an infrequent punishment schedule, losses from deck D still result in a net gain while losses from deck B result in a net loss, making it a disadvantageous deck. Individuals who are quicker to select from these decks may be demonstrating greater punishment avoidance overall, failing to differentiate between the magnitude of the losses. This could be considered an impulsive tendency and appears to relate to the structure of certain prefrontal

regions in males. When interpreted in light of the volumetric difference of the SMA in younger risky and safe drivers, and the behavioural differences reported in chapter four (i.e., that young risky drivers chose more infrequent punishment decks than young safe drivers), negative correlations between time to select from B and D and GM volume in the SMA may be further evidence of the link between the SMA, impulsivity and a history of risky driving.

6.4.1.5 Hypothesis five: there are correlations between GM volume and self-reported aberrant driving behaviours

GM volume in these regions was also correlated against DBQ scores as an alternative measure of driving risk. No significant correlations were found in any of the analytical models. Therefore the current study failed to replicate the relationship between SMA volume and DBQ scores reported by Sakai et al. (2012). This may be due to the different age range employed by the current study, as the oldest participant was 56, compared to the 65 to 76 age range employed by Sakai et al. (2012).

6.4.2 Limitations and future considerations

As the PFC has been noted to continue to develop into the mid 20s (Giedd, 2004; Gogtay et al., 2004; Sowell et al., 2003), the current study expected to find some GM volume decline with age within the younger models. Effects were detected an and uncorrected level of $p<.001$, however these did not pass the FWE correction procedure. This may be due to the narrow age range used in these models, reducing the power to detect an effect. In the current study 'younger' participants were aged between 18 and 24, a period of just seven years. Effects were observed,

however, when all younger and older males were combined, an age range of 39 years, possibly due the exaggerated nature of the differences between these two groups.

Previous cross-sectional studies that have reported tissue volume changes with age typically have a larger age range, for example a range of 15 years (Giedd, Snell, et al., 1996; Giedd, Vaituzis, et al., 1996), 29 years (Sowell et al., 1999), 24 years (Sowell, Thompson, et al., 2001), 81 years (Sowell et al., 2003) and 23 years (Tamnes et al., 2010). As recommended in chapter five, future research into this topic could replicate the current methodologies in a younger sample of drivers from a State in the US where the driving age is 14[36]. If employing a cross-sectional design such a study would have greater power to detect changes in brain structure between mid adolescence and the mid 20s because of a greater age range. This may give insight into whether some individuals take risks while driving due to individual differences in the rate of development of certain brain regions. Furthermore, concurrent DTI and VBM could be used to link risky driving behaviour to both regional GM development and changes in WM tracts that link cortical and subcortical GM regions.

Ideally, however, a longitudinal design would be adopted. As there is huge variability in brain size and structure within children and adolescents, and wide-ranging individual differences in the developmental trajectories of regions, large samples are needed in order for cross-sectional studies to detect human brain development (Giedd et al., 1999). Longitudinal studies have increased power to

[36] As mentioned in chapter five, learning permits can be obtained from the age of 14 from States such as; Arkansas, Iowa, Michigan, Montana and Wyoming (VDriveUSA, 2013)

detect changes due to development and so fewer participants are needed compared to cross-sectional designs (Giedd, Snell, et al., 1996). While there is strong evidence from concurrent longitudinal and cross-sectional studies to suggest that differences between older and younger groups are primarily due to age (Giedd et al., 1999), the validity of such cross-sectional findings is often called into question (Sowell et al., 2004). Cross-sectional VBM studies have been criticised for lacking the ability to detect valid "change" in brain structure with age, i.e. brain maturation, due to the difficulty in assembling comparable cohorts at different ages (Sowell et al., 2004). As high rescan reliability has been reported in MR images (Giedd et al., 1995), longitudinal methods are increasingly used in the developmental literature instead of the quicker, cross-sectional approach.

An issue of note with using a voxel-based structural analysis is that the boundary between grey and white matter is not so clearly defined (Annese, Pitiot, Dinov, & Toga, 2004). As the VBM approach assigns each voxel the probability of it being a certain tissue type, some WM voxels may be included in the analysis of GM volume, and vice versa. Using a large, cross-sectional adult sample, Sowell et al. (2003) investigated the diverging results that can be obtained by using different methods of segmenting the frontal lobe into its subregions. The study employed three segmentation techniques, manual tracing, semiautomatic ("Talairach boxes" volumetric method) and VBM. While all three methods showed that GM volume in the PFC as a whole decreased with age, some variation in the slope of regional GM decline was observed between methodologies. For example, in the manual tracing method, lateral and orbital frontal regions showed the most rapid GM decline with

age, but the semiautomatic and VBM approaches showed the strongest decline in lateral frontal and cingulate regions.

The authors concluded that semiautomated and VBM methods are no substitute for manual volumetry (Sowell et al., 2003). A manual approach, however, would present another set of potential limitations, as, without standardisation, it would be difficult to compare results across studies. The degree of expertise of the researcher segmenting the regions would greatly influence the results obtained and not every research group will be able to employ such an expert. The solution, therefore, is for the continued use of the current, standardised VBM techniques. Each new version of the VBM toolbox, distributed with updates of SPM, contains more advanced methods of segmentation (Ashburner & Friston, 2001). Each update improves the accuracy and efficiency of the segmentation for the benefit of future studies.

6.4.3 Conclusion

In conclusion, the present study provides preliminary evidence to suggest that the SMA may serve as a vulnerability marker for younger individuals who make more risky decisions while driving. This finding was unique to younger individuals but was common to both young males and females. This may be the first evidence to suggest that individual differences in risky driving tendencies may be linked to brain structure. With a cross-sectional study, however, it is difficult to determine whether different trajectories in SMA development may lead some individuals to engage in more risk-taking behaviours or whether risk-taking propensities have some influence on the development of this region. Furthermore, the current sample may have been insufficient in size to detect true, between groups

differences in brain volume. There may also have been other factors that impacted on brain volume in the current sample, such as engagement in bring drinking (Lisdahl et al., 2013), that were not controlled for. The limitation of such cross-sectional research may be addressed with longitudinal studies of brain development and risk-taking behaviour, or replications of the current methodology in a larger, cross-sectional sample, with the ability to control for more, potentially confounding variables.

7 Chapter Seven: General Discussion

The current thesis examined the relationship between risky driving and impulsivity. Male drivers and drivers under the age of 25 are overrepresented in both non-fatal and fatal collision statistics, in Ireland and internationally (OECD & ECMT, 2006; RSA, 2015c; Sarma et al., 2013; WHO, 2015). Therefore, the current study included only samples of young male and female drivers, in addition to an older male driver group. Furthermore, despite a tendency of the literature to generalise risk-taking to these groups as a whole, research suggests that not all males and younger drivers will take risks (Dryfoos, 1991; Sercombe, 2014). It is proffered that heightened impulsivity may be an individual difference factor that predisposes some individuals to drive riskily (Brandau et al., 2011; O'Brien & Gormley, 2013; Wickens et al., 2008).

To investigate whether drivers with a history of risk-taking were more impulsive that those without, the current study compared such individuals on various measures of impulsiveness, including an examination of the neural correlates of impulsivity. Drivers were classified as 'risky' if they had penalty points on their licence and/or had been culpable in a collision. Chapter two reported the validity of these criteria in differentiating between drivers likely to be risk takers or not. Using an online survey it was found that, with this classification method, risky drivers reported significantly more aberrant driving behaviours than safe drivers, providing support for the use of these criteria.

Survey respondents that met the criteria for risk, gender and age necessary to form the groups of interest for the study (males and females under 25 and males over 28) were recruited for participation in the main experiment. Various

measures of impulsivity were taken during this follow up session, including psychometric measures prior to scanning (Barratt's Impulsiveness Scale, BIS-11, and Kirby's Delay Discounting Questionnaire, DDQ) and three behavioural tasks while participants were scanned with fMRI (the Iowa Gambling Task, IGT, Monetary Incentive Delay task, MID, and Go/No Go task). Chapters four and five reviewed the results of these data; with chapter four devoted to the analysis of the psychometric and behavioural data and chapter five the results of the functional analysis. Furthermore, as MR images were also obtained from participants, chapter six explored potential differences in the volume of structures implicated in impulsive processes between groups.

The main findings of these chapters are summarised under three main hypotheses below; *hypothesis one:* a decline in collision involvement with age is due to declining impulsivity from adolescence to adulthood, *hypothesis two:* there are gender differences in impulsivity that may contribute to an overrepresentation of males in collision statistics, and, *hypothesis three:* risky drivers, regardless of age or gender, are more impulsive than safe drivers.

7.1 Hypothesis One: Impulsivity Declines with Age

7.1.1 Summary of results

While males overall are overrepresented in collision statistics, young males are even further overrepresented. The current study posited that high levels of impulsivity in younger males, compared to older, may contribute to this increased risk. No difference in the number of self-reported aberrant driving tendencies was observed between younger and older males, however, younger males were found

to be more impulsive on several of the psychometric and behavioural measures of the current study. Younger males, compared to older, scored higher on three of the six subscales of the BIS-11 and demonstrated a greater preference for smaller immediate rewards when choosing between them and larger, delayed rewards on Kirby's DDQ, consistent with previous research (da Matta et al., 2012; Löckenhoff et al., 2011; Steinberg et al., 2008). Older males had longer reaction times on certain conditions of the behavioural tasks. This is more likely to reflect normal age related changes in reaction times, rather than a decline in impulsiveness. No differences were found in the number of successful inhibitions made on the Go/No Go task, neither were differences in performance on the IGT or MID observed, in accordance with the findings of previous studies (Balodis & Potenza, 2015; Christakou, Brammer, et al., 2009; Geier & Luna, 2009; Liston et al., 2006). Therefore, other than differences in reaction time between younger and older males, no difference in performance on the behavioural tasks was observed.

Despite observing extensive grey matter (GM) volume decline in the striatum and prefrontal regions with age in males (consistent with finding by; Giedd, 2004; Giedd et al., 2006; Gogtay et al., 2004; Nunnemann et al., 2009; Raz et al., 2005; Sowell et al., 2003; Walhovd et al., 2011), functional differences between older and younger males were not found in these regions. In fact, the only region to demonstrate significant changes in activation with age (the hippocampus) did not decrease in volume with age (contrary to previous functional and structural findings by; X. Li et al., 2010; Pruessner et al., 2001).

Younger male drivers scored higher on the (lack of) self-control subscale of the BIS-11 than older males, suggesting that self-control increases with age. Scores on

this subscale also positively correlated with the intensity of the BOLD response in the supplementary motor area (SMA) when males made successful inhibitions on the Go/No Go task, compared to errors of commission. As no differences in the number of successful and unsuccessful inhibitions in this task were observed between groups, greater activation in this region, implicated in action monitoring, response preparation and cancellation (Aron, 2007; Ikeda et al., 1999; Whelan et al., 2012), could indicate that individuals with less self-control (i.e. scored high on this subscale) required a greater neural effort to inhibit their responses in this task. Furthermore, GM volume in the SMA declined with age, corresponding with an increased time to select from infrequent punishment decks on the IGT. Thus, while activation in the SMA did not differ between younger and older males, it appears to relate to impulsive-like traits and actions that have been observed to decline with age.

7.1.2 Theoretical implications

The above suggests that trait impulsivity (including impulsive choice traits) and certain indicators of impulsive action (i.e. faster reaction times) decline with age. Furthermore, a reduction in GM volume was observed in prefrontal regions implicated in the decline of impulsive action from adolescence to adulthood, likely reflecting normal maturational processes, rather than degeneration. Yet, no differences in activation were observed in these regions during an experimental task designed to measure an aspect of impulsive action; behavioural inhibition. Using a variation of the Go/No Go paradigm, Durston et al. (2006) found that activation in task-relevant regions became more focused with age. The current study did not replicate these activational findings and observed no difference in

performance on the Go/No Go task (as measured by the number of successful inhibitions made) between younger and older groups. This may be due to the age range of the 'younger' group in the current study, 18 to 24, compared to the nine to 11 year old age range employed by the Durston et al. (2006) study. Therefore, it is likely that, while aspects of impulsivity decline with age, coinciding with the development of the prefrontal cortex (PFC), an already functioning system of inhibition exists in individuals over 18 years of age.

According to Casey et al. (2008) and Steinberg (2008), behaviours that reflect impulsive choice tendencies, such as an inability to delay gratification and decision-making without consideration of the future consequences, are likely to occur in adolescence due to the immature PFC's inability to regulate reward-driven impulses. They posit that this is due to the early maturation of reward processing regions, such as the nucleus accumbens (NAcc), relative to the on going development of the PFC into the mid twenties, and is likely to result in inappropriate responses to rewarding stimuli, such as hyperactivation in some instances (as seen by; Ernst et al., 2005; Galvan et al., 2006) and hypoactivation in others (e.g. Bjork et al., 2004). The current study tested this hypothesis by comparing the neural responses of younger and older males to the rewarding stimuli of the IGT and MID tasks. No differences in activation were observed. Furthermore, striatal regions were observed to decline in GM volume at a similar rate to PFC regions with age, contrary to the expectation of Casey et al. (2008). As mentioned in chapter five, Casey et al. (2008), along with the others who laid the foundation for this theory (such as; Bjork et al., 2004; Ernst et al., 2005; Galvan et al., 2006), used younger adolescents than those of the current study

(approximately 12 to 17 years of age) and, yet, this theory is often generalised to risk-taking observed up to the age of 25, with the decline in risk-taking coinciding with the maturation of the PFC. As both prefrontal and striatal regions were observed to continue to mature from late adolescence to adulthood and neither demonstrated significant activational differences between younger and older males, the applicability of Casey et al. (2008)'s neurobiological theory to risk-taking in an *older* adolescent/young adult sample is called into question. Instead, increased risk-taking during this age (18 to 24) may be attributed to individual differences in impulsivity, including activational and structural differences, as will be discussed in hypothesis three below.

7.2 Hypothesis Two: There are Gender Differences in Impulsivity

7.2.1 Summary of results

In this study, younger males reported significantly more highway-code (HC) violations than females, while females were found to self-report making more errors and lapses of attention while driving than males (consistent with the findings of; Berdoulat et al., 2013; De Winter & Dodou, 2010; Reason et al., 1990). It was hypothesised that higher levels of impulsivity in males could explain why they are overrepresented in collision statistics (Sarma et al., 2013). In the current sample, only modest evidence was found to support this hypothesis. No gender differences were found on the BIS-11 or performance on the Go/No Go task, consistent with previous research (Cross et al., 2011; Patton et al., 1995). Furthermore, no differences in activation were observed and, while some small volumetric differences were found, only one of these has been previously implicated in impulsive processes, the orbitofrontal cortex (OFC).

The OFC has been found to activate in response to monetary rewards (e.g. Knutson et al., 2001) and is implicated in determining the appropriate response to a rewarding stimulus (Volkow & Baler, 2012). Greater OFC volume in females, despite larger overall brain size in males, may explain the few detected gender differences in psychometric and behavioural measures related to reward in the present study. For example, while no differences in IGT performance were found (consistent with the findings of; Bayard et al., 2011; Bechara et al., 2000), females took longer to select from decks B and D, which delivered infrequent, large punishments. Faster reaction times in males in this case may reflect a greater sensitivity to rewards, without consideration of the magnitude of the potential punishment that could occur. While previous research has not reported gender differences in performance on the MID (see meta-analysis by Balodis & Potenza, 2015), the present study found that males hit more of the target windows than females, which could indicate a greater motivation to obtain positive feedback on the task. Finally, contrary to previous research that suggested that females would discount future rewards to greater extent than males (Petry et al., 2002), young males in the current study chose more immediate rewards than females when the delayed rewards were small in magnitude. These findings combined provide limited evidence of different behavioural responses to hypothetical rewards and rewarding stimuli in younger males and females.

7.2.2 Theoretical implications

There is little to suggest that higher levels of impulsivity, as assessed by the current measures, contribute to a greater amount of deliberate risk-taking while driving in males, compared to females, as indexed by higher scores on the HC

violation subscale of the DBQ. Furthermore, slower reaction times in females, while potentially meaningful, are unlikely to relate to the greater number of error and lapse behaviours reported by females on the DBQ. Therefore, the current study was unable to identify differences between younger males and females that would extend so far as to explain the overrepresentation of males in collision statistics.

7.3 Hypothesis Three: Risky Drivers are More Impulsive Than Safe

7.3.1 Summary of results

Older male (29+), younger male (18 to 24) and younger female drivers were further divided into 'risky' and 'safe' driver groups based on their self-reported history of driving offences and collision involvement. Participants were classified as risky if they had been culpable in a collision and/or had penalty points on their driving licence and safe drivers were those with no penalty points who also had not been involved in a collision. When risky and safe participants in the main sample were compared, risky drivers reported deliberately breaking the law while driving (HC violations) to a greater extent than safe drivers, regardless of age or gender. Furthermore, when females were excluded, risky male drivers also reported significantly more aggressive tendencies. While this demonstrates a difference in aberrant driving between male and females, it primarily serves to justify the separation of these participants into discrete groups for the subsequent analysis of impulsivity in drivers. The main findings of this investigation are summarised below.

When given the choice between a smaller, immediate reward and a larger, delayed reward in Kirby's DDQ, the risky drivers of the current study chose the immediate reward more often than safe drivers. Correlations between the number of immediate rewards selected and the HC violations subscale of the DBQ also indicated that greater delay discounting tendencies were associated with more deliberate violations while driving. Furthermore, HC violations correlated with motor impulsivity on the BIS-11 and risky drivers scored significantly higher on the (lack of) self-control subscale. These findings suggest that drivers who take deliberate risks, such as exceeding the speed limit, are likely to act in the present, without consideration of future consequences and are motivated by immediate, rather than long-term gains.

This was also evident in young risky drivers' preference for decks in the IGT that delivered infrequent punishments (decks B and D). Despite an overall preference for infrequent punishment decks in both male and female young risky drivers, interactions between risk status and gender/age were observed for the intensity of activation in some regions when selecting from/getting feedback from these decks. When males were selecting from infrequent punishment decks, young risky males had significantly greater activation in the anterior cingulate cortex (ACC) than young safe males, but the opposite pattern was found in older males. Similarly, when participants won money from these decks, young risky males demonstrated greater activation in the insula than young safe males, while young safe females had greater activation than risky females in this region. Furthermore, when risky younger males lost money from frequent punishment decks (A and C), a greater response in the left inferior frontal gyrus (IFG) was observed when

compared to safe younger males. Differences in reward processing (activation in the OFC and striatum) were also observed in males during certain feedback conditions of the IGT, such as when they received feedback from deck B.

In the context of frequent and infrequent punishment decks, differences in the employment of these regions, implicated in the monitoring of feedback (left IFG; Walton et al., 2004), action selection (ACC; J. W. Brown & Braver, 2007; Carter et al., 1998; Carter & Van Veen, 2007), weighing the costs of a risky decision (insula; Burke et al., 2013) and reward processing (the striatum and OFC; Hampton & O'Doherty, 2007; Knutson & Cooper, 2005; Knutson & Wimmer, 2007; Krawczyk, 2002), suggest that risky and safe drivers may place a different saliency on frequent and infrequent punishment decks and, possibly, encode the risks associated with gains and losses from these decks differently. Furthermore, some of these differences were found to interact with age and gender, supporting the hypothesis that there are particular characteristics of young risky male drivers that may relate to their overrepresentation in collision statistics.

Further differences between risky and safe drivers in reward processing and frontal regions were observed in the MID task, despite no behavioural differences between these groups on the task. When all younger males and females were combined, young risky drivers demonstrated greater activation in the SMA than safe in certain conditions, while safe younger drivers had more activation in the ACC and SMA in others. Similar differences between risky and safe were observed when all males (younger and older) were combined, and when younger male drivers were examined separate to all other groups. Additionally, risky and safe younger male drivers differed in regions that have been found to be activated in

response to rewarding stimuli, such as the dorsolateral prefrontal cortex (DLPFC) and caudate (Knutson et al., 2001; Knutson et al., 2000; Nestor et al., 2010). However, one thing that was common to all three analytical models applied to the MID was differences between driver groups in the intensity of activation of the SMA, the implications of which will be discussed in greater detail below.

During the Go/No Go task, no differences in performance were found between risky and safe groups. Furthermore, no differences in activation between risky and safe drivers were observed in younger drivers, regardless of whether young males and females were combined or not. However, when risk groups were compared in all males, younger and older combined, safe males had a greater response in the SMA and left parietal lobule when making successful inhibitions, over errors of omission, than risky males (inferior parietal lobule implicated in modulating attention during cognitively demanding tasks; Krueger et al., 2007; and the SMA in response preparation; Whelan et al., 2012). Furthermore, also during this condition, there was an interaction in the DLPFC (thought to be responsible for response selection; Bunge, Dudukovic, et al., 2002; Rubia et al., 2001) such that young safe males had a significantly greater response than young risky males, while there was no difference between risky and safe older males. As there were no differences in the number of successful inhibitions made between groups, and neither successful inhibitions, nor errors of omission, involve a motor response, this finding suggests safe and risky males may employ different mechanisms when inhibiting behavioural responses. This could have implications for driver behaviour as a greater capacity for control over interfering thoughts and actions may make some males more resilient towards driving risk.

It has been said that, in the study of impulsivity, it is advantageous to combine fMRI methods with brain morphometry, as fMRI is limited to correlational analysis of the activation that may be associated with specific conditions in tasks measuring aspects of impulsiveness, while the analysis of GM volume is not restricted to certain paradigms or conditions (Schilling et al., 2013). As such, voxel based morphometry (VBM) was utilised in the present study to identify whether there were volumetric differences between risky and safe drivers in any of the regions implicated in impulsive processes. Safe younger drivers, independent of gender, were found to have significantly greater GM density in the SMA than young risky drivers but no significant differences were observed in older males after corrections for multiple comparisons were made. The SMA may play particular role in motor-inhibition (Aron, 2007), in mediating response selection (Whelan et al., 2012) and in the preparation of actions (Ikeda et al., 1999). As earlier summarised, activational differences in the SMA were observed between risky and safe drivers on certain conditions of the behavioural tasks. In addition to this, positive correlations between SMA activation and the HC and aggressive violation subscales of the DBQ were also observed in males. While no groups differed in their ability to inhibit responses to lure stimuli on the Go/No Go task, differences in the intensity of recruitment of the SMA may be a mechanism to compensate for the size of the SMA when preparing to make and inhibit behavioural responses on the tasks. If this is the case, disruption to this mechanism may result in an inability to compensate for this deficit, which could impact on performance in such an attentionaly-demanding task as driving. The above evidence may be combined to suggest that the function and structure of the SMA may be a vulnerability marker for risk-taking while driving.

7.3.2 Theoretical implications

Driving is a complicated task that involves the integration of several behaviours such as selecting and dividing attention, visuomotor integration, visuospatial interpretation and decision-making (Calhoun et al., 2002). Chapter one (section 1.5) reviewed the findings of studies that have investigated the regions of the brain that may be involved in the complicated process of driving, identifying several key regions. In summary, Uchiyama et al. (2003) found that, while the SMA and ACC were recruited when participants were passively viewing driving scenarios, greater activation in these regions was elicited when participants were actually actively involved in the task. When a driving scenario became more complex, Schweizer et al. (2013) observed increasing engagement of the DLPFC, thought to be involved with regulating executive functions such as attention, decision-making and multitasking behaviours (Christoff & Gabrieli, 2000). The function of the ACC and DLPFC, therefore, may be essential to safe driving, exerting top-down control over regions to direct behaviour towards certain goals and sub-goals and to reduce the effect of distractors, as has been observed in other research (Bunge et al., 2000; Kondo et al., 2004; E. E. Smith et al., 2001). Finally, Spiers and Maguire (2007) identified regions that may be particularly important in driving manoeuvres. The SMA, ACC and insula, along with other regions, activated when participants were swerving to avoid colliding with an obstacle and the pre-SMA was identified as a region crucial for initiating actions to stop a vehicle.

With the current criteria utilised to differentiate between drivers who are likely to be risk takers or not, significant differences in the function (in the case of the ACC,

insula, DLPFC and SMA) and structure (SMA) of the above regions suggested to be involved in the process of driving were found between groups. Data from the present study was unable to support the *first* hypothesis, that declining impulsivity with age explains the overrepresentation of younger drivers in collision statistics. However, in light of the current findings, there may be an underlying neurological basis for increased risk-taking in some individuals, such that differences in the function and structure of the neural correlates of impulsivity, in addition to differences in trait impulsivity, may contribute to risk-taking while driving. Thus, this study provides modest evidence to support the third hypothesis, that impulsivity may be an individual difference factor that contributes to heightened risk-taking in drivers. Furthermore, as some, but not all, differences interacted with either age or gender, there is reason to suspect that the extent to which impulsivity relates to collision involvement in young males, young females and older males will vary, which may contribute to the different prevalence of collision involvement in these groups.

In summary, the risky and safe drivers of the current study differed on psychometric measures of impulsivity, displaying greater discounting of the value of distant rewards and demonstrating a tendency to act without consideration of future consequences. This was also reflected in their performance on the IGT, with younger risky drivers preferring decks that delivered infrequent punishments to frequent, as evidenced by a greater number of selections from decks B and D. Furthermore, while no other behavioural differences were found, these groups differed significantly in the recruitment of regions implicated in both impulsive processes and the execution of successful driving manoeuvres. The presence of

activational differences in the absence of behavioural ones is useful for fMRI power, however, this can promote only speculation as to meaning behind such differences, and the extent to which these may relate to decision made while driving.

Therefore this study provides limited evidence that different neural mechanisms may be related to a tendency to take risks while driving. However, this evidence is supplemented by the structure of the SMA in young drivers, where those with a history of risk-taking while driving had less GM density in the SMA than those without, a region identified as being part of a network recruited when completing manoeuvres to avoid collisions. The combined functional and structural findings of the present study, however tentatively, provide preliminary evidence to suggest that a propensity towards problematic driving may be associated with underlying neural processes and brain structure.

7.4 Reducing Risk-taking in Drivers

Dryfoos (1991) proposed that risk-taking propensities can be addressed before problem behaviours emerge. In their review of existing interventions designed to reduce high risk behaviour, Dryfoos (1990) found that successful interventions had the following elements in common; individual attention (a consistent adult who may provide counselling, mentoring or other one-to-one support), involvement of parents, school involvement (educational and non-educational interventions, e.g. after school activities) and community-wide approaches. Furthermore, Dryfoos (1990, 1991) urged that no one intervention is 100% effective, and that combinations of approaches have the most potential in addressing problems before they arise. As such, the successful reduction of risky

driving in individuals who may be predisposed is likely to require a combination of programmes, for example Graduated Driver Licence (GDL) schemes in conjunction with specific interventions for reducing impulsivity.

GDLs are employed by many countries as a method of protecting drivers from future risk from the time they begin to learn. They involve the cooperation of parents, police, insurance companies, policy makers and other government bodies to ensure their success (Williams, 2006). Certain restrictions are placed on learner and novice drivers that are thought to allow them to obtain on-road driving experience under conditions of low risk, and keeping them out of high risk situations (Williams, 2006). High risk behaviours such as speeding and driving while under the influence of alcohol are just some of the targets of GDL schemes. To reduce these behaviours, the World Health Organisation (WHO) recommended that learner and novice drivers be prohibited from driving on roads with high speed limits, such as motorways, and have a lower legal blood alcohol limit (BAC) (WHO, 2012a). In their 2006 report, the European Conference of Ministers of Transport (ECMT) suggested that this BAC be set to a maximum of 0.02g/dl in learner and novice drivers (OECD & ECMT, 2006). Further suggestions by this report included placing restrictions on driving at night with young passengers, requiring at least 50 (and optimally 120) hours of pre-licence driving, having an minimum age of licensure of 18 years and making it easier for novice drivers to lose their licence by increasing the penalty points associated with driving offences, such as speeding. The OECD and ECMT (2006) report also notes the importance of the advertisement campaigns that should accompany changes to policy such as

those mentioned above, as they have the potential to shape the attitudes of younger individuals who have not yet begun to drive.

