Examination of Financial Decision-Making in Sub-Threshold Gamblers

by

Victoria Yung Mei Suen

A thesis submitted in partial fulfillment of the requirements for the degree of

Doctor of Philosophy

Department of Psychiatry

University of Alberta

Abstract

Gambling is a recreational activity enjoyed by many; however, for some this enjoyment can lead to the development of Gambling Disorder with financial, personal, and psychological ramifications. The study of pathological gamblers has targeted the psychological, behavioural and neurobiological aspects and points to dysfunctions in decision-making and reward processing as common across pathological gamblers. Brain regions typically studied include the ventromedial prefrontal cortex, the anterior cingulate cortex, and the ventral striatum. What is less clear is how the spectrum of gamblers compares to non-gamblers. Considerable attention has been paid to those who are clinically diagnosed; however, there remains a group of frequent gamblers who do not meet the requirements for diagnosis, a sub-threshold group. We examined the brain activation patterns of sub-threshold gamblers in an investment decision-making task to determine if and how decision-making in this group differs from those who do not gamble. Additionally, it is well recognized that individuals follow "Expert" advice, even when flawed and offers no advantage, and sometimes leads to disadvantages. The neurobiology underlying this is uncertain, and in particular there is an incomplete understanding of which brain regions are most involved when individuals chose to disobey an expert. To study this we examined functional magnetic resonance imaging (fMRI) differences during an investment game where non-gambling subjects received differentially credible investment advice. We also wished to examine how sub-threshold gamblers compare to non-gamblers in the presence of advice.

Participants (n = 64) played a novel investment game developed specifically for this study, in which they could Buy or Not Buy a sequence of stocks. The better they did, the more

money they made. In our first study, non-gamblers received either "Expert" advice or "Peer" advice. Those receiving Expert advice were told the advice came from a certified financial "Expert". Those receiving Peer Advice were told the advice was that of the student administering the scans, who deliberately dressed and acted casually. Both streams of advice were predetermined and identical. The advice was scripted to be helpful initially, but progressively worsened as the task continued, becoming 100% wrong by the end of the task. In our second study, non-gamblers and gamblers both completed the task under the assertion that the advice being received was from an "Expert". Psychological measures were also administered to determine links between patterns of brain activation and psychological traits.

Subjects receiving Expert Advice followed the advice significantly longer on average, even though this was progressively worse advice. Thus, following Expert advice had poorer consequences for individuals, but this did not dissuade them from continuing to follow the advice. In contrast, when subjects disobeyed Expert advice they exhibited significant anterior cingulate cortex and superior frontal gyrus activation relative to those disobeying Peer advice. These findings may suggest that in subjects who defy authority, or believe they are doing so (in this case by disobeying an "Expert") there is increased activation of these two brain regions. This may have relevance to several areas of behaviour, and the potential role of these two brain regions in regard to disobedience behaviour requires further study. Sub-threshold gamblers displayed more rational decision-making by following bad advice less than non-gamblers. However, in contrast to previous research linking ventromedial prefrontal cortex deactivation to decision-making in pathological

gamblers, our sub-threshold gamblers did not display this pattern of deactivation. Our gamblers reported greater risk tolerance across multiple domains, not just greater financial risk tolerance. However, our findings also failed to support previous suggestions that impulsivity significantly explains gambling severity, is linked to lowered self-esteem or greater rates of adverse childhood experience thus gambling related psychological factors remain unclear. Our study indicates that dysfunctions in decision-making and reward processing may differ across non-gamblers, sub-threshold gamblers, and pathological gamblers thus future research including all three groups may be pertinent.

Preface

This thesis is an original work by Victoria Suen. The research project, of which this thesis is a part, received research ethics approval from the University of Alberta Research Ethics Board, Project Name "Examination of regional brain changes occurring during cognitive choices: an fMRI study utilizing an investment paradigm.", No. 14124, January 31, 2011.

Some of the research conducted for this thesis forms part of a research collaboration within the University of Alberta, led by Dr. Peter H. Silverstone from the Department of Psychiatry, with Dr. Randall K. Morck as lead advisor from the Alberta School of Business. Dr. Matthew R.G. Brown and myself designed the task referred to in Chapter 5. The literature reviews in chapters 2-4 are my original work and the data analyses in chapters 6-8 are my original work, with input from Dr. Matthew R.G. Brown. Dr. Ivor Cribben and myself conducted the data analyses in chapter 9.

Parts of this thesis have been published as Suen, V.Y.M., M.R.G. Brown, R.K. Morck and P.H. Silverstone (2014). "Regional brain changes occurring in disobedience to "experts" in financial decision-making." PLoS One 9:e87321. I was responsible for the data collection as well as the manuscript composition with input from M.R.G. Brown, R.K. Morck and P.H. Silverstone. M.R.G. Brown assisted me with the analysis.

Acknowledgements

I would like to thank Dr. Peter Silverstone for his enthusiasm, encouragement, and continuous support of my PhD research. The opportunities that Dr. Silverstone has presented me with, along with the belief that he has in me, have allowed me to grow as a student, researcher, as well as a person, and I am so very thankful. I would like to thank my co-supervisor, Dr. Randall Morck, for the immense knowledge that he brought to this research. I truly believe him to be an exceptionally gifted teacher and researcher. I would also like to thank Dr. Alan Wilman for his input into this research throughout the past 4 years and for helping me to understand the complexities of fMRI despite my wariness of all things related to physics.

My sincere thanks extend to the members of my examination committee Dr. Frank MacMaster, Dr. Garry Smith, and Dr. Ivor Cribben for the in-depth and thought-provoking discussion.

Particular thanks also goes out to Dr. Matthew Brown, without whom this research could not have occurred, and Andrea Shafer who mentored me through the confusion that is spm8. I must also thank Dr. Esther Fujiwara who provided me with tremendous guidance and support when the stress of grad school was almost too much to bear.

I am so grateful for all that Tara Checknita has done for me. I will miss the continuous flow of candy, the bonding over beach vacation stories during lunches, and the comfort of knowing that someone is always looking out for me.

To the friends I have made here in Edmonton, you kept me sane when life was full of insanity. The memories and laughter that we shared are irreplaceable. To my friends back in Winnipeg, I thank you for keeping me grounded and reminding me that I will always have a home there.

Stephanie Graham, you have always been and will always be my person.

My love and gratitude go to Scott Travis, who experienced the ups and downs of this degree with me. Thank you for being my comfort when things got tough. Thank you for always encouraging me. I could not have gotten through these past 4 years without you.

And finally, I thank my entire family for their unwavering support. I feel so fortunate to have siblings and family who I can share this accomplishment with. To my parents, Victor Suen and Eva Wong: You have given me such a full life, ripe with opportunities to learn and grow. Any success that I have achieved or will achieve is due to the belief you put and the values that you instilled in me. I am so fortunate to call you my parents and I hope that I will continue to make you proud.

List of Tables

List of Figures

List of Abbreviations

ACC – anterior cingulate cortex

ACE – Adverse Childhood Experience

AMPH – D-amphetamine

ANCOVA – analysis of covariance

ANOVA – analysis of variance

BART – Balloon Analog Risk Task

BIS-11 – Barratt Impulsivity Scale

BOLD – blood-oxygen-level dependent signal

CS – Conditioned Stimulus

DG – Dictator Game

dlPFC – dorsolateral prefrontal cortex

dmPFC – dorsomedial prefrontal cortex

DOSPERT – Domain-Specific Risk-Taking Scale

DSM – Diagnostic Statistical Manual

EEG - Electroencephalography

EMGs – Electronic gambling machines

fMRI – functional magnetic resonance imaging

GABS – Gambling Attitudes and Beliefs Scale

GDT – Game of Dice Task

ICD – Impulse control disorder

IGT – Iowa Gambling Task

MEG - magnetoencephalography

MNI – Montreal Neurological Institute

NAcc – Nucleus Accumbens

NaCl – Sodium Chloride

OFC – orbitofrontal cortex

PD – Prisoner's Dilemma Game

PET – Positron emission tomography

PGSI – Problem Gambling Severity Index

sAA – alpha-amylase concentration

TG – Trust Game

UCS – unconditioned stimulus

UG – Ultimatum Game

vlPFC – ventrolateral prefrontal cortex

vmPFC – ventromedial prefrontal cortex

WCST – Wisconsin Card Sorting Task

Chapter 1. Introduction

1.1 Introduction

Risks are inherent in life and to thrive in society. These occur in multiple areas of existence, but in current societies financial risks are particularly important. This can be as simple as the decision to buy a house (compared to renting) or to lease a car (compared to buying). However, it is also clear that financial risks can yield massive profits for an individual or company. Despite the importance of managing financial risks, decision-making in this area is often completed with significant uncertainty. Information must be gathered and processed so that any decision is a function of payoffs and probabilities; thus, economic models have suggested that investors are rational beings, relying on mathematically defined consistencies to determine when to buy, not buy, or sell a stock (Arrow 1951, Arrow and Debreu 1954). How economically successful in life we are is dependent on how well we can decipher this information. How successful an economy remains is dependent, in part, on how investors make financial decisions in the stock market. However, all the information required to make rational, mathematical decisions is often either not available, or difficult, or time-consuming to obtain. This opens individuals up to using cognitive shortcuts (known in economics as bounded rationality; Simon 1957) in order to aid in the decision-making process. In doing so, decisions can be made at a much lower cost (both temporally and cognitively) to the individual (Simon 1957, Simon et al. 1995). Prospect theory (Kahneman and Tversky 1979, Tversky and Kahneman 1992) provides a model of human decision-making that includes additional psychological factors.

Financial gambling (casinos, bingo halls, video lottery terminals; VLTs, online gambling and sports betting) is becoming increasingly popular and provides massive annual revenue (Smith 2013). It has been estimated that between 50-80% of the general population will partake in gambling at least once annually (Abbott and Volberg 1995, Welte et al. 2002). While for most individuals this type of gambling represents a relatively harmless social function, for about 15% of frequent gamblers, and 1.6% of the general population, gambling can sometimes have severe detrimental outcomes (Wardle et al. 2007). With the release of the new Diagnostic and Statistical Manual of Mental Disorders (5th ed., APA 2013) the reclassification of gambling disorder (formerly pathological gambling) from an "Impulse-Control Disorder" to a "Substance-Related

and Addictive Disorder", suggests that gambling is a type of addiction, similar to drug or alcohol abuse (Petry et al. 2013).

The parallels between gambling and investing in the stock market are obvious; however, little research has been conducted on the two in relation to one another. Paying for advice from "financial experts" can be one of the shortcuts that less savvy or time-pressed investors may take in order to get involved in the stock market. The benefits being that these experts have a supposed authority regarding the entire process. This is despite the fact that, for investors, blindly following the advice of an expert puts one at risk of investing in potentially higher risk stocks than the individual investor might otherwise have purchased. As the expert, one is not immune to the same judgment heuristics that affect the general population. Only, once influenced, the experts may not only invest irrationally themselves, but also advise others to do so as well. This is repeatedly seen, where experts start following whatever fashion is currently proposed. Interestingly this may start early and involve some element of self-selection, for example Sjoberg and Engelberg (2009) have implicated students studying finance degrees as being greater endorsers for gambling-type risk attitudes than others. If those working in our financial markets show such similarities to people who enjoy gambling, part of our economic health is reliant on our understanding of how this population functions, psychologically and neurobiologically.

The field of neuroscience has the potential for enhancing our understanding of economic behaviour by combining our understanding of cognition, psychology, and neural correlates with the mathematical models prominent in economics (Sanfey 2007). It is also important to take our social environment into account when studying decision-making, as the world we live in is socially complex, and rarely does decision-making with clearly defined probabilities and outcomes alone present itself. Employing tasks derived from Game Theory, researchers have begun to form a more detailed picture of how social decision-making occurs – in both biological and behavioural terms. Neuroscientific methods, such as functional magnetic resonance imaging, have also opened up avenues of new exploration into clinical populations, with pathological gamblers being one of the often-studied groups.

1.2 functional Magnetic Resonance Imaging (fMRI)

As a noninvasive tool, the introduction of functional magnetic resonance imaging (fMRI) was a significant step forward in neuroscience as a means of assessing brain function. Previous tools included far more invasive techniques such as positron emission tomography (PET), which involves creating images based on the movement of injected radioactive material. Unlike PET, which is limited by poor temporal resolution (the ability to distinguish changes in a signal across time), fMRI has the ability to reflect brain activation with both good spatial (the ability to distinguish changes in an image across different spatial locations) and temporal resolution (Huettel et al. 2004).

fMRI is a noninvasive tool in that it measures signal changes in the brain via changes in the magnetization between oxygen-rich (arterial) vs. oxygen-poor (venous) blood (Huettel et al. 2004). When a body is placed into a strong magnetic field, the atomic nuclei in the body will precess (spin) around an axis that is either parallel (low energy state – typically the state which most nuclei will take) or antiparallel (high energy state) to the magnetic field. When the majority of the nuclei are aligned in this low energy state there is longitudinal magnetization. In order to flip the direction of spin of the nuclei, and thus the magnetization into transverse magnetization, another magnetic field (gradient field) must be applied at a resonant frequency. At this particular frequency, some of the low energy nuclei will absorb the applied energy and change to the high-energy state thereby inducing a state of excitation. When the energy source is removed, some of the nuclei will then release the previously absorbed energy and return to its low energy state, restoring longitudinal magnetization. This release of energy is measured with a coil to recreate the positions of the nuclei, thus creating an image.

fMRI is most commonly implemented using pulse sequences sensitive to T_2* decay; a time constant that describes the decay of the transverse component of net magnetization (Huettel et al. 2004). It is believed that there is an increase in local blood flow to brain regions in which neurons become active thus oxygenated blood comes in and displaces the deoxygenated blood. Deoxygenated hemoglobin (the hemoglobin molecule is responsible for carrying oxygen in red blood cells) is paramagnetic (more magnetic) compared to diamagnetic (resistant to magnetism) oxygenated hemoglobin. Thus, the magnetic field surrounding the paramagnetic deoxygenated

hemoglobin will be distorted and nearby hydrogen protons will experience different field strengths. This causes the nuclei to precess at different frequencies and leads to more rapid decay of transverse magnetization. This is turn, results in more signal in areas of diamagnetic blood in T_2* sensitive images. As fluctuations in oxygenated blood flow to areas of brain activity causes the change in the signal that we interpret as differentiating between active and inactive brain regions, the signal acquired in fMRI is called the blood-oxygen level dependent (BOLD) response. It is important to note that BOLD signal is an indirect measure of the effects of interest (brain region activation) as it measures changes in the magnetic properties of molecules, which in turn reflects changes in concentration of paramagnetic deoxyhemoglobin. MR signal is not increased due to the influx of oxygenated hemoglobin but rather the arrival of oxygenated hemoglobin displaces the deoxygenated hemoglobin, which has been suppressing the MR signal due to its paramagnetic properties. The following research uses fMRI in order to examine the neurobiological aspects of financial decision-making.

1.3 Conclusion

This thesis is a culmination of the research conducted to determine the neurobiological underpinnings of decision-making in the stock market and the influence that advice may have. How does advice from both "experts" or "peers" affect risky investment decisions and does this advice affect non-gamblers and gamblers in the same way? Pathological gamblers have been studied meticulously, but what are the profiles of individuals who gamble frequently yet do not meet the clinical criteria? These are the overarching research questions examined in this thesis that joins together ideas and methods from psychology, economics and neuroscience.

Chapter 2. Decision-Making and Risk Taking

2.1 Decision-making

The overall study of decision-making aims to elucidate our ability to process information and choose a beneficial action. Behavioural, neurobiological, and psychological studies of decision-making abound. Several decision-making and risk-taking tasks are commonly employed to assess risky decision-making, widely studied examples of these being the Iowa Gambling Task (IGT), the Balloon Analog Risk Task (BART), the Wisconsin Card Sorting Task (WCST) and the Game of Dice Task (GDT). Potential underlying brain regions associated with each task have been examined utilizing many techniques, including functional magnetic resonance imaging (fMRI), which has helped clarify similarities and differences between them.

2.1.1 Iowa Gambling Task

2.1.1.1 Concept

The IGT (Bechara et al. 1994) is one of the most validated and widely used measures of decision-making (Brevers et al. 2013b). The task involves asking subjects to select cards, one at a time from any of four decks with the goal to maximize profits. However, the participants are not told how many turns they will have (typically 100) and that two of the decks are advantageous in the long-term (long-term gain with short-term punishment) while the other two are not (long-term punishment with short-term gain). Thus, this task requires participants to learn that in order to gain long-term reward they must endure short-term punishment.

2.1.1.2 Findings

This task is a sensitive measure of impaired decision-making as several populations have been shown to differ significantly in task performance from healthy controls. Several clinical populations have been shown to display decision-making deficits by favouring short-term goals. These include individuals with orbitofrontal (OFC)/ventromedial prefrontal cortex (vmPFC) lesions (Bechara et al. 1994, Bechara et al. 1998, Bechara et al. 2002, Manes et al. 2002), substance addiction leading to frontal lobe dysfunction (Bechara 2001, Bechara and Damasio 2002, Bechara et al. 2002, Bechara and Martin 2004), Parkinson's disease (Thiel et al. 2003),

Huntington's disease (Stout et al. 2001), schizophrenia (Whitney et al. 2004), obsessive-compulsive disorder (Cavedini et al. 2002a) and anorexia nervosa (Cavedini et al. 2004). Following the dual-model process of self-regulation (Bechara 2005, Everitt and Robbins 2005), it is believed that the ability to decide advantageously based on long-term and short-term outcomes is derived from activation from both an impulsive amygdala-striatum based system and a reflective prefrontal based system. The impulsive network promotes automatic, habitual and salient behaviours, while the reflective network is credited with forecasting the future consequences of decisions and employing the inhibitory control when necessary. This reflective network is also thought to include executive functions, which consist of various cognitive abilities to control thought, emotion and action (Brevers et al. 2013a). Overall, it is believed that task deficits are due to impairments in using feedback from previous trials to aid in current decision-making. This is thought to be mediated by OFC/vmPFC dysfunction as well as dysfunction in the limbic network including the amygdala, previously implicated in emotion processing (Bechara et al. 1999, Bechara et al. 2003).

2.1.2 Game of Dice Task

2.1.2.1 Concept

The GDT (Brand et al. 2005a) is a gambling task in which - unlike in the IGT - the rules for both gains and losses are explicit, as are the winning probabilities throughout the entire task. This explicit knowledge of the probabilities allows individuals to plan a long-term strategy in order to increase their outcome. In this task, participants are asked to guess what number on which they think a single die will land in order to maximize their funds. There are four types of guesses: a single number, combination of two possible numbers, combination of three possible numbers or a combination of four possible numbers. Each type of choice is associated with a gain/loss amount should the die match/not match the chosen option. The smaller probability that the choice will be correct the greater the reward/loss. Thus, if a participant chose a single number then the probability of winning would be 1:6 and this option would yield the highest amount of reward/loss. If a participant chose a combination of four numbers the probability of having the correct number increases to 4:6; however, this would yield a smaller reward than any of the other options. All of this is presented visually so that the probabilities can be easily discerned. After the participant makes his selection the die is rolled and the winning number revealed.

Participants receive both visual and auditory feedback and their monetary total is adjusted accordingly. This is repeated for a total of 18 rounds. The three and four number combination choices are considered advantageous choices, as their winning probabilities are greater than 50% and are associated with lower gains but also lower penalties. Disadvantageous choices are the single or two number combination choices, as their winning probabilities are less than 50% and are associated with higher gains but also higher penalties. Thus, the GDT assesses decision-making under risk and uncertainty (Brand et al. 2005a).

2.1.2.2. Findings

Alcoholic Korsakoff patients (patients whose former alcohol abuse led to frontal lobe dysfunction and brain damage) were shown to have impaired performance in this task where healthy controls displayed risk-avoidant decision-making (Brand et al. 2005a). Women with binge eating disorder (Svaldi et al. 2010), individuals diagnosed with bulimia nervosa (Brand et al. 2007a), and Parkinson's patients (Brand et al. 2004) have also shown deficits in this task. Choosing disadvantageous options was correlated with poor performance in the modified WCST in executive functions such as categorization, set-shifting and cognitive flexibility (Brand et al. 2005a).

It is important to note that the IGT has also been found to examine decisions under conditions of ambiguity, as well as the risks involved as the task progresses (Brand et al. 2007b). In the beginning phases of the IGT, probabilities and outcomes are ambiguous; however, as the task progresses participants acquire some knowledge about each of the decks and the decision-making switches from being under conditions of ambiguity to conditions of risk, much like the GDT. Brand et al. (2007) suggest that executive functions are taxed differently based on whether the decision-making is occurring under conditions of ambiguity or risk. They hypothesize that ambiguity does not call upon executive functions but risky decisions more heavily draw on the executive functions system. The OFC and amygdala appear to have important roles in decision-making under conditions of ambiguity. In an fMRI study by Hsu et al. (2005) comparing ambiguous and risky decision-making, activation in the OFC and amygdala corresponded with ambiguous decisions while activation in the dorsal striatum (caudate nucleus) was found to correlate negatively with ambiguity but positively with expected rewards. The authors suggest

that the OFC and amygdala respond to the degree of uncertainty/ambiguity, much like a vigilance evaluation system. Behavioural tests with OFC lesion patients also revealed an inability to distinguish between ambiguity and risk conditions (Hsu et al. 2005).

2.1.3 Wisconsin Card Sorting Task

2.1.3.1 Concept

The WCST (Grant and Berg 1948) tests executive functions in humans. Participants are asked to sort a deck of cards according to some predetermined rule, but are not explicitly told what the rule is, and are given feedback to let the participant know if they have used the correct rule. The three possible rules used to sort the cards are to sort them by the color of the items, the number of items, or by the shape of the items on the card. The rule by which the cards are to be sorted changes throughout the task and the participants must adapt with the change in feedback. Participants are scored based on the number of different categories they were able to achieve as well as the number of perseveration errors (continuing to sort by an old rule once a new one has been established) that occurred.

2.1.3.2 Findings

Previous research has found impairments in patients with lesions in the prefrontal cortex (Milner 1963, Nelson 1976, Stuss et al. 2000) and fMRI studies have supported these findings, linking the prefrontal cortex to set-shifting (Berman et al. 1995, Nagahama et al. 1996, Rogers et al. 2000, Nagahama et al. 2001). There has been some speculation that the basal ganglia is also involved in the WCST as impairments have also been seen in Parkinson's disease patients (Bowen et al. 1975, Gotham et al. 1988). Monchi et al (2001) found that there was a dissociation in activity between the mid-ventrolateral prefrontal cortex (vlPFC) and mid-dorsolateral prefrontal cortex (dlPFC) during the WCST. While both areas were activated during set-shifting, the mid-dlPFC also exhibited an increase in activation during set maintenance. The authors also found that both the caudate and putamen were involved with performance on the WCST, activating during negative feedback trials.

2.1.4 Balloon Analog Risk Test

2.1.4.1 Concept

The BART (Lejuez et al. 2002b) is a computerized measure that mimics real-world risk-taking where riskiness is rewarded up until a certain point, after which continued risk-taking results in poorer outcomes. In the task, participants are presented with 90 balloons, one at a time that they can inflate by pressing a labeled button. For each button press, or air pump into the balloon, money is added to a temporary bank although the amount is not displayed to the participant. Participants may stop inflating the balloon at any time and choose to collect the funds from the temporary bank and proceed to the next balloon. The more the balloon is inflated, the more money is accumulated; however, the balloon may pop and the funds may be lost, thus the more pumps a participant decides to use the increased risk that the balloon will pop. The average number of inflations participants decide to do provides a measurement of risk preference.

2.1.4.2 Findings

Studies have shown that the BART assessment of risk preference correlates with scores on risk-related constructs such as sensation seeking and impulsivity as well as self-reports of risk behaviour occurrence (Lejuez et al. 2002b, Lejuez et al. 2003a, Lejuez et al. 2003b, Hunt et al. 2005, Lejuez et al. 2007). Rao et al. (2008) used a modified version of the BART to determine if the dopamine system in the human brain is activated by risk alone, decision-making alone, or a combination of both. They found that increasing risk coupled with decision-making correlated with activation in the ventral tegmental area, striatum, insula, anterior cingulate cortex (ACC), and dlPFC (Rao et al. 2008) supporting the role of the mesolimbic and frontal dopamine regions in risk during active decision-making. When the participants were forced to passively watch the balloon, without deciding when to stop the inflation, the previous activation pattern was not observed suggesting that it is not risk alone that engages these pathways but rather that an active choice is also required (Rao et al. 2008).

2.1.5 Comparisons between the tasks

It can be seen that each of the tasks has different strengths and weaknesses, and often affect similar regions of the brain, such as ventromedial prefrontal/orbitofrontal cortex (vmPFC/OFC) and the anterior cingulate cortex (ACC). These tasks form a mix of both ambiguous (IGT and WCST) and explicit (GDT and BART) rules of risk and demonstrate the complexity involved in the study of decision-making. As decision-making can occur under a myriad of situations, it is important to have a variety of tasks in order to fully understand how decision-making occurs. Variations of the Go/No-Go tasks, a task in which participants are asked to attend and respond to certain stimuli (a Go trial) while ignoring others (a No-Go trial), are also often used in decision-making studies. Aside from the decision-making process, reward and learning may shift during decision-making and forms another focus in decision-making research.

2.2 Reward Prediction Error

At its most simplistic form, reward prediction error refers to the degree to which a reward is surprising to a subject, a hypothesis derived originally by Schultz et al. (1997). In this pioneering research, behavioural experimental results were combined with measures of physiological response of dopaminergic neurons in primates during a basic reinforcement based learning task. In summary, midbrain dopamine firing was recorded when there was an unpredicted occurrence of juice (the unconditioned stimulus; UCS) prior to learning. Post learning, the conditioned stimulus (CS) predicts the delivery of a reward (the UCS) and thus when the reward arrives according to prediction there is no error in the prediction of the reward, and the dopamine neurons fail to activate to the delivery of the reward. Rather, the neurons fire in response to the CS, the reward-predicting stimulus. Should the CS be presented but no reward follows dopamine neuron activity is depressed at precisely the time when the reward ought to have occurred (Schultz et al. 1997). Schultz et al. (1997) concluded that dopamine neurons appeared to be predictors of how well actual events fit to previously learned predictions about those events: an increase in dopamine firing was exhibited if the event was better than expected, no signal was measurable if the event occurred as expected, and a depressed signal or rate of firing was exhibited if the event was worse than expected. This was termed the Reward Prediction Error

hypothesis. More specifically, this hypothesis states that dopamine encodes the difference between the experienced and predicted reward of an event.

2.2.1 Functional magnetic resonance imaging evidence

With the progression of neuroscience, researchers have adopted fMRI to study reward prediction error more closely. Some areas of interest include the midbrain, ventral striatum, nucleus accumbens (NAcc), amygdala, OFC and medial prefrontal cortex, areas that are all innervated by mesolimbic dopamine pathways (Knutson and Cooper 2005). Specifically, fMRI studies have shown that the ventral striatum is activated preferentially during reward anticipation while the medial prefrontal cortex is preferentially activated during reward outcome (Knutson et al. 2001, O'Doherty et al. 2002). It also appears that reward may manifest itself differently in the brain depending on the timing of the reward and learning. Tanaka et al. (2004) found that during a task in which subjects learned to choose options that lead to monetary gains versus losses, activation in the striatum and lateral OFC correlated with immediate reward prediction while longer-term future reward (small immediate losses leading to long term benefit) correlated with dlPFC and inferior parietal cortex activation. Delgado et al. (2005b) found that the caudate exhibited greater activation in the early learning stages. O'Doherty et al. (2004) had participants learn cues that predicted a juice reward with either active choice or passive association in an fMRI study. They found that while reward prediction error correlated with activity in the ventral striatum for both tasks, the caudate was recruited only during the active choice task. Taken together, these studies point to the ventral striatum and caudate as having an important role in what appears to be reward learning.

2.3 Incentive Salience vs. Reward Prediction Error

In a recent review by Berridge (2012), he outlines the importance of differentiating between reward prediction error and incentive salience. Incentive salience refers to a form of Pavlovian-related "wanting" or motivation for rewards, and is mediated by the mesocorticolimbic brain systems (Robinson and Berridge 1993, Berridge 2007). While "wanting" typically occurs in

conjunction with "liking" this is not always the case, as manipulations involving dopamine can dissociate the two (Berridge and Robinson 1998, Berridge 2007, Smith et al. 2011).

Despite past beliefs that dopamine is responsible for causing pleasure (Wise 1985), it has been shown that dopamine is not required for normal 'liking' reactions (Berridge and Robinson 1998), since patients with dopamine depletion (Parkinson's) report normal ratings of pleasure (Sienkiewicz-Jarosz et al. 2005), and elevating dopamine levels does not appear to enhance pleasure (Smith et al. 2011). Berridge (2012) argues that while dopamine appears to code for reward learning, closer examination of the evidence reveals that it does not actually cause learning in terms of reward but rather causes incentive salience for both learned and unlearned rewards.

Previous animal studies have employed stringent physiological conditions in both training and testing, under which a CS–triggered incentive salience will appear to track learning as that is the only possibility (Berridge 2012). However, in reality, life is not that constant and physiological states can vary. Studies that manipulate physiological state to new levels, never before experienced, have shown that rather than a reward prediction error, dopamine levels may be more linked to incentive salience, or 'wanting'. Thus, dopamine release appears more closely related to "wanting things" than to actually obtaining them. This is demonstrated effectively with the inducement of a salt appetitive state. A prediction error theorist would hypothesize that the CS value will remain the same in the face of this novel state. An incentive salience theorist would predict that this change in physiological state would alter the CS value. Rats were trained to associate an auditory signal (the CS) with a squirt of 10% sodium chloride (NaCl) solution into the mouth – a highly aversive stimulus (UCS) that elicits a fast learning to avoid or turn away from the CS. When a strong salt appetitive state is induced the UCS is no longer aversive but is instead reacted to as pleasant (Berridge et al. 1984, Tindell et al. 2006). Additionally, upon first re-encounter with the previously associated aversive CS in this new salt appetitive state, the neurons in the striatum, specifically the ventral pallidum, fire as strongly for the salt-associated CS as they do to a sweet/positive-associated CS (Tindell et al. 2009). These results support dopamine's role in incentive salience rather than simply reward prediction error.

2.4 vmPFC/OFC role in Reward and Decision-Making

The vmPFC/OFC is involved in the processing of the reward value of stimuli, a key function to decision-making (Krawczyk 2002a). It has recently been suggested as the area in which rewards of all types are valued on a common scale by which comparisons are measured and decisions subsequently made (Levy and Glimcher 2012). Many studies focused on monetary rewards have implicated the medial prefrontal cortex, ventral striatum, posterior cingulate cortex, amygdala and insula in reward magnitude (Kable and Glimcher 2009, Grabenhorst and Rolls 2011, Padoa-Schioppa 2011); however, studies including more than one type of reward have also been conducted to determine how these areas are recruited, if at all, when the decisions are based on non-monetary rewards. The first of such studies was conducted by FitzGerald et al. (2009), who used both money and consumer goods. The researchers found that ventromedial prefrontal cortex and orbitofrontal cortex (vmPFC/OFC) activation was correlated with subjective values for both reward types both in terms of gains and losses. Three reward types, money, food, and consumer goods, were then used to explore this more and all three reward types' subjective values were represented in the vmPFC/OFC (Chib et al. 2009). Some studies have also found evidence for ventral striatum involvement (Izuma et al. 2009, Talmi et al. 2009, Levy and Glimcher 2011). Additionally, studies have found that equal behavioural value does in fact reveal equal BOLD signal in the vmPFC/OFC, providing evidence that these different types of reward are all represented as a single common currency of equal value in these areas (Smith et al. 2010, Levy and Glimcher 2011).

Research with non-human primates has shown the OFC's involvement in motivation, affect and reward processing (Cavada et al. 2000, Hikosaka and Watanabe 2000). The OFC is particularly important to the rapid adjustments in behaviour in response to changes in the environment (Krawczyk 2002a). OFC lesions have been shown to cause deficits in reversal learning, where animals will perseverate on stimuli that had at one time been rewarded but no longer are (Butter 1969, Dias et al. 1996, Dias et al. 1997, Izquierdo and Murray 2004, Murray and Izquierdo 2007). Additionally, animals with damage to the OFC show deficits in Go/No-Go tasks, choosing to respond in no-go trials (Iverson and Mishkin 1970). This pattern of Go/No-Go task deficit has also been shown in patients with frontal-lobe damage (Drewe 1975).

The description of one OFC damaged patient demonstrates the importance of the OFC in daily decision-making (Eslinger and Damasio 1985). Prior to OFC damage, this individual was a model employee, receiving promotions consistently. After the surgery, he began to have difficulty holding a job due to being constantly disorganized and late. His personal life suffered as well, going through two divorces. Some abnormal behaviour that developed included refusing to dispose of useless items such as broken appliances, spending a great deal of time more than usual on personal hygiene, and taking extremely long amounts of time deliberating on small purchases. He did not exhibit any problems in normal problem solving tasks within the laboratory, scored within normal ranges on intelligence testing, and could reason on social or ethical dilemmas; however, despite having this intact knowledge, he seemed incapable of transferring this knowledge towards his day to day functioning. Upon further examination, it appeared that the dysfunction that he suffered was specific to decision-making among options (Saver and Damasio 1991), hence why he scored normally on laboratory tasks of intelligence. In other studies, patients with similar lesions have also been shown to display deficits in financial planning (Goel et al. 1998). In a task where participants were asked to generate a series of long-term plans across a series of life goals, patients provided overall fewer viable solutions. Importantly, the frontal lobe damaged group spent less time working on plans to achieve long term future goals (Goel et al. 1998).

2.4.1 The somatic marker hypothesis

The somatic marker hypothesis was put forward by Damasio (Damasio 1994, Damasio et al. 1996), and proposes how decision-making is guided by emotional processes/states otherwise called somatic markers where the term ‘somatic’ refers to a physiological affective state. Thus, when presented with a stimulus to which a previous outcome association has occurred, the vmPFC reactivates the previously associated body states in one of two ways: the ‘body loop’ where the originally associated somatic signals are activated which sends somatic state information to the somatosensory cortex, or the ‘as if body loop’ where the originally associated somatic signal is not re-experienced but is just sent to the somatosensory cortex (Bechara and Damasio 2005). The resulting somatosensory activations are the markers by which different options can be either positively or negatively biased, allowing for faster deliberation and decision-making. Patients with OFC damage have been shown to display abnormal autonomic

arousal in response to emotionally charged photos (Damasio et al. 1990) as well as abnormal skin conductance responses in an initial version of the IGT (Bechara et al. 1996). This abnormal autonomic response, coupled with deficits in performance of the task, supported a hypothesis that physiological markers are important factors in advantageous decision-making, with the OFC being critical to this process.

2.4.2 Criticism of the somatic marker hypothesis

Some have suggested that the somatic marker hypothesis is overly elaborate in explaining common reversal learning deficits, since the reasoning could be as simple as a lack of forming the appropriate associations between stimuli and responses, a known function of the OFC (Krawczyk 2002a). As well, it has been argued that the OFC and amygdala are recruited directly as a reaction to the experience of a stimulus, and thus the emotion acts as part of the response (Rolls et al. 1996, Rolls 1999). There is evidence to support the view that reward and punishment processing is a specific function of the OFC (Rolls 1999, Rolls 2000). Rolls (2000) also criticizes the somatic marker hypothesis as being inefficient as a great deal of interpretation would be required to analyze massive amounts of somatic input.

2.5 Dorsolateral pre-frontal cortex role in Decision-Making

The dorsolateral pre-frontal cortex (dlPFC) is one of the key areas responsible for keeping and influencing information that is being used in working memory (Goldman-Rakic 1992, Jonides et al. 1993, Belger et al. 1998, Shimamura 2000). Decision-making requires working memory to maintain a focus on goal hierarchies and monitor competing options (Krawczyk 2002a). There is also evidence suggesting that the dlPFC is involved in the processing of relational information and the integration of information. Waltz et al. (1999) tested the importance of the prefrontal cortex in integrating information and found that patients with frontal lobe damage performed poorly on tasks that required the integration of information from two or more sources compared to temporal lobe patients and healthy controls. Coupled with findings that the task used elicited activation in the dlPFC in an fMRI study (Prabhakaran et al. 1997) and that significant activation was also found in the dlPFC in a PET study during a study of transitive inference (Baker et al.

1996), it is believed that the dlPFC plays a key role in mediating relational processing (Krawczyk 2002a). Goldberg et al. (1994) and Podell et al. (1995) assessed decision-making under ambiguous contexts that do not have clearly correct choices, otherwise termed adaptive decision making. In this task, participants were shown a target picture of a shape and asked to choose one of two shapes that varied in similarity to the target shape. In one condition, participants were asked to select the shape that they "liked best" while in another condition they were asked to select the shape that was either most similar or most different. While healthy controls made their choices in the first condition based on a balance of similarity and dissimilarity, patients with damage to the left dlPFC chose shapes that were more dissimilar to the target. In the other two conditions, there were no significant differences between the groups thus showing that frontal lobe damage did not affect directed decision-making but caused dysfunction in undirected decision-making. Another study comparing decision-making under either an explicit rule condition or a no-rule condition found that the right dlPFC was activated during the no-rule condition while the rule conditioned elicited bilateral activity (Goel and Dolan 2000). The authors suggested that the right hemisphere might be more involved in the resolution of ambiguity in the absence of explicit rules. Goel and Grafman (2000) observed a man, P.F., with a right frontal ablation, documenting that when presented with a task to redesign a laboratory (P.F. had previously worked as an architect) P.F. spent more time planning how to complete the project but, consistent with an inability to access prior knowledge, showed deficits in his ability to execute the plans he had previously made. Thus, these observations support the hypothesis that the right dlPFC plays an important role in accessing and processing based on previous knowledge.

2.6 Anterior Cingulate Cortex (ACC) Role in Decision-Making

The ACC has been implicated in decision-making that is highly ambiguous and is believed to contribute to the processing of conflicting options with a high likelihood of making an error (Krawczyk 2002a). Increased ACC activation was elicited when participants made errors in a task of matching a cue letter (A) to a target letter (X) and this activation was greater when the errors were made under high competition (A presented with a Y, or B presented before an X; Carter et al. 1998). The role of the ACC in conflict monitoring has also been suggested

(Botvinick et al. 1999, Botvinick et al. 2001) as trials in which incompatible stimuli were presented elicited ACC activity. Additionally, when an incompatible trial was preceded by a compatible trial, thus when the conflict was the most salient, ACC activation was greater than if the incompatible trial had been preceded by another incompatible trial (Botvinick et al. 1999). The ACC has also been implicated in outcome anticipation as Critchley et al. (2001) found that as outcome uncertainty increased so did activity in the ACC along with the OFC. In this study, the authors also found that higher arousal level as measured by galvanic skin response was associated with activation in the ACC, dlPFC and parietal cortex during the delay period between decision-making and outcome notification.

2.7 Summary of Evidence

It can be seen that the evidence to date suggests that there are several considerations to be aware of when conducting research on decision-making as a whole and that many brain regions have been shown to come into play. Decision-making research must take learning, memory and reward into consideration rather than a focus on simply the act of making a choice. There are complex relationships involved that have ramifications on how decisions occur and how these decisions are manifested neurobiologically. From the reviewed literature, certain brain regions have emerged as playing key roles in the decision-making process. The ventromedial prefrontal cortex/orbitofrontal cortex (vmPFC/OFC) has been implicated in reward, which is a driving force in the decision-making process. Individuals will make choices in order to receive rewards thus an understanding of how these rewards are processed is necessary in order to form a true understanding of decision-making. This area also appears to be involved in planning and motivation, both of which are very obviously linked to decision-making. Using past memories in order to guide future decision-making is also a crucial skill, with the dorsolateral prefrontal cortex (dlPFC) being an area of focus in this research. How this region interacts with the vmPFC/OFC, thus how reward and memory interact may influence how decision-making occurs. Finally, the anterior cingulate cortex (ACC) has emerged as a particular region of interest in risky and ambiguous decision-making. This research is particularly meaningful as decisions made in everyday life, outside of a laboratory, are often risky, with ambiguous associated rewards. In the creation and use of tasks that tap into to all these concepts, and coupling them

with neuroimaging techniques, a more global understanding of decision-making behaviour and biology can be achieved.

Chapter 3. Psychology and Economics

3.1 Social Influence

The idea that social factors influence financial decision-making is not new. Individually and collectively, people's evaluations under uncertainty deviate from mathematically expected values. Rabin (2003) elucidated the following common biases in decision-making: low probability events tend to be either neglected or over represented while high probability events are not fully accounted for; small samples are taken as unreasonably representative; memorable and salient information is more heavily weighted when there is conflicting information; and initial information may bias the interpretation of subsequent information.

Economists recognize that people employ bounded rationality –that is, they take cognitive shortcuts (Simon 1957), such as relying on recent experience, social cues, expert advice, decisions of others, and authority figures when making decisions (Shleifer 2000). Such behaviour is rational if the costs of obtaining a more precise solution exceed the costs of making occasional errors while using an approximate solution. The term "bounded rationality" signifies rationality bounded by the costs of gathering and processing information. Economic selection can favour bounded rationality if such shortcuts leave individuals or firms wealthier in the long run than if they had to bear the costs of obtaining precise solutions.

Psychologists term these cognitive shortcuts *judgmental heuristics* (Tversky and Kahneman 1974, Kahneman et al. 1982, Todd and Girgerenzer 2007). Elaborating on the concept of bounded rationality, Kahneman and Tversky (2011) argue that people routinely employ "fast thinking" (cognitive shortcuts such as deference to experts), but activate "slow thinking" (rational decision-making as in neoclassical economics) if fast thinking fails to converge, positing the latter too metabolically expensive to employ constantly. These tendencies to respond automatically to incomplete information usually help, but sometimes hinder us. Automated behaviour, also evident in animal species in the natural environment (Fox 1974) is evolutionarily advantageous due to its efficiency (Gigerenzer and Goldstein 1996) and in today's complex world, relying on such stereotypes and rules of thumb has arguably become a necessity in navigating daily life (Fiske and Neuberg 1990, Bodenhausen et al. 1999).