GDLs have been reported to have a positive effect on learner and novice driver safety, reducing the number of collisions and associated injuries (Williams, Tefft, & Grabowski, 2012). For example, since their introduction, GDLs are thought to have contributed to an approximate reduction of collision involvement in young drivers by 20 to 40% in the United States (Shope, 2007). It has been observed that the more elements that comprise the GDL programme, i.e. the more restrictions placed on learner and novice drivers, the greater their effectiveness (Chen, Baker, & Li, 2006; Williams et al., 2012). However, despite their potential to save lives, these measures have encountered some resistance prior to their implementation (OECD & ECMT, 2006). Thus, GDLs are not identical across countries and the effectiveness of the individual elements of these programmes that policy makers 'pick and choose' from have not been sufficiently assessed (Ferguson, 2003). Furthermore, rather than targeting the factors that directly lead to risk-taking, GDLs are used to keep learner and novice drivers out of hazardous situations (Ferguson, 2003). Therefore, another element that could be added to the GDL programmes is the inclusion of courses or other interventions that may equip drivers to resist impulsive tendencies and improve attitudes towards safe driver behaviour (Keating, 2007).

7.4.1 Reducing impulsivity in drivers

Such an intervention was proposed and tested by Paaver et al. (2013), who wanted to identify whether interventions targeting impulsivity could decrease risky driving in impulsive people or simply make non-impulsive people behave

even more safely. The intervention was delivered in schools to adolescents that had not yet begun to learn to drive. It consisted of a 45 minute lecture and 45 minutes of group work. A control group was included, where these individuals did not attend either the lecture or group work sessions and received no alternative training.

At the beginning of the session participants completed a booklet of questionnaires, which they collectively named the Adaptive and Maladaptive Impulsivity Scale (AMIS). The AMIS consisted of the BIS-11 (Patton et al., 1995), items from Dickman's Functional and Dysfunctional Impulsivity Scales (Dickman, 1990) and from the Revised NEO Personality Inventory (NEO-PI-R; Costa & McCrae, 1992). During the session they were encouraged to go back over the booklet to mark situations that were of particular relevance to them in order to help them to evaluate their own impulsiveness. The lecture explained the concept of impulsivity to the attendants, differentiated between different types of impulsivity and outlined how they can lead to risky behaviours, such as risky driving. Self-monitoring and self-regulation techniques were also taught, adapted from cognitive behavioural therapy methodology (e.g. conceptualising risky behaviour in the framework of a cognitive model including a situation, automatic thought and reaction), in order to help these adolescents be aware of their own impulsivity and the potential situational triggers of impulsive behaviour.

After a year, the effectiveness of the intervention was assessed by comparing the number and type of driving offences and collisions accrued by participants (obtained from police and insurance company databases) in the control and intervention groups. The intervention group was further divided into groups with

low, medium and high impulsivity to identify whether the effectiveness of the programme depended on the baseline impulsiveness of the participants. These groups were based of the interquartile ranges of total BIS-11 score. The control group was not subdivided. Paaver et al. (2013) found that the intervention was successful in reducing future speeding offences, in that, compared to the control group, fewer adolescents with low to medium baseline impulsivity who received the intervention were caught speeding after a year. However, there was no difference between the number of subsequent speeders in the high impulsivity intervention group compared to control. Furthermore, no differences in the number of drink driving offences were observed between groups, which may be indicative of the limitations of this intervention, but also may be because a year follow up was insufficient time to measure a rarely occurring event such as drink driving.

In summary, while this method was successful in reducing risk in some individuals, its effects did not carry across all types of risky driving and was not effective on individuals with heightened impulsiveness. The intervention may have been more effective if it was reinforced over a number of occasions, rather than a once off session. It is also possible that different results would have been observed if the baseline impulsiveness of the control group was considered. The efficacy of this intervention should be reassessed taking these limitations into consideration. An alternative intervention, cognitive-training, is discussed below.

7.4.2 The potential application of cognitive-training

A potential candidate for reducing the effects of heightened impulsivity could be cognitive-training. The human brain is highly plastic, where interactions with the

environment (e.g. learning) result in rapid cortical functional reorganisation (Dehaene et al., 2010) and the ability to develop compensatory mechanisms (Zanto, Chadick, Satris, & Gazzaley, 2013). Cognitive, or 'brain' training software is thought to have the ability to improve cognitive function (Mishra & Gazzaley, 2014). While not all paradigms have proven successful in this venture, Mishra and Gazzaley (2014, p. 1) claimed that a "blanket statement that all cognitive training is ineffective" is unfair. Primarily used to benefit older adults, the effects of certain brain-training programs have even been found to transfer to untrained cognitive abilities (Anguera et al., 2013; Bherer et al., 2005; Kramer et al., 1999; Mishra & Gazzaley, 2014; Schumacher et al., 1999). Of particular relevance to safe driving is the observed positive effect of brain-training on one's ability to reduce the costs associated with distractors (Erickson et al., 2007) and in improving multitasking capabilities (Anguera et al., 2013; Mishra & Gazzaley, 2014).

The ability to exert control over thoughts and behaviours is critical for successful, goal-directed interaction with a complicated environment (Botvinick et al., 2001). Multitasking is a process that allows simultaneous goals to be accomplished with limited interference (Dux et al., 2009). Anguera et al. (2013) developed the paradigm, 'NeuroRacer', and tested its ability to improve multitasking and other cognitive abilities over a series of experiments. With NeuroRacer, individuals navigate a driving scenario with a control, avoiding obstacles. Three conditions of varying complexity are utilised; sign only (testing perceptual discrimination ability; participants press a button whenever they see a certain target stimulus), drive only (visuomotor tracking task; participants use a joystick to keep the car in the centre of the road) and sign and drive (multitasking condition where

participants have to navigate the car and respond when they see the target stimulus). With no training on the task, the cost of adding a second, multitasking, component was observed to increase with age, i.e. multitasking capabilities declined with age. However, a second experiment demonstrated that individuals who trained on the multitasking condition, compared to those who trained on the individual components of the task separately, significantly improved their multitasking abilities. The benefits of this multitasking training also transferred to other, untrained, cognitive skills such as improved working memory (seen on a delayed recognition task, with and without distraction; Clapp, Rubens, Sabharwal, & Gazzaley, 2011) and sustained attention (assessed with the Test Of Variables of Attention, TOVA; L. M. Greenberg & Waldman, 1993), while other measures trended towards significance; dual-tasking, useful field of view, and change detection task. Furthermore, electroencephalogram (EEG) readings showed greater activation of a long-range network between frontal and posterior brain regions in the group that received multitasking training, a network associated with cognitive control capabilities, such as sustained attention. All of these benefits persisted to a six-month follow up.

This evidence suggests that training on programs targeted at improving cognitive processes, such as resisting the effects of distractors, may have the potential to better equip individuals to deal with the attentional demands of driving and protect against the intrusion of impulsive thoughts. However, longitudinal research would be needed to assess the validity and reliability of such measures and their relation to real world driving behaviours (Mishra & Gazzaley, 2014).

7.5 Limitations and Recommendations

7.5.1 Classification of risky drivers

While care was taken to validate the criteria used to separate risky and safe drivers, it was possible that some individuals may have been misclassified. For instance, participants with risky driving tendencies may have been assigned to the safe group because they had never been collision involved or had not received any penalty points by the date of testing. It is also possible that safe individuals may have been collision involved due to lack of skill, rather than deliberate risk-taking, which meant they would have been classified as risky with the current criteria. This is more likely to apply to the younger drivers of the study, as these would have had less exposure to situations where a collision could have occurred or where the police could have caught them in the act of a traffic violation. Furthermore, while the initial survey data demonstrated the ability of the current criteria to differentiate between drivers likely to be risk takers or not, the participants recruited for the main study only differed on one of the subscales of the DBQ, the HC violations subscale, not all four, as was observed in a larger sample of drivers. It could be the case that a larger number of participants are needed to detect this effect, however it may also indicate that there were misclassifications in sample of the main study. More objective measures of driving tendencies should be useful in future studies that wish to differentiate between risky and safe drivers, such as in-car 'black box' device, which, as mentioned in chapter two, record speed, harsh braking, fast lane changing, collision involvement and other types of reckless manoeuvres. In addition to this, frequency of engagement in other risk behaviours, such as alcohol and drug use,

should be recorded to determine whether results can be limited to a subset of individuals who take risks while driving, or whether they may apply to those likely to engage in everyday risk-taking in general.

7.5.2 Measuring impulsivity

As discussed in chapter four, in the study of impulsivity it is advantageous to use a combination of psychometric and behavioural measures (Cyders & Coskunpinar, 2011, 2012; Gomide Vasconcelos et al., 2014). However, it is also important to note that many different measures are employed by the literature. While there is some overlap between measures, many assess discrete aspects of impulsiveness, such as response inhibition, delay of gratification and impulsive decision-making (Gomide Vasconcelos et al., 2014). Just by changing the measures used in a study, different results can be obtained (Coskunpinar et al., 2013; Cross et al., 2011; MacKillop et al., 2011; Simmonds et al., 2008; Wright et al., 2014). Little has been done to identify an optimal combination of measures for the comprehensive study of the full breadth of impulsivity. Until this is achieved, caution should be taken not to generalise findings beyond the specific component of impulsivity being measured with the particular task or questionnaire of choice (Cyders & Coskunpinar, 2011; G. T. Smith et al., 2003).

The tasks utilised in the current study had all been found by previous research to differentiate between healthy controls and clinical populations, either on a behavioural or neurological level, or both. However, as the current sample comprised entirely of individuals that would be classified as 'non-clinical', as they had no history of neurological, psychiatric or psychological illness, and instead

only differed based on their driving history, the ability of these measures to detect differences was unknown.

As such, within the present data, the IGT was the only behavioural measure that demonstrated meaningful, significant differences between risky and safe drivers. Even so, this difference was found by analysing the IGT in an unconventional way, the number of selections from infrequent punishment decks (a method increasingly adopted by the literature, for examples see; Adida et al., 2011; Lin et al., 2007; Singh & Khan, 2009). Furthermore, the IGT highlighted certain functional differences between these groups, but only in males. This provides some support for the use of the IGT in future research comparing decision-making strategies (i.e. behaviourally) and mechanisms (i.e. functional processes) in 'healthy', but risk-taking, groups. Any such study should consider comparing the number of selections from frequent and infrequent punishment decks, in addition to the traditional method of analysing performance on the IGT.

Furthermore, while participants did not differ in their performance on the MID, consistent with previous research (Balodis & Potenza, 2015), this measure was the most sensitive of the three tasks to functional differences between groups. Therefore, it is recommended that future research utilise the MID when comparing the function of reward processing and prefrontal regions between groups of interest.

No difference in the number of commission errors made on the Go/No Go task were observed (contrary to previous research in aberrant drivers; O'Brien & Gormley, 2013) and no differences in activation, when older males were excluded. In the O'Brien and Gormley (2013) study, the 'offender' group was comprised of

individuals who had been caught speeding. This meant that they had a very homogenous sample, which may have increased their power to detect inhibitory differences when comparing this group to their control. In contrast, the risky group of the current study used combined criteria for risk; collision culpable and/or the accumulation of penalty points. Furthermore, participants differed in the types of collisions that they had been culpable in, were awarded penalty points for different offences[37] and some had both penalty points and had been culpable in a collision. Different inhibitory processes may underline each of these offence types. Therefore, the Go/No Go task may only be suitable for use in future studies that have a single criterion to differentiate between risky and safe drivers.

Three psychometric measures were also used, the BIS-11, Kirby's DDQ and the Manchester DBQ. While the DBQ was useful when validating the criteria used to differentiate between risky and safe participants in the initial survey sample, risky and safe groups in the main study did not differ on all subscales. This may have been due to errors in sampling (i.e., misclassification) or sample size, indicting that the DBQ may not be as reliable when used in smaller samples. As previously mentioned, more objective measures of driver behaviour should be used in future research.

While the BIS-11 has been used to study impulsivity in drivers (e.g. Jakubczyk et al., 2013; O'Brien & Gormley, 2013), these studies tend to either compare groups on the second order subscales (i.e. attention, motor and non-planning) or based on their total score on the BIS-11, which is not sensitive to the subtypes of

[37] While the majority had accrued penalty points for speeding, there was some variation in the types of offences participants had

impulsiveness that these groups may differ on. The current study utilised the first order subscales (i.e. attention, cognitive instability, motor, perseverance, self-control and cognitive complexity) and found that they better identified the discrete aspects of impulsiveness that may differ between individuals with and without a history of aberrant driving behaviours. Future research could concentrate on the three, first order subscales of the BIS-11 that appeared to relate to aberrant driving (i.e. the cognitive instability, motor and self-control subscales), which differentiated between risky and safe drivers and significantly correlating with self-reported aberrant driving tendencies on the DBQ. Alternatively, another measure of impulsivity could be employed to capture associations between driving behaviour and other facets of impulsivity, such as impulsivity under hot and cold emotional contexts (Cross et al., 2011). This could be achieved with the UPPS (the 'Urgency, Premeditation, Perseverance and Sensation seeking' scale; Whiteside & Lynam, 2001), whose subscales have also been found to correlate with the DBQ (e.g. Berdoulat et al., 2013; Pearson et al., 2013).

The psychometric measure that demonstrated the greatest capacity to differentiate between risky and safe drivers was Kirby's DDQ, contrary to what was expected, based on previous research (e.g. Teeters & Murphy, 2015). While these groups differed across the board on this measure, further evidence of the role of delay discounting and risk-taking while driving was observed in younger drivers, where a significantly greater proportion of young risky drivers were classified as having 'impaired' discounting of future rewards, i.e. they were found to place a value on immediate rewards to the degree that has been previously seen

in addicted populations. Therefore Kirby's DDQ was the most sensitive psychometric measure to a history of aberrant driving, particularly in younger individuals, and should be included in any future investigation of the contribution of impulsivity to aberrant driving.

7.5.3 Age range of the current sample

Neuroimaging of the structure and function of the brain in suspected risky and safe drivers was conducted in the current study in an effort to determine the extent to which Casey et al. (2008)'s theory of neurological development applied to a propensity towards risky driving. While evidence of differences in reward processing and executive functions were observed between groups, in addition to a difference in structure, the applicability of Casey et al. (2008)'s theory in the current context was not supported in these data. As discussed in greater detail in chapters five and six, this may have been due to both the minimum age of participants and the age range used. Studies of the development of the structure and function of the adolescent brain typically have used adolescent samples within the approximate range of 12 to 17 years of age (e.g. Galvan, 2010; Giedd, 2004; Gogtay et al., 2004; Sowell et al., 2003), whereas the younger participants in the current study were aged between 18 and 24. Furthermore, larger age ranges could be used, for example a range of 29 years (Sowell et al., 1999), as these exaggerate the differences between groups, making it easier to detect significant effects in cross-sectional samples. Therefore, it is recommended that future research replicate the current methodologies in a country with a younger age of licensure, such as the United States, for example, where the legal driving age is as

young as 14 in some States[38]. This would give an age range of ten years (ages 14 to 24), which spans the period of time for which drivers are most at risk of being involved in a collision. Despite this, the ability of the current data to demonstrate differences between risky and safe drivers within the age range of 18 to 24 was a strength and, while Casey et al. (2008)'s theory of neurobiological development during adolescence was not supported in this older sample, does provide evidence of particular structural and functional vulnerabilities in the brain that could relate to a propensity towards increased risk-taking while driving.

7.5.4 Longitudinal research

As there is huge variability in brain size and structure, and wide-ranging individual differences in the developmental trajectories of regions, it has been noted that large samples are needed in order for cross-sectional studies to detect human brain development (Giedd et al., 1999). Ideally, therefore, this increased age range would be combined with a longitudinal design, increasing the power to detect the relationship between changes in the brain and subsequent, real world risk-taking behaviours (Giedd, Snell, et al., 1996). Furthermore, there is evidence to suggest that a more accurate representation of a construct, such as impulsivity, would be made if psychometric and behavioural measurements are taken across several time-points (Gomide Vasconcelos et al., 2014). Young drivers could be followed every two years during adolescence and young adulthood (i.e., until the age of 24), and less frequently thereafter, until approximately 35 years of age. Rather than generalising from cross-sectional research, this approach would allow

[38] Such as Alaska, Arkansas, Idaho, Iowa, Kansas, Michigan and Montana

researchers to map the changes in brain structure and function with age, along with developments in trait and behavioural impulsivity. If these methodologies are combined with objective measurements of driving behaviour they may have the potential to explain why younger individuals, and young males in particular, are significantly overrepresented in both ordinary and fatal collision statistics.

7.6 Summary and Overall Conclusions

The current study corroborated previous findings that trait impulsivity, and certain behavioural aspects of impulsiveness, decline with age from late adolescence to adulthood, coinciding with extensive prefrontal maturational changes. Despite this, the present study was unable to replicate the activational differences in prefrontal and reward processing regions that have been reported in previous studies that compared younger adolescents to adults. Furthermore, prefrontal and striatal regions were both observed to mature (as indexed by a decline in GM volume) at a similar rate from older adolescence to adulthood, contrary to expectation. Hence, there was no evidence to suggest that the overrepresentation of younger individuals in collision statistics was due to an imbalance between the development of reward processing and prefrontal regions.

Gender differences in impulsivity were also examined in an effort to determine why males are also overrepresented in collisions. Females were slower to respond on several conditions of the behavioural tasks, which may explain why they also reported making more errors and lapses while driving. Despite this, the current data were unable to support the hypothesis that males are involved in more collisions than females due to higher levels of impulsiveness.

However, in a comparison between drivers with and without a history of risky driving, the present study found significant differences in the function (e.g. the ACC, insula, DLPFC and SMA) and structure (SMA) of regions that have been identified as part of the driving network, while also being implicated in processes related to impulsivity, such as behavioural inhibition and impulsive decision-making. These results, while preliminary and exploratory in nature, are further corroborated by the psychometric and behavioural findings, which demonstrated a greater preference for immediate gratification and a lack of consideration of future consequences in risky drivers. These findings tentatively suggest that impulsivity may be an individual difference factor that contributes to heightened risk-taking in drivers.

GDLs involving more restrictions to leaner and novice drivers have potential for reducing the frequency of collision involvement in these high-risk individuals (Chen et al., 2006; Williams et al., 2012). However, as these programmes protect drivers by keeping them out of hazardous situations (Ferguson, 2003), an additional intervention that directly targets impulsiveness could be added to help drivers to respond appropriately should they find themselves in a risky scenario (Keating, 2007). Previously proposed interventions to reduce the impact of impulsivity on driving have not proven effective on individuals with high levels of impulsiveness (Paaver et al., 2013), therefore, alternative approaches need to be considered. One such alternative could be brain-training games, which have been observed to improve multitasking capabilities (Anguera et al., 2013; Mishra & Gazzaley, 2014), reduce the costs associated with distractors (Erickson et al., 2007) and have been found to have benefits that transfer to other, untrained

cognitive abilities (Anguera et al., 2013; Bherer et al., 2005; Kramer et al., 1999; Mishra & Gazzaley, 2014; Schumacher et al., 1999). Future research could examine the efficacy of brain-training as a way of providing individuals with the tools to compensate for heightened impulsivity, utilising a longitudinal design that employs psychometric, behavioural and neuroimaging techniques to study changes in the various facets of impulsivity with age from young adolescence to later adulthood. Within such studies, developmental processes should be related to real-word driving behaviours as a more objective method of measuring risk-taking propensities while driving.

While many have posited the link between brain development, impulsivity and risky driving, a review of the literature to date revealed no other studies directly measuring how potential differences in psychometric or behavioural impulsivity between drivers may relate to underlying differences in the structure and/or function of the brain. As a result, many hypotheses needed to be addressed by the current study, necessitating the application of very stringent corrections for multiple comparisons. As such, it should serve as a preliminary investigation and potentially lays the groundwork for more focused studies and longitudinal work in the future.

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9 Appendices

9.1 Appendix A: Letter of ethical approval

F.A.O. Georgia O'Callaghan

School of Psychology Research Ethics Committee

12 November 2013

Dear Georgia,

Following receipt of amendments, I am pleased to inform you that your application entitled "Impulsiveness and aberrant driving behaviours" has been approved by the School of Psychology Research Ethics Committee.

Yours sincerely,

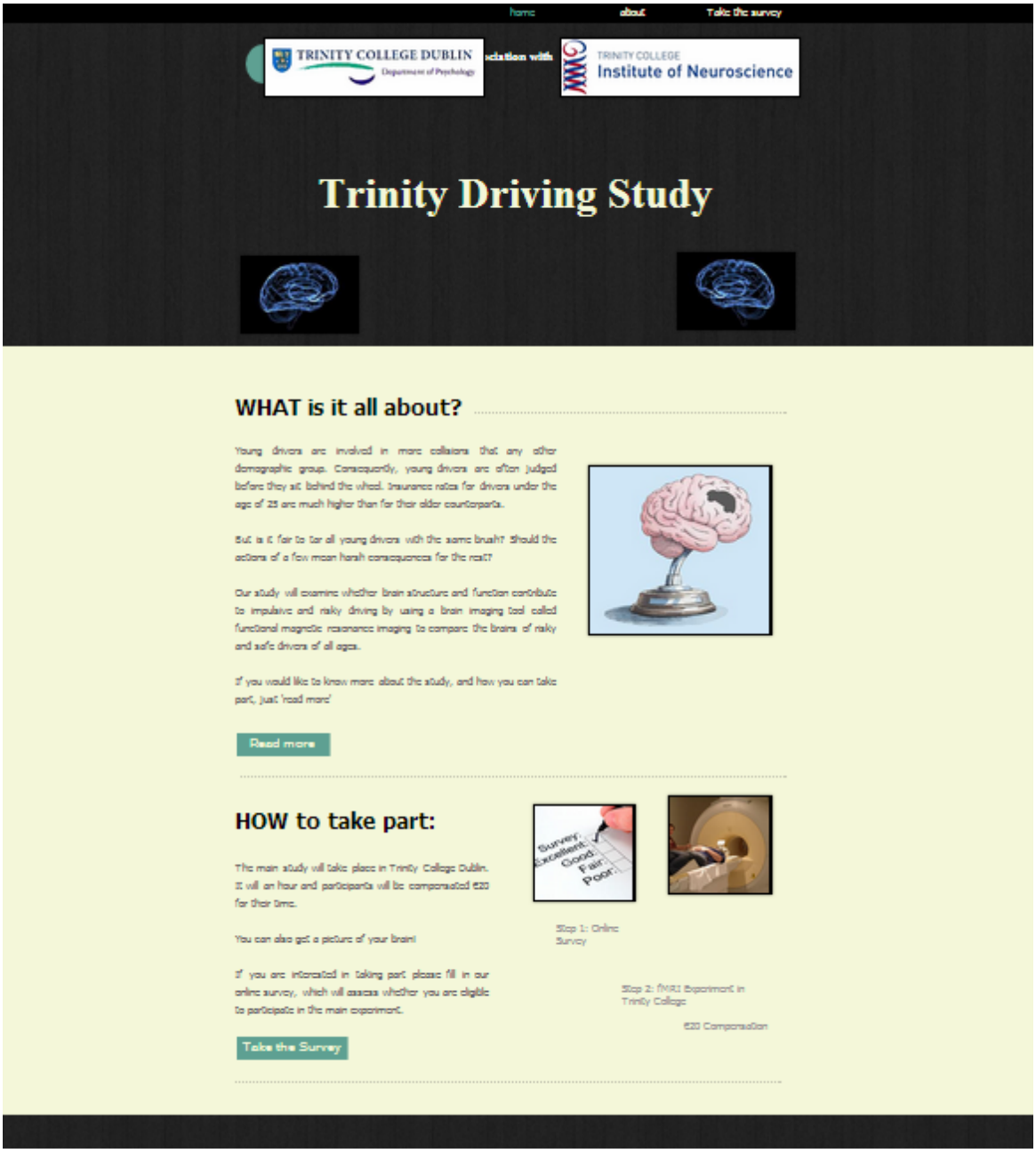
Richard Carson
Chair,
School of Psychology Research Ethics Committee

SCHOOL OF PSY
Arás an Phiarsaig
Trinity College
Dublin 2

9.2 Appendix B: Recruitment website

- **Main page**

Screen shot:



Content:

WHAT is it all about?

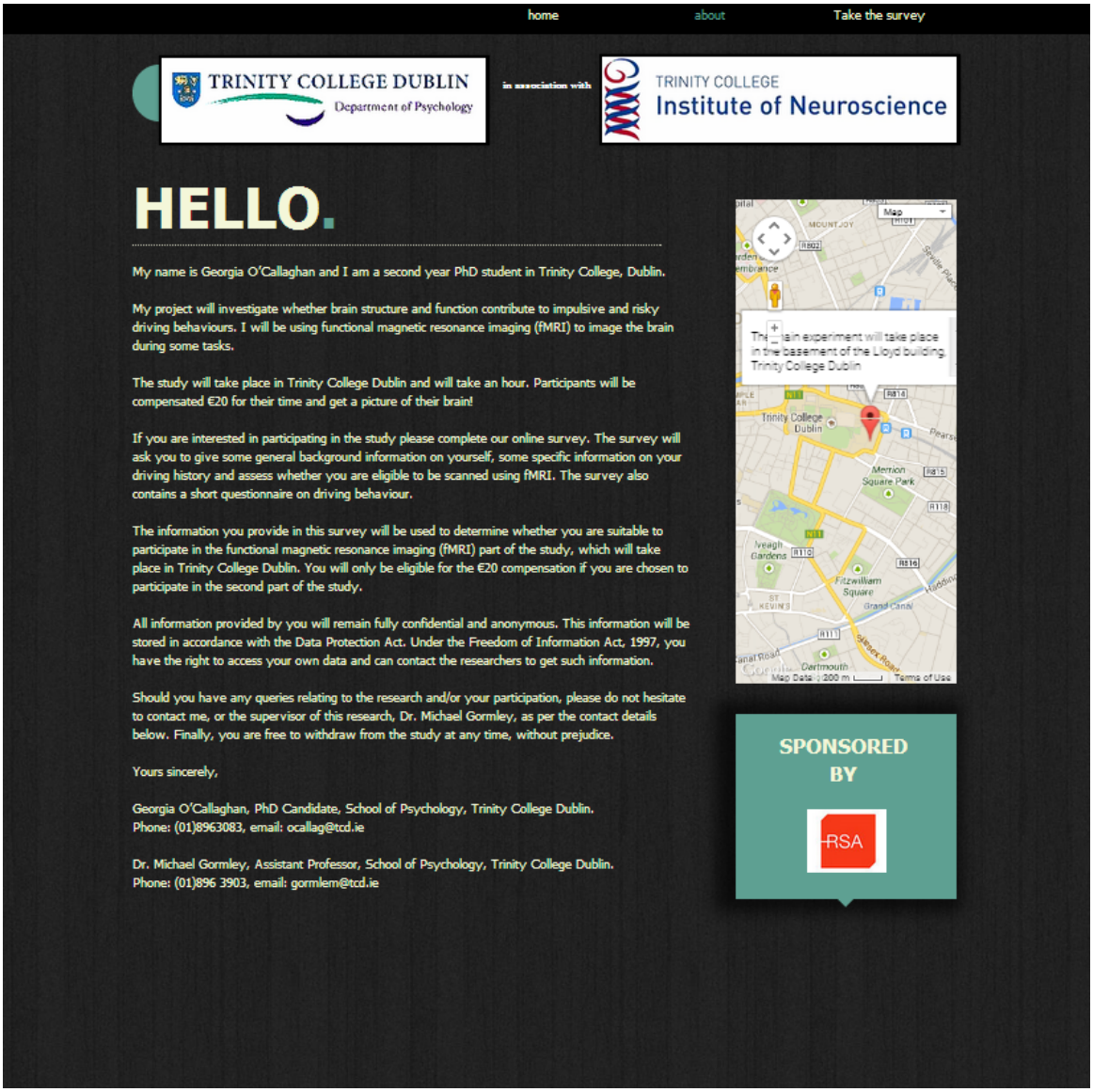
Young drivers are involved in more collisions that any other demographic group. Consequently, young drivers are often judged before they sit behind the wheel. Insurance rates for drivers under the age of 25 are much higher than for their older counterparts. But is it fair to tar all young drivers with the same brush? Should the actions of a few mean harsh consequences for the rest?

Our study will examine whether brain structure and function contribute to impulsive and risky driving by using a brain imaging tool called functional magnetic resonance imaging to compare the brains of risky and safe drivers of all ages.

If you would like to know more about the study, and how you can take part, just 'read more'

- **'About' page:**

Screen shot:



Content:

My name is Georgia O'Callaghan and I am a second year PhD student in Trinity College, Dublin.

My project will investigate whether brain structure and function contribute to impulsive and risky driving behaviours. I will be using functional magnetic resonance imaging (fMRI) to image the brain during some tasks.

The study will take place in Trinity College Dublin and will take less than an hour. Participants will be compensated €20 for their time and get a picture of their brain!

If you are interested in participating in the study please complete our online survey. The survey will ask you to give some general background information on yourself, some specific information on your driving history and assess whether you are eligible to be scanned using fMRI. The survey also contains a short questionnaire on driving behaviour.

The information you provide in this survey will be used to determine whether you are suitable to participate in the functional magnetic resonance imaging (fMRI) part of the study, which will take place in Trinity College Dublin. You will only be eligible for the €20 compensation if you are chosen to participate in the second part of the study.

All information provided by you will remain fully confidential and anonymous. This information will be stored in accordance with the Data Protection Act. Under

the Freedom of Information Act, 1997, you have the right to access your own data and can contact the researchers to get such information.

Should you have any queries relating to the research and/or your participation, please do not hesitate to contact me, or the supervisor of this research, Dr. Michael Gormley, as per the contact details below. Finally, you are free to withdraw from the study at any time, without prejudice.

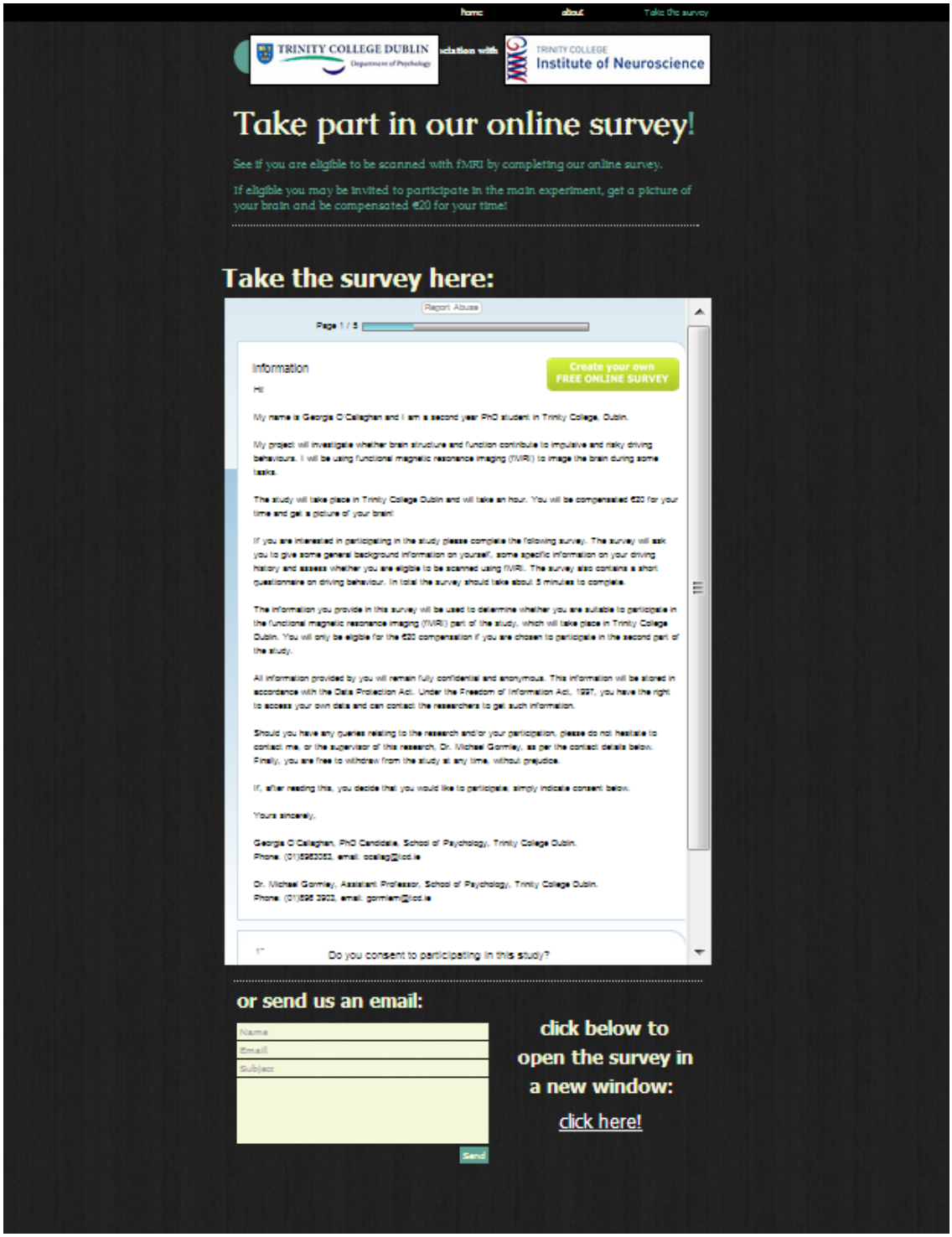
Yours sincerely,

Georgia O'Callaghan, PhD Candidate, School of Psychology, Trinity College Dublin. Phone: (01)8963083, email: ocallag@tcd.ie

Dr. Michael Gormley, Assistant Professor, School of Psychology, Trinity College Dublin. Phone: (01)8963903, email: gormlem@tcd.ie

- **'Take the survey' page:**

Screen shot:



Content:

Take part in our online survey!

See if you are eligible to be scanned with fMRI by completing our online survey.

If eligible you may be invited to participate in the main experiment, get a picture of your brain and be compensated €20 for your time!

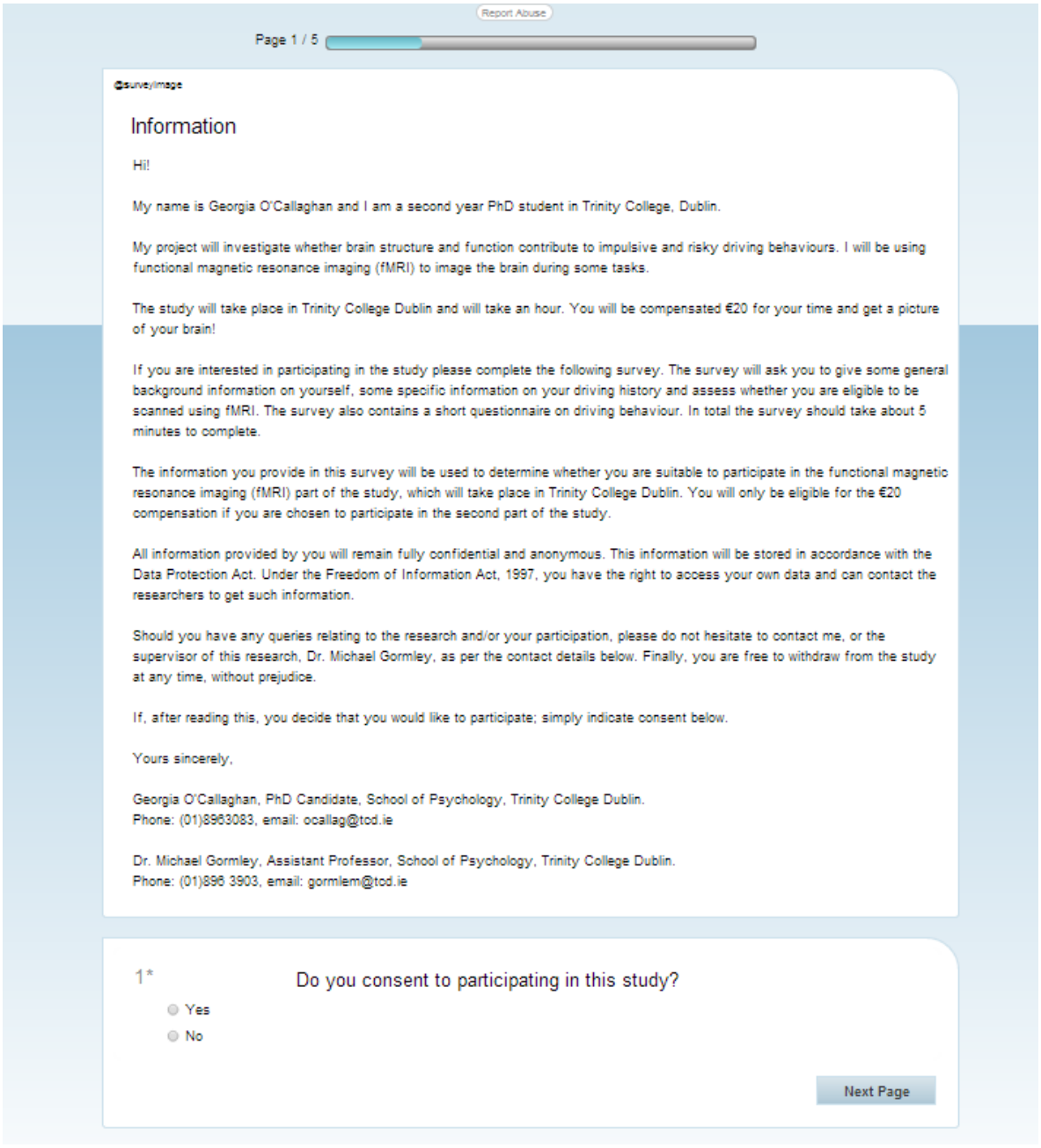
Direct link to the online survey;

http://kwiksurveys.com/s.asp?sid=u6vnt3mwo53104a224817

Or email Georgia at; ocallag@tcd.ie

9.3 Appendix C: Online survey

Sample of the information, consent, MRI screening and debriefing pages of the online survey:



Report Abuse

Page 4 / 5

@survey/image

MRI eligibility

Are you eligible to be scanned using magnetic resonance imaging (MRI)?

19* Please indicate whether you have any of the following;

	Yes	No	Decline to answer
Any type of electronic, mechanical, or magnetic implant?	○	○	○
Cardiac pacemaker?	○	○	○
Aneurysm clip?	○	○	○
Implanted cardiac defibrillator?	○	○	○
Neurostimulator?	○	○	○
Biostimulator?	○	○	○
Any type of internal electrode or wire?	○	○	○
Cochlear implant?	○	○	○
Hearing aid that cannot be removed?	○	○	○
Implanted drug pump (eg, insulin, Baclofen, chemotherapy, pain medicine)?	○	○	○
Halo vest?	○	○	○
Spinal fixation device?	○	○	○
Spinal fusion procedure?	○	○	○
Any type of coil, filter, or stent?	○	○	○
Any type of metal object (eg, shrapnel, bullet, BB)?	○	○	○
Artificial heart valve?	○	○	○

Any type of ear implant?	○	○	○
Penile implant?	○	○	○
Artificial eye?	○	○	○
Eyelid spring?	○	○	○
Any type of implant held in place by a magnet?	○	○	○
Any type of surgical clip or staple?	○	○	○
Any IV access port (eg, Broviac, Port-a-Cath, Hickman, Picc line)?	○	○	○
Medication patch (eg, nitroglycerine, nicotine)?	○	○	○
Shunt?	○	○	○
Artificial limb or joint?	○	○	○
Tissue expander (e.g., breast)?	○	○	○
Removable dentures, false teeth, or partial plate?	○	○	○
Diaphragm, IUD or similar?	○	○	○
Surgical mesh?	○	○	○
Body piercings that cannot be removed?	○	○	○
Wig/hair implants?	○	○	○
Tattoos or tattooed eyeliner?	○	○	○
Do you suffer from claustrophobia?	○	○	○
Are you currently pregnant?	○	○	○

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Report Abuse | Kwik Surveys

Report Abuse

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surveyimage

Debriefing

Thank you for completing the survey!

The details you have provided us will help us assess your eligibility to participate in the main experiment, which will take place in Trinity College Dublin. We will be examining the brain structure and function of younger and older drivers in an effort to determine whether they may influence impulsive and risky driving behaviours. We will be using magnetic resonance imaging (MRI) to do this. MRI is a type of safe, non-invasive brain imaging method which uses a strong magnetic field to take pictures of the brain. You will not be able to feel the magnetic field. For your health and safety you will not be eligible to participate in the main experiment if you met any of the exclusion criteria listed in this survey. This is because there are additional risks for individuals entering the scanner that meet any of these criteria.

If you did not meet any of the exclusion criteria and you are interested in taking part in the main experiment please enter your email address in the box below. If you give your email address you may be contacted by one of the researchers to arrange a date and time, at your convenience, to participate in the main experiment. Scanning for this experiment will take place in the Trinity College's Institute of Neuroscience, located in the Lloyd Building. Detailed directions will be given at a later date. Testing will take an hour and you will be given regular breaks. If selected to participate in this part of the study you will be compensated €20 for your time.

Please be assured that all information provided by you will remain anonymous. Should you have any queries relating to the research and/or your participation, please do not hesitate to contact me, or the supervisor of this research, Dr. Michael Gormley, as per the contact details below.

Georgia O'Callaghan, PhD Candidate,
School of Psychology, Trinity College Dublin
Phone: (01)8963083, email: ocallag@tcd.ie

Dr. Michael Gormley, Assistant Professor,
School of Psychology, Trinity College Dublin
Phone: (01)896 3903, email: gormlem@tcd.ie

The following are contact details for psychological support, should you experience any distress over the issues raised in this project:

Trinity College staff/students:
Trinity College Counselling Service; (01) 896 1407

For participants outside of Trinity College:
Samaritans (1850 609 090), Aware (1890 303 302)

20 * If you would like to participate in the main experiment please send me an email at the following address:

ocallag@tcd.ie

- I would like to participate in the main experiment
- I would not like to participate in the main experiment

Previous Page | Finish Survey

9.4 Appendix D: Manchester Driver Behaviour Questionnaire (DBQ)

Questionnaire with the subscale that each item belonged to indicated.

MANCHESTER DRIVER BEHAVIOUR QUESTIONNAIRE

How often do you do each of the following? Circle the response most appropriate for you.

1. Attempt to drive away from the traffic lights in third gear. [Lapse]

Never **Hardly ever** **Occasionally** **Quite often** **Frequently** **Nearly all the time**

2 Overtake a slow driver on the inside. [Viol(HC)]

Never **Hardly ever** **Occasionally** **Quite often** **Frequently** **Nearly all the time**

3. Drive especially close to the car in front as a signal to its driver to go faster or get out of the way. [Viol (HC)]

Never **Hardly ever** **Occasionally** **Quite often** **Frequently** **Nearly all the time**

4. Attempt to overtake someone that you hadn't noticed to be signalling a right turn. [Error]

Never **Hardly ever** **Occasionally** **Quite often** **Frequently** **Nearly all the time**

5. Forget where you left your car in a car park. [Lapse]

Never **Hardly ever** **Occasionally** **Quite often** **Frequently** **Nearly all the time**

6. Switch on one thing, such as the headlights, when you meant to switch on something else, such as the wipers. [Lapse]

Never **Hardly ever** **Occasionally** **Quite often** **Frequently** **Nearly all the time**

7. Sound your horn to indicate your annoyance to another road user. [Viol (agg)]

Never **Hardly ever** **Occasionally** **Quite often** **Frequently** **Nearly all the time**

8. Realise that you have no clear recollection of the road along which you have just been traveling. [Lapse]

Never	Hardly ever	Occasionally	Quite often	Frequently	Nearly all the time

9. Cross a junction knowing that the traffic lights have already turned against you. [Viol (HC)]

Never	Hardly ever	Occasionally	Quite often	Frequently	Nearly all the time

10. Fail to notice that pedestrians are crossing when turning into a side street from a main road. [Error]

Never	Hardly ever	Occasionally	Quite often	Frequently	Nearly all the time

11. Become angered by another driver's behaviour, and give chase with the intention of giving him/her a piece of your mind. [Viol (agg)]

Never	Hardly ever	Occasionally	Quite often	Frequently	Nearly all the time

12. Misread the signs and exit from a roundabout on the wrong road. [Lapse]

Never	Hardly ever	Occasionally	Quite often	Frequently	Nearly all the time

13. Disregard the speed limit on a residential road. [Viol (HC)]

Never	Hardly ever	Occasionally	Quite often	Frequently	Nearly all the time

14. On turning left, nearly hit a cyclist who has come up on your inside. [Error]

Never	Hardly ever	Occasionally	Quite often	Frequently	Nearly all the time

15. Pull out of a junction so far that the driver with right of way has to stop and let you out. [Viol (agg)]

Never	Hardly ever	Occasionally	Quite often	Frequently	Nearly all the time

16. Queuing to turn left onto a main road, you pay such close attention to the main stream of traffic that you nearly hit the car in front. [Error]

Never **Hardly ever** **Occasionally** **Quite often** **Frequently** **Nearly all the time**

17. Drive even though you realise that you may be over the legal blood-alcohol limit. [Viol (HC)]

Never **Hardly ever** **Occasionally** **Quite often** **Frequently** **Nearly all the time**

18. Have an aversion to a particular class of road user and indicate your hostility by whatever means you can. [Viol (agg)]

Never **Hardly ever** **Occasionally** **Quite often** **Frequently** **Nearly all the time**

19. Underestimate the speed of an oncoming vehicle when overtaking. [Error]

Never **Hardly ever** **Occasionally** **Quite often** **Frequently** **Nearly all the time**

20. Hit something when reversing that you had not previously seen. [Lapse]

Never **Hardly ever** **Occasionally** **Quite often** **Frequently** **Nearly all the time**

21. Intending to drive to destination A, you 'wake up' to find yourself on the road to destination B, perhaps because the latter is your more usual destination. [Lapse]

Never **Hardly ever** **Occasionally** **Quite often** **Frequently** **Nearly all the time**

22. Stay in a motorway lane that you know will be closed ahead until the last minute before forcing your way into the other lane. [Viol (agg)]

Never **Hardly ever** **Occasionally** **Quite often** **Frequently** **Nearly all the time**

23. Get into the wrong lane approaching a roundabout or junction. [Lapse]

Never **Hardly ever** **Occasionally** **Quite often** **Frequently** **Nearly all the time**

24. Miss 'Give way' signs, and narrowly avoid colliding with traffic having right of way. [Error]

Never	**Hardly ever**	**Occasionally**	**Quite often**	**Frequently**	**Nearly all the time**

25. Fail to check your rear view mirror before pulling out, changing lanes etc. [Error]

Never	**Hardly ever**	**Occasionally**	**Quite often**	**Frequently**	**Nearly all the time**

26. Get involved in unofficial 'races' with other drivers. [Viol (agg)]

Never	**Hardly ever**	**Occasionally**	**Quite often**	**Frequently**	**Nearly all the time**

27. Brake too quickly on a slippery road, or steer the wrong way in a skid. [Error]

Never	**Hardly ever**	**Occasionally**	**Quite often**	**Frequently**	**Nearly all the time**

28 Disregard the speed limit on a motorway. [Viol (HC)]

Never	**Hardly ever**	**Occasionally**	**Quite often**	**Frequently**	**Nearly all the time**

Key:
Viol (HC) = Highway Code or normal violations
Viol (agg) = interpersonally aggressive violations

9.5 Appendix E: Advertising the website and online survey

9.5.1 Appendix Ea: Sample of post to online forums

Hi!

My name is Georgia O'Callaghan and I am a second year PhD student in the School of Psychology in Trinity College Dublin.

I am looking for drivers between the age of 18-24 and male drivers 29+ to participate in my study on driving behaviour and the brain. The study will take place in the Lloyd Institute in Trinity College Dublin and will take just an hour and a half. I will be using a special type of MRI (Magnetic Resonance Imaging) to measure brain activity during some games. MRI is a safe, non-invasive technique that uses a magnetic field to take pictures of the brain.

You will be compensated €20 for your time and get pictures of your brain to keep! A specialist will also review the scans so this is a great opportunity to get a free MRI.

To sign up please complete this short online survey to determine your eligibility to be scanned using MRI:

http://kwiksurveys.com/app/rendersur...y87m85f4334126

Or for more information please visit the link below:

http://ocallag.wix.com/yield

Hope to hear from you,

Thanks,

Georgia O'Callaghan

9.5.2 Appendix Eb: Sample of post to Facebook pages

Do you want to earn €20 and get some pictures of your brain?

Are you aged between 18-24 or are you a male over 29? Do you have a full driving license?

If you meet the above criteria and are interested in participating in a study on driving behaviour & the brain in Trinity College Dublin please visit the following link for more information: http://ocallag.wix.com/yield

Thanks!

Georgia O'Callaghan, PhD Candidate,
School of Psychology & Institute of Neuroscience
Trinity College Dublin

9.5.3 Appendix Ec: Email circulated around Irish Universities

Subject:

TCD fMRI Driver Behaviour study - €20 and a picture of your brain!

Body of email:

Hi!

My name is Georgia O'Callaghan and I am a second year PhD student in the School of Psychology here in Trinity College Dublin.

I am looking for **drivers between the age of 18-24 and male drivers 29+** to participate in my study on driving behaviour and the brain. The study will take place in the Lloyd Institute in Trinity College and will take just over an hour. I will be using functional magnetic resonance imaging (fMRI) to image the brain during some games.

You will be compensated **€20** for your time and **get a picture of your brain!**

To sign up please complete this short online survey to determine your eligibility to be scanned using fMRI:

http://kwiksurveys.com/app/rendersurvey.asp?sid=ztn5uma9in9cj4i307034&refer=

Or for more information please visit the link below:

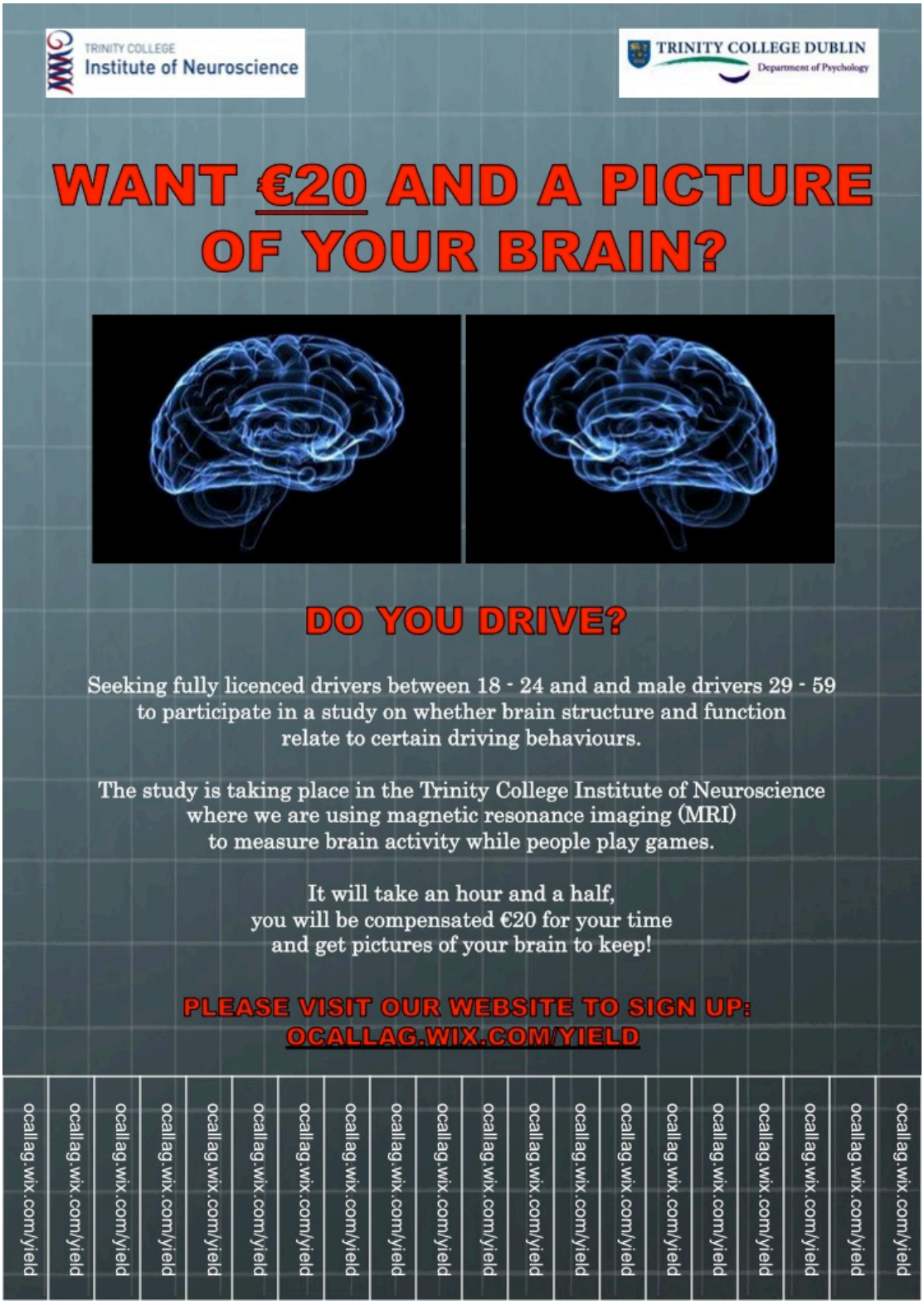
http://ocallag.wix.com/yield

Hope to hear from you,

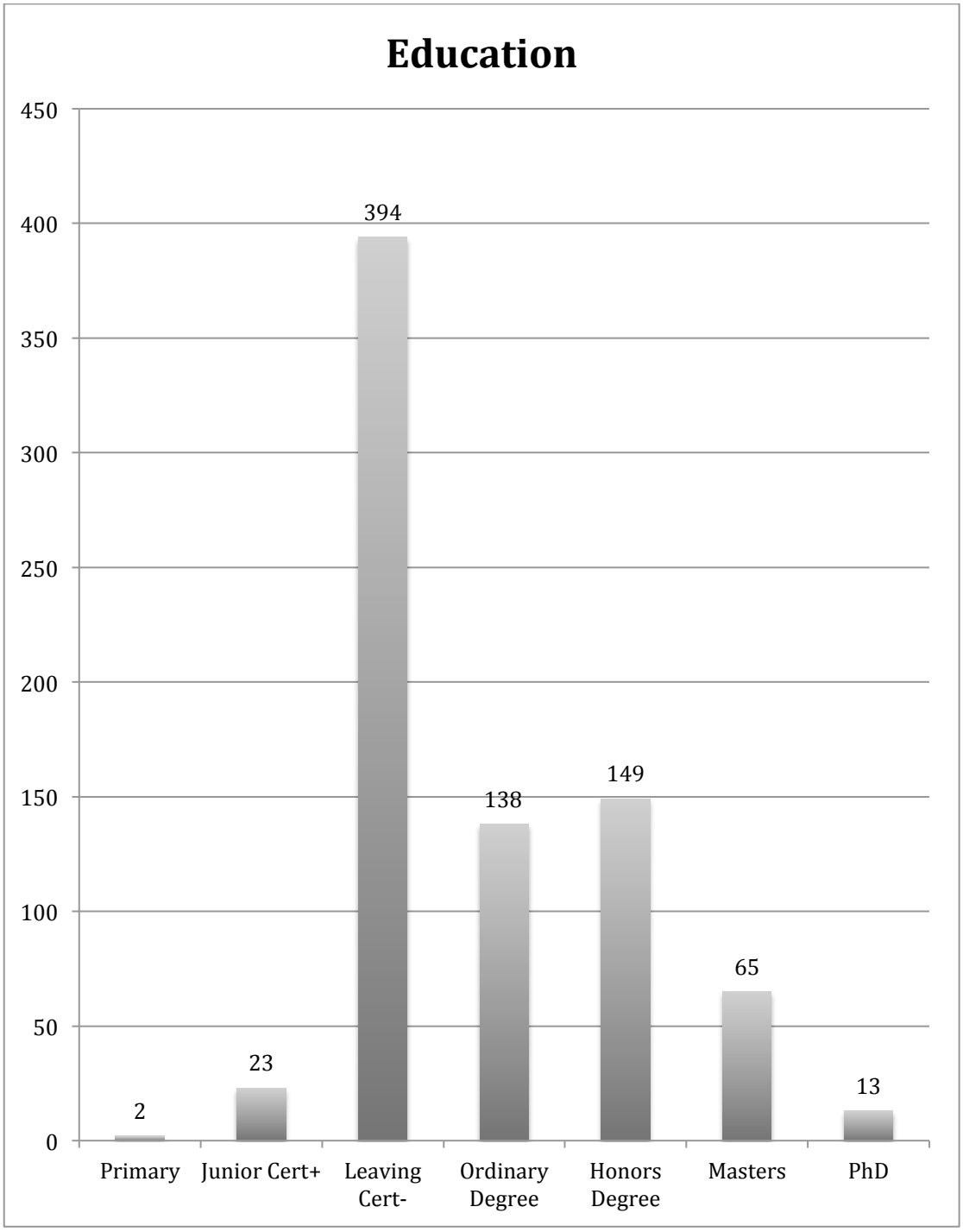
Thanks,

Georgia O'Callaghan
PhD Candidate, Psychology
Trinity College Dublin

9.5.4 *Appendix Ed: Sample of poster displayed in Trinity College and at NCT centres*