One simplistic form of social influence that occurs daily in the realm of sales is the manipulation of prices (Rao and Monroe 1989). Stereotypically, higher prices signal higher quality or otherwise more desirable goods, all else equal. But not all consumers can afford the time and effort to research all aspects of a good and compare it to otherwise similar goods, even if that information is publicly available. They rationally rely on others to have made such comparisons, and conclude that the seller could not remain in business unless their prices were fair. Most of the time this works, and this behaviour is rational if the savings in time and effort outweighs the cost of occasionally falling victim to a sly storeowner who exploits the heuristics on which busy consumers rely.

Looking to others as templates upon which to base one's actions is another judgmental heuristic, and may be of first order importance in stock market decision-making (Welch 2000). This form of bounded rationality can also be cast as a principle of social proof, whereby individuals determine what is correct based on what others think is correct (Lun et al. 2007), on what one thinks others think is correct, or on yet higher orders of recursion (Keynes 1936) that affects financial decisions. In other words, to determine what action is appropriate in a given scenario, we are guided by others' behaviour in what we perceive to be a similar scenario. The extent to which we see others engaging in that behaviour modulates how "correct" we perceive the behaviour to be. When deciding which stocks to buy or sell, people can avoid information gathering and processing costs by looking to other investors and mimicking their actions, the underlying assumption being that these other investors have incurred the cost of becoming informed and that their actions are therefore correct. As the number of investors behaving in a particular fashion increases, so does the strength of the social proof. This could be one of the factors that lead to so-called stock market "bubbles" (Bikhchandani et al. 1992). As ever more investors buy a stock, its price rises ever higher, validating their decisions to buy and causing yet more uninformed investors to buy, and so on in an upward spiral. Moreover, as uninformed buying pushes the stock's price ever higher, the aforementioned heuristic of "expensive = better" can also be activated further stimulating investors to buy the stock, without gathering information and mathematically weighing the odds of losses and gains. In addition to fuelling asset price bubbles, heuristics of social proof are also used in financial advertising. For example,

early advertisements for U.S Savings Bonds depicting a young boy with the caption "Just one of the 50 million Americans who invests in U.S. Savings Bonds" (Cialdini 2009) had a powerful impact, leading the reader to question how 50 million Americans could possibly be wrong.

The heuristic of social proof may also reflect a well-documented "wisdom of the crowd" effect. If each of a large number of individuals has access to an independent source of information, statistically, the Law of Large Numbers in statistics makes the mean of their assessments a more precise estimate than any of their individual assessments is likely to be (Surowiecki 2004). An uninformed person entering the fray might rationally avoid the costs of gathering and processing information by accepting that mean as a sufficient statistic. Indeed this is the basis of the efficient markets hypothesis in economics, which posits that market prices are likely to be more precise estimates of "true" values than are the estimates of any individual market participant. Such considerations may explain our strong desire to appear to be a part of a majority group, rather than a deviant or member of a minority group (Asch 1956). If everyone else partakes in an action, in this case a decision to but or sell a particular stock, no investor wants to remain the lone contrarian. Consistent with this theme, Cooksey (1996) found that participants asked to predict fluctuations in stock prices were influenced by the predictions of other participants, especially when the stock prices varied unsystematically and if the group was a majority. This influence was reduced when the prices varied systematically, indicating that the tendency to follow others would be strongest during times of greater uncertainty in the stock market. Strengthening these concepts of social proof, it has been found that once a group has formed a conclusion or hypothesis, there is a strong reluctance to disengage from it. This leads people to consistently seek out information that affirms their existing opinion, a heuristic known as "confirmation bias" (Watson 1960). Reports of the strength of an investment will be committed to memory and used to validate the choices already made, while warnings of a potential crash or the over-valuation of a stock will be ignored.

Touted financial experts may also exhibit an overconfidence bias (De Bondt and Thaler 1985). Believing oneself to be more capable and skilled than one truly is may be a survival trait in an economy that rewards innovation and boldness. But, coupled with the assurance that others want to take the same action, such beliefs may encourage experts to parade an excessively enthusiastic

confidence in their decisions/advice. New portfolio managers might follow the lead of these more confident and experienced senior investors, again giving rise to "herding" (Chevalier and Ellison 1999, Lamont 2002, Sias 2004), yet another manifestation of the social proof heuristic. This "follow the leader" behaviour among investors can also be modelled as a bounded rationality-based phenomenon called an "information cascade": uninformed investors mimicking the observed choices of people they incorrectly deem to have born the costs of becoming informed (Bikhchandani et al. 1992). Scharfstein and Stein (1990) demonstrated that this herding behaviour might be a product of managers' attempts to improve their reputations as good decision makers. If a newer portfolio manager is not investing as other more experienced ones are, they may be singled out with harmful consequences for their reputations and recruitment of future clients. An added advantage of herding is that, when the decisions turn out to have been wrong, everyone was wrong and blame cannot fall on any individual (Devenow and Welch 1996). Taken together, it can be seen that there can be significant costs (in terms of lost clients and reputation) even if subsequent events prove them right. This behaviour can be very powerful, and particularly when it is linked to aspects of obedience to authority figures, or "experts".

3.2 Obedience and "Experts"

Dubious "experts" populate many fields. Many nontraditional medical practitioners charge high fees for therapies that lack both scientific plausibility and statistically detectable efficacy (Singh and Ernst 2008). Dowsers, clairvoyants, and others charge for similarly unverifiable expertise (Taylor and Balanovski 1979, Bausell 2007), while retail financial advisors charge substantial fees for advice long known to be statistically significantly inferior to buying and holding a randomly selected diversified portfolio of stocks (Inderst and Ottaviani 2012). Professional money managers' substantial underperformance net of fees (Jensen 1968) is estimated to cost investors almost two-thirds of a percent per year (Gruber 1996, French 2008). Including other fees they pay to the brokers and investment advisors who direct them to underperforming money managers' mutual funds (Bergstresser et al. 2009, Del Guercio et al. 2010) raises investors' losses to 2% per year relative to index funds. Compounded over the years until the typical investors' retirement, this constitutes a huge transfer of wealth (Malkiel 2003).

Investors' persistent willingness to bear these costs mars naïve rational agent models and directs economists' attention onto bounded rationality models (Keynes 1936, Simon 1957, Simon et al. 1995), in which agents rationally opt to rely on a cognitive shortcut instead of solving a difficult optimization problem if the expected costs of a precise solution outweigh the expected benefits.

In perhaps the most famous social psychology experiment, Stanley Milgram (1963) demonstrated just how influential an authority figure can be. A surprisingly large fraction of participants, roughly two of every three, obeyed a researcher's orders to inflict obviously painful and potentially lethal electric shocks to another supposed participant (in fact a professional actor in no real danger) for failing to learn word pairs. When questioned about their actions after the experiment, many participants cited the influence of the experimenter in a white lab coat. The experimenter was the authority, the "expert", so even though morally, participants felt an immense amount of conflict, the expert must be correct and should be obeyed.

Looking outside the laboratory, to a real-life scenario, we can look to the story of Mr. Wilson, who laid down on a train track to protest U.S. shipments of military equipment to Nicaragua in September of 1987 (Cialdini 2009). Mr. Wilson and his two co-protesters had informed the navy as well as railroad officials of their intentions and were confident that they would be in no real danger. However, the crew running the train had been ordered by superiors *not* to stop, and so did not stop, nor even slow the train down when the protestors on the train tracks came into view. While the other two protestors escaped without injury, Mr. Wilson remained on the tracks and lost both his legs. Surprisingly, Mr. Wilson did not blame the crew for following orders, and the crew went on to sue Mr. Wilson for interfering with their abilities to complete their assigned task. The power of the desire to obey authority led the crew not just to sever a man's legs, but to believe themselves the victims in this situation. If the crew remained obedient to an authority when lives were at risk, how will an investor behave when an authority advises them on financial decisions?

A strong proclivity to obey legitimate authority may be a form of bounded rationality with roots in prehistory. Economic anthropology posits social hierarchies, which necessitate the general acceptance of a system of authority, allow the coordination of specialized economic activities

characteristic of the "Neolithic transition" from a nomadic hunter-gatherer subsistence economy to agriculture, with its surplus production for sustaining scribes and artists (Cialdini 2009). Milgram's (1963) subjects rationalized administering dangerous electric shocks in terms of deference to his authority as an expert psychologist. Milgram (1974) speculates that evolution selected for *reflexive obedience* to legitimate authority figures, recalling Darwin's (1871)(p. 166) musings that

> "There can be no doubt that a tribe including many members who, from possessing in a high degree the spirit of patriotism, fidelity, obedience, courage, and sympathy, were always ready to give aid to each other and to sacrifice themselves for the common good, would be victorious over most other tribes; and this would be natural selection."

This reasoning suggests that individuals following expert advice should exhibit evidence of positive emotions associated with feelings of loyalty, submission to legitimate authority, and the like. Thus, some behavioural economists posit a *utility of trust*: trusting an expert increases an investor's happiness *ceteris paribus* by decreasing the anxiety associated with risk taking (Gennaioli et al. 2012). This potentially affects the economy by inducing investors to invest more money and bear more risk than they otherwise would.

3.3 Neuroeconomics

Recent work in neuroscience supplements behavioural studies of social and financial decision-making. This new sub-discipline, called neuroeconomics, makes use of behavioural and economic tasks with the addition of neuroimaging technique such as electroencephalography (EEG) and fMRI. In this way, behavioural data can be linked to biology and help to further increase our understanding of financial decision-making

3.3.1 Game Theory

Game Theory (von Neumann and Morgenstern 1947) is a branch of economic theory that models strategic decision-making where the outcomes depend not just on a player's own decision, but on one or many other players' decisions, all of which depend on the first player's decision.

Experimental economics shows that people generally do not exhibit the behaviour that game theory deems rational. Rather, the heuristics associated with bounded rationality are again in evidence.

This suggests that neuroscience can utilize tasks derived from Game Theory to elucidate the neural correlates of social decision-making. These tasks are often simplistic in nature; however, reasoning about the motivations of other players is required. Classic game theory, assumes that all individuals are rational and self-interested, predicts that, in a broad range of situations, individuals' decisions interact to produce a Nash equilibrium (Nash 1950), a situation in which no player could improve their outcome by unilaterally changing their strategy. This can leave all the players trapped in a low level equilibrium, where each player would be rendered worse off if they were the only one to do otherwise, but where all the players could be made better off if they all changed their behaviour in concert to achieve a higher level "cooperative equilibrium". Any such cooperative equilibrium can be unstable, in that each player may be able to gain by changing their behaviour if the other players adhered to the cooperative behaviour. If each player, seeing this, changes their behaviour, the cooperative equilibrium dissolves and the players eventually settle back onto the Nash equilibrium. A simple example illustrating all these points is the Prisoner's Dilemma Game (PD; Table 1).

	Prisoner B cooperates	Prisoner B defects
Prisoner A cooperates	Both serve 1 year	Prisoner A: 3 years Prisoner B: free
Prisoner A defects	Prisoner A: free Prisoner B: 3 years	Both serve 2 years

Table 1. Prisoner's Dilemma Game

In this game, two players must decide whether to cooperate with one another or defect and betray the other. Should both players defect, each would serve 2 years in jail. Should Prisoner A defect while Prisoner B cooperates, Prisoner A will be set free while Prisoner B will serve 3 years and vice versa. Should both players cooperate, each would only serve 1 year in jail. Classic Game Theory predicts that both players will behave in a rational self-interested manner by defecting and a low level equilibrium will be reached. The optimal equilibrium requires both players to trust and cooperate with one another, leading to both serving 1 year in jail.

Other games commonly used in neuroscience experiments include: the Ultimatum Game (UG); used to examine response to fairness, the Trust Game (TG); which like the Prisoner's Dilemma game is used to examine reciprocal exchange; and the Dictator Game (DG); used to examine altruism. These are summarized in Table 2.

Table 2.
Game Theory Tasks

Task	Construct Measured	Description
Ultimatum Game	Fairness	- Player A (the Proposer) divides a sum of money and proposes the division to Player B (the Responder) - The Responder may accept the offer leading to the sum being divided as proposed - The Responder may reject the offer leading to neither participant receiving any sum of money
Dictator Game	Altruism	- Similar to the Ultimatum Game only Player B (the Responder) is a passive recipient and does not have to option to reject the offer
Trust Game	Reciprocal Exchange	- Player A (the Investor) entrusts a portion of an endowment with Player B (the Trustee) - The entrusted money is multiplied by some factor - The Trustee has the opportunity to return money to the Investor, but *is not required to*
Prisoner's Dilemma Game	Reciprocal Exchange	- Both players must simultaneously choose whether to betray or trust their partner without knowing the other players choice - The outcome is an interaction of both players decision

3.3.2 Neuroscience findings

One line of research in neuroeconomics endeavors to discern brain functioning in human subjects making specific financial decisions. Early research focused on individual decisions where only one's own preferences matter. However, as noted above, decision-making often must also account for the likely choices others will make, which can in turn depend on one's own choices. A Game Theory framework has thus become the norm in research into social decision making in general and financial decision-making in particular (Fehr and Camerer 2007, Rilling and Sanfey 2011).

As previously reviewed, neuroscience research supports the hypothesis that the mesolimbic dopamine system houses the common-reward metric of the brain. In addition, the striatum, which is linked to overall reward, also appears to be involved in social decision-making. In an iterated Prisoner's Dilemma game, striatal activation tracked a partner deciding to cooperate or not, with increased activation occurring during reciprocal cooperation and decreased activation during unreciprocated actions (Rilling et al. 2002). This research also suggests that the striatum may encode social prediction errors to guide future social decision-making, as striatal activation was linked to an increased likelihood of cooperation in the following rounds. There is also some evidence to suggest that we find the acts of another player instilling trust in us to be rewarding, as differential activation was found in the vmPFC depending on whether or not money was invested in the Trust Game with or without a threat of a financial penalty for non-repayment (Li et al. 2009).

Reciprocity also appears to have links in the caudate. In the Trust Game, not only was the trustee's caudate activation related to the investor's reciprocity in previous rounds, acting like a signal of "intention to trust", but this signal shifted temporally as the task progressed (King-Casas et al. 2005). Early trials showed the signal occurring post investor decision; while in later trials, the signal occurred before the investor revealed the investment decision. This mimics the shift also seen in the previously mentioned reinforcement learning models of reward prediction errors (Schultz et al. 1997, Schultz 2002). Providing the trustee with information on the investor's general personality, with an either positive or

negative moral spin, can mitigate this effect and reduce the caudate activation (Delgado et al. 2005a). This suggests that pre-existing beliefs reduce the amount of learning necessary on each trial. If the information provided has no moral component, or a neutral moral focus, the caudate activation remained the same as had no information been provided (Delgado et al. 2005a). The caudate was also recruited when choosing to punish a partner who betrayed the reciprocation norm in the Trust Game (de Quervain et al. 2004).

Some hypothesize that one motivation for reciprocal behaviour lies in a drive to minimize negative affect, specifically guilt (Rilling and Sanfey 2011). When a player broke a promise to reciprocate, as compared to following through with the promise, there was activation in the ACC and dlPFC (Baumgartner et al. 2009), both of which have consistently been implicated in conflict and cognitive control (Eriksen and Eriksen 1974, Botvinick et al. 2001, Miller and Cohen 2001, Ullsperger and von Cramon 2001, Pochon et al. 2008). This may suggest that for some, our default tendency is to keep a promise of reciprocity because breaking the promise requires additional cognitive effort. Notably, this appears to hold true only for those with pro-social tendencies; that is, for players who valued the outcomes of others (van den Bos et al. 2009). Greater ventral striatal activity was seen in pro-social players when they chose to reciprocate rather than defect. In contrast, pro-self players, those who did not value the outcomes of others, showed the opposite effect. Additionally, this study reported greater insula activation in pro-social players choosing to defect, while pro-self players showed this greater insula activation when choosing to reciprocate. Taken together these findings suggest that, depending on one's social value orientation, reciprocation may be intrinsically rewarding and defection intrinsically aversive, or vice-versa (van den Bos et al. 2009).

Recent work goes beyond the rewarding (or punishing) effects of social decision-making to study emotional links. Neurobiological research in emotion has reliably identified a set of structures engaged during emotion processing. These include the striatum, caudate, vmPFC, orbitofrontal cortex, ACC, amygdala and insula (Dalgleish 2004). Non-reciprocity and inequity, such as unfair offers in the Ultimatum Game, elicit observable behavioural negative emotional states (Pillutla and Murnighan 1996). As unfair behaviour increased in

the Ultimatum Game, greater activation was elicited in the anterior insula (Sanfey et al. 2003). Additionally, increased activation in this area was predictive of a player rejecting the offer presented (Sanfey et al. 2003). The anterior insula was again implicated in unfairness when increased anterior insula activation in response to unreciprocated cooperation was linked to higher rates of defection in the Prisoner's Dilemma (Rilling et al. 2004). The anterior insula has been linked to physically painful (Derbyshire et al. 1997) and disgusting (Calder et al. 2001) stimuli, as well as interoception (i.e. awareness of internal bodily senses; Critchley, Wiens, Rotshtien, Ohman, & Dolan, 2004). This suggests that this region may play a role in discouraging trust and cooperation by tagging a social interaction as aversive (Sanfey 2007).

As in other decision-making tasks described above, vmPFC patients have been shown to display deficits in these tasks. Lesion patients exhibit less trust and appear to be less trustworthy in the Trust Game (Karjbich et al. 2009). Relevant fMRI findings show that a decision to trust, compared to deciding to reciprocate trust, recruited greater activation in the frontal pole (Krueger et al. 2008). This area has also been linked both to the valuing of future rewards (Kable and Glimcher 2007) and to the protection of long-term interests from immediate demands (Koechlin and Hyafil 2007). For these reasons it has been hypothesized that the vmPFC may register the benefits of successful partnerships for long-term goals, which can aid in the decisions to trust over the fear of potential betrayal (Rilling and Sanfey 2011).

Overall, society runs on the established social norms of cooperation, reciprocity and trust. These social norms aid in the shaping of behaviour, as we are generally highly sensitive to the approval of others (Rilling and Sanfey 2011). When subjects were asked to make decisions regarding donating money to charities, having peers present increased both the rates of donations and activity in the ventral striatum compared to making the decision in the absence of any company (Izuma et al. 2009). This activation occurred in the same region that shows activation when subjects received positive feedback from peers and to the receipt of monetary rewards in non-social tasks (Izuma et al. 2008). Based on these findings it seems plausible that social approval is intrinsically rewarding and may, in part, motivate

norm-abiding decision-making. Previous studies have also found that we can trigger a prediction error signal in the putative reinforcement learning circuitry by creating conflict with a group opinion (Klucharev et al. 2009). When participants learned that their decision differed from a group's, there was activation in the rostral cingulate zone and deactivation in the nucleus accumbens (NAcc). When allowed to reassess and conform to the group, conformity was linked to increased activation in the rostral cingulate zone and even greater deactivation of the NAcc. Additionally, participants who displayed the greatest amount of conformity also displayed greater deactivation in the ventral striatum when in their initial response to discovering their unconformity (Klucharev et al. 2009). Thus it appears that these error-related signals notify us when we deviate from the norm of social conformity and affect our future decision-making. However, it is important to remind ourselves that despite these robust findings there is still individual variability. Behavioural economics studies have shown that some individuals will only make norm-abiding decisions under the threat of punishment (Fehr and Gachter 2002). In this study, the players who showed the greatest amount of change between no threat and threat of punishment conditions also displayed the greatest increase in activation of the lateral OFC and right dlPFC.

Previous work in neuroeconomics finds significantly reduced activation in brain regions associated with problem solving in subjects who are given expert advice and follow it – a phenomenon dubbed *cognitive offloading* (Engelmann et al. 2009). However, the role of strong positive emotions (Darwin 1871, Milgram 1974, Gennaioli et al. 2012) in subjects obeying experts remains uncertain. Moreover, people do not always trust experts. The triggers that switch the brain from Kahneman and Tversky's (2011) its default inexpensive "fast thinking" (cognitive shortcuts such as deference to experts) to the more costly "slow thinking" (rational decision-making as in neoclassical economics) likewise remain uncertain. Decisions regarding investments are of particular interest in economics as these decisions determine an economy's allocation of capital and thus its ability to sustain high living standards. Given a possibly intrinsic motivation to comply with social norms, an improved understanding of the effects of expert advice on financial decision-making promises not just to further our understanding of bounded rationality in economics, but also

to provide both investors and public policy makers with better insights into how financial markers work, or fail to work.

Chapter 4. Gambling

4.1 DSM-5 Criteria

A. Persistent and recurrent problematic gambling behaviour leading to clinically significant impairment or distress, as indicated by the individual exhibiting four (or more) of the following in a 12-month period:

1. Needs to gamble with increasing amounts of money in order to achieve the desired excitement.
2. Is restless or irritable when attempting to cut down or stop gambling.
3. Has made repeated unsuccessful efforts to control, cut back, or stop gambling.
4. Is often preoccupied with gambling (e.g., having persistent thoughts of reliving past gambling experiences, handicapping or planning the next venture, thinking of ways to get money with which to gamble).
5. Often gambles when feeling distressed (e.g., helpless, guilty, anxious, depressed).
6. After losing money gambling, often returns another day to get even ("chasing" one's losses).
7. Lies to conceal the extent of involvement with gambling.
8. Has jeopardized or lost a significant relationship, job, or educational or career opportunity because of gambling.
9. Relies on others to provide money to relieve desperate financial situations caused by gambling.

B. The gambling behaviour is not better explained by a manic episode.

4.2 Psychological and Neurobiological evidence

With the increase in popularity of gambling as a form of entertainment and a focus for social gatherings, problem gambling has become an increasing area of research. Factors repeatedly studied include demographic characteristics (Volberg 1994, Rahman et al. 2012), risk factors/onset (Burge et al. 2006, Yip et al. 2011, Rahman et al. 2012), and treatment options (Petry 2002, Leung and Cottler 2009). Despite this research, there remain

conflicting hypotheses regarding the links between gambling and psychological factors, including risk tolerance, impulsivity, self-esteem, and a possible link to adverse childhood experiences.

With increasing suggestions that gambling is similar to addictions, and that risk tolerance changes are seen in those with addictions (Baler and Volkow 2011), it may useful to further examine risk tolerance as a possible contributing factor in gambling. Previous studies in this area have had mixed results, with some finding physiological evidence of risk tolerance in gamblers compared to non-gamblers (Griffiths 1993), while others find no such evidence (Coventry and Norman 1997). Including several measures of risk tolerance into one study may help clarify this issue, and determine in what areas risk differences between gamblers and non-gamblers may exist. Furthermore, there have been no previous studies examining if risk tolerance in gamblers is specific to the financial domain, or if it expands to other domains such as health, social, recreational and ethical risk tolerance.

Theories of low self-esteem in gamblers stem from the belief that gambling holds many similarities to addictions, whereby addictive behaviour may be preceded by feelings of low self-worth and elicit temporary relief of these negative thoughts through pleasurable experience (Brown 1993, Jacobs 1993, Rosenthal 1993). Other potentially addicting behaviours, such as the newly termed "Internet Gaming Disorder" (APA 2013) have also been linked to lower levels of self-esteem (Niemz et al. 2005), providing support that gamblers may also suffer from lower levels of self-esteem. In contrast, others have posited that gamblers may experience high levels of self-esteem as they view themselves as highly skilled in their gambling device of choice (Kusyszyn and Rutter 1985). This hypothesis may be correct when looking only at domain-specific self-esteem; however, global self-esteem encompasses more than just gambling skills.

Adverse childhood experiences were the focus of a major study by Felitti et al. (1998) on overall mental and physical well-being. They found that higher levels of childhood adversity might account for as much as one-half to two-thirds of drug abuse as well as increase the likelihood for experiencing mental health disorders such as depression and hallucinations (Felitti et al. 1998). It follows that increased levels of childhood adversity

may lead to lower levels of self-esteem and may also increase risk of developing gambling disorder.

It has been suggested that the process by which Gambling Disorder develops consists of four important cognitive-emotional processes (van Holst et al. 2010): behavioural conditioning; increased salience of and response to gambling cues; impulsivity; and impairment of executive functions.

4.2.1 Behavioural conditioning dependent on reward and punishment sensitivity

4.2.1.1 Concept

Common to all gamblers in the initial stages of development of problematic gambling behaviour is the influence of operant and classical conditioning (Blaszczynski and Nower 2002, Redish et al. 2007). Operant conditioning arises when intermittent rewards are provided according to a variable-ratio schedule during gambling which has been reported to produce "drug-induced-high" type arousal (Blaszczynski and Nower 2002). As this stimulus-response pairing repeatedly occurs, the arousal from winning a reward becomes classically conditioned to the stimuli of the gambling surroundings/environment. This pairing can be particularly potent for a gambler who has a series of early wins, as this is predictive of longer gambling continuation (Coventry 2001). Gambling may also act as a negative reinforcer as it can reduce aversive state such as anxiety or depression (Blaszczynski and Nower 2002, Sharpe 2002).

Previous research has implicated a need for more dopamine release due to lower dopamine receptor density in substance abuse populations (Thanos et al. 2001, Volkow et al. 2002). It is hypothesized that these less reward sensitive populations are more likely to engage in reward seeking behaviours and decisions, and that this may serve as a factor in the susceptibility to ongoing gambling behaviour (van Holst et al. 2010). For someone who, neurobiologically, requires more dopamine stimulation in order to experience the same "reward high", larger rewards or longer periods of gambling may be needed compared to individuals who have higher dopamine receptor sensitivity (van Holst et al. 2010).

Little research has been conducted on the role that punishment may have on the development of gambling disorder. Some have suggested that those who are more likely to develop an addiction may have a diminished punishment sensitivity, leading to poor use of feedback which in turn leads to continued disadvantageous choices (Eisen et al. 2001). Thus, it appears that problem gambling may be explained by a combination of both an increased sensitivity to reward and a decreased sensitivity to punishment.

4.2.1.2. Cognitive Behavioural Findings of Reward Processing/Sensitivity

Increased reward seeking behaviour and decreased sensitivity to loss during behavioural tasks have been implicated in previous research on problem gambling (Vitaro et al. 1999, Petry 2001a, Cavedini et al. 2002b). Pathological gamblers displayed worse performance compared to healthy controls on a computerized card task where participants were tasked with playing cards one at a time to either gain or lose money, until they wanted to stop playing any more cards (Goudriaan et al. 2005). After each card either a win or a loss was revealed. At the onset of the task the majority of the cards yielded rewards; however, as the task progressed the cards began to shift towards losses. Gamblers continued to play more cards despite the shift to greater loss compared to healthy controls. The authors concluded that this behavioural difference indicated an increase in reward seeking behaviour or a diminished sensitivity to punishment in the pathological gambling group (Goudriaan et al. 2005).

However, there have also been some self-report scale findings that do not support this proposed increase in reward sensitivity and decrease in punishment sensitivity. Leiserson and Pihl (2007) found that gamblers and controls did not differ significantly on self-reported extraversion (how reward sensitivity was operationalized), sensation seeking (the authors assessment of reward seeking behaviour), or inhibition of reward seeking behaviours. Reward and punishment sensitivity also did not differ between the groups in this study, a finding which was supported by Goudriaan et al. (2005) self-report findings that pathological gamblers and controls did not differ on the Behavioural Inhibition Scale and the Behavioural Activation Scale.

4.2.1.3. Neuroimaging Findings of Reward Processing/Sensitivity

As previously reviewed, areas that are commonly activated during rewarding events include the orbitofrontal cortex (OFC), amygdala, striatum and Nucleus Accumbens (NAcc). A positron emission tomography (PET) study by Hollander et al. (2005), focused on monetary reward processing, found that increased metabolic rate in the visual cortex, cingulate gyrus, putamen and prefrontal areas was associated with gambling for money in pathological gamblers. However, this study did not include a control group thus the significance of these findings is unclear. In an fMRI study that did include a control group, a decrease in the ventral striatal and ventromedial prefrontal cortex (vmPFC) activation during receipt of monetary rewards was found in pathological gamblers compared to healthy controls (Reuter et al. 2005). Additionally, the authors found that there was a negative correlation between the severity of gambling problems and activation in the ventral striatum (Reuter et al. 2005). These results support the hypothesis that gamblers may have a decreased reward sensitivity compared to non-gamblers. de Ruiter et al. (2008) found that pathological gamblers performed poorly compared to nicotine dependent men and healthy controls during an affective switching task. Participants were asked to respond to one of two stimuli presented at each trial and are then given either positive or negative feedback (8:2 ratio). In this study pathological gamblers performed the worst, followed by nicotine dependent men, with healthy controls performing the best.

In monetary gains trials a pattern of ventrolateral prefrontal cortex (vlPFC), frontal operculum, right parietal and occipital cortex, bilateral caudate nucleus and sub-thalamic region activation was found (de Ruiter et al. 2008). vlPFC activation was decreased in the pathological gamblers compared to healthy controls during these monetary gains trials. Activation patterns in the right frontal operculum, insular cortex and sub-thalamic regions were associated with monetary loss, with pathological gamblers showing less vlPFC activation compared to healthy controls (de Ruiter et al. 2008). This decreased vlPFC activation in response to monetary gains in pathological gamblers supports the hypothesis of a lowered reward sensitivity, and the decreased vlPFC activation in response to monetary loss supports the hypothesis of a lowered sensitivity to punishment (Goudriaan et al. 2005).

In summary, findings to date suggest that the ventral striatum, vmPFC and vlPFC are important in reward processing and sensitivity and that these areas are affected in pathological gamblers. This suggests pathological gamblers may be victim of a precursory dysfunctional reward system or that ongoing gambling may lead to the development of dysfunction in these reward related areas. It is, however, not clear if this diminished reward and punishment sensitivity is a precursor or a consequence of gambling behaviour.

4.2.2 Increased salience of and response to gambling cues (cue reactivity)

4.2.2.1 Concept

Cue reactivity has been associated with cravings and attentional bias to addiction-related stimuli, and is also a central characteristic of pathological gambling (Goldstein and Volkow 2002, Kalivas and Volkow 2005, Potenza 2008). Exposures to stimuli previously associated with addictive behaviours have been shown to stimulate relapse amongst substance abuse populations (Grüsser et al. 2004, Heinz et al. 2007). Cravings are defined as a physiological reaction and desire to partake in behaviour such as gambling (van Holst et al. 2010) while attentional bias in addiction is defined as a greater attention to stimuli related to the addiction compared to non-addiction related stimuli (van Holst et al. 2010). Attentional bias is often operationalized as differences in reaction times (Cox et al. 2006). Ongoing gambling or relapse of problem gambling behaviour post treatment is likely contributed to by biased attention and gambling related cue reactivity thus these are believed to play an important role in gambling disorder.

4.2.2.2 Cognitive behavioural findings of attentional bias towards gambling cues

In a study by Zack and Poulos (2003) the influence of a psychostimulant drug (D-amphetamine; AMPH) on gambling motivation was examined. An increase in desire to gamble post AMPH distribution was observed in the pathological gambler group but not in other groups. In a reading task with both gambling relevant and non-relevant words, AMPH produced an increase in reading speed for all words in the non-gambling groups while these improvements were only seen with gambling related words in the gambling group. In addition, in the gambling group, the AMPH actually produced a negative effect on performance of reading the non-gambling related words (Zack and Poulos 2003).

Strengthening these results was the inclusion of a placebo condition, in which no group differences emerged in reading speed (Zack and Poulos 2003). Haloperidol, a selective dopamine D2 antagonist, enhanced the salience of gambling words in problem gamblers in a similar reading task (Zack and Poulos 2007). The desire to gamble during a slot machine game was also increased with the administration of haloperidol; however, pre-game desire was not influenced (Zack and Poulos 2007).

4.2.2.3 Neuroimaging findings of cue reactivity

Previous work with substance dependent populations has found that the amygdala, ACC, OFC and vlPFC are associated with viewing addiction-related cues (Braus et al. 2001, George et al. 2001, Grüsser et al. 2004, Tapert et al. 2004). Potenza et al. (2003b) were the first to conduct an fMRI study on gambling urges. They tasked their participants with viewing a tape designed to evoke emotional and motivational cues to gambling. The pathological gambling group compared to healthy controls exhibited less activation in the cingulate gyrus, OFC, caudate, basal ganglia and thalamic areas (Potenza et al. 2003b). Crockford et al. (2005) found, with a similar gambling movie paradigm, an increased BOLD signal in the right dlPFC, right inferior frontal gyrus, medial frontal gyrus, left parahippocampal region and left occipital cortex when pathological gamblers are presented with gambling-related cues. The authors also found visual processing stream differences between gamblers and controls – pathological gamblers recruited a dorsal visual processing stream while healthy controls recruited a ventral visual stream when viewing the gambling movies (Crockford et al. 2005). While the authors argue that these results suggest that pathological gamblers compared to healthy controls recruit regions that comprise parts of the dlPFC network and are associated with attention, reward expectancy, and behavioural planning for attaining rewards, it is also possible that rather than cue reactivity or craving, the movies elicited a conditioned response as conditioned responses have been linked to activity in the dlPFC and dorsal striatum (Everitt and Robbins 2005). Increases in visual processing, vlPFC, amygdala and parahippocampal gyrus activation have also been demonstrated in gambling subjects compared to heavy smokers and healthy controls in a cue reactivity paradigm with gambling related, smoking related and neutral pictures (Goudriaan et al. 2010). The increased visual stream activation was hypothesized to relate

to an increased salience of the gambling stimuli, while the amygdala and parahippocampal gyrus activation indicates the activation of the emotion, motivation and memory-related circuitry (Goudriaan et al. 2010). Self-reported urges to gamble have also been found to correlate with activity in the temporal pole for pathological gamblers (Balodis et al. 2012b).

It can be seen that cue reactivity primarily involves different regions to reward processing and sensitivity; being focused more on dlPFC, amygdala, and parahippocampal gyrus, among others. That these areas also activate dysfunctionally in gamblers compared to non-gamblers, we can begin to see how gambling addiction is manifested throughout the entire brain and the importance of examining gambling from many different perspectives with varying tasks.

4.2.3 Impulsivity as a trait

4.2.3.1 Concept

Impulsivity is often equated with disinhibition in the field of cognitive neuroscience (van Holst et al. 2010) which represents a state in which mechanisms that usually suppress reward-driven responses are dysfunctional or unable to meet the demands being put upon it (Aron 2007). It is hypothesized that pathological gamblers have deficits that encourage impulsive decision-making.

4.2.3.2 Cognitive behavioural findings of impulsivity

Several studies have suggested that the psychological factor of impulsiveness is linked to various addictions including alcohol and drugs (Cloninger et al. 1988, Luengo et al. 1994, Verdejo-Garcia et al. 2008), but studies of a possible relationship between impulsiveness and gambling have been much more mixed. Thus, some studies have found that, compared to controls, gamblers have high levels of impulsivity (McCormick et al. 1987, Carlton and Manowitz 1994, Castellani et al. 1996, Blasczcynski et al. 1997, Steel and Blaszczynski 1998, Nower et al. 2004, Kertzman et al. 2008), while others have shown that gamblers do not differ, or even have lower levels of impulsivity than controls (Blaszczynski et al. 1986, Dickerson et al. 1987, Allcock and Grace 1988, Blaszczynski et al. 1990). These contradictory findings are further complicated by the fact that in many studies the gamblers

were also substance abusers, while some studies failed to report on history of substance abuse, which may itself have an effect on impulsivity scores. Others have suggested that pathological gambling and substance abuse can have an additive effect on scores of impulsiveness (Petry and Casarella 1999, Petry 2001a). Fuentes et al. (2006) found that pathological gamblers with co-morbid disorders were more impulsive than pathological gamblers without any co-morbid disorders, and healthy controls were the least impulsive of the three groups. Controlling for substance abuse may help clarify the role of personal impulsivity fits into problem gambling.

4.2.3.3 Neuroimaging findings of impulsivity

Potenza et al. (2003a) has carried out one of the few neuroimaging studies on impulsivity and problem gamblers to date. Participants were asked to perform the Stroop task both in and outside the fMRI scanner. Behavioural differences were not found between the groups and overall the task elicited activation in the dorsal anterior cingulate cortex (ACC), bilateral inferior frontal gyrus, right insula and right thalamus. The two groups were only differentiated in the activation patterns of the vlPFC, with pathological gamblers showing decreased activation compared to healthy controls in the left middle and superior frontal gyri (Potenza et al. 2003a). In a recent study by Balodis et al. (2012a) where impulsivity was assessed and participants asked to perform a monetary incentive delay task, the authors found relatively decreased corticostriatal neurocircuitry activation during phases of reward processing. The authors concluded that their results, combined with previous findings in alcohol dependence, suggest that diminished ventral striatal activation to reward anticipation may reflect impulsivity in addictions (Balodis et al. 2012a).

It can be seen that there have been relatively few neuroimaging studies of impulsivity, but it likely involves activation within the anterior cingulate cortex, insula, and thalamus. Again, these findings differ both from the regions involved in reward processing and sensitivity and from those primarily involved in cue reactivity. These findings of different regional activation tend to validate that each of these processes has a differing set of neurological underpinnings, and that they represent meaningful differences.

4.2.4 Impairment of executive functioning

4.2.4.1 Concept

There have been suggestions that pathological gamblers have both attentional and executive dysfunctions (Rugle and Melamed 1993, Specker et al. 1995). Tasks previously discussed in Chapter 2 have been employed to determine the extent of this dysfunction.

4.2.4.2 Cognitive behavioural findings of executive functioning

Much like frontal lesion patients, substance abuse individuals have also demonstrated the disadvantageous preference for the "short-term gain/long-term loss" decks when presented with the Iowa Gambling Task (IGT) (Petry et al. 1998, Yechiam et al. 2005). This pattern, of poorer performance in the IGT has been consistently repeated in pathological gamblers (Cavedini et al. 2002b, Kertzman et al. 2011, Brevers et al. 2012, Brevers et al. 2013b). Pathological gamblers appear to display a stubborn preference for the harmful decks in the IGT. However, there have been some studies that have not found this significant difference between the two populations (Tanabe et al. 2007, Linnet et al. 2011a, Linnet et al. 2011b, De Wilde et al. 2013). This lack of consistent support may be due to small sample sizes as well the heterogeneity of gamblers as a group (strategic vs. non-strategic game preference/psychological profiles) (Brevers et al. 2013). For instance, both pathological gamblers and controls who score highly on sensation seeking displayed significantly increased activity in the ventral striatum during the IGT (Peterson et al. 2010), an area previously linked to anticipation of monetary rewards (Knutson et al. 2003).

Pathological gamblers were also found to be impaired in decision-making using the Game of Dice task (GDT) (Brand et al. 2005b, Labudda et al. 2007). Importantly, in Brand et al. (2005), the gamblers were also assessed with a neuropsychological battery and, as a group, scored within normal ranges. The frequency of disadvantageous decisions was correlated with specific executive functions (categorization, set-shifting, cognitive flexibility and interference susceptibility) but not with personality traits. Pathological gamblers appear to display a failure to use negative feedback after a disadvantageous choice to improve decision-making on following rounds compared to controls (Brand et al. 2005b). Additionally, Labudda et al. (2007) tested the neuroendocrine responses (salivary cortisol

and alpha-amylase concentration; sAA) before and during task performance. Post-hoc analysis revealed that heightened sAA during the task was found in patients who demonstrated *less* disadvantageous decision-making compared to other patients (Labudda et al. 2007). It was proposed that, as a marker of sympathetic nervous system activity, the increase of sAA in patients with less severe decision-making deficits may be indicative of a somatic marker affecting the decision-making process (Labudda et al. 2007).

Both Rugle and Melamed (1993) and Forbush et al. (2008) administered the Wisconsin Card Sorting Task (WCST) to gamblers and controls and found that gamblers performed significantly worse than the controls. Forbush et al. (2008) also administered two other measures of cognitive flexibility, the Controlled Oral Work Association Test and the Trail Making Task A and B, on both of which gamblers performed poorly compared to controls. Marazziti et al. (2008) found supporting evidence for the WCST results as it was the only task (administered alongside a verbal fluency test and the Wechsler memory scale) to show group differences, with pathological gamblers demonstrating greater difficulty on the task than healthy controls.

Delay discounting, the preference of an individual for smaller immediate rewards or larger future/delayed rewards, is another measure often used to examine executive functioning in gamblers. Petry and Casaella (1999) found that gamblers discounted delayed rewards, that is they preferred to take the smaller immediate rewards, at a higher rate than substance dependent and healthy control groups. This group of gamblers, however, was also a substance dependent group and thus it is unclear if gambling alone produces these effects to a greater extent than substance abuse alone. In order to make this distinction, in a later study (Petry 2001b), a pathological gambling group without substance dependence was also recruited and while this group displayed greater delay discounting than healthy controls, the group of gamblers with substance dependence displayed the highest levels of delay discounting suggesting that a combination of both pathological gambling and substance dependence produces the greatest discounting of delayed rewards.

4.2.4.3 Neuroimaging findings of executive functioning

The impulsive amygdala-striatum network (discussed in Chapter 2) is believed to be involved in the process by which reward-seeking transfers from being a controlled to an automatic habitual behaviour (Everitt et al. 1999, Everitt and Robbins 2005). It has been proposed that association clusters of stimuli are created, and are gradually strengthened through classical conditioning (Hofmann et al. 2008, Hofmann et al. 2009). In the case of gambling, when a gambling-related cue is encountered a gambling-focused cluster may activate, automatically triggering a positive incentive value impulse attributed to gambling (Stacy and Wiers 2010). Studies of this implicit association have found that pathological gamblers exhibited only positive, not negative, implicit associations towards gambling cues (Yi and Kanetkar 2010, Brevers et al. 2013c). Positron emission tomography (PET) studies have found that in pathological gamblers poor performance on the IGT was associated with dopaminergic release in the ventral striatum compared to controls (Linnet et al. 2010, Linnet et al. 2011a). Controls, in contrast, show this dopamine release when choosing cards from the advantageous decks (Linnet et al. 2010, Linnet et al. 2011a). An fMRI study (Power et al. 2012) found that pathological gamblers exhibited greater activation in areas linked to the integration of emotional and cognitive input such as the OFC (Rolls and Grabenhorst 2008), reactivity to emotional information such as the amygdala (Bechara et al. 2003), and short-term reward learning such as the caudate nucleus (Haruno and Kawato 2006).