9.6 Appendix F: Education of survey respondents

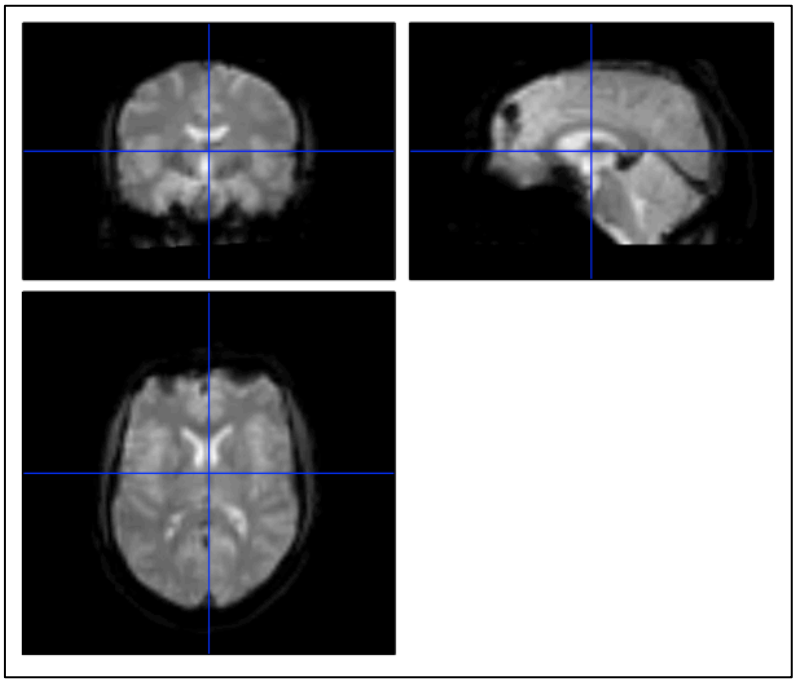


+ = Junior certificate: secondary level education up to 16 years of age approximately

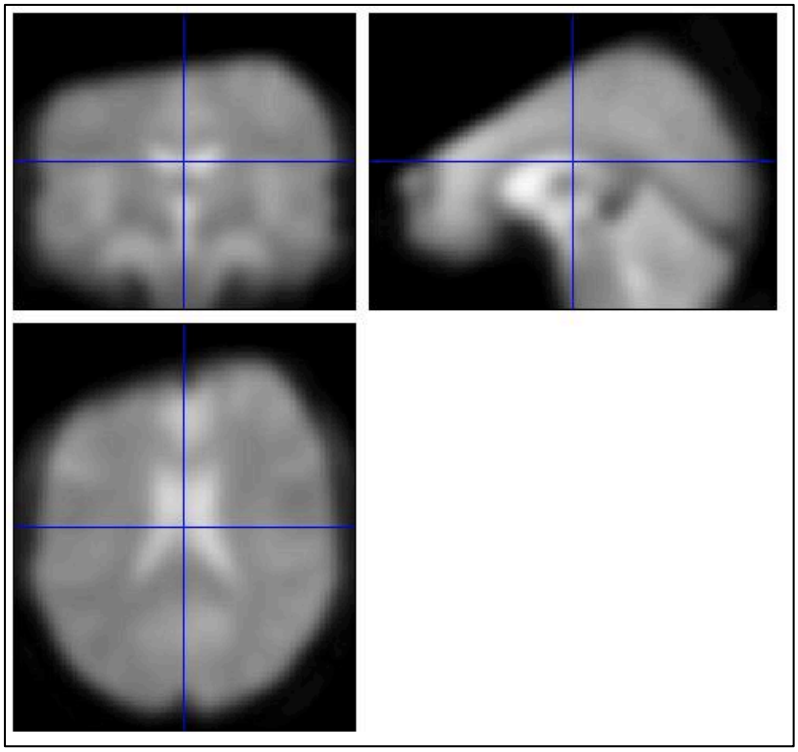
- = Leaving certificate: completion of secondary level education

9.7 Appendix G: Artifact in participant 100

Raw image;



Failed normalisation;



9.8 Appendix H: Letter to participant's General Practitioner (GP)

Insert GP address

Dear **Insert GP's name**

My name is Georgia O'Callaghan and I am a second year PhD student in the School of Psychology, Trinity College Dublin. Your patient **insert patient name here** (D.O.B.) has agreed to participate in my research project, "Impulsiveness and aberrant driving behaviours", taking place in Trinity College's Institute of Neuroscience. The project will investigate whether brain structure and function contribute to impulsive and risky driving behaviours. To do so it will utilise functional magnetic resonance imaging (fMRI) to image the brain during some tasks.

The images of the brain obtained using fMRI are not the type used to look for problems in the brain. However, additional images are obtained for review by a consultant radiologist for the presence of a brain abnormality. In the unlikely event that an abnormality is detected you will be contacted by the radiologist who will recommend that your patient receive a further clinical-quality brain scan. **Insert patient name** has been informed of this practice and has indicated their consent. This process is confidential and the researchers on this project will not be informed if any abnormalities are detected in the scan. All communication will occur between you and the radiologist.

Please be assured that all information provided by your patient shall remain anonymous. Should you have any queries relating to the research please do not hesitate to contact me, or the supervisor of this research, Dr. Michael Gormley, as per the contact details below.

Yours sincerely,

_____

Georgia O'Callaghan

Georgia O'Callaghan
PhD Candidate
School of Psychology
Trinity College Dublin
Dublin 2
Tel: 01 896 3083
Email: ocallag@tcd.ie

Dr. Michael Gormley
Assistant Professor
School of Psychology
Trinity College Dublin
Dublin 2
Tel: 01 896 3903
Email: gormlem@tcd.ie

9.9 Appendix I: Study information sheet

Information sheet:

Does Impulsivity Contribute to Risky Driving?

Dear Participant,

My name is Georgia O'Callaghan and I am a second year PhD student in Trinity College, Dublin. I request your consent to participate in a study on the relationship between impulsivity and driving behaviours. In this study you will be asked to provide some general background information on yourself and be asked to complete two short questionnaires. You will then complete three tasks while in the MRI scanner. MRI is a type of safe, non-invasive brain imaging method which uses a strong magnetic field to take pictures of the brain. You will not feel the magnetic field. You will, however, hear repetitive tapping noises that arise from the scanner around your body. We will provide earplugs or ear phones that you will be required to wear so that it is not too loud.

Scanning will take place in Trinity College Institute of Neuroscience, Lloyd Building, Trinity College Dublin. It will take an hour and you will be given breaks between the tasks. During this time you will lie in the scanner on a narrow couch and we will make you feel as comfortable as possible. The machine will take images of your brain, and we will use this information to learn about the brain's structure and function, and relate this information to performance on the various different tasks and questionnaires you will complete. It is very important that you try not to move your head as this affects the quality of the data. During the scan the experimenter will be in the next room and you can talk to them on an intercom system. You can stop the scan at any time by pressing an emergency button.

You will be asked to perform three tasks that examine decision making and behavioural inhibition. These tasks will be explained to you before entering the scanner and you will be shown examples to make sure you understand them. Some of the tests are designed to be challenging, therefore it is not surprising that you may get frustrated or tired. Do not get discouraged and try to accomplish the tests to the best of your abilities. You will have sufficient breaks during the tests, however please let the experimenter know if you are getting tired and would like another break.

Participation in this study is voluntary and there are no consequences to not participating. You will be compensated €20 for your time. All information provided by you will remain fully confidential. This information will be stored in accordance with the Data Protection Act. Under the Freedom of Information Act, 1997, you have the right to access your own data and can contact the researchers to get such information.

If, after reading this, you decide that you would like to participate; simply sign the consent form. Please be assured that all information provided by you will remain anonymous and your consent form will be stored in a secure filing cabinet, separated from any answers you may provide. Should you have any queries relating to the research and/or your participation, please do not hesitate to contact me, or the supervisor of this research, Dr. Michael Gormley, as per the contact details below. Finally, you are free to withdraw from the study at any time, without prejudice.

Yours sincerely,

Georgia O'Callaghan, PhD Candidate, phone: (01)8963083, email: ocallag@tcd.ie

Dr. Michael Gormley, Assistant Professor, phone: (01)896 3903, email: gormlem@tcd.ie

9.10 Appendix J: Study consent form

Consent form:

Does Impulsivity Contribute to Risky Driving?

Date: _____

I consent to participate in this study. I am aware that participation is voluntary. I understand that I may withdraw from the study at any time without judgement and without consequence. I understand that I may not enter the scanner if I meet any of the criteria outlined in the information sheet as to do so may be a risk to my own health and safety. I understand that my scans will be examined by a professional radiologist, Dr. William Torreggiani in AMNCH (Tallaght Hospital), and that any significant abnormalities will be reported to my GP for appropriate follow-up, if necessary. I am aware that my GP received a letter informing them of this protocol.

I understand that all information provided by me will remain fully confidential and that I may access this information in accordance with the Freedom of Information Act, 1997. I have been informed that I may withdraw from this study at any time without prejudice.

Print Name _____

Signature of Participant: _____

Georgia O'Callaghan
PhD Candidate
School of Psychology
Trinity College Dublin
Dublin 2
Tel: 01 896 3083
Email: ocallag@tcd.ie

Dr. Michael Gormley
Assistant Professor
School of Psychology
Trinity College Dublin
Dublin 2
Tel: 01 896 3903
Email: gormlem@tcd.ie

9.11 Appendix K: Trinity College Institute of Neuroscience (TCIN) MRI information and consent booklet

Assessment of Risk Factors for participant in Magnetic Resonance Imaging experiments

MAGNETIC RESONANCE IMAGING

Name: _____

Phone- _____

Sex: Male [ ] Female [ ]

Date of Birth: _____

Weight: _____kg

Please provide us with the details of another person (e.g., next-of-kin) should we need to contact you in the future.

Name of contact person: _____

Phone: _____

Study: _____

Time in: ____:____ Time Out: ____:____

Investigator: _____

Information:

Your MR study

As part of your study we will obtain a limited number of pictures of your brain. Our research studies are designed to improve our knowledge of the brain. They are not designed for diagnostic or clinical purposes.

Minor changes are sometimes found in completely healthy people. You should be aware that because our pictures are taken for a specific research purpose, not all abnormalities that might be detected by other MR scans are necessarily seen. On extremely rare occasions, an significant abnormality which should be investigated further may be detected. If such a significant abnormality in your brain is detected, the consultant radiologist, Dr William Torreggiani in AMNCH (Tallaght Hospital) will contact your GP, and your GP will make appropriate arrangements for follow-up, should that be required.

Although a significant abnormality is extremely unlikely, you should be aware that if such an abnormality is detected and you are informed, then this knowledge may have consequences for you. Please take the time to consider carefully what it would mean to you if we told you of an abnormality in your brain which might, or might not, affect you later in life. Knowledge of an abnormality may affect your ability to work in certain professions, obtain life or health insurance and other facets of daily living. If you do not want to know, then it is better not to participate.

There are some items that may be **potentially hazardous** for MR scanning. To help us to determine your suitability for an MRI scan and to ensure your safety, please complete the following checklist carefully.

Not all people can have an MRI scan because the strong magnetic field may be hazardous to them.

- People with
  - permanent pacemakers
  - prosthetic heart valves
  - implanted cardiac defibrillators
  - certain types of vascular clips

Cannot have an MRI scan

Instructions for Patient/Volunteers

1. You are urged to use the earplugs or headphones we supply during your MRI examination since some patients may find the noise levels unacceptable, and the noise levels may affect your hearing.
2. Remove all jewellery (e.g., necklaces, pins, rings).
3. Remove all hairpins, bobby pins, barrettes, clips, etc.
4. Remove all dentures, false teeth, and partial dental plates.
5. Remove hearing aides.
6. Remove eyeglasses.
7. Remove your watch, pager, cell phone, credit cards, bankcards, and all other cards with a magnetic strip.
8. Remove body piercing objects.

9. Use gown, if provided, or remove all clothing with metal fasteners, zippers, etc.

CHECK LIST FOR 3T MAGNETIC RESONANCE IMAGING

Do you have any of the following:

Cardiac Pacemaker: Yes No
Have you ever had any surgical procedures? Yes No
If yes, what type and where

.....

.....

The following items may be harmful to you during your MR scan or may interfere with the MR examination. You must provide a "yes" or "no" for every item. Please indicate if you have or have had any of the following:

Yes	No	
____	____	Any type of electronic, mechanical, or magnetic implant Type:
____	____	Cardiac pacemaker
____	____	Aneurysm clip
____	____	Implanted cardiac defibrillator
____	____	Neurostimulator
____	____	Biostimulator Type:
____	____	Any type of internal electrodes or wires
____	____	Cochlear implant
____	____	Hearing aid
____	____	Implanted drug pump (eg, insulin, Baclofen, chemotherapy, pain medicine)
____	____	Halo vest
____	____	Spinal fixation device
____	____	Spinal fusion procedure
____	____	Any type of coil, filter, or stent Type:
____	____	Any type of metal object (eg, shrapnel, bullet, BB)
____	____	Artificial heart valve
____	____	Any type of ear implant
____	____	Penile implant
____	____	Artificial eye
____	____	Eyelid spring
____	____	Any type of implant held in place by a magnet Type:
____	____	Any type of surgical clip or staple
____	____	Any IV access port (eg, Broviac, Port-a-Cath, Hickman, Picc line)
____	____	Medication patch (eg, nitroglycerine, nicotine)
____	____	Shunt
____	____	Artificial limb or joint What and where:
____	____	Tissue expander (eg, breast)
____	____	Removable dentures, false teeth, or partial plate
____	____	Diaphragm, IUD, Pessary Type:
____	____	Surgical mesh Location:
____	____	Body piercing Location:
____	____	Wig, hair implants
____	____	Tattoos or tattooed eyeliner
____	____	Claustrophobia

Handedness: Right / Left

Native Language: _____

I have read and understood this form, and consent to my study being used for research. I have had a chance to ask any questions.

My contact phone number is: _____

My contact address is: _____

_____

_____

Signed (Volunteer): _____

Parent or Guardian if under 18: _____

Witnessed (Researcher/MR staff): _____

Date: _____

Hazard Checklist for MRI Personnel

Yes	No	
____	____	Endotracheal tube
____	____	Swan-ganz catheter
____	____	Extra ventricular device
____	____	Arterial line transducer
____	____	Foley catheter with temperature sensor and/or metal clamp
____	____	Rectal probe
____	____	Esophageal probe
____	____	Tracheotomy tube
____	____	Guidewires

Comments: _____

TRINITY COLLEGE
Institute of Neuroscience
TCIN *from molecules to mind*

GENERAL MRI DATA CONSENT FORM

Trinity College Institute of Neuroscience, (TCIN) is performing research, utilising an MRI scanner at Trinity College, Dublin 2. These research scans, although not full clinical scans, will be read by a radiologist.

In the unlikely event of an irregularity being found, the radiologist, [Dr William Torreggiani of The Adelaide and Meath Hospital Incorporating the National Children's Hospital (AMNCH), Tallaght] will inform the participant's GP, that a proper clinical scan may be required to determine whether or not an irregularity is of clinical significance.

To enable us to perform the research scans the participant agrees to give consent/ permission for:

(i) TCIN to conduct the MRI scan and store MRI scan data of the participant;
(ii) TCIN or Principal Investigator, (PI) to contact participant's GP;
(iii) TCIN radiographer to send MRI scan data to radiologist acting for TCIN;
(iv) Radiologist to store data in a hospital system with same care as other patient data ensuring participant's confidentiality;
(v) Radiologist/ Clinician (acting for TCIN) to contact participant's GP;
(vi) TCIN to store data on the study for a period of at least 5 years or as specified in the specific consent form.

A dated standard letter signed by the appropriate Principal Investigator will be sent to all participants' GPs, it is the responsibility of the Principal Investigator to ensure that this is sent at least two days before scanning to allow for postal delays. The principal investigator is responsible for their project at all times.

The TCIN designated radiologist will be sent data in a form that allows identification so that if a response is required he can act quickly (a copy of this is also held at TCIN). This will be stored in the hospital system with the same rigour and attention to confidentiality as all other medical data, as per the rules of that institution; a copy of this data will also be stored at TCIN. The raw scan data will be stored at TCIN in anonymous form for research purposes as agreed on the consent form of the specific research project.

I agree to the above points and understand that my data will be treated carefully at TCIN and in the hospital system.

Participant's Name and Address_____

_____

Signed by Participant:_____

9.12 Appendix L: Demographic questionnaire of the main study

9.12.1 Appendix La: Demographic questionnaire

1. What is your approximate mileage per year? _____

2. What type of car do you most often drive?

   Using the examples that the researcher provides you, please indicate the category in which the car belongs:

   a) Category A: Compact/Small family car/City car

   b) Category B: Medium car/Midsize family car

   c) Category C: Large car/Large family car

   d) Category D: SUV/4x4

   e) Category E: Executive car

   f) Category F: Luxury car/Convertible

3. Do you own this car personally? _____

4. Was the car bought new or second hand? _____

5. What year is the car? _____

6. What is your occupation? _____

7. What are/were your parents' occupations (if known)?

   Mother: Father: _____

8. Indicate the highest level of education your parents have completed (if known)

   (M = mother, F = father)

   a. Primary: _____

   b. Secondary (Junior/Inter Certificate): _____

   c. Secondary (Leaving Certificate): _____

   d. Third (Certificate/Diploma/Ordinary degree): _____

   e. Third (Honours degree): _____

   f. Fourth (HDip/Postgraduate Certificate/Masters/PhD): _____

9.12.2 Appendix Lb: Booklet illustrating the car categories

Category A:

Compact/Small family car/City car

Examples:

- Ford Ka
- Mitsubishi i-MiEV
- Smart Fortwo
- Toyota Aygo
- Fiat 500
- Chevrolet Cruze
- Mazda3
- Toyota Corolla
- Nissan Micra
- Kia Rio

Engine size range: 0.9 L – 1.4 L

Approximate price of a new model: €11,000 - €18,000

Category B:

Medium car/Midsize family car

Examples:

- Ford focus
- Honda civic
- Samand
- Toyota Auris
- Volkswagen golf
- Audi A3
- Volkswagen polo
- Skoda fabia
- Peugeot 308
- Toyota Prius

Engine size range: 1.4 L – 2.0 L

Approximate price of a new model: €17,000 - €30,000

Category C:

Large car/Large family car

Examples:

- Ford Fusion/Mondeo
- Hyundai Sonata
- Holden Commodore
- IKCO Samand
- Buick LaCrosse
- Opel/Vauxhall Insignia
- Peugeot 508
- Volkswagen Passat
- Mazda 6
- Škoda Superb
- Honda Accord
- Citroen C5

Engine size range: 2.0 L – 3.0 L

Approximate price of a new model: €22,000 - €39,000

Category D:

SUV/4x4

Examples:

- Daihatsu Terios
- Jeep Wrangler
- Mitsubishi Pajero iO
- Suzuki Jimny
- Chevrolet Equinox
- Ford Escape
- Honda CR-V
- Jeep Liberty
- Kia Sportage
- Ford Edge
- Jeep Grand Cherokee
- Volvo XC90

Engine size range: 1.5 L – 6.4 L

Approximate price of a new model: €27,000 - €50,000

Category E:

Executive car

Examples:

- Alfa Romeo 159
- BMW 3 Series
- Mercedes-Benz C-Class
- Chevrolet Impala
- Ford Taurus
- Hyundai Grandeur
- Tesla Model S
- Toyota Avalon
- Chrysler 300
- Cadillac CTS
- Lincoln MKZ
- Lexus GS
- Volvo S80

Engine size range: 2.6 L – 6.1 L

Approximate price of a new model: €35,000 - €50,000

Category F:

Luxury car/Convertible

Examples:

- BMW 7 Series
- Lexus LS
- Mercedes S-Class
- Porsche Panamera
- BMW 6 Series
- Chevrolet Camaro
- Mercedes CLK
- Volvo C70
- Volkswagen Eos
- Ferrari LaFerrari/FF
- Lamborghini Aventador
- Jaguar XK
- Maserati GranTurismo

Engine size range: 2.5 L – 6.5 L

Approximate price of a new model: €40,000 - €95,000

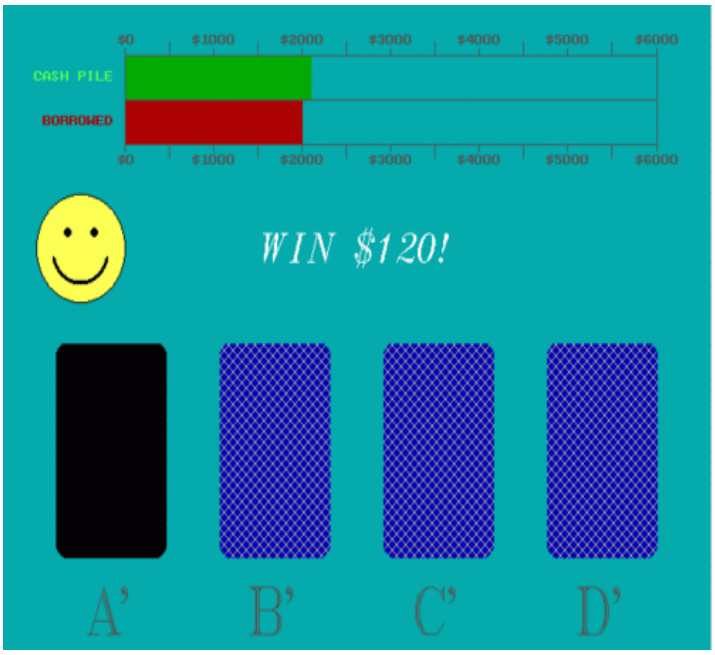
9.13 Appendix M: Iowa Gambling Task (IGT) instructions and example

On the screen there will be four decks of cards, A, B, C, and D. In your hand you will have a response pad with four buttons, each button corresponding to one of the four decks.

On each turn you will use the response pad to select a card from one of the decks. Each time you select a card the colour of the card turns red or black. If the card turns black you will win some money. However, if the card turns red you will win some money but also loose some money. There are equal numbers of red and black cards per deck, so the colour of the cards do not tell you which decks are 'better' in this game.

The amount of money you will win/loose on each turn will vary. You will find out more about this as you go along. Every time you win money the green bar gets bigger, and every time you loose money the green bar gets smaller.

You will begin with a $2,000 'loan' from the bank (the red bar) to start the game. The red bar is there to remind you of how much money you borrowed to play the game, and how much money you will owe the bank at the end. This means that your total winnings for the game will be the green bar, minus the red bar.



You are free to switch from one deck to the other at any time, and as often as you wish.

The goal of the game is to win as much money as possible, and if you can't win, avoid losing money as much as possible. The game will last exactly 15 minutes. You must keep on playing until the computer stops.

It is important to know that just like in a real card game; the computer does not change the order of the cards after the game starts. You may not be able to figure out exactly when you will lose money, but the game is fair. The computer does not make you lose money at random, or make you lose money based on the last card you picked.

You may find all of the decks bad, but some are worse than the others. No matter how much you find yourself losing, you can still win if you stay away from the worst decks.

Please treat the play money in this game as real money, and any decision on what to do with it should be made as if you were using your own money.

Please make your decision as quickly as possible or the deck will be chosen for you!

9.14 Appendix N: Monetary Incentive Delay (MID) task instructions and example

You will only need 1 button for this task.

On each trial a coloured square will appear on screen. The square will be green or blue. The colour of the square indicates whether it is a gain or neutral trail.

Each square will be followed by a target. The target will be a white star inside a white circle. You must press the button when you see the target to win money or avoid losing money.

Green squares indicate gain trials. When the target appears after a green square you must press the button to win money. A picture of a 50c piece will appear to tell you that you won some money. If you do not press the button you will see a sad face, which tells you that you have not won anything on this trial.

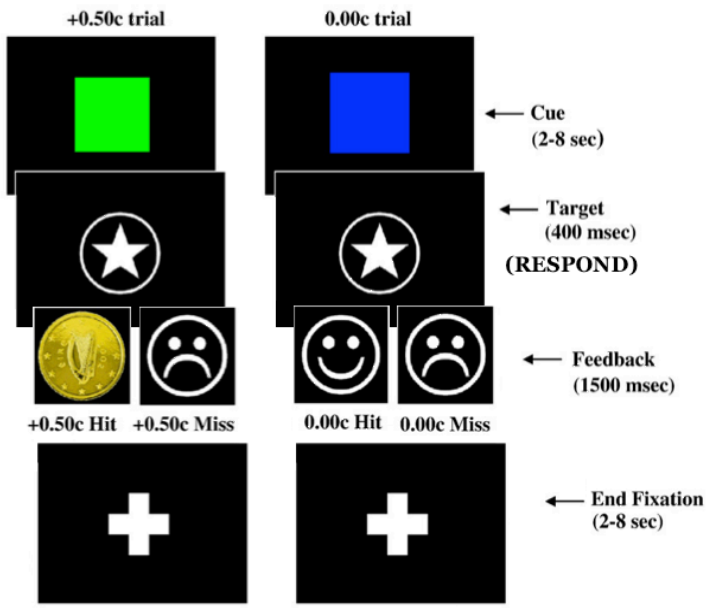
Blue squares indicate neutral trials. You will not win or lose money on these trials. When the target appears after a blue square you must still press the button. When you do you will see a happy face but if you do not you will see a sad face.

At the end of each trial you will see a white cross, this is to tell you to get ready for the next trial. It will be followed immediately after by the next coloured square.

The rewards in this game are hypothetical. You will still receive your full €20s compensation at the end, regardless of your performance on the task. However, please still treat the money in the game as real, and do your best to win as much money as possible!

The target does not appear for long so respond quickly!

An example of the different trial types can be seen in the accompanying figure:



9.15 Appendix O: Go/No Go task instructions and example

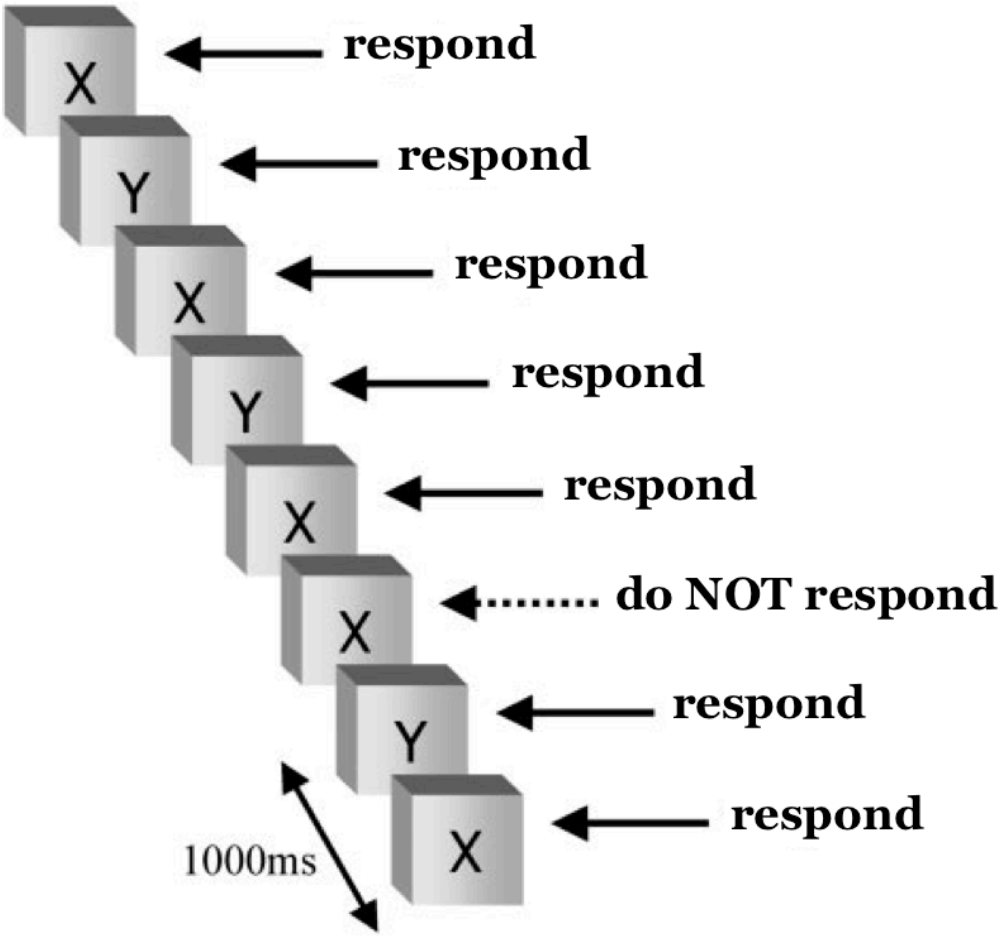
You will only need 1 button for this task.

The letters ''X'' and ''Y'' will be presented on the screen one at a time in an alternating fashion i.e. X, Y, X, Y... and so on.

You must press the button as quickly as possible each time one of these letters appears on screen.

However, if the same letter is presented twice in a row, then you must not respond on the second presentation.

For example:



All you have to do is keep pressing the button each time a letter appears on screen unless a letter is presented twice in a row.

9.16 Appendix P: Possible task run orders

- IGT, MID XY
- IGT, XY, MID
- XY, MID, IGT
- MID, XY, IGT

9.17 Appendix Q: Debriefing sheet

Debriefing:

Does Impulsivity Contribute to Risky Driving?

Dear Participant,

Thank you for taking part in this study on the relationship between impulsivity and driving behaviour. Impulsivity is seen as a tendency to act without considering what the consequences may be. High impulsivity has been linked to many risk taking behaviours but the current study focuses impulsivity in driving. Because of the noted increase in impulsivity during adolescence this study is examining the relationship between age, impulsivity, risky driving behaviours and brain development in the hopes of offering an explanation for why younger drivers are involved in more collisions than older drivers. The aim of this research is to investigate whether those who take risks while driving have higher levels of impulsivity and whether this may be attributed to differences in brain development.

Functional Magnetic Resonance Imaging will allow us investigate whether the brain activity observed during the tasks you have completed differs between individuals of high and low impulsivity and of high and low driving risk. In addition, the questionnaires you have completed will allow us to get an idea of how impulsivity changes with age and whether this change relates to driving behaviours.

Please be assured that all information provided by you will remain anonymous. Should you have any queries relating to the research and/or your participation, please do not hesitate to contact me, or the supervisor of this research, Dr. Michael Gormley, as per the contact details below. In the unlikely event that an abnormality is detected in your scan you will be contacted by your GP. This process is completely confidential and the researchers will not be informed of such abnormalities.

The following are contact details for psychological support, should you experience any distress over the issues raised in this project:

Trinity College staff/students:

Trinity College Counselling Service; (01) 896 1407

For participants outside of Trinity College:

Samaritans (1850 609 090), Aware (1890 303 302)

Yours sincerely,

Georgia O'Callaghan

Georgia O'Callaghan
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Assistant Professor
School of Psychology
Trinity College Dublin
Dublin 2
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Email: gormlem@tcd.ie

9.18 Appendix R: Participant payment receipt

ÁRAS AN PHIARSAIGH

TRINITY COLLEGE

DUBLIN 2, IRELAND

SCHOOL OF PSYCHOLOGY

RESEARCH PARTICIPANT

EXPENSES FORM

TITLE OF STUDY: **Impulsivity and Risky driving** AMOUNT PAID: **€20**

STAFF MEMBER RESPONSIBLE: **Dr. Michael Gormley**

NAME OF PARTICIPANT: _____

DATE OF PARTICIPATION: _____

SIGNATURE OF PARTICIPANT: _____

SIGNATURE OF RESEARCHER: _____

9.19 Appendix S: Barratt's Impulsiveness Scale (BIS-II)

DIRECTIONS:

People differ in the ways they act and think in different situations. This questionnaire will measure some of the ways in which you act and think.

Answer quickly and honestly. Please do not spend too much time on any statement.

Rarely/Never	Occasionally	Often	Almost always/Always
1	2	3	4

Read each statement and put an X in the appropriate square on the right side of this page, where 1 = Rarely/Never, 2 = Occasionally, 3 = Often and 4 = Almost always/Always:

	Question	1	2	3	4
1	I plan tasks carefully				
2	I do things without thinking				
3	I makeup my mind quickly				
4	I am happy-go-lucky				
5	I don't "pay attention"				
6	I have "racing" thoughts				
7	I plan trips well ahead of time				
8	I am self-controlled				
9	I concentrate easily				
10	I save regularly				
11	I "squirm" at plays or lectures				
12	I am a careful thinker				
13	I plan for job security				
14	I say things without thinking				
15	I like to think about complex problems				
16	I change jobs				
17	I act "on impulse"				
18	I get easily bored when solving thought problems				
19	I act on the spur of the moment				
20	I am a steady thinker				
21	I change residences				
22	I buy things on impulse				
23	I can only think about one thing at a time				
24	I change hobbies				
25	I spend or charge more than I earn				
26	I often have extraneous thoughts when thinking				
27	I am more interested in the present than the future				
28	I am restless at the theatre or lectures				
29	I like puzzles				
30	I am future oriented				

9.20 Appendix T: How to score the BIS-II

Scoring the BIS:

Barratt Impulsiveness Scale 11 – Factor Structure and Scoring

2nd Order Factors	**1st Order Factors**	**# of items**	**Items contributing to each subscale**
Attentional	Attention	5	5, 9*, 11, 20*, 28
	Cognitive Instability	3	6, 24, 26
Motor	Motor	7	2, 3, 4, 17, 19, 22, 25
	Perseverance	4	16, 21, 23, 30*
Non-planning	Self-Control	6	1*, 7*, 8*, 12*, 13*, 14
	Cognitive Complexity	5	10*, 15*, 18, 27, 29*

*reverse scored items

9.21 Appendix U: Kirby's Delay Discounting Questionnaire (DDQ)

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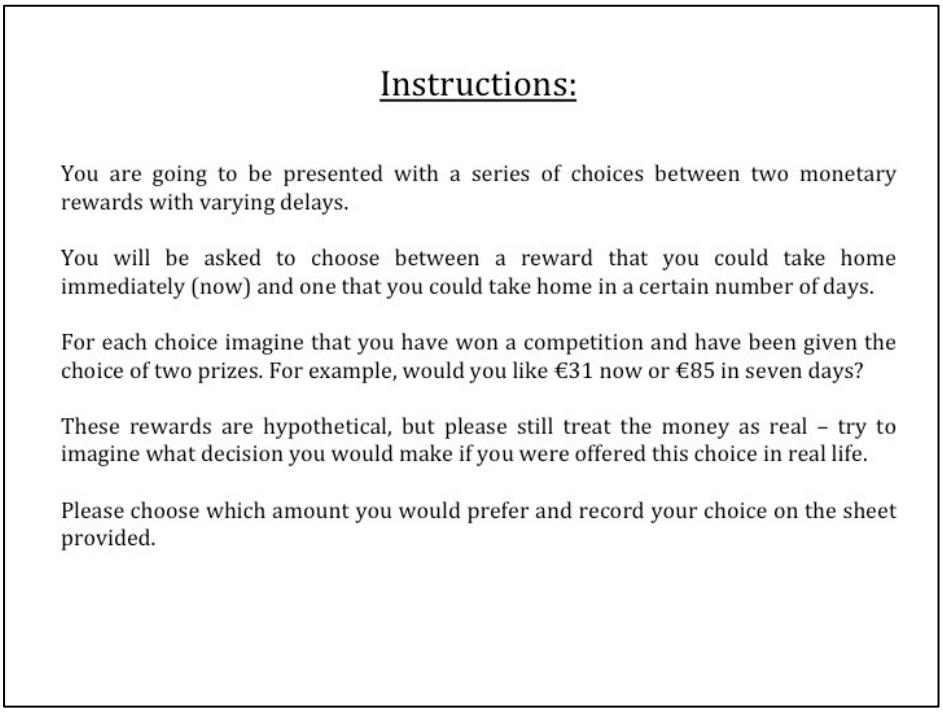

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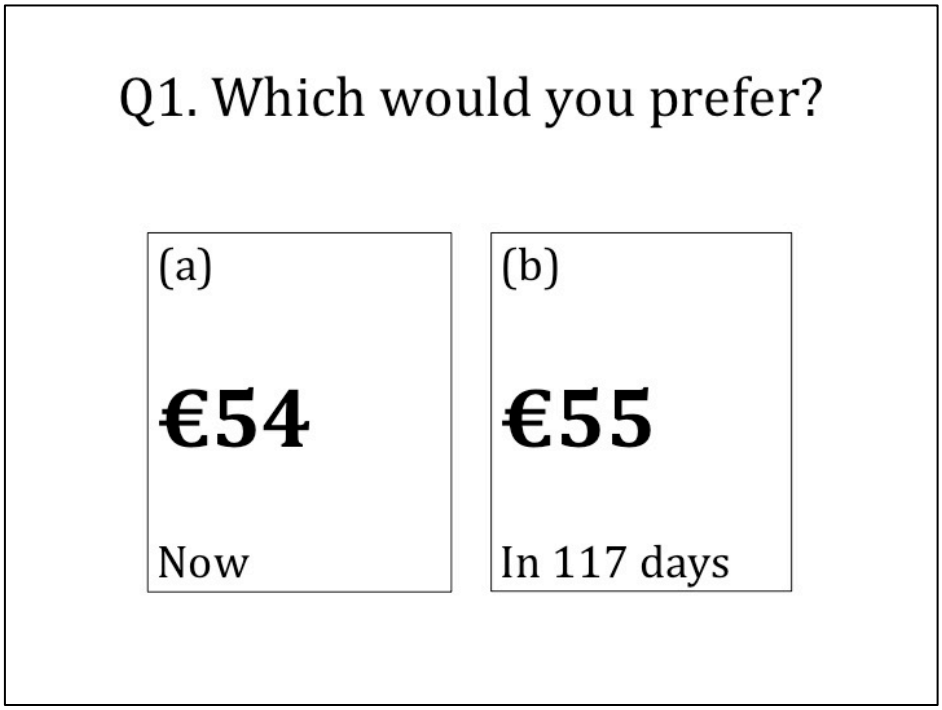
Slide 3:



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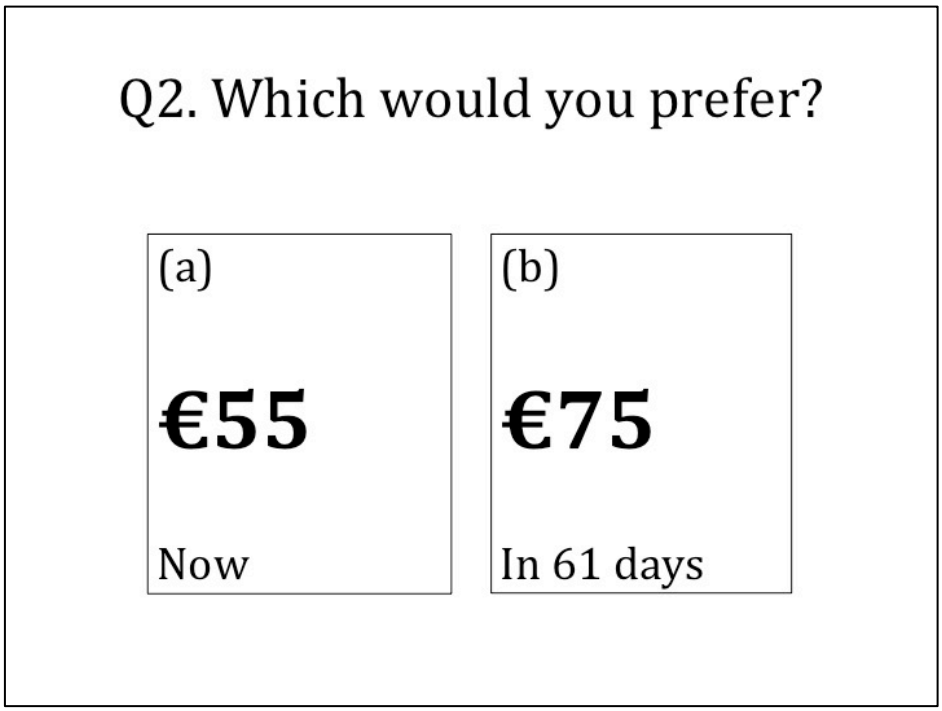

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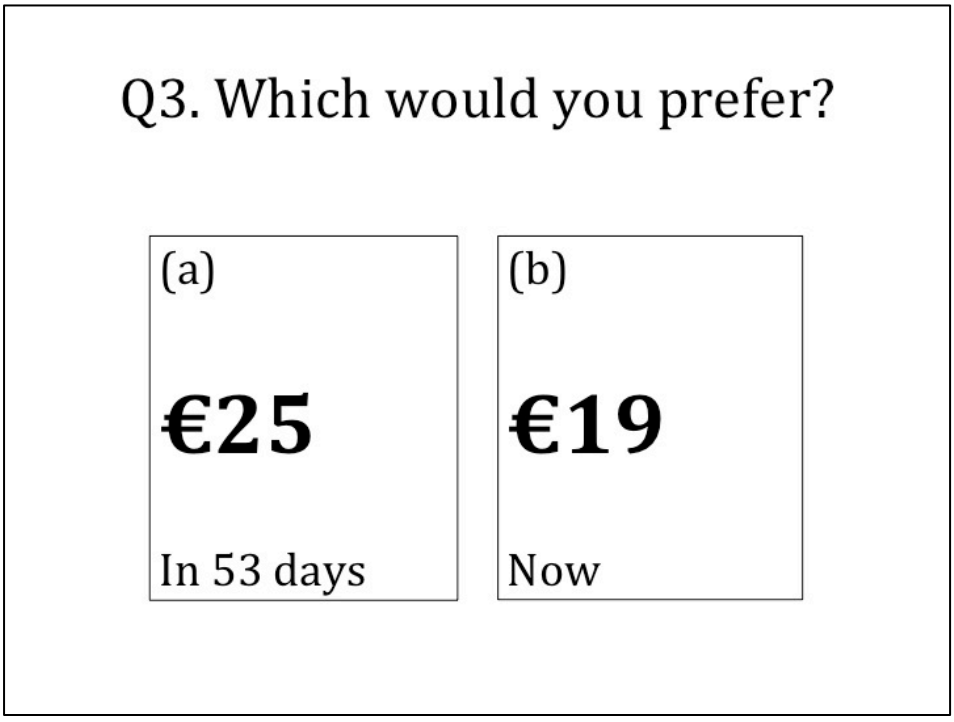

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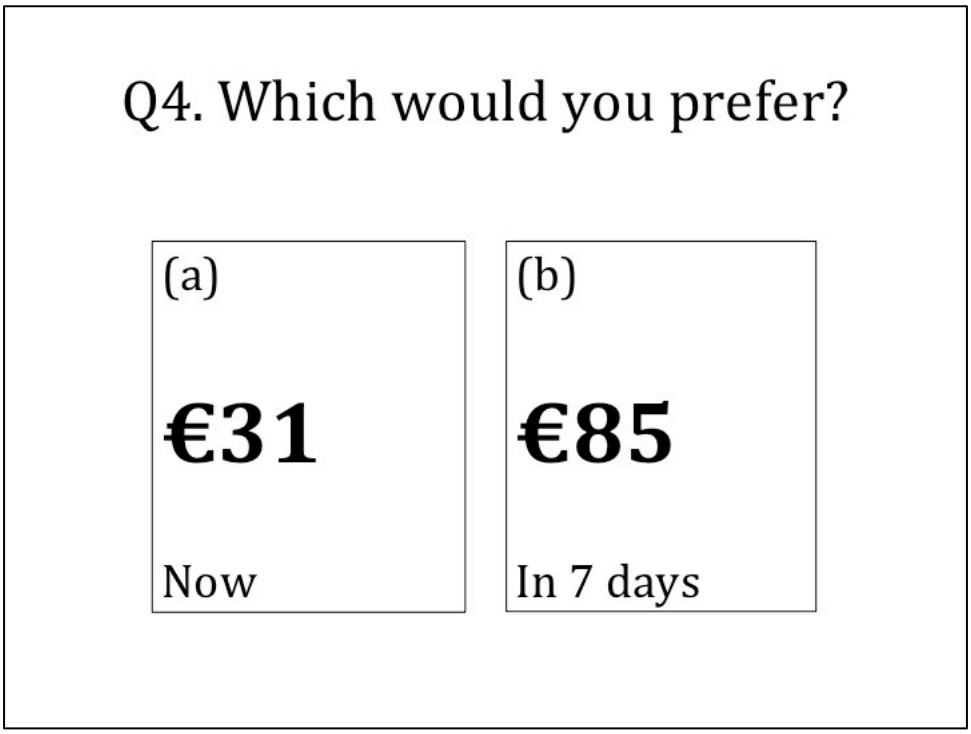

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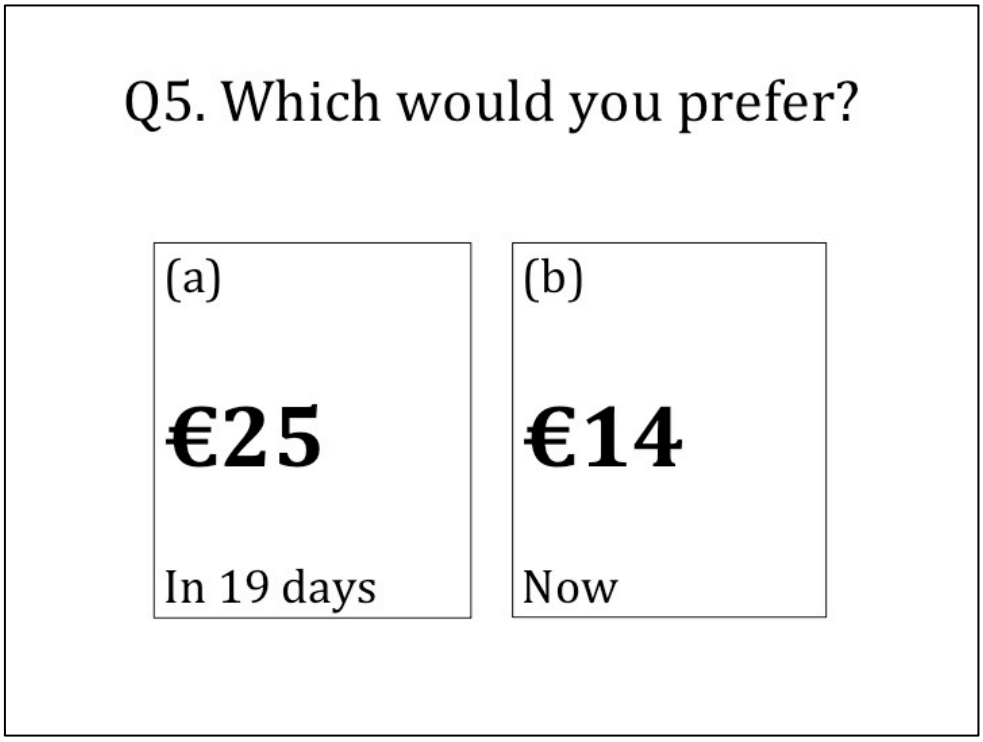

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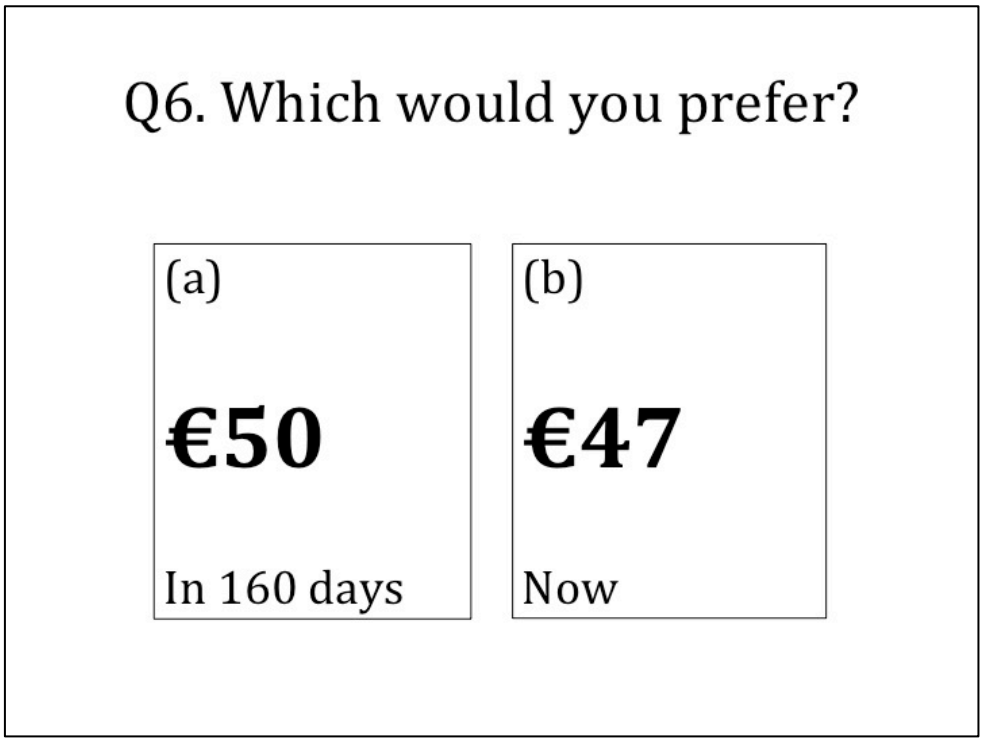

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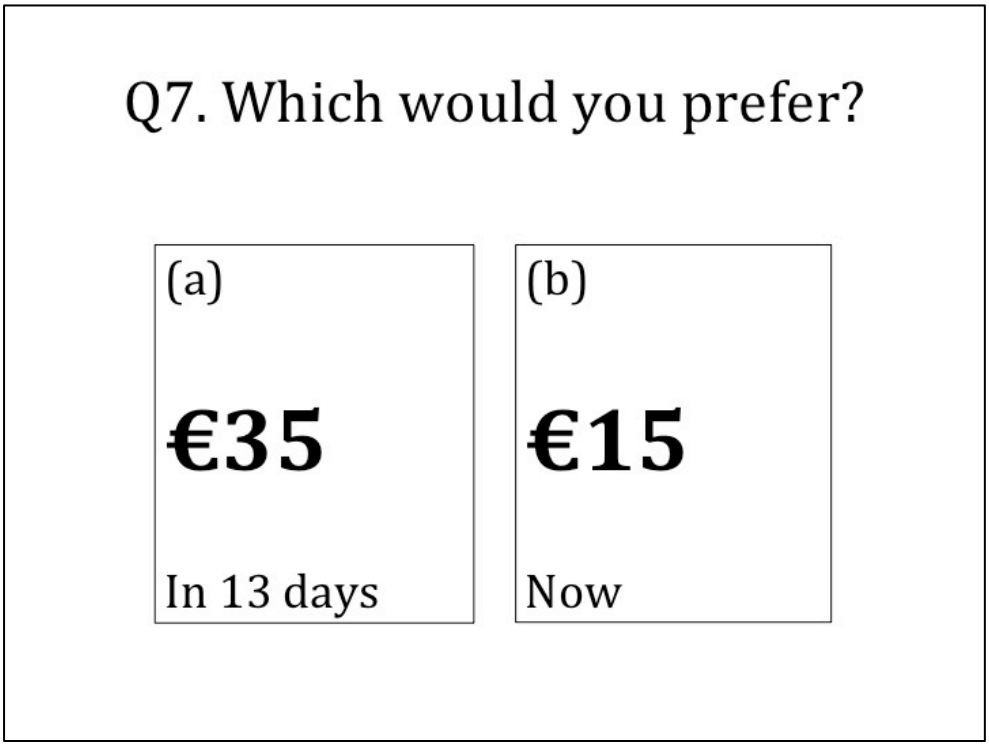

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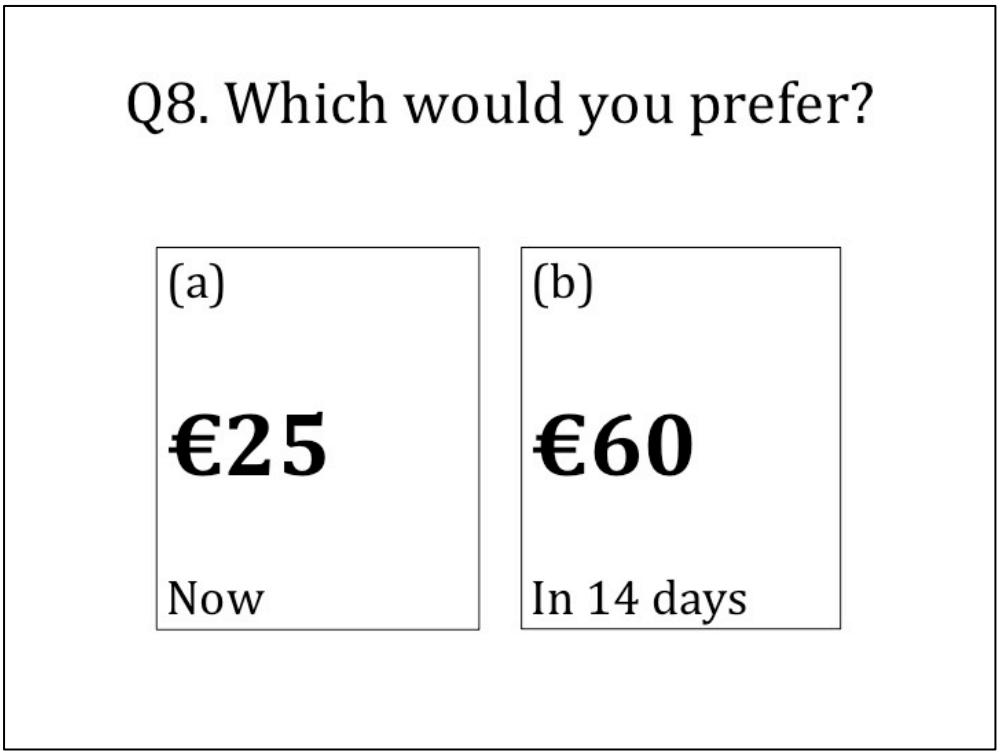