Miedl et al. (2012) also found increased delayed discounting in pathological gamblers in an fMRI study. The authors found a negative correlation between gambling severity and valuation signals in the ventral striatum, vmPFC and ventral tegmental area for delayed rewards (Miedl et al. 2012). Importantly, they found reward representation differences in gamblers depending on condition – neural value correlations increase in delayed discounting and decreased in probability discounting throughout the reward system. In an fMRI study employing the IGT, pathological gamblers exhibited increased OFC, caudate, hippocampus and amygdala activation during high-risk deck selection (Power et al. 2012) which is consistent with previous studies with the addition that previous studies have also implicated the amygdala, OFC as well as the ACC (Ernst et al. 2002, Fukui et al. 2005, Li

et al. 2010). As these regions make up the dopamine reward pathways these results provide support for the hypothesis that an increased salience of immediate potential rewards relative to future losses may be one of the mechanisms by which gambling behaviour is maintained.

Taken together this suggests that there exist several regions of dysfunction within gambling disordered individuals in terms of reward processing which leads to disadvantageous decision-making observed in behavioural studies. It remains to be determined if this dysfunction precedes the onset of gambling behaviour and that those with this neurobiological predisposition are thus more "hard-wired" to developing a gambling disorder or if through experience and reinforcement these neurobiological changes are developed.

4.2.5 The "near-miss" cognitive distortion

Electronic gambling machines (EGMs) are believed to lead to problematic gambling in part due to the reinforcement schedule and also in how the outcome of each gamble is displayed (Parke and Griffiths 2006). A "near-miss" occurs when a loss resembles an actual win in physical display, for example, two out of three matching symbols on a slot machine. These occurrences are hypothesized to play a role in the maintenance of gambling behaviour (Parke and Griffiths 2006). Physiological changes, such as increased heart rate, blood pressure, electrodermal activity and cortisol, are found in gamblers when they are presented with near-misses (Anderson and Brown 1984, Meyer et al. 2000, Coventry and Hudson 2001). fMRI studies have found that near-misses activate overlapping brain regions that are associated with winning such as the ventral striatum, anterior insula and medial prefrontal cortex in pathological gamblers (Clark et al. 2009, Chase and Clark 2010). It has been suggested that this reward-related circuitry activation during near-misses helps to maintain excessive gambling (Clark et al. 2009, Chase and Clark 2010). Interestingly, non-pathological gamblers have also displayed tendencies to initiate new trials quickly as well as increased gambling persistence and higher ratings of chances to win post near-miss outcomes compared to outright wins or losses (Kassinove and Schare 2001, Dixon and Schreiber 2004, Dillen and Dixon 2008, Clark et al. 2009, Billieux et al. 2012). The

susceptibility to gambling-related cognitive distortions of non-pathological gamblers is correlated with anterior insula response to near-misses (Clark et al. 2009). Pathological gamblers, alternatively, show responses in the midbrain rather than the insula correlate with gambling severity (Chase and Clark 2010).

A study (Dymond et al. 2014) was recently conducted combing fMRI with magnetoencephalography (MEG) to examine the near-miss phenomenon in a slot machine task. This is the first study to combine these techniques, which allow the spatial resolution of fMRI and the temporal resolution of MEG to investigate the temporal dynamics and oscillatory changes involved (Dymond et al. 2014). Increases in insula and right OFC BOLD signal and theta power were associated with gambling severity, demonstrating that oscillatory power changes overlapping with previous fMRI findings are found in pathological gambling. This supports the role that reward-related brain responses in near-miss outcomes plays in the maintenance of gambling behaviour (Dymond et al. 2014).

It can be seen that in reinforcing gambling, through "near-miss" cognitive distortions, other brain regions appear particularly important, including the insula and the right OFC. Since activation in the insula was found to be correlated to susceptibility to gambling-related cognitive distortions in non-pathological gamblers during "near-miss" events and has also been found to associate with gambling severity this may implicate this region as another key region in the development and maintenance of gambling disorder. Through studies of various cognitive and behavioural tendencies, we have seen that each recruits a unique pattern of brain regions and that these regions are all important in gambling disorder.

4.3 Current Treatment for Pathological Gambling/Gambling Disorder

There have been many potential therapies proposed to help individuals with pathological gambling. The following is just a brief description of some of the more widely used treatments.

4.3.1 Cognitive-Behavioural Therapy (CBT)

In adapted versions of CBT for pathological gamblers (Sylvain et al. 1997, Petry 2005b, Ladouceur and Lachance 2007a, Ladouceur and Lachance 2007b), individuals are taught to examine positive and negative consequences of their gambling triggers and identify the cognitive biases, such as luck-related beliefs, for their gambling behaviour. Interpersonal and conflict resolution (decisions between short-term enjoyment compared to long term punishment) skills may also be included in CBT as a way to aid the individual in understanding that while gambling may have short-term pleasure, it also has long-term consequences such as problems with friends, family and the legal system (Potenza et al. 2013). Also addressed are financial management skills and debt settlement, two important concerns when it comes to pathological gamblers (Petry 2005a). Some speculations as to the neurobiological effects of CBT have been raised. The development of skills to cope with gambling cues and altering decision-making when these cues appear is posited to enhance prefrontal cortical function and strengthening control over motivation (Potenza et al. 2013). A reduction in the cognitive bias of "near-misses" may involve the balancing of activation in the neural systems that code for conflicting motivational states, while learning financial management skills may alter the processing of immediate vs. delayed rewards in the ventral striatum, insula and vmPFC (Potenza et al. 2013). CBT appears to be effective when delivered in conjunction with pharmacotherapy and is also effective in individuals with co-morbid disorders (Petry 2005a, Toneatto et al. 2009, Champine and Petry 2010) and there appears to be long lasting treatment efficacy (Gooding and Tarrier 2009).

4.3.2 Exposure Therapies

Exposure therapies are based on classical condition. There are several different methods to exposure therapy such as aversion therapy, imaginal desensitization and in-vivo exposure. Aversion therapy involves the behaviour being repeatedly paired with aversive stimuli to reduce the frequency of the behaviour. Imaginal desensitization involves guiding individuals through imaginative experiences of gambling and then learning to control their associated physiological responses. This is to aid them in controlling their behaviour in future exposures to gambling cues. In-vivo exposure involves exposing the individual to

gambling to become desensitized and develop different habits. It is hypothesized that these therapies, which aim to change habitual responses, may alter the habit-based processing of the OFC and dorsal striatal regions (Rogers et al. 2000, Hampshire and Owen 2006, Yin and Knowlton 2006). The reported success of these treatments varies (Tavares et al. 2003, Hodgins and Peden 2008) with imaginal desensitization having been shown to have longer-term reductions in gambling compared to the other forms (McConaghy et al. 1991).

4.3.3 Motivational Therapies

This treatment technique involves "rolling with a patient's resistance and exploring with patients in an unbiased fashion the patients' story regarding their engagement in a specific behaviour" (Potenza et al. 2013). This is a well-validated treatment of substance addictions (Miller 1996, Miller et al. 2003) and it has been found that those who express greater commitment to changing their gambling behaviour have better outcomes than those with weaker expressions of commitment (Hodgins et al. 2009). It is thought that motivational therapies may dampen the addiction-related reward and motivational circuitry (Feldstein Ewing et al. 2011).

4.3.4 Pharmacotherapies

Currently, there has yet to be a medication to be approved for the treatment of gambling disorder/pathological gambling (Grant et al. 2014). A recent meta-analysis reviewed 14 randomized, placebo-controlled pharmacological treatments for pathological gamblers (Bartley and Bloch 2013). They found that opiate antagonists had a small but significant benefit compared to placebo; however, overall the study concluded that the current data provides limited support for any pharmacological agent in treating gambling. Other studies have found some evidence for the use of naltrexone (an opiate antagonist) compared to topiramate, bupropion and escitalopram (Rosenberg et al. 2013). Grant et al. (2014) suggest that the heterogeneity of gamblers may be one source of complication when searching for an effective pharmacotherapy and suggest that future research should use larger sample sizes that are representative of the population including minority groups to increase power as well as include longitudinal assessments.

4.3.5 Treatment outcomes

Despite many treatment options, the outcome for those with gambling is often poor, and response to treatments is often only moderate in terms of impact as well as in terms of the percentage of individuals who are successfully helped. By further understanding of some of the brain mechanisms underlying gambling, it is hoped that treatments may become more effective.

4.4 Summary and rational for study

Based on the reviewed literature, we can see that decision-making, and more specifically decision-making in a gambling disordered population, is highly complex, with many brain regions having been implicated. Forming a better understanding of how financial decision-making is affected may allow for the development of new and more targeted treatment for pathological gamblers. The developmental trajectory of gambling disorder from a neurobiological perspective has also yet to be determined. While it has been established that dysfunctional recruitment of various brain regions related to reward processing, reward sensitivity, executive functions and delayed discounting occurs in gambling disordered individuals, what is less clear is how these dysfunctions develop and when these significant dysfunctions appear. Are individuals biologically predisposed to the development of a gambling problem? Are there any protective factors involved for those who do not develop a gambling disorder? One population that may help to begin to answer these questions is a population of frequent gamblers who do not develop gambling disorder. What does this group of frequent but non-clinical gamblers look like? How does this group behave? What pattern of brain recruitment occurs in this group and is it unique?

In order to explore these questions, a single task was created to capitalize on the similarities between gambling and stock market investing and was used to test both non-gamblers as well as frequent but sub-threshold gamblers. A stock market frame was chosen so that the factor of social influence could also be explored – that is, knowing that gamblers behave differently than non-gamblers in decision-making paradigms, do they also show differences in how their decisions are influenced? Functional magnetic resonance imaging coupled

with psychological surveys provided the opportunity to not only elucidate which brain regions were recruited during the stock market decision-making task but also to determine which, if any, psychological factors may contribute to differences between the groups and the areas of activation. We would expect to see similar patterns of brain activation in this sub-clinical group of gamblers compared to non-gamblers as previously found in pathological gamblers if all these regions truly form the basis of gambling disorder and dysfunctional decision-making. However, this sub-clinical group may display a unique pattern of activation indicating that perhaps not all previously identified regions are as tightly linked to gambling behaviour as a whole as originally thought.

Chapter 5. Methods and Materials

In order to examine the relationship between sub-threshold gamblers vs. non-gamblers, various psychological factors, financial decision-making and how all is manifested in the brain, a battery of psychological measures were administered alongside an fMRI scan where participants were asked to complete a newly developed financial decision-making task. The study was approved by the University of Alberta Health Research Ethics Board.

5.1 Participant Recruitment

Participants were recruited from the University of Alberta and greater Edmonton area. Participants responded to advertisements and indicated interest in the research study. Follow-up calls were placed in order to describe the research study in greater detail. All potential participants received details of the study, and signed an informed consent form. Individuals were screened for the presence of any psychiatric disorder (such as depression), using a semi-structured interview (Appendix A), as well as for any risk associated with having an MRI scan (metal in the body, claustrophobia etc.; Appendix B and C). Recruitment occurred over a 24-month period and a total of 64 adults were recruited into the entire study from a pool of about 100 who were screened.

5.2 Psychological Measures

Cronbach's alpha (α) is reported for each scale, and is a measure of internal reliability, which ranges from zero to one. The closer the score is to one, the greater is the internal reliability.

5.2.1 Gambling.

The Problem Gambling Severity Index (PGSI) (Ferris and Wynne 2001) was used to determine gambling behaviour among participants. This tool was selected due to its short length as one of many psychological measures being delivered. For this screening tool participants were asked to think about the last 12 months and answer nine questions (e.g.

Have you bet more than you could really afford to lose?; α = .913) from 0 (*never*) to 3 (*almost always*). Responses for all questions were summed for a total scale score of problem gambling and used to separate participants into either the control (total scores 0-2) or "Gambler" group (total scores < 3). Total scores of 3-7 indicate a moderate level of problems due to gambling, leading to some negative consequences, with higher scores (8 – 27) indicating greater severity of problems. In this study, those who scored more than 3 were considered to have a gambling problem, while those who scored less than 3 formed the control group.

Additionally, participants also completed Breen and Zuckerman's (1994) Gambling Attitudes and Beliefs Scale (GABS) to assess overall gambling related attitudes. Participants were asked to rate how much they agreed with 35 statements regarding gambling (e.g. Gambling makes me feel really alive) from 1 (*strongly agree*) to 4 (*strongly disagree*). The GABS was scored as the mean of all items, with higher total scores indicating greater endorsement of pro-gambling attitudes and beliefs (α = .908)

5.2.2 Risk Tolerance.

Two measures of risk tolerance were administered. Financial specific risk tolerance scales have not been widely developed as this is typically measured through an entire economic task. Due to the structure and length of our study this was not desirable thus Grable and Lyttons'(1999) 13-item scale was used to determine financial risk tolerance. In this, participants were asked a series of financially related questions (e.g. In terms of experience, how comfortable are you investing in stocks or stock mutual funds?) with ranked multiple-choice answers, each given a score from 1-4. Responses were divided into three subscales: Investment Risk (five items), Risk Comfort and Experience (five items), and Speculative Risk (3 items). Overall risk tolerance was scored as the sum of all responses (α =.698) ranging from 0 – 52. Higher scores reflect greater risk tolerance.

Participants also completed Weber, Blais, and Betz's (2002) 30-item Domain-Specific Risk-Taking (DOSPERT) Scale as a measure of overall risk taking tendencies as this

measure is frequently cited. This measure asked participants to rate willingness to participate in risky activities, as well as asking them to give their overall risk perception of those activities from 1 (*extremely unlikely*) to 7 (*extremely likely*). Scores were calculated by summing the responses. Higher scores on the willingness to participate scale indicate greater likelihood to engage in the activity, and higher scores for the perception of risk scale indicate higher perceived risk of the activity. The DOSPERT assesses both levels of risk in five domains: ethical (e.g. Having an affair with a married man/woman; α's = .773 for willingness to participate and .705 for perception), financial (e.g. Betting a day's income at the horse races; α's = .772 for willingness to participate and .773 for perception), health safety (e.g. Drinking heavily at a social function; α's = .753 for willingness to participate and .820 for perception), social (e.g. Admitting that your tastes are different from those of a friend; α's = .571 for willingness to participate and .695 for perception) and recreational (e.g. Going down a ski run that is beyond your ability; α's = .839 for willingness to participate and .783 for perception).

5.2.3 Impulsivity.

Participants completed the widely used 30-item Barratt Impulsivity Scale [BIS-11; 38] to assess impulsivity. Participants we asked to rate how likely they would be to act or think in different situations (e.g. I do things without thinking) from 1 (*Rarely/Never*) to 4 (*Almost Always/Always*). Responses were summed to obtain scale scores for three second-order factors (Attentional Impulsiveness; 8 items, Motor Impulsiveness; 11 items, Nonplanning Impulsiveness; 11 items) as well as an overall scale score. Higher scores indicate higher levels of impulsivity (α =.747).

5.2.4 Self-esteem.

Self-esteem was measured using Rosenberg's (1965) ten-item scale as it is one of the oldest and most frequently used measure of self-esteem in research. Its short length also contributed to the decision to select this measure. Participants were asked to rate their general feelings about themselves (e.g. On the whole, I am satisfied with myself) from 0 (Strongly Disagree) to 3 (Strongly Agree). Responses were summed to obtain self-esteem score (0-30), with a higher score indicating a higher level of self-esteem (α =.842).

5.2.5 Childhood trauma.

Participants completed the ten-item scale from the Adverse Childhood Experience (ACE) Study (Felitti et al. 1998) to determine presence of trauma during the participant's younger life. This scale is well validated and has been shown to predict a large variety of health outcomes for individuals (Felitti et al. 1998). Participants were asked to indicate whether or not a list of events took place within the first 18 years of life (e.g. Did you live with anyone who was a problem drinker or alcoholic or who used street drugs?). 'Yes' answers were summed to provide a total score (0-10), with a higher score indicating a higher level of adverse childhood experiences ($\alpha = .666$).

5.3 Development of Investment Paradigm

Several phases of development occurred in the creation of the investment paradigm used in the study. In order to create the stock market framing of the task, it was decided that the participants would be asked to choose to either buy or not buy a series of stock options. To ensure that the risk associated with each stock was unambiguous the following information was provided: probability out of 100% of the win and loss potential of the stock as well as the amount of money associated with both the win and loss.

A template of stocks was created to fill all the required trials with all possible combinations of 100% summed totals (10% vs. 90%, 20% vs. 80%, 30% vs. 70%, 40% vs. 60% probability splits were used). Round whole numbers were selected as the value of the financial options for ease of processing and understanding (e.g. 10% of winning $50).

In order to keep the task as short as possible, for the comfort of our participants in the scanner, a 4 second trial length for the decision-making phase of the task was selected. First pilots of the task, proved to be too difficult for participants to both read the on screen information and select a response in that short a time period. The decision-making phase was then increased to 7 seconds, which participants indicated gave them enough time to complete the task.

The outcome of each trial was pre-determined based on the expected value of the stock presented (i.e. the probability of either the win or loss multiplied by the dollar amount associated with that probability). Thus, a participant would win the trial if he/she chose to buy the stock and the expected value of the probability of winning money on the stock was greater than that of losing money. For example, if the stock presented had a 70% chance to win $50 and a 30% chance to lose $100, the expected value of winning is 35 (0.7 x 50) while the expected value of losing is −30 (0.3 x −100). As the expected value of winning outweighs the expected value of losing (the addition of 35 and -30 yields a positive number), the rational decision would be to buy this stock. If participants chose to buy a stock whose expected value for a loss outweighed that for a win, that decision would result in a loss on that trial. For example, if the stock presented had a 40% chance to win $90 and a 60% chance to lose $80, the expected value of winning is 36 (0.4 x 90) and the expected value of losing is −48 (0.6 x −80). As the addition of 36 and -48 yields a negative number, in this case the rational decision would be to not buy the stock. It was possible in theory for a participant to complete the task in an entirely rational manner by simply calculating the expected value for every stock and choosing the appropriate action. Lotteries were assigned low, medium, and high difficulty based on how great a difference there was in expected values - smaller difference in expected values were more difficult trials than ones with large differences in expected values. The presentation of trials was counterbalanced with regard to advice (see below) and difficulty in order to control for order effects.

Trials were followed by one second of feedback, as follows:

- If a participant chose to buy the stock, he/she was informed if that choice resulted with a win or a loss and their total was adjusted accordingly.
- If a participant chose not to buy the stock, the feedback read “No Buy”, and the total remained the same.
- If a participant failed to respond in the allotted time, the feedback read “No Response”, and the total remained the same.

5.3.1 Investment paradigm

Participants completed the investment task that we had developed during their fMRI scan. Participants were told that the investigators were interested in how individuals make investment decisions. On each trial, participants were asked to decide to either 'Buy' or 'Not Buy' a stock based on the following information: the probability of winning a specified amount of money, the probability of losing a specified amount of money, and advice on what action to take. An example of the sequential images shown to each individual per investment decision is shown in Figure 1. Each investment choice was presented for seven seconds and participants were instructed to make their decision within that time frame. All participants were told that advice was being presented to aid in their decision-making process but that they were not required to follow it if they did not want to.

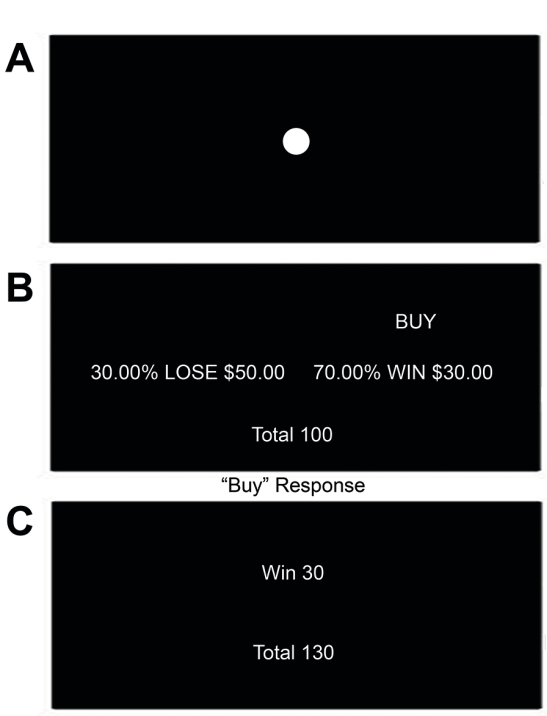

Figure 1. Investment Task

a: *Fixation Point (6-10s): Participants were instructed to attend to the fixation point* ***b:*** *Trial (7s): Participants are presented with a stock and must decide to either "Buy" or "Not Buy". Advice to "Buy" is rational as the expected value of buying the stock (0.7 x 30 = 21) outweighs the expected value of not buying the stock (0.3 x -50 = -15).* ***c:*** *Feedback (1s): Participants are presented with feedback based on their decision (in this case the participant chose to obey the advice and "Buy" thus the trial resulted in a win) and their total is adjusted accordingly.*

Each participant began with a nominal total of $100. If they ended the task with the same amount (or less) in nominal dollars they would take home $45. However, if they increased their earnings in the task, they would be given greater compensation, with a take home amount ranging between $45 - $105. By using real financial incentives participants were more likely to increase the amount of cognitive effort put into the task (Wilcox 1993) and act in ways that more closely mimic real world investing decisions (Wilcox 1993, Harrison 1994).

After completion of the task in the scanner, participants were asked what strategies they used to make their decisions and also for feedback on the advice that was given to them.

5.4 Advice

Healthy participants were divided into two groups based upon what they were told regarding the advice they received about investment decisions during the investment paradigm. Advice as to whether to "Buy" or "Don't Buy" appeared in 80% of the trials (Table 3). Participants in the external expert group ("Expert") were instructed that the advice was given by an outside financial expert with over 20 years of experience in the field of financial investments who had been specifically asked to prepare the advice he would give his own clients in such a situation. Participants in the peer group ("Peer") were instructed that the student who was in charge of running the study was giving the advice. All other aspects of information given to the subjects were identical, as was the training they received. All participants were aware of the order of each task stimulus, what actions they could take and what the consequences of those actions could be. Each participant was allowed a practice run of the task that was equivalent in length to the first run that they would complete in the MRI scanner. During the practice run of 19 trials, seven were "No Advice" trials while the remaining 12 were all "Good Advice" trials. All gamblers were told that an outside financial expert was giving the advice.

Table 3.
Investment task conditions

Trials	Duration of Run	Type of Advice	Type of Buy	Number of Trials
First 1/3 of trials Runs 1 and 2	5 min 30 sec	No Advice	Good Buy	8
			Bad Buy	6
		Good Advice	Good Buy	12
			Bad Buy	12
Second 1/3 of trials Runs 3 and 4	9 min	Good Advice	Good Buy	18
			Bad Buy	16
		Bad Advice	Good Buy	16
			Bad Buy	16
Last 1/3 of trials Runs 5 and 6	5 min 30 sec	No Advice	Good Buy	8
			Bad Buy	6
		Bad Advice	Good Buy	12
			Bad Buy	12

Although participants were not aware of this, all advice was pre-determined and did not differ between groups. The schedule was set up to create credibility for the advice, which would gradually become less and less rational. Thus, the paradigm was divided into six runs. During the first 2 runs, if the suggested advice was followed it would result in a win or no loss (Good Advice). During runs 3 and 4, the advice gradually became less reliable (50:50 mixture of Good Advice and Bad Advice). During runs 5 and 6, whenever the advice was followed this would result in a loss or a failure to win (Bad Advice). Advice

was not given in every trial. Thus, as the advice became increasingly questionable, participants were required to choose between obeying external advice or not, with the only difference between the groups being whether or not they believed it came from an "Expert" or "Peer".

5.5 fMRI acquisition

A 1.5-T Siemens scanner and 8-channel head coil was used for data acquisition at the University of Alberta's Peter S Allen MR Research Centre. Thirty-two axial slices (3 x 3 x 4 mm voxels) were acquired in a descending interleaved order. Functional images were obtained using a gradient echo EPI sequence (TR = 2000 ms, TE = 40 ms, FOV = 256 mm, flip angle = 90°). One hundred forty-four slices were acquired with a T1-weighted pulse sequence in the same location for structural images (MPRAGE, TR = 1670 ms, TE = 3.82 ms, TI = 1100 ms, flip angle - 15°, FOV = 256, 1 mm thick). Images were pre-processed and analyzed using SPM8. Pre-processing steps included 6-parameter rigid body motion correction, slice timing correction, and coregistration to each participants' anatomical image to their functional scans. Structural scans were normalized to the Montreal Neurological Institute (MNI) template, and functional images were normalized to the new anatomical image. Lastly, we performed smoothing using a three-dimensional Gaussian filter (8-mm FWHM). Five participants (four from the "Expert" group; one from the "Peer" group) were excluded from further analyses due to significant movement artifacts that occurred during the scans (pitch, roll or yaw translation greater than 8mm).

Chapter 6. Study 1: Comparison of "Expert" vs. "Peer" Advice in Healthy Controls

6.1 Recruitment and Participants

Participants were recruited from the University of Alberta and greater Edmonton area. Participants responded to advertisements and indicated interest in the research study. Follow-up calls were placed in order to describe the research study in greater detail. All potential participants received details of the study, and signed an informed consent form. Individuals were screened for the presence of any psychiatric disorder (such as depression), using a semi-structured interview (Appendix A), as well as for any risk associated with having an MRI scan (metal in the body, claustrophobia etc.; Appendix B and C). Following this screening a total of 48 individuals (32 males, age range 20 – 39) were recruited into this study. This was a single-blind study in which participants were randomly placed into 4 alternating blocks of either the "Expert" (28 participants) or "Peer" (20 participants) groups.

6.2 Hypotheses

We expected to see brain activation consistent with previous studies of decision-making under conditions of uncertainty. Previous research in risky decision-making has implicated a wide distribution of areas including the ventromedial prefrontal cortex (vmPFC), insular cortex, parietal and temporal cortices as well as areas of the striatum (Paulus et al. 2003, Paulus et al. 2005, Clark et al. 2008). We expected our task would elicit similar patterns of activation at its most basic decision level: choosing to "Buy" or "Not Buy" a stock.

We also anticipated several advice related differences between groups. First, if following expert advice is a cognitive shortcut that minimizes costly "slow thinking" by offloading onto an "Expert", individuals who follow "Expert" advice were expected to exhibit minimal brain activation. Previous research has shown that expert advice can significantly alter decision-making both behaviourally and neurobiologically (Engelmann et al. 2009).

We expected to see a decrease in cognitive effort yielding less activation when advice was presented.

Secondly, if obeying an expert counters anxiety and evokes good feelings associated with "trusting an expert", evidence of less anxiety and/or positive emotions might also be detectable in individuals who follow expert advice. Activation in the ventral striatum, orbitofrontal cortex, ventrolateral prefrontal cortex and anterior insula have been associated with processing positive emotions and rewards (Rolls 2000, McClure et al. 2004, Burgdorf and Panksepp 2006) and were expected to support the hypothesis that following expert advice elevates an investor's utility.

Thirdly, we expected that either individuals receiving "Peer" advice should exhibit less advice-related activation compared to those receiving "Expert" advice or individuals receiving "Peer" advice should be less influenced by that advice and exhibit greater brain activation than those receiving "Expert" advice. Advice provided by a "Peer", if less influential, should lead to decreased activation in the advice related areas activated in individuals provided with "Expert" advice. Moreover, "Peer" advice could lead to more activation associated with problem solving in subjects who deem this advice less valuable than advice from an "Expert"

Lastly, we were particularly interested in the activation elicited when the proposed obedience reflex was disengaged, or more simply when individuals chose to disobey the presented advice. Individuals given financial advice by an "Expert" contrary to their financial interest were expected to exhibit greater response conflict, potentially related to disengaging an obedience reflex, and perhaps stronger negative emotions, when they chose to disregard that advice than would individuals given advice by a "Peer". Negative emotions and punishment have been associated with activation in the amygdala, orbitofrontal cortex, ventrolateral prefrontal cortex and anterior insula (Rolls 2000, Brown et al. 2012) and such activation was expected in decisions to disobey the "Expert" advice. We anticipated differences in brain activation related to response conflict in previously identified brain regions of interest. Certain brain regions are activated in studies of decision-making, including the anterior cingulate cortex (ACC), anterior and posterior

lateral prefrontal cortices, medial frontal cortex, insular cortex, intraparietal sulcus, striatum, and thalamus (Engelmann et al. 2009, Engelmann et al. 2012, Meshi et al. 2012). Previous studies using a variety of paradigms with both animal and human subjects support a central role for the prefrontal cortex in decision-making. The ACC is implicated in particularly complex decisions involving ambiguity, conflict and increased potential for errors (Elliott and Dolan 1998, Krawczyk 2002b, Krawczyk 2002a, Hsu et al. 2005, Kuhnen and Knutson 2005). Therefore, these regions might be involved when individuals choose to "obey" or "disobey" an "Expert".

6.3 Statistical Analysis

Behavioural data on the investment task was analyzed using SPSS 21. To test differences in obedience between the two groups Hotelling's T^2 test was performed on the three dependent variables: percent obedience in Runs 1/2, Runs 3/4, and Runs 5/6 (Runs were grouped based on type of advice presented), with Group ("Expert" or "Peer") as our independent variable.

fMRI data were analyzed using the General Linear Model. During model specification, trials were classified by type of advice (No Advice, Good Advice, Bad Advice), type of buy (Good Buy resulting in a win, Bad Buy resulting in a loss), and decision (Buy, Not Buy). Nuisance predictors included run offsets and six motion parameters. We included the trials from all runs in a single General Linear Model, grouping together run 1 with run 2, run 3 with run 4, and run 5 with run 6, as per the type of advice provided. GLM parameters were estimated using linear least-squares error fitting. We computed the following first-level statistical contrasts separately for each participant: Buy – Did Not Buy, Did Not Buy – Buy, Advice – No Advice, No Advice – Advice, Obedient – Not Obedient, Not Obedient – Obedient (Obedient and Not Obedient trials, respectively, were defined as those in which the participant's choice matched / did not match the advice), Good Advice – Bad Advice, and Bad Advice – Good Advice. We performed three second level analyses on the amplitudes of each contrast: within group t-test across all participants in the "Peer" group to detect significant contrast amplitude, within-group t-test across all participants in the

"Expert" group, and between-groups t-test comparison. For all analysis, we used a voxelwise statistical threshold of $t(40) = 2.0211$ ($p < 0.05$ uncorrected) and a cluster size threshold of k = 201 voxels, yielding $p < 0.05$ corrected for multiple comparisons across both the voxel population as well as the statistical tests. Cluster size threshold level was computed using Monte Carlo simulation.

To examine the effect of obedience on brain activity over time we conducted a 2 (Group; "Expert" vs. "Peer" Advice) x 2 (Run; Obedient versus Not Obedient runs 1 and 2 vs. Obedient versus Not Obedient runs 5 and 6) ANOVA. Four participants (three from the "Expert" group and one from the "Peer" group) were excluded from this analysis due to being 100% obedient in the first two runs thus Obedient versus Not Obedient runs 1 and 2 contrasts could not be computed. To elucidate what was driving the interaction effect, four two sample t-tests were conducted and each used as a mask for the interaction contrast.

6.4 Behavioural Results

Post-scan responses were collected to ensure that participants believed that either an outside financial "Expert" or the student running the experiment ("Peer") was giving the advice. No participants indicated suspicion of the indicated advisor. One participant ("Expert" group) indicated only following advice and not attempting to form independent judgments for each stock and was subsequently excluded from all analysis. The remaining 42 participants acknowledged use of the advice as an aid in decision-making at the beginning of the task. However, in both groups all participants indicated using personal strategies as their main decision tool, citing an increased comfort with the task and a lack of trust in the advisor as the task continued. The assumption of equality of covariance matrices was satisfies for our two-group MANOVA (Box's $M = 13.612$, $p = 0.052$). There was a significant difference between the groups (Expert and Peer) on the combined dependent variable (Run), (*Hotellings* $T^2 = 16.64$, $F(3/38) = 5.272$, $p = 0.004$*; Note.* T^2 = Trace coefficient*(sample size-number of groups) = 0.416*(42-2) = 16.64). Post hoc univariate ANOVAs were conducted to determine the effect of group on each of the runs. A significant difference between groups was found in Runs 5/6 ($F(1, 40) = 16.254$, $p < 0.0001$). Both runs 1/2

($F(1,40) = 0.178$, $p = 0.675$) and 3/4 ($F(1,40) = 3.163$, $p = 0.083$) failed to reach significance (Figure 2).

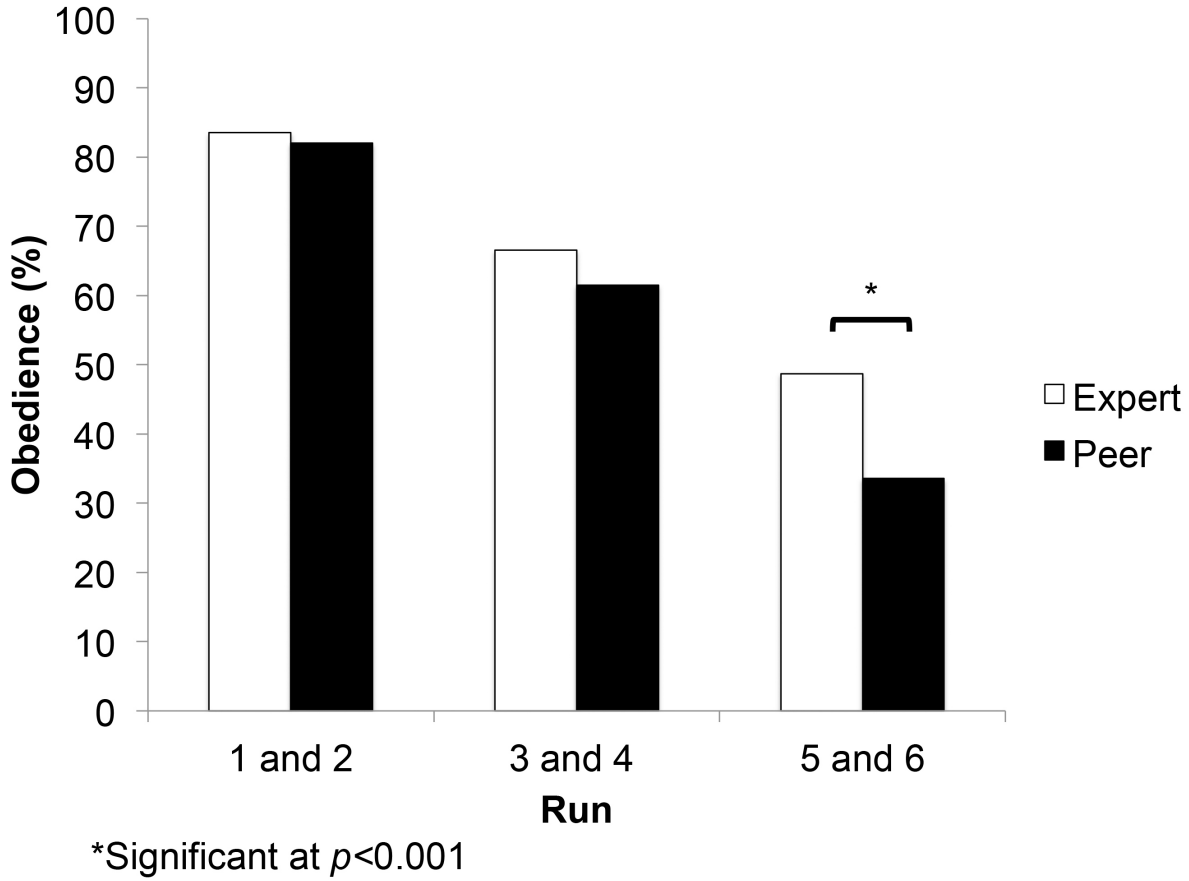

Figure 2. Comparison between groups in obedient decisions

Significant differences in number of obedient decisions in the final two runs of the study ($F(1,40) = 16.254$, $p < 0.0001$). The "Expert" group was significantly more obedient to the advice in the last two runs than the "Peer" group.

6.5 Neuroimaging Results

6.5.1 Obedient versus Not Obedient

Differences emerged between the "Expert" and "Peer" groups when choosing to follow or not follow the advice presented. The "Expert" group displayed significantly greater activation in left anterior cingulate cortex, right superior frontal gyrus, left inferior parietal lobule, left medial frontal gyrus, and left frontal lobe and bilateral temporal lobe white matter (Figure 3) during Not Obedient (or "disobedient") trials (when compared to Obedient trials). On within group tests, when participants in the "Expert" group disobeyed the advice, there was significant activation in bilateral anterior cingulate, right frontopolar cortex, the right pons and left culmen (Figure 4.). In contrast, in the same comparisons the "Peer" group displayed more activation in the right temporal lobe, left insula, right middle occipital gyrus, right hippocampus and left caudate (Figure 5).

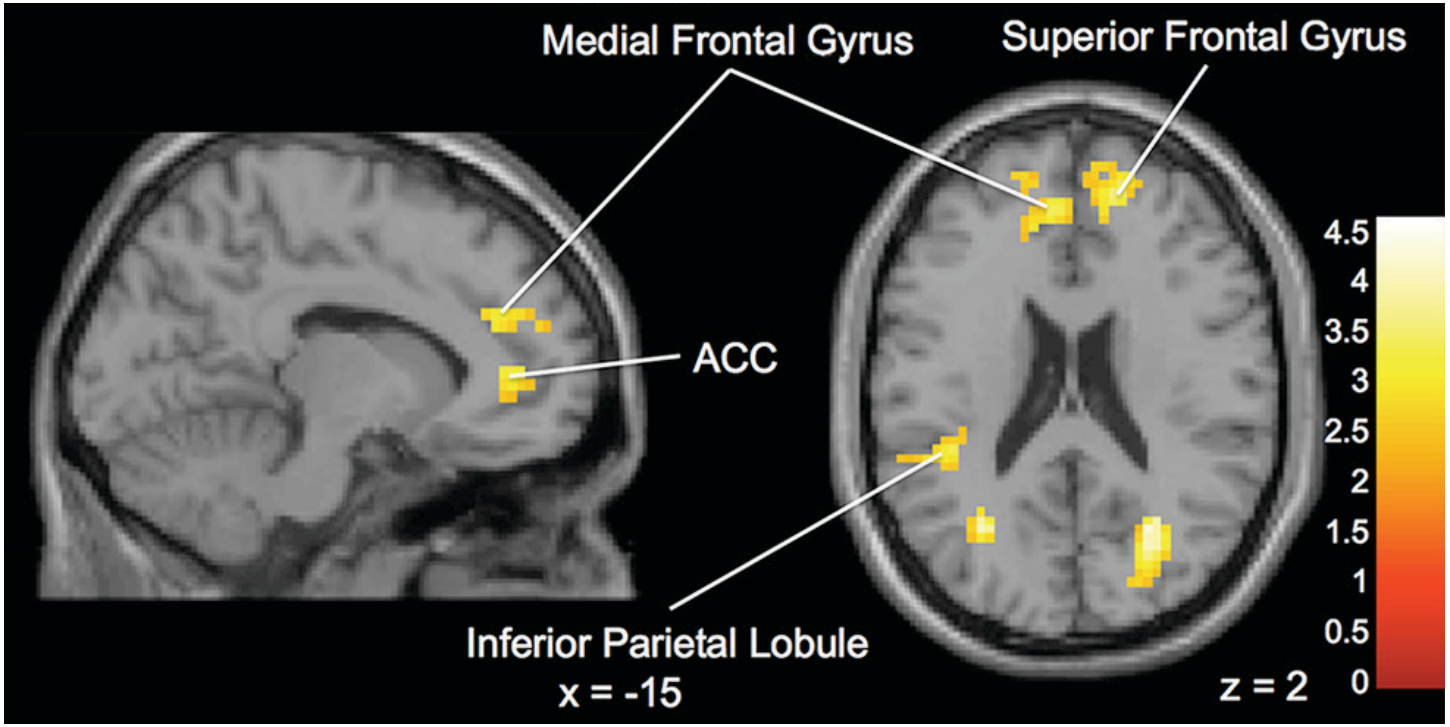

Figure 3. Brain activation for statistical contrast maps *NOT OBEDIENT – OBEDIENT.*

"Expert" group shows increased activation compared to "Peer" group in the ACC, medial frontal gyrus and superior frontal gyrus. "z" and "x" coordinate provided at bottom right corners in MNI space. All results voxelwise statistical threshold at t = 2.0211 (p < 0.05) and a cluster threshold level of k = 201, p < 0.05 corrected for multiple comparisons.