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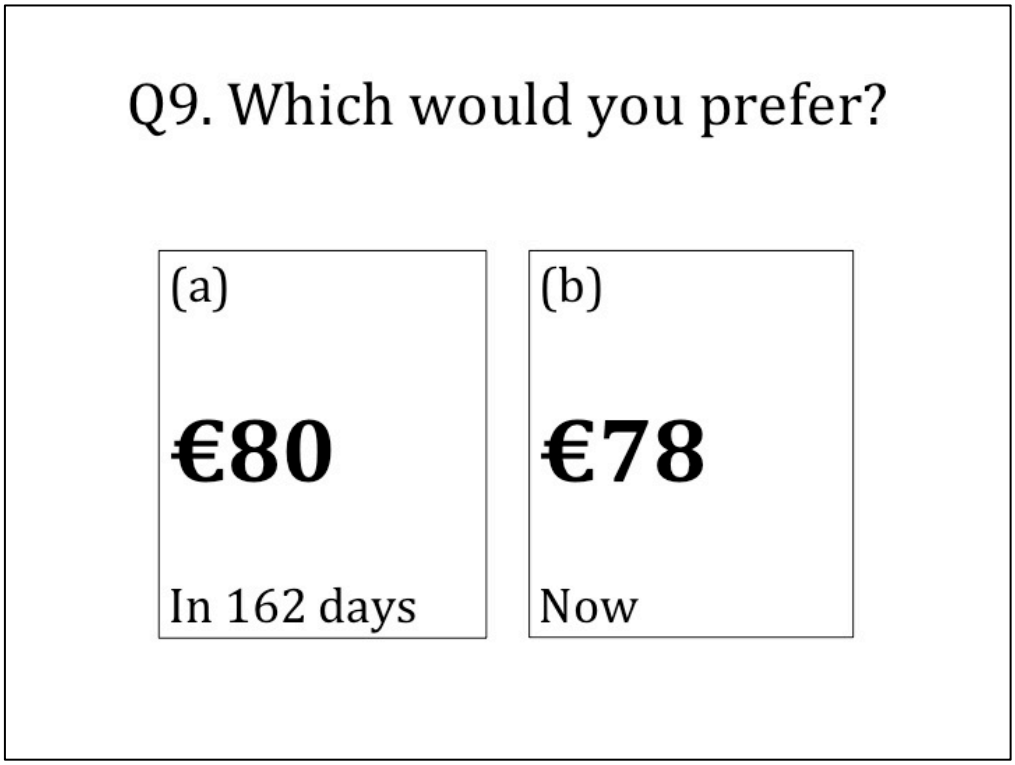

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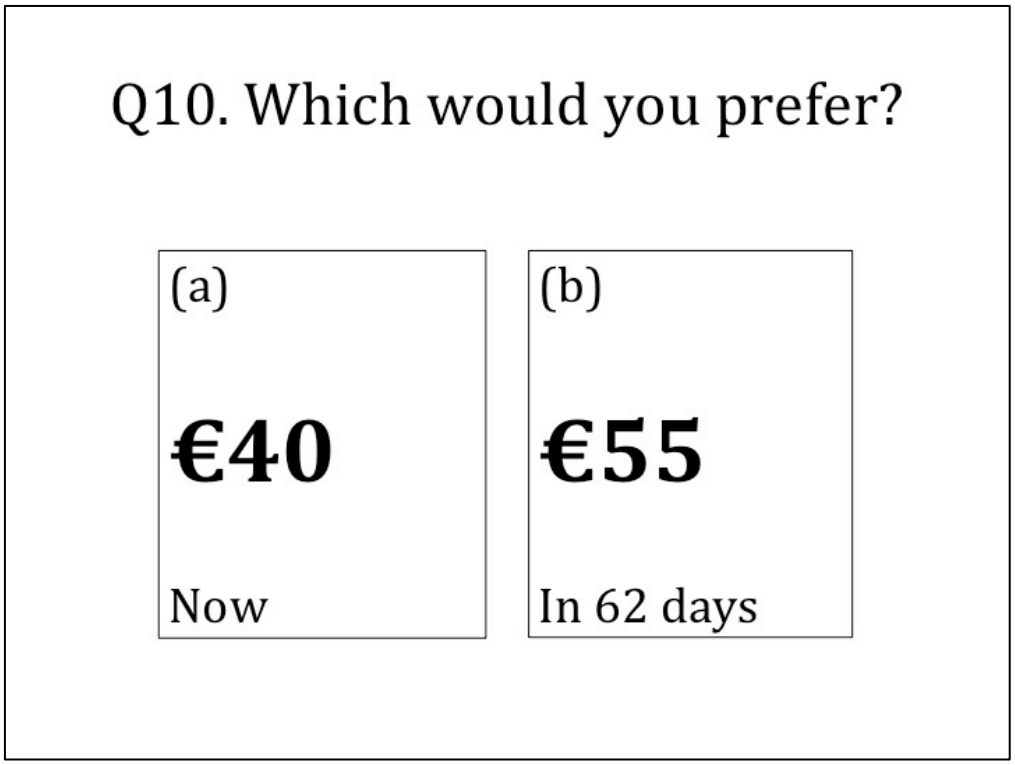

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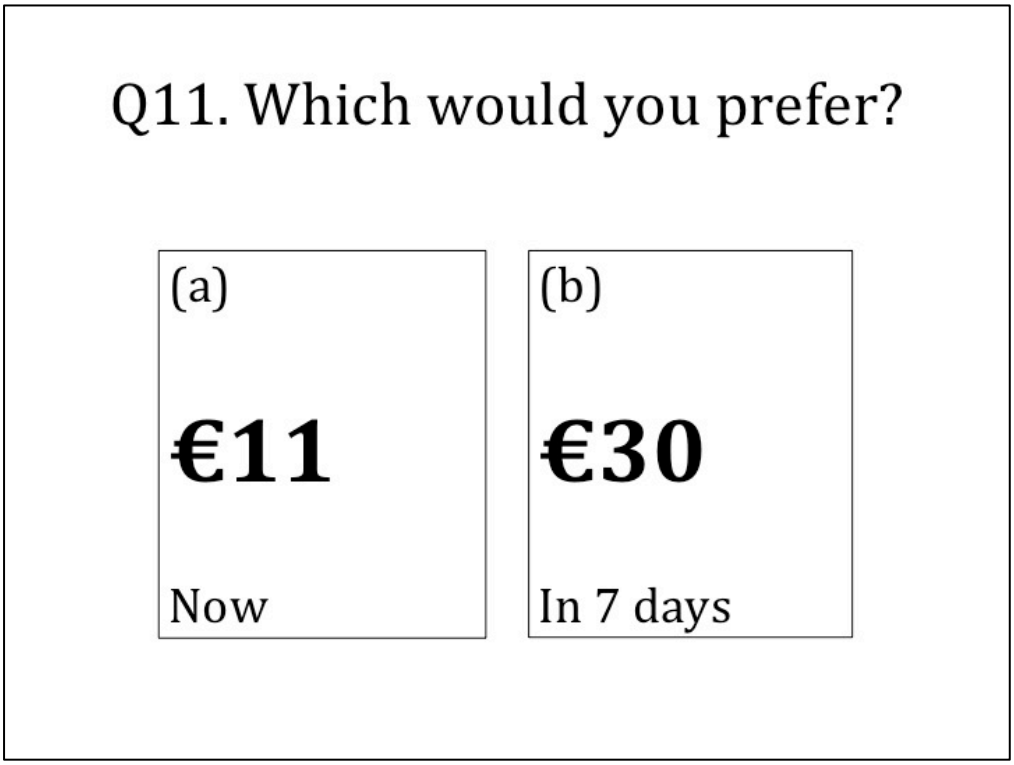

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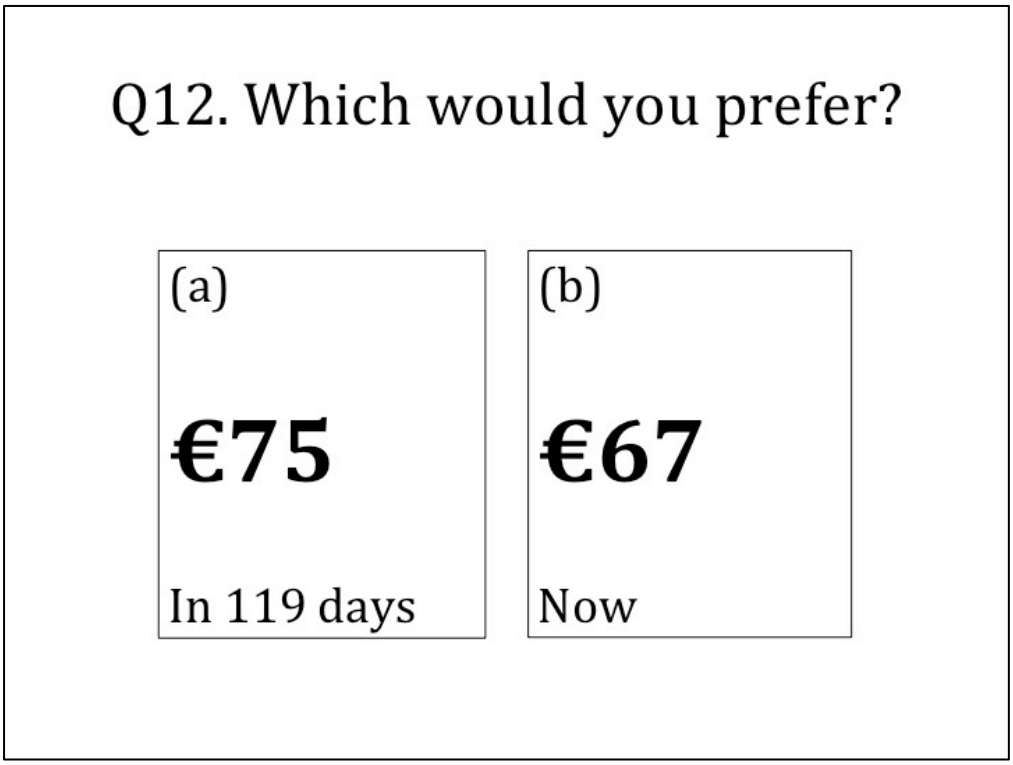

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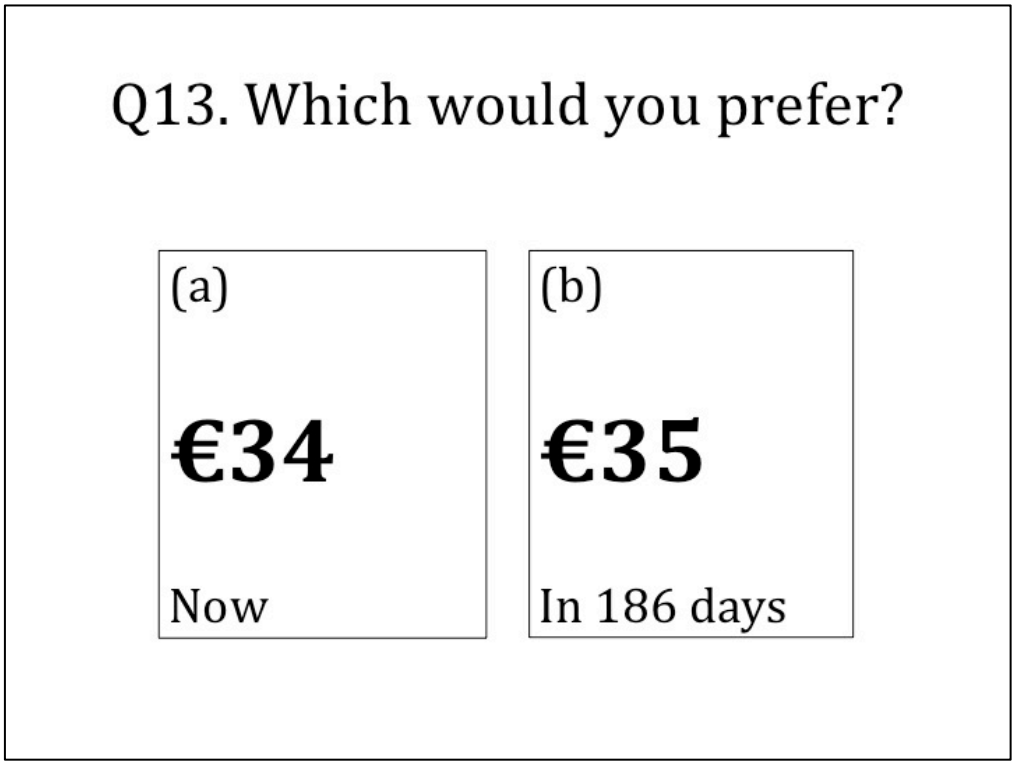

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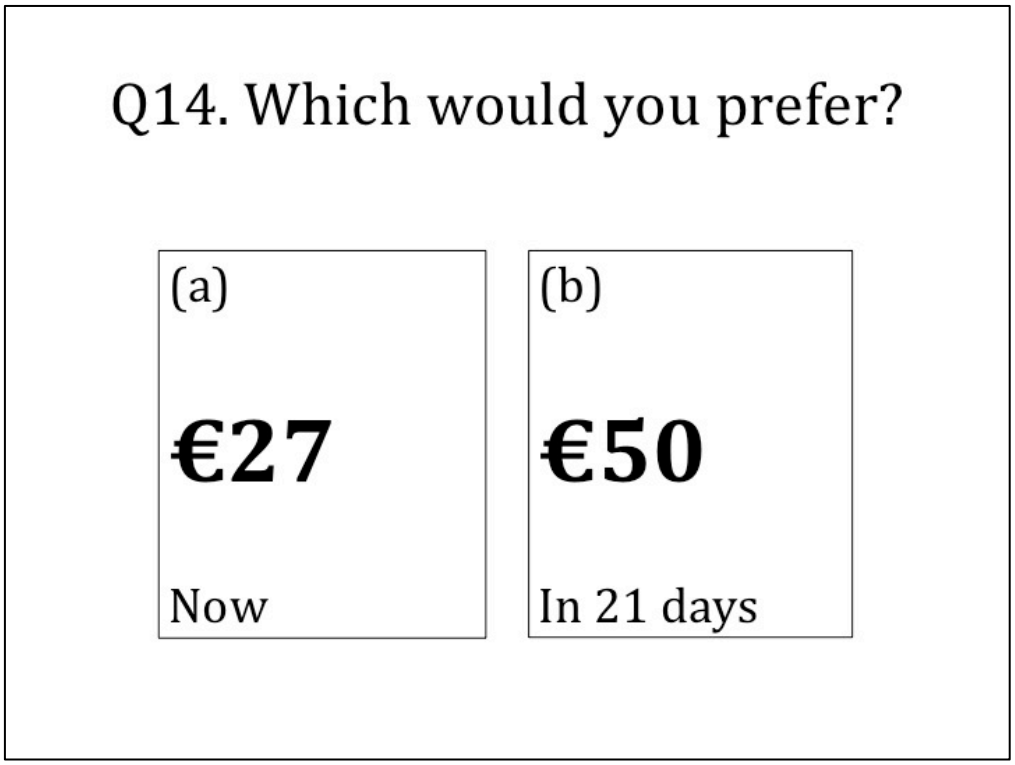

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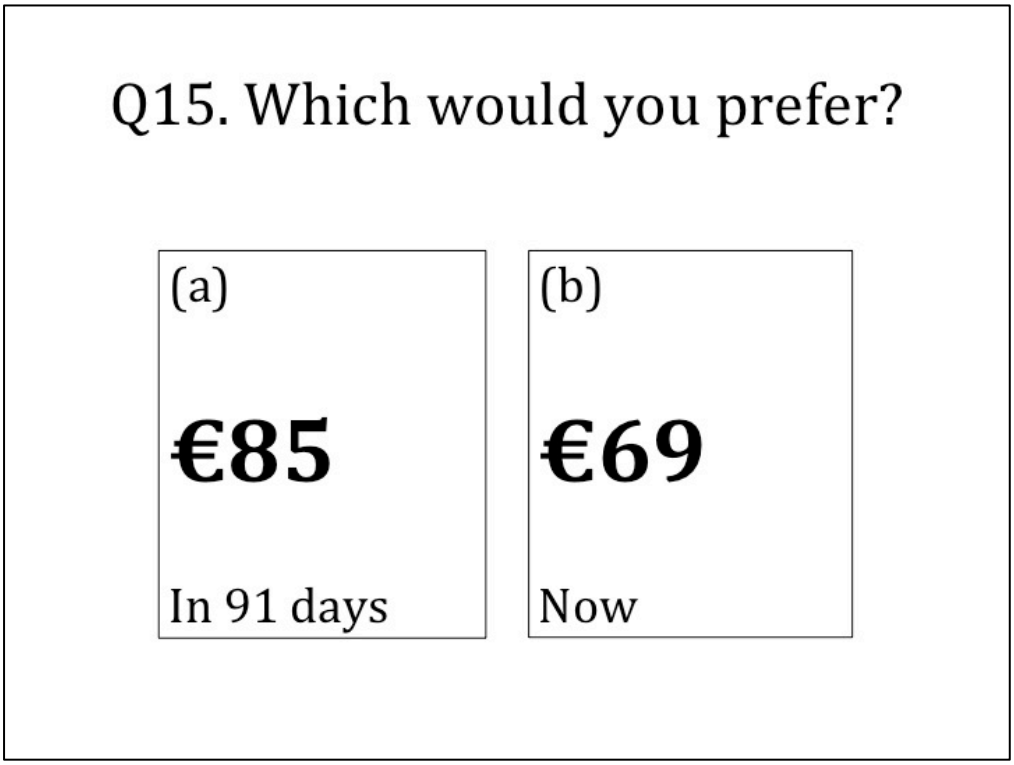

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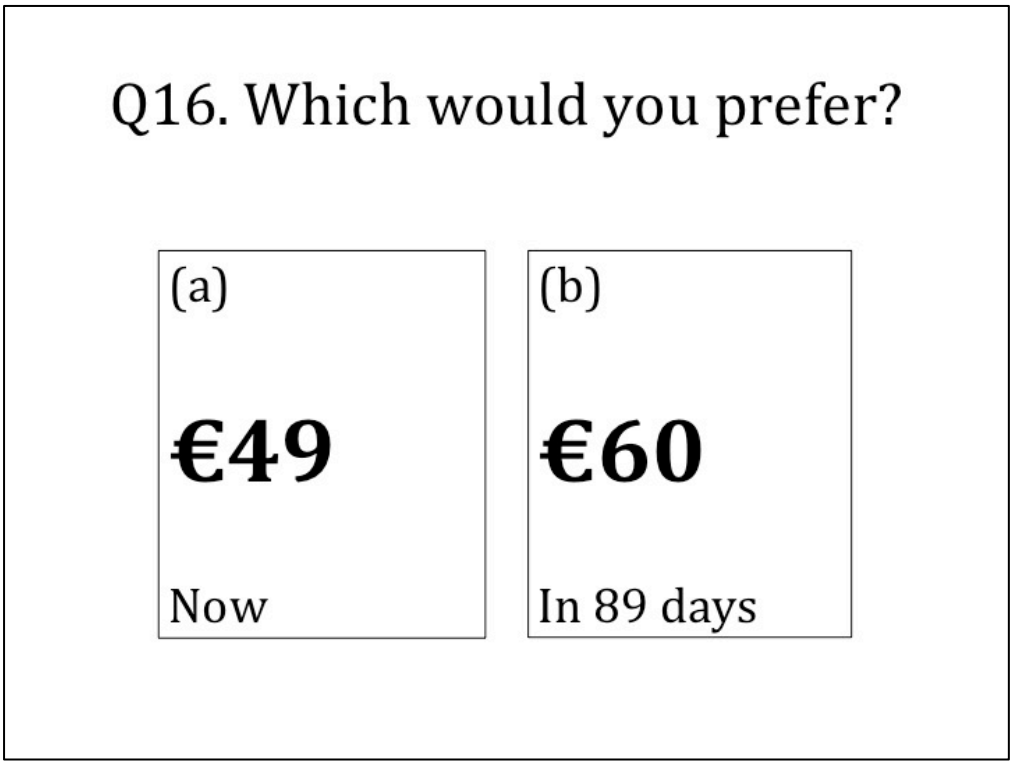

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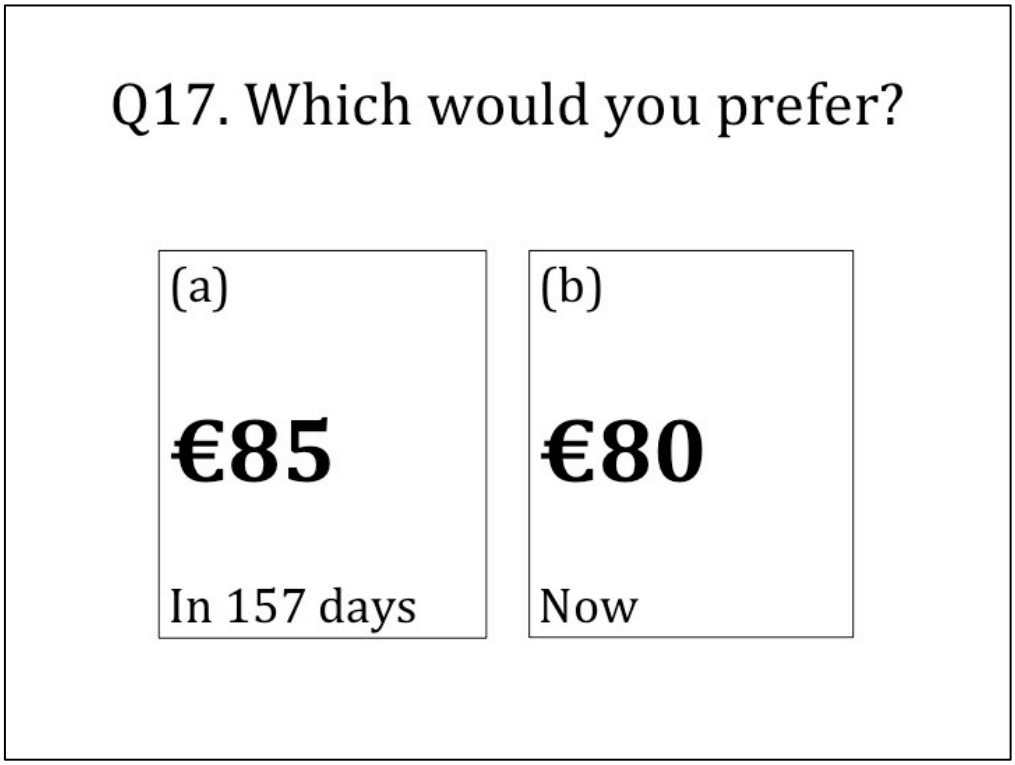

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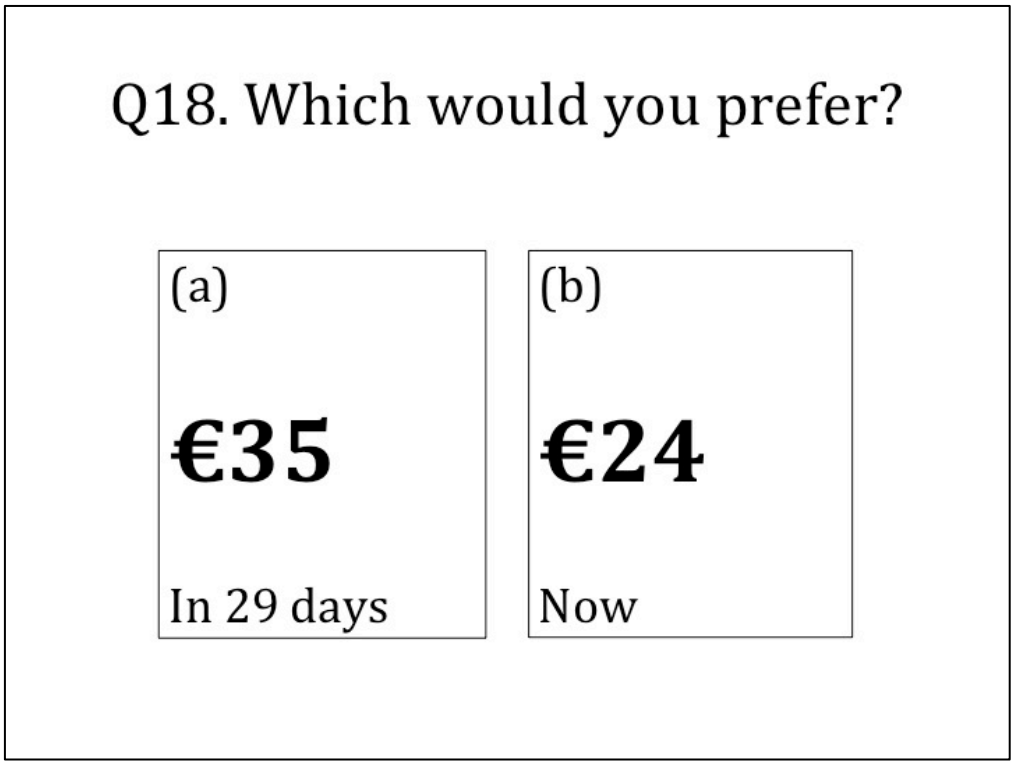

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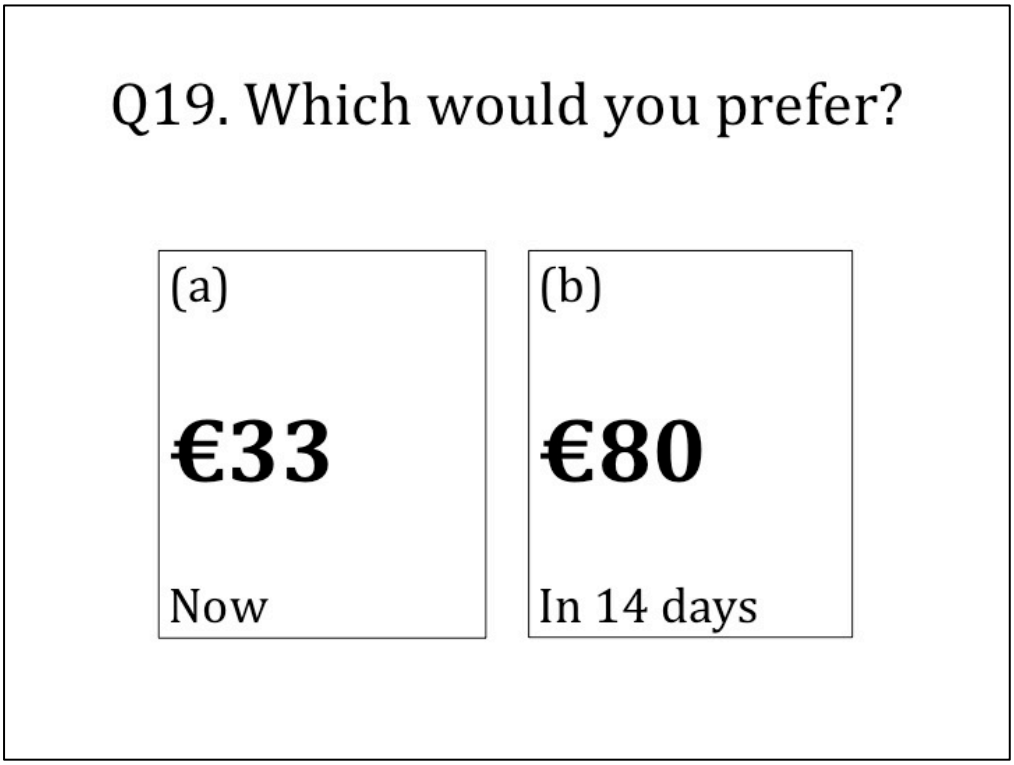