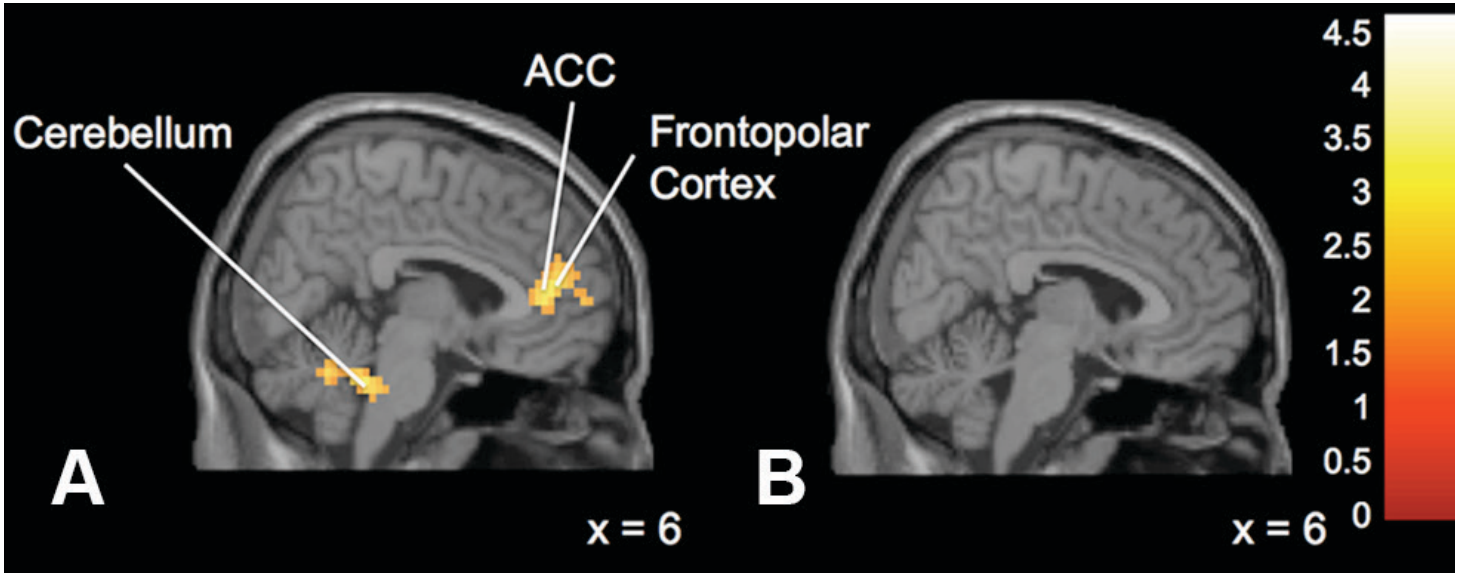

Figure 4. Brain activation for statistical contrast maps *NOT OBEDIENT – OBEDIENT* (within-groups).

a: *"Expert" group. Not Obedient trials elicited activation in the left anterior cingulate cortex, frontopolar cortex and cerebellum in the "Expert" group* ***b:*** *"Peer" group. No significant activation was found in Not Obedient trials in the "Peer" group. "x" coordinate provided at bottom right corners in MNI space. All results voxelwise statistical threshold at $t = 2.500$ ($p < 0.01$) and a cluster threshold level of $p < 0.05$ corrected for multiple comparisons.*

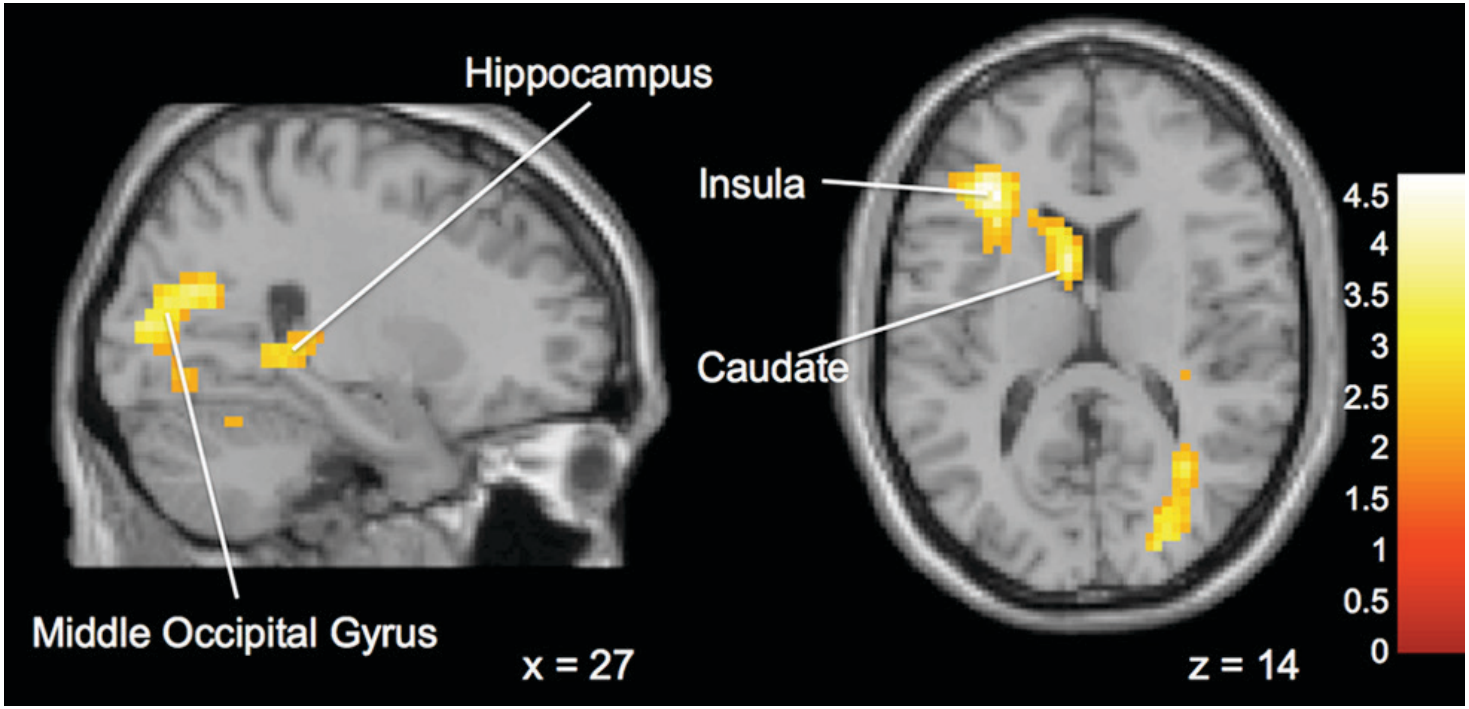

Figure 5. Brain activation for statistical contrast maps *OBEDIENT – NOT OBEDIENT.*

"Peer" group shows increased activation compared to "Expert" group in the middle occipital gyrus, hippocampus, insula and caudate. "z" and "x" coordinate provided at bottom right corners in MNI space. All results voxelwise statistical threshold at t = 2.0211 ($p < 0.05$) and a cluster threshold level of k = 201, $p < 0.05$ corrected for multiple comparisons.

6.5.2 Buy versus Not Buy

No significant differences were found between the “Expert” and “Peer” groups when a decision to “Buy” or “Not Buy” a stock was made. In both the “Expert” and “Peer” groups, there was increased activation in the striatum (specifically the right caudate and left putamen), left pallidum, left middle temporal gyrus, and right cerebellum when participants chose to “Buy” (Figure 6a). In contrast, when participants chose to “Not Buy”, there was significant activation in the right insula, left cerebellum, cingulate gyrus, right middle frontal gyrus and right inferior parietal lobule (Figure 6b).

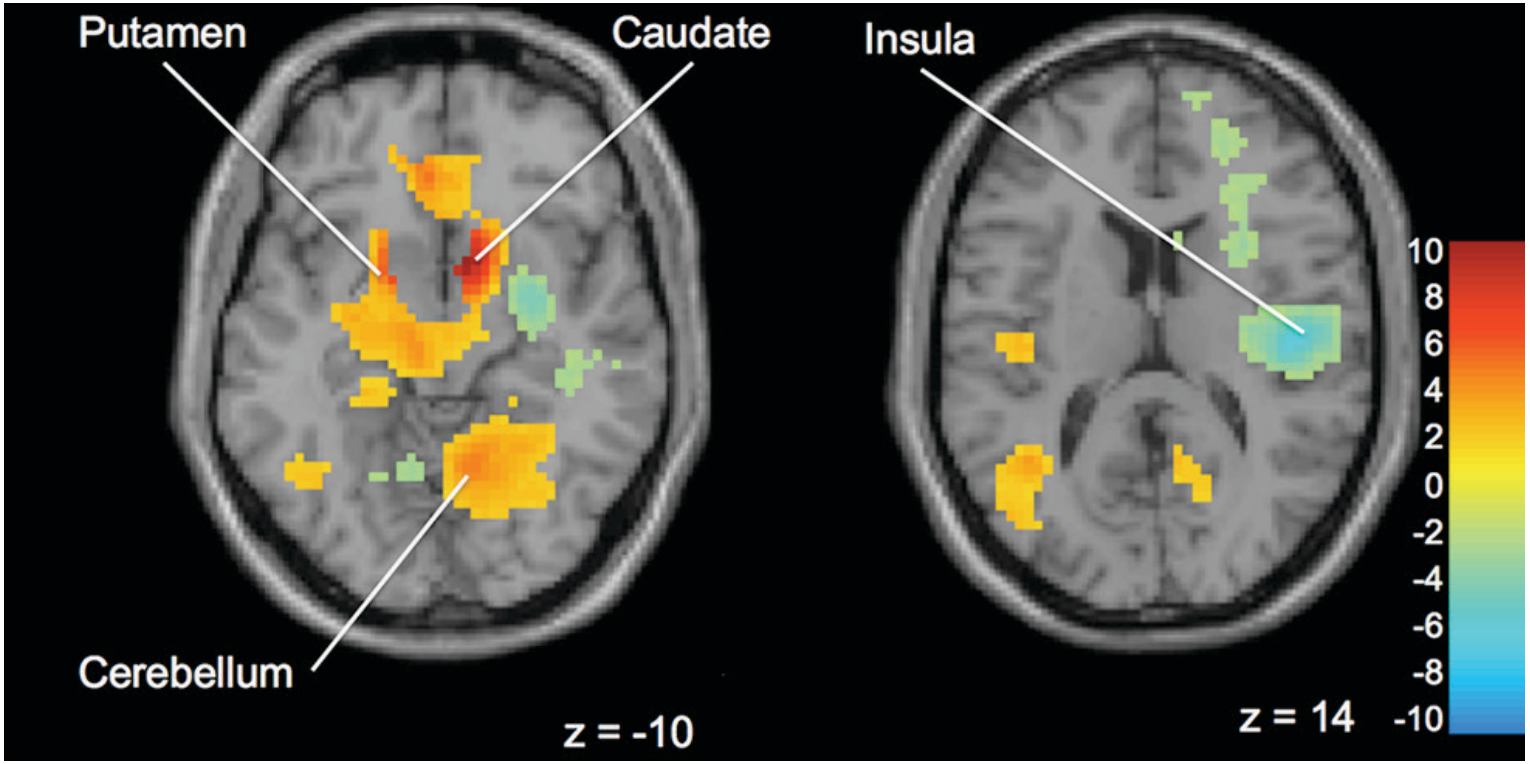

Figure 6. Brain activation for statistical contrast maps *BUY – DID NOT BUY* and *DID NOT BUY – BUY*

a: *Brain activation for statistical contrast maps BUY – DID NOT BUY.* ***Buy*** *trials included a significant cluster of activation in the right caudate and cerebellum in both groups.* ***b:*** *Did Not Buy trials activated the right insula in both groups. "z" coordinate provided at bottom right corners in MNI space. All results voxelwise statistical threshold at t = 2.0211 (p < 0.05) and a cluster threshold level of k = 201, p < 0.05 corrected for multiple comparisons.*

6.5.3 Advice versus No Advice

When comparing Advice to No Advice trials, there was significantly greater activation in the "Peer" group compared to the "Expert" group in the left posterior cingulate cortex, right caudate, left insula, right medial frontal gyrus, left middle frontal gyrus and bilateral frontal lobe white matter (Figure 7a). On within group tests, no significant differences were found in the "Expert" group. However, significant activation emerged in the "Peer" group in the right temporal middle gyrus, left calcarine, left cerebellum, left lingual gyrus, right temporal lobe, left superior temporal gyrus, and left angular gyrus.

In contrast, when comparing activation between groups for No Advice vs. Advice trials, significantly greater activation was found in the "Expert" group than the "Peer" group in the left posterior cingulate, bilateral thalamus, left insula, right caudate, right cingulate gyrus, bilateral medial frontal gyrus, left middle frontal gyrus and left frontal lobe white matter (Figure 7b). On within group tests, there was significant activation in the right caudate, right insula, left putamen, and frontal lobe white matter during No Advice trials as compared to Advice trials in the "Expert" group (Figure 8).

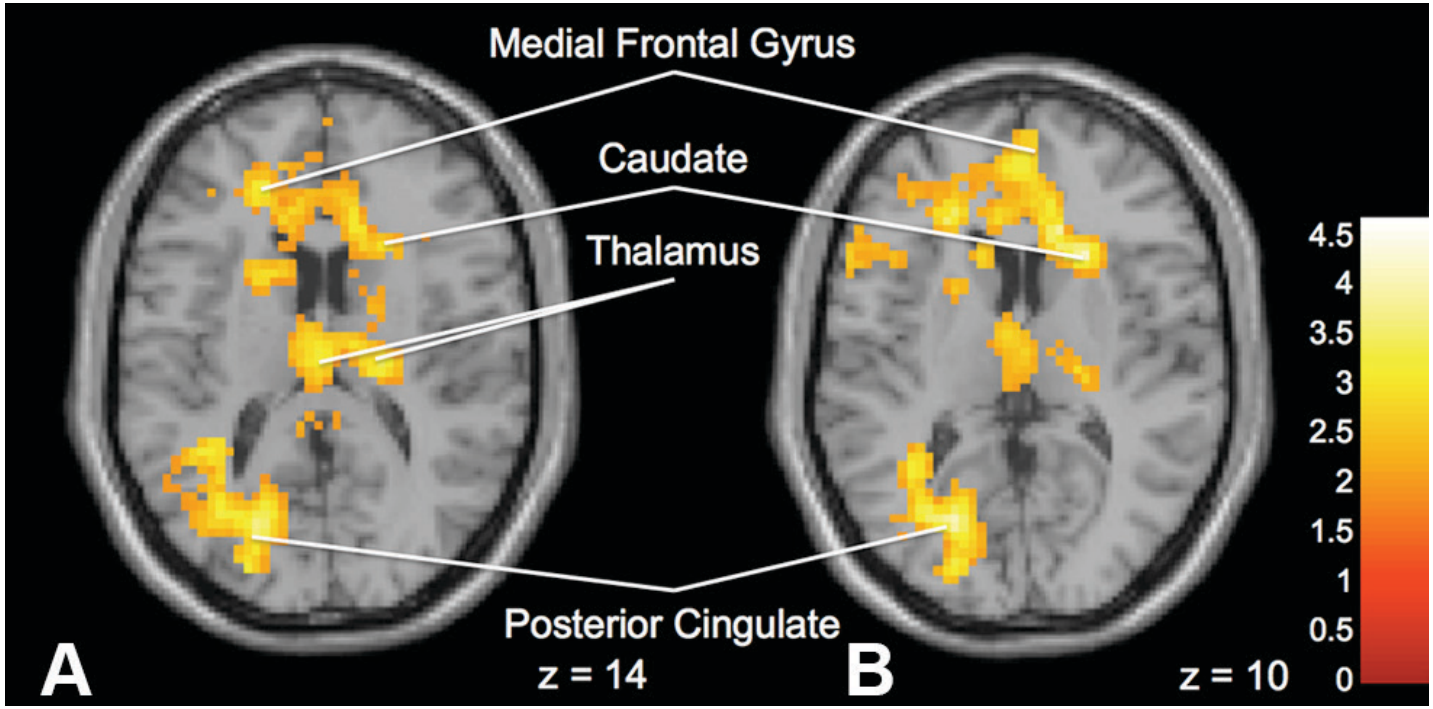

Figure 7. Brain activation for statistical contrast maps *ADVICE – NO ADVICE* and *NO ADVICE - ADVICE*

a. *"Peer" group shows increased activation compared to "Expert" group in the posterior cingulate, medial frontal gyrus and caudate in the ADVICE – NO ADVICE comparison.* ***b.*** *"Expert" group shows increased activation compared to "Peer" group in the posterior cingulate, medial frontal gyrus, caudate and thalamus in the NO ADVICE – ADVICE comparison. "z" coordinate provided at bottom right corners in MNI space. All results voxelwise statistical threshold at $t = 2.0211$ ($p < 0.05$) and a cluster threshold level of $k = 201$, $p < 0.05$ corrected for multiple comparisons.*

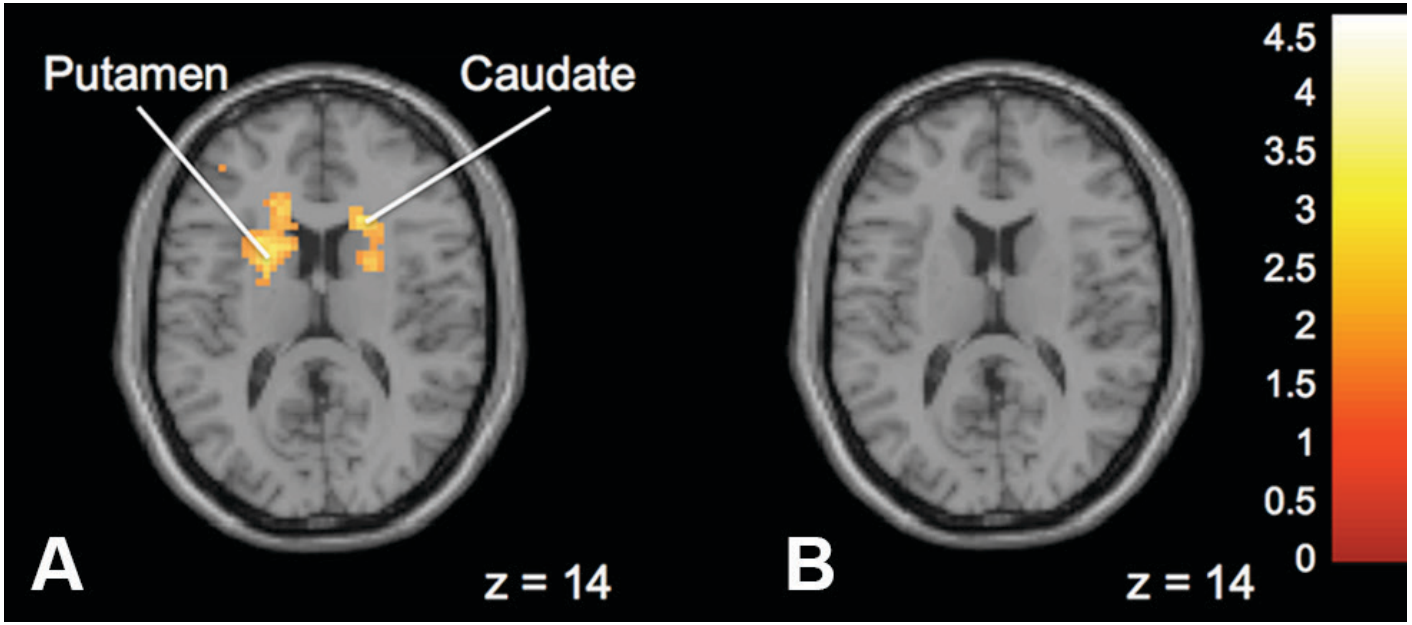

Figure 8. Brain activation for statistical contrast maps *NO ADVICE – ADVICE*

a: *"Expert" group. No Advice trials elicited activation in the right caudate and left putamen for the "Expert" group.* ***b:*** *"Peer" group. No significant activation was found during No Advice trials for the "Peer" group. "z" coordinate provided at bottom right corners in MNI space. All results voxelwise statistical threshold at $t = 2.500$ ($p < 0.01$) and a cluster threshold level of $p < 0.05$ corrected for multiple comparisons.*

6.5.4 Good Advice versus Bad Advice

There were no significant differences between the “Expert” and “Peer” groups when comparing Good Advice with Bad Advice. When combining the groups we found significant differences between Good Advice and Bad Advice, with Good Advice trials eliciting significantly greater activation in the superior temporal gyrus, hippocampus and middle temporal gyrus compared to Bad Advice trials (Figure 9).

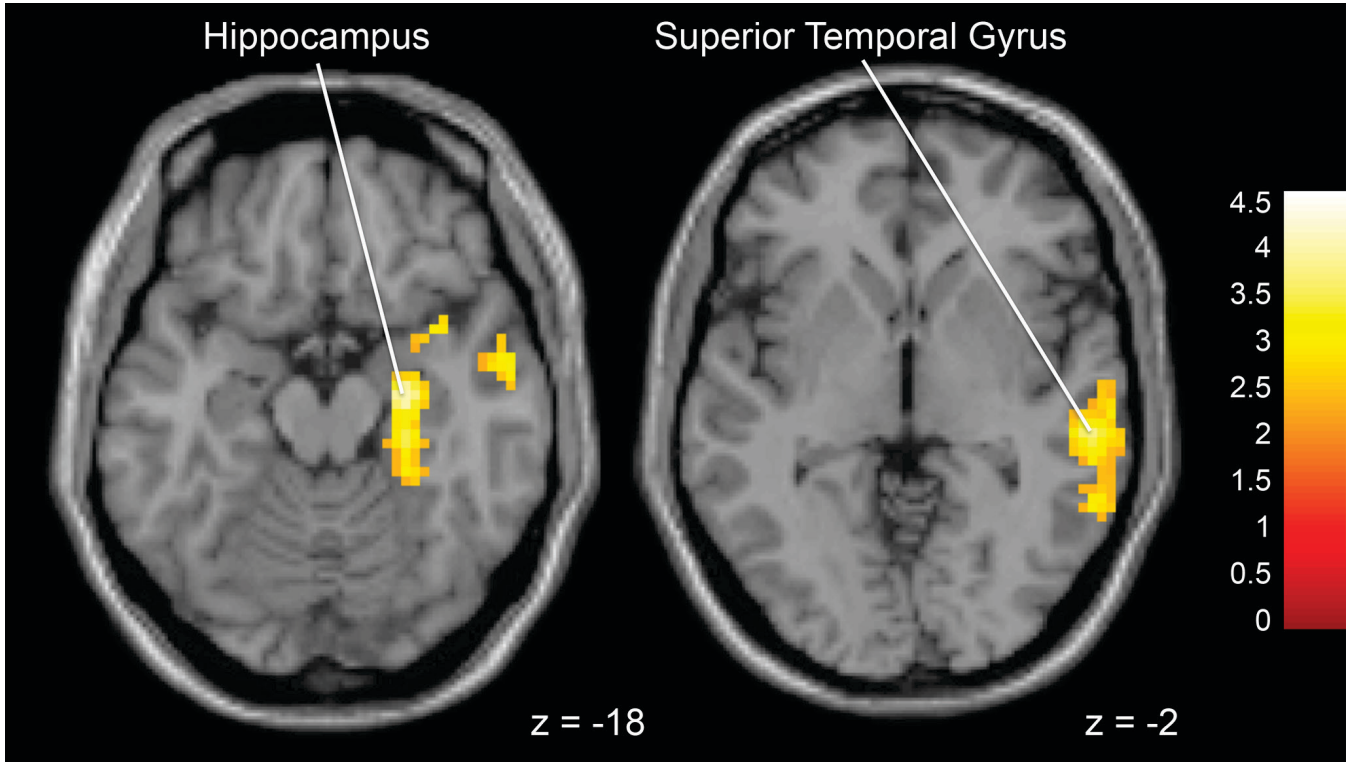

Figure 9. Brain activation for statistical contrast maps *GOOD ADVICE – BAD ADVICE*

Good Advice trials elicited significantly greater activation in the hippocampus and superior temporal gyrus than Bad Advice trials when both the Expert" and "Peer" groups are combined. "z" coordinate provided at bottom right corners in MNI space. All results voxelwise statistical threshold at t = 2.500 (p < 0.01) and a cluster threshold level of p < 0.05 corrected for multiple comparisons.

6.5.5 Obedience over time

While no significant main effects were found for both Group and Run, a significant interaction effect did emerge in the dorsal anterior cingulate gyrus. Further analysis revealed that the "Expert" group showed significant deactivation relative to baseline compared to the "Peer" group in this area during the last two runs. This difference was driven by the "Expert" group demonstrating significant deactivation in this area in the last two runs compared to the first two runs; the "Peer" group did not show significant differences in this region across time (Figure 10).

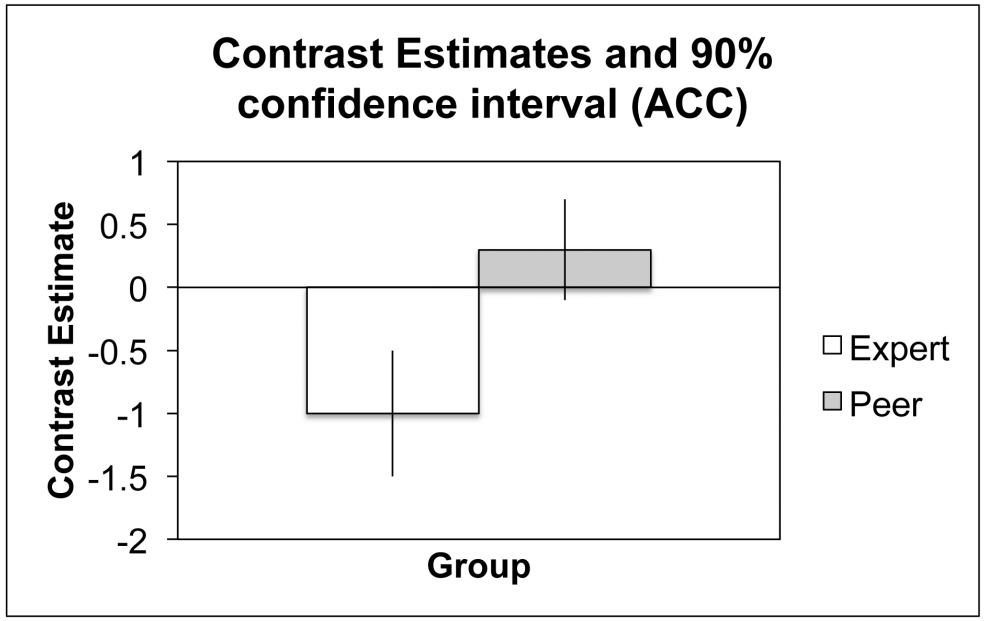

Figure 10. Contrast estimates and 90% confidence interval (ACC)

"Expert" group displayed significantly greater deactivation compared to baseline in the ACC during the last two runs of the task.

6.6.General Discussion

One of the primary goals of this study was to examine the cognitive processes underlying obedience and disobedience to "Experts" in financial decision-making. We found that there was greater activation in the anterior cingulate cortex (ACC) when choosing to disobey an "Expert" rather than a "Peer". These findings for brain changes occurring when a financial "Expert" is disobeyed are consistent with previous research, which has linked changes in the ACC to error detection (Carter et al. 1998, Bush et al. 2000). It has also been suggested that one of the primary functions of the ACC is to monitor conflict (Eriksen and Eriksen 1974, Ullsperger and von Cramon 2001), and then help select an appropriate response (Luu and Pederson 2004). Participants in the "Expert" group are required to consider both the advice of a (supposed) financial "Expert" as well as their own opinions before choosing to buy or not to buy each stock. When choosing to disobey the "Expert", this conflict and integration process may take additional resources compared to choosing to disobey a "Peer". Consistent with this are findings that right superior frontal gyrus activation has been linked to certainty-related processing (Paulus et al. 2004a), and in the present study participants in the "Expert" group may have required more certainty that disobeying the advice was the rational decision to make prior to choosing that action.

Because we employed a large cluster size threshold (k = 201), we applied to our tests an extremely conservative correction for multiple comparisons in all our analyses. This increases the power of our tests, making our findings less susceptible to Type II Errors than are most studies of this sort. Uniquely, the ACC and right superior frontal gyrus activation appears to differentiate between advice from an "Expert" vs. "Peer". This suggests that the influence of advice on financial decision-making has more complex neurobiological links than previously recognized. These results would be consistent with the hypothesis that there exists an obedience reflex to "Expert" advice that can only be disengaged with the occurrence of some conflict processing. These findings would suggest that additional cognitive resources may be required in order to switch off reflexive obedience, and oppose what an authority figure is recommending. However, when it comes to "Peer" advice, no such "reflex" would be present, and thus there would be no conflict processing occuring when the advice is disregarded. This hypothesis is consistent with the findings at each stage

of our study.

In addition to activation in the ACC, there was also activation in the frontopolar cortex at the within groups level in the "Expert" group. In keeping with this finding it has been suggested that this region is important in the integration of multiple cognitive processes when pursuing a single higher behavioural goal (Ramnani and Owen 2004). Furthermore, both the pons and cerebellum are connected via subcortical projections to these prefrontal cortical areas, and have roles in decision-making, learning, working memory as well as the modulation of prefrontal function (Schmahmann and Pandya 2008, Stoodley and Schmahmann 2009, Rosenbloom et al. 2012), which again may explain why these regions are also activated during the investment task. That this activation occurred even though participants in the "Expert" group subjectively cited the same level of use of the advice as the "Peer" group would be consistent with suggestions that the conflict of disobedience and the resolution of this conflict resulting in a disobedient financial decision occur subconsciously.

Previous research has implicated the anterior insula in nonconformity with an expert (Engelmann et al. 2009); however, this activation appears to extend to nonconformity to a peer as no significant differences in activation in this area was found between the two groups. Once again, these results demonstrate that advice modulates activity in the brain in a more complex manner than previously supposed. Based on our results, it appears likely that some, but not all, brain regions affected by advice are differentially affected by whether advice is provided by either an "Expert" or a "Peer". This finding adds to previous research on brain changes occurring during decision-making, and may need to be considered in future studies.

In addition to the primary findings, we also showed support for our first hypothesis in that during the investment task, there was activation when comparing "Buy" vs. "Not Buy" decisions. When participants decided to "Buy", they were taking a risk as the outcome could result in either monetary gain or loss. When participants chose to "Not Buy", they were not risking the loss of any funds, and thus this could also be defined as a 'risk-averse'

trial. Our findings are compatible with the previous literature in that during "Buy" decisions (i.e. risk-seeking choices) there was caudate activation, a region that has previously been linked in other studies to higher risk choices (Kuhnen and Knutson 2005). Conversely, in the "Not Buy", or risk-averse choices, we found insula activation, a finding which has occurred in other studies during risk-avoidance choices (Paulus et al. 2003, Kuhnen and Knutson 2005). The insula is believed to be involved in interoceptive awareness (Critchley et al. 2004). Thus, activation in the insula may indicate the possibility of an aversive outcome, such as punishment, and may lead a participant not to choose the more risky option (Paulus et al. 2003, Critchley et al. 2004), in this case a "Buy" decision in our task. In addition, when comparing "Buy" to "Not Buy" we found that there was increased activation in the pallidum, a region which has previously been shown to precede advantageous actions (Paulus et al. 2004b). Thus, our findings regarding individual decisions for "Buy" and "Not Buy" are compatible with the previous literature, validating our task and lending more support to previous findings.

When comparing "No Advice" to "Advice" trials, we found significantly greater activation in many areas in the "Expert" group compared the "Peer" group. These include frontal lobe, thalamus and left posterior cingulate. We suggest that it is possible in the "Expert" group that little cognitive effort was required when advice was presented, because of an obedience reflex to "Expert" advice, regardless of whether or not the advice was good or bad. In contrast, there was additional cognitive effort expended when no advice was given. Since the data suggests that the "Peer" group generally discounted the advice, they would therefore be expending relatively similar cognitive effort on both the "No Advice" and "Advice" trials, and therefore would not demonstrate any changes between these two activities.

The posterior cingulate has been implicated in the retrieval of episodic memory (Andreasen et al. 1995), semantic information (Hargreaves et al. 2012) as well as self-reflective thought (Johnson et al. 2002) indicating that part of this increased cognitive effort stems from switching from a reliance on the advice to relying on previous trials or previous experience to guide decision-making. Our findings are also compatible with previous research in which

a lack of advice increased activation in the posterior cingulate cortex, inferior frontal gyrus and middle temporal gyrus (Engelmann et al. 2009) and advice modulates activity in the ventromedial pre-frontal cortex (Engelmann et al. 2012). Consistent with previous research on decision-making in risk related tasks, the thalamus has also been implicated (Ernst et al. 2002). In the "Peer" group we found support for our hypothesis that this group would discount the advice, and thus they exerted greater cognitive effort (and more activation in these areas) in "Advice" trials than did the "Expert" group. In this scenario, it is suggested that the cost of gathering and evaluating the information provided on each stock is not seen to outweigh the risk of taking a cognitive shortcut and following the advice of a "Peer", and this results in increased cognitive effort reflected in the brain changes detected by fMRI.

This hypothesis regarding our neuroimaging result was reflected in the behaviour of our participants. Advice from the "Peer" was discounted, as was demonstrated by the significant decrease in obedient decisions compared to the "Expert" group (Figure 2). This follows previous fMRI research that participants value expert advice more than novice advice (Meshi et al. 2012). Participants in the "Peer" group also did not engage in their error detection and conflict monitoring mechanisms when disregarding the advice. In fact, when participants in the "Peer" group followed the advice presented there was greater activation in the hippocampus, insula and caudate. This may indicate that rather than 'following' the advice presented, these participants were making a decision that happened to agree with the advice based on risk assessment and previous trials. Furthermore, no significant activation was found during "No Advice" trials at the within groups level, indicating that the absence of advice may not have produced a similar increase in cognitive effort to that was found in the "Expert" group. Rather, when the experimenter's advice was presented, regions involved in semantic processing (Hargreaves et al. 2012) and adjustments made to optimize performance (Kim et al. 2011) were activated, suggesting more cognitive effort despite the presentation of advice. **What is particularly interesting in the present study is that even though the "Expert" was not present, or ever seen, the importance given to advice from this source had a meaningful impact on brain activation and behaviour.** This finding would support suggestions that even remote authority figures can have profound unconscious effects on financial (and perhaps other)

behaviour, and may in part explain how financial decisions can be significantly influenced by the current "understanding" or "knowledge" as interpreted by intermediaries (Scharfstein and Stein 1990, Banerjee 1992, Trueman 1994, Loh and Stulz 2011).

That there were no significant differences between the "Expert" and "Peer" groups when comparing "Good Advice" and "Bad Advice" indicates that the two groups did not differ in how they differentiated between the good and bad advice. "Good Advice" trials elicited significant activation in the superior temporal gyrus, hippocampus and middle temporal gyrus in both groups. This could indicate that participants were able to differentiate between good and bad advice and were learning and engaging in the "Good Advice" trials compared to "Bad Advice" trials. Nonetheless, it is important to note that all Good Advice trials occurred at the onset of the task and all Bad Advice trials occurred at the end of the task, and it is therefore conceivable that participants were generally more actively concerned about understanding the advice at the onset of the task compared to the end, and that this is reflected in the pattern of brain activation seen.

The interaction between obedience over time and advice lends more support for the role of the anterior cingulate cortex in decision-making as well as obedience. Specifically, the dorsal region of the anterior cingulate gyrus is associated with rational thought process and reward-based decision-making (Bush et al. 2002). At the end of the task, the "Expert" group showed a significant decrease in activation in this region of the ACC compared to when the task first began when obeying the presented advice; however, the "Peer" group did not show this change in activation across time. Decreased activation in the ACC has been found in depressed patients in decision-making/reward anticipation and is believed to reflect a lack of awareness or concern for outcomes (Forbes et al. 2006). Based on these findings coupled with the "Expert" groups greater behavioural obedience compared to the "Peer" group in the final runs, we hypothesize that over time, advice from a seemingly trusted source, such as an expert, may lead to a similar reduced awareness in decision-making as seen in depressed patients. When a peer, with no social context of being particularly trustworthy, provides the advice that advice does not elicit the same pattern of activation, allowing the individual to make more rational decisions over time. In the case of

our task, more rational decision-making led to less obedient decisions later in the task, as demonstrated by our "Peer" group.

Our hypotheses regarding utility of trust and the positive or negative emotions associated with obeying or disobeying the advice were not supported. It is conceivable that our task did not elicit strong enough emotional reactions from participants to reveal significant activation at our high threshold. Previous studies utilizing neuroimaging have shown activation in the ventral striatum (Potenza 2013). It is possible that this did not occur in our study because our task was not positively or negatively rewarding enough (i.e. there was no risk that a participant could lose all their money for participation).

It is important to recognize that this research may illuminate only one aspect of financial decision-making. Personality factors can also be important, for example, one study suggested that those students who have a higher risk taking and more positive attitude to gambling may be more likely to pursue careers in the financial industry (Sjoberg and Engelberg 2009). These factors were explored in our second study.

Chapter 7. Study 2: Psychological Differences between Gamblers and Non-Gamblers

7.1 Recruitment and Participants

Participants were recruited from the University of Alberta campus and surrounding area via online advertising. Following completion of informed consent, all participants were screened for the presence of psychiatric disorders and ongoing alcohol or drug abuse using standardized questionnaires. Any individuals who had ongoing alcohol or drug abuse were excluded from further participation. Participants then completed the Problem Gambling Severity Screen (PGSI) (Ferris and Wynne 2001) and a psychological battery consisting of the Gambling Attitudes and Beliefs Scale (GABS) (Breen and Zuckerman 1994)), two measures of risk tolerance (Grable and Lytton 1999, Weber et al. 2002), the Barratt Impulsivity Scale (BIS-11) (Patton et al. 1995) and the Rosenberg Self-Esteem Scale (Rosenberg 1965); and the Adverse Childhood Experiences Scale (Felitti et al., 1998). Those individuals who scored higher on the PGSI (see previous Gambling section in Chapter 5 for details) were characterized as gamblers, while those who scored lower formed the healthy control group.

A total of 57 individuals entered the study (mean age 25.4 ± 5.32 years, range: 20-48 years) of which 70.2% were male. Based on the scores on the PGSI, there were 41 individuals in the Control group and 16 individuals in the 'Gambler" group. The "Gamblers" exhibited a variety of preferred forms of gambling, including attending casinos regularly, playing internet poker frequently, and betting on sport outcomes, while some reported regularly engaging in a mixture of gambling forms.

7.2 Hypotheses

We carried out a study comparing gamblers with healthy controls in order to examine any psychological differences between the two populations. Based upon the literature we had several hypotheses we wished to examine:

1) Gamblers would endorse more positive gambling related attitudes, and that there would be a significant correlation between the two gambling measures.
2) Gamblers would report higher levels of risk tolerance. We hypothesized that this increased risk tolerance would encompass not only financial risk, but other domains of risk as well.
3) In gamblers who did not have substance abuse issues there would be no association with impulsiveness.
4) Gamblers would report lower levels of self-esteem, potentially linked to a greater report of traumatic experiences as a child. We also expected risk tolerance to be positively correlated with adverse childhood experiences.

7.3 Statistical Analysis

One-sample Kolmogorov-Smirnov tests were run on all scales to test for normality. Based on these results logarithmic transformations were conducted for financial risk tolerance subscales and total score, as well for the DOSPERT (willingness to participate) subscales. One-sample Kolmogorov-Smirnov tests were rerun on the transformed data to ensure normality. Independent samples t-tests were run on the demographics, analysis of covariance (ANCOVA) tests were conducted for all the scales (Table 4), correlation analysis were conducted on the independent variables (Table 5), and multiple linear regression was conducted to determine which measures predicted gambling severity as measured by the PGSI (Table 6) and GABS (Table 7). The assumptions of the multiple linear regression model were checked and verified.

7.4 Results

7.4.1 Between-groups comparisons

In terms of differences between the participants in the two groups, there were no statistically significant differences between controls and "Gamblers" in terms of age ($t(55) = -1.953$, $p = .0.056$), gender ($t(55) = 1.135$, $p = 0.261$), or ethnicity ($t(55) = 1.691$, $p = 0.097$). However, there were statistically significant demographic differences between the

two groups in terms of level of education ($t(55) = 2.951$, $p = 0.005$). Two-way ANOVA analyses were computed to determine if level of education had any interaction effects with the psychological scales. There were main effects for the BIS-11 Nonplanning Impulsiveness subscale ($F(2,55) = 3.438$, $p = 0.039$), PGSI ($F(2,55) = 6.850$, $p = 0.002$), and the DOSPERT Ethical subscale ($F(2,55) = 4.720$, $p = 0.013$). Level of education was negatively correlated with all three measures ($r = -0.287$, $p = 0.031$; $r = -0.427$, $p = 0.001$; and $r = -0.372$, $p = 0.004$, respectively).

As would be anticipated, there were significant differences between the two groups in the GABS, with "Gamblers" scoring higher than controls. Controlling for level of education, significant differences were found between groups on the PGSI (Table 4). There was also a highly significant ($p < 0.01$) correlation between our two gambling measures (Table 5).

"Gamblers" and controls differed significantly on overall financial risk tolerance with "Gamblers" scoring higher than controls (Table 4). When examining the three subscales separately, significant differences were seen only in the Risk Comfort and Experience scale score. DOSPERT scores were significantly different in two domains: Financial and Health Safety. In both domains, "Gamblers" scored higher than controls. Interestingly, across all domains, there were no significant differences in perception of risk (Table 4).

Significant differences between the groups emerged in the BIS-11 Motor Impulsiveness subscale, with "Gamblers" scoring higher than controls (Table 4). No significant differences emerged on the BIS-11 subscales of Attentional Impulsiveness, Non Planning Impulsiveness or the total scale. A correlation analysis determined that impulsivity was partially linked to the gambling measures: total BIS-11 score, as well as subscales Motor Impulsiveness and Non Planning Impulsiveness, were positively correlated with both the GABS and PGSI (Table 5).

No significant differences were found between groups in adverse childhood experiences or in self-esteem, after controlling for education (Table 4). Utilizing a correlational analysis, a negative association was found between the two scales (Table 5): Financial risk tolerance as measured by Grable and Lyons' (1999) scale was correlated with adverse childhood experiences (Table 5).

Table 4.

Analysis of covariance results

	Controls		Gamblers			
Scale	**M**	**SD**	**M**	**SD**	**F (1,54)**	**p-value**
Financial Tolerance (FT) Total	3.264†	0.147	3.407	0.208	5.879	0.019*
FT Investment Risk	2.249†	0.244	2.369	0.292	0.901	0.347
FT Risk Comfort and Experience	2.360†	0.149	2.532	0.203	12.464	0.001***
FT Speculative Risk	1.738†	0.312	1.868	0.380	0.851	0.360
DOSPERT (w) Ethical	2.485†	0.288	2.743	0.545	1.966	0.167
DOSPERT (w) Financial	2.845†	0.325	3.257	0.433	10.315	0.002**
DOSPERT (w) Health Safety	2.914†	0.369	3.243	0.348	5.778	0.020*
DOSPERT (w) Recreational	3.252†	0.397	3.319	0.479	0.433	0.513
DOSPERT (w) Social	3.434†	0.138	3.447	0.196	0.113	0.738
DOSPERT (p) Ethical	26.439	6.108	27.188	6.316	0.253	0.617
DOSPERT (p) Financial	27.732	6.169	25.250	7.289	0.886	0.351
DOSPERT (p) Health Safety	27.342	6.744	26.625	7.329	0.131	0.719
DOSPERT (p) Recreational	22.098	6.196	21.563	6.088	0.263	0.610
DOSPERT (p) Social	15.415	4.336	16.500	6.088	0.454	0.503
BIS-11 Total	61.317	8.214	65.438	7.633	1.578	0.215
BIS-11 Attentional Impulsiveness	17.585	3.633	16.125	3.575	1.16	0.286
BIS-11 Motor Impulsiveness	21.439	3.800	25.375	3.181	9.565	0.003**
BIS-11Nonplanning Impulsiveness	22.293	3.989	23.938	3.021	0.568	0.454
GABS	73.146	9.671	90.688	12.965	23.693	<0.0001***
PGSI	0.244	0.538	7.438	4.163	95.035	<0.0001***
ACE	1.415	1.612	2.000	1.966	0.655	0.422
Rosenberg Self-Esteem	21.732	4.775	21.688	3.825	0.528	0.470

*Note***:** DOSPERT (w): Domain-Specific Risk-Taking Scale – Willingness to participate; DOSPERT (p): Domain-Specific Risk-Taking Scale – Perception of risk; BIS-11: Barratt Impulsivity Scale; GABS: Gambling Attitudes and Belief Scale; PGSI: Problem Gambling Severity Index; ACE: Adverse Childhood Experience.

* significant at $p < 0.05$, ** significant at $p < 0.01$, *** significant at $p < 0.001$

†value after logarithmic transformation

Table 5.

Correlation Matrix for Barratt Impulsivity Scale, Rosenberg Self-esteem Scale, Adverse Childhood Experiences and Gambling Measures

	BIS Attentional	BIS Motor	BIS Nonplanning	BIS Total	GABS	PGSI	Rosenberg Self-esteem	ACE
BIS Attentional	1							
BIS Motor	.087	1						
BIS Nonplanning	.096	.598**	1					
BIS Total	.532**	.805**	.798**	1				
GABS	.028	.470**	.361**	.409**	1			
PGSI	-.117	.454**	.317*	.317*	.713**	1		
Rosenberg Self-esteem	-.231	-.085	-.239	-.255	-.023	-.127	1	
ACE	.211	.428**	.208	.400**	.186	.204	-.332*	1

*significant at $p < 0.05$, **significant at $p < 0.01$

7.4.2 Prediction power of measurements

To determine to what extent the psychological measures collected related to gambling severity, two regressions were performed as measured by both gambling measures: the PGSI and the GABS. When both the PGSI and GABS were regressed against Grable and Lytton's (1999) Financial Risk Tolerance Scale, significant relationships did emerge. However, these relationships became insignificant in the presence of the other independent variables. A significant relationship emerged when regressing total BIS score, BIS Nonplanning subscale and BIS Motor Impulsiveness subscale individually with both the PGSI and GABS; however, these relationships also became insignificant in the presence of the other independent variables. Significant predictors of PGSI included GABS, Age, and the DOSPERT Health and Safety subscale. These predictors explained 61% of the variability in PGSI scores (Table 6). Significant predictors of GABS included PGSI, DOSPERT subscales Financial, Recreational, Social and perception of Recreational risk. These predictors explained 68% of the variability in GABS scores (Table 7).

Table 6.
Multiple Regression Results for Problem Severity Gambling Index (Stepwise)

	Model 1[a]			
	β	SE(β)	t	p
Constant	-14.284	2.227	-6.415	<0.0001
GABS	0.212	0.028	7.535	<0.0001
	Model 2[b]			
	β	SE(β)	t	p
Constant	-18.373	2.385	-7.702	<0.0001
GABS	0.193	0.026	7.318	<0.0001
AGE	0.217	0.065	3.335	0.002
	Model 3[c]			
	β	SE(β)	t	p
Constant	-23.043	3.124	-7.377	<0.0001
GABS	0.181	0.026	6.948	<0.0001
AGE	0.21	0.063	3.337	0.002
DOSPERT (w) Health Safety[†]	1.927	0.87	2.214	0.031

[a.] Adjusted R2 = 0.499
[b.] Adjusted R2 = 0.577
[c.] Adjusted R2 = 0.605
[†] logarithmic transformation

Table 7.

Multiple Regression Results for Gambling Attitudes and Belief Scale (Stepwise)

	Model 1[a]			
	β	SE(β)	t	p
Constant	72.646	1.434	50.655	< 0.0001
PGSI	2.397	0.318	7.535	< 0.0001
	Model 2[b]			
	β	SE(β)	t	p
Constant	39.606	9.153	4.327	< 0.0001
PGSI	1.856	0.324	5.735	< 0.0001
DOSPERT (w) Financial[†]	11.574	3.174	3.647	0.001
	Model 3[c]			
	β	SE(β)	t	p
Constant	23.047	10.79	2.136	0.037
PGSI	1.664	0.316	5.261	< 0.0001
DOSPERT (w) Financial[†]	13.773	3.134	4.394	< 0.0001
DOSPERT (p) Recreational	0.478	0.184	2.596	0.012
	Model 4[d]			
	β	SE(β)	t	p
Constant	0.729	14.932	0.049	0.961
PGSI	1.566	0.310	5.046	< 0.0001
DOSPERT (w) Financial[†]	13.567	3.04	4.463	< 0.0001
DOSPERT (p) Recreational	0.655	0.197	3.317	0.002
DOSPERT (w) Recreational[†]	5.88	2.811	2.095	0.041
	Model 5[e]			
	β	SE(β)	t	p
Constant	49.439	26.204	1.887	0.065
PGSI	1.564	0.299	5.229	< 0.0001
DOSPERT (w) Financial[†]	14.414	2.956	4.877	< 0.0001
DOSPERT (p) Recreational	0.588	0.193	3.049	0.004
DOSPERT (w) Recreational[†]	7.598	2.817	2.698	0.009
DOSPERT (w) Social[†]	-16.095	7.235	-2.225	0.031

[a.] Adjusted R^2 =0.499

[b.] Adjusted R^2 = 0.591

[c.] Adjusted R^2 = 0.630

[d.] Adjusted R^2 = 0.652

[e.] Adjusted R^2 = 0.677

[†] logarithmic transformation

7.5 General Discussion

The purpose of this study was to examine possible psychological differences between gamblers and controls and possible links between these measures.

Our first hypothesis was that gamblers would endorse more positive gambling-related attitudes and that there would be a significant correlation between the two gambling measures. Our results supported this, and we found that those with the highest gambling scores (referred to as "Gamblers") reported endorsing more positive attitudes and irrational beliefs towards gambling than did controls. Since our "Gamblers" were defined by our screening to possess moderate levels of problems due to gambling, it follows that this group should report higher agreement with pro-gambling statements than controls. Our use of the PGSI as a tool to assign participants was supported by the positive correlation between the two measures of gambling (PGSI and GABS), which was also in line with our expectations.

Our second hypothesis was that gamblers would report higher levels of risk tolerance. Our findings also supported this, since they indicate that there are significant differences between "Gamblers" and controls in terms of risk tolerance, both financially and in other non-financial domains. Specifically, "Gamblers" scored higher on financial risk tolerance, which appears to be driven by their risk comfort and experience. In general, gamblers are more likely to be in a situation where financial risk is a factor, and thus it follows that they should feel more comfortable with experiencing such risk. This supports previous research in which a physiological marker of tolerance (heart rate) was linked to gambling behaviour (Griffiths 1993). In the earlier study, regular gamblers were found to have an immediate and significant decrease in heart rate after gambling than non-regular gamblers. This immediate decrease in heart rate shows how gamblers have physiologically adapted to the experience of financial risk compared to non-gamblers. Coupled with our results, we conclude that it is the experience and increased comfort with financial risk that leads, in part, to the adaptation and increased risk tolerance seen in gamblers.

Differences also emerged in a financially-distinct domain, namely risk tolerance in terms of Health and Safety. In this area "Gamblers" reported greater willingness to participate in activities that could be seen as having a greater risk to one's personal health and safety.

These results support our hypothesis that greater risk tolerance is not unique to the financial domain. This is consistent with one view in psychology, in which individuals were believed to demonstrate consistent risk taking and attitudes across domains (Eysenck and Eysenck 1977, Lejuez et al. 2002a), although others have suggested that the stability of such risk-taking may be less clear (Hanoch et al. 2006).

Interestingly, in our study there were no significant differences in perception of risk between the groups. That is, "Gamblers" and controls rated the activities/behaviours similarly in terms of perception of risk. "Gamblers", who reported more willingness to engage in higher risk behaviours, did not view these behaviours as less risky than did the controls. In fact, *despite* the high risk present, these individuals would still be more likely to participate in the behaviours. These results expand on gambler's tendencies towards higher risk and demonstrate that these occur not only in financial scenarios. Gamblers do perceive higher risk situations similar to non-gamblers; however, recognizing this risk does not produce the same deterring effect that is achieved in non-gamblers. Rather, in addition to the anticipated monetary gains, gamblers may wish to seek 'action' in the form of increased excitement or an 'adrenaline rush' when they choose to take financial risk (Lesieur and Rosenthal 1991). This 'action' (also colloquially called an "adrenaline rush") has been suggested as being similar to the euphoric state that drug addicts seek, and supports previous researchers who have likened gambling to substance based addictions (Levinson, Gernstein, & Maloff, 1983; Moran, 1970, Petry et al., 2013).

Our third hypothesis was that, in gamblers who did not have substance abuse issues, there would be no association with impulsiveness. However, the findings were contrary to our hypothesis, and significant differences emerged for impulsivity between the groups. Differences were found for the motor impulsiveness subscale, with "Gamblers" reporting greater impulsiveness than controls. This subscale refers to items such as "I do things without thinking" and "I act on the spur of the moment". Thus. "Gamblers" reported greater impulsivity regarding actions. It has been suggested that there are two facets of impulsivity: reward-driven (a goal-focused approach behaviour) and rash (an individual's inability to halt approach behaviour) impulsivity (Dawe and Loxton 2004). In the case of gambling, rash impulsivity refers to gambling in spite of knowing the potential punishments. Previous

research has implicated impulsivity with gambling in both men and women (Loxton et al. 2008), and our results support this finding as rash impulsivity shares similarities with the motor impulsiveness subscale. This rash impulsivity and increased motor impulsiveness may also explain why gamblers' similar perception to non-gamblers of high-risk situations is not a deterrent from engaging in risky behaviours.

It should be noted that overall impulsivity was not significantly different between the groups, although with a larger sample it is conceivable that this result may become significant. As well, we found that all but one subscale (Attentional Impulsiveness) was correlated with both the GABS and PGSI. This is consistent with previous research, which has found correlational links between other measures of impulsivity (specifically Eysenck's Impulsivity scale) and gambling measures (PGSI and Gambling Involvement; (Mishra et al. 2010) Thus, our results lend support to the argument that impulsivity, or certain aspects of impulsivity at the very least, is related to gambling behaviour.

Our fourth hypothesis was that gamblers would report lower levels of self-esteem, potentially linked to a greater report of traumatic experiences as a child. However, this hypothesis was not supported, and we found no statistically significant differences between gamblers and controls for self-esteem or for adverse childhood experiences. Some previous studies have had similar findings, with some finding no relationship between self-esteem and gambling (Volberg et al. 1997) or between gambling, self-esteem, and addictions overall (Greenberg et al. 1999). Our results may indicate that rather than gambling acting as a coping mechanism for low self-esteem, it is rather greater willingness to engage in risky behaviours despite the perceived risk that differentiates gamblers to controls. Nonetheless, our results do point to a decrease in self-esteem when there is the presence of childhood traumatic events, which is supported by previous studies that have linked the two (Browne and Finkelhor 1986, Low et al. 2000). Based on these results, it appears that neither self-esteem nor traumatic events early in life are differential factors between gamblers and non-gamblers. While the ACE study (Felitti et al., 1998) has provided evidence that childhood trauma can have long lasting detrimental health outcomes and has been linked to drug abuse, no evidence has yet been found linking such trauma to the development of gambling disorder. Clearly, a larger study would be required to more definitively determine these

findings, and such a study should also compare those with more severe gambling problems, as well as the group that we studied.

In terms of the relative importance of each component, GABS score, age, and the Health and Safety DOSPERT subscale significantly explained 60.5% of the variance in PGSI scores. Interestingly, both financial risk tolerance measures did not significantly contribute to the model in the presence of the other independent variables indicating that, at least in these groups, significant differences in financial risk tolerance do not in fact have any bearing on frequency of gambling behaviour. However, based on the moderate explanatory power of the model, it is likely that the current measures do not encompass the full picture of problem gambling. The PGSI, DOSPERT Financial, Recreational, Social, and perception of Recreational risk subscales significantly explained 67.7% of the variability in GABS score. While this model fairs only slightly better than the previous model, it does lend some support to our group comparisons with "Gamblers" endorsing greater risk tolerance in more than just the financial domain. It should be noted that when the PGSI and GABS was regressed against only Grable and Lytton's (1999) Financial Risk Tolerance Scale, a significant relationship did emerge. Thus, there is a significant relationship between risk tolerance and both gambling severity and gambling attitudes; however, other measures produced better explanatory relationships. In keeping with some previous findings (Mishra et al. 2010), neither gambling measure was significantly predicted by the impulsivity measure in the presence of all independent variables, providing support for the hypothesis that impulsivity may not be the best predictor for gambling nor hold the strongest relationship to gambling. While our sample of gamblers and controls differed on their scores of one subscale of impulsivity (Motor Impulsiveness), this difference is only a predicting factor on reported gambling behaviour or attitudes and beliefs when no other variables were included. However, overall impulsivity and the Nonplanning scale individually were also significant predictors of both gambling measures and so impulsivity cannot be completely discounted when discussing gambling disorder. Self-esteem and adverse childhood experiences also failed to be significant in both models.

Post hoc analyses were conducted to determine if age or gender had any significant relationship to our risk tolerance or impulsivity variables. Interestingly, no significant

relationships emerged for impulsivity, suggesting that impulsivity remains stable throughout life and between genders. A maturation (age) relationship emerged with both Grable and Lytton's (1999) Financial Risk Tolerance Scale (with age explaining 9.3% or the variance in financial risk tolerance) and the DOSPERT Social subscale (with age explaining 5.9% of the variance in the DOSPERT Social subscale) while a gender effect emerged for DOSPERT Ethical perception of risk subscale (with gender explaining 10.5% of the variance in the DOSPERT Ethical perception of risk subscale). While these predictors all reached significance, they remain relatively weak predictors of the risk tolerance scales thus we conclude that these relationships are not of significant interest or importance.

Chapter 8. Study 3: Neurobiological Comparison of Gamblers and Non-Gamblers

8.1 Recruitment and Participants

Participants were recruited from the University of Alberta campus and surrounding area via online advertising. The study was approved by the University of Alberta Health Research Ethics Board. All potential participants received details of the study, and signed an informed consent form. Individuals were screened for the presence of any psychiatric disorder (such as depression), using a semi-structured interview (Appendix A), as well as for any risk associated with having an MRI scan (metal in the body, claustrophobia etc.; Appendix B and C). Any individuals who had ongoing alcohol or drug abuse were excluded from further participation, as were any participants unable to have an MRI scan. Participants then completed the psychological battery previously discussed. Those individuals who scored higher on the PGSI (see previous Gambling section in Chapter 5 for details) were characterized as gamblers, while those who scored lower formed the healthy control group.

A total of 39 individuals entered the study (mean age 26.13 ± 6.23 years, range: 28 years) of which 74.4% were male. Based on the scores on the PGSI, there were 23 individuals in the Control group and 16 individuals in the 'Gambler" group.

8.2 Hypotheses

In the current study, we wished to examine if the differences consistently found in pathological gamblers also appear with subclinical gamblers or if they have their own unique neurobiological effects. Based on the previous literature we had several hypotheses:

1) We predicted that our subclinical "Gamblers" would differ from "Controls" on the overall task and overall feedback phases of the task. Regions anticipated to show differences include the vmPFC and reward pathway regions such as the striatum.

2) "Gamblers" would perform poorly on the task compared to 'Controls" as indicated by lower financial outcome in the task, mimicking the dysfunctional decision-making patterns seen in previous research and gambling tasks with pathological gamblers. Additionally, we expected "Gamblers" not to be as obedient/affected by the advice presented during the task compared to "Controls".
3) Since we do not expect "Gamblers" to follow the advice provided by the supposed expert, we hypothesize that there will be no differences in brain activation when comparing Advice trials to No Advice trials within the "Gamblers" but that differences will emerge when comparing "Gamblers" to "Controls". Regions of hypothesized differences are the ACC and prefrontal cortex.

8.3 Statistical Analysis

Behavioural data on the investment task was analyzed using SPSS 21. An ANOVA and independent samples two-tailed *t*-test were performed to determine differences between the groups in terms of age and gender. To test differences in obedience between the groups Hotellings T^2 test was performed on the three dependent variables: percent obedience in Runs 1/2, Runs 3/4, and Runs 5/6 (Runs were grouped based on type of advice presented), with Group ("Gambler" or "Control") as our independent variable.

fMRI data were analyzed using the General Linear Model. Trials were classified by type of advice (No Advice, Good Advice, Bad Advice), type of buy (Good Buy resulting in a win, Bad Buy resulting in a loss), decision (Buy, Did Not Buy), and feedback (Win, Lose) during model specification. Nuisance predictors included run offsets and six motion parameters. We included the trials from all runs in a single GLM, grouping together run 1 with run 2, run 3 with run 4, and run 5 with run 6, as per the type of advice (Good, Bad or both) provided. GLM parameters were estimated using linear least-squares error fitting. We computed the following first-level statistical contrasts separately for each participant: Buy – Did Not Buy, Did Not Buy – Buy, Advice – No Advice, No Advice – Advice, Obedient – Not Obedient, Not Obedient – Obedient. (Obedient and Not Obedient trials, respectively, were defined as those in which the participant's choice matched / did not match the advice),

Win – Lose and Lose – Win. We performed three second level analyses on the amplitudes of each contrast: within group t-test across all participants in the "Control" group to detect significant contrast amplitude, within-group t-test across all participants in the "Gamblers" group, and between-groups t-test comparison. For all analysis, we used a voxelwise statistical threshold of $t(37) = 2.0262$ ($p < 0.05$ uncorrected) and a cluster size threshold of k = 201 voxels, yielding $p < 0.05$ corrected for multiple comparisons across both the voxel population as well as the statistical tests. Cluster size threshold level was computed using Monte Carlo simulation.

Post hoc analysis of reaction time data was conducted using SPSS 21.

8.4 Behavioural Results

There were no significant differences in age or gender between the two groups; however, gamblers scored significantly higher on the PGSI ($t(37) = -8.160$, $p < .0001$).

The assumption of equality of covariance matrices was satisfied for our two-group MANOVA (Box's $M = 6.44$, $p = 0.44$). There was a significant difference between the groups (Gamblers and Non Gamblers) on the combined dependent variable (Run), (*Hotellings* $T^2 = 11.25$, $F(3/35) = 3.549$, $p = 0.024$*; Note.* T^2 = Trace coefficient*(sample size-number of groups) = 0.304*(39-2) = 11.25). Post hoc univariate ANOVAs were conducted to determine the effect of group on each of the Runs (Figure 11). A significant difference between the groups only appeared in Runs 5/6 ($F(1,37) = 5.416$, $p = 0.026$). Both runs 1/2 ($F(1,37) = 0.63$, $p = 0.43$) and 3/4 ($F(1,37) = 0.144$, $p = .71$) failed to reach significance.

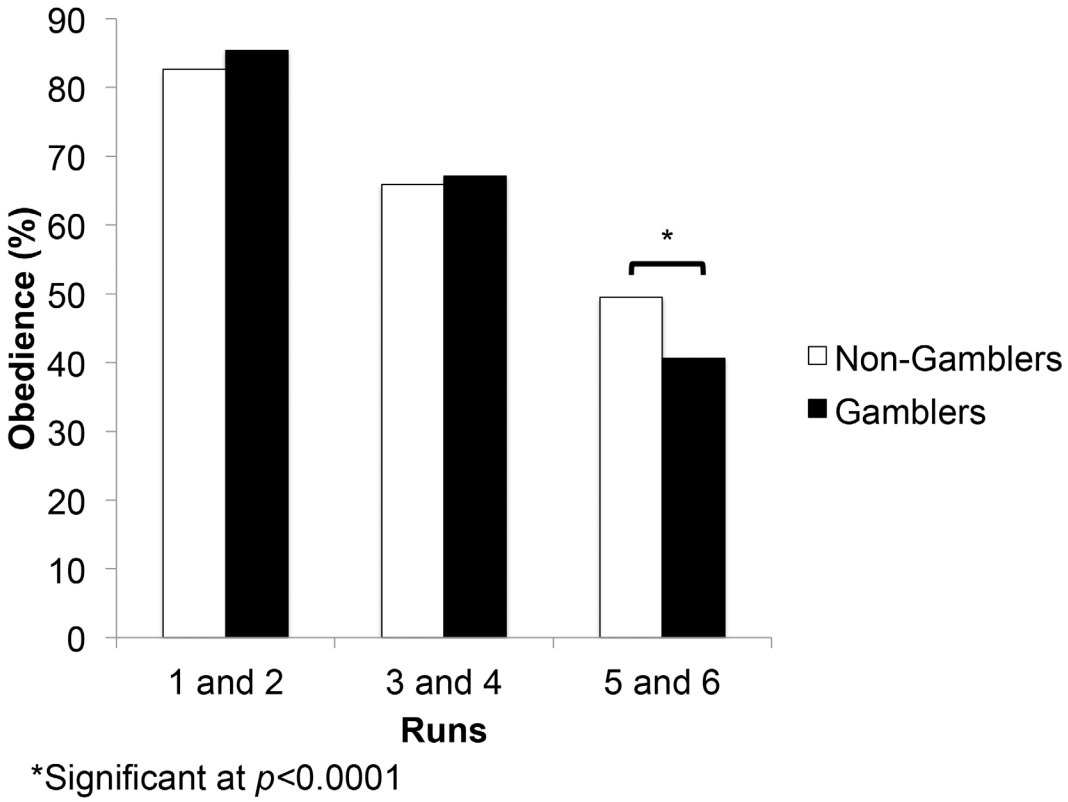

Figure 11. Comparison between Non-Gamblers and Gamblers in obedient decisions

Significant differences in number of obedient decisions in the final two runs of the study ($F(1,40) = 16.254$, $p < 0.0001$). The "Expert" group was significantly more obedient to the advice in the last two runs than the "Peer" group.

8.4.1 Task Performance

Total monetary score at the end of the task determined task performance with higher performance indicated by a higher total score. There were no significant differences between the groups in task performance however this effect did approach significance favoring Gamblers ($t(37) = -1.872$, $p = 0.069$, Cohen's $d = -0.625$).

8.4.2 Reaction Times

Reaction time analyses revealed no significant differences in overall reaction times throughout the task between groups.

8.4.2.1 2 (Group; Non-Gambler, Gambler) x 2 (Obedience; Obedient, Not Obedient) ANOVA

A main effect for Obedience emerged ($F(1,72) = 8.124$, $p = 0.006$) with Not Obedient ($M = 3.574$ seconds, $SD = 0.757$ seconds) decisions taking longer than Obedient decisions ($M = 3.135$ seconds, $SD = 0.596$ seconds). There was no main effect of group or interaction effect.

8.4.2.2 2 (Group; Non-Gambler, Gambler) x 2 (Good Advice Obedience; Good Advice Obedient, Good Advice Not Obedient) ANOVA

A main effect emerged for Good Advice Obedience ($F(1,72) = 14.776$, $p < 0.0001$) with Not Obedient ($M = 3.717$, $SD = 0.828$) decisions being slower than Obedient ($M = 3.073$, $SD = 0.605$) decisions when the advice presented was good.

8.4.2.3 2 (Group; Non-Gambler, Gambler) x 2 (Bad Advice Obedience; Bad Advice Obedient, Bad Advice Not Obedient) ANOVA

No significant effects were found.

8.4.2.4 2 (Group; Non-Gambler, Gambler) x 2 (Advice; Good Advice, Bad Advice) ANOVA

A main effect approached significance for group ($F(1,72) = 3.178$, $p = 0.079$) with Gamblers ($M = 3.509$ seconds, $SD = 0.117$ seconds) being slower than Non-Gamblers ($M = 3.236$ seconds, $SD = 0.099$ seconds). There was no main effect of Advice or interaction effect.

8.5 Neuroimaging Results

8.5.1 Overall Task

There were no group differences when comparing overall task activation during all presentation/decision phases of the task. Differences in activation did emerge when comparing the two groups during the feedback phase of the task. “Gamblers” displayed greater activation in bilateral insula, thalamus and dmPFC (Figure 12).

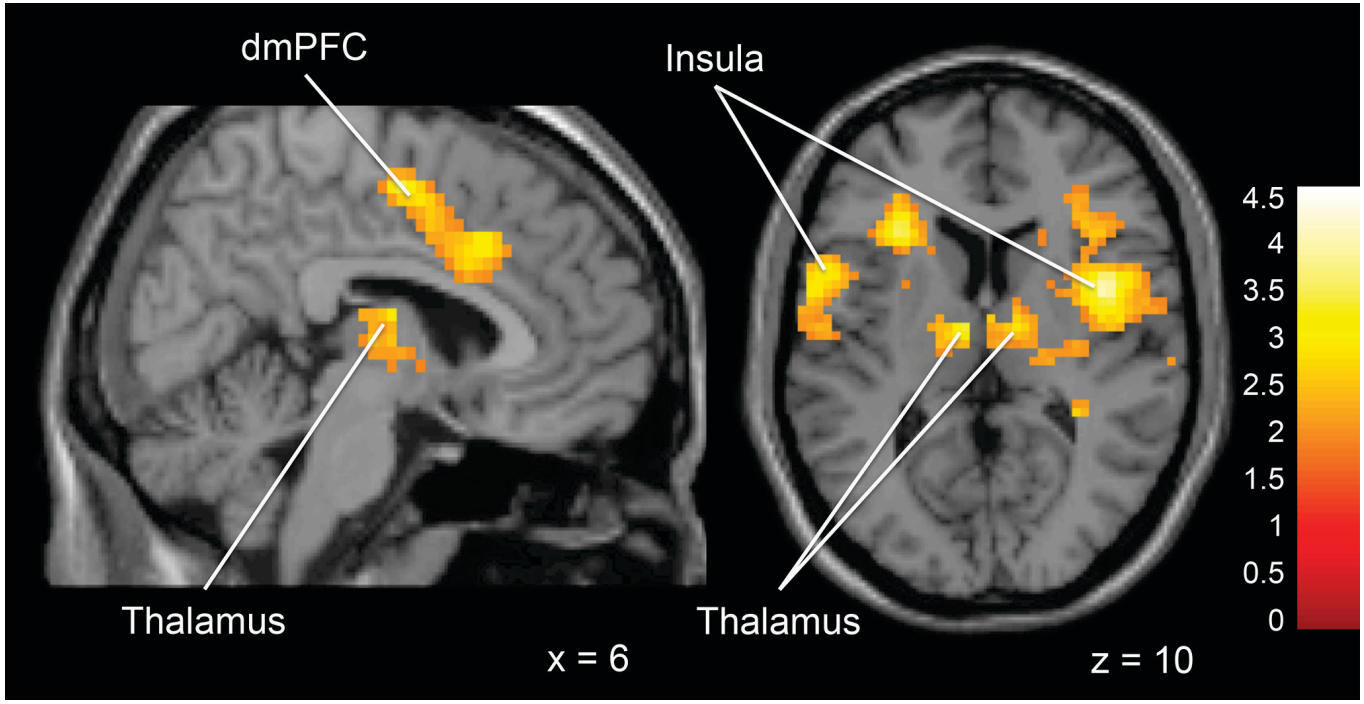

Figure 12. Brain activation for statistical contrast maps Overall Task Feedback Phase

"Gamblers" show increased activation in bilateral insula, thalamus and dorsal-medial prefrontal cortex. "x" and "z" coordinate provided at bottom right corners in MNI space. All results voxelwise statistical threshold at t = 2.0211 (p <0.05) and a cluster threshold level of k = 201, p < 0.05 corrected for multiple comparisons.

8.5.2 Obedient vs. Not Obedient

Significant differences emerged when comparing Obedient to Not Obedient trials in the middle runs (mixed good and bad advice). There was significant activation compared to baseline for Obedient compared to Not Obedient trials in “Gamblers” in the left inferior parietal lobule, insula, medial frontal gyrus and the ventral anterior cingulate cortex compared to “Non-Gamblers” (Figure 13). There was significant activation compared to baseline for Not Obedient compared to Obedient trials in “Non-gamblers” in both the dorsal and ventral anterior cingulate cortex compared to “Gamblers”. No significant differences between the groups emerged when comparing the first two runs and the final two runs.

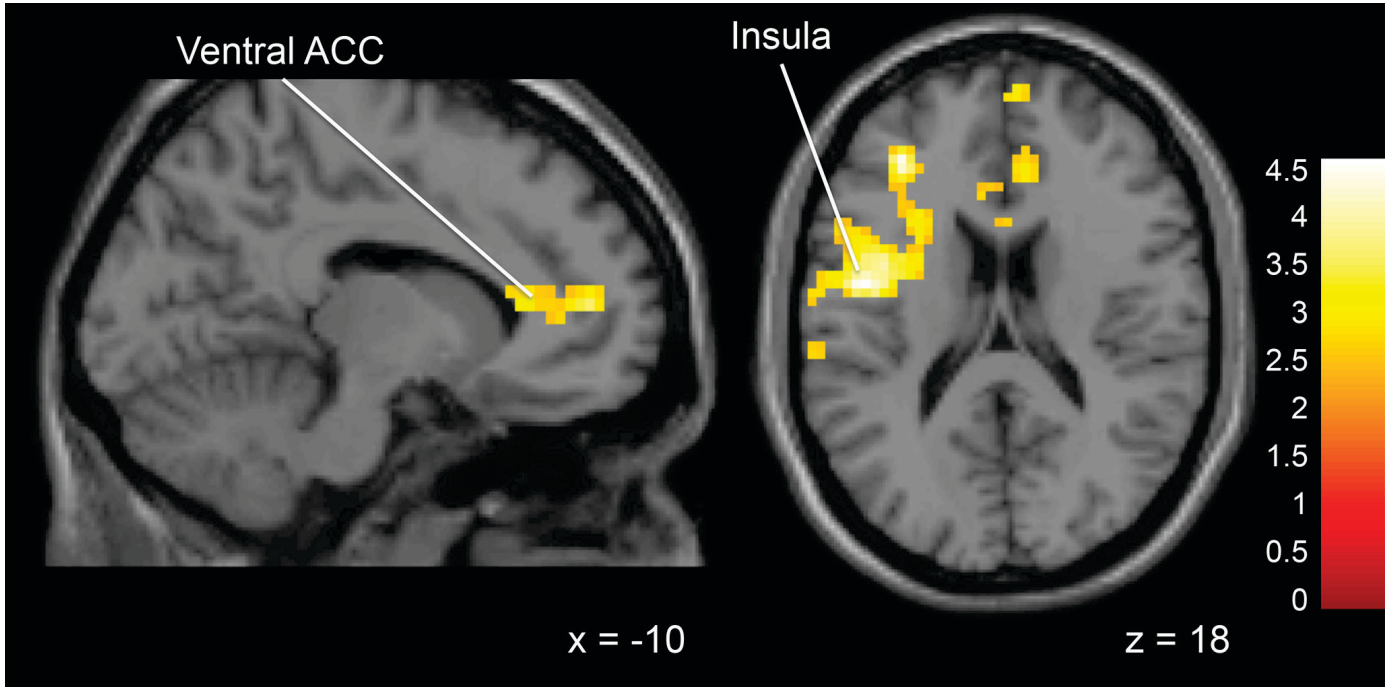

Figure 13. Brain activation for statistical contrast maps OBEDIENT – NOT OBEDIENT in Sub-threshold Gamblers

"Gamblers" display greater activation in the ventral ACC and insula. "x" and "z" coordinate provided at bottom right corners in MNI space. All results voxelwise statistical threshold at t = 2.0211 (p <0.05) and a cluster threshold level of k = 201, p < 0.05 corrected for multiple comparisons.

8.5.3 Advice vs. No Advice

Differences emerged between “Gamblers” and “Non-gamblers” when comparing Advice to No Advice trials with “Gamblers” displaying significant activation in the superior frontal gyrus and the anterior cingulate gyrus compared to baseline than “non-Gamblers” during Advice trials (Figure 14). At the within-groups level, differences between Advice and No Advice trials did emerge in “Gamblers” with Advice trials recruiting the occipital lobe and No Advice trials recruiting the left putamen and left precentral gyrus (Figure 15).

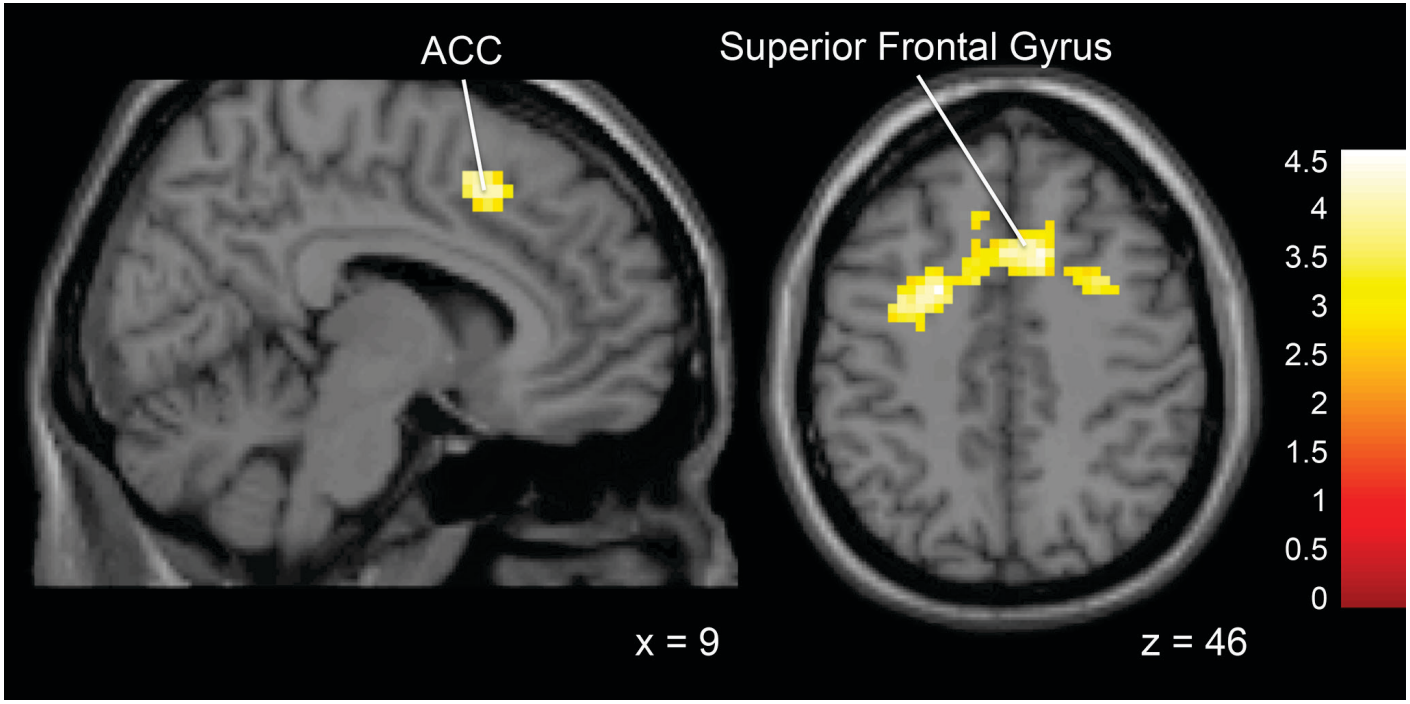

Figure 14. Brain activation for statistical contrast map ADVICE – NO ADVICE in Gamblers

"Gamblers" show greater activation in the superior frontal gyrus and anterior cingulate gyrus compared to non-Gamblers. "x" and "z" coordinate provided at bottom right corners in MNI space. All results voxelwise statistical threshold at t = 2.0211 (p <0.05) and a cluster threshold level of k = 201, p < 0.05 corrected for multiple comparisons.

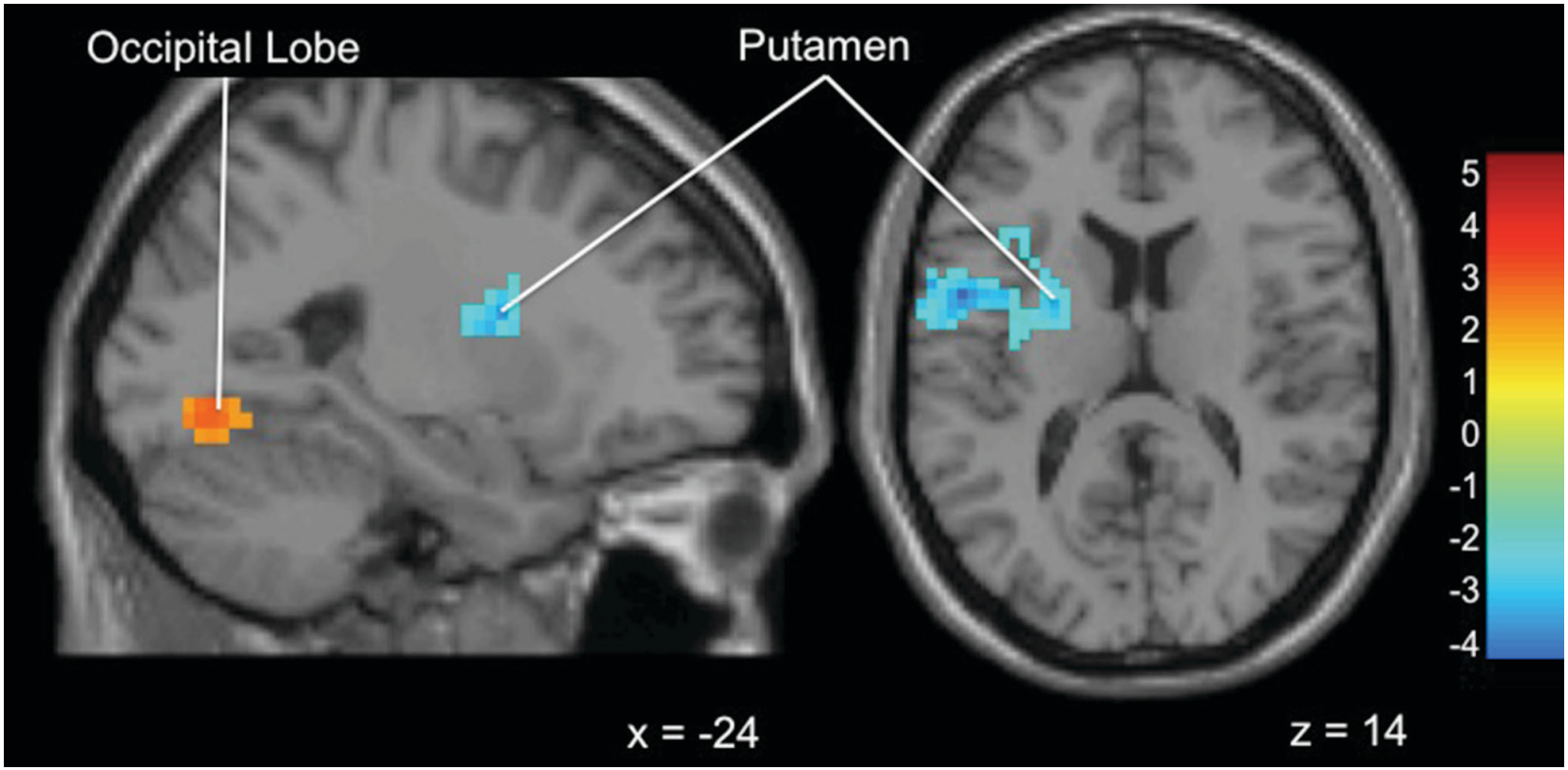

Figure 15. Brain activation for statistical contrast map ADVICE – No ADVICE (Within groups sub-threshold gamblers)

At the within groups level, "Gamblers" show activation in the occipital lobe during Advice trials and greater activation in the putamen in No Advice trials. "x" and "z" coordinate provided at bottom right corners in MNI space. All results voxelwise statistical threshold at t = 2.0211 (p <0.05) and a cluster threshold level of k = 201, p < 0.05 corrected for multiple comparisons.

8.5.4. Good Advice vs. Bad Advice

No significant differences emerged between groups when comparing Good Advice trials to Bad Advice trials. No significant difference emerged between Good and Bad Advice trials when combining the groups together.

8.5.5 Buy vs. Did Not Buy

Differences emerged when comparing Buy to Did Not Buy trials. “Gamblers” displayed significantly greater activation in the bilateral precuneus while “Non-gamblers” displayed significant deactivation in the posterior cingulate cortex and right inferior parietal lobule in Buy compared to Did Not Buy trials. “Non-gamblers” showed significantly greater activation compared to baseline in the posterior cingulate gyrus in Did Not Buy compared to Buy trials (Figure 16).