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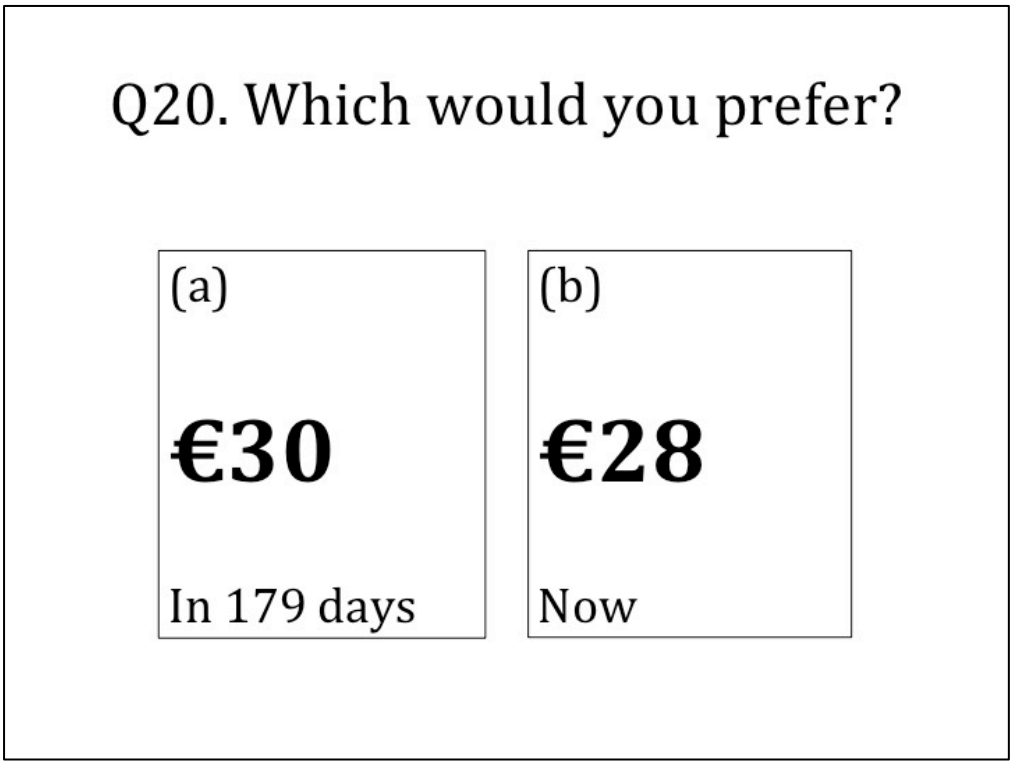

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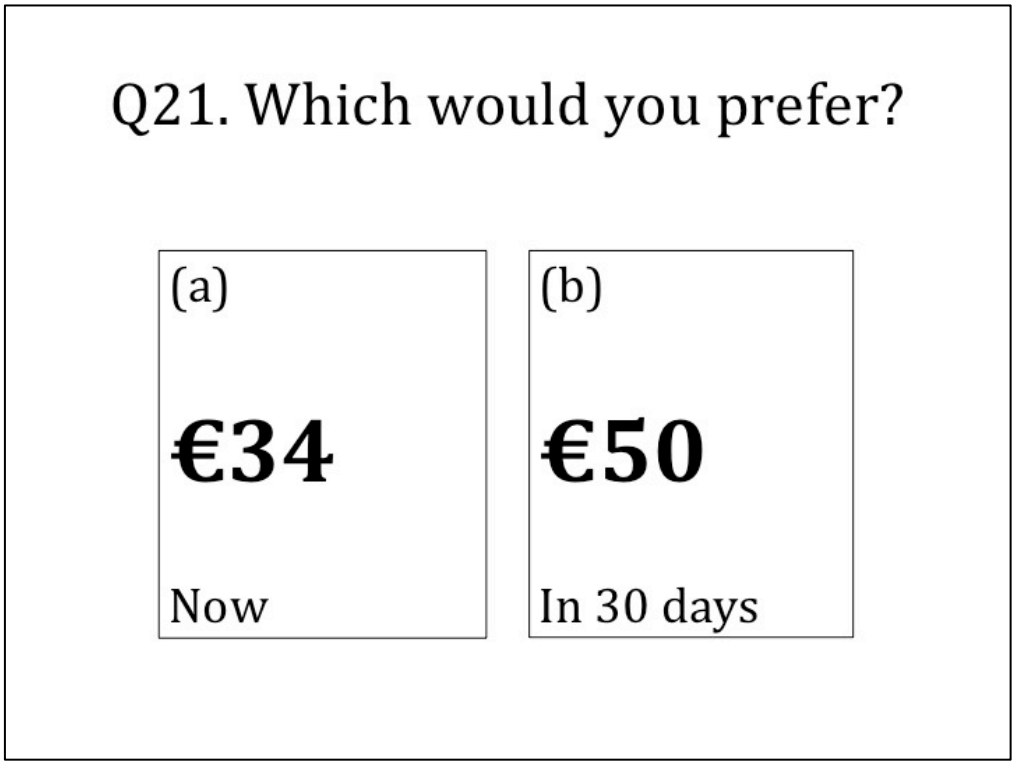

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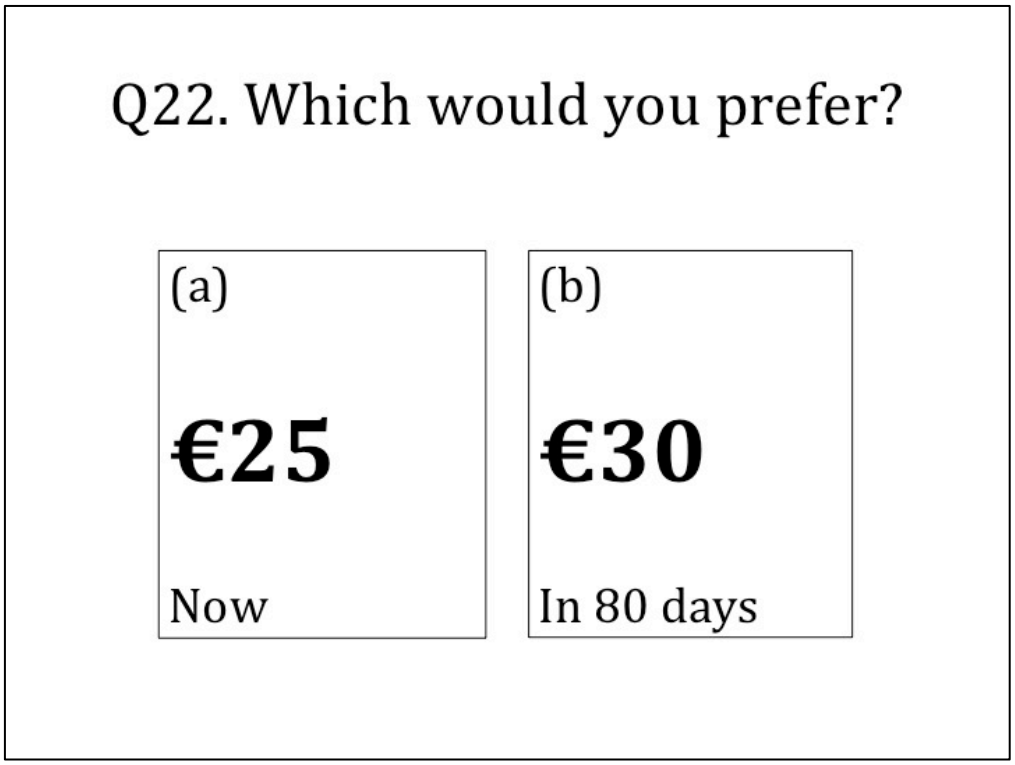

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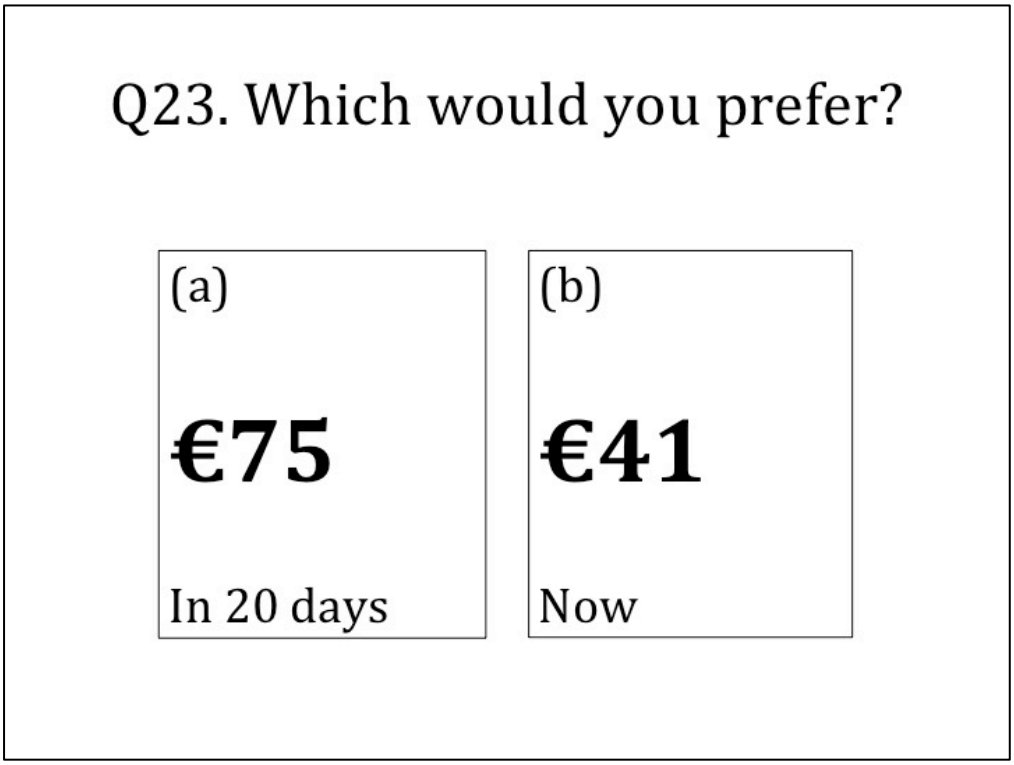

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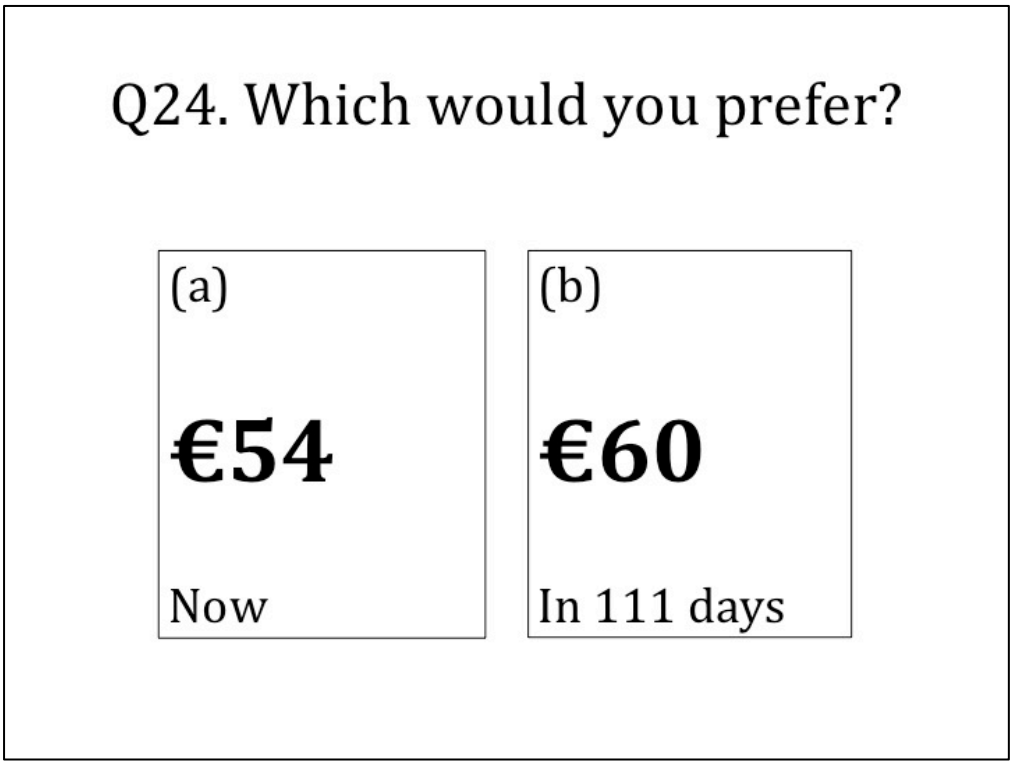

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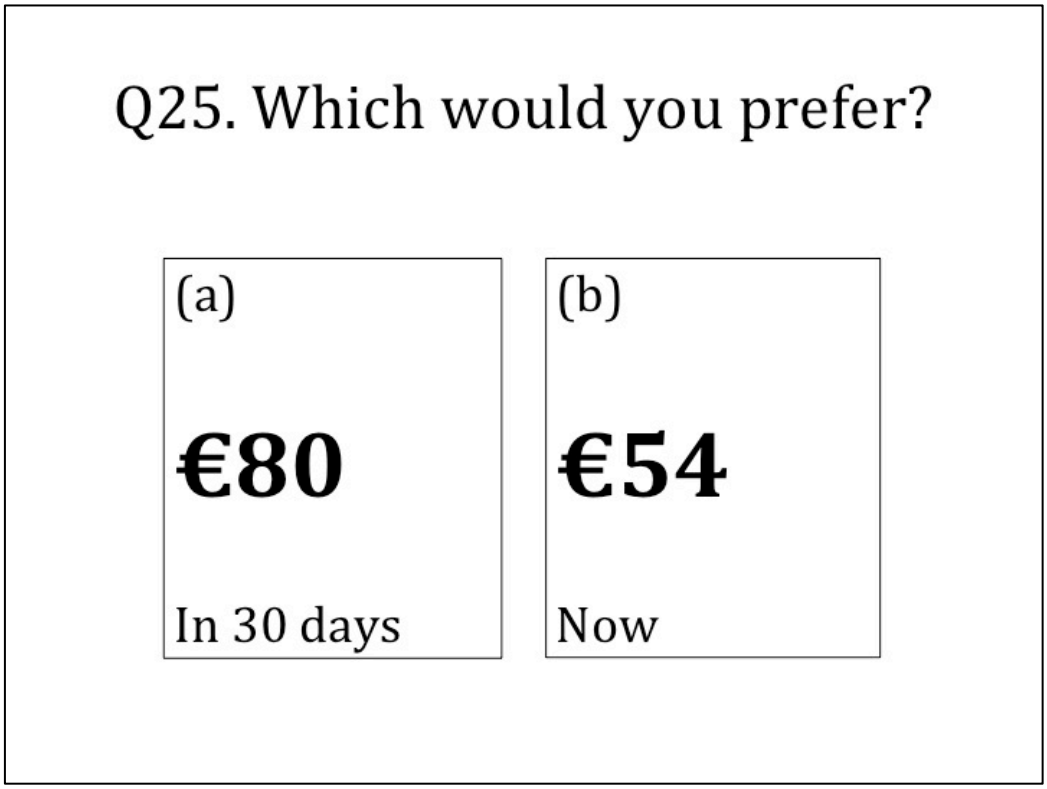

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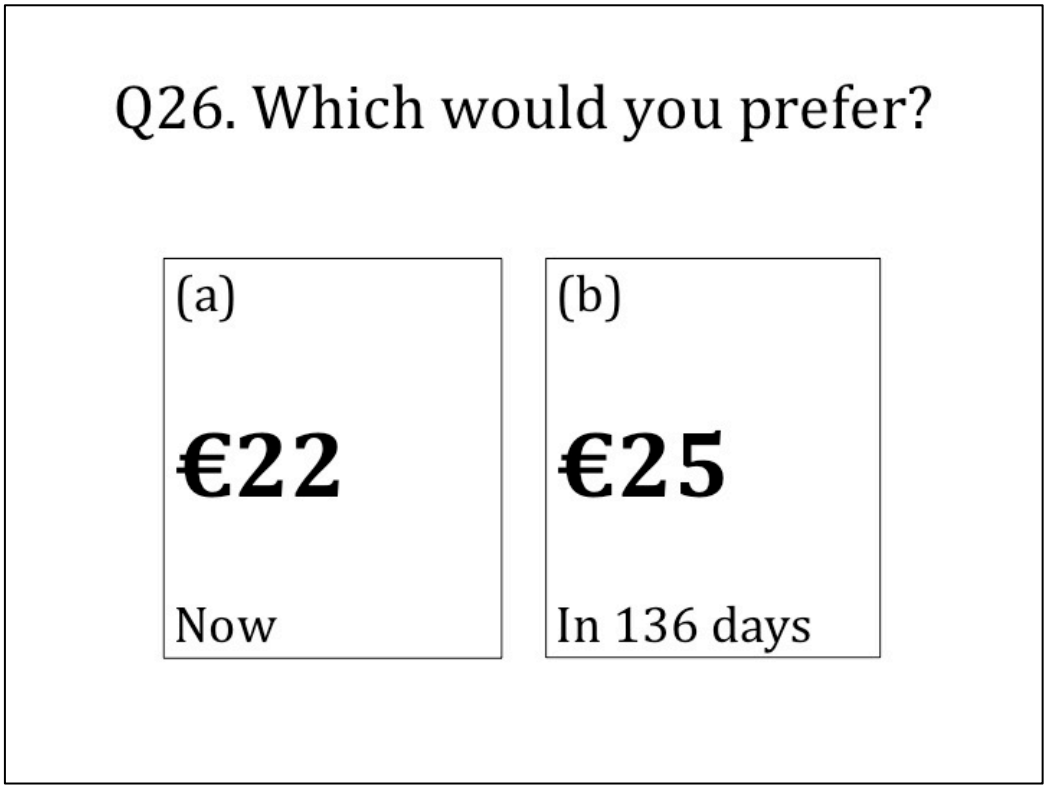

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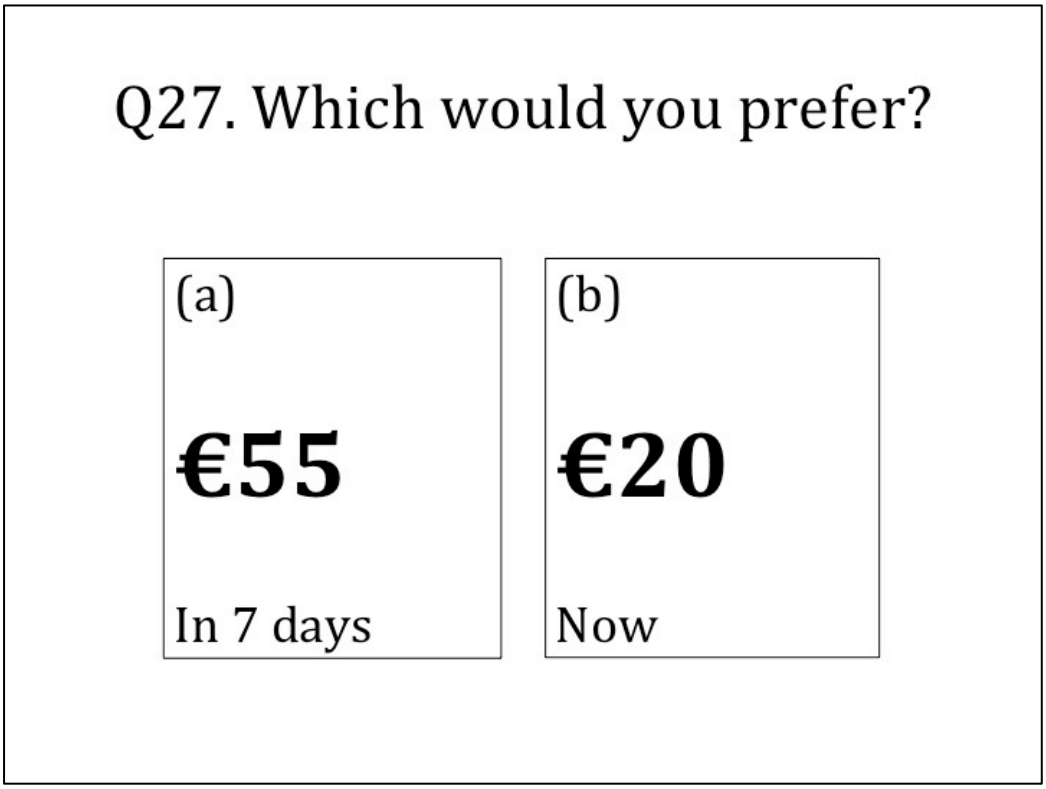

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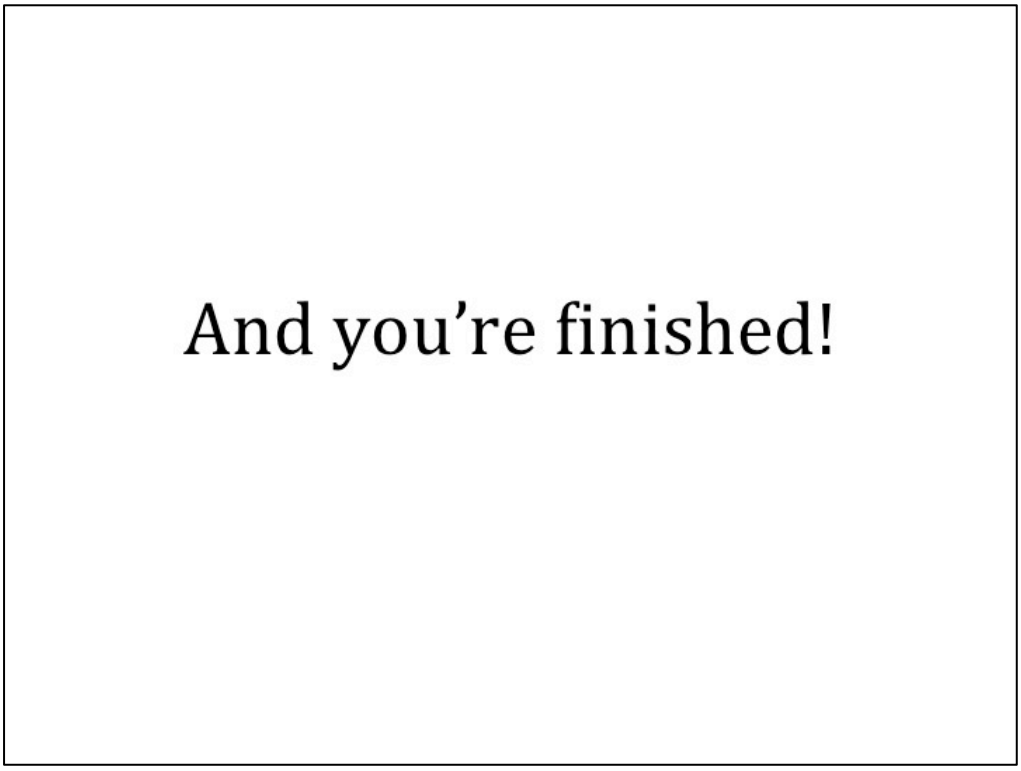


9.22 Appendix V: Kirby's DDQ answer sheet

Please take these choices seriously; you should answer *every question* as though this was real money that you could be offered.

Which of the following choices would prefer? (please circle your answer)

1	**(a)**	or:	**(b)**
2	**(a)**	or:	**(b)**
3	**(a)**	or:	**(b)**
4	**(a)**	or:	**(b)**
5	**(a)**	or:	**(b)**
6	**(a)**	or:	**(b)**
7	**(a)**	or:	**(b)**
8	**(a)**	or:	**(b)**
9	**(a)**	or:	**(b)**
10	**(a)**	or:	**(b)**
11	**(a)**	or:	**(b)**
12	**(a)**	or:	**(b)**
13	**(a)**	or:	**(b)**
14	**(a)**	or:	**(b)**
15	**(a)**	or:	**(b)**
16	**(a)**	or:	**(b)**
17	**(a)**	or:	**(b)**
18	**(a)**	or:	**(b)**
19	**(a)**	or:	**(b)**
20	**(a)**	or:	**(b)**
21	**(a)**	or:	**(b)**
22	**(a)**	or:	**(b)**
23	**(a)**	or:	**(b)**
24	**(a)**	or:	**(b)**
25	**(a)**	or:	**(b)**
26	**(a)**	or:	**(b)**
27	**(a)**	or:	**(b)**

9.23 Appendix W: Scoring Kirby's DDQ

9.23.1 Appendix Wa: DDQ items ordered by rank

Choice trial values, their associated discount rates (*k*). '*k* at Indiff' indicates the value of the discount rate at which the immediate and delayed rewards are of equal value. SIR = smaller, immediate reward; LDR = larger delayed reward. S, M and L represent the small, medium and large delayed reward categories, respectively. Delays are in days.

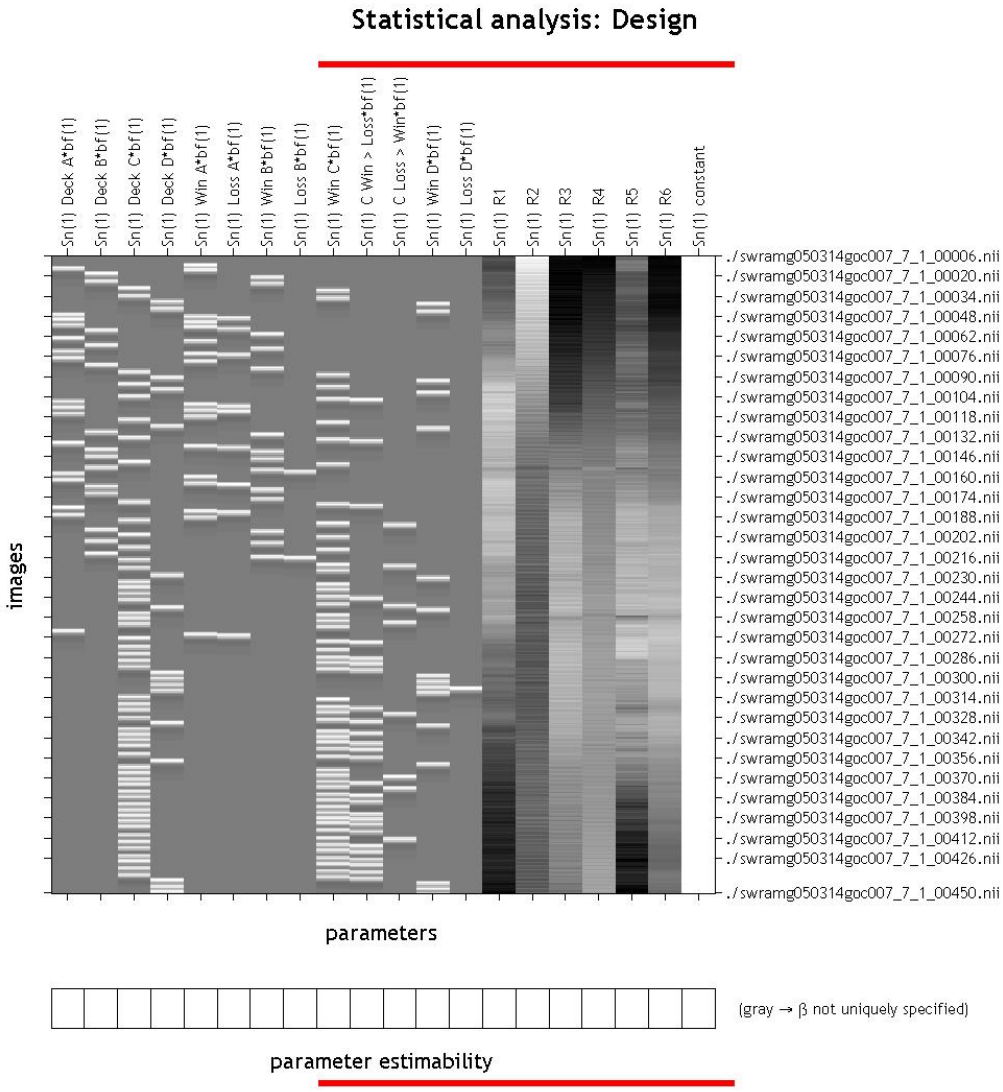
Order	Reward values			*k* at indiff.	*k* rank	LDR size
	SIR	LDR	Delay[a]			
13	$34	$35	186	.00016	1	S
1	$54	$55	117	.00016	1	M
9	$78	$80	162	.00016	1	L
20	$28	$30	179	.00040	2	S
6	$47	$50	160	.00040	2	M
17	$80	$85	157	.00040	2	L
26	$22	$25	136	.0010	3	S
24	$54	$60	111	.0010	3	M
12	$67	$75	119	.0010	3	L
22	$25	$30	80	.0025	4	S
16	$49	$60	89	.0025	4	M
15	$69	$85	91	.0025	4	L
3	$19	$25	53	.0060	5	S
10	$40	$55	62	.0060	5	M
2	$55	$75	61	.0060	5	L
18	$24	$35	29	.016	6	S
21	$34	$50	30	.016	6	M
25	$54	$80	30	.016	6	L
5	$14	$25	19	.041	7	S
14	$27	$50	21	.041	7	M
23	$41	$75	20	.041	7	L
7	$15	$35	13	.10	8	S
8	$25	$60	14	.10	8	M
19	$33	$80	14	.10	8	L
11	$11	$30	7	.25	9	S
27	$20	$55	7	.25	9	M
4	$31	$85	7	.25	9	L

9.23.2 Appendix Wb: DDQ items ordered by delayed reward magnitude

Choice trial values, their associated discount rates (*k*). '*k* at Indiff' indicates the value of the discount rate at which the immediate and delayed rewards are of equal value. SIR = smaller, immediate reward; LDR = larger delayed reward. S, M and L represent the small, medium and large delayed reward categories, respectively. Delays are in days.