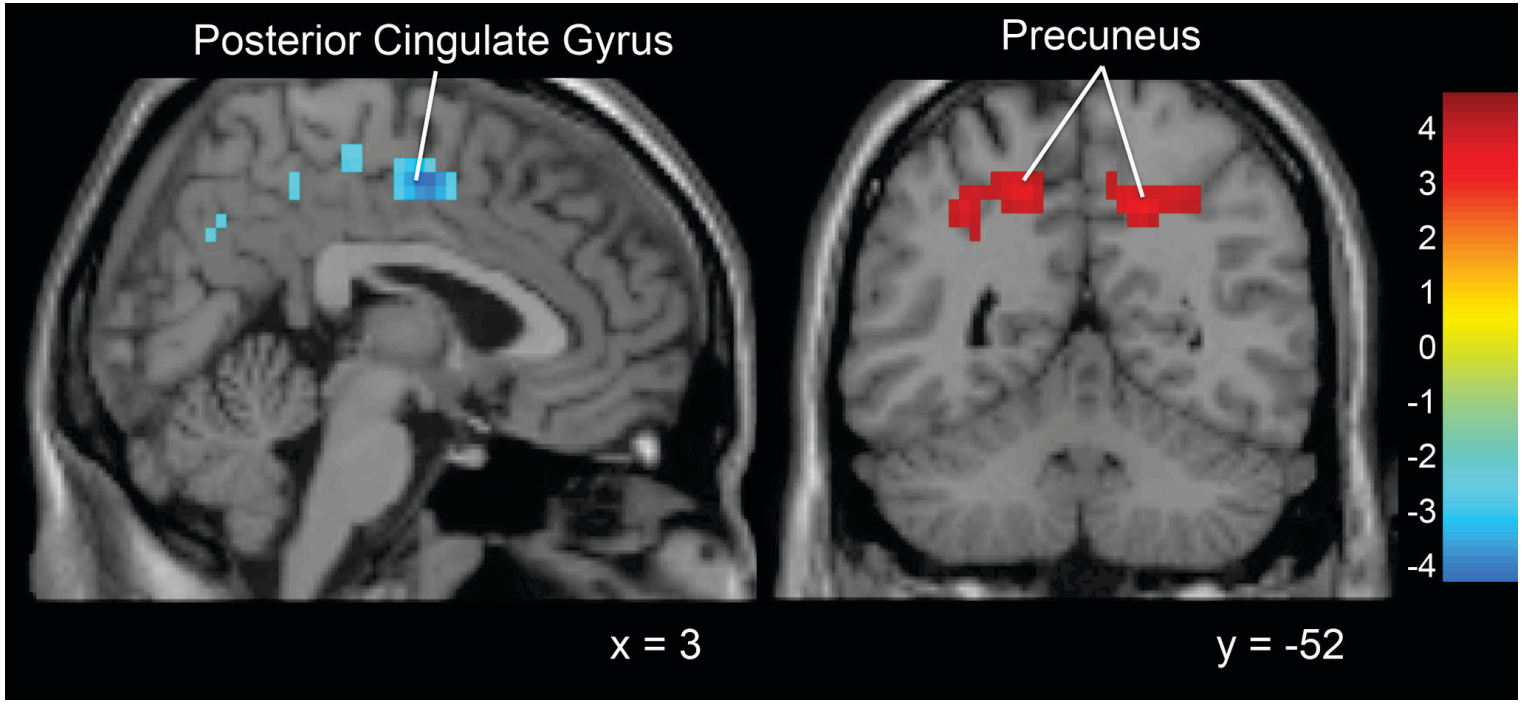

Figure 16. Brain activation for statistical contrast map for BUY – DID NOT BUY

"Non-gamblers" show significant deactivation in the posterior cingulate during Did Not Buy trials while "Gamblers" showed greater activation in bilateral precuneus. "x" and "y" coordinate provided at bottom right corners in MNI space. All results voxelwise statistical threshold at t = 2.0211 (p <0.05) and a cluster threshold level of k = 201, p < 0.05 corrected for multiple comparisons.

8.5.6 Win vs. Lose

Significant differences emerged when comparing Win feedback and Lose feedback. “Non-gamblers” showed significant activation while “Gamblers” showed significant deactivation compared to baseline in the right inferior frontal gyrus, bilateral medial frontal gyrus and right insula when receiving Win feedback (Figure 17). There was significant deactivation compared to baseline in the right thalamus and right superior frontal gyrus in “Gamblers” (Figure 18) while “Non-gamblers” displayed significant activation in the left insula and right dorsal medial prefrontal cortex compared to baseline when receiving Win feedback (Figure 19).

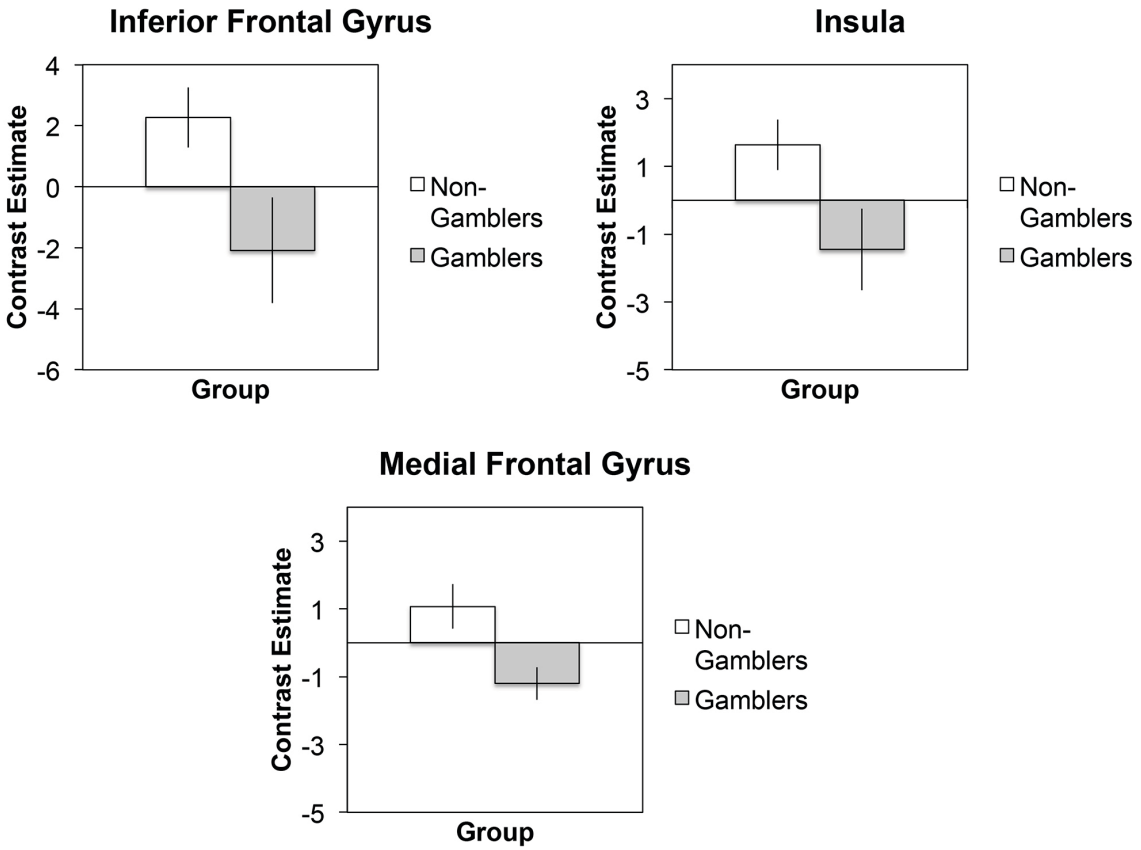

Figure 17. Contrast estimate and 90% confidence interval (inferior frontal gyrus, insula, medial frontal gyrus) for Win feedback

"Non-gamblers" show significantly greater activation and "Gamblers" show deactivation in the inferior frontal gyrus, insula and medial frontal gyrus when receiving "Win" feedback.

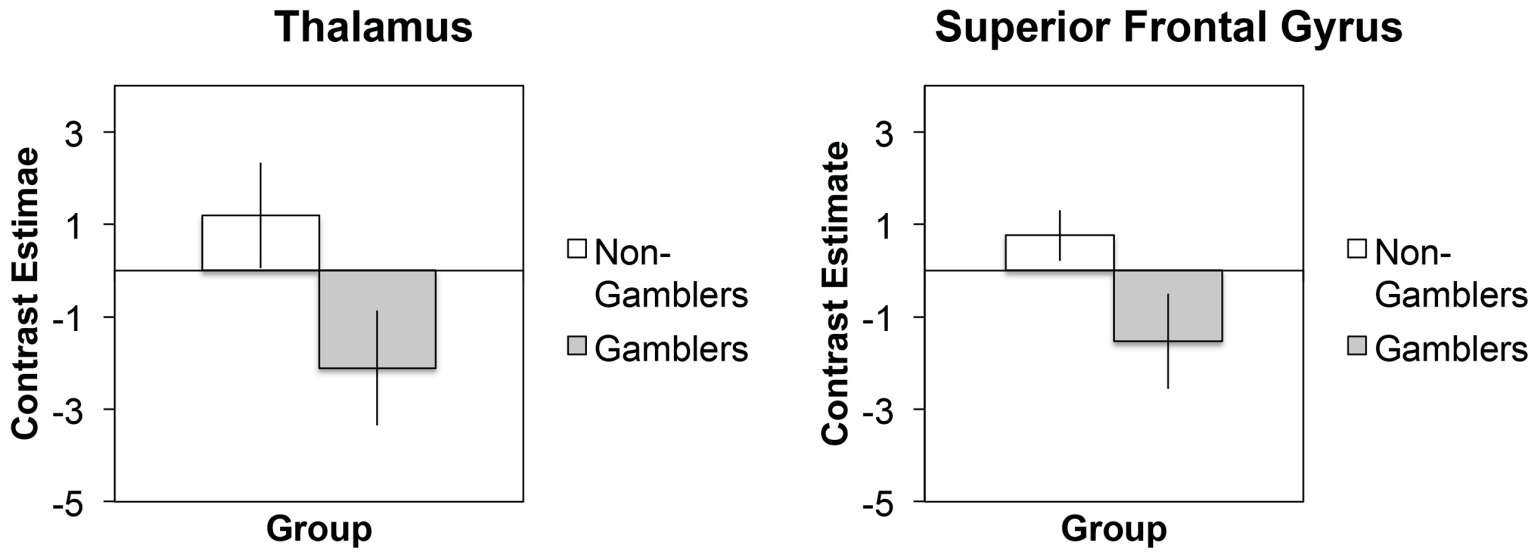

Figure 18. Contrast estimate and 90% confidence interval (thalamus, superior frontal gyrus) for Win feedback

Compared to baseline "Gamblers" display significant deactivation in the thalamus and superior frontal gyrus when receiving "Win" feedback.

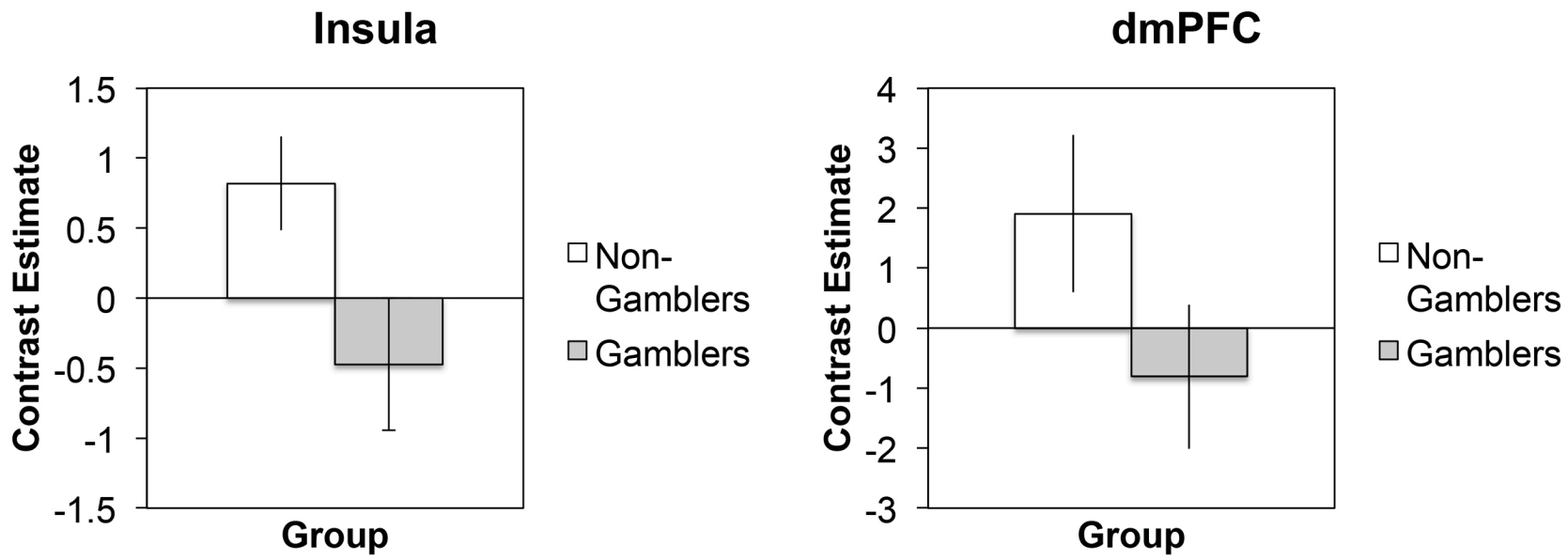

Figure 19. Contrast estimate and 90% confidence interval (insula, dmPFC) for Win feedback

Compared to baseline, "Non-Gamblers" display significant activation in the insula and dmPFC when receiving "Win" feedback. "Gamblers" do not display significant change compared to baseline.

8.6 General Discussion

We were interested in examining how sub-threshold gamblers respond in our investment task and how this manifests itself neurobiologically. Based on previous research it has been well established that pathological gamblers show deactivation in the vmPFC during gambling tasks; however, this pattern of activation was not found in our sub-threshold group. Interestingly, no significant differences appeared in the overall decision-making phases of the task. Previous research with pathological gamblers suggests that gamblers display dysfunctional brain activation in several regions during decision-making including the ACC, OFC, vlPFC and amygdala (Braus et al. 2001, George et al. 2001, Grüsser et al. 2004, Tapert et al. 2004); however, these patterns were not replicated with sub-threshold gamblers. Thus from our findings it is possible that these areas, typically dysfunctionally recruited in pathological gamblers, may represent regions associated with the severity of gambling behaviour.

In contrast, when the task was broken down to only look at "Buy" compared to "Did Not Buy" decisions, some significant differences did emerge. Sub-threshold gamblers displayed activation in the precuneus during Buy trials compared to non-gamblers. The precuneus has been linked to episodic memory (Lundstrom et al. 2003) thus it is possible that in this sub-threshold group decisions to buy may be triggered by retrieval of context-related (gambling) memories. Additionally deactivation in the posterior cingulate cortex may signify a lack of emotional salience associated with choosing to gamble/buy a stock in non-gamblers as this region has been linked to memory of emotional stimuli (Maddock et al. 2003). A suppression of activation in this region suggests that the decision to buy a stock in this task does not present strong positive or negative emotional valence in non-gamblers compared to sub-threshold gamblers. This may indicate that decisions to gamble are not as strongly encoded in memory for non-gamblers.

Our overall task analysis yielded differences between the two groups (gamblers and non-gamblers) during the feedback phase, partially supporting our first hypothesis, indicating that some of the reward pathways (insula and thalamus) are affected in those who gamble but do not meet the criteria for Gambling Disorder compared to those who do not gamble.

This provides evidence that sub-threshold gamblers have some dysfunctional reward pathways compared to non-gamblers in regards to financial decision-making and may form part of the mechanism that leads to continued and increased levels of gambling. When comparing Win feedback to Lose feedback, several differences emerged between the groups. The inferior frontal gyrus has been implicated in GO/NO-GO tasks and is believed to be involved in response inhibition (Garavan et al. 1999, Bunge et al. 2002, Rubia et al. 2003). A dampening of activation in this area would be consistent with the possibility that after sub-threshold gamblers are presented with winning feedback, they may be less likely to 'stop while they are ahead', whereas a win may signal when it is time to quit in non-gamblers. Such a suggestion is compatible with previous findings, for example diminished activation in gamblers compared to controls in response to reward has been suggested (Reuter et al. 2005, Balodis et al. 2012a), and our results support this finding with regard to the insula. Our findings suggest that one of the key elements separating sub-threshold gamblers and pathological gamblers may be the addition of decreased activity in both the vmPFC and ventral striatal area (Reuter et al. 2005, Balodis et al. 2012a). This in turn could lead to greater reward processing dysfunction, followed by increased likelihood of development of Gambling Disorder.

Our second hypothesis was that the sub-threshold group would perform poorly compared to controls, but this was not supported. This implies that in sub-threshold gamblers, decision-making does not appear to be significantly impaired. Previous work has established that pathological gamblers perform significantly worse, often by taking higher risk options, on a variety of tasks such as the IGT (Cavedini et al. 2002b, Kertzman et al. 2011, Brevers et al. 2012, Brevers et al. 2013b), GDT (Brand et al. 2005b, Labudda et al. 2007), and the WCST (Rugle and Melamed 1993, Forbush et al. 2008). Our results suggest that this harmful decision-making pattern is not yet significantly present in our gambling group (who were sub-threshold). This finding is in keeping with the lack of significant findings in brain activation in the overall task, as with this lack of significant neurobiological difference we may not see any significant behavioural differences.

In contrast, we found that obedience to the presented advice did differ between the groups, with "Non-gamblers" making more obedient decisions than "Gamblers" on the overall task. This supported the second part of our second hypothesis, When examining the task during each successive run, we found that the groups differed significantly in the final two runs, where the advice being given was all bad advice. These results may imply that the gamblers were in fact making more rational decisions than the controls, and that this decision-making was superior to that of the non-gambling control group. Interestingly, this more rational decision-making during the final runs of the task was not enough to significantly improve final performance/financial outcome, as neither group outperformed the other.

These findings provides some evidence that not all gambling behaviour leads to irrational decision-making, and that perhaps a moderate degree of gambling propensity could potentially shield investors from following poor advice and in the long run result in more profitable decisions. However, such a suggestion is speculative at present, even though differences in obedient decisions between the two groups continued to grow larger throughout the task. Significant differences in BOLD signal between the two groups was only found in the middle trials of the task, seemingly at odds with our current behavioural results. However one way to explain both these findings is that it is conceivable that in the middle trials, when the advice was mixed both good and bad advice, our sub-threshold group was learning not to trust the advice but by the final runs were no longer concerned with advice at all, negating any large obedience or disobedience effects. Consistent with this suggestion was that the sub-threshold group recruited the insula and ventral ACC during obedient trials, while the non-gamblers recruited the dorsal ACC when making non-obedient decisions. In choosing to not follow the advice of the expert our non-gamblers displayed (similar to the "Expert" group participants in Study 1) activation in an area linked to error detection (Carter et al. 1998, Bush et al. 2000) as well as violation of expectancy (Somerville et al. 2006). Meanwhile, in sub-threshold gamblers obedient trials recruited activation in regions associated with interoception (Critchley et al. 2004), risk-avoidance (Paulus et al. 2003, Kuhnen and Knutson 2005) and sensitivity to social and emotional evaluation (Somerville et al. 2006). The activation of the ventral ACC may signify a desire

of the sub-threshold gamblers to appear likeable when following advice that, based on the insula recruitment, may no longer seem sound.

Contrary to our third hypothesis, differences did emerge in Gamblers when comparing Advice to No Advice trials. Additional information was presented on the screen during Advice trials thus greater activation in the occipital lobe is to be expected. No Advice trials produced activation in the putamen suggesting that sub-threshold gamblers did associate No Advice trials with having greater risk as increased risk associated with the BART recruited greater activation in the striatum (Rao et al. 2008). However, when comparing our two groups, greater activation of the ACC, the error detection center of the brain (Carter et al. 1998, Bush et al. 2000) and superior frontal gyrus, believed to contribute to cognitive functions (Boisgueheneuc et al. 2006), during Advice trials in the gambling group does suggest that this group was less affected by the advice. If the advice were influencing decision-making, we would expect that in the presence of advice there would be a decrease of cognitive effort. That this pattern of activation was not found in the sub-threshold gamblers suggests that the non-gamblers made use of the advice to a greater extent that the sub-threshold gamblers. Interestingly, neither group displayed differential activation between good or bad advice. This suggests that neither group really differentiated between the two types of advice, as we would expect that if this difference was known Bad Advice trials would have elicited greater frontal lobe activation connected to greater effort in decision-making.

Post-hoc analysis of reaction times indicated that throughout the overall task there were no significant differences between the “Gamblers” and “Controls”. Overall, choosing not to follow the presented advice took longer than choosing to follow it for both groups suggesting that both groups were taking the advice into account to some degree, once again, providing support that sub-threshold gamblers were not completely immune to the advice. When looking at only Good Advice trials, this pattern of disobedient decisions taking longer than obedient decisions was repeated; yet, this same effect was not seen when comparing on Bad Advice trials, suggesting that when the advice was not sound participants were able to decide not to follow it more easily. However, upon comparing

Good and Bad Advice trials, no differences between overall reaction times was seen, supporting our fMRI findings. Supporting our fMRI results, sub-threshold gamblers were slightly slower than non-gamblers during Advice trials as this difference approached significance. This suggests that in the presence of advice non-gamblers were quicker to make their decisions, possibly due to the use of the advice as a cognitive shortcut in their decision-making process, whereas the sub-threshold gamblers did not make as great a use of the advice thus requiring more time to consider what action to take.

It is important to note that while our gamblers did not meet the requirements for gambling disorder and were not diagnosed they still reported high levels of frequent gambling, thus this group can still be considered regular and experienced gamblers. We suggest that, based on the DSM-5 criteria of significant distress for diagnosis, this *lack* of distress leading to overall *unimpaired* decision-making of sub-threshold gamblers has neurobiological underpinnings that differ from both non-gamblers but also pathological gamblers. While overall decision-making brain region recruitment does not yet differ significantly between non-gamblers and sub-threshold gamblers, the reward pathways do. We hypothesize that the vmPFC in particular plays one of the most important roles in the development of Gambling Disorder, as frequent gamblers who do not meet the criteria fail to show the pattern of deactivation so robustly found (Potenza 2013) in this area. However, in order to truly test this hypothesis a third group of diagnosed gamblers is required, and this is suggested as a future study.

Chapter 9. Study 4: Predictive Ability of Neurobiological BOLD Response by Psychological Profile

9.1 Recruitment and Participants

Participants were recruited from the University of Alberta campus and surrounding area via online advertising. The study was approved by the University of Alberta Health Research Ethics Board. All potential participants received details of the study, and signed an informed consent form. Individuals were screened for the presence of any psychiatric disorder (such as depression), using a semi-structured interview (Appendix A), as well as for any risk associated with having an MRI scan (metal in the body, claustrophobia etc.; Appendix B and C). Any individuals who had ongoing alcohol or drug abuse were excluded from further participation, as were any participants unable to have an MRI scan. Participants then completed a psychological battery consisting of the PGSI (Ferris and Wynne 2001), GABS (Breen and Zuckerman 1994), two measures of risk tolerance (Grable and Lytton 1999, Weber et al. 2002), the BIS-11 (Patton et al. 1995), the Rosenberg Self-Esteem Scale (Rosenberg 1965), and the ACE Scale (Felitti et al., 1998). A total of 39 individuals entered the study (mean age 26.13 ± 6.23 years, range: 28 years) of which 74.4% were male.

9.2 Hypotheses

In this study we wished to explore the predictive capabilities of a battery of psychological variables in the recruitment of brain regions during the investment task that we had developed (Chapter 5). We hypothesized that PGSI and GABS score, as our measures of gambling, would emerge as significant predictors of brain activation in our investment task, particularly in the vmPFC as this area has been linked to gambling behaviour (Potenza 2013). As links between gambling, impulsivity, risk tolerance, and self-esteem have been suggested, we hypothesized that these psychological scales may also be significant predictors of brain activity in an investment decision-making task. Other regions commonly associated with decision-making and risk taking include the cingulate cortex (Eriksen and

Eriksen 1974, Carter et al. 1998, Bush et al. 2000, Ullsperger and von Cramon 2001), inferior frontal gyrus (Crockford et al. 2005), insula (Paulus et al. 2003, Kuhnen and Knutson 2005, Engelmann et al. 2009), and striatum (Kuhnen and Knutson 2005, Rao et al. 2008) thus we selected these as our regions of interest for exploration.

9.3 Statistical Analysis

Based on the previous literature, beta values for six brain regions (Cingulate, Inferior Frontal Gyrus, Insula, Striatum, Striatum and Insula combined, and vmPFC) were extracted for four contrasts of interest: Buy – Did Not Buy, Obedient – Not Obedient, Win – Lose, and No Advice – Advice for each participant and formed our dependent variables. These areas were selected based on involvement during a variety of other decision-making tasks such as the IGT, GDT, WCST and BART. The independent variables were formed from the psychological survey responses previously mentioned in Chapter 7, total scale scores were calculated creating twelve variables in total (Table 8). Analyses were conducted using the statistical analysis program "R".

Table 8.
Independent Variables for Regression Analyses

Group	Controls or Gamblers
Age	
Gender	Male or Female
Education	
BIS_Total	Impulsivity Total Scale Score
RSE_Total	Rosenberg Self Esteem Scale
GABS_Total	Gambling Attitudes and Beliefs Scale
PGSI_Total	Problem Gambling Severity Index
Risk_Total†	Financial Risk Tolerance Total Scale
ACE_Total†	Adverse Childhood Experiences
DOSPERT_Total	DOSPERT Total Scale Score
DOSPERT_percTotal	DOSPERT Perception Total Scale Score

Normality of the dependent variables was assessed using QQ plots (Figure 20), which plot the data against a normal distribution. A sparse regression technique called the lasso (Tibshirani, 1996) was estimated on the data to determine important independent variables. We used a sparse regression technique as the number of independent variables was similar in size to the number of data points and hence ordinary multiple regression would fail in this context. Using the results from the sparse regression estimation, we kept the independent variables with non-zero beta estimates in the model and refit the data using ordinary multiple regression in order to reduce bias. Finally, non-significant variables were removed from this regression and the model was refitted if necessary. In total, 24 regressions were calculated (six dependent variables for each of the four contrasts of interest).

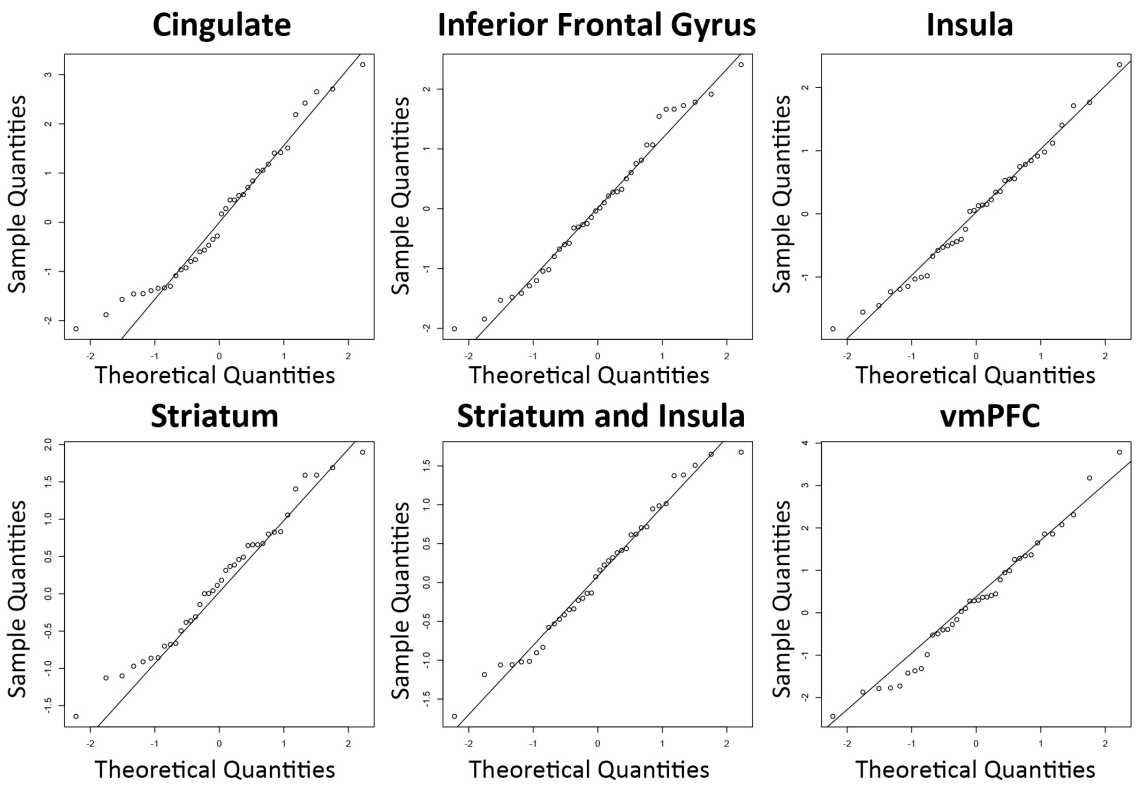

Figure 20. QQ-plots of dependent variables

QQ-plots of the six dependent variables of interest: cingulate, inferior frontal gyrus, insula, striatum, striatum and insula, and vmPFC. If the data is normally distributed then a linear progression is expected (where x=y). All the QQ plots showed acceptable distributions and were not indicative of non-normal data.

9.4 Results

While 24 regressions were calculated, only eight produced significant models. When looking at the Buy vs. Did Not Buy contrast, activation in the Cingulate was predicted by Gender and DOSPERT total perception of risk (Table 9) while activation in the Inferior Frontal Gyrus was predicted by DOSPERT totally perception of risk (Table 9). In the Obedient vs. Not Obedient contrast, Cingulate activation was weakly predicted by Education while PGSI score predicted Inferior Frontal Gyrus activation (Table 10). No variables were found to significantly explain the response activation in the Insula, Striatum, Striatum and Insula combined, and vmPFC for either contrast. No significant variables were found to significantly explain the response in any of the brain regions for the Win vs. Lose response. No Advice vs. Advice yielded significant models in four brain regions (Table 11). Both DOSPERT total willingness to participate and DOSPERT total perception of risk significantly predicted the variability in the Cingulate response, DOSPERT total perception of risk predicted the response in the Striatum, GABS score predicted response in the Striatum and Insula combined, and vmPFC activation was predicted by the BIS total score (Table 11).

Table 9.

Regression Results for Buy vs. Did Not Buy

	Cingulate[a]			
	β	SE(β)	t	p
Constant	-2.333	1.093	-2.135	0.040
Gender	-1.557	0.513	-3.037	0.005
DOSPERT (p)	0.116	0.046	2.542	0.016
	Inferior Frontal Gyrus[b]			
	β	SE(β)	t	p
Constant	-2.101	0.889	-2.364	0.024
DOSPERT (p)	0.088	0.0356	2.496	0.018

[a]Adjusted R2 = 0.200

[b]Adjusted R2 = 0.121

Table 10.

Regression Results for Obedient vs. Not Obedient

	Cingulate[a]			
	β	SE(β)	t	p
Constant	1.132	0.54	2.095	0.433
Education	-0.424	0.199	-2.126	0.041
	Inferior Frontal Gyrus[b]			
	β	SE(β)	t	p
Constant	-0.071	0.121	-0.59	0.560
PGSI	0.052	0.022	2.334	0.025

[a]Adjusted R2 = 0.087

[b]Adjusted R2 = 0.107

Table 11.

Regression Results for No Advice vs. Advice

			Cingulate[a]	
	β	SE(β)	t	p
Constant	5.803	1.368	4.242	0.0002
DOSPERT (w)	-0.114	0.031	-3.732	0.0006
DOSPERT (p)	-0.123	0.036	-3.432	0.002
			Striatum[b]	
	β	SE(β)	t	p
Constant	2.089	0.741	2.817	0.008
DOSPERT (p)	-0.072	0.030	-2.441	0.020
			Striatum and Insula[c]	
	β	SE(β)	t	p
Constant	2.349	0.780	3.011	0.005
GABS	-0.026	0.010	-2.709	0.010
			vmPFC[d]	
	β	SE(β)	t	p
Constant	5.519	1.791	3.082	0.004
BIS	-0.085	0.028	-2.977	0.005

[a]Adjusted R2 = 0.299

[b]Adjusted R2 = 0.118

[c]Adjusted R2 = 0.146

[d]Adjusted R2 = 0.175

9.5 General Discussion

In this final study we wished to explore the predictive strength of a battery of psychological scales on brain activation during an investment task. Based on previous research citing the vmPFC as one of the key areas of dysfunction in gamblers during decision-making tasks (Potenza 2013), we were surprised to find that neither of our gambling surveys proved to be significant predictors of activation in this region across all the conditions of our investment task. The only model for vmPFC activation that was significant included our measure of impulsivity (BIS) as the only independent variable when looking at Advice compared to No Advice trial BOLD signal. It appears that the lower impulsivity score reported by an individual, the greater disparity in brain activation between advice and no advice trials. If an individual is highly impulsive then they are likely going to make decisions more rashly and therefore would put less thought into their decisions, regardless of the presence of advice. It is interesting that this effect appears in the vmPFC, where decision-making dysfunctions have been repeatedly reported as this suggests that one of the key components involved with decision-making dysfunction in this region is impulsivity.

Overall risk tolerance emerged as significant predictors of activation in both the cingulate cortex and striatum when comparing No Advice to Advice trials. 30% of the variability in cingulate cortical activation was predicted by both the willingness to participate in risky behaviours as well as the perception of risk associated with behaviours. A lower score on both these measures was linked to greater differences in signal strength in the cingulate cortex during No Advice and Advice trials. As previously discussed, the cingulate cortex is involved in decision-making, in particular conflict monitoring (Eriksen and Eriksen 1974, Ullsperger and von Cramon 2001) and error detection (Carter et al. 1998, Bush et al. 2000) as well as sensitivity to social and emotional evaluation (Somerville et al. 2006). A lower level of risk tolerance leading to greater activation in this region may indicate that those who are less comfortable with risk will defer to advice when available and would thus have a greater difference in recruitment of the cingulate cortex between No Advice and Advice trials. As this group may also be more keen to avoid risk, a task that requires them to choose risky options may increase overall levels of activation.

The striatum has been implicated in increased risk during decision-making (Rao et al. 2008) thus it follows that lower tolerance for perceived levels of risk associated with social, ethical, financial, health and recreational behaviours predicted greater activation differences in this region between No Advice and Advice trials. If an individual reports a lower level of risk tolerance we would expect trials with no aid to be perceived as especially risky and elicit greater activation than for an individual with a higher level of risk tolerance.

Gambling attitudes predicted 14.6% of the variability in combined brain activation in the striatum and insula for No Advice vs. Advice trials indicating that as gambling attitudes became more positively endorsed, the difference between No Advice and Advice trial brain activation decreased. Let us suppose that the difference in BOLD signal between No Advice and Advice trials increases in these areas when participants are using advice due to an increased level of risk associated to No Advice trials (Rao et al. 2008). It then follows that when the advice is taken away (No Advice trials), an increase in pro-gambling beliefs (likely indicative of increased gambling behaviour) results in a decrease in associated risk of these trials; in other words, those with higher pro-gambling beliefs may view No Advice trials as less risky that those with lower pro-gambling beliefs. Potentially, it is greater experience with risky financial decisions that make No Advice trials less threatening/risky.

Two regions found to be significant in several conditions include the cingulate cortex and inferior frontal gyrus. When comparing differences in activation between Buy and Did Not Buy trials, both gender and DOSPERT perception of risk were significant contributors to the model of cingulate activation. The DOSPERT perception of risk was also the only significant contributor to the model of inferior frontal gyrus activation. It appears that as tolerance to perceived overall risk decreases (when events are increasingly rated as risky by an individual), so does the signal in both the cingulate and inferior frontal gyrus during decisions to buy a stock. Deciding to buy a stock would be considered a risky decision as in this situation the outcome is uncertain as this trial may yield either a win or a loss. Therefore, it appears that risk perception may be associated with both the cingulate and the inferior frontal gyrus, and this finding is consistent with previous research that these areas are linked to perception (Phillips et al. 2003, Shamay-Tsoory et al. 2009). As the entire

cingulate was selected as a region of interest, we cannot specify if it was the anterior or posterior cingulate cortex or both that is associated with the DOSPERT. It should also be noted that both models are fairly week, only explaining 20% and 12% of the variability in the BOLD signal, and therefore other variables not currently measured are likely to play a larger role.

When looking at Obedient vs. Not Obedient trials, once again activation in both the cingulate cortex and the inferior frontal gyrus produce significant models. Education appears to explain roughly 9% of the variability in the difference between obedient and not obedient brain activation. One potential explanation is that a higher level of education may lead an individual to feel less conflicted about disobeying an outsiders advice which could manifest itself as a weaker signal from the ACC, this conflict region of the brain (Eriksen and Eriksen 1974, Ullsperger and von Cramon 2001). However, more research would need to be conducted to either confirm or refute this hypothesis. PGSI score predicted 10.7% of the variability in activation in the inferior frontal gyrus. In pathological gamblers, the inferior frontal gyrus was found to exhibit greater activation in response to gambling cues compared to healthy controls (Crockford et al. 2005). While that study only measured activation in this region during a passive visual task, our findings may point to a role of the inferior frontal gyrus in the decision to be obedient or not obedient to outside expert advice, with those with increased levels of gambling displaying greater signal level disparity.

The strongest model that we found only predicted roughly 30% of the variability in our dependent variables. Thus it is clear that we are not capturing all the meaningful traits in our psychological battery. We chose to focus on six brain regions due to the frequency with which they appear in decision-making literature and thus we expected that these regions would yield the most significant results. In the future, more comprehensive batteries should be taken into consideration as well as additional brain regions. However, as with all good research, they should be hypothesis-led and should consider the existing literature when selecting regions of interest. A highly psychologically variable population may also help to increase the power of the models, for example, including diagnosed pathological gamblers who will increase the spread of scores across the gambling screens.

This study was the first study, to our knowledge, to consider how psychological variables can model brain activation in an investment decision-making task, but given the sample size these results should be considered preliminary. There are many future directions that this research may continue to take that may help in understanding the connection between psychological factors, decision-making and recruitment of various brain regions.

Chapter 10. Conclusion

10.1 Overall conclusion

The present research focuses on investment decision-making and the influence of advice on brain activation as well as how the recruitment of brain regions during decision-making may differ amongst controls and sub-threshold gamblers. The results suggest that when individuals defy authority, or believe they are doing this (in this case by disobeying an outside financial expert) to make an investment decision there is increased activation of the Anterior cingulate cortex (ACC) and superior frontal gyrus, and that **such activation is in part responsible for "disobedience" to expert advice**. This is a highly novel finding, as there have been no previous studies which have identified the brain regions responsible for "disobedience". These results specify the differences in activation to the level of source (expert or peer) and benefit of advice (good or bad). Increased awareness of this may allow strategies to be developed to help both individuals and groups avoid inappropriate financial decisions. It is also possible that these results may have wider implications about the brain mechanisms underlying obedience. When the advice is sought from someone deemed to be an expert, it is conceivable that this influence can have negative outcomes for individuals, as they might offload cognitively and defer to the expert without forming independent judgments. It is particularly important to note that in our study the expert providing the advice was not present, or ever seen; however, the advice had a meaningful impact on both brain activation and behaviour.

In expanding this research to include sub-threshold gambling, potential decision-making benefits associated with gambling were explored. That this group of sub-threshold gamblers displayed more rational decision-making in the face of wayward advice suggests that, to some degree, a propensity towards gambling may be beneficial in avoiding typical social influence pitfalls. It is also conceivable that the difference between remaining a sub-threshold *non-distressed* gambler instead of a pathological *distressed* gambler may be linked to several specific brain regions including the vmPFC and ventral striatum as these regions appear to remain functionally intact in sub-threshold gamblers but not in pathological gamblers. It remains to be concluded if sub-threshold gamblers who do not

cross over into gambling disorder are neurobiologically different than sub-threshold gamblers who do make that transition. The possibility remains that those individuals who are likely to fall into gambling disorder may already show some dysfunctional recruitment of these brain regions prior to the onset of the gambling problem. Alternatively, gambling behaviour may alter the normal pattern of brain activation and, as gambling behaviour progresses so does the level of dysfunctional brain activation. Further longitudinal studies may help to clarify these uncertainties.

Research has shown that gambling disorder shares some commonalities with substance abuse; hence it's reclassification into the same category in the newest edition of the DSM. However, despite much research in the field, there are still conflicting theories on some of the psychological links to gambling. Our results provide support for suggestions that those experiencing gambling problems endorse more positive attitudes and beliefs towards gambling. Our primary psychological finding is that gamblers have greater risk tolerance across multiple domains, not just greater financial risk tolerance, even though they do not have a difference in terms of their risk assessment. Our findings also failed to support previous suggestions that impulsivity significantly explains gambling severity in the presence of other variables or that this is linked to lowered self-esteem or greater rates of adverse childhood experience. The psychological factors, which lead some individuals to change from enjoying gambling as an occasional social activity to one that can have catastrophic personal impacts for that individual, remain uncertain. Additionally, while certain brain regions level of activation during investment decision-making may be partially explained by the psychological factors measured it is clear, due to the low explanatory powers of the models, that these factors do not encompass the full psychological story of decision-making.

10.2 Limitations and Future Directions

This research had several limitations to consider. The present studies were not double-blind studies, so it is possible that some biases may have existed. Furthermore, most of the individuals taking part were university students or university aged individuals and it may

not be appropriate to generalize these findings to the general population. While our control in the first study of having "Peer" advice was one that served the purpose of our study, it should be noted that a true control would have involved an additional scenario where participants were told that the advice was random. Nonetheless, given the size of our studies, the robustness of the findings about brain activation when individuals defy the advice of an alleged financial "Expert", and that the findings are compatible with the existing literature, we believe these findings add meaningfully to the existing understanding of why individuals make irrational financial decisions, particularly when under the influence of advice.

Another limitation was that the number of gamblers was relatively small. This finding means that our results need to be replicated in larger studies. Additionally, since they were recruited via the Internet, they may preferentially represent certain groups of gamblers (young, internet gamblers). They were also in the top of the "moderate" range for gambling scores, so individuals with more severe gambling problems may have given a different outcome. Furthermore, although there were no differences in age, sex, or ethnicity between our two groups, there was a difference in education. This difference in level of education was likely due to how our sample was recruited, with many of the controls coming from the University of Alberta while many of our gamblers were recruited from the wider community. However, it is important to note that while the proportion of university educated participants favored the control group, there was still a mix of both university and high-school educated individuals in both groups. As well, this difference in level of education was taken into consideration and controlled for in our analyses. In order to test the generalizability of our results, a larger and more varied sample is required.

Finally, we did not test a group of pathological gamblers, and thus our results cannot be taken as generalizing to this group. However, they do point out differences between both controls and sub-threshold gamblers (in our study) and between our findings in sub-threshold gamblers and those with pathological gambling (from the literature). This would suggest they may actually be a useful group to study in future. For these reasons an ideal

subsequent study would be to conduct a study with this investment task in three groups: non-gamblers, sub-threshold gamblers, and diagnosed pathological gamblers. Only then could we conclude if all three groups have unique patterns of brain activation, as the possibility remains that the level of BOLD signal difference between sub-threshold gamblers and pathological gamblers may not reach significance. It is possible that sub-threshold gamblers are quite similar to pathological gamblers in brain pattern activation but certain regions were not yet able to meet statistical significance in comparison to non-gamblers. The next step in this research would involve conducting this study to elucidate any differences. Additionally, in order to determine if brain dysfunction or problem gambling behaviour occur together or one precedes the other, a longitudinal study would be necessary.

10.3 Conclusion

Taken together, in these studies the first major finding from this thesis is that it supports suggestions of an obedience to "Expert" advice, even when no "Expert" is actually seen. Our second conclusion is that it is conceivable that the mechanism by which this occurs could involve cognitive offloading, which occurs when "Expert" advice is present. These processes may, in part, explain some of the reasons why individuals choose to follow the advice of "Experts", financial and otherwise. Our third conclusion is that while the psychological factors related to gambling, as well as their relation to differences in recruitment of various brain regions, remain unclear it appears that sub-threshold gamblers differ from non-gamblers in reward processing. From this, we conclude that this group likely has its own unique pattern of activation during decision-making. Further studying this group may help to guide our understanding of Gambling Disorder by specifying how dysfunction differs across non-gamblers, sub-threshold gamblers, and pathological gamblers.

References

Abbott, M. W. and R. A. Volberg (1995). "The New Zealand national survey of problem and pathological gambling." Journal of Gambling Studies **12**: 143-160.

Allcock, C. C. and D. M. Grace (1988). "Pathological gamblers are neither impulsive nor sensation-seekers." Australian and New Zealand Journal of Psychiatry **22**(3): 307-311.

Anderson, G. and R. I. Brown (1984). "Real and laboratory gambling, senstation-seeking and arousal." British Journal of Psychology **75**: 401-410.

Andreasen, N. C., D. S. O'Leary, T. Cizadlo, S. Arndt, K. Rezai and G. L. Watkins (1995). "Remembering the past: Two facets of episodic memory explored with positron emission tomography." The American Journal of Psychiatry **152**: 1576-1585.

Aron, A. R. (2007). "The Neural Basis of Inhibition in Cognitive Control." The Neuroscientist **13**(3): 214-228.

Arrow, K. J. (1951). Social choice and individual values. New York, Wiley.

Arrow, K. J. and G. Debreu (1954). "Existence of an equilibrium for a competitice economy." Econometrica **22**(3): 265-290.

Asch, S. E. (1956). "Studies of independence and conformity: A minority of one against a unanimous majority." Psychological Monographs **70**: Whole No. 416.

Association, A. P. (2013). Diagnostic and statistical manual of mental disorders. Arlington, VA, American Psychiatric Publishing.

Baker, S., R. Dolan and C. Frith (1996). "The functional anatomy of logic: a PET study of inferential reasoning." Neuroimage **3**(3): S218.

Baler, R. D. and N. D. Volkow (2011). "Addiction as a Systems Failure: Focus on Adolescence and Smoking." Journal of the American Academy of Child & Adolescent Psychiatry **50**: 329-339.

Balodis, I. M., H. Kober, P. D. Worhunsky, M. C. Stevens, G. D. Pearlson and M. N. Potenza (2012a). "Diminished Frontostriatal Activity During Processing of Monetary Rewards and Losses in Pathological Gambling." Biological Psychiatry **71**(8): 749-757.

Balodis, I. M., C. M. Lacadie and M. N. Potenza (2012b). "Self-reported gambling urge intensity correlates with temporal pole activation in men with pathological gambling." Journal of Gambling Studies **28**: 493-513.

Banerjee, A. (1992). "A simple model of herd behavior." Quarterly Journal of Economics **107**: 797-817.

Bartley, C. A. and M. H. Bloch (2013). "Meta-analysis: pharmacological treatment of pathological gambling." Expert Review of Neurotherapeutics **13**(8): 887-894.

Baumgartner, T., U. Fischbacher, A. Feierabend, K. Lutz and E. Fehr (2009). "The neural circuitry of a broken promise." Neuron **64**: 756-770.

Bausell, R. B. (2007). Snake oil science. New York, Oxford University Press.

Bechara, A. (2001). "Neurobiology of decision-making: Risk and reward." Seminars in Clinical Neuropsychiatry **6**: 205-216.

Bechara, A. (2005). "Decision making, impulse control and loss of willpower to resist drugs: a neurocognitive perspective." Nature Neuroscience **8**(11): 1458-1463.

Bechara, A. and A. Damasio (2005). "The somatic marker hypothesis: A neural theory of economic decision." Games and Economic Behavior **52**: 336-372.

Bechara, A., A. Damasio, H. Damasio and S. W. Anderson (1994). "Insensitivity to future consequences following damage to human prefrontal cortex." Cognition **50**: 7-15.

Bechara, A. and H. Damasio (2002). "Decision-making and addiction (part 1): Impaired activation of somatic states in substance dependent individuals when pondering decisions with negative future consequences." Neuropsychologia **40**: 1675-1689.

Bechara, A., H. Damasio and A. R. Damasio (2003). "Role of the Amygdala in Decision‐Making." Annals of the New York Academy of Sciences **985**(1): 356-369.

Bechara, A., H. Damasio, A. R. Damasio and G. P. Lee (1999). "Different contributions of the human amygdala and ventromedial prefrontal cortex to decision-making." The Journal of Neuroscience **19**(13): 5473-5481.

Bechara, A., H. Damasio, D. Tranel and S. W. Anderson (1998). "Dissociation of working memory from decision making within the human prefrontal cortex." The Journal of Neuroscience **18**(1): 428-437.

Bechara, A., S. Dolan and A. Hindes (2002). "Decision-making and addiction (part II): myopia for the future or hypersensitivity to reward?" Neuropsychologia **40**(10): 1690-1705.

Bechara, A. and E. M. Martin (2004). "Impaired decision making related to working memory deficits in individuals with substance addictions." Neuropsychology **18**(1): 152.

Bechara, A., D. Tranel, H. Damasio and A. R. Damasio (1996). "Failure to Respond Autonomically to Anticipated Future Outcomes Following Damage to Prefrontal Cortex." Cerebral Cortex **6**(2): 215-225.

Belger, A., A. Puce, J. H. Krystal, J. C. Gore, P. Goldman-Rakic and G. McCarthy (1998). "Dissociation of mnemonic and perceptual processes during spatial and nonspatial working memory using fMRI." Human Brain Mapping **6**(1): 14-32.

Bergstresser, D., J. Chalmers and P. Tufan (2009). "Assessing the costs and benefits of brokers in the mutual fund industry." Review of Financial Studies **22**: 4129-4156.

Berman, K. F., J. L. Ostrem, C. Randolph, J. Gold, T. E. Goldberg, R. Coppola, R. E. Carson, P. Herscovitch and D. R. Weinberger (1995). "Physiological activation of a cortical network during performance of the Wisconsin Card Sorting Task: A positron emission tomography study." Neuropsychologia **33**: 1027-1046.

Berridge, K. C. (2007). "The debate over dopamine's role in reward: the case for incentive salience." Psychopharmacology **191**(3): 391-431.

Berridge, K. C. (2012). "From prediction error to incentive salience: Mesolimbic computation of reward motivation." European Journal of Neuroscience **35**: 1123-1143.

Berridge, K. C., F. W. Flynn, J. Schulkin and H. J. Grill (1984). "Sodium depletion enhances salt palatability in rats." Behavioral Neuroscience **98**(4): 652-660.

Berridge, K. C. and T. E. Robinson (1998). "What is the role of dopamine in reward: hedonic impact, reward learning, or incentive salience?" Brain Research Reviews **28**(3): 309-369.

Bikhchandani, S., D. Hirshleifer and I. Welch (1992). "A theory of fads, fashion, custom and cultural change as informational cascades." Journal of Political Economy **100**: 992-1026.

Billieux, J., M. Van der Linden, Y. Khazaal, D. Zullino and L. Clark (2012). "Trait gambling cognitions predict near-miss experiences and persistence in laboratory slot machine gambling." British Journal of Psychology **103**(3): 412-427.

Blasczcynski, A., Z. Steel and N. McConaghy (1997). "Impulsivity in pathological gambling: The antisocial impulsivist." Addiction **92**: 75-87.

Blaszczynski, A., N. McConaghy and A. Frankova (1990). "Boredom proneness in pathological gambling." Psychological Reports **67**(1): 35-42.

Blaszczynski, A. and L. Nower (2002). "A pathways model of problem and pathological gambling." Addiction **97**(5): 487-499.

Blaszczynski, A., A. C. Wilson and N. McConaghy (1986). "Sensation seeking and pathological gambling." British Journal of Addiction **81**(1): 113-117.

Bodenhausen, G. V., C. N. Macrae and J. W. Sherman (1999). On the dialectics of discrimination: Dual processes in social stereotyping. Dual-process theories in social psychology. S. Chaiken and Y. Trope. New York, Guilford**:** 271-290.

Boisgueheneuc, F. d., R. Levy, E. Volle, M. Seassau, H. Duffau, S. Kinkingnehun, Y. Samson, S. Zhang and B. Dubois (2006). "Functions of the left superior frontal gyrus in humans: a lesion study." Brain **129**(12): 3315-3328.

Botvinick, M., L. E. Nystrom, K. Fissell, C. S. Carter and J. D. Cohen (1999). "Conflict monitoring versus selection-for-action in anterior cingulate cortex." Nature **402**(6758): 179-181.

Botvinick, M. M., T. S. Braver, D. M. Barch, C. S. Carter and J. D. Cohen (2001). "Conflict monitoring and cognitive control." Psychological Review **108**(3): 624-652.

Bowen, F. P., R. D. Kamienny, M. M. Burns and M. Yahr (1975). "Parkinsonism: Effects of levodopa treatment on concept formation." Neurology **25**: 701-704.

Brand, M., C. Franke-Sievert, G. E. Jacoby, H. J. Markowitsch and B. Tuschen-Caffier (2007a). "Neuropsychological correlates of decision making in patients with bulimia nervosa." Neuropsychology **21**(6): 742-750.

Brand, M., E. Fujiwara, S. Borsutzky, E. Kalbe, J. Kessler and H. J. Markowitsch (2005a). "Decision-making deficits of korsakoff patients in a new gambling task with explicit rules: Associations with executive functions." Neuropsychology **19**: 267-277.

Brand, M., E. Kalbe, K. Labudda, E. Fujiwara, J. Kessler and H. J. Markowitsch (2005b). "Decision-making impairments in patients with pathological gambling." Psychiatry Research **133**(1): 91-99.

Brand, M., K. Labudda, E. Kalbe, R. Hilker, D. Emmans, G. Fuchs, J. Kessler and H. J. Markowitsch (2004). "Decision-making impairments in patients with Parkinson's disease." Behavioral Neurology **15**: 77-85.

Brand, M., E. C. Recknor, F. Grabenhorst and A. Bechara (2007b). "Decisions under ambiguity and decisions under risk: Correlations with executive functions and comparisons of two different gambling tasks with implicit and explicit rules." Journal of Clinical and Experimental Neuropsychology **29**(1): 86-99.

Braus, D. F., J. Wrase, S. Grüsser, D. Hermann, M. Ruf, H. Flor, K. Mann and A. Heinz (2001). "Alcohol-associated stimuli activate the ventral striatum in abstinent alcoholics." Journal of Neural Transmission **108**(7): 887-894.

Breen, R. B. and M. Zuckerman (1994). The gambling beliefs and attitudes survey. Unpublished instrument. University of Delaware.

Brevers, D., A. Bechara, A. Cleeremans and X. Noël (2013a). "Iowa Gambling Task (IGT): Twenty years aftehr - gambling disorder and IGT." Frontiers in Psychology **4**: 665-679.

Brevers, D., A. Cleeremans, A. Bechara, M. Greisen, C. Kornreich, P. Verbanck and X. Noël (2013b). "Impaired Self-Awareness in Pathological Gamblers." Journal of Gambling Studies **29**(1): 119-129.

Brevers, D., A. Cleeremans, A. E. Goudriaan, A. Bechara, C. Kornreich, P. Verbanck and X. Noël (2012). "Decision making under ambiguity but not under risk is related to problem gambling severity." Psychiatry Research **200**(2–3): 568-574.

Brevers, D., A. Cleeremans, C. Hermant, H. Tibboel, C. Kornreich, P. Verbanck and X. Noël (2013c). "Implicit gambling attitudes in problem gamblers: Positive but not negative implicit associations." Journal of Behavior Therapy and Experimental Psychiatry **44**(1): 94-97.

Brown, M. R. G., R. M. Lebel, F. Dolcos, A. H. Wilman, P. H. Silverstone, H. Pazderka, E. Fujiwara, T. C. Wild, A. M. Carroll, O. Hodlevskyy, L. Zedkova, L. Zwaigenbaum,

A. H. Thompson, A. J. Greenshaw and S. M. Dursun (2012). "Effects of emotional context on impulse control." Neuroimage **63**(1): 434-446.

Brown, R. I. F. (1993). Some contributions of the study of gambling to the study of other addictions. Gambling Behavior and Gambling Problems. W. R. Eadington and J. A. Cornelius**:** 241-272.

Browne, A. and D. Finkelhor (1986). "Impact of child sexual abuse: A review of the research." Psychological Bulletin **99**: 66-77.

Bunge, S. A., N. M. Dudukovic, M. E. Thomason, C. J. Vaidya and J. D. E. Gabrieli (2002). "Immature frontal lobe contributions to cognitive control in children: Evidence from fMRI." Neuron **33**(2): 301-311.

Burgdorf, J. and J. Panksepp (2006). "The neurobiology of positive emotions." Neuroscience & Biobehavioral Reviews **30**(2): 173-187.

Burge, A., R. Pietrzak and N. Petry (2006). "Pre/Early Adolescent Onset of Gambling and Psychosocial Problems in Treatment-Seeking Pathological Gamblers." Journal of Gambling Studies **22**(3): 263-274.

Bush, G., P. Luu and M. I. Posner (2000). "Cognitive and emotional influences in anterior cingulate cortex." Trends in Cognitive Sciences **4**(6): 215-222.

Bush, G., B. A. Vogt, J. Holmes, A. M. Dale, D. Greve, M. A. Jenike and B. R. Rosen (2002). "Dorsal anterior cingulate cortex: A role in reward-based decision making." Proceedings of the National Academy of Sciences **99**(1): 523-528.

Butter, C. M. (1969). "Perseveration in extinction and in discrimination reversal tasks following selective frontal ablations in Macaca mulatta." Physiology & Behavior **4**(2): 163-171.

Calder, A. J., A. D. Lawrence and A. W. Young (2001). "Neuropsychology of fear and loathing." Nature Reviews Neuroscience **2**: 352-363.

Carlton, P. L. and P. Manowitz (1994). "Factors determining the severity of pathological gambling in males." Journal of Gambling Studies **10**(2): 147-157.

Carter, C. S., T. S. Braver, D. M. Barch, M. M. Botvinick, D. Noll and J. D. Cohen (1998). "Anterior cingulate cortex, error detection, and the online monitoring of performance." Science **280**(5364): 747-749.

Castellani, B., E. Wootton, L. Rugle, R. Wedgeworth, K. Prabucki and R. Olson (1996). "Homelessness, negative affect, and coping among veterans with gambling problems who misused substances." Psychiatric Services **47**(3): 298-299.

Cavada, C., T. Compañy, J. Tejedor, R. J. Cruz-Rizzolo and F. Reinoso-Suárez (2000). "The Anatomical Connections of the Macaque Monkey Orbitofrontal Cortex. A Review." Cerebral Cortex **10**(3): 220-242.

Cavedini, P., T. Bassi, A. Ubbiali, A. Casolari, S. Giordani, C. Zorzi and L. Bellodi (2004). "Neuropsychological investigation of decision-making in anorexia nervosa." Psychiatry Research **15**: 259-266.

Cavedini, P., G. Riboldi, A. D'Annucci, P. Belotti, M. Cisima and L. Bellodi (2002a). "Decision-making heterogenity in obsessive-compulsive disorder: Ventromedial prefrontal cortex function predicts different treatment outcomes." Neuropsychologia **40**: 205-211.

Cavedini, P., G. Riboldi, R. Keller, A. D'Annucci and L. Bellodi (2002b). "Frontal lobe dysfunction in pathological gambling patients." Biological Psychiatry **51**(4): 334-341.

Champine, R. B. and N. M. Petry (2010). "Pathological gamblers respond equally well to cognitive-behavioral therapy regardless of other mental health treatment status." American Journal on Addiction **19**: 550-556.

Chase, H. W. and L. Clark (2010). "Gambling severity predicts midbrain response to near-miss outcomes." The Journal of Neuroscience **30**: 6180-6187.

Chevalier, J. and G. Ellison (1999). "Career Concerns of Mutual Fund Managers." The Quarterly Journal of Economics **114**(2): 389-432.

Chib, V. S., A. Rangel, S. Simojo and J. O'Doherty (2009). "Evidence for a Common Representation of Decision Values for Dissimilar Goods in Human Ventromedial Prefrontal Cortex." Journal of Neuroscience **29**: 12315-12320.

Cialdini, R. B. (2009). Influence: Science and practice. Boston, Allyn and Bacon, Inc.

Clark, L., A. Bechara, H. Damasio, M. R. F. Aitken, B. J. Sahakian and T. W. Robbins (2008). "Differential effects of insular and ventromedial prefrontal cortex lesions on risky decision-making." Brain **131**(5): 1311-1322.

Clark, L., A. J. Lawrence, F. Astley-Jones and N. Gray (2009). "Gambling Near-Misses Enhance Motivation to Gamble and Recruit Win-Related Brain Circuitry." Neuron **61**(3): 481-490.

Cloninger, C. R., S. Sigvardsson and M. Bohman (1988). "Childhood personality predicts alcohol abuse in young adults." Alcohol Clin Exp Res **12**: 494-505.

Cooksey, R. W. (1996). Judgment analysis: Theory, methods, and applications. San Diego, CA, Academic.

Coventry, K. R. (2001). "Mood state, arousal, decision making, and persistence at gaming." Addiction **96**(1860-1861).

Coventry, K. R. and J. Hudson (2001). "Gender differences, physiological arousal and the role of winning in fruit machine gamblers." Addiction **96**(6): 871-879.

Coventry, K. R. and A. C. Norman (1997). "Arousal, sensation seeking and frequency of gambling in off-course horse racing bettors." British Journal of Psychology **88**(4): 671-681.

Cox, W. M., J. S. Fadardi and E. M. Pothos (2006). "The Addiction-Stroop test: Theoretical considerations and procedural recommendations." Psychological Bulletin **132**(3): 443-476.

Critchley, H. D., C. J. Mathias and R. J. Dolan (2001). "Neural Activity in the Human Brain Relating to Uncertainty and Arousal during Anticipation." Neuron **29**(2): 537-545.

Critchley, H. D., S. Wiens, P. Rotshtien, A. Ohman and R. J. Dolan (2004). "Neural systems supporting interoceptive awareness." Nature Neuroscience **7**: 189-195.

Crockford, D. N., B. Goodyear, J. Edwards, J. Quickfall and N. el-Guebaly (2005). "Cue-Induced Brain Activity in Pathological Gamblers." Biological Psychiatry **58**(10): 787-795.

Dalgleish, T. (2004). "The emotional brain." Nature Reviews Neuroscience **5**(7): 583-589.

Damasio, A. (1994). Descartes' error: Emotion, reason, and the human brain. New York: NY, Putnam Publishing.

Damasio, A. R., B. J. Everitt and D. Bishop (1996). "The Somatic Marker Hypothesis and the Possible Functions of the Prefrontal Cortex [and Discussion]." Philosophical

Transactions of the Royal Society of London. Series B: Biological Sciences **351**(1346): 1413-1420.

Damasio, A. R., D. Tranel and H. Damasio (1990). "Individuals with sociopathic behavior caused by frontal damage fail to respond autonomically to social stimuli." Behavioural Brain Research **41**(2): 81-94.

Darwin, C. (1871). The descent of man, and selection inrelation to sex. London, John Murray.

Dawe, S. and N. J. Loxton (2004). "The role of impulsivity in the development of substance use and eating disorders." Neuroscience and Biobehavioral Reviews **28**: 343-351.

De Bondt, W. F. and R. H. Thaler (1985). "Does the stock market overreact?" Journal of Finance **40**: 793-805.

de Quervain, D. J., U. Fischbacher, V. Treyer, M. Schellhammer, U. Schnyder, A. Buck and E. Fehr (2004). "The neural basis of altruistic punishment." Science **305**: 1254-1258.

de Ruiter, M. B., D. J. Veltman, A. E. Goudriaan, J. Oosterlaan, Z. Sjoerds and W. van den Brink (2008). "Response Perseveration and Ventral Prefrontal Sensitivity to Reward and Punishment in Male Problem Gamblers and Smokers." Neuropsychopharmacology **34**(4): 1027-1038.

De Wilde, B., A. E. Goudriaan, B. Sabbe, W. Hulstijn and G. Dom (2013). "Relapse in pathological gamblers: A pilot study on the predictive value of different impulsivity measures." Journal of Behavioral Addictions **2**: 23-30.

Del Guercio, D., J. Reuter and P. A. Tkac (2010). "Broker incentives and mutual fund market segmentation." NBER working paper No. 16312.

Delgado, M. R., R. H. Frank and E. A. Phelps (2005a). "Perceptions of moral character modulate the neural systems of reward during the trust game." Nature Neuroscience **8**: 1611-1618.

Delgado, M. R., M. M. Miller, S. Inatia and E. A. Phelps (2005b). "An fMRI study of reward-related probability learning." Neuroimage **24**: 862-873.

Derbyshire, S. W. G., A. K. P. Jones, F. Gyulai, S. Clark, D. Townsend and L. L. Firestone (1997). "Pain processing during three levels of noxious stimulation produces differential patterns of central activity." Pain **73**(3): 431-445.

Devenow, A. and I. Welch (1996). "Rational herding in financial economics." European Economic Review **40**(3,Äì5): 603-615.

Dias, R., T. W. Robbins and A. C. Roberts (1996). "Dissociation in prefrontal cortex of affective and attentional shifts." Nature **380**(6569): 69-72.

Dias, R., T. W. Robbins and A. C. Roberts (1997). "Dissociable forms of inhibitory control within prefrontal cortex with an analog of the Wisconsin Card Sort Test: Restriction to novel situations and independence from "on-line" processing." The Journal of Neuroscience **17**: 9285-9297.

Dickerson, M., J. Hinchy and J. Fabre (1987). "Chasing, Arousal and Sensation Seeking in Off-course Gamblers." British Journal of Addiction **82**(6): 673-680.

Dillen, J. and M. R. Dixon (2008). "The impact of jackpot and near-miss magnitude on rate and subjective probability of slot machine gamblers." Analysis of Gambling Behavior **2**: 121-134.

Dixon, M. R. and J. E. Schreiber (2004). "Near-miss effects on response latencies and win estimations of slot machine players." Psychological Record **54**: 335-348.

Drewe, E. A. (1975). "Go- no go learning after frontal lobe lesions in humans." Cortex **11**(1): 8-16.

Dymond, S., N. S. Lawrence, B. T. Dunkley, K. S. L. Yuen, E. C. Hinton, M. R. Dixon, W. M. Cox, A. E. Hoon, A. Munnelly, S. D. Muthukumaraswamy and K. D. Singh (2014). "Almost winning: Induced MEG theta power in insula and orbitofrontal cortex increases during gambling near-misses and is associated with BOLD signal and gambling severity." Neuroimage(0).

Eisen, S. A., W. S. Slutske, M. J. Lyons, J. Lassman, H. Xian, R. Toomey, S. Chantarujikapong and M. T. Tsuang (2001). "THe genetics of pathological gambling." Seminars in Clinical Neuropsychiatry **6**: 195-204.

Elliott, R. and R. J. Dolan (1998). "Activation of different anterior cingulate foci in association with hypothesis testing ad response selection." Neuroimage **8**: 17-29.

Engelmann, J. B., C. M. Capra, C. Noussair and G. S. Berns (2009). "Expert Financial Advice Neurobiologically Offloads Financial Decision-Making under Risk." PLOS ONE **4**(3): e4957.

Engelmann, J. B., S. Moore, C. Monica Capra and G. S. Berns (2012). "Differential neurobiological effects of expert advice on risky choice in adolescents and adults." Social Cognitive and Affective Neuroscience **7**(5): 557-567.

Eriksen, B. A. and C. W. Eriksen (1974). "Effects of noise letters upon identification of atarget letter in a non-search task." Perception and Psychophysics **16**: 143-149.

Ernst, M., K. Bolla, M. Mouratidis, C. Contoreggi, J. A. Matochik, V. Kurian, J.-L. Cadet, A. S. Kimes and E. D. London (2002). "Decision-making in a Risk-taking Task: A PET Study." Neuropsychopharmacology **26**(5): 682-691.

Eslinger, P. J. and A. Damasio (1985). "Severe disturbance of higher cognition after bilateral frontal lobe ablation: Patient EVR." Neurology **35**(12): 1731-1741.

Everitt, B. J., J. A. Parkinson, M. C. Olmstead, M. Arroyo, P. Robledo and T. W. Robbins (1999). "Associative Processes in Addiction and Reward The Role of Amygdala-Ventral Striatal Subsystems." Annals of the New York Academy of Sciences **877**(1): 412-438.

Everitt, B. J. and T. W. Robbins (2005). "Neural systems of reinforcement for drug addiction: from actions to habits to compulsion." Nature Neuroscience **8**(11): 1481-1489.

Eysenck, S. B. and H. J. Eysenck (1977). "The place of impulsivness in a dimensional system of personality description." British Journal of Social and Clinical Psychology **16**: 57-68.

Fehr, E. and C. F. Camerer (2007). "Social neuroeconomics: The neural circuitry of social preferences." Trends in Cognitive Sciences **11**: 419-427.

Fehr, E. and S. Gachter (2002). "Altruistic punishment in humans." Nature **415**(6868): 137-140.

Feldstein Ewing, S. W., F. M. Filbey, C. S. Hendershot, A. D. McEachern and K. Hutchison (2011). "Proposed model of the neurobiological machanisms underlying pyschosocial alcohol interventaions: The example of motivational interviewing." Journal of Studies on Alcohol and Drugs **72**: 903-916.

Felitti, V. J., R. F. Anda, D. Nordenberg, D. F. Williamson, A. M. Spitz, E. V., M. P. Koss and J. S. Marks (1998). "Relationship of childhood abuse and household

dysfunction to many of the leading causes of death in adults: The adverse childhood experiences (ACE) study." American Journal of Preventive Medicine **14**: 245-258.

Ferris, J. and H. Wynne (2001). The Canadian Problem Gambling Index: Final Report. Ottawa, Canadian Centre on Substance Abuse.

Fiske, S. T. and S. L. Neuberg (1990). A continuum of impression formation: Influences of information and motivation on attention and interpretation. Advances in experimental social psychology. M. P. Zanna. New York, Academic Press. **23:** 1-74.

FitzGerald, T. H. B., B. Seymour and R. J. Dolan (2009). "The Role of Human Orbitofrontal Cortex Value Comparison for Incommensurable Objects." Journal of Neuroscience **29**(26): 8388-8395.

Forbes, E. E., J. Christopher May, G. J. Siegle, C. D. Ladouceur, N. D. Ryan, C. S. Carter, B. Birmaher, D. A. Axelson and R. E. Dahl (2006). "Reward-related decision-making in pediatric major depressive disorder: an fMRI study." Journal of Child Psychology and Psychiatry **47**(10): 1031-1040.

Forbush, K. T., M. Shaw, M. A. Graeber, L. Hovick, V. J. Meyer, D. J. Moser, J. Bayless, D. Watson and D. W. Black (2008). "Neuropsychological characterisitics and personality traits in pathological gambling." CNS Spectrums **13**: 306-315.

Fox, M. W. (1974). Concepts in ethology: Animal and human behavior. Minneapolis, University of Minnesota Press.

French, K. (2008). "American finance association presidential address: The cost of active investing." Journal of Finance **63**: 1537-1573.

Fuentes, D., H. Tavares, R. Artes and C. Gorenstein (2006). "Self-reported and neuropsychological measures of impulsivity in pathological gambling." Journal of the International Neuropsychological Society **12**: 907-912.

Fukui, H., T. Murai, H. Fukuyama, T. Hayashi and T. Hanakawa (2005). "Functional activity related to risk anticipation during performance of the Iowa gambling task." Neuroimage **24**(1): 253-259.

Garavan, H., T. J. Ross and E. A. Stein (1999). "Right hemispheric dominance of inhibitory control: An event-related functional MRI study." Proceedings of the National Academy of Sciences of the United States of America **96**(14): 8301-8306.

Gennaioli, N., A. Shleifer and R. W. Vishny (2012). "Money doctors." NBER working paper 18174.

George, M. S., R. F. Anton, C. Bloomer and et al. (2001). "ACtivation of prefrontal cortex and anterior thalamus in alcoholic subjects on exposure to alcohol-specific cues." Archives of General Psychiatry **58**(4): 345-352.

Gigerenzer, G. and D. G. Goldstein (1996). "Reasoning the fast and frugal way: Models of bounded rationalilty." Psychological Review **103**: 650-669.

Goel, V. and R. J. Dolan (2000). "Anatomical Segregation of Component Processes in an Inductive Inference Task." Journal of Cognitive Neuroscience **12**(1): 110-119.

Goel, V. and J. Grafman (2000). "Role of the right prefrontal cortex in ill-structured planning." Cognitive Neuropsychology **17**(5): 415-436.

Goel, V., J. Grafman, J. Tajik, S. Gana and D. Danto (1998). "A study of the performance of patients with frontal lobe lesions in a financial planning task." Brain **120**: 1805-1822.

Goldberg, E., R. Harner, M. Lovell, K. Podell and S. Riggio (1994). "Cognitive Bias, Functional Cortical Geometry, and the Frontal Lobes: Laterality, Sex, and Handedness." Journal of Cognitive Neuroscience **6**(3): 276-296.

Goldman-Rakic, P. S. (1992). "Working memory and the mind." Scientific American **267**: 111-117.

Goldstein, R. Z. and N. D. Volkow (2002). "Drug addiction and its underlying neurobiological basis: Neuroimaging evidence for the involvement of the frontal cortex." American Journal of Psychiatry **159**: 1642-1652.

Gooding, P. and N. Tarrier (2009). "A systematic review and meta-analysis of cognitive-behavioural interventions to reduce problem gambling: Hedging our bets?" Behaviour Research and Therapy **47**(7): 592-607.

Gotham, A. M., R. G. Brown and C. D. Marsden (1988). "'Frontal' cognitive function in patients with Parkinson's disease 'on'and 'off' levodopa." Brain **111**(2): 299-321.

Goudriaan, A. E., M. B. De Ruiter, W. Van Den Brink, J. Oosterlaan and D. J. Veltman (2010). "Brain activation patterns associated with cue reactivity and craving in abstinent problem gamblers, heavy smokers and healthy controls: an fMRI study." Addiction biology **15**(4): 491-503.

Goudriaan, A. E., J. Oosterlaan, E. de Beurs and W. can den Brink (2005). "Decision making in pathological gambling: A compariosn between pathological gamblers, alcohol dependents, persons with Tourette syndrome, and normal controls." Brain Research. Cognitive brain research **23**: 137-151.

Grabenhorst, F. and E. T. Rolls (2011). "Value, pleasure and choice in the ventral prefrontal cortex." Trends in Cognitive Sciences **15**(2): 56-67.

Grable, J. and R. H. Lytton (1999). "Financial risk tolerance revisited: The development of a risk assessment instrument." Financial Services Review **8**: 163-181.

Grant, D. A. and E. A. Berg (1948). "A behavioral analysis of degree of reinforcement and ease of shifting to new responses in a Weigel-type card-sorting problem." Journal of Experimental Psychology **38**: 404-411.

Grant, J. E., B. L. Odlaug and L. R. N. Schreiber (2014). "Pharmacological treatments in pathological gambling." British Journal of Clinical Pharmacology **77**(2): 375-381.

Greenberg, J. L., S. E. Lewis and D. K. Dodd (1999). "Overlapping addictions and self-esteem among college men and women." Addictive Behaviors **24**(4): 565-571.

Griffiths, M. (1993). "Tolerance in gambling: An objective measure using the psychophysiological analysis of male fruit machine gamblers." Addictive Behaviors **18**(3): 365-372.

Gruber, M. J. (1996). "Another puzzle: The growth in actively managed mutual funds." Journal of Finance **51**: 783-810.

Grüsser, S., J. Wrase, S. Klein, D. Hermann, M. Smolka, M. Ruf, W. Weber-Fahr, H. Flor, K. Mann, D. Braus and A. Heinz (2004). "Cue-induced activation of the striatum and medial prefrontal cortex is associated with subsequent relapse in abstinent alcoholics." Psychopharmacology **175**(3): 296-302.

Hampshire, A. and A. M. Owen (2006). "Fractionating Attentional Control Using Event-Related fMRI." Cerebral Cortex **16**(12): 1679-1689.

Hanoch, Y., J. G. Johnson and A. Wilke (2006). "Domain Specificity in Experimental Measures and Participant Recruitment: An Application to Risk-Taking Behavior." Psychological Science **17**(4): 300-304.

Hargreaves, I. S., M. White, P. M. Pexman, D. Pittman and B. G. Goodyear (2012). "The question shapes the answer: The neural correlates of task differences reveal dynamic semantic processing." Brain and Language **120**(1): 73-78.

Harrison, G. W. (1994). "Expected utility theory and the experimentalists." Empirical Economics **19**: 223-253.

Haruno, M. and M. Kawato (2006). "Different Neural Correlates of Reward Expectation and Reward Expectation Error in the Putamen and Caudate Nucleus During Stimulus-Action-Reward Association Learning." Journal of Neurophysiology **95**(2): 948-959.

Heinz, A., J. Wrase, T. Kahnt, A. Beck, Z. Bromand, S. M. Grüsser, T. Kienast, M. N. Smolka, H. Flor and K. Mann (2007). "Brain Activation Elicited by Affectively Positive Stimuli Is Associated With a Lower Risk of Relapse in Detoxified Alcoholic Subjects." Alcoholism: Clinical and Experimental Research **31**(7): 1138-1147.

Hikosaka, K. and M. Watanabe (2000). "Delay Activity of Orbital and Lateral Prefrontal Neurons of the Monkey Varying with Different Rewards." Cerebral Cortex **10**(3): 263-271.

Hodgins, D. C., L. E. Ching and J. McEwen (2009). "Strength of commitment language in motivational interviewing and gambling outcomes." Psychology of Addictive Behaviors **23**(1): 122-130.

Hodgins, D. C. and N. Peden (2008). "Cognitive-behavioral treatment for impulse control disorders." Revista Brasileira de Psiquiatria **30**: S31-S40.

Hofmann, W., M. Friese and F. Strack (2009). "Impulse and self-control from dual-systems perspective." Perspectives on Psychological Sciences **4**: 162-176.

Hofmann, W., M. Friese and R. W. Wiers (2008). "Impulsive versus reflective influences on health beahvior: A theoretical framework and empirical review." Health Psychology Review **2**: 111-137.

Hollander, E., S. Pallanti, N. B. Rossi, E. Sood, B. R. Baker and M. S. Buchsbaum (2005). "Imaging monetary reward in pathological gamblers." The World Journal of Biological Psychiatry **6**(2): 113-120.

Hsu, M., M. Bhatt, R. Adolphs, D. Tranel and C. F. Camerer (2005). "Neural systems responding to degress of uncertainty in human decision-making." Science **310**: 1680-1683.

Huettel, S. A., A. W. Song and G. McCarthy (2004). Functional magnetic resonance imaging. Basingstoke: Palgrace, Sunderland, Mass, Sinauer Associates.

Hunt, M. K., D. R. Hopko, R. Bare, C. W. Lejuez and E. V. Robinson (2005). "Construct Validity of the Balloon Analog Risk Task (BART): Associations With Psychopathy and Impulsivity." Assessment **12**(4): 416-428.

Inderst, R. and M. Ottaviani (2012). "Financial advice." Journal of Economic Literature **50**(494-512).

Iverson, S. D. and M. Mishkin (1970). "Perseverative interference in moneky following selective lesions of the inferior prefrontal convexity." Experimental Brain Research **11**: 367-386.

Izquierdo, A. and E. A. Murray (2004). "Combined unilateral lesions of the amygdala and orbital prefrontal cortex impair affective processing in rhesus monkeys." Journal of Neurophysiology **91**(5): 2023-2039.

Izuma, K., D. N. Saito and N. Sadato (2008). "Processing of social and monetary rewards in the human striatum." Neuron **58**: 284-294.

Izuma, K., D. N. Saito and N. Sadato (2009). "Processing of the incentive for social approval in the ventral striatum during charitable donation." Journal of Cognitive Neuroscience **22**: 621-631.

Jacobs, D. F. (1993). Evidence supporting a general theory of addiction. Gambling Behavior and Problem Gambling. W. R. Eadington and J. A. Cornelius**:** 287-294.

Jensen, M. (1968). "The performance of mutual funds in the period 1945-1964." Journal of Finance **23**: 389-416.

Johnson, S. C., L. C. Baxter, L. S. Wilder, J. G. Pipe, J. E. Heiserman and G. P. Prigatano (2002). "Neural correlates of self‐reflection." Brain **125**(8): 1808-1814.

Jonides, J., E. E. Smith, R. A. Koeppe, E. Awh, S. Minoshima and M. A. Mintun (1993). "Spatial working memory in humans as revealed by PET." Nature **363**: 623-625.

Kable, J. W. and P. W. Glimcher (2007). "The neural correlates of subjective value during intertemporal choice." Nature Neuroscience **10**(12): 1625-1633.

Kable, J. W. and P. W. Glimcher (2009). "The Neurobiology of Decision: Consensus and Controversy." Neuron **63**(6): 733-745.

Kahneman, D., P. Slovic and A. Tversky (1982). Judgement under uncertainty: Heuristics and biases. New York, Cambridge University Press.

Kahneman, D. and A. Tversky (1979). "Prospect theory: An analysis of decision under risk." Econometrica **47**: 263-291.

Kahneman, D. and A. Tversky (2011). Thinking, fast and slow, Macmillan.

Kalivas, P. W. and N. D. Volkow (2005). "The neural basis of addiction: A pathology of motivation and choice." American Journal of Psychiatry **162**: 1403-1413.

Karjbich, I., R. Adolphs, D. Tranel, N. Denburg and C. F. Camerer (2009). "Economic games quantify diminished sense of guilt in patients with damage to the prefrontal cortex." Journal of Neuroscience **29**: 2188-2193.

Kassinove, J. I. and M. L. Schare (2001). "Effects of the "near miss" and the "big win" on persistence at slot machine gambling." Psychology of Addictive Behaviors **15**(2): 155-158.

Kertzman, S., H. Lidogoster, A. Aizer, M. Kotler and P. N. Dannon (2011). "Risk-taking decisions in pathological gamblers is not a result of their impaired inhibition ability." Psychiatry Research **188**(1): 71-77.

Kertzman, S., K. Lowengrub, A. Aizer, M. Vainder, M. Kotler and P. N. Dannon (2008). "Go–no-go performance in pathological gamblers." Psychiatry Research **161**(1): 1-10.

Keynes, J. M. (1936). The State of Long-term Expectation. General Theory of Employment, Interest and Money, Macmillan.

Kim, K. K., P. Karunanayaka, M. D. Privitera, S. K. Holland and J. P. Szaflarski (2011). "Semantic association investigated with functional MRI and independent component analysis." Epilepsy & Behavior **20**(4): 613-622.

King-Casas, B., D. Tomlin, C. Anen, C. F. Camerer, S. R. Quartz and P. R. Montague (2005). "Getting to Know You: Reputation and Trust in a Two-Person Economic Exchange." Science **308**(5718): 78-83.

Klucharev, V., K. Hytönen, M. Rijpkema, A. Smidts and G. Fernández (2009). "Reinforcement Learning Signal Predicts Social Conformity." Neuron **61**(1): 140-151.

Knutson, B. and J. C. Cooper (2005). "Functional magnetic resonance imaging of reward prediction." Current Opinion in Neurology **18**(4): 411-417.

Knutson, B., G. W. Fong, C. M. Adams, J. L. Varner and D. Hommer (2001). "Dissociation of reward anticipation and outcome with event-related fMRI." NeuroReport **12**(17): 3683-3687.

Knutson, B., G. W. Fong, S. M. Bennett, C. M. Adams and D. Hommer (2003). "A region of mesial prefrontal cortex tracks monetarily rewarding outcomes: characterization with rapid event-related fMRI." Neuroimage **18**(2): 263-272.

Koechlin, E. and A. Hyafil (2007). "Anterior Prefrontal Function and the Limits of Human Decision-Making." Science **318**(5850): 594-598.

Krawczyk, D. C. (2002a). "Contributions of the prefrontal cortex to the neural basis of human decision making." Neuroscience & Biobehavioral Reviews **26**(6): 631-664.

Krawczyk, D. C. (2002b). "Contributions of the prefrontal cortex to the neural basis of human decision making." Neurosci. Biobehav. R. **26**: 631-664.

Krueger, F., J. Grafman and K. McCabe (2008). "Neural correlates of economic game playing." Philosophical Transactions of the Royal Society B: Biological Sciences **363**: 3859-3874.

Kuhnen, C. M. and B. Knutson (2005). "The neural basis of financial risk taking." Neuron **47**: 763-770.

Kusyszyn, H. R. and R. Rutter (1985). "Personality characteristics of male heavy gamblers, light gamblers, non-gamblers, and lottery players." Journal of Gambling Behavior **1**: 59-63.

Labudda, K., O. T. Wolf, H. J. Markowitsch and M. Brand (2007). "Decision-making and neuroendocrine responses in pathological gamblers." Psychiatry Research **153**(3): 233-243.

Ladouceur, R. and S. Lachance (2007a). Overcoming pathological gambling: Therapist guide. New York, NY, Oxford University Press.

Ladouceur, R. and S. Lachance (2007b). Overcoming your patholgoical gambling: Workbook. New York, NY, Oxford University Press.

Lamont, O. A. (2002). "Macroeconomic forecasts and microeconomic forecasters." Journal of Economic Behavior & Organization **48**(3): 265-280.

Leiserson, V. and R. Pihl (2007). "Reward-Sensitivity, Inhibition of Reward-Seeking, and Dorsolateral Prefrontal Working Memory Function in Problem Gamblers not in Treatment." Journal of Gambling Studies **23**(4): 435-455.

Lejuez, C. W., W. Aklin, S. Daughters, M. Zvolensky, C. Kahler and M. Gwadz (2007). "Reliability and Validity of the Youth Version of the Balloon Analogue Risk Task (BART–Y) in the Assessment of Risk-Taking Behavior Among Inner-City Adolescents." Journal of Clinical Child & Adolescent Psychology **36**(1): 106-111.

Lejuez, C. W., W. M. Aklin, H. A. Jones, J. B. Richards, D. R. Strong, C. W. Kahler and J. P. Read (2003a). "The balloon analogue risk task (BART) differentiates smokers and nonsmokers." Experimental and Clinical Psychopharmacology **11**(1): 26-33.

Lejuez, C. W., W. M. Aklin, M. J. Zvolensky and C. M. Pedulla (2003b). "Evaluation of the Balloon Analogue Risk Task (BART) as a predictor of adolescent real-world risk-taking behaviours." Journal of Adolescence **26**(4): 475-479.

Lejuez, C. W., J. P. Read, C. W. Kahler, J. B. Richards, S. E. Ramsey, G. I. Stuart, D. R. Strong and R. A. Brown (2002a). "Evaluation of a behavioral measure of risk taking: The balloon analogue risk task (BART)." Journal of Experimental Psychology: Applied **8**: 75-84.

Lejuez, C. W., J. P. Read, C. W. Kahler, J. B. Richards, S. E. Ramsey, G. L. Stuart, D. R. Strong and R. A. Brown (2002b). "Evaluation of a behavioral measure of risk taking: The Balloon Analogue Risk Task (BART)." Journal of Experimental Psychology: Applied **8**(2): 75-84.

Lesieur, H. and R. Rosenthal (1991). "Pathological gambling: A review of the literature (prepared for the American Psychiatric Association task force on DSM-IV committee on disorders of impulse control not elsewhere classified)." Journal of Gambling Studies **7**(1): 5-39.

Leung, K. S. and L. B. Cottler (2009). "Treatment of pathological gambling." Current Opinion on Psychiatry **22**: 69-74.

Levinson, P. K., D. R. Gernstein and D. R. Maloff (1983). Commonalities in substance abuse and habitual behaviors. Lexington, MA, Lexington Books.

Levy, D. J. and P. W. Glimcher (2011). "Comparing Apples and Oranges: Using Reward-Specific and Reward-General Subjective Value Representation in the Brain." Journal of Neuroscience **31**: 14693-14707.

Levy, D. J. and P. W. Glimcher (2012). "The root of all value: a neural common currency for choice." Current Opinion in Neurobiology **22**(6): 1027-1038.

Li, J., E. Xiao, D. Houser and P. R. Montague (2009). "Neural responses to sanction threats in two-party economic exchange." Proceedings of the National Academy of Sciences.

Li, X., Z.-L. Lu, A. D'Argembeau, M. Ng and A. Bechara (2010). "The Iowa Gambling Task in fMRI images." Human Brain Mapping **31**(3): 410-423.

Linnet, J., A. Møller, E. Peterson, A. Gjedde and D. Doudet (2011a). "Dopamine release in ventral striatum during Iowa Gambling Task performance is associated with increased excitement levels in pathological gambling." Addiction **106**(2): 383-390.

Linnet, J., A. MØLler, E. Peterson, A. Gjedde and D. Doudet (2011b). "Inverse association between dopaminergic neurotransmission and Iowa Gambling Task performance in pathological gamblers and healthy controls." Scandinavian Journal of Psychology **52**(1): 28-34.

Linnet, J., E. Peterson, D. J. Doudet, A. Gjedde and A. Møller (2010). "Dopamine release in ventral striatum of pathological gamblers losing money." Acta Psychiatrica Scandinavica **122**(4): 326-333.

Loh, R. and R. Stulz (2011). "When are analyst recommendation changes influential?" Review of Financial Studies **24**: 593.

Low, G., D. Jones, A. MaCleod, M. Power and C. Duggan (2000). "Childhood trauma, dissociation and self-harming behaviour: A pilot study." British Journal of Medical Psychology **73**(2): 269-278.

Loxton, N. J., D. Nguyen, L. Casey and S. Dawe (2008). "Reward drive, rash impulsivity and punishment sensitivity in problem gamblers." Personality and Individual Differences **45**(2): 167-173.

Luengo, M. A., M. T. Carrillo-de-la-Pena, J. M. Otero and E. Romero (1994). "A shotr-term longitudinal study of impulsivity and antisocial beahvior." Journal of Personality and Social Psychology **66**: 542-548.

Lun, J., S. Sinclair, E. R. Whitechurch and C. Glenn (2007). "Why do I think what you think? Epistemic social tuning and implicit prejudice." Journal of Personality and Social Psychology **93**: 957-972.

Lundstrom, B. N., K. M. Petersson, J. Andersson, M. Johansson, P. Fransson and M. Ingvar (2003). "Isolating the retrieval of imagined pictures during episodic memory: activation of the left precuneus and left prefrontal cortex." Neuroimage **20**(4): 1934-1943.

Luu, P. and S. M. Pederson (2004). The anterior cingulate cortex: Regulating actions in context. Cognitive neuroscience of attention. M. I. Posner. New York, Guilford Press.

Maddock, R. J., A. S. Garrett and M. H. Buonocore (2003). "Posterior cingulate cortex activation by emotional words: fMRI evidence from a valence decision task." Human Brain Mapping **18**(1): 30-41.

Malkiel, B. G. (2003). A Random Walk Down Wall Street, Norton.

Manes, F., B. Sahakian, L. Clark, R. Rogers, N. Antoun, M. Aitken and T. Robbins (2002). "Decision‐making processes following damage to the prefrontal cortex." Brain **125**(3): 624-639.

Marazziti, D., L. Dell'Osso, C. Conversano, G. Consoli, L. Vivarelli, F. Mungai, E. Di Nasso and F. Golia (2008). "Executive function abnormalities in pathological gamblers." Clinical Practive and Epidemiology in Mental Health **4**: 7.

McClure, S. M., M. K. York and P. R. Montague (2004). "The Neural Substrates of Reward Processing in Humans: The Modern Role of fMRI." The Neuroscientist **10**(3): 260-268.

McConaghy, N., A. Blaszczynski and A. Frankova (1991). "Comparison of imaginal desensitisation with other behavioural treatments of pathological gambling. A two- to nine-year follow-up." The British Journal of Psychiatry **159**(3): 390-393.

McCormick, R. A., J. Taber, N. Kruedelbach and A. Russo (1987). "Personality profiles of hospitalized pathological gamblers: The California Personality Invertory." Journal of Clinical Psychology **43**(5): 521-527.

Meshi, D., G. Biele, C. W. Korn and H. R. Heekeren (2012). "How expert advice influences decision making." PLOS ONE **7**(11): e49748.

Meyer, G., B. P. Hauffa, M. Schedlowski, C. Pawlak, M. A. Stadler and M. S. Exton (2000). "Casino gambling increases heart rate and salivary cortisol in regular gamblers." Biological Psychiatry **48**(9): 948-953.

Miedl, S. F., J. Peters and C. Büchel (2012). "ALtered neural reward representations in pathological gamblers revealed by delay and probability discounting." Archives of General Psychiatry **69**(2): 177-186.

Milgram, S. (1963). "Behavioral study of obedience." Journal of Abnormal & Social Psychology **67**(4): 371-378.

Milgram, S. (1974). Obedience to authority: An experimental view. New York, Harpercollins.

Miller, E. K. and J. D. Cohen (2001). "An integrative theory of prefrontal cortex function." Annual Review of Neuroscience **24**: 167-202.

Miller, W. R. (1996). "Motivational interviewing: Research, practice, and puzzles." Addictive Behaviors **21**(6): 835-842.

Miller, W. R., C. E. Yahne and J. S. Tonigan (2003). "Motivational interviewing in drug abuse services: A randomized trial." Journal of Consulting and Clinical Psychology **71**(4): 754-763.

Milner, B. (1963). "Effects of different brain lesions on card sorting: The role of the frontal lobes." Archives of Neurology **9**(1): 90-00.

Mishra, S., M. L. Lalumière and R. J. Williams (2010). "Gambling as a form of risk-taking: Individual differences in personality, risk-accepting attitudes, and behavioral preferences for risk." Personality and Individual Differences **49**(6): 616-621.

Monchi, O., M. Petrides, V. Petre, K. Worsley and A. Dagher (2001). "Wisconsinc Card Sorting revisitied: Distinct neural circuits participating in different stages of the task identified by event-related functional magnetic resonance imaging." The Journal of Neuroscience **21**: 7733-7741.

Moran, E. (1970). "Varieties of pathological gambling." British Journal of Psychiatry **116**: 593-597.

Murray, E. A. and A. Izquierdo (2007). "Orbitofrontal Cortex and Amygdala Contributions to Affect and Action in Primates." Annals of the New York Academy of Sciences **1121**(1): 273-296.

Nagahama, Y., H. Fukuyama, H. Yamauchi, S. Matsuzaki, J. Konishi, H. Shibasaki and J. Kimura (1996). "Cerebral activation during performance of a card sorting test." Brain **119**(5): 1667-1675.

Nagahama, Y., T. Okada, Y. Katsumi, T. Hayashi, H. Yamauchi, C. Oyanagi, J. Konishi, H. Fukuyama and H. Shibasaki (2001). "Dissociable Mechanisms of Attentional Control within the Human Prefrontal Cortex." Cerebral Cortex **11**(1): 85-92.

Nash, J. F. (1950). "Equilibrium points in n-person games." PNAS **36**: 48-49.

Nelson, H. (1976). "A modified card sorting response sensitive to frontal lobe defects." Cortex **12**: 313-324.

Niemz, K., M. Griffiths and P. Banyard (2005). "Prevalence of pathological internet use among university students and correlations with self-esteem, the general health questionnaire (GHQ), and disinhibition." CyberPsychology & Behavior **8**: 562-570.

Nower, L., J. L. Derevensky and R. Gupta (2004). "The Relationship of Impulsivity, Sensation Seeking, Coping, and Substance Use in Youth Gamblers." Psychology of Addictive Behaviors **18**(1): 49-55.

O'Doherty, J., P. Dayan, J. Schultz, R. Deichmann, K. Friston and R. J. Dolan (2004). "Dissociable Roles of Ventral and Dorsal Striatum in Instrumental Conditioning." Science **304**(5669): 452-454.

O'Doherty, J. P., R. Deichmann, H. D. Critchley and R. J. Dolan (2002). "Neural Responses during Anticipation of a Primary Taste Reward." Neuron **33**(5): 815-826.

Padoa-Schioppa, C. (2011). "Neurobiology of Economic Choice: A Good-Based Model." Annual Review of Neuroscience **34**(1): 333-359.

Parke, J. and M. Griffiths (2006). "The psychology of the fruit machineL The role of structural characterisitics (revisted)." International Journal of Mental Health and Addiction **4**: 151-179.

Patton, J. M., M. S. Stanford and E. S. Barratt (1995). "Factor structure of the Barratt Impulsiveness Scale." Journal of Clinical Psychology **51**: 768-774.

Paulus, M. P., J. S. Feinstein, D. Leland and A. N. Simmons (2005). "Superior temporal gyrus and insula provide response and outcome-dependent information during assessment and action selection in a decision-making situation." Neuroimage **25**(2): 607-615.

Paulus, M. P., J. S. Feinstein, A. Simmons and M. B. Stein (2004a). "Anterior cingulate activation in high trait anxious subjects is related to altered error processing during decision making." Biological Psychiatry **55**: 1179-1187.

Paulus, M. P., J. S. Feinstein, S. F. Tapert and T. T. Liu (2004b). "Trend detection via temporal difference model predicts inferior prefrontal cortex activation during acquisition of advantageous action selection." Neuroimage **21**(2): 733-743.

Paulus, M. P., C. Rogalsky, A. Simmons, J. S. Feinstein and M. B. Stein (2003). "Increased activation in the right insula during risk-taking decision making is related to harm avoidance and neuroticism." Neuroimage **19**(4): 1439-1448.

Peterson, E., A. Møller, D. J. Doudet, C. J. Bailey, K. V. Hansen, A. Rodell, J. Linnet and A. Gjedde (2010). "Pathological gambling: Relation of skin conductance response to dopaminergic neurotransmission and sensation-seeking." European Neuropsychopharmacology **20**(11): 766-775.

Petry, N. (2001a). "Substance abuse, pathological gambling, and impulsiveness." Drug and Alcohol Dependence **63**(1): 29-38.

Petry, N. (2002). "A Comparison of Young, Middle-Aged, and Older Adult Treatment-Seeking Pathological Gamblers." The Gerontologist **42**(1): 92-99.

Petry, N. (2005a). "Gamblers Anonymous and Cognitive-Behavioral Therapies for Pathological Gamblers." Journal of Gambling Studies **21**(1): 27-33.

Petry, N., C. Blanco, M. Auriacombe, G. Borges, K. Bucholz, T. Crowley, B. Grant, D. Hasin and C. O'Brien (2013). "An Overview of and Rationale for Changes Proposed for Pathological Gambling in DSM-5." Journal of Gambling Studies: 1-10.

Petry, N. and T. Casarella (1999). "Excessive discounting of delayed rewards in substance abusers with gambling problems." Drug and Alcohol Dependence **56**(1): 25-32.

Petry, N. M. (2001b). "Pathological gamblers, with and without substance abuse disorders, discount delayed rewards at high rates." Journal of Abnormal Psychology **110**(3): 482-487.

Petry, N. M. (2005b). Pathological gambling: Etiology, comorbidity, and treatment. Washington, DC, US, American Psychological Association.

Petry, N. M., W. K. Bickel and M. Arnett (1998). "Shortened time horizons and insensitivity to future consequences in heroin addicts." Addiction **93**(5): 729-738.

Phillips, M. L., W. C. Drevets, S. L. Rauch and R. Lane (2003). "Neurobiology of emotion perception I: The neural basis of normal emotion perception." Biological Psychiatry **54**(5): 504-514.

Pillutla, M. M. and J. K. Murnighan (1996). "Unfairness, Anger, and Spite: Emotional Rejections of Ultimatum Offers." Organizational Behavior and Human Decision Processes **68**(3): 208-224.

Pochon, J. B., J. Riis, A. G. Sanfey, L. E. Nystrom and J. D. Cohen (2008). "Functional imaging of decision conflict." Journal of Neuroscience **28**: 3468-3473.

Podell, K., M. Lovell, M. Zimmerman and E. Goldberg (1995). "The cognitive bias task and lateralized frontal lobe functions in males." The Journal of Neuropsychiatry and Clinical Neuroscience **7**: 491-501.

Potenza, M. N. (2008). "The neurobiology of pathological gambling and drug addiction: an overview and new findings." Philosophical Transactions of the Royal Society B: Biological Sciences **363**(1507): 3181-3189.

Potenza, M. N. (2013). "Neurobiology of gambling behaviors." Current Opinion in Neurobiology **23**(4): 660-667.

Potenza, M. N., I. M. Balodis, C. A. Franco, S. Bullock, J. Xu, T. Chung and J. E. Grant (2013). "Neurobiological considerations in understanding behavioural treatments for pathological gambling." Psychology of Addictive Behaviors **27**: 380-392.

Potenza, M. N., H. C. Leung, H. P. Blumberg, B. S. Peterson, R. K. Fulbright, C. M. Lacadie, P. Skudlarski and J. C. Gore (2003a). "An fMRI Stroop task study of ventromedial prefrontal cortical function in pathological gamblers." American Journal of Psychiatry **160**: 1990-1994.

Potenza, M. N., M. A. Steinberg, P. Skudlarski, R. K. Fulbright, C. M. Lacadie, M. K. Wilber, B. J. Rounsavile, J. C. Gore and B. E. Wexler (2003b). "Gambling urgers in pathological gambling: A functional magnetic resonance imaging study." Archives of General Psychiatry **60**: 828-836.

Power, Y., B. Goodyear and D. Crockford (2012). "Neural Correlates of Pathological Gamblers Preference for Immediate Rewards During the Iowa Gambling Task: An fMRI Study." Journal of Gambling Studies **28**(4): 623-636.

Prabhakaran, V., J. A. L. Smith, J. E. Desmond, G. H. Glover and J. D. E. Gabrieli (1997). "Neural Substrates of Fluid Reasoning: An fMRI Study of Neocortical Activation during Performance of the Raven's Progressive Matrices Test." Cognitive Psychology **33**(1): 43-63.

Rabin, M. (2003). "The Nobel memorial prize for Daniel Kahneman." Scandinavian Journal of Economics **105**: 157-180.

Rahman, A. S., C. E. Pilver, R. A. Desai, M. A. Steinberg, L. Rugle, S. Krishnan-Sarin and M. N. Potenza (2012). "The relationship between age of gambling onset and adolescent problematic gambling severity." Journal of Psychiatric Research **46**(5): 675-683.

Ramnani, N. and A. Owen (2004). "Anterior prefrontal cortex: Insights into function from anatomy and neuroimaging." Nature Reviews Neuroscience **5**: 184-194.

Rao, A. R. and K. B. Monroe (1989). "The effect of price, brand name, and store name on buyer's perceptions of product quality." Journal of Marketing Research **26**: 351-357.

Rao, H., M. Korczykowski, J. Pluta, A. Hoang and J. A. Detre (2008). "Neural correlates of voluntary and involuntary risk taking in the human brain: An fMRI Study of the Balloon Analog Risk Task (BART)." Neuroimage **42**(2): 902-910.

Redish, A. D., S. Jensen, A. Johnson and Z. Kurthnelson (2007). "Reconciling reinforcement learning models with behavioral extinction and renewal: Implications for addiction, relapse, and problem gambling." Psychological Review **114**: 784-805.

Reuter, J., T. Raedler, M. Rose, I. Hand, J. Glascher and C. Buchel (2005). "Pathological gambling is linked to reduced activation of the mesolimbic reward system." Nature Neuroscience **8**(2): 147-148.

Rilling, J. K., D. A. Gutman, T. R. Zeh, G. Pagnoni, G. S. Berns and C. D. Kilts (2002). "A Neural Basis for Social Cooperation." Neuron **35**(2): 395-405.

Rilling, J. K. and A. G. Sanfey (2011). "The Neuroscience of Social Decision-Making." Annual Review of Psychology **62**(1): 23-48.

Rilling, J. K., A. G. Sanfey, J. A. Aronson, L. E. Nystrom and J. D. Cohen (2004). "Opposing BOLD responses to reciprocated and unreciproated altruism in putative reward pathways." NeuroReport **15**: 2239-2243.

Robinson, T. E. and K. C. Berridge (1993). "The neural basis of drug craving: An incentive-sensitization theory of addiction." Brain Research Reviews **18**(3): 247-291.

Rogers, R., T. C. Andrews, P. M. Grasby, D. J. Brooks and T. W. Robbins (2000). "Contrasting cortical and subcortical activations produced by attentional-set shifting and reversal learning in humans." Journal of Cognitive Neuroscience **12**: 142-162.

Rolls, E. T. (1999). The neural basis of emotion. The brain and emotion. E. T. Rolls. New York, Oxford University Press**:** 112-137.

Rolls, E. T. (2000). "The Orbitofrontal Cortex and Reward." Cerebral Cortex **10**(3): 284-294.

Rolls, E. T., B. J. Everitt and A. Roberts (1996). "The Orbitofrontal Cortex [and Discussion]." Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences **351**(1346): 1433-1444.

Rolls, E. T. and F. Grabenhorst (2008). "The orbitofrontal cortex and beyond: From affect to decision-making." Progress in Neurobiology **86**(3): 216-244.

Rosenberg, M. (1965). Society and the adolescents self-image. Princeton NJ, Princeton University Press.

Rosenberg, O., L. K. Dinur and P. N. Dannon (2013). "Four-year follow-up study of pharmacological treatment in pathological gamblers." Clinical Neuropharmacology **36**: 42-45.

Rosenbloom, M., H., J. D. Schmahmann and B. H. Price (2012). "The functional neuroanatomy of decision-making." The Journal of Neuropsychiatry and Clinical Neurosciences **24**: 266-277.

Rosenthal, R. J. (1993). Some causes of pathological gambling. Gambling Behavior and Problem Gambling. W. R. Eadington and J. A. Cornelius**:** 143-148.

Rubia, K., A. B. Smith, M. J. Brammer and E. Taylor (2003). "Right inferior prefrontal cortex mediates response inhibition while mesial prefrontal cortex is responsible for error detection." Neuroimage **20**(1): 351-358.

Rugle, L. and L. Melamed (1993). "Neuropsychological Assessment of Attention Problems in Pathological Gamblers." The Journal of Nervous and Mental Disease **181**(2): 107-112.

Sanfey, A. G. (2007). "Social decision-making: Insights from game theory and neuroscience." Science **318**: 598-602.

Sanfey, A. G., J. K. Rilling, J. A. Aronson, L. E. Nystrom and J. D. Cohen (2003). "The Neural Basis of Economic Decision-Making in the Ultimatum Game." Science **300**(5626): 1755-1758.

Saver, J. L. and A. Damasio (1991). "Preserved access and processing of social knowledge in a patient with acquired sociopathy due to ventromedial frontal damage." Neuropsychologia **29**(12): 1241-1249.

Scharfstein, D. and J. Stein (1990). "Herd behavior and investment." American Economic Review **80**: 465-479.

Schmahmann, J. D. and D. N. Pandya (2008). "Disconnection syndromes of basal ganglia, thalamus and cerebrocerebellar systems." Cortex **44**: 1037-1066.

Schultz, W. (2002). "Getting Formal with Dopamine and Reward." Neuron **36**(2): 241-263.

Schultz, W., P. Dayan and P. R. Montague (1997). "A Neural Substrate of Prediction and Reward." Science **275**(5306): 1593-1599.

Shamay-Tsoory, S. G., J. Aharon-Peretz and D. Perry (2009). "Two systems for empathy: a double dissociation between emotional and cognitive empathy in inferior frontal gyrus versus ventromedial prefrontal lesions." Brain **132**(3): 617-627.

Sharpe, L. (2002). "A reformulated cognitive–behavioral model of problem gambling: A biopsychosocial perspective." Clinical Psychology Review **22**(1): 1-25.

Shimamura, A. (2000). "The role of the prefrontal cortex in dynamic filtering." Psychobiology **28**(2): 207-218.

Shleifer, A. (2000). Inefficient markets: An introduction to behavioral finance. Oxford and New York, Oxford University Press, Clarendon Lectures in Economics.

Sias, R. W. (2004). "Institutional herding." Review of Financial Studies **17**: 165-206.

Sienkiewicz-Jarosz, H., A. Scinska, W. Kuran, D. Ryglewicz, A. Rogowski, E. Wrobel, A. Korkosz, A. Kukwa, W. Kostowski and P. Bienkowski (2005). "Taste responses in patients with Parkinson's disease." Journal of Neurology, Neurosurgery & Psychiatry **76**(1): 40-46.

Simon, H. A. (1957). Models of man: Social and rational; Mathematical essays on rational human behavior in a social setting. New York, Wiley.

Simon, H. A., M. Egidi and R. L. Marris (1995). Economics, bounded rationality and the cognitive revolution. Aldershot, England; Brookfield, Vt., USA, E. Elgar.

Singh, S. and E. Ernst (2008). Trick or treatment? Alternative medicine on trial. New York, Bantam.

Sjoberg, L. and E. Engelberg (2009). "Attitudes to economic risk taking, sensation seeking, and values of business students specializing in finance." Journal of Behavioral Finance **10**: 33-43.

Smith, D. V., B. Y. Hayden, T. K. Truong, A. W. Song, M. L. Platt and S. A. Huettel (2010). "Distinct Value Signals in Anterior and Posterior Ventromedial Prefrontal Cortex." Journal of Neuroscience **30**: 2490-2495.

Smith, G. (2013). "The nature and scope of gambling in Canada." Addiction: n/a-n/a.

Smith, K. S., K. C. Berridge and J. W. Aldridge (2011). "Disentangling pleasure from incentive salience and learning signals in brain reward circuitry." Proceedings of the National Academy of Sciences **108**(27): E255–E264.

Somerville, L. H., T. F. Heatherton and W. M. Kelley (2006). "Anterior cingulate cortex responds differentially to expectancy violation and social rejection." Nature Neuroscience **9**(8): 1007-1008.

Specker, S. M., G. A. Carlson, G. A. Christension and M. Marcotte (1995). "Impulse control disorders and attention deficit disorder in pathological gamblers." Annals of Clinical Psychiatry **7**: 175-179.

Stacy, A. W. and R. W. Wiers (2010). "Implicit Cognition and Addiction: A Tool for Explaining Paradoxical Behavior." Annual Review of Clinical Psychology **6**(1): 551-575.

Steel, Z. and A. Blaszczynski (1998). "Impulsivity, personality disorders and pathological gambling severity." Addiction **93**(6): 895-905.

Stoodley, C. J. and J. D. Schmahmann (2009). "Functional topography in the human cerebellum: A meta-analysis of neuroimaging studies." Neuroimage **44**: 489-501.

Stout, J. C., W. C. Rodawalt and E. R. Siemers (2001). "Risky decision making in Huntington's disease." Journal of the International Neuropsychological Society **7**: 92-101.

Stuss, D. T., B. Levine, M. P. Alexander, J. Hong, C. Palumbo, L. Hamer, K. J. Murphy and D. Izukawa (2000). "Wisconsin Card Sorting Test performance in patients with focal frontal and posterior brain damage: effects of lesion location and test structure on separable cognitive processes." Neuropsychologia **38**(4): 388-402.

Surowiecki, J. (2004). The wisdom of crowds: Why the many are smarter than the few and how collective wisdom shapes business, economies, societies, and nations. London, Little Brown.

Svaldi, J., M. Brand and B. Tuschen-Caffer (2010). "Decision-making impairments in women with binge eating disorder." Appetite **54**: 84-92.

Sylvain, C., R. Ladouceur and J.-M. Boisvert (1997). "Cognitive and behavioral treatment of pathological gambling: A controlled study." Journal of Consulting and Clinical Psychology **65**(5): 727-732.

Talmi, D., P. Dayan, S. J. Kiebel, C. D. Frith and R. J. Dolan (2009). "How humans integrate the prospects of pain and reward during choice." Journal of Neuroscience **29**: 14617-14626.

Tanabe, J., L. Thompson, E. Claus, M. Dalwani, K. Hutchison and M. T. Banich (2007). "Prefrontal cortex activity is reduced in gambling and nongambling substance users during decision-making." Human Brain Mapping **28**(12): 1276-1286.

Tanaka, S. C., K. Doya, G. Okada, K. Ueda, Y. Okamoto and S. Yamawaki (2004). "Prediction of immediate and future rewards differentially recruits cortico-basal ganglia loops." Nature Neuroscience **7**(8): 887-893.

Tapert, S. F., G. G. Brown, M. V. Baratta and S. A. Brown (2004). "fMRI BOLD response to alcohol stimuli in alcohol dependent young women." Addictive Behaviors **29**(1): 33-50.

Tavares, H., M. L. Zilberman and N. el-Guebaly (2003). "Are there cognitive and behavioural approaches specific to the treatment of pathological gambling?" Canadian Journal of Psychiatry **48**: 22-27.

Taylor, J. G. and E. Balanovski (1979). "Is there any scientific explanaiton of the paranormal?" Nature **279**: 631-633.

Thanos, P. K., N. D. Volkow, P. Freimuth, H. Umegaki, H. Ikari, G. Roth, D. K. Ingram and R. Hitzemann (2001). "Overexpression of dopamine D2 receptors reduces alcohol self-administration." Journal of Neurochemistry **78**(5): 1094-1103.

Thiel, A., R. Hilker, J. Kessler, B. Habedank, K. Herholz and W. D. Heiss (2003). "Activation of basal ganglia loops in idiopathic Parkinson's disease: a PET study." Journal of Neural Transmission **110**(11): 1289-1301.

Tindell, A. J., K. S. Smith, K. C. Berridge and J. W. Aldridge (2009). "Dynamic computation of incentive salience: "Wanting" what was never "liked"." Journal of Neuroscience **29**: 12220-12228.

Tindell, A. J., K. S. Smith, S. Peciña, K. C. Berridge and J. W. Aldridge (2006). "Ventral Pallidum Firing Codes Hedonic Reward: When a Bad Taste Turns Good." Journal of Neurophysiology **96**(5): 2399-2409.

Todd, P. M. and G. Girgerenzer (2007). "Environments that make us smart." Current Directions in Psychological Science **16**: 167-171.

Toneatto, T., B. Brands and P. Selby (2009). "A randomized, double-blind, placebo-controlled trial of naltrexone in the treatment of concurrent alcohol use disorder and pathological gambling." American Journal on Addiction **18**: 219-225.

Trueman, B. (1994). "Analyst forecasts and herding behavior." Review of Financial Studies **7**: 97-124.

Tversky, A. and D. Kahneman (1974). "Judgment under uncertainty: Heuristics and biases." Science **185**(4157): 1124-1131.

Tversky, A. and D. Kahneman (1992). "Advances in prospect theory: Cumulative representation of uncertainty." Journal of Risk and Uncertainty **5**(4): 297-323.

Ullsperger, M. and D. Y. von Cramon (2001). "Subprocesses of performance monitoring: A dissociation of error processing and response competition revealed by event-related fMRI and ERPs." Neuroimage **14**: 1387-1401.

van den Bos, W., E. van Dijk, M. Westenberg, S. A. Rombouts and E. A. Crone (2009). "What motivates repayment? Neural correlates of reciprocity in the Trust Game." Social Cognitive and Affective Neuroscience **4**: 294-304.

van Holst, R. J., W. van den Brink, D. J. Veltman and A. E. Goudriaan (2010). "Why gamblers fail to win: A review of cognitive and neuroimaging findings in pathological gambling." Neuroscience & Biobehavioral Reviews **34**(1): 87-107.

Verdejo-Garcia, A., A. J. Lawrence and L. Clark (2008). "Impulsivity as a vulnerability marker for substance use disorders: Review of findings from high-risk research, problem gamblers and genetic addociation studies." Neuroscience & Biobehavioral Reviews **32**: 777-810.

Vitaro, F., L. Arseneault and R. E. Tremblay (1999). "Impulsivity predicts problem gambling in low SES adolescent males." Addiction **94**(4): 565-575.

Volberg, R. A. (1994). "The prevalence and demographics of pathological gamblers: Implications for public health." American Journal of Public Health **84**: 237-241.

Volberg, R. A., D. C. Reitzes and J. Boles (1997). "Exploring the links between gambling, problem gambling, and self‐esteem." Deviant Behavior **18**(4): 321-342.

Volkow, N. D., J. S. Fowler and G.-J. Wang (2002). "Role of dopamine in drug reinforcement and addiction in humans: results from imaging studies." Behavioural Pharmacology **13**(5-6): 355-366.

von Neumann, J. and O. Morgenstern (1947). Theory of Games and Economic Behavior. Princeton, NJ, Princeton University Press.

Waltz, J. A., B. J. Knowlton, K. J. Holyoak, K. B. Boone, F. S. Mishkin, M. de Menezes Santos, C. R. Thomas and B. L. Miller (1999). "A System for Relational Reasoning in Human Prefrontal Cortex." Psychological Science **10**(2): 119-125.

Wardle, H., K. Sproston, J. Orford, B. Erens, M. Griffiths and R. Constantine (2007). British Gambling Prevelance Survey. London, National Centre for Social Research.

Watson, P. C. (1960). "On the failure to eliminate hypothesies in a conceptual task." Quarterly Journal of Experimental Psychology **12**: 129-140.

Weber, E. U., A.-R. Blais and N. Betz (2002). "A domain-specific risk-attitude scale: Measuring risk perceptions and risk behaviors." Journal of Behavioral Decision Making **15**: 263-290.

Welch, I. (2000). "Herding among security analysts." Journal of Financial Economics **58**: 369.

Welte, J. W., G. M. Barnes, W. F. Wieczorek, M. C. Tidwell and J. Parker (2002). "Gambling participation in the U.S. - results from a national survey." Journal of Gambling Studies **18**: 313-337.

Whitney, K. A., P. S. Fastenau, J. D. Evans and P. H. Lysaker (2004). "Comparative neuropsychological function in obsessive-compulsive disorder and schizophrenia with and without obsessive-compulsive symptoms." Schizophrenia Research **69**: 75-83.

Wilcox, N. (1993). "Lottery choice, incentives, complexity and decision time." American Economic Journal **103**: 1397-1417.

Wise, R. A. (1985). "The anhedonia hypothesis: Mark III." Behavioral and Brain Sciences **8**(01): 178-186.

Yechiam, E., J. R. Busemeyer, J. C. Stout and A. Bechara (2005). "Using Cognitive Models to Map Relations Between Neuropsychological Disorders and Human Decision-Making Deficits." Psychological Science **16**(12): 973-978.

Yi, S. and V. Kanetkar (2010). "Implicit measures of attitudes toward gambling: An exploratory study." Journal of Gambling Issues: 140-163.

Yin, H. H. and B. J. Knowlton (2006). "The role of the basal ganglia in habit formation." Nature Reviews Neuroscience **7**(6): 464-476.

Yip, S. W., R. A. Desai, M. A. Steinberg, L. Rugle, D. A. Cavallo, S. Krishnan-Sarin and M. N. Potenza (2011). "Health/Functioning Characteristics, Gambling Behaviors, and Gambling-Related Motivations in Adolescents Stratified by Gambling Problem Severity: Findings from a High School Survey." The American Journal on Addictions **20**(6): 495-508.

Zack, M. and C. X. Poulos (2003). "Amphetamine Primes Motivation to Gamble and Gambling-Related Semantic Networks in Problem Gamblers." Neuropsychopharmacology **29**(1): 195-207.

Zack, M. and C. X. Poulos (2007). "A D2 Antagonist Enhances the Rewarding and Priming Effects of a Gambling Episode in Pathological Gamblers." Neuropsychopharmacology **32**(8): 1678-1686.

Appendix A. Screening questions for recruitment interview

Do you have any of the following conditions:	Yes	No
Schizophrenia	___	___
Mood disorder	___	___
Bipolar disorder	___	___
Depression	___	___
Personality disorder	___	___
Learning disability	___	___
Attention deficit/Hyperactivity disorder	___	___

Do you use recreational drugs (e.g.: marijuana, cocaine, heroin, amphetamines) on a regular basis?	___	___

**"Yes" to any of the above were grounds for exclusion in the study

Appendix B. MRI screening form (Male)

Alberta Health Services

Patient History and MRI Screening (Male)

Covenant Health

Name: _____ Hospital #: _____

The following items may interfere with your Magnetic Resonance Imaging examination, and some can be potentially hazardous.
Please indicate if you have the following:

Section 1

Yes	No	
☐	☐	Cardiac Pacemaker / Automatic Defibrillator
☐	☐	Aneurysm Clip(s)
☐	☐	Implanted Insulin Pump
☐	☐	Implanted Drug Infusion Device
☐	☐	Bone Growth or Bio Stimulator
☐	☐	Neurostimulator
☐	☐	Epicardial Leads
☐	☐	Cochlear Implant
☐	☐	Intra-vascular Coils
☐	☐	Swan-Ganz Catheter

Section 2

Yes	No	
☐	☐	Stents
☐	☐	Any type of surgical clip or staple(s)
☐	☐	Heart Valve Prosthesis
☐	☐	Vena Cava Filter
☐	☐	Middle Ear Implant
☐	☐	Penile Prosthesis
☐	☐	Eye Prosthesis
☐	☐	Shrapnel or Bullet
☐	☐	Magnetically operated devices
☐	☐	Wire Sutures
☐	☐	Silver impregnated dressing (Acticoat, Actisorb Plus, Aquacel)

Section 3

Yes	No	
☐	☐	Intraventricular Shunt
☐	☐	Intracranial Pressure Monitor
☐	☐	Wire Mesh
☐	☐	Artificial Limb or Joint
☐	☐	Any orthopedic item(s) (i.e. pins, rods, screws, nails, clips, plates, wire, etc.)
☐	☐	Dentures or any type of removable dental item
☐	☐	Hearing Aid
☐	☐	Tattoos
☐	☐	Body Piercings
☐	☐	Transdermal Patches (i.e. nicotine, nitroglycerine, etc.)

Yes	No	
☐	☐	**Worked as welder, lathe operator, sheet metal worker or any similar occupation that may result in a metallic foreign object in your eyes.**
☐	☐	**Have you ever had an endoscopy (gastroscopy/colonoscopy) procedure in 2011 or later and is there any possibility that you have a Metallic Clip**

Have you ever had any surgical procedure or operation? ☐ Yes ☐ No

Type _____ Year _____

Type _____ Year _____

Type _____ Year _____

Have you **EVER** had any metal fragments in your eyes, or had an injury to your eyes with metal? ☐ Yes ☐ No

Do you have a history of kidney failure or are you on kidney dialysis? ☐ Yes ☐ No

Patient Weight _____ lb / kg Patient Height _____ in / cm

I have answered the above questions to the best of my ability.
The MRI examination has been explained to me and I have had my questions answered to my satisfaction.

Signature of Patient or Guardian _____ Date _____

Witness / Technologist _____

CH-0186 Aug 2007

PAGE 1 OF 1

Appendix C. MRI screening form (Female)

Capital Health
EDMONTON AREA

Patient History and MRI Screening (Female)

CARITAS HEALTH GROUP

Name: _____ Hospital #: _____

The following items may interfere with your Magnetic Resonance Imaging examination, and some can be potentially hazardous.
Please indicate if you have the following:

Section 1

Yes	No	
☐	☐	Cardiac Pacemaker / Automatic Defibrillator
☐	☐	Aneurysm Clip(s)
☐	☐	Implanted Insulin Pump
☐	☐	Implanted Drug Infusion Device
☐	☐	Bone Growth or Bio Stimulator
☐	☐	Neurostimulator
☐	☐	Epicardial Leads
☐	☐	Cochlear Implant
☐	☐	Intra-vascular Coils
☐	☐	Swan-Ganz Catheter

Section 2

Yes	No	
☐	☐	Stents
☐	☐	Any type of surgical clip or staple(s)
☐	☐	Heart Valve Prosthesis
☐	☐	Vena Cava Filter
☐	☐	Middle Ear Implant
☐	☐	Eye Prosthesis
☐	☐	Shrapnel or Bullet
☐	☐	Magnetically operated devices
☐	☐	Wire Sutures
☐	☐	Silver impregnated dressing (Acticoat, Actisorb Plus, Aquacel)

Section 3

Yes	No	
☐	☐	Diaphragm or IUD
☐	☐	Intraventricular Shunt
☐	☐	Intracranial Pressure Monitor
☐	☐	Wire Mesh
☐	☐	Artificial Limb or Joint
☐	☐	Any orthopedic item(s) (i.e. pins, rods, screws, nails, clips, plates, wire, etc.)
☐	☐	Dentures or any type of removable dental item
☐	☐	Hearing Aid
☐	☐	Tattoos
☐	☐	Body Piercings
☐	☐	Transdermal Patches (i.e. nicotine, nitroglycerine, etc.)

Yes	No	
☐	☐	Worked as welder, lathe operator, sheet metal worker or any similar occupation that may result in a metallic foreign object in your eyes.
☐	☐	Have you ever had an endoscopy (gastroscopy/colonoscopy) procedure in 2011 or later and is there any possibility that you have a Metallic Clip

Have you ever had any surgical procedure or operation? ☐ Yes ☐ No

Type _____ Year _____

Type _____ Year _____

Type _____ Year _____

Have you **EVER** had any metal fragments in your eyes, or had an injury to your eyes with metal? ☐ Yes ☐ No

Do you have a history of kidney failure or are you on kidney dialysis? ☐ Yes ☐ No

Are you pregnant or do you suspect that you are pregnant? ☐ Yes ☐ No LMP _____

Patient Weight _____ lb / kg Patient Height _____ in / cm

I have answered the above questions to the best of my ability.
The MRI examination has been explained to me and I have had my questions answered to my satisfaction.

Signature of Patient or Guardian _____ Date _____

Witness / Technologist _____

CH-0185 Aug 2007

PAGE 1 OF 1