	Reward values				
Order	SIR	LDR	Delay[a]	*k* at Indiff.	LDR size
13	$34	$35	186	0.00016	S
20	$28	$30	179	0.00040	S
26	$22	$25	136	0.0010	S
22	$25	$30	80	0.0025	S
3	$19	$25	53	0.0060	S
18	$24	$35	29	0.016	S
5	$14	$25	19	0.041	S
7	$15	$35	13	0.10	S
11	$11	$30	7	0.25	S
1	$54	$55	117	0.00016	M
6	$47	$50	160	0.00040	M
24	$54	$60	111	0.0010	M
16	$49	$60	89	0.0025	M
10	$40	$55	62	0.0060	M
21	$34	$50	30	0.016	M
14	$27	$50	21	0.041	M
8	$25	$60	14	0.10	M
27	$20	$55	7	0.25	M
9	$78	$80	162	0.00016	L
17	$80	$85	157	0.00040	L
12	$67	$75	119	0.0010	L
15	$69	$85	91	0.0025	L
2	$55	$75	61	0.0060	L
25	$54	$80	30	0.016	L
23	$41	$75	20	0.041	L
19	$33	$80	14	0.10	L
4	$31	$85	7	0.25	L

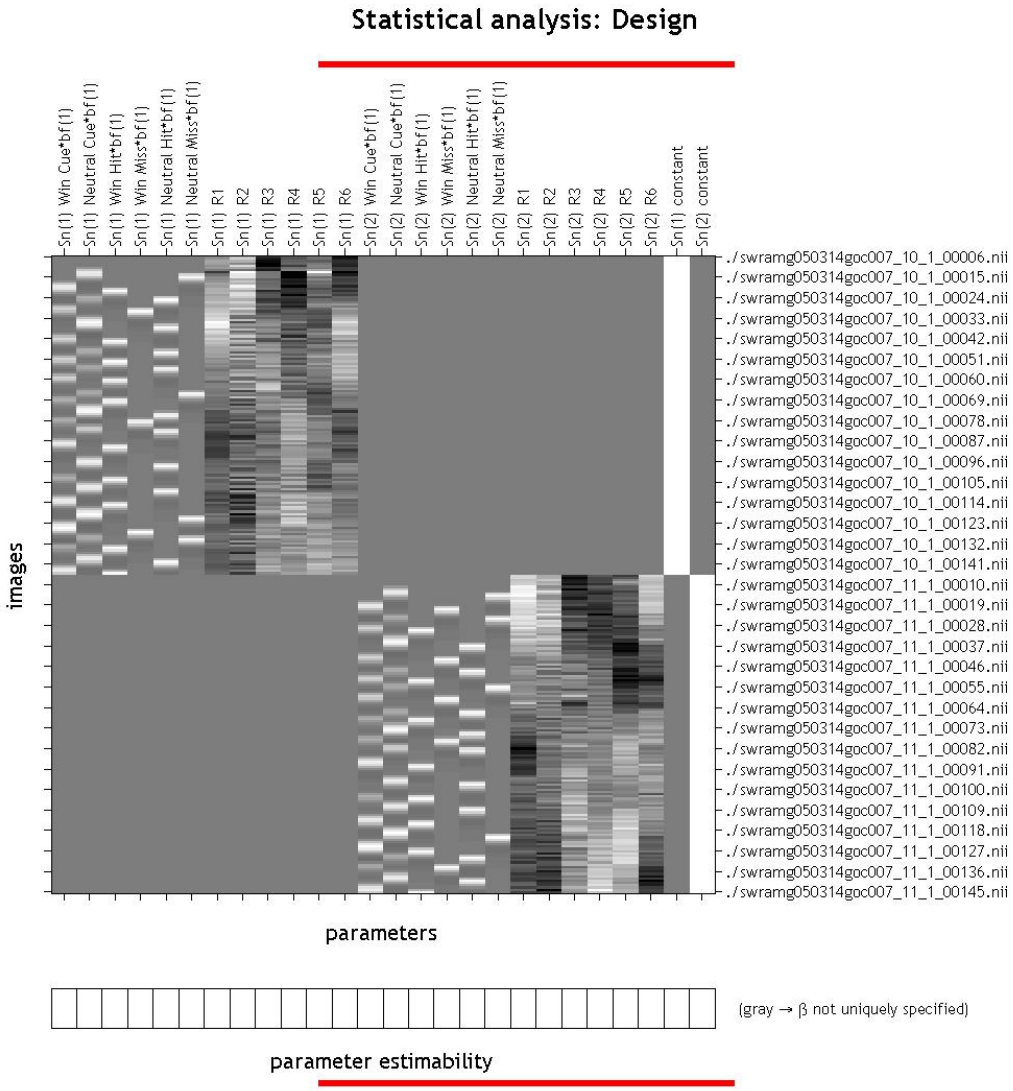
9.24 Appendix X: Sample IGT first level design matrix, participant 7



Design description...

Basis functions :	hrf
Number of sessions :	1
Trials per session :	13
Interscan interval :	2.00 {s}
High pass Filter :	Cutoff: 128 {s}
Global calculation :	mean voxel value
Grand mean scaling :	session specific
Global normalisation :	None

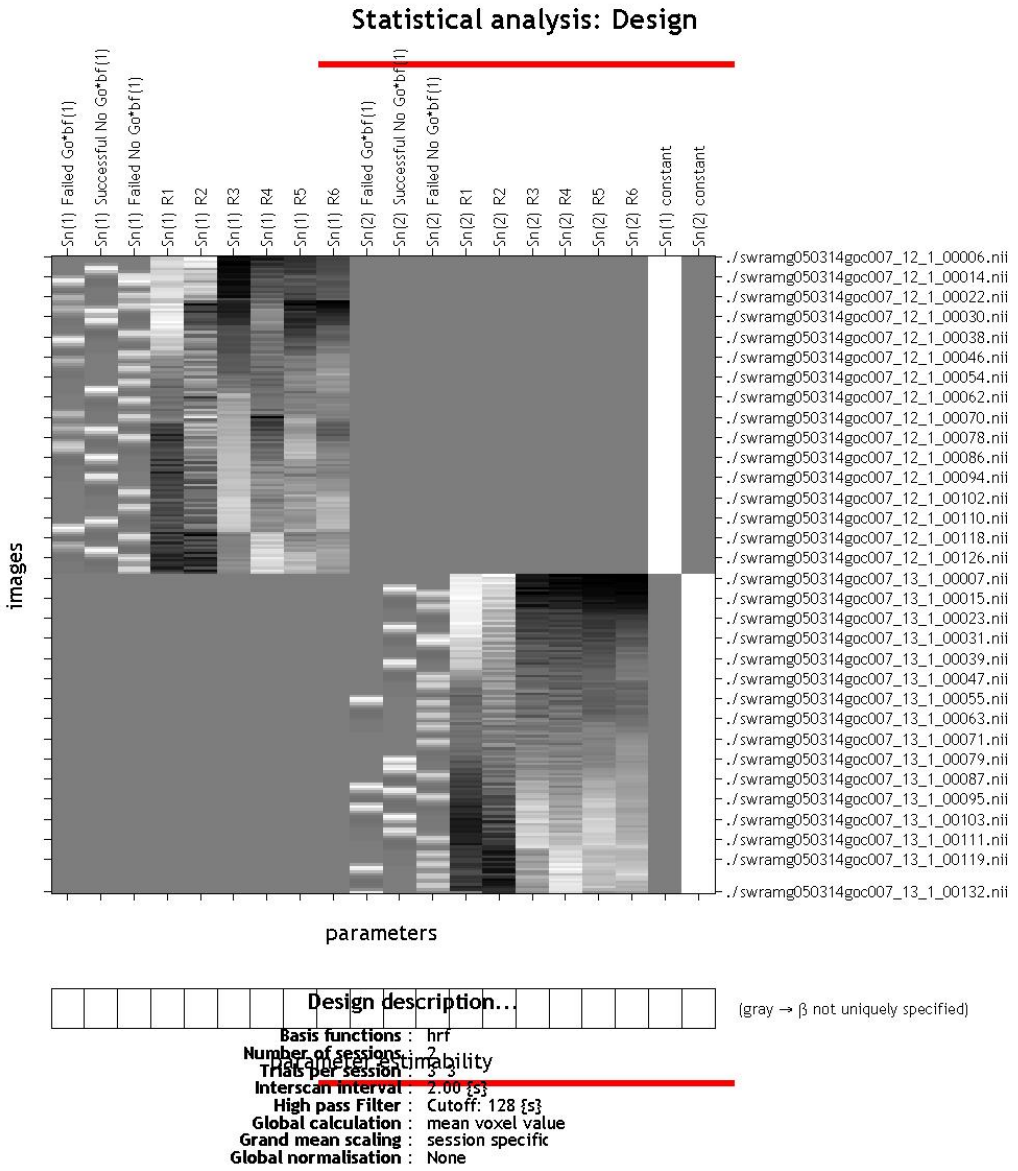
9.25 Appendix Y: Sample MID first level design matrix, participant 7



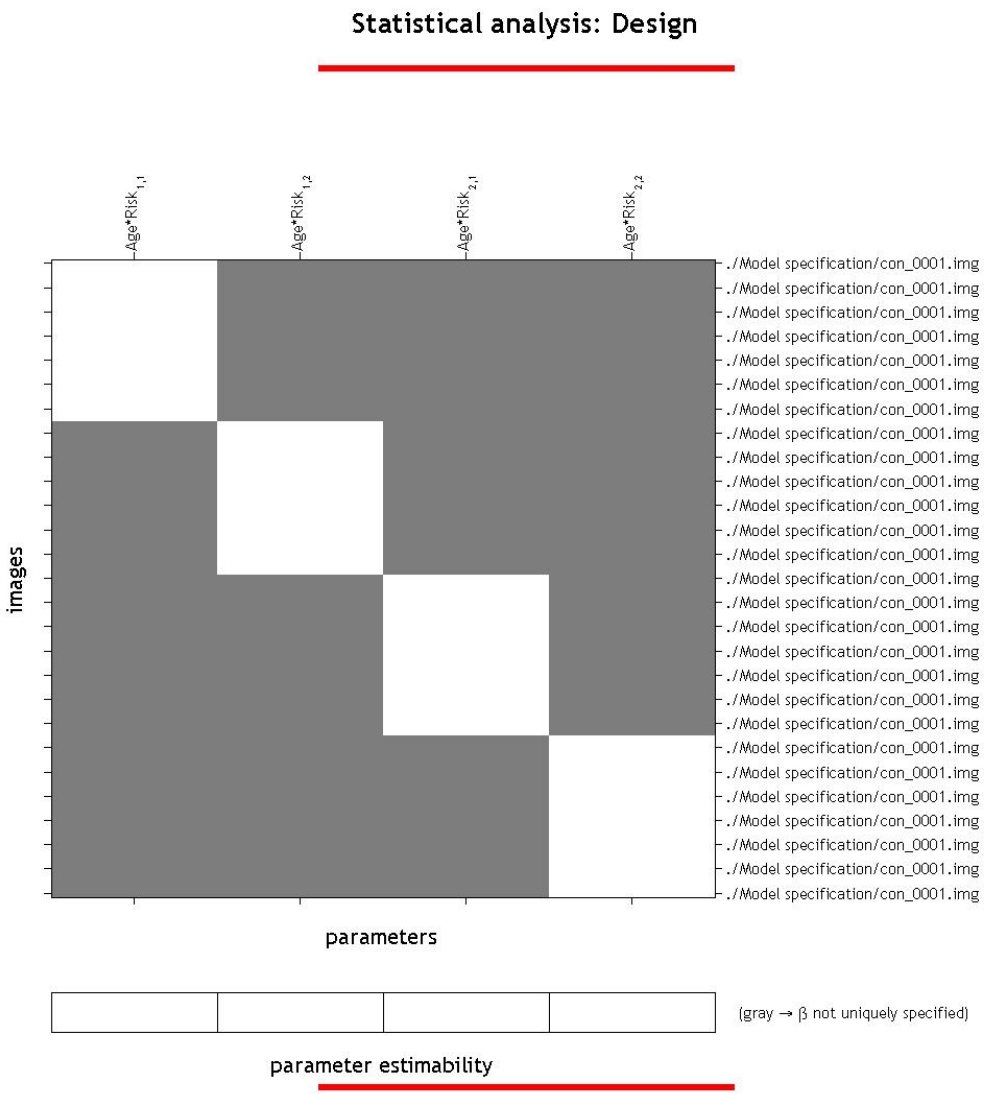
Design description...

Basis functions :	hrf
Number of sessions :	2
Trials per session :	6 6
Interscan interval :	2.00 {s}
High pass Filter :	Cutoff: 128 {s}
Global calculation :	mean voxel value
Grand mean scaling :	session specific
Global normalisation :	None

9.26 Appendix Z: Sample Go/No Go first level design matrix, participant 7

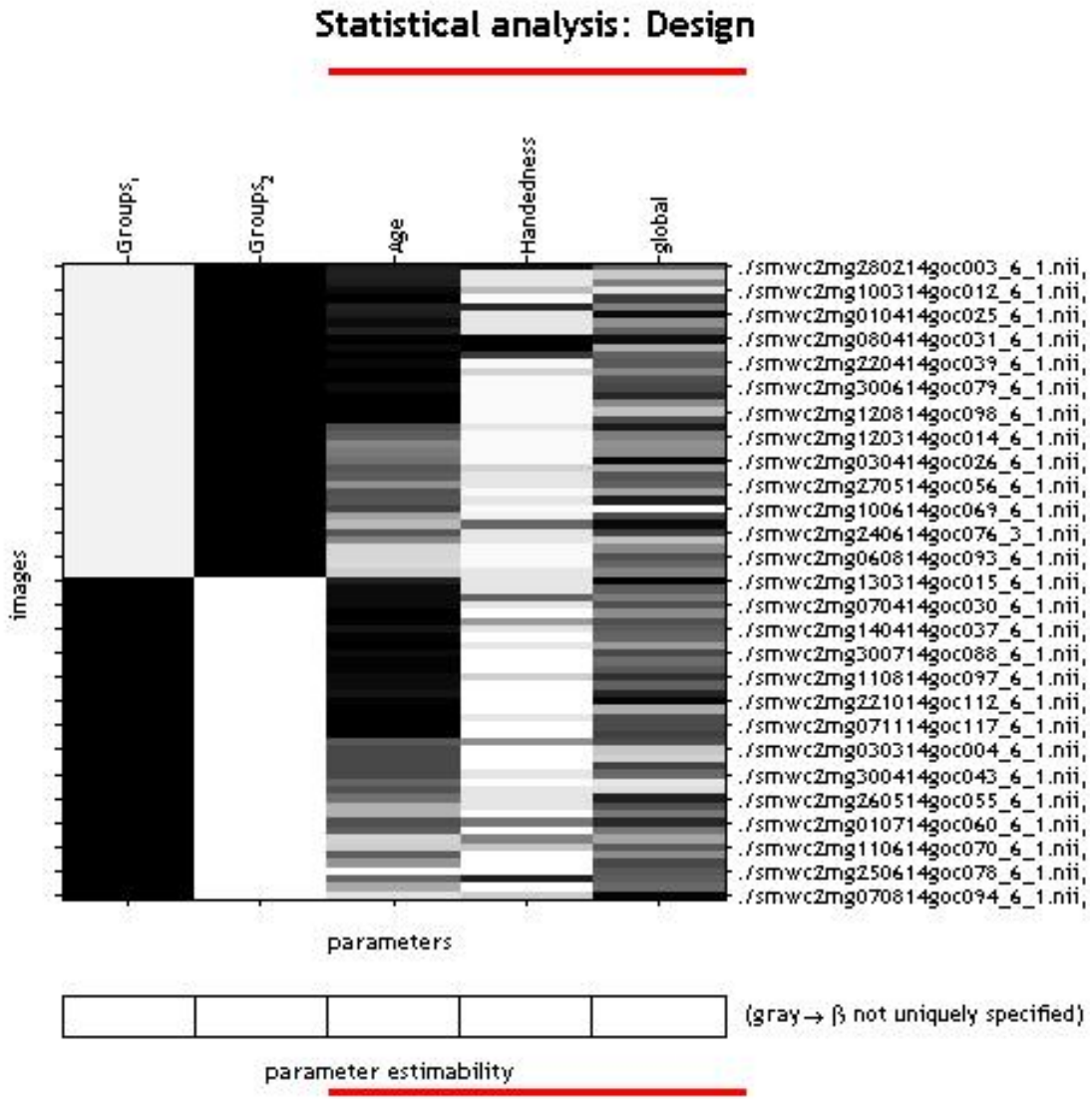


9.27 Appendix AA: Sample second level factorial design matrix



9.28 Appendix AB: Sample VBM group level design matrices

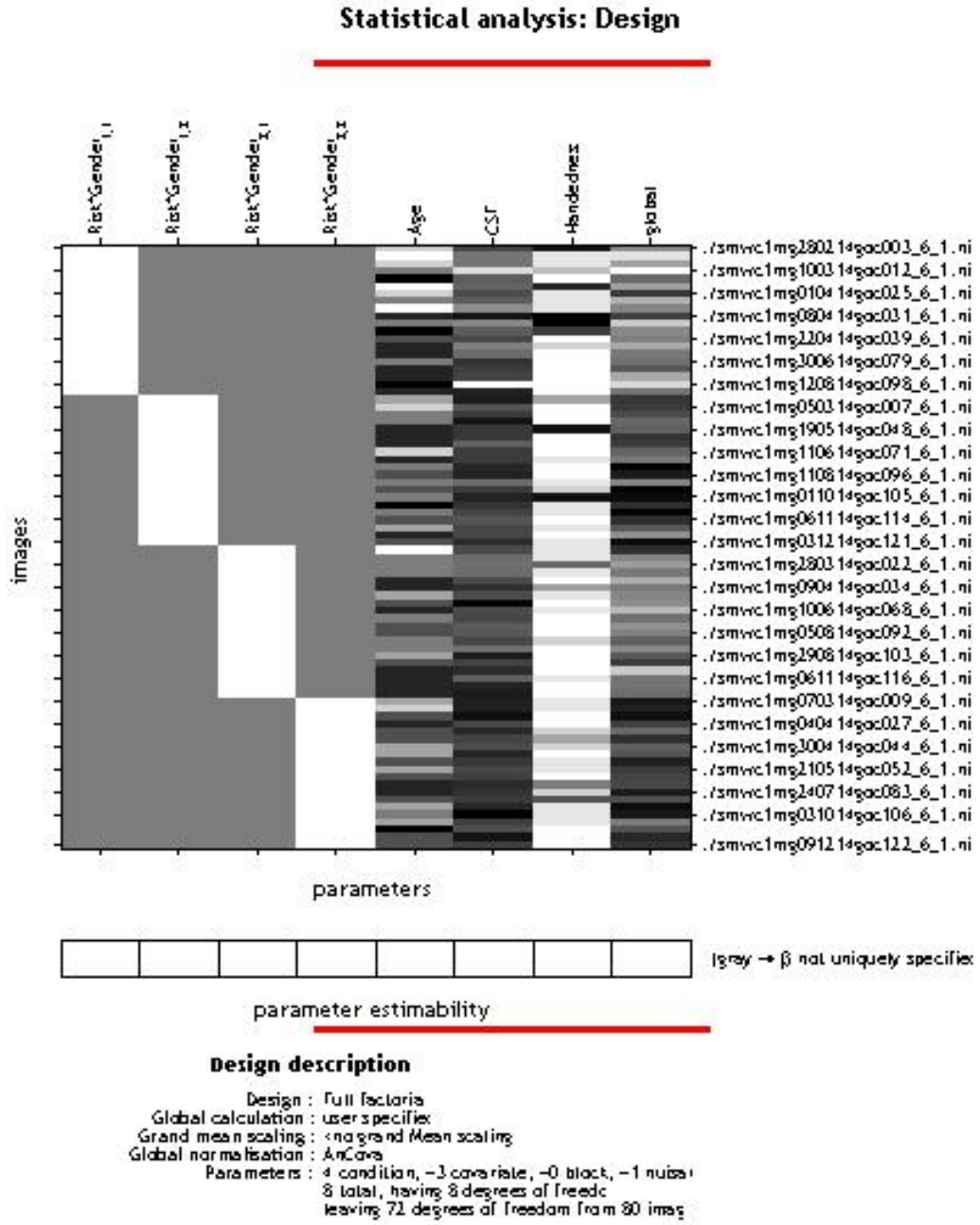
9.28.1 Appendix ABa: All males



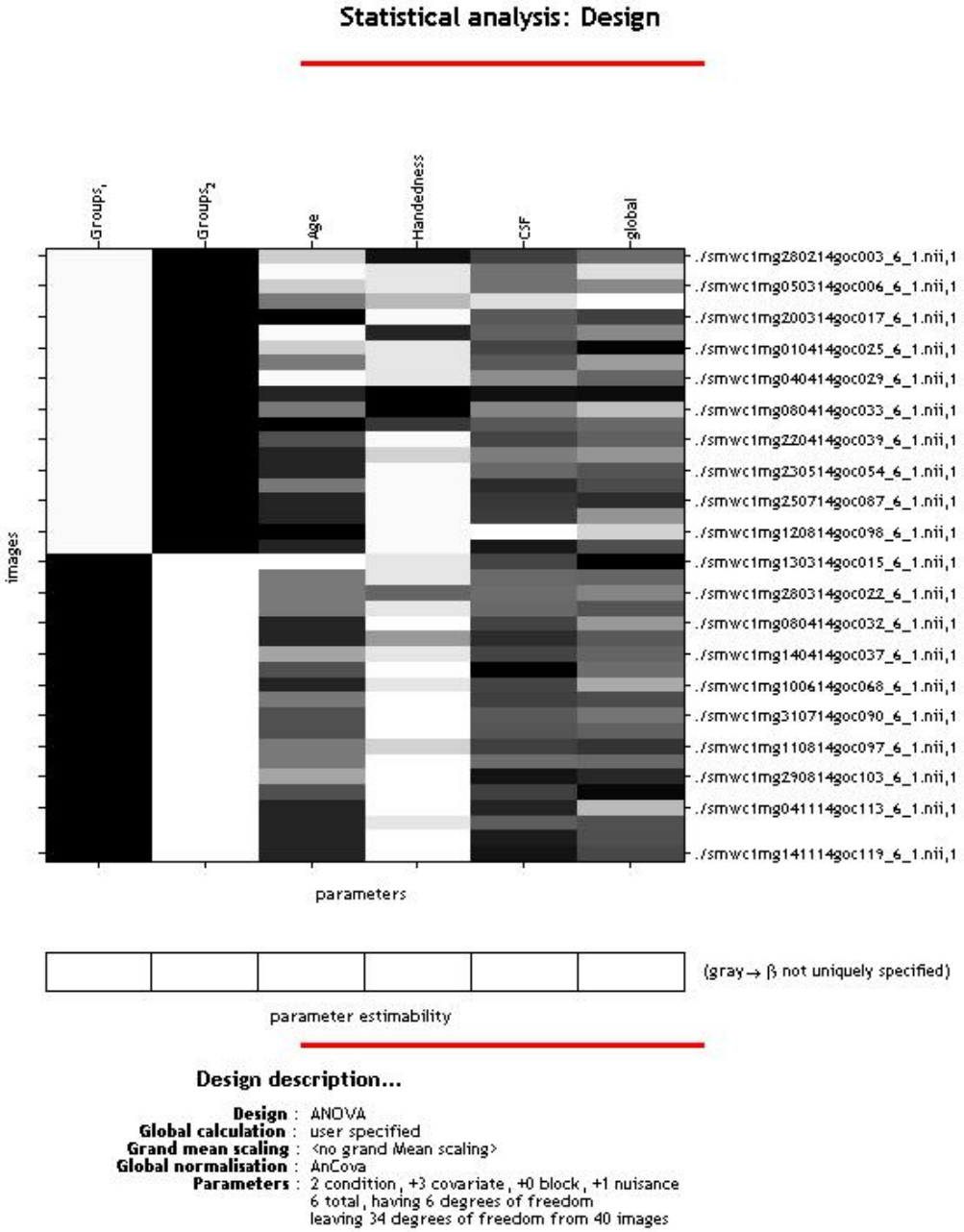
Design description...

Design : ANOVA
Global calculation : user specified
Grand mean scaling : <no grand Mean scaling>
Global normalisation : AnCova
Parameters : 2 condition, +2 covariate, +0 block, +1 nuisance
5 total, having 5 degrees of freedom
leaving 74 degrees of freedom from 79 images

9.28.2 Appendix ABb: All younger individuals



9.28.3 Appendix ABc: Younger males



Design description...

Design : ANOVA
Global calculation : user specified
Grand mean scaling : <no grand Mean scaling>
Global normalisation : AnCova
Parameters : 2 condition, +3 covariate, +0 block, +1 nuisance
6 total, having 6 degrees of freedom
leaving 34 degrees of freedom from 40 images

9.29 Appendix AC: Edinburgh Handedness Inventory (EHI)

EDINBURGH HANDEDNESS INVENTORY

Name .....

Participant Number.....

Please indicate your preference in the use of hands in the following activities *by putting + in the appropriate column.* Where the preference is so strong that you would never try to use the other hand unless absolutely forced to, *put* ++. If in any case you are really indifferent *put* + in both columns.

Some of the activities require both hands. In these cases the part of the task, or object, for which hand preference is wanted is indicated in brackets.

Please try to answer all questions, and only leave a blank if you have no experience of the object or task.

		LEFT	RIGHT
1	Writing		
2	Drawing		
3	Throwing		
4	Scissors		
5	Toothbrush		
6	Knife (without fork)		
7	Spoon		
8	Broom (upper hand)		
9	Striking match (match)		
10	Opening box (lid)		
i	Which foot do you prefer to kick with?		
ii	Which eye do you use when using only one?		

L.Q: Leave these spaces blank DECILE: