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Affective and Cognitive Influences on Decision Making in Obesity

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2010
Declaration

I hereby declare that:

(a) the work contained in this thesis has not been submitted as an exercise for a degree at this or any other university,

(b) this thesis is the result of my own investigations, and the contributions of others are duly acknowledged in the text wherever included,

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Summary

Significant increases in the prevalence of overweight and obesity have been observed worldwide, the direct and indirect costs of which are huge for society. The most rapid increases have been seen in those who were already overweight or obese, rendering the morbidly obese a particularly vulnerable group. The increased prevalence of obesity has been closely associated with increasing energy supply per capita in western countries, highlighting the role of individual choice and decision making in the regulation of diet and weight. Indeed, obesity can be viewed as the cumulative result of thousands small decisions that have the outcome that caloric intake exceeds caloric expenditure. Poor decision making is also seen to affect the ability to commit to weight loss interventions. As such, this thesis aimed to examine decision making, and specifically affective and cognitive influences on decision making, in a morbidly obese population, through using a neuropsychological and food diary approach.

Chapter 1 reviewed early research on decision making in psychology, which focused on rational decision making and indeed the limits to human decision making, through to the more recent emphasis on affective influences on decision making. Historical approaches to decision making in obesity, including the concepts of restraint, external and emotional eating, seen to address cognitive, sensory, and affective influences on decision making around energy intake, respectively, were discussed. Subsequently, recent neuropsychological approaches applied to decision making in obesity, including the Iowa Gambling Task (IGT) based on the Somatic Marker Hypothesis (SMH) were detailed.

Chapter 2 aimed to investigate decision making under uncertainty in morbidly obese individuals (n = 42) using the IGT, compared to a healthy comparison group (n = 50). Accordingly, obese participants were found to be significantly impaired in overall task performance and task learning. These deficits were independent of age, gender, education, eating pathology, and severity of the condition as measured by BMI. As these IGT decision making deficits were in line with previous investigations of other disordered eating populations, Chapter 3 used the IGT to compare decision making processes across Anorexia Nervosa (n = 22), Bulimia Nervosa (n = 17) and obesity (n = 18). Results showed that the three clinical groups were significantly impaired on the IGT relative to the comparison group, but were not significantly different from each other, supporting a broad profile of shared decision
making deficits in disordered eating females. As such, the IGT proved a useful tool in revealing common decision making impairments in the inability to regulate food intake.

Chapter 4 sought to further specify the decision making deficits in morbid obesity revealed in Chapter 2, through the inclusion of psychophysiological measures (skin conductance response (SCR) and heart rate) and additional neuropsychological and psychometric measures. It also aimed to assess the validity of the SMH. Accordingly, IGT deficits were again revealed in obese (n = 21), compared to a comparison (n = 20) participants. However, intelligence was found to remove group differences. Inconsistent with the SMH, 'somatic marker' signals did not differentiate between these groups. The Soochow Gambling Task, revealed that both obese and comparison participants were impaired on this task, responding to immediate reinforcement, rather than to long-term expected value, and therefore showing the normal tendency to underweight rare events and (or) to be loss averse. In assessing decision making under explicit conditions, obese participants were unimpaired on the Game of Dice Task. Addressing the validity of the SMH, 'somatic marker' activity failed to discriminate between impaired vs. non-impaired performers on the IGT and was thus unsupportive of the SMH. An alternative interpretation of this hypothesis was discussed.

Chapter 5 aimed to investigate decision making in morbid obesity in a naturalistic environment using the food diary methodology. Invoking the concepts of restraint, external and emotional eating, the relationships between these self-reported eating styles and intake were examined. Restraint was the strongest predictor of lower overall energy intake, external eating was related to higher intake in females only and emotional eating was unrelated to intake. The same pattern existed for the consumption of top-shelf foods, but for the entire sample. A second research question concerning whether self-reported emotional eaters ate more when emotional found that for both positive and negative emotional states, self-reported emotional eater status had no impact on overall food intake or on the consumption of specific food types. The predictive validity of emotional eater scales and indeed the construct of emotional eating itself were debated.

Chapter 6 summarised the findings and their implications for understanding affective and cognitive influences on decision making in morbid obesity. Alternative interpretations of the SMH as a theoretical framework were articulated. Finally, discussion focused on limitations to the individual studies, in addition to suggestions for future research.
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Publications Arising from the Present Work


# Table of Contents

## DECLARATION

I

## SUMMARY

II

## ACKNOWLEDGEMENTS

IV

## PUBLICATIONS ARISING FROM THE PRESENT WORK

V

## TABLE OF CONTENTS

VI

## LIST OF TABLES

XII

## LIST OF FIGURES

XIII

## CHAPTER 1: INTRODUCTION

1

1.1 The rise in obesity

1

1.2 Decision making and obesity

3

1.3 Psychological approaches to decision making

4

1.4 Historical approaches to decision making in obesity

7

1.4.1 Psychosomatic theories of obesity

7

1.4.1.1 The Kaplan and Kaplan psychosomatic theory of obesity

8

1.4.1.2 Bruch’s theory

8

1.4.2 Externality theory

9

1.4.3 Restraint theory

10

1.5 Neuropsychological approaches to decision making

12

1.5.1 Decision making and the Ventromedial prefrontal cortex (VmPFC)

12

1.5.2 The Somatic Marker Hypothesis (SMH)

14

1.5.3 The Iowa Gambling Task (IGT)

15

1.5.4 Interpreting IGT performance

16

1.5.4.1 Task analysis

16

1.5.4.2 The Expectancy Valence Model

16

1.5.4.3 The Variant IGT: E’ F’ G’ H’

17

1.5.5 The IGT and psychophysiological support

18

1.5.6 IGT and clinical application

19

1.5.7 Individual differences in IGT performance

20

1.6 Critique of the IGT

24
1.6.1 Strengths of the paradigm
1.6.2 Weakness of the IGT
  1.6.2.1 Cognitive penetrability of the reward / punishment schedule
  1.6.2.2 Psychophysiological issues
  1.6.2.3 Variability in control participant performance
  1.6.2.4 ABCD vs. A'B'C'D'
  1.6.2.5 Specificity
  1.6.2.6 Task design

1.7 Aims of the thesis

CHAPTER 2: IMPAIRED DECISION MAKING AMONG MORBIDLY OBESE ADULTS

2.1 Introduction
  2.1.1 Decision making and eating disorders
  2.1.2 IGT, addiction and obesity
  2.1.3 Aims of the current study

2.2 Method
  2.2.1 Participants & procedure
  2.2.2 Measures
    2.2.2.1 Anthropometric Measurements
    2.2.2.2 Psychometric measures
    2.2.2.3 Decision making
  2.2.3 Data analysis

2.3 Results
  2.3.1 Sample characteristics
  2.3.2 Behavioural performance on the IGT
  2.3.3 Task learning: Net scores blocks 1–5
  2.3.4 Eating pathology and impulsivity
  2.3.5 Clinical impairment on the IGT

2.4 Discussion
  2.4.1 IGT performance
  2.4.2 IGT and eating disorders
  2.4.3 Explicit decision making
  2.4.4 Decision making and executive functioning
  2.4.5 Clinical comparisons
  2.4.6 Study limitations

2.5 Conclusion

CHAPTER 3: ANOREXIA, BULIMIA AND OBESITY: SHARED DECISION MAKING DEFICS ON THE IOWA GAMBLING TASK (IGT)

3.1 Introduction
  3.1.1 Pathological eating behaviour
5.1.3 Experimental studies: Emotional eating and intake when emotional
5.1.4 Aims of the current study

5.2 Method
5.2.1 Participants & procedure
5.2.2 Measures
  5.2.2.1 Diary development
  5.2.2.2 Anthropometric measurements
5.2.3 Data analysis
  5.2.3.1 Missing data

5.3 Results
5.3.1 Sample characteristics
5.3.2 Theories of overeating and variables under study
5.3.3 Eating style and overall intake
5.3.4 Eating style and food type
5.3.5 Mood and self-reported eating style
5.3.6 Eating style and intake when emotional
5.3.7 Regulation of intake
5.3.8 Self-reported bingers and non-bingers
  5.3.8.1 Portions during participant-identified binge episodes

5.4 Discussion
5.4.1 Eating style and overall food intake
5.4.2 Eating style and food type
5.4.3 Emotional eater status and intake when emotional
5.4.4 The dominance of restraint
5.4.5 Study limitations
5.4.6 Application of the findings

5.5 Conclusion

CHAPTER 6: GENERAL DISCUSSION

6.1 Summary of empirical chapters
6.1.1 Affective decision making in obesity
6.1.2 Obesity and addiction
6.1.3 Affective decision making across disordered eating groups
6.1.4 Affective decision making and food intake
6.1.5 Psychophysiological brakes
6.1.6 Cognitive influences on decision making
6.1.7 Individual difference variables and decision making
6.1.8 Integrating neuropsychological and naturalistic approaches
6.1.9 The validity of the SMH
6.1.10 Limitations of the research
6.1.11 Future research

6.2 Conclusion
**REFERENCES**

**APPENDIX 1: PSYCHOPHYSIOLOGY AND THE IGT**

**APPENDIX 2: ETHICAL APPROVAL LETTERS**

**APPENDIX 3: STUDY PROTOCOL FOR CHAPTERS 2 AND 3**

**APPENDIX 4: GAIN-LOSS STRUCTURE ON THE ORIGINAL IGT AND SGT**

**APPENDIX 5: PROTOCOL FOR CHAPTER 4**

**APPENDIX 7: PROTOCOL FOR FOOD DIARY STUDY**
List of Tables

TABLE 2-1. REWARD STRUCTURE FOR THE IGT (BECHARA ET AL., 1994). 38
TABLE 2-2. CLINICAL DEMOGRAPHIC PROFILE OF PARTICIPANTS. 40
TABLE 2-3. PROFILE OF PARTICIPANTS ON PSYCHOMETRIC MEASURES. 42
TABLE 2-4. INTERCORRELATIONS BETWEEN PSYCHOMETRIC MEASURES FOR BOTH THE OBESE GROUP (OVER THE DIAGONAL) AND THE COMPARISON GROUP (BELOW THE DIAGONAL). 43
TABLE 3-1. CLINICAL DEMOGRAPHIC PROFILE OF FEMALE COMPARISON, AN, BN, AND OBESE PARTICIPANTS. 53
TABLE 4-1. GAIN-LOSS STRUCTURE IN ORIGINAL SGT (LIN ET AL., 2009). 73
TABLE 4-2. CLINICAL DEMOGRAPHIC AND PSYCHOMETRIC PROFILE OF PARTICIPANTS. 79
TABLE 4-3. IGT MEMORY ASSESSMENT. FREQUENCIES FOR THE DISADVANTAGEOUS AND ADVANTAGEOUS DECKS ARE PRESENTED. 86
TABLE 4-4. SGT MEMORY ASSESSMENT. FREQUENCIES FOR THE DISADVANTAGEOUS AND ADVANTAGEOUS DECKS ARE PRESENTED. 93
TABLE 5-1. CLINICAL DEMOGRAPHIC PROFILE OF PARTICIPANTS. 126
TABLE 5-2. SAMPLE MEANS ON RESTRAINT, EMOTIONAL, AND EXTERNAL EATING DEBQ SUBSCALES AND CLASSIFICATION AS PER SCALE NORMS. 127
TABLE 5-3. INTERCORRELATIONS BETWEEN VARIABLES UNDER STUDY. 127
TABLE 5-4. HIERARCHICAL MULTIPLE REGRESSION ANALYSES FOR THEORIES AND OVERALL FOOD INTAKE. 128
TABLE 5-5. HIERARCHICAL MULTIPLE REGRESSION ANALYSES FOR TOP-SHELF FOODS. 129
TABLE 5-6. HIERARCHICAL MULTIPLE REGRESSION ANALYSES FOR EMOTIONAL AND RESTRAINT EATING. 129
TABLE 5-7. HIERARCHICAL MULTIPLE REGRESSION ANALYSES FOR NA AND OVERALL FOOD INTAKE. 130
TABLE 5-8. HIERARCHICAL MULTIPLE REGRESSION ANALYSES FOR PA AND OVERALL FOOD INTAKE. 130
List of Figures

FIGURE 1-1. THE RUNAWAY WEIGHT GAIN TRAIN (SWINBURN & EGGER, 2004). 2


FIGURE 2-1. MEAN NET SCORES \([C&D - A&B]\) ON THE IGT FOR THE COMPARISON AND OBESE GROUPS. 40

FIGURE 2-2. MEAN NET SCORES \([(C&D - A&B)]\) ON THE IGT ACROSS EACH BLOCK OF 20 TRIALS. POSITIVE SCORES REFLECT ADVANTAGEOUS PERFORMANCE WHILE NEGATIVE SCORES REFLECT DISADVANTAGEOUS PERFORMANCE. 41

FIGURE 2-3. MEAN NET SCORES FOR TRIALS 1-50 AND TRIALS 51-100 FOR THE COMPARISON AND OBESE GROUPS. 42

FIGURE 3-1. MEAN NET SCORES \([(C&D - A&B)]\) ON THE IGT FOR EACH GROUP. 54

FIGURE 3-2. MEAN NET SCORES \([(C&D - A&B)]\) FOR BLOCKS 1-5 FOR EACH GROUP. POSITIVE SCORES REFLECT ADVANTAGEOUS PERFORMANCE WHILE NEGATIVE SCORES REFLECT DISADVANTAGEOUS PERFORMANCE. ERRORS BARS ARE STANDARD ERRORS. 55

FIGURE 4-1. ILLUSTRATION OF THE FIXED ALTERNATIVES PROVIDED IN THE GDT. 74

FIGURE 4-2. MEAN NET SCORES \([(C'&D' - A'&B')]\) ON THE IGT FOR THE COMPARISON AND OBESE GROUPS. ERROR BARS ARE STANDARD ERRORS. 80

FIGURE 4-3. MEAN NET SCORES \([(C'&D' - A'&B')]\) ON THE IGT ACROSS EACH BLOCK OF 20 TRIALS. POSITIVE SCORES REFLECT ADVANTAGEOUS PERFORMANCE WHILE NEGATIVE SCORES REFLECT DISADVANTAGEOUS PERFORMANCE. ERROR BARS ARE STANDARD ERRORS. 81

FIGURE 4-4. MEAN IGT NET SCORES \([(C'&D' - A'&B')]\) FOR TRIALS 1-50 AND TRIALS 51-100 FOR THE COMPARISON AND OBESE GROUPS. ERROR BARS ARE STANDARD ERRORS. 82

FIGURE 4-5. IGT MEAN PROBABILITY OF SHIFT AND STAY IN THE CONTINUING CHOICE. ERROR BARS ARE STANDARD ERRORS. 82

FIGURE 4-6. MEAN TOTAL ANTICIPATORY SCR FOR DISADVANTAGEOUS \((A', B')\) AND ADVANTAGEOUS \((C', D')\) DECKS. 83

FIGURE 4-7. MEAN ANTICIPATORY HEART RATE \((IBi0 - IBi-1)\) FOR DISADVANTAGEOUS \((A', B')\) AND ADVANTAGEOUS \((C', D')\) DECKS. ERROR BARS ARE STANDARD ERRORS. 84
FIGURE 4-8. MEAN RESPONSE SCR FOR DISADVANTAGEOUS (A', B') AND ADVANTAGEOUS (C', D') DECKS. ERROR BARS ARE STANDARD ERRORS.

FIGURE 4-9. DECK * REINFORCEMENT INTERACTION FOR RESPONSE SCR. ERROR BARS ARE STANDARD ERRORS.

FIGURE 4-10. MEAN RESPONSE HEART RATE (IBI+2 – IBI-1) FOR DISADVANTAGEOUS (A', B') AND ADVANTAGEOUS (C', D') DECKS. ERROR BARS ARE STANDARD ERRORS.

FIGURE 4-11. MEAN NUMBER OF CARDS SELECTED FROM EACH DECK ON THE SGT.

FIGURE 4-12. MEAN NET SCORES ([(C&D – A&B)] ON THE SGT ACROSS EACH BLOCK OF 20 TRIALS. POSITIVE SCORES REFLECT ADVANTAGEOUS PERFORMANCE WHILE NEGATIVE SCORES REFLECT DISADVANTAGEOUS PERFORMANCE. ERROR BARS ARE STANDARD ERRORS.

FIGURE 4-13. MEAN SGT NET SCORES ([(C&D – A&B]) FOR TRIALS 1-50 AND TRIALS 51-100 FOR THE COMPARISON AND OBESE GROUPS. ERROR BARS ARE STANDARD ERRORS.

FIGURE 4-14. SGT MEAN PROBABILITY OF SHIFT AND STAY IN THE CONTINUING CHOICE.

FIGURE 4-15. MEAN TOTAL ANTICIPATORY SCR FOR DISADVANTAGEOUS (A, B) AND ADVANTAGEOUS (C, D) DECKS.

FIGURE 4-16. MEAN ANTICIPATORY HEART RATE (IBI0 – IBI-1) FOR DISADVANTAGEOUS (A, B) AND ADVANTAGEOUS (C, D) DECKS. ERROR BARS ARE STANDARD ERRORS.

FIGURE 4-17. MEAN RESPONSE SCR FOR DISADVANTAGEOUS (A, B) AND ADVANTAGEOUS (C, D) DECKS. ERROR BARS ARE STANDARD ERRORS.

FIGURE 4-18. DECK * REINFORCEMENT INTERACTION FOR RESPONSE SCR. ERROR BARS ARE STANDARD ERRORS.

FIGURE 4-19. MEAN RESPONSE HEART RATE (IBI+2 – IBI-1) FOR DISADVANTAGEOUS (A, B) AND ADVANTAGEOUS (C, D) DECKS. ERROR BARS ARE STANDARD ERRORS.

FIGURE 4-20. MEAN FREQUENCY OF CHOICE FROM EACH OF THE FOUR CATEGORIES ON THE GDT. ERROR BARS ARE STANDARD ERRORS.

FIGURE 4-21. MEAN TOTAL ANTICIPATORY SCR FOR DISADVANTAGEOUS (LINES 1 AND 2) AND ADVANTAGEOUS (LINES 3 AND 4) CATEGORIES. ERROR BARS ARE STANDARD ERRORS.

FIGURE 4-22. MEAN ANTICIPATORY HEART RATE (IBI0 – IBI-1) FOR DISADVANTAGEOUS (LINES 1 AND 2) AND ADVANTAGEOUS (LINES 3 AND 4) CATEGORIES. ERROR BARS ARE STANDARD ERRORS.

FIGURE 4-23. MEAN TOTAL RESPONSE SCR FOR DISADVANTAGEOUS (LINES 1 AND 2) AND ADVANTAGEOUS (LINES 3 AND 4) CATEGORIES. ERROR BARS ARE STANDARD ERRORS.

FIGURE 4-24. MEAN RESPONSE HEART RATE (IBI0+1– IBI-2) FOR DISADVANTAGEOUS (LINES 1 AND 2) AND ADVANTAGEOUS (LINES 3 AND FOUR) CATEGORIES. ERROR BARS ARE STANDARD ERRORS.
FIGURE 4-25. MEAN ANTICIPATORY SCR FOR DISADVANTAGEOUS (A', B') AND ADVANTAGEOUS (C', D') DECKS. ERROR BARS ARE STANDARD ERRORS.  

FIGURE 4-26. MEAN ANTICIPATORY HEART RATE (IBI - IBI-1) FOR DISADVANTAGEOUS (A', B') AND ADVANTAGEOUS (C', D') DECKS. ERROR BARS ARE STANDARD ERRORS. 

FIGURE 4-27. MEAN RESPONSE SCR FOR DISADVANTAGEOUS (A', B') AND ADVANTAGEOUS (C', D') DECKS. ERROR BARS ARE STANDARD ERRORS. 

FIGURE 4-28. MEAN RESPONSE HEART RATE (IBI+2 - IBI-1) FOR DISADVANTAGEOUS (A', B') AND ADVANTAGEOUS (C', D') DECKS. ERROR BARS ARE STANDARD ERRORS.
Chapter 1: Introduction

"To eat is a necessity, but to eat intelligently is an art." ~ La Rochefoucauld

1.1 The rise in obesity

Increases in the prevalence of overweight and obesity among both adults and children have been observed worldwide. Obesity can be defined as a state in which there is an excess of adipose tissue (Hetherington, 2007) or a disease whereby excess body fat has accumulated to the extent that health may be adversely affected (World Health Organisation [WHO], 2000). It is most frequently measured in terms of Body Mass Index (BMI), that is, weight (kg) as a function of height (m²). Accordingly, a BMI of 20 – 24.9 is classified as a healthy weight, 25 – 29.9 as overweight and ≥ 30 as obese. Obesity is further specified as Class I (30 – 34.9), Class II (35 – 39.9) and Class III (≥ 40) (WHO, 2000). In the United States, between 1980 and 2002, obesity prevalence doubled in adults aged 20 years or older (Ogden et al., 2006), with the most rapid increase seen in those considered extremely obese (i.e. (BMI) ≥ 40) (Boeka & Lokken, 2008). Within Great Britain, the prevalence of obesity among adults almost tripled during the same period (Rennie & Jebb, 2005). In Ireland, the most recent Survey of Lifestyle, Attitudes, and Nutrition in Ireland ([SLÁN]; Harrington et al., 2008) found, based on measured data, 38% of adults were overweight (44% men and 31% women) and almost one-quarter (23%) were obese (22% men and 23% women). There figures compared to estimates of 39% overweight and 18% obesity in 1999 (National Taskforce on Obesity, 2005). Globally, the prevalence of obesity has increased by greater than 75% since 1980, leading the WHO (2000) to proclaim a global obesity epidemic. As obesity is associated with many diseases, such as cardiovascular disease, type II diabetes, functional disability, and musculo-skeletal disorders, the direct and indirect costs of obesity for a society are huge (Silventoinen et al., 2004). Obesity is a major contributor to preventive death, and poses a significant public health challenge (National Heart, Lung, and Blood Institute [NHLBI], 1998).

To describe this public health challenge of obesity, Swinburn and Egger (2004) invoked the analogy of a ‘runaway weight gain train’ (see Figure 1-1). The train is at high momentum from the downhill slope of obesogenic (obesity promoting) environments whereby unhealthy choices are the easy, default choices, resulting in a high intake of energy dense foods and beverages and in reduced physical activity. Examples would include the heavy promotion of fast food outlets, in addition to transport systems and urban design that inhibit both active
transport and active recreation. The train is then further perpetuated or even accelerated by a series of cycles or positive feedback loops, which include movement inertia, mechanical dysfunction, psychological dysfunction, dieting and low socioeconomic status (SES). Movement inertia, most apparent in people who are substantially overweight, is created by the fact that a heavy body weight is a disincentive for movement and physical activity, which further promotes weight gain. Closely related to this are mechanical problems associated with increased body weight, such as arthritis, arthralgia, low back pain, chest wall and diaphragm restriction, incontinence, obstructive sleep apnoea, and cellulitis, all of which may result in pain and difficulty walking, daytime somnolence and fatigue, which further limit physical activity and increase weight gain. The psychological dysfunction cycle concerns the fact that for some obese individuals, body dissatisfaction and social stigmatisation (see Wadden & Stunkard, 1985) may lead to depression, anxiety, low self-esteem and guilt, which may in turn lead to increased intake (e.g. binge and comfort eating) or reduced physical activity (e.g. lethargy, lack of motivation). Unsuccessful dieting and the consequent sense of personal failure associated with this may exacerbate this cycle. The dieting cycle itself refers to the unsustainable nature of diets, given they are often hypocaloric resulting in marked weight loss. The metabolic response to a large weight loss is vigorous and the subsequent weight that is regained is disproportionately higher in fat. Finally, a higher prevalence of obesity is found in groups with a lower SES, especially among women. This relationship also seems to be bidirectional as obesity leads to reduced opportunities for jobs, education, marriage and social inclusion. A lower income reduces the range of healthy choices (e.g. fresh fruit and vegetables) and low SES neighbourhoods are usually more obesogenic than higher income neighbourhoods, evidencing the perpetuating nature of the cycle (Swinburn & Egger, 2004).

![Figure 1-1. The runaway weight gain train (Swinburn & Egger, 2004).](image-url)
The brakes of the train including social (e.g. social discrimination of being overweight or obese), cognitive (e.g. knowledge about the causes of obesity; cognitive effort to control weight gain), personal (e.g. personal physical discomfort associated with obesity which reduces quality of life) and physiological (e.g. metabolic reactions to weight gain such as reductions in appetite, increased fat oxidation, increased energy expenditure, insulin resistance, increase sympathetic nervous system activation, increased leptin concentrations) factors, which should be sufficient to slow it down, are seen as weak by comparison to both the obesogenic environment as well as the aforementioned cycles. While acknowledging the importance of the environmental context and social factors, the present thesis focuses on factors within the individual which may help explain the rise in obesity. In terms of Swinburn and Egger's model (2004), the thesis addresses ineffectual brakes at the cognitive and psychophysiological levels of the 'runaway weight gain train' through the investigation of decision making in a morbidly obese population. However, the importance of environmental context as an ecological framework for decision making will be addressed in Chapter 5.

1.2 Decision making and obesity

The increase in the prevalence of overweight and obesity has been closely associated with increasing energy supply per capita in western countries (Silventoinen et al., 2004), highlighting the role of individual choice and decision making in dietary and weight regulation. Davis, Levitan, Muglia, Bewell, and Kennedy (2004) argued that unlike times in our evolutionary history when food was scarce and the next meal unpredictable, current human eating is not just a passive response to salient environmental triggers and powerful physiological drives, it is fundamentally about making a choice between short-lived and overabundant rewards in the face of a disadvantageous long-term outcome if the behaviour is done in excess. In an environment where food, particularly high calorie energy dense food, is readily available, poor decision making can make certain individuals vulnerable to overeating. Indeed, obesity can be regarded as the result of thousands of small choices that have the outcome that caloric intake exceeds the decision maker's caloric expenditure (Weber & Johnson, 2009). Furthermore, decision making deficits are seen to contribute not only to the development of an obese state, but also to present significant barriers to weight management with arguments in the literature that it is such deficits that lead to an inability to commit to weight loss interventions (Biddle & Dovey, 2009). As such, the decision making processes underlying obesity, affecting both the development and management of the condition, warrant elucidation.
1.3 Psychological approaches to decision making

Before delineating the specific approaches taken to investigating decision making in obesity in this thesis, brief consideration will be given to aspects of judgement and decision making (JDM) research which have been the focus of psychological investigation.

In the 1950s, research on decision making in psychology very much focused on the limitations of human decision making. Simon (1956) criticised rational models of decision making, such as those adopted by classical economists, as ignoring situational and personal constraints such as time pressure and limited cognitive capacity. Indeed, he proposed that the mind had evolved short-cut strategies that deliver reasonable solutions to real-world problems, an idea known as bounded rationality. In the 1970's, this view gave way to a body of research on mental short cuts or heuristics. Much of this research focused on the systematic errors (biases) that can occur through the use of heuristics, such as in the representativeness, availability, anchoring and adjustment heuristics, and the hindsight bias. Assessing decision making under risk and uncertainty has also been of interest to psychologists. For example, under risky conditions, expected value theory states that each potential outcome should be weighted by the probability of its occurrence, with the expected value rendered the sum of weighted outcomes. The rational decision maker should therefore choose the alternative with the highest expected value. This view was challenged by expected utility theory (Bernoulli, 1954 [1738]) which proposed that people are actually trying to maximise utility (the desirability) of each action's possible outcomes, not value. Prospect theory (Kahneman & Tversky, 1979b), considered a psychological variant of subjective expected utility theory, was developed as a theory of how people actually make decisions between two alternatives under risk. It recognised that the reference point against which gains and losses are defined may be an expectation rather than the status quo (Hardman, 2009). In attempts to explain decision making under uncertainty, cumulative prospect theory was developed (Tversky & Kahneman, 1992) which was later combined with support theory (Tversky & Koehler, 1994) into a two-stage model (Fox & Tversky, 1998). The model argues that people make an assessment of the probability of uncertain events, which is then weighted and modified according to the decision maker's own knowledge of the domain in question. Other approaches to decisions under uncertainty and risk have placed greater emphasis on the stages of processing involved in decision making, as in, for example, decision field theory (Busemeyer & Johnson, 2004) and the priority heuristic (Brandstätter, Gigerenzer, & Hertwig, 2006). In decision field theory the deliberations of a decision maker involve
switching attention between alternatives and thinking about their consequences, after which an overall feeling of desirability in relation to each action accumulates. Once a threshold is passed, then the successful action is chosen. By contrast, the priority heuristic argues that decisions under risk do not involve the computation of expectations. Instead, people give sequential attention to outcomes and probabilities according to an order of prioritisation.

JDM has also addressed judgement and choice over time. For example, studies have found that people are frequently too optimistic about the time it will take them to complete a task. The planning fallacy, which suggests that people tend to take the ‘inside view’ of a task, focusing on how the various steps of a plan will lead to success, but neglecting past experience and factors which could interfere with task completion, has been used to explain such optimism (Kahneman & Tversky, 1979a). Attention has also been given to the fact that in many domains, people prefer immediate rewards to future rewards, referred to as time preference (Frederick, Loewenstein, & O’Donoghue, 2002). With regards to the quantification of intertemporal choice, the discounted utility model (Samuelson, 1937) proposed that people discount future rewards at a constant rate. However, for monetary outcomes, hyperbolic discounting (i.e. declining discount rates with increasing time) appears to be a more accurate description of behaviour (Thaler, 1981).

Dual-process explanations have been invoked to distinguish between intuitive and reflective decision making processes. Accordingly, psychological models have distinguished between a rapid, automatic and effortless, associative and intuitive process (System 1) and a slower, rule-governed, analytic, deliberate and effortful process (System 2) (Kahneman, 2003). There is debate about the extent and way in which the two systems interact (Weber & Johnson, 2009). As an example, serial interventionist models put System 2 into a supervisory role because System 2 knows the analytic rules that the intuitive System 1 is prone to violate and thus can intervene to correct erroneous intuitive judgments (Kahneman, 2003).

More recently, JDM research has focused on dynamic decision making which is largely laboratory based, examining decision making in microworlds; simulated environments in which people typically try to control a complex system in which initial decisions may affect later decisions (Hardman, 2009). Microworlds enable researchers to investigate a variety of factors such as cognitive ability, type of feedback, timing of feedback, strategy use, and knowledge acquisition. However, performance on such tasks is usually far from optimal.
Explanations of learning in dynamic decision making tasks include the use of production rules, the retrieval of instances or exemplars, and connectionism (Hardman, 2009). Dynamic tasks have also been employed in the context of risk in neuropsychological tasks such as the Columbia Card Task (Figner, Mackinlay, Wilkening, & Weber, 2009) and the Balloon Analogue Risk Task (Lejuez et al., 2002), which aim to predict risk taking in dynamic environments. Thus, the decision making task is used in an artificial way to reflect a behaviour. In contrast to the above approach, researchers who are interested in real-world contexts now identify themselves as working within a field called naturalistic decision making (NDM). NDM is less concerned with psychological processes at the moment of choice and more concerned with the processes leading up to the moment of choice (e.g. planning, perception, comprehension, and forecasting). Under the heading of Cognitive Task Analysis, techniques used in this domain include the use of concurrent verbal reports and the critical decision method, which involves the telling and retelling of a previously encountered situation. Theories of NDM include image theory (Beach, 1990) and recognition primed decision making (RPD; Klein, 1998). Image theory proposes that people screen out decision options that violate their basic rules, while the RPD model is an attempt to explain how critical decisions are made under time pressure and stress through the recognition of the situation as prototypical and consequent use of knowledge to generate a single course of action. There is also a host of literature on social decision making not addressed here; however, comprehensive reviews of JDM research can be found in Weber and Johnson (2009) and Lowenstein, Rick and Cohen (2008).

This brief overview of some of the areas of decision making historically addressed by psychology demonstrates a marked focus on cognitive processes and indeed the limitations of those processes. Weber and Johnson (2009) argued that the cognitive revolution may have been too focused on analytic and computational processes, and that this overemphasis has since been corrected by the more recent emotional revolution which, through documenting affective processes and depicting them as automatic and essentially effort-free inputs that orient and motivate adaptive behaviour, has put affective processes on a footing equal to cognitive ones. They describe the four functions of affect as: affect as spotlight, affect as information, affect as common currency and affect as motivator. Affect as spotlight argues that emotions experienced by the decision maker focus attention on features of the environment that matter for emotion-appropriate action tendencies. For example, feelings of fear or worry focus attention on the source of the apparent threat and ready flight responses (Loewenstein, Weber, Hsee, & Welch, 2001). Affect as information outlines that emotions
experienced while making a decision are incorporated as information into choices. Thus, positive and negative past associations with available choice outcomes contribute to new decisions. Affect as common currency views experienced emotions as a common currency on which the effects of different outcome dimensions and variations in decision context can be integrated. Finally, affect as motivator draws on affect construal theory, which shows that the effect of affective reactions is not satisfactorily attributed to the emotion's valence and intensity, but rather is influenced by other situational appraisals; that is, affect is constructed (Ellsworth & Sherer, 2003). Thus while emotions can be similar in valence or intensity (e.g. fear versus anger) they result in very different judgements or choices, because they motivate different action tendencies (Weber & Johnson, 2009). The relationship between emotion and JDM is an area that has received considerable attention since the mid-1990s. For example, the risk-as-feelings hypothesis (Loewenstein et al., 2001) proposes a two-way relationship between cognitions and emotions such they each may influence the other. In addition, both cognitions and emotions may act directly on behaviour unmediated by the other. Particular focus in this thesis will be given to such affective decision making processes with respect to obesity, while later chapters (Chapters 4 and 5) will pay more attention to cognitive influences. This will be addressed through the use of both historical approaches as well as current neuropsychological approaches to decision making in obesity. The former of these will first be addressed.

1.4 Historical approaches to decision making in obesity

Historically, three dominant perspectives in obesity have considered decision making around food and food choice: psychosomatic theories (Bruch, 1974; Ganley, 1989); externality theory (Schachter, Goldman, & Gordon, 1968); and restraint theory (Herman & Polivy, 1975). Psychosomatic theories can be seen to tap primarily emotional influences, restraint cognitive influences and externality sensory influences on eating. A brief outline of these theories will be provided here.

1.4.1 Psychosomatic theories of obesity

Psychosomatic theory focuses on the subject of emotional eating and nearly all such theories view obesity as the result of pathological personality structures or processes (Allison & Heshka, 1993).
1.4.1.1 The Kaplan and Kaplan psychosomatic theory of obesity

Kaplan and Kaplan (1957 as cited in, Canetti, Bachar, & Berry, 2002) proposed that obese people overeat when anxious and eating reduces this anxiety. The mechanism by which eating reduces anxiety may involve differential effects of protein and carbohydrate intakes affecting the synthesis of brain neurotransmitters, in particular serotonin, although learning factors are also probably involved. Furthermore, they hypothesised that the acts of eating and intense fear or anxiety are physiologically incompatible, leading these emotions to be temporarily diminished. Thus, obese individuals are unable to distinguish between hunger and anxiety because they have learnt to eat in response to both states. Such eating in order to reduce anxiety may lead to compulsive overeating and obesity (Canetti et al., 2002).

1.4.1.2 Bruch’s theory

Bruch (1974) linked overeating to faulty hunger awareness. A normal response to emotional arousal or stress is loss of appetite, as emotional arousal inhibits gastric motility and leads to liberation of sugar from the liver into the bloodstream; physiological states similar to the chief peripheral physiological correlates of satiety (Van Strien & Ouwens, 2003). However, at the heart of Bruch’s theory is the hypothesis that the obese have failed to learn to discriminate between the physiological symptoms accompanying food deprivation and internal states of arousal characteristic of emotional states such as fear, anger and anxiety (Schachter et al., 1968). Thus, incorrect and confusing early experiences have interfered with the ability to: 1) recognise hunger and satiation; and 2) differentiate hunger (the urge to eat) from other signals of discomfort independent of food deprivation. As such, hunger is rendered a state that is not universally applied to an identical set of physiological symptoms (Schachter et al., 1968), and the individual will choose to overeat in response to ‘emotional tension’ and ‘uncomfortable sensations and feelings’ (Canetti et al., 2002). If this emotional eating occurs frequently it may lead ultimately to weight gain and obesity.

An extensive review by Ganley (1989) found that the conclusion that obese people engage in significantly more emotional eating than non-obese individuals was quite robust, thus providing support to psychosomatic theories. However, Ganley (1989) also found that the picture was a complex one, in which emotions appear to influence eating by obese participants, only if individual differences in food choice and in the type of emotion precipitating eating are considered. Accordingly, emotional eating was found to usually involve high-calorie or high-carbohydrate food, often with idiosyncratic choices, and different
emotions (e.g. anger, depression, boredom) were found to precipitate emotional eating in different obese individuals. Attention was also drawn to the secrecy around such eating, and its episodic nature, often related to overall stress. Such eating was found to reduce negative affect, especially for emotions such as anger, loneliness, boredom and depression. The generality of the emotion-eating relationship in the obese was supported by the replication of this finding in obese subjects not currently in treatment, refuting claims that the relationship is merely an artefact of a biased sample that seeks treatment (Ganley, 1989). Allison and Heshka (1993), however, question the obesity-emotional eating association arguing that many studies previously considered supportive are substantially flawed with regard to control groups, failure to control Type-I error rate and, the possibility of confounding of social desirability and other response sets with self-reports of emotional eating. While the issue of social desirability remains a concern in overweight and obese individuals (Van Strien & Ouwens, 2003), several lines of contemporary research have supported the association between negative emotions and food intake in both normal weight and overweight populations (e.g. Arnow, Kenardy, & Agras, 1995; Kubiak, Vögele, Siering, Schiel, & Weber, 2008; Macht, Haupt, & Ellgring, 2005; Macht & Simons, 2000; Newman, O’Connor, & Conner, 2007; O’Connor, Jones, Conner, McMillan, & Ferguson, 2008; O’Connor & O’Connor, 2004) and indeed in disordered eating groups such as Anorexia Nervosa (AN) and Bulimia Nervosa (BN) (Van Strien, Engels, Leeuwe, & Snoek, 2005). Positive emotions are also seen to increase food intake although this area has been less well researched (Canetti et al., 2002; Dubé, LeBel, & Lu, 2005). In addition, the influence of emotions on eating behaviour were found to be stronger in obese than non-obese people and in people on diets than in non-dieters (Canetti et al., 2002).

1.4.2 Externality theory

Externality theory (Schachter et al., 1968; Schachter & Rodin, 1974) centres on ‘external eating’, regardless of internal state. Internal factors are defined as those visceral, physiological states varying with food deprivation such as gastric motility, blood sugar concentration and tissue needs. External factors are those which, while affecting food intake, are unrelated to nutrition, including cognitive and sensory cues such as the taste and smell of food, social stimuli and habit (Nisbett, 1968). Support for the theory was derived from findings that gastric motility and self-report of hunger were highly related in normal weight individuals and unrelated in obese individuals. Further, Schachter et al. (1968) manipulated two states that have a visceral effect, food deprivation and fear, and found that these manipulations directly affected the amounts eaten by normal individuals but had no effect on
obese participants (though a recent replication of this study with modifications to the original design (Van Strien & Ouwens, 2003) found emotional eating moderated the intake-deprivation relationship). Similarly, manipulation of the external food-relevant cue, ‘dinner time’ through the use of doctored clocks, found that obese individuals ate more when they thought they were eating after their regular dinner hour than they did when they thought they were eating before it, while there was no such effect for normal weight participants (Schachter & Gross, 1968). Thus, in external eating there is an insensitivity to internal cues of hunger and satiety, with the decision to overeat being driven by a heightened sensitivity to food cues (Van Strien, Schippers, & Cox, 1995).

Both psychosomatic and externality theories operate on the same basic premise, that the individual’s misperception of internal state prior to eating is considered a causal factor in the development of obesity. However, the theories differ on the trigger for such overeating. In psychosomatic theory it is psychic states, and in externality theory it is external food-related stimuli, which are thought to precipitate the decision to over-eat. In addition, both theories also specify that ‘obese eating patterns’, like emotional and external eating behaviour, precede dieting rather than being caused by dieting (Van Strien et al., 1995). This is in contrast to the final theory to be addressed here, that of restraint eating, whereby external and emotional eating are considered consequences of intense dieting (Herman & Polivy, 1975).

1.4.3 Restraint theory

Restraint theory (Herman & Mack, 1975; Herman & Polivy, 1975) was originally conceived based on the notion of Nisbett’s (1972) set-point theory. Nisbett hypothesised that both normal weight and obese individuals eat so as to bring their weight into line with a biologically determined set-point, derived from the number of fat cells in the body. More recent work has challenged the original view that the number of fat cells remains constant after adolescence; however, the concept of set point remains viable with the evident stability of weight levels over time demanding the consideration of such a homeostatic mechanism to account for it (Herman & Mack, 1975). Opposing this biological set-point, individuals are also subject to the pressure of cultural and social demands, such as fashionable ideals of slimness and medical disapproval of overweight, to restrain their eating in order to maintain an ‘ideal’ body weight which is fairly low in absolute terms. As such, restraint theory views eating as a result of both physiological and sensory cues (e.g. hunger, satiety) but also a self-sustained cognitive resistance to or acceptance of physiological / sensory demands on the other hand.
Counterregulation is defined as the breakdown of restrictive control so that suppressed eating behaviour is disinhibited and excessive food intake occurs (Van Strien, Frijters, Bergers, & Defares, 1986). It may occur when self-control processes which monitor dieting behaviour are undermined by, for example, the consumption of alcohol or high caloric food, anxiety or depression. The continuous restraint of hunger sensations may also lead to loss of contact with internal feelings of hunger and satiety. Accordingly, restraint theory views both ‘external’ and ‘emotional eating’ as the consequence of intense dieting (Van Strien, Frijters, Bergers et al., 1986). As an example of the role of internal versus cognitive factors in the counterregulatory effect, Polivy (1976) created a situation where participants consumed an 8 oz serving of chocolate pudding; half received a high calorie (750 kcal) and half a low calorie (325 kcal) pudding. Within the high and low calorie groups, half of the participants in each group were told that the pudding was high or low in calories, respectively. The design thus permitted a comparison of the effect of actual calories to that of perceived calories on the subsequent consumption of sandwiches. Restrained eaters who had a high calorie preload subsequently ate 20 per cent more than restrained eaters with a low calorie preload, seemingly supporting the internal physiological interpretation in counterregulation. However, an examination of restrained eaters who had perceived the preload as high in calories, compared to those who perceived it as low in calories showed that the former group out-ate the latter by 61 per cent, supporting the cognitive interpretation. Of note, within other disordered eating groups, patients suffering from AN score significantly higher on the Restraint Scale (Herman & Polivy, 1975) than normal restrained college students. However, it is bulimic anorectics who score the highest on the Restraint Scale, scoring significantly higher than their anorexic peers (Polivy, 1978).

The theories considered here provided a useful framework for understanding emotional, sensory and cognitive influences on decision making around eating behaviour. While the literature cited above is quite dated, there has been a resurgence of interest in this area in the last decade. Contemporary research has been concerned with the relationship between these eating styles and both actual food intake and body weight, as well as the moderational role of emotional eating on intake, when in emotional states. Such recent
empirical evidence will be reviewed in Chapter 5 as a preface to an empirical food diary study on the relationships between self-reported eating behaviour and actual food intake in morbidly obese individuals.

This early research (in particular psychosomatic theories) highlighted the role of affect in decision making. In line with this, our discussion will now move to recent research in the neuropsychological domain which has used emotion as a lens to investigate decision making. Preliminary research on affective decision making from a neuropsychological perspective, through to its more recent application in profiling decision making deficits in various psychiatric, clinical and health conditions, including obesity, will now be considered.

1.5 Neuropsychological approaches to decision making

1.5.1 Decision making and the Ventromedial prefrontal cortex (VmPFC)

It has been argued that throughout the history of neuropsychology, the psychological capacities associated with the prefrontal region of the brain have remained enigmatic and elusive. However, the region has firmly been established as providing the neural substrate for higher order capacities such as planning, reasoning, self-awareness, empathy, emotion modulation and most notably decision making. Further, the portion of the prefrontal region formed by the orbital and lower mesial sectors (ventromedial; VM) has been considered as the neural basis for personality (Tranel, Bechara, & Damasio, 2000). Early work on affective decision making centred on patients with VmPFC lesions, who show severe impairment in personal and social decision making, despite otherwise normal intellectual functions. After such lesions, the patients have difficulties learning from previous mistakes, as reflected by repeated engagement in decisions that have deleterious consequences. An infamous example of this was the case of Phineas Gage. In 1848, Gage was a successful foreman on the railway. However, following a blasting accident, a tamping iron went through his eye socket extensively damaging his frontal cortex (see Figure 1-2 for portrait). While Gage appeared to have no intellectual impairment from the accident, he started to display odd decision making and social behaviours. Previously a conservative family man, he subsequently could not hold down a job, made risky financial decisions and his family relations broke down. Indeed the personality profile displayed by Gage and other such patients was later designated as 'acquired sociopathy' (Tranel et al., 2000) because of its similarities to that characterised in
clinical psychology and psychiatry as psychopathic (or sociopathic) (American Psychiatric Association [APA], 2000).


Modern neuroimaging techniques applied to Gage’s skull found the tamping iron ablated a portion of the frontal lobe centred around the VmPFC (the portions of the frontal lobe above the eye sockets) (A. R. Damasio, 1994; H. Damasio, Graboeski, Frank, Galaburda, & Damasio, 1994). Further, research by Damasio and his team at the Iowa Laboratory revealed that VmPFC patients had difficulty in expressing emotion and experiencing feelings relative to complex personal and social situations, for example, as in the expression and experience of embarrassment (Tranel et al., 2000). Neuropsychological investigation also showed that VmPFC damage altered psychophysiological response and reported emotional experience to emotional but not neutral stimuli (A. R. Damasio, Tranel, & Damasio, 1990, 1991). Accordingly, skin conductance response (SCR), a highly sensitive index of autonomic responsivity especially with regards to emotional stimuli (as will be discussed later in 1.5.5), was used in conjunction with targets (pictures of emotionally charged stimuli such as mutilations and nudes) and non-targets (pictures of emotionally neutral stimuli such as farm scenes and abstract patterns). While normal and brain-damaged controls displayed a large SCR response to targets and little or no response to non-targets, VmPFC participants generated almost no response to the targets and failed to show the standard target-non-target SCR difference, despite the fact that their ability to generate SCRs to basic physical stimuli was intact.
These findings were crucial in the development of Damasio's (1994) influential Somatic Marker Hypothesis (SMH), which was introduced in an attempt to understand and explain the decision making deficits of Gage and other VmPFC patients. The SMH argues that the decision making deficits found following VmPFC damage are due to an inability to use emotion-based signals generated from the body (or 'somatic markers') when appraising different response options. As such, the SMH extends the influence of somatic processes to the regulation of decision making and emotion (Dunn, Dalgleish, & Lawrence, 2006).

1.5.2 The Somatic Marker Hypothesis (SMH)

The SMH provides a systems-level neuro-anatomical and cognitive framework for decision making and the influence of emotion on decision making (Bechara, Damasio, & Damasio, 2000). The theory proposes that 'somatic marker' biasing signals from the body are represented and regulated in the emotion circuitry of the brain, particularly the VmPFC to help regulate decision making in situations of complexity and uncertainty. Accordingly, when making decisions, a signal ('somatic marker') indicates our emotional reaction to a response option, based on previously acquired associations between that class of stimuli and a certain class of somatic states (Tranel et al., 2000). That is, the ability to choose present options, which favourably influence future outcomes, depends crucially on an accumulated 'emotional memory' of the consequences of past interactions with similar events (Davis et al., 2008). In defining these markers Damasio (1994) stated "When the bad outcome connected with a given response option comes into mind, however fleetingly, you experience an unpleasant gut feeling. Because the feeling is about the body, I gave the phenomenon the technical term somatic state ('soma' is Greek for body); and because it 'marks' an image, I called it a marker" (p.173).

'Somatic markers' can reflect actions of the body proper (the 'body' loop) or the brain's representation of the action expected to take place in the body (the 'as-if' loop) (Dunn et al., 2006). Both mechanisms may operate either overtly (consciously) or covertly (nonconsciously). The VmPFC is regarded as the crucial area of the brain that integrates actual or predicted bioregulatory state representations with potential response options (Dunn et al., 2006). These 'somatic markers' serve as an indicator of the value of what is represented either overtly as a conscious perception in the form of a feeling, or covertly modifying appetitive / aversive behaviour (Tranel et al., 2000). Further, particularly in situations of complexity and uncertainty, they help to reduce the problem space to a tractable size by
marking response options with an ‘emotional’ signal (Dunn et al., 2006), and thus facilitates the operation of logical reasoning (Tranel et al., 2000). ‘Somatic markers’, whether or not they are perceived consciously in the form of feelings, are seen to provide the ‘go’, ‘stop’ and ‘turn’ signals needed for much decision making and planning (Tranel et al., 2000). Decision making is thus viewed as a combination of ‘high reason’, carrying out a logical cost-benefit analysis of a given action, and marker signals, indicating how rewarding or punishing an action is likely to be in complex situations where more detailed cost-benefit analysis is not possible.

Damasio (1994) argued that damage to the VmPFC and other structures involved in the representation and regulation of body-state (amygdala, insula, somatosensory cortex, cingulate, basal ganglia and brain-stem nuclei) leads to impaired decision making because the somatic marking system can no longer be activated. As such, these individuals are unable to use prior emotional experience to guide them towards previous advantageous choices and away from disadvantageous choices, a profile characterised as ‘myopia for the future’, where the individual is unable to predict long-term punishments and rewards based on previous experience (Dunn et al., 2006). In the absence of ‘somatic markers’, individuals may resort to a strategy of deciding on an option based on extremely slow and laborious logic operations. As a result, decision making may fail to be timely, accurate and propitious, or indeed become random or impulsive; corresponding with the real-world behaviour of patients with VmPFC damage (Tranel et al., 2000).

1.5.3 The Iowa Gambling Task (IGT)

Empirical support for the SMH is largely based on performance of the IGT, an experimental paradigm originally designed for the functional assessment of VmPFC patients and as a measure of emotional decision making. The IGT is a card game which tests the ability to postpone immediate reward for a longer-term successful outcome (Apkarian et al., 2004). It simulates, in real time, decision making strategy by factoring uncertainty, reward and punishment (Bechara, Damasio, Damasio, & Anderson, 1994). Participants choose between four decks of cards that yield high immediate gain but larger future losses (disadvantageous decks, A and B) and decks that yield lower immediate gain but a smaller future loss (advantageous decks, C and D). Thus, participants have to forego short-term benefit for long-term profit. Crucially, it is argued that this reward / punishment schedule is opaque, such that participants are unlikely to be able to perform an exact calculation of net gains and losses. Instead, participants must rely more on ‘intuitive’ decision making processes, in particular the
activation of ‘somatic marker’ biasing signals. In this way, the IGT is a test of implicit decision making or decision making under uncertainty (at least until task contingencies are learned), in contrast to tests of an explicit nature where the contingencies of the task are known and obvious as in, for example, the Game of Dice Task ([GDT]; Brand, Kalbe et al., 2005).

1.5.4 Interpreting IGT performance

1.5.4.1 Task analysis

IGT performance is generally evaluated in terms of total net score on the task, that is, the total number of cards chosen from the advantageous decks (C and D) minus those chosen from the disadvantageous decks (A and B). Lower scores therefore represent poorer performance and negative scores indicate a relative preference for choices from the disadvantageous decks. As an index of learning and the strategy used by participants across the trial, IGT performance is analysed by blocks of 20 cards, forming blocks 1 – 5. Similarly, net scores on the IGT for the first 50 trials are often compared with net scores over the last 50 trials. Performance is also commonly examined through ‘response shift’ strategy, that is, the mean probability that participants will shift deck or stay on that deck in the continuing choice, for both advantageous and disadvantageous choices.

1.5.4.2 The Expectancy Valence Model

An alternative approach to decomposing performance on the IGT has been through the use of cognitive modelling by mathematical psychologists. A formal decision model, the Expectancy Valence Learning Model, was developed for the IGT, to examine individual differences in cognitive, motivational and response style processes inherent to the task (Busemeyer & Stout, 2002). The model states that as individuals make selections and experience payoffs, they develop emotional reactions termed valances for each of the decks. These valences are assumed to vary across individuals in line with differences in attention to win versus loss experiences, the rate at which these valences are updated as the task progresses, as well as fidelity of choices to the valences they assign to the decks (Garavan & Stout, 2005). Accordingly, the model derives three parameters describing the individual’s: 1) weight given to wins versus losses \(w\); 2) update \(a\); and, 3) sensitivity \(d\) parameters. The first of these indicates the motivational significance of wins or losses for the individual with smaller values of \(w\) indicating lower sensitivity to losses. The update parameter represents learning / memory of the contingencies by determining the relative influence of new
experiences on expectancies. Large values of $a$ indicate strong recency effects and more rapid forgetting of past outcomes, while small values of $a$ indicate the persistence of influences over longer spans of selections (Stout, Busemeyer, Lin, Grant, & Bonson, 2004). Finally, the sensitivity parameter ($\delta$) is indicative of choice consistency, that is, the consistency with which the decision maker applies expectancies when making selections. When sensitivity is very low, choices are inconsistent, random, reckless, impulsive and independent of expectancies. Conversely, when sensitivity is very high, the deck that has the maximum expectancy will almost certainly be chosen (Stout et al., 2004). In summary, the model allows individual difference measures of the relevant cognitive processes to be extracted from the same performance data that one hopes to explain (Stout et al., 2004). These parameter values are used as quantitative estimates of three aspects of decision processes that can be examined between groups or in association with other demographic and clinical factors (Garavan & Stout, 2005).

The Expectancy Valence model has been applied in this way to decompose the performance of Huntington’s patents (Busemeyer & Stout, 2002) as well as cocaine abusers (Stout et al., 2004). For patients with Huntington’s disease, cognitive modelling analyses indicated that learning and memory processes rather than motivational processes were responsible for the decision making deficits on the IGT, while for drug abusers motivational and choice consistency factors were primarily responsible (Stout et al., 2004). These differences illustrate the value of applying a formal model to decision data from clinical groups in that although the behavioural results from cocaine abusers and Huntington’s patients appear similar, their performance deficits have different underlying theoretical explanations. Further, the use of cognitive modelling to study psychological processes provides a method for building a bridge between neurophysiology and behaviour (Stout et al., 2004).

1.5.4.3 The Variant IGT: $E' F' G' H'$

Finally, it should be noted that a variant of the IGT, $E' F' G' H'$ (Bechara, Dolan, & Hindes, 2002), was introduced in an attempt to further interpret performance deficits on the IGT. The variant task reverses the reward / punishment contingencies so that punishment is immediate and reward delayed and intermittent. The advantageous decks ($E'$, $G'$) yield high immediate punishment but higher delayed reward, while the disadvantageous decks ($F'$ $H'$) yield low immediate punishment and lower delayed reward. The rationale behind the introduction of this variant IGT was to differentiate various profiles of performance on the IGT.
Accordingly, individuals hypersensitive to reward (and potentially hyposensitive to punishment) would be expected to be impaired on the original IGT (where reward is immediate and punishment delayed) but not on the variant task (overcoming the reluctance of normal controls to sample the advantageous decks because of the higher costs associated with these decks before receiving a reward), while individuals insensitive to future consequences, positive or negative, would be expected to be impaired on both tasks. SCR activity was also included in interpreting these profiles (see Bechara et al., 2002).

1.5.5 The IGT and psychophysiological support

Key support for the hypothesised role of 'somatic markers' in performance on the IGT was derived from the identification of a physiological correlate of success on the decision making task (see Appendix 1 for a non-technical index of terms related to psychophysiological assessment and the IGT). Using SCR (introduced in 1.5.1) as an index of somatic state activation, it was found that amongst control subjects, choosing advantageously on the IGT was a correlate of the development of anticipatory SCRs (those preceding a choice), which were larger for selections from 'risky' than from 'safe' decks. Conversely, the absence of anticipatory SCRs in VmPFC patients similarly correlated with impaired task performance (Bechara, Damasio, Damasio, & Lee, 1999). In summary, intact individuals typically learn to adopt an advantageous response strategy during the course of the task, and develop anticipatory SCRs preceding disadvantageous choices, while VmPFC patients show a disadvantageous strategy and the absence of 'somatic markers' in the form of SCR (Bechara, Damasio, Tranel, & Damasio, 1997; Bechara, Tranel, Damasio, & Damasio, 1996). In testing the assertion that the reward / punishment schedule of the IGT is opaque, Bechara et al. (1997) identified four learning periods on the IGT underlying normal performance, and based on behavioural self-report: the pre-punishment period (the start of the game when participants sample the decks and before they encounter a loss); pre-hunch period (participants continue to choose cards from various decks but profess no knowledge of what is happening in the game); hunch period (the period when participants report 'liking' or 'disliking' certain decks and can guess which decks are safe or risky) and, conceptual period (when participants can articulate accurately the nature of the task or identify with certainty the good and bad decks and why they are good or bad (Bechara et al., 1997)). Importantly, anticipatory SCR activity and increased selection from the good decks began to take place for the control group in the pre-hunch period and was sustained throughout the task. Thus, nonconscious biasing signals (SCRs) were found to guide behaviour in normal controls before
conscious knowledge was available, while VmPFC patients who reached the conceptual period (50%) both failed to generate SCRs and to choose advantageously (Bechara et al., 1997).

While skin conductance (SC) reactivity has typically been used as the primary index of sympathetic bodily arousal, 'somatic marker' activity was recently extended to the cardiovascular domain of arousal based on the fact that the SMH suggests a general sensitivity of the autonomic system to activity associated with cognitively driven changes in body states of arousal (Crone, Somsen, Van Beek, & Van der Molen, 2004; Crone, Somsen, Zanolie, & Van der Molen, 2006; Crone & Van der Molen, 2004, 2007). This was achieved through examining heart rate variability (HRV) in interbeat interval (IBI), defined as the time in milliseconds (Ms) between consecutive R waves of an electrocardiogram (EKG) (Kumar, Weippert, Vilbrandt, Kreuzfeld, & Stoll, 2007). Accordingly, Crone et al. (2004) found anticipatory heart rate slowing was higher preceding disadvantageous relative to advantageous response options, but for good performers only, while heart rate slowing increased following loss relative to reward outcomes among all (bad, moderate and good) performers.

1.5.6 IGT and clinical application

As alluded to previously, the IGT, as a test of decision making under uncertain conditions and the most frequently used neuropsychological measure of emotionally-based decision making (Davis et al., 2008), has been used to assess the decision making capacity of a wide range of clinical groups. Preliminary studies incorporated the IGT in the areas of substance dependence and drug addiction, based on the real-life behavioural similarities between VmPFC patients and some substance dependent individuals (SDI), in terms of their preferences for choices that bring immediate benefit even if coupled with negative future consequences (e.g. loss of job, home, family and friends). Thus, the choices to be made in the IGT are similar to those of individuals with an addiction, in that they must continually weigh the short-term rewards against the long-term consequences of their behaviour (Fein, Klein, & Finn, 2004). Addressing a non-substance dependent form of addiction, pathological gamblers have shown impairment on the task (Cavedini, Riboldi, Keller, D'Annucci, & Bellodi, 2002; Goudriaan, Oosterlaan, de Beurs, & Van den Brink, 2005). The task has also been used to profile decision making impairments in neuropsychiatric conditions such as obsessive-compulsive disorder (OCD) (Cavedini, Gorini, & Bellodi, 2006; Lawrence et al., 2006; Starcke, Tuschen-Caffier, Markowitsch, & Brand, 2009; Starcke, Tuschen-Caffier, Markowitsch, & Brand, 2010), schizophrenia (Bark, Dieckmann, Bogerts, & Northoff, 2005; Shurman, Horan, &
The task has also been applied to health, profiling decision making in conditions such as HIV (Gonzalez, Wardle, Jacobus, Vassileva, & Martin-Thormeyer, 2010) and chronic pain (Apkarian et al., 2004). Similarly, in using the premise that the task assesses the ability to forego an immediate reward for a long-term better outcome, the IGT has been used to characterise the behaviour of disordered eating groups such as in obesity (Davis et al., 2004; Pignatti et al., 2006), Binge Eating Disorder (BED; Davis, Patte, Curtis, & Reid, 2010), AN (Cavedini, Bassi, Ubbiali et al., 2004; Cavedini, Zorzi et al., 2006; Tchanturia et al., 2007), and BN (Boeka & Lokkenz, 2006; Liao et al., 2009). However, while the IGT has been used in a wide range of clinical studies, only a subset of these has included psychophysiological measures (e.g. Bechara et al., 1996; Campbell et al., 2004; Jenkinson, Baker, Edelstyn, & Ellis, 2008; Lawrence et al., 2006; Liao et al., 2009; Starcke et al., 2009; Suzuki, Hirotta, Takasawa, & Shigemasu, 2003; Tchanturia et al., 2007). Of note, the application of the IGT as a tool for measuring the ability to forego an immediate reward for a longer-term better outcome, ties in with recent approaches in the health psychology literature such as that of Temporal Self-Regulation Theory ([TST]; Hall & Fong, 2007). Consistent with the premises of the IGT, they argue that the rationality of human behaviour largely depends on the temporal frame adopted in that behaviours judged to be maladaptive in the long-run are usually driven by a strongly favourable balance of immediate costs and benefits. As such, the model, which incorporates thinking about temporal aspects of behavioural contingencies and the biological roots of self-regulation, presents a framework for understanding instances of seemingly ‘self-defeating’ behaviour that have important implications for physical health.

1.5.7 Individual differences in IGT performance

A common trend in the literature has been to look for individual difference variables which may moderate IGT performance, independent of, or in addition to, clinical classification. Education level is one such variable. While results from IGT studies which included education level as a secondary factor in their analyses have been collectively inconclusive, Davis et al. (2008) carried out a systematic investigation into the effect of education level on IGT performance in both the original (Bechara et al., 1994) and variant (Bechara et al., 2002) IGT. Accordingly, they found that learning on the IGT was moderated by both task and education...
level. For the original, widely used IGT, performance tended to improve more rapidly and reach a higher eventual positive score, as level of education increased thus refuting claims by Bechara (2007) that education has only a 'minor' influence on IGT performance, and by Evans, Kemish and Turnbull (2004) that education was inversely correlated with IGT performance. Consistent with Bechara (2007), age and gender were non-significant effects in the model. This argument was further supported by recent findings that cognitive intelligence (IQ) but not emotional intelligence (EIQ) was predictive of IGT performance (Demaree, Burns, & DeDonno, 2010) and that education level was found to remove group differences in IGT performance between obese women (with and without BED) and normal controls (Davis et al., 2010).

Inconsistent with these findings is a recent review on the associations between executive functions (inhibition, set-shifting, and working memory), intelligence, and IGT performance (Toplak, Sorge, Benoit, West, & Stanovich, 2010). The review, incorporating neurological, neuropsychiatric, and non-clinical child and adult studies, found that only a small proportion of studies reported a significant relationship between IGT performance and cognitive abilities. Accordingly they found that of studies examining the association between IGT performance and inhibition only 24% of correlational analyses reported a statistically significant association. In the set-shifting, working memory and intelligence domains, only 13%, 16% and 32% of correlational analyses, respectively, reported a significant association. Of the minority of studies that reported statistically significant effects, associations were small to modest ($r = .18, .15, .06, .23$ for the inhibition, set-shifting, working memory and intelligence domains respectively) and confidence intervals were large, indicating that considerable variability in performance on the IGT is not captured by current measures of executive function and intelligence and supporting the separability between decision making on the IGT and cognitive abilities. As highlighted by Toplak et al. (2010) the small number of associations that were obtained between IGT performance and these neuropsychological indices may be indicative of the presence of multiple deficits in functioning in clinical populations, in contrast to findings in healthy samples (Davis et al., 2008; Demaree et al., 2010). As such, decision making performance may be somewhat contingent on intact neuropsychological abilities, for example, to maintain and update information during the IGT task. In addition, perhaps it is too simplistic to assume the moderating effect of individual difference variables would be true of both clinical and non-clinical samples.
Research examining the personality correlates of poor IGT performance is very much in its infancy. An exploratory investigation in a large sample of healthy adults found higher scores on measures of impulsivity using the Barratt Impulsivity Scale ([BIS]; Patton, Stanford, & Barratt, 1995), sensitivity to reward and to punishment measured by the Sensitivity to Punishment and Sensitivity to Reward Questionnaire ([SPSRQ]; Torrubia, Avila, Molto, & Caseras, 2001), and addictive personality assessed by the Addiction Scale of the Eysenck Personality Questionnaire-Revised ([EPQ-R]; Eysenck & Eysenck, 1991), were all significantly associated with some performance deficits (e.g. failure to learn) on the IGT (A'B'C'D'). Contrary to expectation, none of these personality traits were related to performance on the variant, E' F' G' H' version of the task, although these null findings may relate to a lack of counterbalancing the order of task presentation. Sensitivity to punishment showed the largest effect size and was the only personality variable to correlate with all measures of performance deficits on the IGT (impaired vs. non-impaired, learner vs. non-learner, number of advantageous choices). The pattern of findings for impulsivity was similar to those of Crone, Vendel and Van der Molen (2003), who found performance on the IGT was modulated by cognitive disinhibition, a key feature of impulsivity, as measured by a subscale of Zuckerman's Sensation Seeking Scale (Zuckerman, Eysenck, & Eysenck, 1978). However, inconsistent with the findings of Davis et al. (2007) cognitively disinhibited participants were impaired on the standard but not the variant (E' F' G' H') IGT. Behavioural disinhibition failed to influence IGT performance. As cognitively high-disinhibited individuals performed equal or even better than low-disinhibited individuals on the variant reversed version of the IGT (E' F' G' H') they concluded that participants who were bad performers on the IGT were best characterised as reward-prone rather than as future-insensitive. However, recent research has indicated that among healthy subjects, impulsivity (or cognitive disinhibition) and reward sensitivity do not represent the same construct (Franken, Muris, & Rassin, 2005). This argument is supported by findings that individual differences in sensitivity to reward and to some extent self-reported decision making style, but not impulsive personality traits, were found to play a role in IGT performance (Franken & Muris, 2005). Sensitivity to reward and punishment was assessed by the Behavioural Inhibition System and Behavioural Approach System (BIS/BAS) questionnaire (Carver & White, 1994), impulsive personality traits by the Dickman Impulsivity Inventory ([DII]; Dickman, 1990) and decision making by the Adolescent Decision Making Questionnaire ([ADMQ]; Tuinstra, Van Sonderen, Groothoff, Van den Heuvel, & Post, 2000). Accordingly, using linear regression, they found higher scores on the IGT were positively associated with scores on the BAS Reward
Responsiveness scale (but not to BIS) and negatively associated with only the panic decision making style, though they argue that sample characteristics (young college students) may have contributed to the latter finding. In summary, it seems impulsivity and cognitive disinhibition may be related to impaired IGT performance. The involvement of sensitivity to reward (and punishment) in this relationship is unclear, with suggestions that higher reward sensitivity may enhance (Franken & Muris, 2005) or have deleterious effect on performance (Crone et al., 2003; Davis, Patte, Tweed et al., 2007).

Within the domain of disordered eating groups, of particular relevance to this thesis, the impact of mood and severity of eating pathology, has been evaluated in relation to IGT performance. For example, in assessing the decision making of BN patients, Beck Depression Inventory ([BDI-II]; Beck, Steer, & Brown, 1996) scores and the Bulimia Test Revised ([BULIT-R]; Thelen, Farmer, Wonderlich, & Smith, 1991), which assesses the frequency of bulimic behaviours were evaluated (Boeka & Lokkenz, 2006). Accordingly, BULIT-R score was the only variable found to uniquely predict IGT performance. Correlational analyses revealed that as bulimic severity (as measured by the BULIT-R and Eating Disorder Examination Questionnaire ([EDE-Q]; Fairburn & Beglin, 1994)) increased, IGT performance decreased. There were no significant correlations between BDI-II scores and IGT performance. These findings are in contrast to a study of AN individuals, healthy controls and individuals with long-term recovery from AN, which found that BDI scores were significantly correlated with IGT performance in all three groups (Tchanturia et al., 2007). To our knowledge, the only study to investigate the relationship between eating pathology and IGT performance in an overweight and obese population found emotional eating did significantly mediate the relationship between decision making and BMI, each having an independent relationship with BMI (Davis et al., 2004).

A related aspect of eating pathology that has been investigated in relation to IGT performance in healthy women is the role of restraint eating behaviour as measured by the Dutch Eating Behaviour Questionnaire ([DEBQ]; Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986). Several researchers have argued that the population of restraint eaters consists of two populations: unsuccessful dieters who score high on restraint and high on disinhibition and successful dieters who score high on restraint but low on disinhibition. Drawing on this view, Kuijer, de Ridder, Ouwheand, Houx, and Van den Bos (2008) examined the relationships between restraint eating in combination with individual differences in self-control (akin to disinhibition) and IGT performance; a task which they argued mimics the uncertainty of
dieting. Results showed that self-control (assessed by the dispositional Self-Control Scale (Tangney, Baumeister, & Boone, 2004)) moderated the association between restraint and decision making with restraint eating related to worse decision making when self-control was low, but to better decision making when self-control was high. These findings point to a role of self-control (or disinhibition) in IGT performance, this time in moderating the impact of self-reported eating style on decision making.

A final factor that should be considered here which may impact IGT performance is apathy (or lack of motivation) (Dunn et al., 2006). Thus, rather than being unable to make advantageous decisions on the IGT, VmPFC patients and other impaired groups may simply not care enough about the negative outcomes to actively avoid them. Such apathy could produce behavioural deficits and a failure to generate anticipatory SCRs. In support, a recent study drawing a distinction between different aspects of future-directed thinking found VmPFC damage left temporal discounting (the subjective devaluation of reward as a function of delay) intact but impaired future time perspective (a measure of the length of an individual’s self-defined future) (Fellows & Farah, 2005b). This deficit in future time perspective was found to correlate with symptoms of apathy rather than impulsivity, suggesting that apathy may deserve more attention in understanding impaired future thinking and decision making following frontal lobe damage (Dunn et al., 2006), and by extension in understanding the deficits of other groups impaired on the IGT.

1.6 Critique of the IGT

1.6.1 Strengths of the paradigm

Dunn et al. (2006) provide a comprehensive overview of the strengths and weaknesses of the IGT paradigm and its ability to support the SMH. A brief consideration of salient points will be made here. Dominant strengths of the paradigm include the fact that impairment on the task following brain damage to the neural regions implicated in the SMH (e.g. VmPFC, amygdala), and normative performance in healthy controls, have been extensively validated within the Iowa laboratory and have been generally replicated outside of it. It is seen to be robust to changes in its parameters, for example, using real financial rewards or facsimile money, different time delays on the task, or when using a manual or computerised form of the task (Dunn et al., 2006). It has proved to be a sensitive measure of impaired decision making in a wide range of neurological and psychiatric conditions characterised by real life decision
making impairments as previously discussed. Further, its predictive validity has been established, for example, in predicting response to pharmacotherapy in OCD (Cavedini, Bassi, Zorzi, & Bellodi, 2004), as well as to a behavioural treatment program for AN (Cavedini, Zorzi et al., 2006). However, as would be expected from a paradigm which has stimulated a considerable amount of research, a substantial body of criticism has also been levied at the task.

1.6.2 Weakness of the IGT

1.6.2.1 Cognitive penetrability of the reward / punishment schedule

The first issue to be addressed here centres on the cognitive penetrability of the task's reward / punishment schedule. In replicating Bechara et al.'s (1997) original study measuring conscious awareness of task contingencies on the IGT using more detailed, focused questions, Maia and McClelland (2004) found that advantageous performance on the task was nearly always accompanied by verbal reports of reasonably accurate quantitative and qualitative knowledge about the outcomes of the decks that was sufficient to guide behaviour. This lead the authors to conclude that the IGT can be performed through access to conscious, explicit knowledge, and that it is therefore inaccurate to claim that task acquisition necessarily requires the generation of nonconscious 'somatic marker' signals. These claims were refuted by Bechara and colleagues (Bechara, Damasio, Tranel, & Damasio, 2005) who argued that the emotion-based rather than the implicit nature of biasing signals is core to the SMH and it is therefore not problematic if the IGT is more transparent than previously thought. Further, they argued that SCRs appear sufficiently early in the IGT to precede conscious knowledge. Dunn et al. (2006) believe that participants have at least some conscious awareness of the reinforcement contingencies used in the task, and that it is unclear whether this is best understood as a full rational understanding of the reward / punishment schedule or simply a heuristic strategy whereby participants can label the valance of the decks as good or bad. The former interpretation would undermine the utility of the SMH, while the latter, though challenging the penetrability of the task, is consistent with the broader claims of SMH that emotion (even as a conscious verbal affective label) can guide decision making.

1.6.2.2 Psychophysiological issues

A second issue highlighted by Dunn et al. (2006) concerns the interpretation of psychophysiological data on the IGT. For example, Crone et al. (2004) found that 'somatic
marker' signals (anticipatory SCR, heart rate slowing) were greater for disadvantageous than advantageous decks for good IGT performers only. The fact that moderate performers could acquire the task without such physiological differentiation between the decks suggests 'somatic markers' are not necessary or sufficient to do well on the paradigm. More generally, criticisms centre on the limited replication of psychophysiological data on the IGT, and over-reliance on SCR as the psychophysiological measurement of choice (Dunn et al., 2006). Controversy also exists over whether anticipatory (Crone et al., 2004) or feedback SCR (Suzuki et al., 2003) is a more appropriate measure of somatic marker activity.

Of the studies which do incorporate SCR data, there are discrepancies in methodological approach, for example inter-trial intervals range from a minimum of six (e.g. Bechara et al., 1999; Lawrence et al., 2006) to much longer intervals of 10 (Crone et al., 2004) or 20 seconds (Suzuki et al., 2003). There is similarly substantial variation in SCR analysis, including area under the curve analysis (Bechara et al., 1999), average total amplitude (Liao et al., 2009; Tchanturia et al., 2007), amplitude of the first SCR peak (Carter & Pasqualini, 2004), amplitude of the largest SCR peak (Suzuki et al., 2003), or alternatively the number of SCR fluctuations (Lawrence et al., 2006). This variability in the analysis of SCR activity undoubtedly has an impact on the ability to compare decision making capacity across studies employing potentially disparate psychophysiological methodologies, and in terms of the broader conclusions drawn from these studies regarding the SMH.

On a related note, the psychophysiological data linking 'somatic marker' generation with successful performance are correlational only, and so no causal conclusions about the role of body-state feedback on decision making can be reliably drawn. Indeed, the study of patients with altered feedback from the body, such as in spinal cord damage or pure autonomic failure (PAF), have failed to support the SMH, with such patients showing normal or improved performance on the IGT. In the case of spinal cord damage, it is possible that feedback from the hormonal route and nerves outside the spinal cord are more important than the afferent feedback sent via the spinal cord, or that people who have lived with long-term spinal cord damage have adapted and make more use of the 'as-if' loop. Explaining away such negative findings with recourse to peripheral feedback remaining intact or the 'as-if' loop, raises questions about the testability of the SMH (Dunn et al., 2006). Further, in the case of PAF, patients have both body-state feedback and regions of the 'as-if' loop compromised, and so the lack of impairment in this group is difficult to explain (Dunn et al., 2006).
1.6.2.3 Variability in control participant performance

A third area of contention is that of the variability in control participant performance on the IGT, whereby some control participants do not learn to select from the advantageous decks over time (Dunn et al., 2006). For example, Bechara and Damasio (2002) found that 33% of their normal controls performed disadvantageously on the IGT, some of whom showed normal anticipatory SCR patterns and others of whom showed a profile similar to VmPFC patients. The former group, with impaired behavioural performance but normal anticipatory SCRs were later characterised as high-risk takers, consciously overriding 'somatic markers' with conscious deliberation. Similarly, Crone et al. (2004) in their sample of healthy participants, identified subgroups of bad, moderate and good performers. Such variability in control participant performance has led to calls that IGT performance should perhaps additionally be compared to objective criteria (e.g. chance levels). Further, it has been suggested that measures of the quality of everyday decision making should be included as an ecological check to assess if control participants who are impaired on the IGT, are similarly impaired in everyday decision making.

1.6.2.4 ABCDvs. A'B'C'D'

A potentially problematic subject, which is relatively ignored in the literature, is the change from the original IGT reward / punishment scheduled introduced by Bechara and his colleagues in 2000 (Bechara, Tranel, & Damasio, 2000). This new version of the task, A'B'C'D', though analogous to the original task, ABCD (Bechara et al., 1994), changed the frequency or magnitude of delayed punishment relative to immediate reward. Accordingly, the discrepancy between reward and punishment in the disadvantageous decks (A' and B') was rendered larger in the negative direction (towards larger loss) while the difference between reward and punishment in the advantageous decks (C' and D') was made larger in the positive direction (towards larger gain). In deck A' the frequency of delayed punishment was increased by 10% in every block of ten cards, while the magnitude of an individual delayed punishment remained the same. In deck B', the magnitude of an individual delayed punishment, relative to an immediate reward, was increased in every block of ten cards (by an equal amount to the increase in A') but the frequency of delayed punishment remained the same. There were parallel increases in the frequency and magnitude of delayed punishment in decks C' and D'. Also the total number of cards in each deck was changed from 40 to 60. The rationale behind the change in structure was based on the move from the original manual version of the task (Bechara et al., 1994) to a computerised version. Accordingly, when Bechara and colleagues
tried to convert the task to a computerised version they encountered problems with normal controls whereby many of them would assume that the computer was punishing them at random and their performance would change (A. Bechara, personal communication, February 11, 2010). To avoid this they introduced the A'B'C'D' schedule in their computerised IGT (Bechara, 2007) and in studies thereafter (e.g. Bechara & Damasio, 2002; Bechara et al., 2001; Bechara et al., 2002). While some researchers have adopted this later version of the task (e.g. Fein et al., 2004), the majority of other researchers have seemingly programmed the IGT based on the original version of the task, for example across domains such as drug addiction (Mazas, Finn, & Steinmetz, 2000; Rotheram-Fuller, Shoptaw, Berman, & London, 2004; Whitlow et al., 2004), psychiatric (Bark et al., 2005; Nielen, Veltman, de Jong, Mulder, & den Boer, 2002; Shurman et al., 2005) and health (Brogan, Hevey, & Pignatti, 2010; Cavedini, Bassi, Ubbiali et al., 2004; Davis et al., 2004; Pignatti et al., 2006) conditions. Further, in some studies it is not quite clear which version of the task was used (Apkarian et al., 2004; Bosanac et al., 2007). Given the concerns by Bechara (A. Bechara, personal communication, February 11, 2010) on the effects of the computerised original reward schedule on IGT performance in control participants, questions are raised about the validity of applying this schedule. Further, the ability to draw comparisons across clinical groups using different versions of the task (ABCD or A'B'C'D') is limited. As the only known paper to address the different versions of the IGT (Bechara, Tranel et al., 2000) found no significant differences on performance, this issue may not be critical. However, sample size was small, and given the wide-spread application of the IGT it would seem necessary that this difference at least be addressed and quantified in the literature.

1.6.2.5 Specificity

The specificity or discriminative validity of the IGT paradigm was another issue of concern identified by Dunn et al. (2006), as mentioned earlier in the context of the need to further decompose IGT performance. Accordingly, the point was made that as the majority of patient groups show deficits on the task, this undermines the usefulness of the paradigm in better understanding decision making difficulties across different disorders. Perhaps more papers which attempt to compare decision making profiles on the IGT, such as recent papers in disordered eating (Bosanac et al., 2007; Brogan et al., 2010; Davis et al., 2010), are required to further knowledge in this area.
1.6.2.6 Task design

The design of the IGT has also been criticised on a number of levels. For example, deck position is not counterbalanced in the task, meaning preferential selection from good or bad decks could reflect a location bias rather than a genuine decision making deficit (Dunn et al., 2006). The task is also designed in such a way as an initial preference is induced for the disadvantageous decks (due to high rewards on these decks) (Fellows & Farah, 2005a) necessitating intact reversal learning for successful task performance. This has lead to arguments that trials should be classified based on net outcomes, rather than on eventual reinforcement.

1.7 Aims of the thesis

The aim of this thesis was to elucidate affective and cognitive influences on decision making in morbid obesity. The thesis focused on a morbidly obese population for a number of reasons. Within the current rise in obesity over the last two decades, the most rapid increase in obesity has been seen in those considered to be extremely obese (BMI ≥ 40) (Boeka & Lokken, 2008). For example, in the United States, the BMI subgroups experiencing the most rapid growth are 35 or higher (23 million) and 40 or higher (8 million) (Buchwald et al., 2004). From an Irish perspective, while levels of overweight have remained quite stable over the past decade, levels of obesity have risen by 5%, with this rise particularly prevalent in women (7%) (Harrington et al., 2008). The vicious cycles of the 'runaway weight gain train' described earlier may contribute to this increasing right skewness of the frequency distribution of BMI (Swinburn & Egger, 2004). As alluded to previously, the rise in the prevalence of obesity is associated with increases in the rates of obesity comorbidities (e.g. type 2 diabetes, hyperlipidemia, hypertension, obstructive sleep apnea, heart disease, stroke, asthma, back and lower extremity weight-bearing degenerative problems, several forms of cancer, and psychosocial problems such as depression), which are responsible for more than 2.5 million deaths per year worldwide. The loss of life expectancy due to morbid obesity is profound. For example, in comparison with a normal weight individual, a 25-year-old morbidly obese man has a 22% reduction in expected remaining lifespan, representing an approximate loss of 12 years of life (Buchwald et al., 2004). Aside from these population trends highlighting the vulnerability of this morbidly obese group, the fact that the dominant neuropsychological method applied here, the IGT, has been derived largely from the field of addiction research (e.g. Bechara & Damasio, 2002), points to the relevance of this method for a severely obese population. Indeed, the parallels between obesity and conventional addiction shall be
elucidated in Chapter 2. Finally, from a treatment perspective, with regards to the extremely obese, there is considerable evidence that traditional nonsurgical treatments, such as diet, exercise and pharmacotherapy, are ineffective for achieving long term, substantial weight loss (Buchwald et al., 2004). As such, by better understanding the decision making capacities of this group, it is anticipated that research of this kind could inform new behavioural treatments for obesity. If it were possible to change decision making in this extreme group, and by doing so positively impact upon weight outcomes, such strategies could potentially have tangible effects on weight outcomes in both overweight and moderately obese groups.

The structure of the thesis is as follows: The next two empirical chapters address affective decision making in a morbidly obese population taking a neuropsychological approach. Accordingly, Chapter 2 reports preliminary investigations using the IGT to demonstrate impairments in affective decision making under uncertainty in a morbidly obese population. Chapter 3 extends this work through using the IGT to investigate parallels between impaired decision making in morbid obesity and other forms of disordered eating, such as AN and BN. Based on conclusions drawn in Chapters 2 and 3, Chapter 4 aims to further specify decision making impairments in obesity using psychophysiological measures, individual difference measures (e.g. intelligence) and additional neuropsychological tasks which tap cognitive aspects of decision making. In adopting a more naturalistic approach, the final empirical chapter, Chapter 5, employs the food diary methodology as a more ecologically valid way of assessing decision making and particularly the role of affective and cognitive influences. Accordingly, using the concepts of emotional, external and restraint eating, the self-reported relationships between these eating styles and actual food intake are investigated. Chapter 6 is a general discussion where the main findings of the empirical chapters (Chapters 2 – 5) are summarised and conclusions regarding affective and cognitive influences on decision making are drawn.
2.1 Introduction

2.1.1 Decision making and eating disorders

The preference for high immediate reward (despite long-term negative consequences) observed in VmPFC patients, and discussed in Chapter 1, can be seen to characterise the behaviour of individuals with pathological eating behaviours. For example, immediate reward through the relief of anxiety or tension is obtained in AN through the chronic restriction of food intake, in BN through binge eating / purging, and in BED through compulsive overeating, despite often severe long-term psychological and medical consequences. As such, the IGT, the most frequently used measure of affective decision making under uncertainty, has been used to profile decision making deficits in these disordered eating groups.

IGT Impairment was found in 59 inpatients (96% female) with acute stage AN, compared with 82 healthy control participants (52% female) (Cavedini, Bassi, Ubbiali et al., 2004). Impairments were not found on other basic measures of cognition (e.g. the Wisconsin Card Sorting Task [WCST]; Berg, 1948), leading the authors to conclude that decision making deficits were unlikely to be a non-specific reflection of the negative effects of starvation. In addition, impairments were independent of illness severity such as Y-Cornell scores or BMI (Cavedini, Bassi, Ubbiali et al., 2004). The pattern of performance differed between subtypes of AN; the AN restricting subtype (AN-R) demonstrated an increasing preference for the disadvantageous decks, while the AN binge eating / purging subtype (AN-BE) did not follow a clearly advantageous or disadvantageous strategy on the IGT. The IGT has also been employed prognostically in the assessment of treatment outcomes in 38 female AN patients, following a cognitive-behavioural treatment programme (Cavedini, Zorzi et al., 2006). Using receiver operated curve analysis based on a larger (n = 100) independent sample of anorectic patients, they identified a cut-off of >51 cards selected from the disadvantageous decks as indicative of bad performance. AN patients with a ‘good’ IGT decision making profile at baseline showed significantly greater improvements in nutritional status after the treatment. As such, the authors suggest that anorectic patients with normal decision making ability
succeeded in taking advantage of a treatment programme based on the operant conditioning paradigm during cognitive-behavioural therapy, while the inability to identify an adequate decision making strategy prevented those with bad IGT performances from taking significant advantage of the same programme. In support of the SMH (A. R. Damasio, 1994), IGT impairment in 29 females (both outpatient and inpatient) with AN was associated with significantly attenuated anticipatory somatic marker signals, compared with both healthy controls (n = 29) and females with long-term recovery (healthy BMI and minimum one year’s menstruation) from AN (n = 14) (Tchanturia et al., 2007).

In a sample of undergraduate females with a current (n = 16) or past (n = 4) diagnosis of BN, significant impairment was found in comparison to healthy controls (n = 20) (Boeka & Lokkenz, 2006), with IGT performance negatively correlated with bulimic symptomatology as measured by the BULIT-R (Thelen et al., 1991) and EDE-Q (Fairburn & Beglin, 1994). However, failing to support the SMH and the specified role of affective biasing signals, IGT deficits in 26 female BN outpatients were not associated with impaired anticipatory SCR (Liao et al., 2009). In addition, deficits were unrelated to depression or impulsivity scores, but were related to obsessive-compulsive symptoms in line with findings that impairments in IGT performance have been found in obsessive-compulsive patients (Lawrence et al., 2006).

In contrast to the above findings, combining underweight and ‘weight-recovered’ AN (n = 16) and BN (n = 13) females, research found no significant differences between any of the eating disorder groups and controls on the IGT (Bosanac et al., 2007); however, the eating disorder groups scored lower and lack of significance may be due to low statistical power. A second study comparing eating disordered groups currently in treatment found IGT deficits in AN (n = 22), BN (n = 17), and obese (n = 18) females compared to a comparison group (n = 20), but with no significant differences between these clinical groups, highlighting possible shared decision making impairments in the inability to regulate food intake (Brogan et al., 2010). In contrast to decision making in uncertain circumstances as in the IGT (at least until task contingencies are learned), decision making in explicit situations has also been investigated in disordered eating. Using the GDT (Brand, Kalbe et al., 2005), in which decisions are required between different alternatives explicitly related to a specific amount of gain or loss, with stable, obvious winning probabilities, females with BN (n = 14) chose disadvantageous alternatives significantly more frequently than did comparison participants, with task performance related to executive functioning (Brand, Franke-Sievert, Jacoby, Markowitsch, &
Tuschen-Caffier, 2007). Accordingly, the frequency of disadvantageous decisions in the GDT was significantly correlated with the interference sheet of the Color-Word Interference Test and the Trail Making Test Part B (see Lezak, Howieson, & Loring, 2004 for test references), indicating that the longer patients needed for the interference sheet of the Trail Making Test Part B, the more disadvantageous alternatives they selected in the GDT. However, task performance was not related to other neuropsychological functions (intelligence, reasoning, speed of information processing, working memory, attention, executive function, and theory of mind functions), personality or disease-specific variables (e.g. duration of BN).

2.1.2 IGT, addiction and obesity

As mentioned in Chapter 1, the IGT has been used to profile the decision making capacities of a wide range of clinical groups, including SDI (Bechara & Damasio, 2002; Bechara et al., 2001), due to the behavioural similarities between some SDI and VmPFC patients in terms of their preferences for choices that bring immediate benefit even if coupled with negative future consequences (Bechara et al., 2001). Substance dependent groups showing impaired performance on the IGT include: polysubstance abusers (Grant, Contoreggi, & London, 2000); long-term heavy marijuana users (Whitlow et al., 2004); cocaine abusers (Bartzokis et al., 2000; Monterosso, Ehrman, Napier, O'Brien, & Childress, 2001); alcoholics (Mazas et al., 2000); heroin addicts (Petry, Bickel, & Arnett, 1998); and opiate-dependent individuals (Rotheram-Fuller et al., 2004). Thus, the IGT can be conceptualised as a task which looks at the prefrontal systems underlying the 'will power' to control behaviour (Pignatti et al., 2006).

Volkow and Wise (2005) argued for the potential cross-fertilisation between the fields of addiction and obesity research. They propose that obesity and addiction (or drug dependence) are special cases of the consequences of ingestive behaviour gone awry, with each developing in some but not all individuals and each subject to genetic predispositions and the availability of a powerful reinforcer. A range of studies now supports the contention that food, particularly highly palatable food, can be conceptualised as a 'drug' and has the potential for abuse (Bruinsma & Taren, 1999; Pelchat, 2002), especially in the case of compulsive overeating (Davis & Carter, 2009). Such studies allude to the common neural circuits thought to underlie food and drug rewards, including opioid, serotonergic and dopaminergic pathways but also common behavioural characteristics, such as the fact that eating disorders tend to cluster with drug and alcohol abuse both in individuals and families.
(Del Parigi, Chen, Salbe, Reiman, & Tataranni, 2003). The most clearly established common neurobiological mechanism of food and drug intake is their ability to activate the dopamine-containing link in brain reward circuitry (Volkow & Wise, 2005). In addition, in examining causal attributions in an obese population, an 'addictive personality' was identified by morbidly obese participants as a distal cause of their obesity (Brogan & Hevey, 2009). Such casual attributions are seen to have a profound effect on every stage of clinical management (Sensky, 1997), guiding motivated cognitions and behaviours and affecting overall psychological adjustment (Roesch & Weiner, 2001).

Adopting this cross-fertilisation approach between the fields of addiction and obesity research, Davis et al. (2004) hypothesised that poor decision making abilities may characterise many overweight individuals in the same manner as SDI. As such, they applied the IGT, to understand decision making in overweight and obesity. Accordingly, they found evidence that poor decision making was associated with being overweight (BMI > 25) in a small heterogeneous sample of female overweight and obese participants (n = 15), in comparison to normal weight female participants (n = 26), all of whom were recruited from advertisements placed in the university and at local hospitals. High-BMI participants showed significant performance impairments on the IGT and failed to learn on the task as evidenced in their flat learning curve across the task's five performance blocks, and their net deficit in advantageous choices on each block. In addition, they hypothesised that the relationship between overweight and poor decision making would be mediated by emotional eating, as assessed by the Emotional Eating Scale ([EES]; Arnow et al., 1995). Using the meditational procedures outlined by Baron and Kenny (1986) they found that while emotional eating did mediate the relationship between decision making and BMI, its effect was relatively small, leading the authors to conclude that both decision making and emotional eating had relatively independent relationships with BMI. As such, poor decision making does not seem to contribute to an increase in BMI by fostering overeating during periods of negative emotion (Davis et al., 2004).

As highlighted in Chapter 1, the frequency distribution of BMI within the obesity epidemic has been found to be increasingly skewed to the right, whereby the already overweight and obese have gained the most weight (Swinburn & Egger, 2004). Accordingly, the work of Davis et al. (2004), which used the IGT to characterise decision making deficits in overweight and obese populations, was extended to a small sample of 20 moderately obese
(BMI > 34) individuals (6 males, 14 females) (Pignatti et al., 2006) who were free from both previous psychological or psychiatric diagnoses and from disordered eating as assessed by a number of eating measures such as the Eating Disorders Inventory-2 ([EDI-2]; Garner, 1991). Compared with 20 normal weight controls (10 males, 10 females) the morbidly obese participants were similarly found to be significantly impaired in overall performance on the IGT (Pignatti et al., 2006). However, unlike the overweight and obese participants in Davis’ (2004) study, both groups demonstrated learning on the task with significant improvements across blocks two – five of the task’s five trial blocks. More recently, in a community sample, no differences on the IGT and a delay discounting measure (Richards, Zhang, Mitchell, & de Wit, 1999) were found between obese women with \( n = 65 \) and without BED \( n = 73 \), compared to a normal weight control group \( n = 71 \) (Davis et al., 2010). Accordingly, on the IGT both the obese and BED groups demonstrated lower mean scores and less task learning than the control group, and had higher indifferent points on the delay discounting task. However, when education was added to the model, those with a higher education performed better on both tasks, and group differences were no longer significant (Davis et al., 2010). As there were no differences between the obese and BED group, the authors concluded that the tendency to gain weight is strongly related to poor self-regulation, irrespective of the means by which the over-consumption of food occurs. Of note, explicit risk tasking has also been investigated in BED using the GDT (Svaldi, Brand, & Tuschen-Caffier, 2010). Similar to women with BN, a community sample of women with BED \( n = 17 \), who were free from previous psychological or psychiatric diagnoses, made significantly more disadvantageous decisions compared to healthy controls \( n = 18 \) and showed an inability to advantageously use feedback processing. These deficits were unrelated to eating pathology as assessed by the EDE-Q (Fairburn & Beglin, 1994) and depression (Beck, Steer, & Carbin, 1988).

2.1.3 Aims of the current study

The purpose of this study was to replicate the preliminary work of Pignatti et al. (2006) with a larger sample of extremely obese individuals and compare their performance to a healthy comparison group matched for age, gender and education. In addition, as per the previous research discussed in Chapter 1 on the use of individual difference variables that may relate to IGT performance, measures of eating pathology (Arnow et al., 1995; Van Strien, Frijters, Bergers et al., 1986) were included to assess their relationship with IGT performance. In addition, in line with Franken and Muris (2005), a measure of self-reported decision making style (Strathman, Gleicher, Boninger, & Edwards, 1994) which relates to impulsivity was
incorporated, to assess its relationship with the IGT, a behavioural measure of decision making.

The following hypotheses were made: 1) individuals with obesity would have significantly lower net scores on the IGT than a matched healthy weight comparison group; 2) the obese group would fail to learn on the IGT; 3) IGT net scores would be unrelated to eating pathology as assessed by standardised psychometric measurements, based on previous findings in overweight and obese populations (Davis et al., 2004; Svaldi et al., 2010).

2.2 Method

2.2.1 Participants & procedure

Obese group: A convenience sample of 42 participants (12 male, 30 female), from 26 – 74 years of age, were recruited by post from the records of a National Weight Management Clinic at a general hospital. Inclusion criteria were: attendance at the weight management clinic; ability to give informed consent; and a BMI >30. Comparison group: 50 participants (17 male, 33 female), from 20 – 75 years of age, matched for age, gender, and education, were recruited from the university’s participant panel. Participants were paid a small stipend to cover expenses. Exclusion criteria common to both groups were underage (<18 years), and failure to complete the compulsory education period (minimum 8 years of formal education). The study received ethical approval from both the university and hospital ethics committees (see Appendix 2 for ethical approval documentation). After provision of written informed consent participants were tested in the university’s psychology department. Please see Appendix 3 for a full copy of the study protocol.

2.2.2 Measures

2.2.2.1 Anthropometric Measurements

BMI (weight in kilograms / height in meters squared) measurements were taken in the standing position, without shoes and in light clothing, and calculated to the nearest 0.1Kg and 0.01m. Weight was measured using a high capacity digital scales with a wide platform (Seca bathroom scales; Model 813) designed for use in the medical sector. Height was measured using a wall-mounted tape measure (Seca; Model 206).
2.2.2.2 Psychometric measures

Participants completed the EES (Arnow et al., 1995), a 25-item adjective checklist that assesses the desire to eat in response to negative mood. The scale has three subscales: anger/frustration (e.g. resentful, discouraged, inadequate), anxiety (e.g. jittery, uneasy, worried) and depression (e.g. sad, lonely, bored). Participants rate their desire to eat in response to these mood states on a scale of 1 (no desire to eat) – 5 (an overwhelming urge to eat). Higher score indicate higher levels of emotional eating. The authors report good construct, discriminative and criterion validity, as well as acceptable alpha coefficients (Arnow et al., 1995). In addition, the scale has been validated with both obese binge eaters and non-eating disordered women (Waller & Osman, 1998). All three EES subscales (Anger/Frustration, Anxiety, and Depression) are reliable predictors of the EDI-2 (Garner, 1991) Bulimia, Ineffectiveness and Interpersonal Distrust, while EES Depression scores are associated with EDI-2 Body Dissatisfaction. Cronbach’s alpha for the Anger/Frustration (.94), Anxiety (.89) and Depression (.81) subscales of the EES, indicated good internal consistency in the sample.

The 33-item DEBQ (Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986) has three subscales measuring restraint (10 items), emotional (13 items) and external eating (10 items). The restraint and externality scales from DEBQ were administered here, elevated scores on which are associated with eating pathology and obesity. Examples of items from the restraint scale include: 'Do you try to eat less at mealtimes than you would like to eat'; and 'how often do you refuse food or drink offered because you are concerned about your weight?' From the external eating scale, items include: 'If you see others eating, do you also have a desire to eat' and ‘if you see or smell something delicious do you have a desire to eat it?’ Response categories range from 1 (never) to 5 (very often). Higher scores reflect higher levels of the given eating style. The DEBQ subscales have good internal reliability and good concurrent, construct and predictive validity (Van Strien, 2005). In the current sample, Cronbach’s alpha for the restraint (.91) and externality (.84) subscales from the DEBQ indicated good internal consistency.

Finally, the Consideration of Future Consequences scale ([CFC]; Strathman et al., 1994) was administered, which assesses the extent to which people consider distant versus immediate consequences of potential behaviour, and therefore is akin to a self-report measure of impulsivity. The scale has 12 items, examples of which include: 'I consider how things might be in the future, and try to influence those things with my day-to-day behaviour'
and ‘I only act to satisfy immediate concerns, figuring the future will take care of itself’. Participants must rate the extent to which the 12 statements are true of them using the response categories from 1 (extremely uncharacteristic) to 5 (extremely characteristic). Higher scores are indicative of a greater consideration of future consequences. The scale has acceptable reliability and validity (Strathman et al., 1994) and Cronbach’s alpha (.84) for the current sample indicated good internal consistency.

2.2.2.3 Decision making

Decision making was assessed using a computerised analogue version of the original IGT (Bechara et al., 1994). Participants are given a loan of $2000 play money and are told to make choices that maximise their gains, with minimal instructions regarding the rules of the task. Over a series of 100 trials, they have to choose between four identical decks of cards (A, B, C, and D) some of which yield high immediate gain ($100) but larger future losses (bad decks, A and B) and others which yield lower immediate gain ($50) but a smaller future loss (good decks, C and D). Thus, participants have to forego short-term benefit for long-term profit. The pre-programmed schedules of reward and punishment were based on the original version of this task (Bechara et al., 1994). The reward structure for the IGT is illustrated in Table 2-1.

<table>
<thead>
<tr>
<th>Deck</th>
<th>Reward</th>
<th>Punishment</th>
<th>Net Profit (Over 10 trials)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Win $100 (100% trials)</td>
<td>Lose $150 to $350 (50% of trials)</td>
<td>-$250</td>
</tr>
<tr>
<td>B</td>
<td>Win $100 (100% trials)</td>
<td>Lose $1250 (10% of trials)</td>
<td>-$250</td>
</tr>
<tr>
<td>C</td>
<td>Win $50 (100% trials)</td>
<td>Lose $25 to $75 (50% of trials)</td>
<td>+$250</td>
</tr>
<tr>
<td>D</td>
<td>Win $50 (100% trials)</td>
<td>Lose $250 (10% of trials)</td>
<td>+$250</td>
</tr>
</tbody>
</table>

In brief, every 10 cards from decks A and B gain $1000, but deck A incurs five unpredictable punishments ranging from $150 - $350 bringing the total loss to $1250, while deck B incurs one large punishment of $1250. Every 10 cards from deck C or D amounts to a gain of $500, but the punishments incurred are also smaller ranging from $25 - $75 in deck C with a total loss of $250, and a single loss of $250 in deck D. Thus, per 10 cards, decks A and B
lead to an overall net loss of $250 while decks C and D lead to an overall net gain of $250. On decks A and C, punishments are more frequent, but of smaller magnitude, while on decks B and D, punishments are less frequent, but of higher magnitude. Decks A and B are thus 'disadvantageous' because they cost the most in the long run, while decks C and D are 'advantageous' because they result in an overall gain in the long run.

Following Whitlow et al. (2004), participants were told that the participant who accumulated the largest amount of money over the course of the study would win a real monetary bonus of $100, to motivate participants to perform as well as possible.

2.2.3 Data analysis

Performance on the IGT was measured by: 1) a global outcome score (net score); and 2) a net score for each consecutive block of 20 cards. Net scores are derived from the total number of cards chosen from the advantageous decks (C & D) minus those chosen from the disadvantageous decks (A & B). Lower scores represent poorer performance, and negative scores indicate a relative preference for choices from the disadvantageous decks. Analysis of IGT performance by blocks of 20 cards (blocks 1 - 5) provides an index of learning and the strategy used by participants across the task.

2.3 Results

2.3.1 Sample characteristics

The clinical demographic profile is summarised in Table 2-2. The groups were matched for age, gender, and education. The only significant difference between the groups was in BMI as expected ($t (90) = -11.30, p<.001$). Within the obese group, participant BMIs ranged from 30.6 - 74.4; 29% with class I obesity, 24% with class II, and the majority of participants, 48%, with class III obesity (WHO, 2000). Seventy-four percent of participants were currently involved in a weight management programme. Thirty-five participants (83%) had previously used prescription weight loss drugs and thirteen participants (31%) had received bariatric surgery. There were no significant differences between participants who had bariatric surgery and those who did not on any of the analyses below, and so results are presented for the entire sample.
Table 2-2. Clinical demographic profile of participants.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Comparison Group</th>
<th>Obese Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 50)</td>
<td>(n = 42)</td>
</tr>
<tr>
<td>(n(%))</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>17 (34.0)</td>
<td>12 (28.6)</td>
</tr>
<tr>
<td>Female</td>
<td>33 (66.0)</td>
<td>30 (71.4)</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (Years)</td>
<td>47.34 (16.34)</td>
<td>52.24 (10.89)</td>
</tr>
<tr>
<td>Education (Years)</td>
<td>13.86 (3.32)</td>
<td>13.95 (3.43)</td>
</tr>
<tr>
<td>BMI (Kg/m(^2))</td>
<td>24.36 (3.78)</td>
<td>41.45 (9.17) *</td>
</tr>
</tbody>
</table>

BMI = Body Mass Index; *\(p<.001\).

2.3.2 Behavioural performance on the IGT

The mean net scores of the comparison \((M = 16.36, SD = 29.00)\) and obese groups \((M = 0.48, SD = 23.59)\) on the IGT, are presented in Figure 2-1. The difference between the groups represented a standardised \(d\) of .6 (Cohen, 1988). A between groups ANCOVA, controlling for age, gender, education and BMI, found a significant difference on IGT global net scores \((F(1, 86) = 5.83, p=.02)\). In terms of card selection, both the control \((t(49) = 6.96, p<.001)\) and obese groups \((t(41) = 7.22, p<.001)\) showed a significant preference for infrequent punishment, selecting more cards from decks B and D than decks A and C.

![Mean Net Score on the IGT](Image)

**Figure 2-1.** Mean net scores \([C&D - A&B]\) on the IGT for the comparison and obese groups.
2.3.3 Task learning: Net scores blocks 1–5

Block net scores for the comparison and obese groups are presented in Figure 2-2. ANCOVA with group as the between subject factor and block as the within subject factor revealed a statistically significant interaction ($F(4, 360) = 2.59, p=.03$). Post hoc analyses revealed that the obese group showed no significant change over block ($F(4, 164) = 0.41, p=.80$) and that the groups differed significantly at block 3, ($F(1, 90) = 4.68, p=.03$), block 4 ($F(1, 90) = 10.94, p=.001$), and block 5 ($F(1, 90) = 7.06, p=.009$). Thus, while the comparison participants showed an increasing preference for advantageous decks across the task, the obese participants failed to learn across the task. Furthermore, the obese group did not demonstrate a clear strategy, with no shift in preference towards either the advantageous or disadvantageous decks. This pattern held after controlling for age, gender, education and BMI.

![Figure 2-2. Mean net scores (\([C&D - A&B]\)) on the IGT across each block of 20 trials. Positive scores reflect advantageous performance while negative scores reflect disadvantageous performance.](image)

This failure to learn across the task is similarly illustrated in Figure 2-3, which contrasts net scores on the IGT for the first 50 trials with net scores over the last 50 trials in both groups. Only the comparison group showed significant learning between the two time periods ($t(49) = -2.51, p=.02$). The difference in net scores between the two groups at time two (trials 51 – 100) was significant ($t(90) = 3.69, p<.001$).
2.3.4 Eating pathology and impulsivity

The participant profile on these measures is summarised in Table 2-3. The obese group had significantly higher mean scores on the restraint scale \( t(89) = -2.86, p=.005 \), and the anger / frustration \( t(90) = -2.65, p=.009 \) and depression \( t(87) = -3.09, p=.003 \) subscales of the EES.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Comparison (n = 50) Mean (SD)</th>
<th>Obese (n = 42) Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DEBQ Restraint</td>
<td>2.55 (1.06)</td>
<td>3.11 (.75)*</td>
</tr>
<tr>
<td>DEBQ Externality</td>
<td>2.98 (0.72)</td>
<td>3.00 (0.77)</td>
</tr>
<tr>
<td>EES Anger / Frustration</td>
<td>18.48 (8.93)</td>
<td>25.31 (11.95)*</td>
</tr>
<tr>
<td>EES Anxiety</td>
<td>15.98 (6.99)</td>
<td>17.56 (7.80)</td>
</tr>
<tr>
<td>EES Depression</td>
<td>11.35 (4.67)</td>
<td>14.12 (5.31)*</td>
</tr>
<tr>
<td>CFC</td>
<td>38.32 (8.78)</td>
<td>38.02 (9.86)</td>
</tr>
</tbody>
</table>

DEBQ = Dutch Eating Behaviour Questionnaire; EES = Emotional Eating Scale; CFC = Consideration of Future Consequences Scale; *p<.01.

The correlations between the psychometric measures and IGT performance are summarised in Table 2-4 for both the obese and the comparison group. In both groups, subscales of the EES scales were positively correlated with external eating. EES subscales also positively correlated with each other. Standard regression analyses showed none of the psychometric measures could uniquely predict overall net score or net score over the last 50
trials, for both the sample as a whole and for each group separately. Sub-group analysis within the obese group comparing high (BMI>41) versus low BMI groups, and good versus bad performers (IGT < 10) on the task similarly revealed no predictive relationships between these measures and IGT performance.

Table 2-4. Intercorrelations between psychometric measures for both the obese group (over the diagonal) and the comparison group (below the diagonal).

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>DEBQ Restraint (1)</td>
<td>x</td>
<td>-.22</td>
<td>-.05</td>
<td>-.03</td>
<td>-.05</td>
<td>-.07</td>
<td>-.23</td>
</tr>
<tr>
<td>DEBQ Externality (2)</td>
<td>-.18</td>
<td>x</td>
<td>.67**</td>
<td>.54**</td>
<td>.45**</td>
<td>-.11</td>
<td>.22</td>
</tr>
<tr>
<td>EES Anger/Frustration (3)</td>
<td>.10</td>
<td>.63**</td>
<td>x</td>
<td>.72**</td>
<td>.76**</td>
<td>-.02</td>
<td>.01</td>
</tr>
<tr>
<td>EES Anxiety (4)</td>
<td>.03</td>
<td>.58**</td>
<td>.81**</td>
<td>x</td>
<td>.72**</td>
<td>-.08</td>
<td>-.06</td>
</tr>
<tr>
<td>EES Depression (5)</td>
<td>.22</td>
<td>.47**</td>
<td>.70**</td>
<td>.62**</td>
<td>x</td>
<td>.09</td>
<td>.12</td>
</tr>
<tr>
<td>CFC (6)</td>
<td>.11</td>
<td>-.18</td>
<td>-.33*</td>
<td>-.34*</td>
<td>-.25</td>
<td>x</td>
<td>-.09</td>
</tr>
<tr>
<td>IGT Net Score (7)</td>
<td>.06</td>
<td>-.16</td>
<td>-.09</td>
<td>-.07</td>
<td>.07</td>
<td>.19</td>
<td>x</td>
</tr>
</tbody>
</table>

**p<.001; *p<.05

2.3.5 Clinical impairment on the IGT

According to the criterion established by Bechara et al. (2001), whereby the maximum net score of VmPFC patients was below ten, 69% of obese participants fell within this VmPFC range, compared to 38% of the comparison group. This difference was statistically significant ($\chi^2 (1, 92) = 8.81, p=.003$). Of note, performance of both groups was not uniform, with 16 comparison participants (32%) performing below the mean of the obese group, and 9 obese participants (21%) performing above the mean of the comparison group.

2.4 Discussion

2.4.1 IGT performance

Hypothesis one was confirmed with obese participants demonstrating significantly lower net scores on the IGT compared to a matched healthy comparison group. Differences between the groups in terms of Cohen's (1988) $d$ was just above a medium sized effect. Consistent with hypothesis two, in contrast to the comparison group, obese participants failed to learn across the task. As suggested in previous research (Cavedini, Bassi, Ubbiali et al., 2004), the failure to develop a specific strategy on the IGT could be the expression of random choice behaviour, or it could be the product of a real inability to follow a specific strategy.
during the IGT, with participants unable to maximise an immediate reward (by choosing the disadvantageous decks) or programme a delayed reward (by choosing the advantageous decks). This potential reward deficit ties in with recent findings that reward sensitivity is inversely related to BMI in those with moderate and severe obesity (Davis & Fox, 2008). The inverted-U relationship between sensitivity to reward and BMI showed that among normal weight and overweight participants (BMI 18 - 30) this relationship was in the positive direction, but changed to a negative association in the cohort who were moderately and morbidly obese. This curvilinear relationship integrates the opposing perspectives that both a reward deficiency syndrome and a heightened sensitivity to reward are linked to a higher BMI.

The absence of a clear preference for either the advantageous or disadvantageous decks (as in VmPFC patients) also suggests that the obese group did not have a severe impairment in impulse control; further supported by the finding that the CFC scale, a self-report measure of the weight given to future versus immediate consequences, showed no relationship with IGT performance. The fact that there was little overlap between this self-report measure of decision making, and the IGT a behavioural measure of decision making, may indicate that people are poor assessors of their own decision making. This would fit with the notion that people have limited insight into their own motives for behaviour (Berridge, 2004; Nisbett & Wilson, 1977). Alternatively, the IGT and CFC may tap different constructs, the former potentially related to more implicit and the CFC to explicit decision making. The overall performance of the obese participants (i.e. in terms of net score) was in line with previous work using the IGT with obese individuals (Davis et al., 2004; Davis et al., 2010; Pignatti et al., 2006). However, unlike the obese participants in Pignatti et al.'s (2006) study who showed learning on the task, the pattern of performance here over the five blocks and the failure to develop a specific strategy, was similar to that observed by Davis et al. (2004) in a high BMI group, and Davis et al. (2010) in obese groups with and without BED. However, unlike the findings of the latter study (Davis et al., 2010), education level was not found to negate group effects. The lack of learning revealed here was also reflected in BED participants' inability to advantageously use feedback on the GDT, a test of explicit decision making (Svaldi et al., 2010).

In line with hypothesis three, IGT net scores were unrelated to eating pathology both for the sample as a whole, and for each group separately. This is similar to recent findings in obese individuals where emotional eating was not found to substantially mediate the
relationship between decision making and BMI (Davis et al., 2004) and indeed to research in individuals with BED (Svaldi et al., 2010) where disadvantageous decision making on the GDT was unrelated to eating pathology or binge frequency. However, in contrast to these results, in BN, IGT performance was found to be negatively correlated with bulimic symptomatology (Boeka & Lokkenz, 2006). As such, it would seem that the impact of eating pathology on decision making may differ according to the disordered eating population under consideration, and the nature (implicit or explicit) of the decision making task. In the context of obesity, this impact seems to be minimal. With reference to other potential variables which may impact decision making impairment, as was the case in AN (Cavedini, Bassi, Ubbiali et al., 2004), IGT impairment was unrelated to BMI, and therefore independent of illness severity.

2.4.2 IGT and eating disorders

The IGT has revealed common decision making impairments in the inability to regulate eating behaviour in AN, BN, BED and obesity (Boeka & Lokkenz, 2006; Bosanac et al., 2007; Brogan et al., 2010; Cavedini, Bassi, Ubbiali et al., 2004; Cavedini, Zorzi et al., 2006; Davis et al., 2004; Davis et al., 2010; Liao et al., 2009; Pignatti et al., 2006; Tchanturia et al., 2007). However, the IGT is a complex task that taps many aspects of decision making. Potential mechanisms for these common decision making deficits suggested in the literature include a model of learned helplessness and deficits in reversal learning (Brogan et al., 2010). Indeed the learned helplessness view is supported here by the fact that obese participants preferred to stick with infrequent punishment decks (B and D), rather than emotional decks (A and C), perhaps indicating a ‘survival’ strategy on a net score of zero. To further specify these decision making deficits in both obesity and other eating disorders, future studies should incorporate both the original and variant IGT (E' F' G' H' which reverses the task’s reward contingencies). This would help identify whether such deficits are due to hypersensitivity to reward / insensitivity to punishment, or a general ‘myopia’ for future consequences both positive and negative, as is suggested here through the absence of strategy on the task. As discussed in Chapter 1, individuals hypersensitive to reward (and potentially hyposensitive to punishment) would be expected to be impaired on the original IGT (where reward is immediate and punishment delayed) but not on the variant task (overcoming the reluctance of normal controls to sample the advantageous decks because of the higher costs associated with these decks before receiving a reward). With regards to psychophysiological measures, they would be expected to show abnormally high reward SCR as well as anticipatory SCR when expecting a large gain. Individuals insensitive to future consequences, positive or negative, would be
expected to be impaired on both tasks, together with normal reward SCR but impaired anticipatory SCR (i.e. consistent with the pattern shown by VmPFC patients) (Bechara et al., 2002).

Indeed, at a more basic level, research including psychophysiological measures (e.g. SCR, heart rate) is required to investigate whether IGT decision making deficits in obesity are related to impaired affective biasing signals as in AN (Tchanturia et al., 2007) or unrelated to such signals as in BN (Liao et al., 2009), thus fully testing the premises of the SMH. The Dynamic Filtering Hypothesis (DFH) (Rule, Shimamura, & Knight, 2002; Shimamura, 2000), which views the prefrontal cortex as a selective gating or filtering mechanism for controlling information processing, could also be considered. The DFH offers a neural account of orbitofrontal cortex function with respect to monitoring and controlling emotion processing. Accordingly, the DFH views impaired affective decision making as a result of a failure to regulate or gate posterior cortical (and likely subcortical) regions involved in the expression of emotions. Techniques such as Event Related Potential (ERP) analysis can be employed to test for disinhibition of neural activity in response to affectively laden stimuli.

2.4.3 Explicit decision making

As individuals with eating disorders often explicitly know the deleterious long-term consequences of their disturbed eating behaviour (Svaldi et al., 2010) yet choose to persist in spite of this, it is unsurprising that deficits on an explicit decision making task (e.g., the GDT), have been revealed in both BN (Brand, Franke-Sievert et al., 2007) and BED (Svaldi et al., 2010). In overeating and obesity, decisions regarding the consumption of palatable versus healthy foods contain a similar conscious choice: palatable foods result in a short-term gain (e.g. taste) but long-term negative consequences (e.g. overweight). In light of the commonalities between these eating disorder groups on the IGT, future research should extend this work to explicit tests of decision making in obese and other pathological eating groups. The fact that unlike the comparison group, the obese group failed in decisions under ambiguity (first 50 trials) and under risk (last 50 trials) when the contingencies of the task may be better understood (defined by Bechera et al. (1997) as the hunch and conceptual periods), would suggest a potential deficit on tasks such as the GDT.
2.4.4 Decision making and executive functioning

Deficits were independent of eating pathology, indicating that dysfunctional decision making may potentially be embedded in a broader context of reduced executive function. This view is supported by recent literature on neuropsychological functioning in extremely obese individuals showing evidence of specific cognitive dysfunction on tests of executive functioning (planning, problem solving, mental flexibility) independent of medial co-morbidities (Boeka & Lokken, 2008). Such executive dysfunction is associated with the inability to control aberrant behaviours, such as chronic overeating. In addition, as mentioned in Chapter 1, the relationship between restrained eating and IGT decision making was found to be moderated by self-reported dispositional self-control, with restrained eating related to worse decision making when self-control was low, but to better decision making when self-control was high (Kuijer et al., 2008). These studies provide preliminary indications of the potential neuropsychological impairments which may underpin deficits on the IGT, and highlight the importance of assessing executive function in future decision making studies of overweight and obesity. This issue will be returned to in Chapter 6.

2.4.5 Clinical comparisons

In the context of previous studies with SDI both the obese participants here, and the high BMI group studied previously (Davis et al., 2004), showed greater performance impairments in terms of overall net scores and learning across the task than investigations of polydrug users (Bechara & Damasio, 2002; Grant et al., 2000). The percentage of obese participants scoring in the VmPFC range (69%) was similar to that found in a study of polydrug SDI (63%) (Bechara & Damasio, 2002) and alcohol and stimulant SDI (61%) (Bechara et al., 2001). The percentage of the study's comparison group which fell in this range (38%) was also similar to the control groups in these studies with figures of 37% (Bechara & Damasio, 2002) and 33% (Bechara et al., 2001) reported respectively. The broader implication of this is that obese individuals may share similar decision making strategies with conventional SDI.

2.4.6 Study limitations

This sample of severely obese individuals, though significantly greater than previous studies in this area, was only moderate in size. In addition, it comprised individuals currently, or previously enrolled in a weight management clinic thus limiting the study's ability to represent the general population of morbidly obese individuals. Data were not obtained on
illness duration, comorbid somatic (e.g. diabetes, cardiac history) or psychiatric disorders (e.g. Diagnostic and Statistical Manual of Mental Disorders ([DSM-IV-TR]; APA, 2000) axis I or II diagnoses), history of drug abuse, IQ, or executive functioning (e.g. self-control), which may impact upon IGT performance. It is suggested that future research control for these factors in assessing decision making on the IGT.

In terms of more general criticisms of the IGT mentioned in Chapter 1 and relevant here, the different reward schedules in a modified version of the original IGT used by researchers (Bechara & Damasio, 2002; Bechara et al., 2001) limits conclusions drawn from comparisons across studies. The degree to which such changes in the reward/punishment schedule impact on performance is unclear. Similarly, there was also considerable individual variation on the IGT whereby not all obese participants were impaired on the task, and similarly a substantial percentage of the comparison group were impaired on the task. Such findings raise questions about the ecological validity of the IGT paradigm, as comparison participants can perform badly on the task yet presumably function adequately in terms of everyday decision making (Dunn et al., 2006). The cognitive penetrability of the task's reward/punishment schedule was not assessed, and therefore the degree of conscious knowledge participants had about the task is unknown. Finally, a single task such as the IGT is limited in its ability to comprehensively assess decision-making deficits.

2.5 Conclusion

The IGT is one of the most frequently used measures of affective decision making and decision making under uncertain conditions. Obese individuals were significantly impaired on the IGT compared to a matched comparison group and showed a failure to learn across the task. These deficits were independent of age, gender, education, BMI and eating pathology. The findings were in line with previous investigations of eating disorders, indicating that the IGT reveals decision making impairments in the inability to regulate food intake. However, future research is required to further specify the source of this deficit and the degree to which it relates to eating pathology itself and executive function more generally. Decision making deficits may not only contribute to the development of an obese state, but present significant barriers to weight management leading to an inability to commit to weight loss interventions. As such, future research should move towards developing interventions which improve decision making capacity and measure subsequent impact on psychological and physical outcome.
Chapter 3: Anorexia, Bulimia and Obesity: Shared Decision Making

Deficits on the Iowa Gambling Task (IGT)

“There is a charm about the forbidden that makes it unspeakably desirable.” ~ Mark Twain

3.1 Introduction

3.1.1 Pathological eating behaviour

Eating disorders are defined as severe disturbances in eating behaviour characterised by preoccupation with weight concerns and unhealthy efforts to control weight. The vast majority of cases consist of AN and BN, two sometimes overlapping syndromes, the occurrence of which has largely been confined to affluent Westernised cultures (Weiten, 2001). AN involves intense fear of gaining weight, disturbed body image, refusal to maintain normal weight and dangerous measures to lose weight. As referred to in Chapter 2, two subtypes of AN have been observed. In AN-R people drastically reduce their intake of food essentially starving themselves, while in AN-BE individuals attempt to lose weight by forcing themselves to vomit after meals, by misusing laxatives and diuretics, and by engaging in excessive exercise (Garfinkel, 1995). BN involves habitually engaging in out-of-control overeating followed by unhealthy compensatory efforts such as those mentioned in AN-BE (e.g. self-induced vomiting, fasting, abuse of laxatives and diuretics, and excessive exercise). Eating binges are usually carried out in secret and are followed by intense guilt and concern about gaining weight, these feelings then motivating the aforementioned unhealthy compensatory behaviours. Commonalities between these two disorders include a morbid fear of becoming obese, preoccupation with food, and rigid, maladaptive approaches to controlling weight that are grounded in all-or-none thinking (Weiten, 2001).

3.1.2 Pathological eating behaviour – immediate versus long-term reward

As highlighted in Chapter 2, AN and BN have been characterised by the tendency to make decisions that have positive short-term consequences, but that may result in long-term negative outcomes (Brand, Franke-Sievert et al., 2007). In AN, individuals severely restrict their caloric intake in order to obtain an immediate reward (the relief of anxiety elicited by food phobia) while ignoring the progressive and inevitable decline in their physical health (Cavedini, Bassi, Ubbiali et al., 2004). Similarly, in BN, there is a tendency to engage in self-
damaging behaviours (binge eating / purging) that provide immediate benefit (relief of tension and guilt), at the cost of negative medical consequences (Boeka & Lokkenz, 2006). As demonstrated in Chapter 2 and in previous research, the diminished ability to assess future consequences has also shown to be characteristic of overweight and obese individuals, conditions that are also attributable (at least in part) to inadequate eating behaviours (Davis et al., 2004; Davis et al., 2010; Pignatti et al., 2006). These individuals choose to overeat (often palatable energy-dense foods) despite their long-term negative health consequences. Thus, common to AN, BN and obesity, is an inability to appropriately regulate eating behaviour.

3.1.3 The IGT and eating pathology

Despite calls for direct comparisons of the decision making profile in disordered eating groups (Brand, Franke-Sievert et al., 2007), research typically has assessed each group separately using the IGT. Such studies using the IGT in disordered eating groups were considered in detail in Chapter 2, and therefore will only be briefly summarised here. Accordingly, significant impairment on the IGT was revealed in the acute stage of AN, independent of illness severity or BMI (Cavedini, Bassi, Ubbiali et al., 2004). In addition, female AN patients who had a better IGT decision making profile at baseline showed a significantly greater improvement in nutritional status after a cognitive-behavioural treatment programme (Cavedini, Zorzi et al., 2006). Consistent with the SMH (A. R. Damasio, 1994), deficient decision making ability in females with AN was associated with impaired affective processes, specifically, significantly attenuated somatic marker signals (indexed by SCRs), compared to both healthy controls and females with long-term recovery from AN (healthy BMI and minimum one year’s menstruation) (Tchanturia et al., 2007). Significant impairment was found in undergraduate women with BN, wherein IGT performance negatively correlated with bulimic symptomatology (Boeka & Lokkenz, 2006). A more recent study found that female BN patients performed poorly on the IGT (Liao et al., 2009), but with no impairment in affective biasing signals as measured by SCR (in contrast to results with AN; Tchanturia et al., 2007). In a comparison of AN females (who were either underweight or recovered to normal weight) and BN females, research found no significant differences on the IGT between these eating disorder groups and controls (Bosanac et al., 2007). However, effects were in the expected direction, with the eating disorder groups tending to score lower than controls, and failure to reach significance may be attributable to low statistical power. Finally, both healthy women with a high BMI (BMI>25) (Davis et al., 2004), and individuals with moderate (BMI>34) and severe obesity (BMI>40) (Brogan, Hevey, O'Callaghan, Yoder, & O'Shea, In Press; Davis et al.,
2010; Pignatti et al., 2006) performed disadvantageously on the IGT. A comparison of obese women with and without BED found that both groups had lower mean IGT scores than a normal weight group, but were not significantly different from each other. When education was included in the analyses, those with a higher education performed better and the group differences were not significant (Davis et al., 2010).

3.1.4 Aims of the current study

Separate examinations of AN, BN and obesity have revealed significant impairments in affective decision making under uncertainty as measured by IGT performance. This study furthers this research by directly comparing the decision making profiles of AN, BN and obese females on the IGT, and evaluating their performance in relation to a healthy comparison group.

3.2 Method

3.2.1 Participants & procedure

3.2.1.1 AN and BN participants

Twenty-two female participants who met the DSM-IV-TR (APA, 2000) criteria for AN and 17 female participants who met the BN criteria were recruited from a rehabilitation programme in an Italian hospital specialised in the treatment of obesity and eating disorders. The AN group ranged in age from 18 - 46 years, and the BN group from 19 – 38 years. All patients underwent diagnostic interviews conducted by a trained psychiatrist and a psychologist. Participants were not tested during an acute stage of illness.

3.2.1.2 Obese participants

Eighteen female obese individuals were recruited from an Irish hospital specialised in obesity management. Participants ranged in age from 30 – 73 years.

Exclusion criteria common to all three clinical groups were: underage (<18 years); and, failure to complete the compulsory education period (minimum 8 years of formal education). Additional criteria for the AN and BN groups were: disease duration of <1 year; a comorbid personality disorder or other pervasive psychopathology; and, oppositional behaviours during the experimental session (two participants removed). For the obese group other criteria
included a diagnosis of psychopathology, BED, or a documented history of binge eating by the hospital's clinical psychologist.

3.2.1.3 Comparison participants

The comparison group comprised 20 normal weight females matched on age and education to the AN and BN groups, and education only to the obese group, with no history of psychiatric or neurological illness and no current clinical diagnosis. The group ranged in age from 18 – 40 years.

Both studies received ethical approval from the relevant hospital ethics committees, and all participants gave written informed consent (see Appendix 2 for ethical approval for the Irish sample). The study protocol for the Irish sample was as described in Chapter 2 (see Appendix 3).

3.2.2 Measures

3.2.2.1 Anthropometric measurements

BMI (weight in kilograms/height in meters$^2$) was measured to the nearest 0.1Kg and 0.01m. For the Irish sample, weight was measured using a high capacity digital scales with a wide platform (Seca bathroom scales; Model 813) designed for use in the medical sector. Height was measured using a wall-mounted tape measure (Seca; Model 206). For the Italian sample, height and weight were measured using the hospital equipment.

3.2.2.2 Decision making

Decision making was assessed using a computerised analogue version of the original IGT (Bechara et al., 1994) as described in Chapter 2.

3.2.3 Data analysis

IGT performance between groups (comparison group, AN, BN, obese) was examined by a global outcome score (net score) and a net score for each consecutive block of 20 cards. Net scores are derived from the total number of cards chosen from the advantageous decks (C & D) minus the number of cards chosen from the disadvantageous decks (A & B). Lower scores represent poorer performance, and negative scores indicate a relative preference for
choices from the disadvantageous decks. A total net score of <10 was established by Bechara et al. (2001) as the threshold for impaired performance on the IGT, given the maximum score achieved by VmPFC patients was <10. Analysis of IGT performance by blocks of 20 cards (blocks 1 – 5) provides an index of learning and strategy used by participants across the trials.

A mixed factorial ANCOVA between the comparison, AN, BN and obese groups compared differences on IGT global net scores, while a 4 (group) x 5 (block) repeated measures ANCOVA was carried out to examine group differences in block net scores. Post hoc analyses were conducted using the Sidak adjustment for multiple comparisons.

3.3 Results

3.3.1 Sample characteristics

The demographic profile of female participants is summarised in Table 3-1. There were significant differences between the groups in BMI ($F (3, 74) = 63.76, p<.001$) with the obese and BN groups significantly higher than the AN or comparison group, and in age ($F (3, 74) = 36.48, p<.001$) due to the older age of the obese group. Age was therefore entered as a covariate in all analyses. Ten obese participants (55%) had received bariatric surgery; as there were no significant differences between these participants and the non-bariatric surgery group on global net or block net scores, results are presented for the entire obese group.

<table>
<thead>
<tr>
<th></th>
<th>NC Mean (SD)</th>
<th>AN Mean (SD)</th>
<th>BN Mean (SD)</th>
<th>Obese Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>27.75 (6.99)*</td>
<td>29.09 (7.36)*</td>
<td>29.94 (6.41)*</td>
<td>52.11 (11.65)b</td>
</tr>
<tr>
<td>Education (Years)</td>
<td>12.15 (2.92)</td>
<td>12.96 (2.51)</td>
<td>11.65 (3.33)</td>
<td>14.00 (2.91)</td>
</tr>
<tr>
<td>BMI</td>
<td>21.55 (1.42)*</td>
<td>16.03 (2.04)b</td>
<td>31.87 (9.42)c</td>
<td>36.20 (5.04)c</td>
</tr>
</tbody>
</table>

BMI = Body Mass Index; *b,c Different superscript indicates significant differences between means.

3.3.2 Global net scores

The mean net scores for each group are presented in Figure 3-1. Controlling for age, there was a significant difference on IGT global net score ($F (3, 73) = 11.06, p<.001$). Post-hoc analysis revealed the comparison group was significantly different to the AN ($p<.001$), BN ($p<.001$) and obese groups ($p=.004$); however the three clinical groups were not significantly different from each other.
Figure 3-1. Mean net scores ([C&D - A&B]) on the IGT for each group.

3.3.3 Block net scores

The mean block net scores for each group are presented in Figure 3-2. Controlling for age there was a significant main effect for Group ($F (3, 73) = 12.06, p<.001$), but not for Block ($F (3.49, 254.97) = 0.26, p = .88$), and a significant Group x Block interaction ($F (10.48, 254.97) = 3.23, p<.001$). Post hoc analysis revealed no significant group differences at blocks 1 and 2. However, the comparison group was significantly different to the AN, BN and obese groups at blocks 3 ($p<.01$) and 4 ($p<.01$), and from only the AN ($p<.05$) and obese ($p<.05$) groups only at block 5. The three clinical groups were not significantly different from each other in any block.

3.3.4 IGT impairment

61% of AN, 77% of BN, and 72% of obese participants met the threshold (IGT<10) for impairment on the task (Bechara et al., 2001). By contrast, only 15% of the comparison group fell within this range.
3.4 Discussion

All three clinical groups were significantly impaired on the IGT relative to the comparison group. The majority of participants within the clinical groups reached the threshold for impairment on the IGT (although the clinical utility of this cut-off remains to be established). However, the clinical groups were not significantly different from each other, providing evidence of a possible shared inability to modulate reward and punishment in a long-term perspective amongst females with disordered eating. The IGT is a complex task that taps many aspects of decision making; a number of possible explanations for this shared decision making profile are discussed.

As the net scores for the clinical groups generally did not fall within the negative range on the IGT (as in VmPFC patients), this indicates that participants were somewhat sensitive to punishment; consequently their performance deficit may lie in faulty reward processing. The relatively flat learning curves of the three clinical groups (Figure 3-2) suggest that they could neither maximise an immediate reward (by choosing the disadvantageous decks) or a delayed reward (by choosing the advantageous decks). This may indicate learned helplessness (Hiroto & Seligman, 1975). The learned helplessness model of depression, based largely on animal research, proposed that depression is caused by learned helplessness, that is, passive 'giving up' behaviour, produced by exposure to unavoidable aversive events. Related to this is a pessimistic explanatory style, whereby people tend to attribute their setbacks to personal
rather than situational factors, and tend to draw global conclusions about their personal inadequacies based on these setbacks (Seligman, 1990). It is possible that participants had such a cognitive reaction to the IGT developing an expectancy that the task was uncontrollable, with responding unrelated to reinforcement, and that their perceived failure to win on the task was a reflection on their personal abilities as opposed to the nature of the task itself. This explanation may account for the flat performance of the clinical groups (with block net scores centring on zero) and may mimic their attempts to ameliorate their real-life pathological eating behaviours. For the obese group who showed a decline in performance over the last twenty trials (unlike the AN and BN groups who showed modest improvements from blocks four to five) the task may mirror a likely long standing pattern of failed weight loss attempts. This interpretation is supported by the fact that this was an obese population in treatment at a specialised weight management clinic that required a medical referral, made after all other avenues (e.g. community diet and exercise programmes) had been exhausted.

Unlike the comparison group who began to select more cards from the advantageous decks and less from the disadvantageous decks by the second block, the three clinical groups failed to learn across the task. As normal performance on the IGT appears to require reversal learning, in that the fixed card order induces an initial preference for the ultimately riskier decks (due to higher initial gains on these decks), this may provide an alternative explanation for the shared deficits in the female eating disordered groups. Reversal learning is defined as the ability to adapt associative learning when the reward and punishment value of two stimuli switch (Fellows & Farah, 2003). Imitating the choices on the IGT, individuals with pathological eating behaviour have to learn to reverse behaviours that may once have been highly rewarding (e.g. dieting in AN, binging in BN, and overeating palatable foods in obesity) but ultimately bring higher costs in terms of deleterious physical and psychological outcomes. Therefore, reversal learning deficits present a viable explanation for the shared decision making profile and inability to learn in the three clinical groups on the IGT. In a shuffle variant of the IGT wherein the card order was changed, eliminating the need for reversal learning, Fellows and Farah (2005a) found the performance of VmPFC patients improved to control levels. Future testing of the three clinical groups on these variant measures would inform this reversal learning hypothesis. This issue will be returned to in Chapter 6.
3.4.1 Limitations and future research

The obese group were significantly older than the AN, BN and comparison group; however, this was controlled for statistically and research suggests that the capacity for adaptive self-regulation is a relatively stable disposition throughout the lifespan (Davis et al., 2010). Future research should also take into account factors that were not controlled for here, such as illness duration, IQ, psychopathology, impulsivity, and medication use, which may impact IGT performance. Further, the results are generalisable to females only and sample sizes were small, consequently replication with a larger group including males is desirable.

Impaired IGT performance among the three female pathological eating groups highlights the importance of adaptive decision making for successful self-regulation of eating behaviour. In order to understand the involvement of affective biasing signals in this shared decision making profile, and therefore the ability of SMH to account for these common IGT deficits, future research should profile these eating disorder groups on the IGT with the inclusion of psychophysiological measures, which at present have only been applied to AN and BN. In addition, following previous work with SDI (Bechara & Damasio, 2002), and as discussed in Chapter 2, use of both the original and variant IGT (in which the schedules of reward and punishment are reversed so that punishment is immediate and reward is delayed) in AN, BN and obesity would allow differentiation of the nature of this deficit; specifically, whether it is attributable to hypersensitivity to reward / insensitivity to punishment, or a general 'myopia' for future consequences (positive and negative) with behaviour guided by immediate prospects. As stated in Chapter 2, future research should investigate whether interventions could improve IGT decision making capacity (in terms of the ability to modulate reward and punishment in a long-term perspective), and to assess subsequent impact on psychological and physical outcomes.

3.5 Conclusion

Females with AN, BN and obesity were significantly impaired on the IGT with reference to a comparison group on both overall task performance and learning across the task. The three clinical groups did not differ significantly from each other, indicating that IGT performance may represent a common neuropsychological correlate of the patients' deficient eating behaviour.
Chapter 4: Explicit vs. Implicit Decision Making in Morbid Obesity & the Role of Affective ‘Somatic Marker’ signals

"When making a decision of minor importance, I have always found it advantageous to consider all the pros and cons. In vital matters, however, such as the choice of a mate or a profession, the decision should come from the unconscious, from somewhere within ourselves. In the important decisions of personal life, we should be governed, I think, by the deep inner needs of our nature." ~ Sigmund Freud

4.1 Introduction

The results from Chapters 2 and 3, together with the findings from previous research, allow us to conclude that both obese (Davis et al., 2004; 2010; Pignatti et al., 2006) and morbidly obese individuals (Brogan et al., In Press) are significantly impaired in decision making as assessed by the IGT. Further, the types of maladaptive decisions made in obesity around the regulation of food intake, which often involve the need to forego an immediate reward for a longer-term better outcome, are descriptively similar to those in other disordered eating groups, such as in AN and BN. The IGT has also proved to be a useful tool in revealing such shared deficits (Brogan et al., 2010). IGT deficits in obesity seem to be unrelated to aspects of eating pathology or severity of the condition as measured by BMI (Brogan et al., In Press; Davis et al., 2004) and appear to hold across obese women with and without BED (Davis et al., 2010). The purpose of this study was to further specify decision making deficits revealed in obesity and those factors that may influence decision making capacity. This was achieved through the inclusion of psychophysiological measures, additional neuropsychological measures of decision making and a battery of psychometric measures.

4.1.1 Psychophysiology measures and the IGT

As discussed in Chapter 1, the IGT provides the main pillar of behavioural support for the SMH (A. R. Damasio, 1994). However, in order to fully test this hypothesis, psychophysiological data are required in conjunction with IGT performance. The premises of the SMH were articulated in Chapter 1. In addition, information on SCR and heart rate (the typical indicators of ‘somatic marker’ states), and the interpretation of psychophysiological data in relation to IGT performance, can be found in Appendix 1. In summary, when using SCR as an index of somatic state activation, successful performance on the IGT is correlated with

58
the development of anticipatory SCRs (those preceding a choice) which are larger for disadvantageous than advantageous decks, and deficient performance (as in VmPFC patients) with the absence of anticipatory SCRs (Bechara & Damasio, 2002; Bechara et al., 1999; Bechara et al., 1997; Bechara et al., 1996). Anticipatory SCR preceding disadvantageous choices is suggested to represent risk-related behaviour that is associated with the magnitude of future loss, and is presumed to rely on VmPFC convergence zones (Bechara et al., 1996). The relationship between IGT performance and SCR provides support for the SMH, as adaptive decision making is influenced by emotional responses via feedback from autonomic somatic changes (Tchanturia et al., 2007). While the SMH primarily emphasises the importance of anticipatory SCR, feedback or appraisal SCR following reward and punishment are also typically assessed as a function of which deck is chosen (Dunn et al., 2006). The rationale for this is that 'somatic markers' are acquired by experiencing the physiological outcomes (i.e., appraisal or feedback physiology) to prior decisions. Therefore those who show poor or absent appraisal physiology should be unable to develop anticipatory 'somatic markers' and decide advantageously (Jenkinson et al., 2008). Indeed, some researchers have suggested that appraisal SCR may be more important to decision making than anticipatory SCR (Suzuki et al., 2003).

The above interpretation of the SMH, and the role of anticipatory 'somatic marker' signals in facilitating adaptive decision making on the IGT, has been challenged by findings that a proportion of the normal population are as impaired as VmPFC patients on the IGT (selecting mostly disadvantageous choices) yet do develop anticipatory SCR (Bechara, Damasio et al., 2000). Bechara and Damasio (2002) have since characterised such individuals as 'high risk-takers', because they have overridden their somatic signals by conscious deliberation. However, it was also observed that SC rise following loss was less pronounced for poorly performing control participants, suggesting that poor performance could also have resulted from decreased somatic activity to performance outcomes, resulting in less efficient updating of context information to adjust performance strategy (Bechara & Damasio, 2002). As summarised by Crone et al. (2004) three possibilities for why subgroups of intact individuals perform badly on the IGT include the high-risk taking, and reduced autonomic activity to outcomes (reward and punishment) resulting in inefficient context updating views expressed above. A third possibility is that poorly performing participants may show normal autonomic activity following the outcome of choices, but may fail to integrate the acquired stimulus-response associations in the formation of 'somatic markers'.
In previous studies SC reactivity has been used as the primary index of sympathetic bodily arousal and so less research has assessed 'somatic marker' states using heart rate data (Crone et al., 2004; Crone et al., 2006). As per the assumption that the SMH specifies a general sensitivity of the autonomic system to activity associated with cognitively driven changes in body states of arousal, Crone et al. (2004) extended 'somatic marker' activity to the cardiovascular domain of arousal. Previous research has shown that heart rate slows in preparation for a voluntary response, and that slowing is more pronounced when preparing for an aversive event (a shock threat; with slowing largest when the shock is unavoidable) (Somsen, Van der Molen, & Orlebeke, 1983). As such, they hypothesised that heart rate slowing is associated with the extent to which individuals restrict attention to potentially aversive stimuli and that consequently there may be an important association between risk anticipation and anticipatory heart rate slowing. Following Bechara and Damasio (2002), they expected that heart rate slowing would be larger preceding disadvantageous decisions. They investigated these expectations in a student sample (30 men, 66 women) classified into bad (n = 29), moderate (n = 34) and good (n = 33) IGT performers. Their results indicated that anticipatory heart rate slowing (and SC level) was higher preceding disadvantageous relative to advantageous choices, but for good performers only. With regards to response heart rate, heart rate slowed and SC level increased following loss relative to reward outcomes (where heart rate quickened), and these changes were similar for all performance groups.

4.1.2 Previous tests of the SMH

As mentioned in Chapter 1, only a limited number of IGT studies have included psychophysiological data (Dunn et al., 2006). Of the studies that have included psychophysiological measures, varying degrees of support for the IGT have been provided, both in studies of healthy and clinical groups. For example, a study of 30 healthy women on the IGT found success on the task was positively correlated with anticipatory SCR with no difference in pattern between real and fake money conditions (Carter & Pasqualini, 2004). In addition, the stronger the autonomic response (SCR) preceding disadvantageous choices, the greater the success of the individual in learning the rules of the task. The authors concluded that the results show clear support for the SMH and suggest that it can be used to predict learning performance within a healthy population, thus lending more generalisable support to the theory.
In contrast to the above findings, a study of 40 healthy students found that low response (appraisal) SCRs were related to persistence in risky decisions (Suzuki et al., 2003). Although anticipatory SCRs were greater for disadvantageous than advantageous decks, they failed to show a time-course increase in amplitude inconsistent with the idea that anticipatory SCRs are acquired through experience, and there was no relationship between anticipatory SCRs and performance. As such, the authors concluded that anticipatory SCRs and their effect on performance were not confirmed and that variation in covert physiological *appraisal* underlies individual differences in decision making. Similarly, in a study of 41 healthy students who were classified as impaired or non-impaired on the IGT, no differences in SCR were found between these groups (Jenkinson et al., 2008). For the sample as a whole, greater SC rises were borderline significant when anticipating choices from bad decks compared with good decks and a significantly greater SC rise followed a reward from a bad deck. The findings were viewed as constraining the SMH by suggesting that autonomic activity may discriminate between good and bad decks (i.e. good vs. bad decision making) by reflecting the magnitude of gains and losses (bad decks yield rewards and punishments of high magnitude compared to good decks), but is independent of long-term consequences and does not discriminate between overall good and bad performance (i.e. normal vs. impaired decision makers).

Within the clinical domain findings are also found to be ambivalent. In a study of SDI (n = 46), normal controls (n = 49), and VM patients (n = 10), a subgroup of SDI showed defective performance on the IGT coupled with impaired anticipatory SCR, but normal SCR to punishment and normal acquisition of conditioned SCR to an aversive sound, supporting the hypothesis that poor decision making in some SDI is associated with defective somatic state activation (Bechara & Damasio, 2002). In contrast to this supportive result, a study of 15 Huntington’s disease patients and 16 healthy controls found that IGT impairment was associated with significant differences in feedback SCR only (Campbell et al., 2004). The magnitude of response SCRs of Huntington's patients were significantly lower than those produced by the healthy controls for both advantageous and disadvantageous decks and for losses compared to wins. They concluded that the findings indicated a reduced impact of loss on decision making. There were no significant differences between the groups in anticipatory SCRs for both deck types and so the SMH was not supported. Similarly, in a study of 39 OCD patients, compared with 40 controls, no differences between the groups were found in IGT performance. Anticipatory SCR showed a main effect of reinforcement (loss > wins) but no effect of deck, group or any interactions. However, a subgroup of OCD patients with
prominent hoarding symptoms was found to be impaired on the IGT. Those with low hoarding symptoms showed enhanced autonomic responses during the anticipatory phase, while those with high hoarding showed a smaller number of SCR fluctuations particularly during the response phase of the IGT. (Lawrence et al., 2006). In summary, the strength of the evidence supporting the SMH is equivocal (Jenkinson et al., 2008). It is difficult to ascertain why such diversity in findings exists, but is perhaps attributable to the inherently large amount of individual variation in human SCR (Dawson, Schell, & Filion, 2000), imprecision in matching physiological activity to a card choice (Carter & Pasqualini, 2004) and the substantial variability in the measurement and analysis of SCR data in conjunction with the IGT as discussed in Chapter 1 (see section 1.6.2.2).

4.1.3 The SMH in disordered eating

The general literature on studies of obesity and disordered eating using the IGT has been reviewed in Chapters 2 and 3. To the best of the author’s knowledge, no previous obesity studies incorporating the IGT have included psychophysiological data. Within the domain of disordered eating more generally, there are two known investigations of the IGT which have incorporated such data, one in AN (Tchanturia et al., 2007) and one in BN (Liao et al., 2009), both of which used SCR data only as an indicator of ‘somatic marker’ response. Accordingly, IGT impairment in 29 females (both outpatients and inpatients) with AN was associated with significantly attenuated anticipatory ‘somatic marker’ signals, compared with both a community sample of healthy controls (n = 29) and females with long-term recovery (healthy BMI and minimum one year’s menstruation) from AN (n = 14) (Tchanturia et al., 2007). The AN group also showed significantly reduced SCR after losses (to both advantageous and disadvantageous decks) compared with the control group. IGT performance and the SCR of recovered AN patients did not differ from healthy controls. The authors argued that clinically significant levels of depression in the AN group, the effects of starvation itself (given poor decision making was only present in currently ill patients) on metabolism potentially resulting in lower skin temperatures and conductivity, or alternatively a centrally mediated low sensitivity to punishment, may account for these findings. Overall, as the AN group showed reduced anticipatory SCR to all choices (not only to disadvantageous choices) the SMH was partially supported.

In the BN study, IGT deficits in 26 female BN (18 purging and 8 non-purging) outpatients at an eating disorder unit, were not associated with impaired anticipatory SCR
(Liao et al., 2009) compared to 51 healthy controls recruited from a community sample. The BN group generated levels of both anticipatory and response SCR that were comparable to those in the healthy control group. For both groups, anticipatory responses to advantageous and disadvantageous choices were not significantly different. This study included the AN data from the previously cited study for comparison purposes (Tchanturia et al., 2007) and found that in all three groups (AN, BN, healthy control) response SCR was greater to losses than wins, and to losses arising from a disadvantageous (large losses) rather than an advantageous deck. As such, the authors argued the groups were equally sensitive to punishment, but on the basis of their IGT performance did not seem to learn from this. Response SCR to reward outcomes from the disadvantageous or advantageous decks did not elicit significantly different SCR. As mentioned previously, IGT deficits were unrelated to depression or impulsivity (BIS scores; Patton et al., 1995), but were associated with obsessive-compulsive traits. Overall the findings show that in BN, impaired IGT performance is not related to decreased SCR and the data in both the BN and control group were not supportive of the SMH.

As per previous calls in the literature (Brogan et al., In Press; Brogan et al., 2010), measures of SC and heart rate were included in this study to assess if decision making deficits in morbid obesity are related to the impaired activation of 'somatic marker' states and are therefore explained by the SMH. Returning to the 'runaway weight gain train' model (Swinburn & Egger, 2004), the inclusion of such measures evaluates potential ineffectual psychophysiological brakes as a contributory factor in the rising trend of obesity. In addition, by looking at the sample as a function of successful and unsuccessful IGT performance, independent of group (obese, comparison); this study can also provide a broader test of the SMH.

4.1.4 The Soochow Gambling Task (SGT)

Chiu et al. (2008) have argued that two distinct aspects of decision making are confounded in the original IGT (Bechara et al., 1994), that of expected value (EV) and gain-loss frequency. Accordingly, they highlight that for the bad (low EV; - $250) and good (high EV; + $250) decks of the IGT, on average, for each 10-card unit, bad deck A contains 5 gains and 5 losses and B 9 gains and 1 loss. Good deck C contains 6.25 gains, 2.5 standoffs and 1.25 losses, while D contains 9 gains and 1 loss. In summary, bad decks (A and B) contain 14 gains and 6 losses, while good decks (C and D) have 15.25 gains, 2.5 standoffs and only 2.5 losses (Chiu et al., 2008). Thus, while both good (high EV) and bad (low EV) decks have a similar number of
gains, the good decks have significantly fewer losses. As such, they maintain that it is unclear whether participants' choices of good decks in the IGT are driven by improved EV or gain-loss frequency.

To address the possible confound between long-term outcome (EV) and immediate gain-loss frequency variables in the original IGT (Bechara et al., 1994), they developed a modified IGT, the SGT, which links the advantageous decks C and D (those with high EV) with low net gain frequencies and disadvantageous decks A and B (low EV) with high net gain frequencies, while maintaining identical expected values to those in the IGT (see Appendix 4 for a comparison of the reward structure in the original IGT and SGT). Based on the predictions of EV (a basic assumption of IGT, whereby participants are foresighted and learn to respond to the long-term better value of the good decks) participants should prefer to choose good decks C and D (high EV) than bad decks A and B (high frequency gain). In a study of 48 healthy students (24 males, 24 females) in which the relative contributions of EV and gain-loss frequency in the SGT were explored, it was found that immediate reinforcement prevailed over the effect of EV, with the majority of participants choosing bad decks A and B and failing to learn to select the advantageous decks across the task (Lin, Chiu, & Huang, 2009). In addition, participants were not found to 'hunch' the EV throughout the game. The authors argued that the results demonstrated that gain-loss frequency, rather than EV, guided decision makers in high ambiguity gambles, and was more powerful than that of final outcome. Additionally, it was found that choice behaviour followed the 'gain-stay, lose-randomise' strategy, whereby participants favoured high frequency gains to high frequency losses, to cope with the uncertain situation (Lin et al., 2009). From the point of view of Erev and Barron (2005), participants' choices deviate from the advantageous decks in the SGT because of the normal tendency of decision makers to underweight rare events and (or) to be loss averse. They argued that their findings, although conflicting with the SMH, are consistent with behavioural and affective decision literature, indicating that normal individuals are often short-sighted when making decisions in the stock market or real life (Chiu et al., 2008).

These findings were later replicated in a large and small value version of the SGT, with participants preferring decks A and B and failing to learn across the task (Lin et al., 2009). In addition, they were further supported by a study of 36 healthy (18 males, 18 females), predominantly college students, which compared their performance on the IGT and SGT. Accordingly, they found that on the IGT, consistent with previous research, participants chose
more cards from the advantageous decks as the task progressed, with a significant learning curve demonstrated across the task. However, on the SGT, the same participants chose fewer cards from the advantageous decks (even though the decks had the same expected values as those in the IGT), and they failed to learn across the task (Ahn, Busemeyer, Wagenmakers, & Stout, 2008). Further, later research demonstrated that even if the number of SGT trials was increased to 200 trials (from 120), participants, on average, still consistently chose more cards from disadvantageous decks with little learning in later blocks (Chiu, Lin, Lin, & Huang, 2006). While psychophysiological data have not been included in SGT studies, Chiu et al. (2008) concluded that the findings lead to three possible interpretations for SMH: 1) the ‘somatic marker’ system may guide decision making behaviour via rough-estimation processing (gain-loss frequency), and not a precise calculation (EV: probability x value); 2) the ‘somatic marker’ system may only contribute to generating subjective feelings (consciousness) and may not be immediately related to decision guidance; 3) the operation of ‘somatic markers’ may be involved in gathering long-term memory, but may not direct choice behaviour in situations of uncertainty (Chiu et al., 2008).

The SGT was included in this study of morbidly obese individuals by way of exploring the relative contributions of EV and gain-loss frequency in an obese population. In line with previous interpretations of SGT performance (Ahn et al., 2008), this inclusion allowed us to indirectly assess whether obese individuals show the normal tendency of decision makers to underweight rare events and (or) to be loss averse.

4.1.5 Explicit decision making

Decision making situations in real life differ regarding their explicitness of positive and negative consequences as well as the directness of probabilities for reward and punishment. In neuropsychological research, decisions under ambiguity and decisions under risk / explicit conditions are differentiated (Brand, Labudda, & Markowitsch, 2006). While decision making impairments in obesity have been demonstrated under ambiguous or uncertain conditions using the IGT, one of the most frequently used tests of decisions under ambiguity, decision making capacity has not been investigated in explicit conditions. Indeed, it has been argued that in terms of food choice, the decision to eat highly palatable, energy dense foods, instead of a healthier alternative is in many ways an explicit one. It is explicitly known that the former results in a short-term positive outcome (e.g., taste) but will most likely lead to negative consequences in the long run (e.g., high cholesterol, overweight) (Brand, Franke-Sievert et al., 2006).
As such, the assessment of decision making capacity under explicit conditions is relevant for obesity.

Explicit decision making or decision making under risk can be measured by a task that offers clear rules for gains and losses and stable winning probabilities, as found in the GDT (Brand et al., 2006). The GDT requires participants to decide between different fixed alternatives that are explicitly related to a specific amount of gain or loss and that have obvious winning probabilities. As such, participants can calculate the risk associated with each alternative from the very beginning of the task and can use the feedback from previous trials to apply strategies to maximise profit (Brand, Franke-Sievert et al., 2007). The task was also designed to assess the possible influence of executive functions on decision making in the gambling situation (Brand, Fujiwara et al., 2005). Disadvantageous performance on the task has been correlated with low performance in categorisation and mental flexibility, measured with the modified WCST and generally associated with the dorsolateral section of the prefrontal cortex (Brand et al., 2006).

The differences between the IGT and GDT have been clearly articulated in the literature (Brand, Fujiwara et al., 2005), with the main difference concerning the explicitness of the rules for gains and losses in the latter. Accordingly, on the GDT, the different probabilities of the choices that win or lose money can be reasoned easily and the varying amounts of gains and losses according to their probability of occurrence are obvious. In addition, in the GDT participants are told they have 18 throws to increase their starting capital and are given feedback on the remaining throws. Common to both tasks is the fact that some choices may lead to high short-term gains but long-term negative consequences because of their low probability of reward, while other choices may lead to moderate short-term gains but to a positive long-term outcome because of their higher probability of reward (Brand, Fujiwara et al., 2005).

4.1.6 Explicit decision making in disordered eating

While the GDT has not been applied to a purely obese sample, there are two known investigations of explicit decision making, using the GDT in disordered eating, one in BN (Brand, Franke-Sievert et al., 2007) and one in BED (Svaldi et al., 2010). As detailed in Chapter 2, females with BN (n = 14) chose disadvantageous alternatives significantly more frequently than did 15 healthy comparison participants (Brand, Franke-Sievert et al., 2007). As such, the
final balance for BN participants was significantly lower (and in the negative range) than it was for comparison participants who on average had a positive balance. However, although GDT performance was impaired in this BN group, their overall performance was less severely disturbed than in other patient groups, such as those with pathological gambling (Brand, Kalbe et al., 2005) who selected the disadvantageous alternatives an average of 10 out of 18 times compared to an average of 5 times in the BN sample. In addition, as mentioned in Chapter 2, task performance was related to executive functioning measures of cognitive flexibility but not to other neuropsychological functions (intelligence, reasoning, speed of information processing, working memory, attention, executive function, and theory of mind functions), education, age, BMI and other disease-specific variables (e.g. duration of BN).

In the BED study using the GDT, also cited in Chapter 2, a community sample of women with BED (n = 17; BMI = 32.8), who were free from previous psychological or psychiatric diagnoses, made significantly more disadvantageous decisions compared to overweight healthy controls (n = 18; BMI = 30.7), and finished with a significantly lower net score and final balance, similarly in the negative range (Svaldi et al., 2010). The female BED participants also showed an inability to advantageously use feedback processing whereby they changed their game strategy significantly less often than healthy controls in response to negative feedback after a risky choice. Similarly, they continued with the selection of a safe choice in response to positive feedback after a safe choice, significantly less often than controls. These deficits were unrelated to binge frequency as assessed by the EDE-Q (Fairburn & Beglin, 1994), BMI, BDI scores (Beck et al., 1988), age and years of education. However, GDT net score was negatively correlated with the BAS fun seeking subscale (Carver & White, 1994), hence the greater the number of riskier decisions made (lower net score) the higher the score on the fun seeking subscale, which is linked to impulsivity.

4.1.7 Psychophysiological measures and the GDT

Like the IGT, much of the GDT research has not incorporated psychophysiological measures in the study of Korsakoff patients (Brand, Fujiwara et al., 2005), pathological gamblers (Brand, Kalbe et al., 2005), Parkinson’s disease patients (Brand et al., 2004), and opiate dependence individuals (Brand, Roth-Bauer, Driessen, & Markowitsch, 2008), all of whom have shown significant impairments on the task. This is likely related to the fact that an underlying premise of GDT research is the assumption that in a decision situation with explicit rules, individuals can decide advantageously without using unconscious biasing signals.
(somatic markers) (Brand et al., 2006), rendering the acquisition of psychophysiological data less relevant. Accordingly, it is argued that the GDT can be solved using two methods, cognitive functioning as well as emotional feedback processing. The task can be solved not only via the processing of previous decision outcomes, but also strategically, via executive functions such as the categorisation of options, the recognition of probabilities and the magnitude of gains and losses and through strategy application. Therefore, the generation of 'somatic markers' that function as warning signals prior to a risky choice might not be a necessary predecessor for deciding advantageously (Starcke et al., 2009). In line with this, a study of patients with amygdala lesions (n = 3) and healthy comparison participants (n = 20) using the GDT and SCR measures, found the patients generated lower anticipatory and feedback SCR than healthy participants. However, within the healthy group, participants generated heightened feedback responses after risky but not after safe decisions, while no differences in anticipatory SCR to risky or safe choices were observed (Brand, Grabenhorst, Starcke, Vandekerckhove, & Markowitsch, 2007). The authors concluded that the lack of modulations in anticipatory SCRs may have occurred due to the explicit nature of the task, and that unconscious anticipatory somatic signals functioning as a warning signal prior to a risky choice are less important for advantageous decisions, while feedback signals may play a more important role.

A similar finding was obtained in an investigation of OCD patients incorporating psychophysiological data (Starcke et al., 2009). Accordingly, 14 patients with OCD were found to be impaired on the IGT but unimpaired on the GDT in comparison to 15 control participants, reflecting other GDT findings with this patient group (Starcke et al., 2010). With regards to the IGT, no effect of deck, group or any interactions were observed for anticipatory SCR. Response SCR for the entire sample was higher after disadvantageous decisions than after advantageous decisions and after losses compared to gains. In addition, compared to the patient group, healthy subjects had higher SCR to losses than gains. With reference to the GDT, no effect of choice, group or any interactions were observed for anticipatory SCR. For response SCR, there was only an effect of outcome, indicating for the entire sample, SCR generations were higher after losses in comparison to gains. The effect for choice failed to reach significance, although on a descriptive level, SCRs were higher after risky than after safe choices. These results were largely in line with previous investigations including SCR measures with healthy participants on the GDT, where feedback but not anticipatory SCR was related to GDT performance (Brand, Grabenhorst et al., 2007). The authors concluded the results
demonstrated a dissociation of decision making under ambiguity and risk in OCD patients, a finding confirmed by physiological data. The psychophysiological results for the IGT were consistent with previous investigations emphasising deficient response SCR in OCD (Lawrence et al., 2006) but similarly failed to support the SMH. For the GDT, the lack of difference between the groups in task performance was corroborated by equivalence in both anticipatory and feedback SCR patterns.

This study sought to extend the decision making literature in severe obesity to understanding explicit decision making using the GDT. The fact that morbidly obese individuals have been shown to fail on the IGT in decisions under ambiguity (first 50 trials) and under risk (last 50 trials) when the contingencies of the task may be better understood, would suggest a potential deficit on tasks such as the GDT (Brogan et al., In Press). In addition, the finding that similar impairments in decision making capacity have been demonstrated in obese women with and without BED (Davis et al., 2010), together with the fact that obese BED females have found to be impaired on the GDT (Svaldi et al., 2010), further supports this hypothesis.

Finally, to assess individual differences that may influence decision making capacity, a number of psychometric measures were included in the current study. As discussed in Chapter 1, the relationship between intelligence and IGT performance is inconsistent (Davis et al., 2008; Demaree et al., 2010; Toplak et al., 2010). However, as specific cognitive dysfunction has been revealed in extremely obese individuals (Boeka & Lokken, 2008) a measure of intelligence warranted inclusion in this study. In line with the suggestion that lack of motivation or apathy may explain deficient IGT performance (Dunn et al., 2006) a measure of current general health / psychological distress, which may affect task motivation, was also incorporated. Finally, cognitive disinhibition and impulsivity (Crone et al., 2003; Davis, Patte, Tweed et al., 2007) have been associated with impaired IGT performance, while the BAS (also linked to impulsivity) fun seeking subscale (Carver & White, 1994) has been negatively correlated with GDT performance in obese BED women (Svaldi et al., 2010). On a related issue, obese women have been found to show greater delay discounting than healthy women (Weller, Cook lli, Avsar, & Cox, 2008). As such, a measure of impulsivity was included in the study. Much of the information on the potential role of these variables in influencing decision making capacity is derived from the IGT, the most prominent decision task used here, but is likely relevant to decision making as assessed by the GDT and SGT also.
4.1.8 Aims of the current study

The aim of this study was to further specify decision making deficits in morbid obesity. This was achieved through the deployment of psychophysiological measures (SCR and heart rate) with the IGT, to assess if IGT decision making deficits in obesity are related to impaired affective biasing signals in line with the premises of the SMH (A. R. Damasio, 1994). This also facilitated the assessment of a secondary research question; whether the data as whole, analysed by successful or unsuccessful task performance, are supportive of the SMH. Moving towards more cognitive aspects of decision making, a second decision task, the SGT, was incorporated to explore the relative contributions of EV and gain-loss frequency in decision making in obesity, and consequently whether obese individuals show the normal tendency of decision makers to underweight rare events and (or) to be loss averse. Similarly, a third decision task, the GDT, was used to assess decision making capacity in severely obese individuals under explicit or risky conditions, in contrast to the implicit or uncertain nature of the IGT. Psychophysiological measures were obtained on all three tasks in line with and extending the previous literature. Finally, individual difference variables were incorporated to assess their influence, if any, on decision making capacity.

4.2 Method

4.2.1 Participants & Procedure

Obese group: A convenience sample of 21 participants (10 male, 11 female), from 24 – 62 years of age, was recruited at several induction days for a weight management program run by a National Weight Management Clinic at a suburban general hospital. After an oral presentation by the researcher outlining the project, individuals had the option of providing their contact details for participation. Participants were also recruited through posters placed around the university campus and at local libraries. Inclusion criteria were: ability to give informed consent; a minimum of eight years of formal education; and a BMI >30.

Comparison group: 20 participants (5 male, 15 female), from 20 – 59 years of age, matched for education, were recruited from individuals attending the university’s psychology evening course, and from posters placed around the university campus. Inclusion criteria were as above, but with a BMI <30.
Participation in the research was voluntary; however undergraduate psychology students were given research credits as part of the internal psychology research participation program. Exclusion criteria common to both groups were underage (<18 years), and current acute psychiatric disorder. Accordingly, one participant with active schizophrenia was excluded from the obese group. The study received ethical approval from both the university and hospital ethics committees (see Appendix 2 for ethical approval documentation). After provision of written informed consent participants were tested in the university’s psychology department. Please see Appendix 5 for a full copy of the study protocol.

4.2.2 Measures

4.2.2.1 Anthropometric Measurements

These were recorded as specified in Chapter 2.

4.2.2.2 Psychometric measures

The Verbal Subtest from the Wechsler Adult Intelligence Scale-Third Edition ([WAIS-III]; Wechsler, 1997) was used as a measure of general intelligence (g). The subset has 33 words of increasing difficulty (e.g. bed, ship, sentence, diverse) which participants must define in response to the phrase ‘Can you tell me what _____ means’. Using the WAIS manual participant responses are scored on a scale of 0 – 2, up to a maximum score of 66. A discontinue rule is invoked should participants obtain a score of zero on more than six consecutive items. This subtest is shown to correlate .83 with g for the WAIS-III (ages 16 – 89), and to have good reliability (e.g. test-retest reliability of .96; split half reliability of > .85 in 79% of studies) and validity and thus is regarded as a good measure of g (Kaufman & Lichtenberger, 2006).

Participants completed the General Health Questionnaire ([GHQ-12]; Goldberg, 1992) a shorter version of the GHQ-60 (Goldberg, 1978) as a measure of current psychological distress. Each of the questionnaire’s 12 items asks whether the respondent has experienced a particular symptom or item of behaviour using a four-point scale: less than usual, no more than usual, rather more than usual, or much more than usual. Sample items include ‘Have you recently’: ‘Been able to concentrate on whatever you’re doing?’ ‘Lost much sleep over worry?’ ‘Been feeling unhappy and depressed?’ Higher scores indicate greater probability of clinical disorder. Cronbach’s alpha for the GHQ-12 ranged from .82 - .90 in a series of studies.
Split-half reliability was .83 and test-retest reliability was .73. Its validity has been evaluated by assessing its sensitivity in detecting cases of psychiatric disorder against standardised interviews of disorder, all relevant studies of which have produced satisfactory sensitivity and specificity figures (Goldberg & Williams, 1988). Cronbach’s alpha in the current sample (.83) showed good internal consistency for the GHQ.

The 15-item short form of the BIS (Patton et al., 1995), the BIS15 (Spinella, 2007) was used to assess impulsivity. The BIS15 has three subscales, non-planning (NP), attention impulsivity (A), and motor impulsivity (M). Sample items include: ‘I plan tasks carefully’ (inverted item, NP); ‘I am restless at lectures or talks’ (A); and ‘I say things without thinking’ (M). Response categories range from 1 (rarely / never) – 5 (almost always), with higher scores indicative of higher levels of the measured trait. The authors report good validity and reliability, with a Cronbach’s alpha of .81 and a correlation of .94 with the longer version of the questionnaire (Spinella, 2007). Cronbach’s alpha coefficients for the current sample were .72 (NP), .74 (A), and .71 (M) demonstrating good internal consistency.

4.2.2.3 Decision making measures

IGT: The IGT has been described in Chapter 2. To facilitate psychophysiological data acquisition the IGT (Bechara et al., 1994; Bechara, Tranel et al., 2000) was programmed in E-prime (Psychology Software Tools Inc., 2005). In contrast to the IGT used in Chapters 2 and 3, which was based on the original reward schedule (Bechara et al., 1994), the newer reward schedule A’ B’ C’ D’ (Bechara, Tranel et al., 2000) was employed here.

SGT: The SGT (Chiu et al., 2008) was also programmed in E-prime. As a variation of the IGT, the SGT contains four decks with a total of 100 trials. The ‘bad’ decks (defined by EV, as in the IGT) have worse EV and better gain-loss frequency, while conversely the ‘good’ decks have better EV and worse gain-loss frequency (see Table 4-1 for description and Appendix 4 for a comparison of the structure in the original IGT and SGT). Thus per ten cards, in the symmetrically designed SGT, decks A and B yield bad long-term outcome (-€500) but a high frequency gain (eight gains [A: +€200, B: +€100], two losses [A: -€1050, B: -€650]), while decks C and D yield a good final outcome +(€500) but high-frequency losses (eight losses [C: -€200, D: -€100], two gains [C: +1050, D+650]) (Lin et al., 2009). Of note, in the IGT, gains and losses are presented to participants separately, while in the SGT only the net gains are presented. In
addition, the task enlarges the difference between positive and negative expected values to make the difference more noticeable than in the IGT.

Table 4-1. Gain-loss structure in original SGT (Lin et al., 2009).

<table>
<thead>
<tr>
<th>Card Sequence</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>200</td>
<td>100</td>
<td>-200</td>
<td>-100</td>
</tr>
<tr>
<td>2</td>
<td>200</td>
<td>100</td>
<td>-200</td>
<td>-100</td>
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<tr>
<td>3</td>
<td>200</td>
<td>100</td>
<td>-200</td>
<td>-100</td>
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<tr>
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<td>5</td>
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<td>650</td>
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<td>-100</td>
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<tr>
<td>7</td>
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<td>-200</td>
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<td>10</td>
<td>-1050</td>
<td>-650</td>
<td>1050</td>
<td>650</td>
</tr>
<tr>
<td>Final Outcome</td>
<td>-500</td>
<td>-500</td>
<td>500</td>
<td>500</td>
</tr>
</tbody>
</table>

Gain-loss frequency

<table>
<thead>
<tr>
<th>Gain-loss frequency</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>8 gains</td>
<td>8 gains</td>
<td>2 gains</td>
<td>2 losses</td>
<td>2 losses</td>
</tr>
<tr>
<td>2 losses</td>
<td>2 losses</td>
<td>8 losses</td>
<td>8 losses</td>
<td></td>
</tr>
</tbody>
</table>

The order of the IGT and SGT were counterbalanced, so that half of participants completed the IGT first and half the SGT first.

Memory assessment: Following Chiu et al. (2008), participants’ memory for their behaviour and preferences during the IGT and SGT was assessed after the completion of each task through a short questionnaire: 1) Which deck do you experience the highest frequency gain; 2) Which deck do experience the highest frequency loss? 3) Which deck do you earn the largest overall amount of money? 4) Which deck do you lose the largest overall amount of money? 5) Which deck do you prefer most to choose? 6) Which deck do you dislike most to choose?

GDT: In the computerised GDT (Brand, Fujiwara et al., 2005), developed for the neuropsychological investigation of decision making in explicit situations, participants are instructed that the goal of the game is to win as much fictitious money as possible within 18 throws of a die. Participants are given a fictitious starting capital of €1000 and are told that they can continue to play even if they have a negative balance. They are explicitly briefed on the rules for winning and losing and guided through examples of the possible gains and losses associated with each alternative (Brand, Franke-Sievert et al., 2007). Before each throw, individuals must choose from fixed alternatives (shown in Figure 4-1) (Brand, Grabenhorst et
that range from a single number (winning probability = 1:6 or 16.67%) to a combination of two (winning probability = 2:6 or 33.33%), three (winning probability = 3:6 or 50%) or four numbers (winning probability = 4:6 or 66.67%), which number or combination of numbers contains the number that will be thrown in the next trial (Brand, Franke-Sievert et al., 2007). These alternatives are constantly displayed throughout the task. When the thrown number is congruent with the selected number or a number within the selected combination, the subject wins the specified gain. Conversely, when the thrown number is incongruent with the selected number(s) the amount is subtracted from the current balance. The results of the throws are pseudorandomised, meaning that each of the six possible numbers occurs three times during task performance, but in a balanced order (Brand, Fujiwara et al., 2005). After each trial, feedback on the immediate gain or loss is shown on the screen, in addition to the current balance and the number of trials remaining.

<table>
<thead>
<tr>
<th>Categories of Alternatives</th>
<th>Possible Numbers and Combinations of Numbers</th>
<th>Winning Probability</th>
<th>Gains/ Losses</th>
</tr>
</thead>
<tbody>
<tr>
<td>One Number</td>
<td><img src="image" alt="One Number" /></td>
<td>1/6</td>
<td>1,000 €</td>
</tr>
<tr>
<td>Two Numbers</td>
<td><img src="image" alt="Two Numbers" /></td>
<td>2/6</td>
<td>500 €</td>
</tr>
<tr>
<td>Three Numbers</td>
<td><img src="image" alt="Three Numbers" /></td>
<td>3/6</td>
<td>200 €</td>
</tr>
<tr>
<td>Four Numbers</td>
<td><img src="image" alt="Four Numbers" /></td>
<td>4/6</td>
<td>100 €</td>
</tr>
</tbody>
</table>

Figure 4-1. Illustration of the fixed alternatives provided in the GDT. Participants have to choose between alternatives that consist of either a single number or a combination of two, three or four numbers. Only the shown combinations are possible. On the right hand side the gains and losses associated with the different alternatives are constantly displayed. The options of one and two numbers are analysed as “risky” because they have a winning probability of less than 50% and most likely results in high long-term losses. The options of a combination of three and four numbers are analysed as “non-risky” because they have a winning probability of 50% and higher, most likely leading to a positive balance in the long run (Brand, Fujiwara et al., 2005).

The contingencies shown in Figure 4-1 show that altogether the GDT consists of 14 alternatives assigned to four categories which are differently linked to gains and losses (€100 – €1000) with unlikely alternatives having the highest amount of gain or loss (Brand et al., 2008). Accordingly, the choice of single number (€1000 gain / loss) or a combination of two numbers (€500 gain / loss) is evaluated as ‘disadvantageous’ or ‘risky’ as they are associated with
winning probabilities of less than 34%, and so result in rare high gains and frequent high losses in the long run. By contrast, choices of combinations of three (€200 gain / loss) or four (€100 gain / loss) numbers are deemed ‘advantageous’ or ‘non-risky’ decisions, as they are linked to winning probabilities of 50% or higher, leading to low gains but also low penalties and ultimately a moderate positive long-term balance (Brand, Franke-Sievert et al., 2007).

4.2.2.4 Psychophysiological measures

SC and EKG were obtained using the PsychLab SC-EKG stand-alone unit (Contact Precision Instruments, London, UK). Room temperature was set to 23°C to control for diurnal effects. The sampling rate was 1000 Hz. A baseline recording of at least 5 minutes was taken before the first decision making task (IGT or SGT).

SC was recorded using a constant voltage (0.5V) with two 8mm diameter silver chloride disc electrodes (BioMed electrodes, California) positioned on the medial phalange of the index and middle finger of the non-dominant hand using double-sided adhesive collars and non-saline electrode gel. Both electrodes were connected to a Psychlab SC5 24-bit digital skin conductance amplifier which relayed the signal to a host laptop running the PsychlabAcquire1 software via standard USB cable. SC data were DC coupled. SC values were transformed to microsiemens (µS) and values were analysed off-line using the PsychlabS software. In line with previous eating disorder (ED) studies (Liao et al., 2009; Tchanturia et al., 2007), mean SCR for each participant was based on all responses generated for each trial (including zero responses). A constant of 1 was added to all means to account for zero values. As in previous research (Campbell et al., 2004), means were then transformed using a Log10 transformation. The data were screened manually usually boxplots for any potential extreme outliers, which were then removed.

EKG was recorded from standard dot snapper electrodes positioned either side of the heart, on the right collar bone and above the left waist. A 40Hz low pass and 10Hz high pass filter were set, in addition to a hum filter to reduce interference. IBIs, defined as the time in Ms between consecutive R waves of an EKG (see Appendix 1 and section 1.5.5. for more information), were obtained by analysing stored raw EKG wave off-line and were screened for physiologically impossible readings and artefacts using the PsychlabS software. The algorithm used to screen heart rate in producing the data was ±40% variation about the mean of the previous 20 beats. Allowed beats were then updated into the running average used to screen
subsequent intervals. Before the initial 20 beats were detected, initial IBI limits of 500ms (120 bpm) – 1600ms (37.5 bpm) were used for allowable interval readings. In addition, extreme outliers were manually removed based on boxplots.

4.2.3 Data analysis

4.2.3.1 Psychophysiological analysis: SCR

IGT and SGT: For these tasks E-prime sent automatic event marker signals to the Psychlab system (via a parallel port) so that stimulus presentations, responses by participants and feedback could be identified in the Psychlab recording. A minimum inter-trial interval of nine seconds between card selections was set to ensure adequate time for measuring SCR. Following Suzuki et al. (2003), before each card selection, the anticipatory phase, a message appeared on the screen reading ‘Please consider your next choice’ for five seconds duration, after which a message saying ‘Please select your next card’ appeared. During this time the decks were displayed continuously on the screen and the subject could ponder which deck to choose next. However, if the subject clicked the mouse to select a card during that time interval, the computer did not respond and no record was generated. Participants could take longer than the allocated five seconds and so the anticipatory phase was variable, while the feedback or response (win / loss) phase was fixed at 4 seconds. For the purpose of analysing the data, the SCR reading was divided into: 1) anticipatory SCR (four seconds preceding card selection); and, 2) response SCR (five seconds after a card selection). To account for the 0.8sec SCR onset time, the measurement window was shifted forward by one second. The response SCR time window was selected to reflect existing research (Bechara et al., 1999; Bechara et al., 2002; Crone et al., 2004; Lawrence et al., 2006). However, we extended the minimum anticipatory SCR window of one second adopted by some studies (e.g. Lawrence et al., 2006; Liao et al., 2009; Tchanturia et al., 2007) to a window of four seconds in line with longer intervals adopted by other researchers (Campbell et al., 2004; Crone et al., 2004; Jenkinson et al., 2008), based on the rationale that a SCR typically occurs 1 – 5 seconds after stimulus onset (Naqvi & Bechara, 2006). The timings established here considered the ability to capture stimulus elicited changes in physiology, while maintaining a reasonable length of time for participants to complete the task. For both anticipatory and response SCR, total fluctuation amplitude (difference between SC onset and peak with a minimum amplitude of .01 μs) in the specified time windows were summed (see Appendix 1 for more information). As such, this
measurement assessed the change in SC from each participant's individual onset value. When there was no fluctuation associated with a card selection, "0" was recorded.

**GDT:** For the GDT, events markers were set manually during data collection so whenever a decision was made or feedback received, a marker was set in the SCR data. Following Starcke et al. (2009) the inter-trial interval was set at 6 seconds to ensure that trials' SCR's did not overlap and to allow SC levels to return to baseline (see Bechara et al., 1999). Subjects were instructed not to make decisions during the inter-trial interval. The time window for analysing SCR activity was five seconds before the selection and five seconds after feedback. Unlike the IGT / SGT, on the GDT, there is a 2.5s delay between selection and feedback for each trial that is not included in the analysis, therefore the time window for analysing feedback SCRs is 5 seconds after the feedback (not after the selection). Mean total fluctuation amplitude was summed for: 1) anticipatory SCR (five seconds preceding a card selection); and 2) response SCR (five seconds after feedback).

**4.2.3.2 Psychophysiological Analysis: EKG**

For all three tasks (IGT, SGT, GDT) in line with previous research (Crone et al., 2004; Crone et al., 2006): 1) Anticipatory heart rate was computed on the basis of the difference between the IBI concurrent to the response (IBIO) and the IBI preceding the response (IBI-1), based on previous reports showing that response uncertainty effects become prominent at the IBI in which the response is committed (Somsen et al., 1983); 2) Response heart rate was computed on the basis of the difference between the first IBI following the presentation of feedback (IBI+1) and the 2nd IBI preceding the response (IBI-2).

**4.2.3.3 Task analysis**

**SGT & IGT:** Performance was measured by: 1) a global outcome score (net score); 2) a net score for each consecutive block of 20 cards; 3) split half net scores for trials 1 – 50 compared to 51 – 100; and 4) frequency of response shift (percent) or stay in the continuing choice. Net scores are derived from the total number of cards chosen from the advantageous decks (C & D) minus those chosen from the disadvantageous decks (A & B). Lower scores represent poorer performance, and negative scores indicate a relative preference for choices from the disadvantageous decks. Analysis of IGT and SGT performance by blocks of 20 cards
(blocks 1–5), and split half scores (trials 1 – 50 vs. 51 – 100) provides an index of learning and the strategy used by participants across the trial.

GDT: Performance on the GDT was calculated by: 1) a net score; 2) frequency of choices from each of the four different categories (one number, two numbers, three and four numbers) with one single number being the riskiest and a combination of 4 numbers being the safest; 3) frequency (percent) of use of negative feedback (high loss) following a risky choice to shift to a non-risky alternative in the succeeding trial. Similar to the IGT / SGT, net scores are derived from the total number of advantageous or non-risky choices (three and four numbers) minus the total number of disadvantageous or risky choices (one and two numbers). Therefore a positive overall net score indicates non-risky performance.

Based on findings in section 2.3.2, a medium effect size of .60 was expected for the IGT. Power was calculated as .99 to detect an effect size of .6 as significant at the $p = .05$ level using independent samples $t$-test. As such, even though sample size was considerably smaller in this study than in Chapter 2, power was deemed sufficient.

Memory assessment for the IGT and SGT:

As only a brief assessment of the degree of conscious knowledge of the tasks was desired, these results were reported as frequencies of choices from the disadvantageous and advantageous decks.

4.2.3.4 Psychometric Questionnaires: Missing data

There were very little missing data in the sample. However, the GHQ total (Goldberg, 1992) was prorated for one individual. For the subscales of the BIS15 (Spinella, 2007), the average of the items that individual answered was used for two participants.

Regression: Following Liao, et al. (2009) regression analysis was performed on both groups to find the best unique predictors in performance on all three tasks, using task performance (net score) as the dependent variable and WAIS-III, GHQ, and subscales A, M and NP scores from the BIS-15 as independent variables.
4.3 Results

4.3.1 Sample characteristics

The clinical demographic profile and summary of psychometric measures are summarised in Table 4-2. Seventy-five percent of the comparison group (n = 15) were female, in contrast to 52% (n = 11) of the obese group. There were no significant differences between groups in relation to gender (χ² (1, 41) = 2.26, p = .13). There was a significant difference between the groups in age (F(1, 39) = 6.19, p = .02), the obese group significantly older than the comparison group, and so age was entered as a covariate in all analyses. Significant group differences were also found on the BIS15-NP (F(1, 39) = 6.41, p = .02), with the obese group higher than comparison participants, and on the WAIS-III (F(1, 34) = 6.18, p = .02), wherein the obese group scored lower than the comparison group. WAIS-III data were not available on five participants as they were not native English speakers. As expected, the groups differed significantly in BMI (F(1, 38) = 69.56, p < .001). Within the control group 9 participants (45%) were of a healthy weight and 11 participants (55%) were overweight. Within the obese group, participant BMIs ranged from 31.05 – 73.85; 19% with class I obesity, 9.5% with class II, and the majority of participants, 72.5%, with class III obesity (WHO, 2000).

| Table 4-2. Clinical Demographic and Psychometric Profile of Participants. |
|---------------------------------|-----------------|-----------------|
|                                | **Comparison Group** (n = 20) | **Obese Group** (n = 21) |
| **M (SD)**                     | **M (SD)**       | **M (SD)**       |
| Age (Years)                    | 39.15 (12.21)    | 48 (10.54)*      |
| Education (Years)              | 17.05 (3.24)     | 15.81 (5.67)     |
| BMI                            | 24.64 (2.77)     | 44.54 (10.30)**  |
| WAIS-III (Verbal Score)        | 56.94 (5.37)     | 48.16 (13.64)*   |
| GHQ-12 Total Score             | 10.19 (3.74)     | 13.76 (7.49)     |
| BIS15 NP                       | 9.05 (3.35)      | 11.7 (3.35)*     |
| BIS15 A                        | 8.58 (2.57)      | 9.61 (3.16)      |
| BIS15 M                        | 10.55 (1.23)     | 11.43 (1.89)     |

BMI = Body Mass Index; WAIS III = Wechsler Adult Intelligence Scale-Third Edition; GHQ-12 = General Health Questionnaire-12; BIS15 = Barrett Impulsivity Scale-15 – NP = Non-planning, A = Attention impulsivity, M = Motor impulsivity; *p < .05. ** p < .001

Medical history: 10% of the obese group had heart disease, 33% diabetes, 28.6% arthritis and 28.6% sleep apnea, while only 5% of comparison participants had diabetes, 5% sleep apnea and 10% arthritis. Ninety percent of the obese group were medicated for these conditions in contrast to 26.3% of the comparison group. Psychiatric history: 23.8% of obese participants and one participant in the comparison group were diagnosed with depression.
Two obese participants (9.5%) were diagnosed with BED and one obese participant had schizophrenia but was deemed to be stable. Current weight management: 66.7% of obese participants were currently involved in a national weight management programme and 19% were using prescription weight loss drugs. None of the participants had received bariatric surgery.

4.3.2 Results for the IGT

4.3.2.1 Behavioural Performance on the IGT

A between groups ANCOVA, controlling for age, education and BMI, found a significant difference on IGT global net scores \((F(1, 35) = 4.87, p = .03, \eta^2 = .12)\). The mean adjusted net scores of the comparison \((M = 29.89, SE = 9.4)\) and obese groups \((M = -6.39, SE = 9.4)\) on the IGT are presented in Figure 4-2. As mentioned previously there were significant differences between the groups in general intelligence as measured by the WAIS-III. When WAIS-III score was added as a covariate to the model, the difference in IGT net score was no longer significant \((F(1, 29) = 2.31, p = .14, \eta^2 = .074)\). In terms of card selection, both the control \((t(19) = 4.09, p = .001)\) and obese groups \((t(20) = 3.76, p = .001)\) showed a significant preference for infrequent punishment, selecting more cards from decks B and D than decks A and C.

![IGT: Mean Net Score](image)

**Figure 4-2.** Mean net scores \([C'&D' - A'&B']\) on the IGT for the comparison and obese groups. Error bars are standard errors.
4.3.2.2 IGT Task learning: Net scores blocks 1–5

ANCOVA, controlling for age, education and BMI, with group as the between subject factor and block as the within subject factor revealed a main effect of group ($F (1, 35) = 4.87, p=.03, \eta^2 = .122$), block ($F (2.26, 78.98) = 3.17, p=.04, \eta^2 = .083$), and a statistically significant interaction ($F (2.26, 78.98) = 5.62, p<.001, \eta^2 = .138$). Post hoc analyses revealed that the groups differed significantly at block 3 ($F (1, 35) = 6.03, p=.02, \eta^2 = .147$), block 4 ($F (1, 35) = 7.57, p=.009, \eta^2 = .178$), and block 5 ($F (1, 35) = 6.06, p=.019, \eta^2 = .148$). Adjusted block net scores for the comparison and obese groups are presented in Figure 4-3. Thus, while the comparison participants showed an increasing preference for advantageous decks across the task, the obese participants failed to learn across the task. Furthermore, the obese group did not demonstrate a clear strategy, with no shift in preference towards either the advantageous or disadvantageous decks. When WAIS-III score was added as a covariate to the model there was a main effect of block ($F (1.8, 52.09) = 3.54, p=.04, \eta^2 = .109$), and the interaction remained significant ($F (1.8, 52.09) = 3.42, p=.04, \eta^2 = .105$). Group was no longer significant.

Figure 4-3. Mean net scores ($[C'&D' - A'&B']$) on the IGT across each block of 20 trials. Positive scores reflect advantageous performance while negative scores reflect disadvantageous performance. Error bars are standard errors.

4.3.2.3 IGT Split half net scores

This failure to learn across the task is similarly illustrated in Figure 4-4, which contrasts adjusted net scores on the IGT for the first 50 trials with net scores over the last 50 trials in both groups. The difference in net scores between the two groups at time two (trials 51–100) was significant ($F (1, 39) = 7.32, p=0.1, \eta^2 = .173$).
4.3.2.4 IGT Response shift or stay in the continuing choice

Mean probability of shift or stay in the continuing choice is illustrated in Figure 4-5. There were no differences between the groups in the continuing choice after a bad deck (A' & B'). However, after a good deck (C' & D'), the obese group were significantly more likely to switch to a bad deck (A' & B') \( (t(39) = -2.36, p=.02) \), while the comparison group were significantly more like to stay with a good deck (C' & D') \( (t(39) = 2.37, p=.02) \).
4.3.2.5 IGT: Anticipatory SCR and heart rate

The mean total fluctuation amplitude for anticipatory SCRs (see Figure 4-6) and mean anticipatory heart rate (IBO - IBI-1) (see Figure 4-7) was calculated separately for the disadvantageous decks (A', B') and the advantageous decks (C', D') decks; the reinforcement irrelevant for anticipatory SCR, as participants do not know whether they will win or lose when deciding which card to pick. There were no significant differences between the groups in average total anticipatory SCR ($t(31) = -1.72, p=.09$). A repeated measures ANOVA with group (obese, comparison) as the between subject factor and deck (disadvantageous, advantageous) as the within subject factor showed a main effect of deck ($F(1, 31) = 10.12, p=.003, \eta^2 = .25$) with both groups showing higher anticipatory SCR to the disadvantageous decks. There was no effect of group ($F(1, 31) = 2.95, p=.096, \eta^2 = .09$) and a non-significant interaction ($F(1, 31) = .53, p=.473, \eta^2 = .02$).

![IGT: Mean Anticipatory SCR](image)

**Figure 4-6.** Mean total anticipatory SCR for disadvantageous (A', B') and advantageous (C', D') decks.

As shown in Figure 4-7, there were no significant differences between the groups in average total anticipatory heart rate ($t(23) = -1.97, p=.06$). IBI difference scores were similarly submitted to a 2 group (obese, comparison) X 2 deck (disadvantageous, advantageous) repeated measures ANOVA. There was no effect of group ($F(1, 23) = 3.89, p=.06, \eta^2 = .15$), or deck ($F(1, 23) = .50, p=.49, \eta^2 = .02$), and a non-significant interaction ($F(1, 23) = .96, p=.34, \eta^2 = .04$). The obese group showed heart rate slowing (larger IBI intervals) to both advantageous and disadvantageous decks, while the comparison group showed slowing to the advantageous decks only and very little change in IBI to disadvantageous decks.
Figure 4-7. Mean anticipatory heart rate (IBI0 - IBI-1) for disadvantageous (A', B') and advantageous (C', D') decks. Error bars are standard errors.

4.3.2.6 IGT: Response SCR and heart rate

The mean total fluctuation amplitude for response SCRs (see Figure 4-8) and mean response heart rate (IBI+1 - IBI-2) (see Figure 4-10) was calculated separately for wins (no associated loss) and for losses (loss or a net loss) from the disadvantageous decks (A', B') and the advantageous decks (C', D').

There were no significant differences between the groups in average total response SCR (t (30) = -1.54, p=.14). A repeated measures ANOVA with group (obese, comparison) as
the between subject factor and deck (disadvantageous, advantageous) and reinforcement (win, loss) as the within subject factors, showed a deck x reinforcement interaction ($F(1, 30) = 9.83, p=.004, \eta^2 = .247$) for response SCR.

The deck x reinforcement interaction is plotted in Figure 4-9. Post hoc analysis using the Sidak adjustment for multiple comparisons showed mean response SCR to a win from a disadvantageous deck was significantly higher than mean response SCR to a win from an advantageous deck ($p=.04$). The difference between response SCR to loss from a disadvantageous and advantageous deck was non-significant ($p=.43$). The overall pattern indicates that SCR is dependent not just on win or loss, but also on deck type, with a win on a disadvantageous deck generating a similar response to a loss on an advantageous deck. There were no other main effects or significant interactions.

![IGT: Deck * reinforcement interaction](image)

Figure 4-9. Deck * reinforcement interaction for response SCR. Error bars are standard errors.

As shown in Figure 4-10, there were no significant differences between the groups in average total response heart rate ($t(19) = -.91, p=.38$). The IBI response difference scores to wins and losses were similarly submitted to a 2 group (obese, comparison) X 2 deck (advantageous, disadvantageous) X 2 reinforcement (win, loss) repeated measures ANOVA. There were no main effects or significant interactions.
4.3.3 Memory assessment for the IGT

Memory assessment for the IGT is presented in Table 4-3 below. The majority of participants in both the comparison and obese groups clearly identified that they earned the least on the disadvantageous decks. However, participants were less certain of which decks they earned the most on, with only 61% of the comparison group and 38% of the obese group, correctly identifying the advantageous decks.

Table 4-3. IGT memory assessment. Frequencies for the disadvantageous and advantageous decks are presented.

<table>
<thead>
<tr>
<th></th>
<th>Comparison Group</th>
<th></th>
<th>Obese Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Disadvantageous: n (%)</td>
<td>Advantageous: n (%)</td>
<td>Disadvantageous: n (%)</td>
</tr>
<tr>
<td>Frequency Gain</td>
<td>4 (22%)</td>
<td>14 (78%)</td>
<td>14 (66.7%)</td>
</tr>
<tr>
<td></td>
<td>17 (89%)</td>
<td>2 (11%)</td>
<td>15 (79%)</td>
</tr>
<tr>
<td>Frequency Loss</td>
<td>7 (39%)</td>
<td>11 (61%)</td>
<td>13 (62%)</td>
</tr>
<tr>
<td>Earn most</td>
<td>18 (100%)</td>
<td>0</td>
<td>19 (90%)</td>
</tr>
<tr>
<td>Earn least</td>
<td>6 (33%)</td>
<td>12 (67%)</td>
<td>9 (45%)</td>
</tr>
<tr>
<td>Prefer</td>
<td>15 (83%)</td>
<td>3 (17%)</td>
<td>12 (63%)</td>
</tr>
<tr>
<td>Dislike</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
4.3.4 Results for the SGT

4.3.4.1 Behavioural Performance on the SGT

A between groups ANCOVA, controlling for age, education and BMI, found no significant differences on SGT adjusted net scores ($F(1, 39) = 3.56, p = .07, \eta^2 = .092$) between the comparison ($M = -60.20, SE = 8.92$) and obese groups ($M = -30.79, SE = 8.92$). When WAIS-III score was added as a covariate to the model this pattern remained the same. In terms of card selection, as shown in Figure 4-11 it is clear from the net scores that both the comparison ($t(19) = -6.62, p < .001$) and obese groups ($t(20) = -7.75, p < .001$) showed a significant preference for disadvantageous decks (A & B) rather than advantageous decks (C & D).

![SGT: Mean Deck Selections](image)

**Figure 4-11.** Mean number of cards selected from each deck on the SGT.

4.3.4.2 SGT Task learning: Net scores blocks 1–5

ANCOVA controlling for age, education and BMI, with group as the between subject factor and block as the within subject factor revealed a main effect of block ($F(2.69, 94.2) = 3.01, p=.04, \eta^2 = .079$), but not of group, and a non-significant interaction ($F(2.69, 94.2) = 2.31, p=.08, \eta^2 = .062$). Adding WAIS-III score as a covariate to the model did not change these findings. Adjusted block net scores for the comparison and obese groups are presented in Figure 4-12.
Figure 4-12. Mean net scores ([C&D - A&B]) on the SGT across each block of 20 trials. Positive scores reflect advantageous performance while negative scores reflect disadvantageous performance. Error bars are standard errors.

4.3.4.3 SGT Split half net scores

Figure 4-13 contrasts adjusted net scores on the SGT for the first 50 trials with net scores over the last 50 trials in both groups. The difference in net scores between the two groups were non-significant at time one (trials 1-50) \( F(1, 35) = 2.21, p=.14, \eta^2 = .05 \) and time two (trials 51-100) \( F(1, 35) = 2.78, p=.10, \eta^2 = .07 \).

Figure 4-13. Mean SGT net scores ([C&D - A&B]) for trials 1-50 and trials 51-100 for the comparison and obese groups. Error bars are standard errors.
4.3.4.4 **SGT Response shift or stay in the continuing choice**

Response shift or stay in the continuing choice is shown in Figure 4-14. There were no differences between the groups in the continuing choice after a bad (A & B) or good Deck (C & D). For both groups, after a bad deck choice, an average of 80% of continuing selections remained on those decks. After a good choice, it was on average 50:50 whether participants would remain on those choices.

![Bar chart showing SGT: Percentage Continuing Card Selection](image)

**Figure 4-14.** SGT Mean probability of shift and stay in the continuing choice.

4.3.4.5 **SGT: Anticipatory SCR and heart rate**

The mean total fluctuation amplitude for anticipatory SCRs (see Figure 4-15) and mean anticipatory heart rate (IB0 - IBI-1) (see Figure 4-16) was calculated separately for the disadvantageous decks (A, B) and the advantageous decks (C, D) decks. There were no significant differences between the groups in average total anticipatory SCR ($t(32) = -1.30, p=.2$). A repeated measures ANOVA with group (obese, comparison) as the between subject factor and deck (disadvantageous, advantageous) as the within subject factor showed a main effect of deck ($F(1, 32) = 7.56, p=.01, \eta^2 = .19$) with both groups showing higher SCR to the advantageous decks than disadvantageous decks. There was no effect of group ($F(1, 32) = 1.71, p=.2, \eta^2 = .05$) and a non-significant interaction ($F(1, 32) = .14, p=.72, \eta^2 = .004$).
Figure 4-15. Mean total anticipatory SCR for disadvantageous (A, B) and advantageous (C, D) decks.

As shown in Figure 4-16, there were no significant differences between the groups in average total anticipatory IBI ($t (25) = -1.19$, $p=.24$). IBI difference scores were similarly submitted to a 2 group (obese, comparison) X 2 deck (disadvantageous, advantageous) repeated measures ANOVA. There was no effect of group ($F (1, 25) = 1.44$, $p=.24$, $\eta^2 = .05$), or deck ($F (1, 25) = .001$, $p=.97$, $\eta^2 = .0$), and a non-significant interaction ($F (1, 25) = .89$, $p=.35$, $\eta^2 = .03$). Both groups showed heart rate slowing (larger IBI intervals) in anticipation of response to both advantageous and disadvantageous decks. The pattern of performance indicated the comparison group showed somewhat greater slowing to the disadvantageous decks, while the obese group showed somewhat more slowing to advantageous decks.

Figure 4-16. Mean anticipatory heart rate (IBI0 - IBI-1) for disadvantageous (A, B) and advantageous (C, D) decks. Error bars are standard errors.
4.3.4.6 SGT: Response SCR and heart rate

The mean total fluctuation amplitude for response SCRs (see Figure 4-17) and mean response heart rate (IBI+1 - IBI-2) (see Figure 4-19) was calculated separately for wins (no associated loss) and for losses (loss or a net loss) from the disadvantageous decks (A, B) and the advantageous decks (C, D). There were no significant differences between the groups in average total response SCR \( t(26) = -.15, p=.88 \). A repeated measures ANOVA with group (obese, comparison) as the between subject factor and deck (disadvantageous, advantageous) and reinforcement (win, loss) as the within subject factors, showed a main effect of reinforcement \( F(1, 26) = 4.77, p=.04, \eta^2 = .155 \) indicating SCR was higher for the losses than for wins in both groups. There was also a deck x reinforcement interaction \( F(1, 26) = 15.48, p=.001, \eta^2 = .37 \).

![SGT: Mean Response SCR after Reward & Punishment](image)

**Figure 4-17.** Mean response SCR for disadvantageous (A, B) and advantageous (C, D) decks. Error bars are standard errors.

The deck x reinforcement interaction is plotted in Figure 4-18. Post hoc analysis using the Sidak adjustment for multiple comparison showed mean response SCR to a loss from a disadvantageous deck was significantly different than mean response SCR to a loss from an advantageous deck \( p=.004 \). The difference between response SCR to a win from a disadvantageous and advantageous deck was non-significant \( p=.7 \). As in the IGT, the overall pattern indicates that SCR is dependent not just on win or loss, but also on deck type. There were no other main effects or significant interactions.
As shown in Figure 4-19, there were no significant differences between the groups in average total response IBI ($t(20) = 1.72$, $p=.10$). The IBI response difference scores to wins and losses were similarly submitted to a 2 group (obese, comparison) X 2 deck (advantageous, disadvantageous) X 2 reinforcement (win, loss) repeated measures ANOVA. There was a main effect of reinforcement ($F(1, 20) = 13.62$, $p=.001$, $\eta^2 = .41$) indicating overall heart rate slowing was greater for losses than wins in both groups. There was a marginally non-significant deck x reinforcement x group interaction ($F(1, 20) = 4.22$, $p=.05$, $\eta^2 = .179$). However, post hoc $t$-tests did not show any significant effects. There were no other main effects or significant interactions.
4.3.5 Memory assessment for the SGT

Memory assessment for the SGT is presented in Table 4-4 below. The majority of participants in both groups correctly identified that they experienced the highest frequency gain on the disadvantageous decks, and to a lesser extent the highest frequency loss on the advantageous decks. However, with regards to EV, 60% or more of participants in both groups stated that they earned the most on the disadvantageous decks, while 47% and above identified that they earned the least on these disadvantageous decks also. Overall, participants in both groups strongly preferred the disadvantageous decks.

Table 4-4. SGT memory assessment. Frequencies for the disadvantageous and advantageous decks are presented.

<table>
<thead>
<tr>
<th></th>
<th>Comparison Group</th>
<th>Obese Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Disadvantageous: n (%)</td>
<td>Disadvantageous: n (%)</td>
</tr>
<tr>
<td></td>
<td>Advantageous: n (%)</td>
<td>Advantageous: n (%)</td>
</tr>
<tr>
<td>Frequency Gain</td>
<td>16 (80%)</td>
<td>19 (90.5%)</td>
</tr>
<tr>
<td></td>
<td>4 (20%)</td>
<td>2 (9.5%)</td>
</tr>
<tr>
<td>Frequency Loss</td>
<td>6 (30%)</td>
<td>9 (40%)</td>
</tr>
<tr>
<td></td>
<td>14 (70%)</td>
<td>12 (60%)</td>
</tr>
<tr>
<td>Earn most</td>
<td>12 (60%)</td>
<td>13 (62%)</td>
</tr>
<tr>
<td></td>
<td>8 (40%)</td>
<td>8 (38%)</td>
</tr>
<tr>
<td>Earn least</td>
<td>13 (65%)</td>
<td>9 (47%)</td>
</tr>
<tr>
<td></td>
<td>7 (35%)</td>
<td>10 (53%)</td>
</tr>
<tr>
<td>Prefer</td>
<td>17 (85%)</td>
<td>20 (95%)</td>
</tr>
<tr>
<td></td>
<td>3 (15%)</td>
<td>1 (5%)</td>
</tr>
<tr>
<td>Dislike</td>
<td>3 (15%)</td>
<td>4 (20%)</td>
</tr>
<tr>
<td></td>
<td>17 (85%)</td>
<td>16 (80%)</td>
</tr>
</tbody>
</table>

4.3.6 Results for the GDT

4.3.6.1 Behavioural performance on the GDT

A between groups ANCOVA, controlling for age, education and BMI, found no significant difference on GDT global net scores ($F (1, 35) = 1.09, p = .3, \eta^2 = .03$). When WAIS-III score was added as a covariate to the model this result remained unchanged. GDT performance of the comparison and obese groups across the four 4 categories are shown in Figure 4-20. ANCOVA controlling for age, education and BMI, with group as the between subject factor and choice as the within subject factor revealed no main effects for choice ($F (2.16, 75.56) = 1.02, p=.37, \eta^2 = .028$), group ($F (1, 35) = .32, p=.58, \eta^2 = .09$), and a non-significant interaction ($F (2.16, 75.56) = .89, p=.42, \eta^2 = .025$). There were no differences
between the groups in the number of disadvantageous / risky \( (F(1, 35) = 1.09, p=.30, \eta^2 = .03) \), or advantageous / safe choices made \( (F(1, 35) = 1.09, p=.30, \eta^2 = .03) \) or in total money at the end of the task \( (F(1, 35) = .99, p=.33, \eta^2 = .03) \).

Figure 4-20. Mean frequency of choice from each of the four categories on the GDT. Error bars are standard errors.

4.3.6.2 Use of negative feedback after a risky choice

Results showed that following a risky choice, there was no significant difference between the comparison \( (M = 62.91\%, SD = 35.97) \) and obese group \( (M = 67.37\%, SD = 32.39) \) in the percentage use of negative feedback (high loss) as a cue to shift to a non-risky alternative in the succeeding trial \( (F(1, 27) = .12, p=.73, \eta^2 = .01) \). Note that in this analysis, participants who never chose a risky alternative or those who did not receive negative feedback after choosing a risky alternative were necessarily excluded. Sample size for this analysis was \( n = 15 \) in the obese group, and \( n = 14 \) in the comparison group. The use of negative feedback as a cue to shift to a non-risky option in the trial following a risky decision was correlated with GDT performance in both the comparison \( (r = .92, p<.001) \) and obese group \( (r = .91, p<.001) \). Seventeen (81\%) of obese participants and 15 (75\%) of comparison participants chose a risky alternative at least once during the GDT.

4.3.6.3 GDT: Anticipatory SCR and anticipatory heart rate

Analogous to the IGT, mean total fluctuation amplitude for anticipatory SCRs (see Figure 4-21) and mean anticipatory heart rate \( (180 - IBI-1) \) (see Figure 4-22) was computed separately for the disadvantageous choices (one or two numbers) and the advantageous
choices (three and four numbers). A repeated measures ANOVA with group (obese, comparison) as the between subject factor and choice (disadvantageous, advantageous) as the within subject factor showed no effect of choice ($F(1, 6) = 1.66, p=.25, \eta^2 = .22$), group ($F(1, 6) = .44, p=.53, \eta^2 = .07$) or any interactions ($F(1, 6) = .43, p=.54, \eta^2 = .06$).

As shown in Figure 4-22, IBI difference scores were similarly submitted to a 2 group (obese, comparison) X 2 choice (disadvantageous, advantageous) repeated measures ANCOVA. No effect of choice ($F(1, 5) = .59, p=.48, \eta^2 = .12$), group ($F(1, 5) = 1.89, p=.23, \eta^2 = .27$) or any interactions ($F(1, 5) = .12, p=.74, \eta^2 = .02$) were observed. On a descriptive level, both groups showed greater heart rate slowing to disadvantageous choices. On advantageous choices, the comparison group showed slowing, although less than that to disadvantageous choices, while the obese group showed recovery to baseline.
It must be noted that participants had to have made a choice from all disadvantageous (one or two numbers) and advantageous (three or four numbers) categories to be included in the above ANOVA which compared psychophysiological data across these categories. As such, only eight participants could be included in the SCR and seven (due to loss of data) in the IBI analyses between risky and safe choices.

4.3.6.4  **GDT: Response SCR and response heart rate**

Analogous to the IGT, mean total fluctuation amplitude for response SCRs (see Figure 4-23) and mean response heart rate (IBI+1 - IBI-2) (see Figure 4-24) was calculated separately for wins and for losses from the disadvantageous choices (one and two numbers) and the advantageous decks (three and four numbers). As there were insufficient numbers across the eight categories to compute a 2 group (obese, comparison) x 2 choice (disadvantageous, advantageous) x 2 reinforcement (win, loss) ANOVA, three Mann-Whitney U tests were used to assess the difference in response SCR between groups for choice and reinforcement (as there were not data for obese participants for a win from the disadvantageous decks this item was not included for comparison). A Bonferroni correction set the new alpha level at .01. No significant differences were observed on any of the comparisons.

![Figure 4-23. Mean total Response SCR for disadvantageous (lines 1 and 2) and advantageous (lines 3 and 4) categories. Error bars are standard errors.](image)

For response heart rate (see Figure 4-24) three Mann-Whitney U tests were similarly used to assess the difference in response SCR between groups for choice and reinforcement (as there was not data for comparison participants for a win from the disadvantageous decks...
this item was not included for comparison). A Bonferroni correction set the new alpha level at .01. No significant differences were observed on any of the comparisons.

![GDT: Mean Response Heart Rate after Reward & Punishment](image)

**Figure 4-24.** Mean response heart rate (IBI+1–IBI-2) for disadvantageous (Lines 1 and 2) and advantageous (lines 3 and four) categories. Error bars are standard errors.

### 4.3.7 Predictors of task performance

Multiple regression analysis was performed on both groups separately to find the best predictors in performance on all three tasks using net score as the dependent variable and WAIS-III, GHQ total, and subscale A, M and NP scores from the BIS-15 as independent variables. Considering both the sample as a whole and each group separately, only GHQ total was predictive of SGT net score for the control group ($\beta = -.618$, $t (16) = -2.32$, $p=.04$).

### 4.3.8 Impaired vs. Non-impaired performers on the IGT

As a way of broadly assessing the premises of the SMH, participants in both groups were classified as impaired (net score <10) or non-impaired on the IGT, as per the criteria established by Bechara and Damasio (2001). Accordingly, 50% ($n = 10$) of the control group, and 67% ($n = 14$) of the obese group were classified as impaired on the IGT. The analysis was as previously applied to the IGT for the obese and comparison participants.

#### 4.3.8.1 IGT: Anticipatory SCR and heart rate

Mean total fluctuation amplitude for anticipatory SCRs (see Figure 4-25) and mean anticipatory heart rate (IB0 – IBI-1) (see Figure 4-26) is presented for non-impaired and impaired performers. A repeated measures ANOVA with group (non-impaired, impaired) as
the between subject factor and deck (disadvantageous, advantageous) as the within subject factor showed a main effect of deck \( F(1, 23) = 12.89, p = .001, \eta^2 = .29 \) with both groups showing higher anticipatory SCR to the disadvantageous decks. There was no effect of group \( F(1, 23) = 1.38, p = .25, \eta^2 = .05 \) and a non-significant interaction \( F(1, 23) = 3.19, p = .08, \eta^2 = .22 \).

![Figure 4-25. Mean anticipatory SCR for disadvantageous (A', B') and advantageous (C', D') decks. Error bars are standard errors.](image)

As shown in Figure 4-26, IBI difference scores were similarly submitted to a 2 group (non-impaired, impaired) X 2 deck (disadvantageous, advantageous) repeated measures ANOVA. There was no effect of group \( F(1, 23) = 1.38, p = .25, \eta^2 = .05 \), or deck \( F(1, 23) = 1.95, p = .17, \eta^2 = .07 \), and a non-significant interaction \( F(1, 23) = 3.19, p = .08, \eta^2 = .122 \). The impaired group showed heart rate slowing (larger IBI intervals) to both advantageous and disadvantageous decks, while the non-impaired group showed slowing to the advantageous decks only and somewhat reduced IBI to disadvantageous decks.
4.3.8.2 IGT: Response SCR and heart rate

Mean total fluctuation amplitude for response SCRs (see Figure 4-27) and mean response heart rate (IBI+1 - IBI-2) (see Figure 4-28) is presented for non-impaired and impaired performers. A repeated measures ANOVA with group (non-impaired, impaired) as the between subject factor and deck (disadvantageous, advantageous) and reinforcement (win, loss) as the within subject factors showed a deck x reinforcement interaction ($F(1, 30) = 8.69, p = .006, \eta^2 = .225$) for response SCR. Post hoc analysis using Sidak showed, as previously articulated, that mean response SCR to a win from a disadvantageous deck was significantly higher than mean response SCR to a win from an advantageous deck ($p = .04$). There was no difference between response SCR to a loss from a disadvantageous and advantageous deck. There was also a reinforcement x group interaction ($F(1, 30) = 7.38, p = .01, \eta^2 = .20$). Post hoc analysis showed that for the impaired group, SCR to loss ($M = .015, SD = .02$) was greater than SCR for wins ($M = .008, SD = .01$); ($F(1, 18) = 6.59, p = .01$). The difference between SCR to loss ($M = .008, SD = .011$) and wins ($M = .005, SD = .006$) for the non-impaired group was non-significant; ($F(1, 12) = 1.20, p = .29$). There were no other main effects or significant interactions.
IBI response difference scores (see Figure 4-28) to wins and losses were similarly submitted to a 2 group (non-impaired, impaired) X 2 deck (advantageous, disadvantageous) X 2 reinforcement (win, loss) repeated measures ANOVA. There were no main effects or significant interactions.

Figure 4-27. Mean response SCR for disadvantageous (A', B') and advantageous (C', D') decks. Error bars are standard errors.

Figure 4-28. Mean response heart rate (IBI+2 – IBI-1) for disadvantageous (A', B') and advantageous (C', D') decks. Error bars are standard errors.
4.4 Discussion

4.4.1 Behavioural performance on the IGT

The results from the IGT were broadly in line with those from Chapters 2 and 3. Accordingly, the morbidly obese group were significantly impaired on the IGT, compared with the comparison group, in terms of both overall performance (net score) and task learning. As in previous investigations (Davis et al., 2004) and in Chapter 2, the obese participants did not demonstrate a clear strategy and performance declined towards the end of the task (see Chapter 3). Further, consistent with Chapter 2, both groups showed a significant preference for infrequent punishment. Previous investigations of obese women (with and without BED) have found that education removed group differences in IGT performance between obese and comparison participants, with those of a higher education performing significantly better (Davis et al., 2010). While education did not have such an effect in the current sample, intelligence did: when intelligence (WAIS-III) scores were added as a covariate to the model, group differences in both overall performance and block learning were no longer significant. Similar findings have been found in OCD where adding education to the IGT reduced the effect of block (Lawrence et al., 2006). However, in the current sample, the interaction term between block scores and group remained significant, indicating that even when controlling for intelligence, the obese group failed to learn at the same rate as the comparison group.

Looking at strategy and the ability to use feedback on the IGT, there were no differences between the groups in terms of choices after a bad deck, indicating the groups were equally sensitive to the punishments associated with the decks. However, after a good deck, the obese group were significantly more likely to switch to a bad deck, while the comparison group were more likely to stay with a good deck. This may tie in with the reversal learning hypothesis previously proposed to explain decision making deficits under uncertain conditions in obesity and other eating disorders (Brogan et al., 2010), whereby participants do not unlearn that choices which were once rewarding are associated with higher long-term costs. Alternatively, it may suggest a random decision making style in the obese group with participants oscillating between the good and bad decks and thus failing to learn to stick with the better decks. In a similar manner, 100% of the comparison group, and 90% of the obese group, correctly identified that they earned the least on the disadvantageous decks. However, 61% of the comparison group, in contrast to 38% of the obese group correctly identified that they earned the most on the advantageous decks. Thus, it seems that
the obese group have greater difficulty intuiting and sticking with advantageous choices on the IGT.

4.4.2 IGT and psychophysiological measures

Anticipatory SCR was higher for disadvantageous than advantageous decks for the sample as a whole, but did not differentiate between the groups, thus contradicting predictions drawn from the SMH that obese participants' impairment on the IGT would be related to deficient anticipatory 'somatic marker' signals. These findings are similar to those obtained in BN (Liao et al., 2009), OCD (Starcke et al., 2009) and Huntington's disease (Campbell et al., 2004) where anticipatory SCR did not differentiate between the intact performance of healthy controls and impaired decision making in these clinical groups. However, they contrast those in AN, where lower anticipatory SCR was associated with deficient IGT performance (Tchanturia et al., 2007). For anticipatory heart rate, as per previous research, the general pattern of heart rate slowing preceding selection was found. However, unlike this research, and inconsistent with the SMH, heart rate slowing did not differentiate between deck or group (Crone et al., 2004). For response SCR, an interaction between deck and reinforcement showed that response SCR to a win from a disadvantageous deck was significantly higher than to a win from an advantageous deck, highlighting that feedback SCR is sensitive not just to reinforcement (win or loss) but also to deck type (disadvantageous, advantageous). This finding is in line with previous research which found SC level was greater following reward from a disadvantageous deck (Jenkinson et al., 2008). An interaction following punishment was not found in both Jenkinson et al.'s and the current study. However, a deck x reinforcement interaction for punishment, whereby losses arising from the disadvantageous rather than advantageous decks elicited a significantly higher SCR, was observed by Liao et al. (2009) for their sample of BN, AN, and healthy controls. The pattern of findings for response heart rate, whereby heart rate slowed to all outcomes, was different to that reported by Crone et al. (2004) who showed that heart rate slowed following loss relative to reward outcomes. Response heart rate did not differentiate between group, deck or reinforcement in the current sample.

The explanations invoked to explain the performance of control participants who perform badly on the IGT, yet generate normal 'somatic marker' signals, seem relevant here in the context of obese participants who were impaired on the task, but unimpaired in anticipatory 'somatic markers'. Accordingly, the obese participants' profile fits that of 'high-
risk' takers as defined by Bechara and Damasio (2002), choosing to override 'somatic marker' signals with conscious deliberation. The explanation that outcomes (reward and punishment) may trigger less autonomic activity resulting in inefficient context updating seems less applicable here in consideration of the deck x reinforcement interaction (Bechara et al., 1999). Similarly, the explanation that poorly performing participants may have failed to integrate acquired stimulus-response associations in the formation of 'somatic markers' (Crone et al., 2004) is not relevant as the obese participants generated anticipatory markers.

4.4.3 Behavioural performance on the SGT

Both groups were significantly and equally impaired on the SGT. Accordingly, there were no overall differences between the groups in net score, with both groups showing a significant preference for the disadvantageous decks (high frequency gain, but low EV) compared to the advantageous decks (low frequency gain, but high EV). As such, it would seem that in line with previous findings (Chiu et al., 2008; Lin et al., 2009), both groups were guided by gain-loss frequency rather than EV in these high-ambiguity gambles. With regards to learning across the task, there was a main effect of block only, indicating that over time, the groups did begin to learn to select more from the advantageous decks, in contrast to previous results in this area where no effect of block was found (Ahn et al., 2008; Chiu et al., 2006). While there was no effect of group, or no difference in the net scores between the groups for the last 50 trials (51-100), on a descriptive level the obese group seemed to show a steeper learning curve than comparison participants with an upward trend over the last three blocks, in contrast to declining performance in the comparison group. However, of note, co-variation for age had a significant negative impact on the performance of the comparison group. Accordingly, when age was adjusted for, there appeared to be a strong negative correlation between age and performance in the comparison group, with older participants performing significantly worse. This effect, though present, did not have the same degree of impact in the obese group. The fact that older participants in the comparison group were found to have very poor SGT performance, may be explained in part by the finding that loss aversion (i.e. which leads to disadvantageous deck selection and poor SGT performance) increases with both age and income (Eric, Simon, & Andreas, 2006). While income was not directly measured in the comparison group, the group had significantly higher intelligence scores, which have been positively associated with income (Murray, 2002). In addition, GHQ scores negatively predicted net score on the SGT for the comparison group only, indicating that a stress
response may have contributed to impaired performance (and increased loss aversion) in this group.

The overall deviation from the advantageous decks in the sample suggests that both groups showed the normal tendency of decision makers to underweight rare events and (or) to be loss averse (Erev & Barron, 2005). Underweighting of rare events refers to the tendency to underweight small probabilities of rare events and prefer the alternative that provides the best outcome most of the time. Loss aversion refers to the tendency to prefer the alternative that decreases the probability of losses even if it is associated with a lower expected gain (Ahn et al., 2008). These tendencies fit in with a potential implicit ‘gain-stay, lose-randomise’ strategy demonstrated by participants to cope with the uncertain situation, whereby after a bad choice (associated with frequent gains, and infrequent large losses), an average of 80% of continuing selections remained on that choice, while after a good choice (associated with frequent losses, and infrequent large gains) it was 50:50 as to whether participants would remain on those choices. This tendency, also found in a previous study of healthy student participants, has been observed in human and animal appetitive and avoidance experiments in which reward or punishment is encountered (Chiu et al., 2008). Both groups of participants seemed to intuit the frequency gain structure of the task, with 80% of the comparison group and 90% of the control group reporting that they won most often on the disadvantageous decks. As per the loss aversion assumption, the majority of participants in both groups (>85%) reported preferring these disadvantageous decks. With regards to EV, 60 – 62% of participants in both groups reported they earned the most on the disadvantageous decks. However, 65% of comparison participants and 47% of obese participants reported that they earned the least on these decks also. As such, it would seem that participants did not have a clear sense of EV throughout the task.

4.4.4 SGT and psychophysiological measures

In terms of psychophysiological measures on the SGT, both groups showed higher anticipatory SCR to the advantageous decks. Similar to the greater anticipatory SCR shown to the disadvantageous decks of the IGT (higher magnitude losses), this may be attributable to the higher frequency of punishments associated with these decks in the SGT. In support, Crone et al. (2004) found that for good IGT performers, choices associated with frequent but small punishments (similar to the advantageous decks on the SGT) were preceded by larger SCRs and heart rate slowing, suggesting that these choices are experienced as more aversive
than choices with infrequent but large punishments (as in the disadvantageous decks of the SGT). For anticipatory heart rate, similar to the IGT, both groups showed slowing in anticipation of a response; however, there was no effect of group or deck, or any significant interactions. For feedback responses, there was a main effect of reinforcement, with greater response SCR and heart rate slowing to losses than wins. This is in line with the broad tenet of neuroeconomics that responses to loss are more unpleasant than responses to winning are pleasant (Liao et al., 2009). For response SCR, replicating findings for the IGT, there was a deck x reinforcement interaction, indicating that feedback SCR is sensitive to both variables. The interaction was for loss outcomes on this occasion, with a loss to an advantageous deck (high frequency of loss) significantly higher than to a loss from a disadvantageous deck (high frequency of gains), again likely attributable to the higher frequency of punishments associated with these decks.

The results here for the sample as a whole provide support for the view that participants respond to immediate gain or loss and not final outcome in decision making tasks under uncertainty. This interpretation is further supported by the finding that participants in both groups chose infrequent punishment decks B and D significantly more often than frequent punishment (but smaller magnitude) decks A and C on the IGT, both in the current sample and in previous research (Brogan et al., In Press). This is in line with the prominent Deck B phenomenon cited in the literature (Lin, Chiu, & Huang, 2007). For example, Dunn et al. (2006) found that of 38 IGT related studies only five utilised the ‘four-deck format’ to display their findings. However, all five studies demonstrated that Deck B was chosen more frequently than deck A, and four out of the five studies showed that deck B was chosen more frequently than deck C or D. Such findings indicate the importance of gain frequency in decision making for both these groups, contributing to their disadvantageous SGT performance. However, as the obese group was impaired on both the SGT and the IGT, unlike the comparison group who were impaired on the SGT only, this suggests that the IGT requires the intuition of more than just the frequency of gains and losses for successful task performance. Indeed, as outlined by Yechiam, Stout, Busemeyer, Rock, and Finn (2005) and drawing on the Expectancy Valence model discussed in Chapter 1 (Busemeyer & Stout, 2002), decision making impairments on the IGT can be broken down into three basic components. The first is a motivational factor producing a tendency to be attracted by gains and to ignore losses; the second is a learning rate factor, producing a tendency to focus on recent events and to forget or rapidly discount past losses; the third is a response factor causing choices to
be made erratically owing to factors such as loss of interest, boredom or tiredness (Yechiam et al., 2005). The SGT potentially tapped the first component here, that is, the motivational factor, and demonstrated intact performance of obese participants in this regard as they were equally sensitive to loss as the comparison group. As such, impaired IGT performance may derive from one of the latter two factors, that is the update and sensitivity parameters of the Expectancy Valence model (Busemeyer & Stout, 2002). However, consideration must be given to the fact that feedback is presented in the form of a net outcome on the SGT (in contrast to the IGT where punishment follows reward), and differences in EV between the good and bad decks are more salient, both of which may make the SGT more explicit and interpretable than the IGT. This brings us to our test of explicit decision making, the GDT.

4.4.5 Behavioural performance on the GDT

On the GDT, the explicit test of decision making employed here, there were no differences between the groups in overall net score, or in the frequency of choices from each of the four categories. In addition, there were no differences between the groups in the use of negative feedback as a cue to shift to a non-risky alternative in the succeeding trial. The intact use of feedback broadly contrasts findings on the IGT where obese participants were significantly more likely to switch to a bad deck, after a good deck, than comparison participants who were more likely to stay on that deck, thus demonstrating impaired use of feedback. This lack of impairment is somewhat surprising, given obese participants were found to be impaired on the latter half of the IGT (trials 51 – 100) where the contingencies of the task may be better understood (i.e. more explicit; defined as the hunch and conceptual periods) (Brogan et al., In Press), and that obese BED females were impaired on the GDT (Svaldi et al., 2010). However, it seems from the current data, that the obese participants did not gain a clear understanding of the IGT, particularly with regard to the positive EV of the advantageous decks. Further, a clear difference between the current sample and previous studies in the disordered eating domain using the GDT, is that the majority of the current sample was binge-free (except for two participants), while the previous studies involved binge eating groups including BN (Brand, Franke-Sievert et al., 2007) and BED (Svaldi et al., 2010), both of whom showed impairments on the task. It could be argued that the eating behaviour of individuals with BN and BED, which involves bouts of compulsive overeating, is in many ways more explicitly deleterious to health, than the thousands of small choices whereby caloric intake exceeds caloric expenditure, resulting in obesity (Weber & Johnson, 2009). Additionally, the BN study found GDT performance was associated with components of
executive functioning, for example, cognitive flexibility as measured by the Trail Making Test Part B in addition to the Colour-Word Interference Test (although scores were in the normal range), whereby the longer the patients needed on the tasks the more disadvantageous alternatives they selected on the GDT. However, executive functioning was not measured in the current sample and so its potential impact on GDT performance is unknown. In summary, it seems that at the outset, when the decision is an explicit one, in which the contingencies of the task are comprehensible and the rules for reward and punishment are stable, obese individuals perform as well as normal weight individuals.

4.4.6 GDT and psychophysiological measures

For anticipatory SCR, no effects of choice, group or any interactions were found, in line with previous investigations of OCD participants and healthy controls, who were both unimpaired on the GDT (Starcke et al., 2010). This pattern was the same for anticipatory heart rate. The finding that there were no differences in anticipatory SCR to choice (risky or safe) is similar to that in previous research of healthy participants (Brand, Grabenhorst et al., 2007), and is consistent with the view that the GDT can be successfully performed using cognitive strategies and without the presence of anticipatory somatic biasing signals. However, on a descriptive level, both groups showed greater SCR and heart rate slowing to disadvantageous compared to advantageous choices. No effects were similarly observed for response SCR and heart rate. This contrasts previous findings which found heightened response SCR to risky choices (Brand, Grabenhorst et al., 2007; Starcke et al., 2009). Interestingly, for response heart rate on the GDT, participants showed heart rate acceleration after reward and punishment outcomes, in contrast to the previous two tasks where participants generally showed heart rate slowing after response. This altered physiological reaction may have been related to the explicit nature of the task creating more certainty of the outcomes, or the fact that this task was always administered last. Caution must be expressed when interpreting the above physiological data. Due to loss of data for technical reasons, as well as the fact that participants were required to have made choices from the disadvantageous categories to be included in the analysis, sample size was very small on these comparisons and power to detect effects consequently reduced.
4.4.7 The IGT: Impaired versus non-impaired decision makers

A final research question posed that remains to be addressed, is whether the IGT data analysed by successful or unsuccessful (net score <10) (Bechara & Damasio, 2002) performance, would support the SMH. In line with the findings from the comparison of obese and non-obese participants, anticipatory SCR was higher to the disadvantageous than advantageous decks for the sample as a whole, with no effect of group or any interactions observed. Larger SCRs to risky decks are consistent with the claim that electrodermal responses are proportional to affect value and especially sensitive to the negative one (Fowles, 1980). There were no effects of anticipatory heart rate. For response SCR, there was a deck x reinforcement interaction (as in the IGT and SGT previously) where response SCR to a win from a disadvantageous deck was significantly higher than that from an advantageous deck. In addition, a reinforcement x group interaction showed that for the impaired group, SCR for loss was greater relative to SCR for wins. This contrasts previous results in OCD (Starcke et al., 2009), AN (Tchanturia et al., 2007) and Huntington’s disease (Campbell et al., 2004), where reduced autonomic responses to loss were associated with impaired performance and conversely enhanced responses with successful performance. This perhaps indicates that increased sensitivity to loss enhances IGT performance up to a threshold point, after which high sensitivity to loss may result in impaired performance. There were no effects for response heart rate.

What do these findings mean for the SMH? Crucial to the SMH is the argument that heightened anticipatory ‘somatic marker’ signals to the disadvantageous decks are correlated with successful task performance and the absence of such signals with impaired performance. As such, these nonconscious biasing signals guide successful decision making under situations of uncertainty. However, anticipatory SCR failed to distinguish between good and bad decision makers in the current sample, that is, between normal and impaired performance on the IGT. These findings are echoed by Suzuki et al. (2003) and Jenkinson et al. (2008) who found anticipatory ‘somatic marker’ signals did not discriminate between impaired and non-impaired decision makers on the IGT in healthy individuals. While the SMH emphasises the importance of anticipatory ‘somatic markers’ in guiding future decisions, as mentioned previously, more recent research has suggested that differences in appraisal SCR may underlie decision making. Consistent with this view, Suzuki et al. (2003) found low SCRs for appraising the monetary outcomes of disadvantageous decisions were related to impaired performance in healthy participants. Similarly, as discussed above, reduced autonomic response to loss has
been associated with impaired decision making in various clinical groups. However, this issue is still under debate with other researchers finding that appraisal physiology is independent of IGT performance (Crone et al., 2004), and concluding that decision making impairments in poor performers arise from a weak somatic response generated by secondary (i.e. acquired) rather than primary (stimuli directly set as pleasurable or aversive automatically eliciting a somatic response) inducers of reward and punishment. Irrespective of this debate, in the current sample, autonomic responses to wins and losses in the impaired and non-impaired group did not interact with deck type (advantageous, disadvantageous), failing to support the SMH.

The pattern of SCR findings here broadly replicates those of Jenkinson et al. (2008), who examined the validity of the SMH in a population of healthy individuals using the IGT. In accordance with the results here, they found that for the sample as a whole, SCR was greater when anticipating choices from the bad decks compared with the good decks and greater following a reward from a bad deck. As articulated by Jenkinson et al., these findings support previous claims that 'somatic markers' influence decision making in terms of immediate deck selections when performing the IGT, by physiologically distinguishing between good and bad options prior to making a choice (Bechara & Damasio, 2002; Bechara et al., 1999; Bechara et al., 2002; Crone et al., 2004), and immediately after reward (Suzuki et al., 2003). That is, 'somatic markers' distinguished between good vs. bad decision making. However, the lack of differences between normal (non-impaired) and impaired performers, in anticipatory and / or appraisal autonomic responses for disadvantageous decisions, suggests that 'somatic markers' failed to discriminate between good and bad decision makers on the IGT. As such, these conclusions corroborate those of Jenkinson et al. that autonomic activity does not discriminate between good and bad decision makers in terms of overall performance on the IGT, but instead reflects the immediate decision at hand. Further, the pattern of findings here are consistent with proposals that overall performance on the IGT is independent of physiological activity which reflects only the magnitude of immediate reward or punishment resulting from the chosen deck (Tomb, Hauser, Deldin, & Caramazza, 2002). In support, previous research also found greater SCR for anticipation to the disadvantageous decks (Suzuki et al., 2003) and response to rewards (Jenkinson et al.) and punishments (Liao et al., 2009) from the disadvantageous decks (high magnitude gain and loss) compared with advantageous decks (low magnitude gain and loss). Such effects existed in these studies despite failing to distinguish between normal and impaired performance on the task. A caveat to these
conclusions is that unlike previous investigations where healthy samples were used to explore the validity of the SMH (Jenkinson et al., 2008; Suzuki et al., 2003), conclusions here were based on a mixed sample of obese and non-obese individuals and so does not represent a pure test of the SMH.

4.4.8 Limitations and future research

The sample used in this study was small. While power was deemed sufficient with regards to behavioural performance on the IGT, replication with a larger sample is desirable, particularly with reference to psychophysiological data. This point is rendered more salient taking into account the notoriously large individual differences in human SCR (Dawson et al., 2000). Similarly, the error bars for the heart rate graphs demonstrate the substantial individual differences in HRV. On a related note, the fact the study involved obese individuals may have had an influence on the SC data. The relationship between core temperature and obesity has received limited attention in the literature. A number of studies has suggested an inverse association between temperature and obesity (Landsberg, Young, Leonard, Linsenmeier, & Turek, 2009). Others have found that while core body temperature does not differ significantly between obese and non-obese individuals, augmented heat release from the hands may offset heat retention in areas of the body with greater adiposity, thereby helping to maintain normothermia in obesity (Savastano et al., 2009). These findings, while only in a small sample of 23 obese and 13 normal weight participants, could present significant challenges to the use of SC data in obese populations. Similarly, there is evidence that long-term HRV, as measured by the SD of all normal RR intervals (SDANN) is attenuated in obese participants and improves with weight loss and bariatric surgery, which would also impact on IBI measurement here (Karason, Mølgaard, Wikstrand, & Sjöström, 1999; Perugini et al., 2010). The issue seems to be subject to controversy, with other researchers finding no difference between HRV in lean, overweight and obese individuals (Taçoğ, Açığöz, Kocaman, Özdemir, & Čengel, 2010). Reassuringly, in the current sample, there were no differences in the average anticipatory and response SC and heart rate data in obese and non-obese individuals.

The inclusion of heart rate in addition to SC measures was progressive taking into account the criticism that the IGT over-relies on one source of psychophysiological data (Dunn et al., 2006). However, unlike previous investigations using heart rate measures with the IGT (Crone et al., 2004), heart rate did not relate to IGT performance and was less discriminative of
decision making on all three tasks than SCR. It could be speculated that perhaps this was related to the difference in samples in the two studies, undergraduate students in Crone et al. (2004), and healthy, overweight, and obese adults in the present study. Indeed, the range of HRV was smaller in the present study compared to Crone et al., perhaps attributable to the fact that HRV is known to decline with increasing age (O'Brien, O'Hare, & Corrall, 1986; Reardon & Malik, 1996). A restriction of range due to the older sample used in this study may have reduced the ability to detect effects. However, it should be noted that Crone et al. found anticipatory heart rate only differentiated good from bad and moderate performers on the IGT, while response heart rate was similar for all performance groups.

Concerning the acquisition of psychophysiological data, the matching of an individual's card choice to a specific SCR or IBI event is regarded as imprecise (Carter & Pasqualini, 2004). In addition, although a baseline measure of five minutes was taken here to allow participants to adjust to the testing environment, it is impossible to separate out physiological activation to the very act of doing the tasks, versus the tasks themselves. The data both here and in the previous research on impaired IGT / GDT performance and altered anticipatory / feedback SCR is an association only and causality should not be inferred. As discussed in Chapter 1, there is substantial variability in the measurement and analysis of SCR data across studies, with such difference in methodology undoubtedly impacting the ability to compare these studies and their differing degrees of support for the SMH. On a similar topic, also mentioned in Chapter 1, there has been substantial variability in the time windows adopted in measuring physiological (SCR and heart rate) activity. While the time windows in the present study were carefully chosen based on previous research, as well as the desire to ensure a reasonable length of time for completion of the tasks, it is possible that the relatively brief windows used to capture event related changes may have resulted in overlap of anticipatory and appraisal physiological activity, leading to a failure to clearly distinguish these events types. However, the windows adopted were longer than those of previous researchers who have found significant effects in physiological activity (e.g. Bechara & Damasio, 2002; Crone et al., 2004), while generally adhering to the recommended time window of 1 – 4s or shorter to capture stimulus-related activity and to avoid contamination of responses by nonspecific changes in autonomic arousal (Dawson et al., 2000).

The high proportion of healthy participants whose performance on the IGT was classified as impaired (50%), is conspicuous compared to previous reports of 37% (Bechara &
Damasio, 2002) and 38% (Brogan et al., In Press). However, Davis et al. (2007) found 46% of healthy females were impaired on the task, which is more fitting with the results in the comparison group here, 75% of whom were female. In addition, the comparison group in the current study involved university students. Jenkinson et al. (2008) who used university students in their study found 61% of healthy individuals were impaired. The fact that the comparison group involved overweight individuals may also have influenced the degree of impairment in this group, given overweight individuals have shown to be impaired on the IGT (Davis et al., 2004).

Concerning findings from the tasks themselves, conclusions from the SGT that obese individuals demonstrate the normal tendencies to underweight rare events and (or) to be loss averse, and that IGT impairments do not stem from a motivational deficit to wins and losses, are constrained by the fact that the task may have been more explicit than the uncertain IGT. This may be attributable to the presentation of outcomes on the SGT in the form of a net score, and the enlargement of the differences in EV between the good and bad decks. As shown from the GDT, obese individuals are not impaired on decision making tasks of an explicit nature. As such, both sensitivity to frequency of gain and loss, but also the potentially explicit nature of the task, may have contributed to performance. Finally, with regard to interpretations from the GDT, in contrast to previous disordered eating studies, the impact of executive functioning on performance was not assessed.

Based on the expressed limitations in sample size, it is recommended that future research replicate the decision making tasks used here, including psychophysiological measures, with larger samples. In addition, it is anticipated that the application of the Expectancy Valence model (Busemeyer & Stout, 2002) would further inform the nature of decision making deficits on the IGT and the ways in which they contrast with other clinical groups.

4.5 Summary of findings

In line with previous results using the original IGT (Bechara et al., 1994), morbidly obese participants were significantly impaired on the newer version of the task (Bechara, Tranel et al., 2000). Inconsistent with the premises of the SMH, the obese group showed heightened anticipatory SCR to disadvantageous decks, demonstrating that IGT deficits in obesity are not associated with impaired affective biasing signals. There were similarly no
differences between the groups in response SCR. According to Bechara and Damasio (2002) such poor IGT performers, who show normal activity following outcomes and develop anticipatory SCRs, are high-risk takers, because they consciously override the information derived from 'somatic markers'. With regards to conscious knowledge of the task, obese participants did not seem to gain a clear understanding of the EV of the decks by the end of the task. In an attempt to further specify cognitive decision making deficits in obesity, two other decision tasks, the SGT and the GDT were employed. The SGT sought to explore the relative contributions of EV and gain-loss frequency to decision making in obesity. As the obese group, like the comparison group, were significantly impaired on this task preferring the high-gain frequency but ultimately low EV disadvantageous decks, over the low-gain frequency but high EV advantageous decks, it can be concluded that both groups responded to immediate reinforcement as opposed to long-term EV. Extrapolating from this, it can also be concluded that obese participants showed the normal tendency to underweight rare events and (or) be loss averse. This finding is in accord with the prominent Deck B phenomenon on the IGT (Lin et al., 2007), which has also been confirmed in a morbidly obese population (Brogan et al., In Press). These results present a problem for the SMH, which argues that participants are foresighted in learning to respond to long-term EV on the IGT. However, the fact that obese participants were impaired on the IGT, but unimpaired on the SGT, highlights that while EV and gain-loss frequency may be compounded on the IGT, an intact response to gain-loss frequency may be necessary but not sufficient for successful performance on the IGT. Indeed, in line with the Expectancy Valence model (Busemeyer & Stout, 2002) which aims to decompose IGT performance into its constituent parts, their performance on the SGT would suggest obese participants would not be impaired on the motivation parameter but deficits may consequently stem from the update or sensitivity parameters. Psychophysiological data did not discriminate between the groups on the SGT. For the sample as a whole, anticipatory SCR was higher to the advantageous decks (likely attributable to the higher frequency of loss associated with these decks), and response SCR and heart rate slowing were greater for losses than wins, in line with the view that responses to loss are more unpleasant than responses to winning are pleasant.

In contrast to previous studies in disordered eating groups, obese participants showed intact performance on the GDT, indicating that their decision making is unimpaired in explicit situations when the rules of the task are comprehensible and the gain / loss contingencies stable. Psychophysiological data did not discriminate between group or decision making for
the sample as a whole on the GDT. With respect to the individual difference variables incorporated in the study, intelligence was found to play a significant role on the IGT only, removing group differences in overall task performance. The psychometric measures of impulsivity and general health (as a proxy for task motivation) included in the study did not uniquely predict performance on any of the tasks. Finally, in order to address the validity of the SMH, the psychophysiological profile of impaired vs. non-impaired decision makers on the IGT was examined. In line with previous findings it was concluded that 'somatic marker' signals may only discriminate between good and bad decks (i.e. good vs. bad decision making) by reflecting the magnitude of gains and losses, but is independent of long-term consequences and does not discriminate between good vs. bad decision makers (Jenkinson et al., 2008). Indeed, this conclusion echoes the first of Chiu et al.'s (2008) interpretations of the possible role of 'somatic marker' signals in decision making. Accordingly, in light of the SGT findings in healthy participants, they proposed that the 'somatic marker' system may guide decision making behaviour via rough-estimation processing (gain - loss frequency) and not a precise calculation (EV: probability x value). However, it is incompatible with their second and third interpretations where they proposed that the 'somatic marker' system may not be immediately related to decision guidance, and that 'somatic markers' may not globally direct choice behaviour in situations of uncertainty.
Chapter 5: Restraint, External and Emotional Eating: A Food Diary
Assessment of the Relationships between Self-Reported Eating Style and Food Intake

"Stressed spelled backwards is desserts. Coincidence? I think not!" ~ Author Unknown

5.1 Introduction

Psychological factors play an important role in the initiation of both healthy and disordered eating (Jackson, Cooper, Mintz, & Albino, 2003). Chapters 2, 3 and 4 addressed such factors using a relatively structured, lab-based, neuropsychological approach measuring discrete aspects of implicit and explicit decision making and the potential influence of psychophysiology on these processes in line with the theoretical framework specified by the SMH (A. R. Damasio, 1994). However, decision making around food and food choice occurs in a context, including for example, the individual's home, work and social environment, all of which are embedded in the broader obesogenic environment. This obesogenic environment, forming the downhill slope of the 'runaway weight gain train' (see Chapter 1; Swinburn & Egger, 2004), gives rise to restricted opportunities for physical activity and high availability and active promotion (e.g. advertising and marketing) of high fat, sugar and energy dense foods (Snoek, Van Strien, Janssens, & Engels, 2007). Indeed, the importance of the environmental context is highlighted by the Analysis Grid for Environments Linked to Obesity (ANGELO) framework (Swinburn, Egger, & Raza, 1999) and more recently the Obesity Policy Action (OPA) framework (Sacks, Swinburn, & Lawrence, 2009), which identifies a combination of socio-ecological (upstream), lifestyle (midstream) and health services (downstream) policy targets to control the growing obesity epidemic. With this in mind, the final empirical chapter of this thesis sought to take a more naturalistic and ecologically valid approach to assessing affective and cognitive influences on decision making in morbidly obese individuals.

A fundamental question in the context of rising trends in obesity is why some individuals in the obesogenic environment are able to keep their weight in balance for years, while others become overweight, perhaps even in childhood (Snoek et al., 2007). Focusing on rising food intake as the dominant factor in the obesity epidemic (Silventoinen et al., 2004), increasing attention has been directed towards understanding why people eat and indeed overeat. Particularly important in this regard has been the development of questionnaires and
scales, which assess the dominant psychological theories on triggers of overeating: restraint, external and emotional eating. These theories can be seen to assess cognitive (restraint eating), sensory (external), and affective (emotional eating) aspects of decision making around food and food choice. The constructs or eating styles have been operationalised by a number of psychometric measures and have been widely cited in the literature. For example, emotional eating in the EES (Arnow et al., 1995), the Emotional Overeating Questionnaire (EOQ; Masheb & Grilo, 2006), and the Eating and Appraisal Due to Emotions and Stress (EADES; Ozier et al., 2007); restraint in the Restraint Scale (Herman & Polivy, 1975) and the Three-Factor Eating Questionnaire (TFEQ; Stunkard & Messik, 1985); and, all three perhaps most widely in the DEBQ (Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986). These scales are increasingly associated with eating pathology and obesity, in both adults (Van Strien et al., 2005) and children (Braet & Van Strien, 1997). However, evidence regarding the relationships between these self-reported eating styles and food intake, and therefore more generally decision making around food intake, has been inconsistent, leading to suggestions that it may be complex to adequately assess one’s own eating behaviour (Evers, de Ridder & Adriaanse, 2009). Studies which aimed to assess this relationship between self-reported eating style and food intake have used divergent methodologies including the food diary methodology, the Food Frequency Questionnaire (FFQ), as well as more structured lab-based approaches. There has also been substantial variability in the measurement of food intake, with some studies addressing overall energy intake, and others focusing on more specific aspects of dietary intake (e.g. fats, carbohydrates) or snacking behaviour. In addition, some research has focused on the moderating effect of these variables in the hassles-snacking relationship and eating under stress. Examples of such studies are considered below.

5.1.1 Food diary and FFQ studies

The Stanislas Family study (Lluch, Herbeth, Méjean, & Siest, 2000) assessed the relationships between dietary intake, eating style and overweight in a large sample of healthy male and female adults and children, through the completion of three-day dietary records in tandem with the DEBQ (Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986). They found that dietary restraint was positively correlated with being overweight, and associated with lower energy intake in all groups (men, women, boys and girls) a finding which has been replicated in the literature (French, Jeffery, & Wing, 1994; Klesges, Isbell, & Klesges, 1992; Van Strien, Frijters, Van Staveren, Defares, & Deurenberg, 1986). External eating was associated with higher energy intakes (except in men), and there was no relationship between emotional
eating and energy intake (Luch et al., 2000). Their findings were very similar to those found using the DEBQ in conjunction with a diet history taken by a dietitian in a study of healthy adolescents where restraint was predictive of lower intake, external eating of higher intake and emotional eating was unrelated to intake (Wardle et al., 1992). In a food diary study focusing specifically on snack intake and emotional eating, normal weight participants completed seven-day snack diaries in combination with emotional experiences, with daily registering to minimise recall bias (Adriaanse, de Bidder, & Evers, 2010). Results showed that emotional eater scales did not predict caloric intake in response to negative emotion. Instead, snack intake was better predicted by habit strength and by restraint eating.

Comparable results were obtained in a study of healthy female students which assessed eating style with the DEBQ (Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986) and energy intake over a one-month period using a self-report FFQ (Anschutz, Van Strien, Van De Ven, & Engels, 2009). Using structural equation modelling and controlling for BMI and physical activity, they found that restraint eating was consistently negatively related to total energy intake, fat and carbohydrate intake. External eating was positively related, and emotional eating was unrelated to all dependent variables. An additional study using a FFQ and the DEBQ, but taking a more longitudinal approach, followed patients newly diagnosed with Type 2 diabetes over a four year period and found that: (a) increases in restraint eating were associated with a decrease in the intake of fat and saturated fat, (b) decreases in external eating with decreases in intake of energy and fat, while (c) emotional eating did not change over the four year period or explain variance in the change in food intake measures (Van Strien & Van de Laar, 2008).

A revised version of the TFEQ (Stunkard & Messik, 1985), the TFEQ-R18 (Karlsson, Persson, Sjostrom, & Sullivan, 2000), which comprises three different scales corresponding to cognitive restraint, emotional eating, and uncontrolled eating, was used to define the relationships between eating behaviour and food intake at the population level similarly using a FFQ (De Lauzon et al., 2004). Accordingly they found that the association between TFEQ-18 scores and energy intake was largely similar in both genders but different among middle aged adults compared to teenagers and young adults. With regards to overall food intake, girls who scored higher on restraint eating had a lower energy intake, while in adult men energy intake increased with uncontrolled eating (defined as the inability to limit food intake and an extreme sensitivity to external stimuli). With reference to specific food groups, in adults, higher
cognitive restraint was positively associated with healthy food groups (e.g. fat reduced foods or green vegetables) and negatively associated with French fries, sugar and confectionary. Energy-dense foods were positively associated with uncontrolled eating and emotional eaters had a higher consumption of snacking foods such as oleaginous fruits (e.g. peanuts) and cakes/pastries/biscuits. In teenagers and young adults, there were fewer associations between TFEQ-18 scores and reported intake of specific food groups, with the majority of associations relating to cognitive restraint. Accordingly, teenagers and young adults who exhibited high cognitive restraint reported consumption of fewer energy-dense foods (e.g. sugar, French fries, processed meat) but not of more 'healthy foods' as was the case in adults. Only alcoholic beverages were positively related to uncontrolled eating (De Lauzon et al., 2004).

At this juncture it seems that studies using the food diary and FFQ methodologies to assess the general relationship between self-reported eating style and food intake within healthy populations have largely shown a role for restraint eating in predicting lower intake, external eating higher intake, and emotional eating being unrelated to intake. In addition, high cognitive restraint has been positively associated with the consumption of healthy food choices, and negatively associated with unhealthy foods choices, fat, saturated fat and carbohydrate intake. By contrast, external eating has been positively associated with such unhealthy food choices. However, when studies addressing this relationship within the context of daily hassles and stress-induced eating are considered, this picture becomes more complex.

5.1.2 Eating styles as moderators of stress-induced eating

In a study addressing the hassle-eating behaviour relationship and its moderators, daily hassles were associated with increased consumption of high fat/sugar snacks and with a reduction in main meals and vegetable consumption (O'Connor et al., 2008). However, there was a significantly stronger positive association between daily hassles and between-meal snacking for individuals who were high on restraint, emotional, and external eating (as measured by the DEBQ; Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986) and disinhibition (as measured by the TFEQ; Stunkard & Messik, 1985), or who were female or obese. These results indicate that individuals differ in their level of vulnerability to stress-induced eating. When the impact of the multiple moderators was considered simultaneously using multilevel random coefficient modelling, emotional eating reliably emerged as the pre-eminent moderating variable of the stress-between meal snacking relationship. Similarly, in a study of normal weight (pre-menopausal) women using a 14-day daily hassles and snack
intake diary, significant positive associations were found between daily hassles and snack intake within high but not low cortisol reactors in response to stress, in line with previous research which found food intake is promoted by cortisol reactivity to stress (Newman et al., 2007). Restraint, emotional, and external eating (again measured by the DEBQ) were significantly positively associated with snack intake in the overall sample. However, the associations for emotional and restraint eating were significantly stronger for high cortisol reactors to stress. A possible explanation for this finding is that individuals high in these eating style variables produce high levels of cortisol, which then has an appetitive effect. While further investigations of the relationship between eating style and cortisol output are needed to further elucidate this finding, the authors suggest that individual differences in cortisol reactivity to stress may in part contribute to an understanding of the moderating effect of eating style on stress-induced eating.

In contrast to the above, largely positive findings, a study of healthy weight students using a seven-day diary found that only external eating moderated the hassles-snacking relationship, with those high on external eating showing significant positive relationships between hassles and snacking, while those low on external eating showed no significant relationship (Conner, Fitter, & Fletcher, 1999). Neither emotional nor restraint eating were significant moderators of the hassles-snacks relationship. Similar null findings were obtained by O'Connor and O'Connor (2004) in a study using two-week snack diaries during a stressful (examination) and non-stressful period, which found that all three eating style variables measured by the DEBQ (Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986) did not have a significant influence on eating behaviour under stress in a student population.

Due to the inconsistency in findings of these studies, reporting that some, all, or none of the eating style variables moderate the stress-eating behaviour relationship, later studies have since adopted a lab-based approach and have paid particular attention to the concept of emotional eating. These studies employed the lab environment in an attempt to standardise the emotional encounter, and both internal (e.g. degree of hunger) and external states (e.g. food accessibility) (Evers et al., 2009).

5.1.3 Experimental studies: Emotional eating and intake when emotional

Oliver, Wardle, and Gibson (2000) devised a lab-based study involving healthy men and women where anticipation of a speech performance was used as a stressor in the
experimental group, while the control group listened to a neutral passage of text. Subsequently, food intake was measured through the provision of an ad libitum meal which included sweet, salty, or bland high- and low-fat foods, and eating style was assessed by the DEBQ (Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986). While stress did not alter overall intake, they found that stressed emotional eaters ate more sweet, high-fat foods and a more energy-dense meal than unstressed and non-emotional eaters. Dietary restraint did not significantly affect appetitive responses to stress.

Contrary to the above findings, Evers et al. (2009) argued that the evidence that self-reported emotional eaters truly increase their food intake during emotional encounters is inconclusive. As such, in a series of lab-based experiments they tested whether emotional eater scales capture the tendency to eat, when feeling emotional in healthy women. Procedures for emotion induction employed across the experiments included the use of vignettes, film excerpts, recall, or the provision of false feedback. Emotion induction was assessed at baseline and after induction by asking participants how strongly they experience different emotions. In control conditions, either positive or no emotions were induced. Hunger state was standardised across all four studies and food consumption was assessed by bogus (hoax) taste tests of both sweet and savoury foods including chocolate, crisps, raisins, crackers, cookies or fruit. At the end of the study, participants completed the DEBQ to assess eating style (Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986). Results showed that self-reported emotional eaters did not increase food intake during emotional encounters. As such, it seems that the relationship between self-reported emotional eating and actual eating behaviour remains to be determined even in the laboratory environment.

5.1.4 Aims of the current study

As is evident from the literature reviewed here, the relationship between self-reported eating style and actual food intake is somewhat inconsistent. Studies using the food diary or FFQ methodology have largely shown a role for self-reported cognitive restraint, and external eating in predicting dietary intake, but not for affective influences as measured by emotional eating, while studies assessing the moderating role of these variables in the hassles-eating behaviour relationship do not indicate a clear pattern.

A large number of studies in this domain have focused solely on cognitive restraint theory (De Castro, 1995; Van Strien, Frijters, Van Staveren et al., 1986), normal weight
participants (Conner et al., 1999; Lluch et al., 2000; Newman et al., 2007; Oliver et al., 2000) and often female only participants (Adriaanse et al., 2010; Anschutz et al., 2009; Evers et al., 2009; O'Connor & O'Connor, 2004). However, as these scales are so widely associated with eating pathology and obesity, the extension of this research to a morbidly obese population would seem highly pertinent. As such, this study’s first research question aimed to investigate the relationships between self-reported eating style (cognitive restraint, emotional and external eating) and eating behaviour, in a morbidly obese population including both male and female participants. In keeping with the aim of this chapter, to assess decision making around food and food choice in a context-relevant or ecologically valid setting, the food diary methodology was used to achieve this. Eating behaviour was assessed in terms of total food intake and food type as per a food pyramid analysis of intake (Harrington et al., 2008). It was considered important to differentiate food types given within the context of comfort food consumption and emotional eating, the type of food eaten can be linked to affect asymmetry, with foods higher in sugar and fat content more efficient in alleviating negative affect (Dubé et al., 2005; Ganley, 1989) and low calorie foods more efficient in increasing positive affect (Dubé et al., 2005). Research has also shown that personality traits (e.g. sensitivity to reward) can influence body weight indirectly via preferences for food high in fat and sugar (Davis, Patte, Levitan et al., 2007). Data were also obtained on ‘participant-identified binges’ in order to assess this relationship for binger and non-bingers following the criteria laid down by previous research in this area (Raymond, Neumeyer, Warren, Lee, & Peterson, 2003).

The study’s second research question concerned the role of self-reported emotional eater status as a moderator of food intake when in emotional states, previously investigated both in the laboratory (Evers et al., 2009) and in the snack diary above (Adriaanse et al., 2010). While this assumption has been tested in healthy weight, female samples, both of these studies have called for the investigation of this research question in an obese population, where the effects of emotional eating may be more pronounced. Within the time frame of the diary adopted here (five days), it is likely that people would experience both good and bad days, given that for example, previous research has shown people experience up to five hassles a day (Newman et al., 2007). To assess this, following Adriaanse et al. (2010), a measure of emotional state was included in the diary. It was deemed important to address both positive and negative emotions since both can motivate eating (Arnow et al., 1995; Macht, 1999). Consequently, this study’s second research question was: in an obese population, do self-reported emotional eaters eat more when emotional?
5.2 Method

5.2.1 Participants & procedure

A convenience sample of 57 participants (20 male, 37 female), from 16 – 70 years of age were recruited from the records of a National Weight Management Clinic at a general hospital. As part of the induction process to this clinic, patients are required to complete a standard 10-day food diary recording their daily intake. Included in their induction pack was the five-day “reflective diary” devised for this study, which they had the option of completing in conjunction with days one to five of the compulsory standard diary. The standard food diary asked participants to record everything they ate and drank throughout the day and the time of consumption, while the reflective diary was to be completed at the end of each day. Both diaries were completed by hand. A total of 235 diaries were administered (batch 1 = 122; batch 2 = 113) of which 69 were returned, an overall response rate of 29.36%. Fifty-seven diaries were of sufficient quality for this study. Diaries were discarded from the analysis if participants did not complete a minimum of three of the five days (60%) of the diary, or if incorrect response categories were used when answering the questionnaires (e.g. yes / no instead of the specified Likert scale). All participants provided written informed consent on the first page of the reflective diary and then completed basic demographic information.

Inclusion criteria were: medical referral for attendance at the weight management clinic; ability to give informed consent; and a BMI >30. Exclusion criteria were failure to complete both diaries (the standard and reflective diary) and the compulsory education period (minimum 8 years of formal education). The study received ethical approval from both the university and hospital ethics committees (see Appendix 2 for ethical approval documentation).

5.2.2 Measures

5.2.2.1 Diary development

Reflective diary: The reflective diary (see Appendix 7 for diary protocol) ran from Friday – Tuesday (5 days inclusive) including a weekend. A five-day food diary was adopted since assessing food intake at a given moment in time is not considered a valid method of dietary assessment, with a minimum window of 24 hours necessitated (Stubbs, Johnstone, O'Reilly, & Poppitt, 1998). As part of the instructions for the reflective diary participants were
informed of the importance of accurate information in research and asked to be as honest as possible in completing their diary. They were asked to complete the diary every night before going to bed, and to record the date and time of their entry. They were encouraged not to miss a day, but advised that if they did miss a day to leave that day blank. The diary was developed using: standardised psychometric measures; the assessment criteria for binge eating; and both Likert scale and open-ended questions.

Psychometric measures: Standardised measures included in the diary were the international Positive and Negative Affect Schedule (PANAS) Short Form (I-PANAS-SF) as a daily measure of mood (Thompson, 2007) and a shortened version of the DEBQ (Van Strien, Frijters, Bergers et al., 1986) as a daily measure of eating style. The 10-item I-PANAS-SF has two subscales, Positive Affect (PA) and Negative Affect (NA). Accordingly, participants were asked to rate the extent to which they experienced various positive (e.g. alert, inspired, attentive) and negative (e.g. upset, hostile, ashamed) emotions that day, using the response categories 1 (very slightly or not at all) – 5 (extremely), with higher scores indicative of higher PA or NA respectively. The I-PANAS-SF subscales have been found to have good internal reliability, temporal stability, cross-cultural factorial invariance, and convergent and criterion-related validity. Correlations between the short and full form subscales (Watson, Clark, & Tellegen, 1988) were found to be .92 (p < .01) for PA and .95 (p < .01) for NA (Thompson, 2007). Cronbach's alpha for the PA (.79) and NA (.65) subscales in the current sample showed adequate internal consistency. The 33-item DEBQ (as described in Chapter 2) has three subscales measuring restraint, emotional and external eating. A shortened version of the scale was used here incorporating: items 2, 3, 4, 7, 8 from the restraint eating subscale; 11, 13, 14, 18, 19, 23 from the emotional eating subscale; and 37, 39, 40, 42 and 43 from the external eating subscale. However, the items were re-worded so that they reflected past eating behaviour. For example: From the restraint scale, "Do you try to eat less at mealtimes than you would like to eat" was changed to "Did you try to eat less at mealtimes that you would like to eat"; from the emotional eating scale, "Do you have a desire to eat if you are depressed or discouraged" was changed to, "Did you have a desire to eat if you were depressed or discouraged"; and from the external eating scale, "If you see others eating, do you also have a desire to eat" was changed to “If you saw others eating did you also have a desire to eat”. Response categories ranged from 1 (never) – 5 (very often). For emotional eating items only, there was also an additional 'not relevant' response category, if that particular emotion was not experienced that day. Higher scores reflect higher levels of that given eating style. The
DEBQ subscales have good internal reliability and good concurrent, construct and predictive validity (Van Strien, 2005). Cronbach's alpha for the restraint (.88), emotional (.82), and external eating (.83) subscales indicated good internal consistency in the current sample.

Binge eating: Following previous research (Raymond et al., 2003), data were obtained on 'participant-identified binges' defined as eating episodes where the participant reported that they overate and experienced a loss of control regardless of the size (kilocalories) of the binge. This categorisation relates to that of a subjective binge episode as identified by Fairburn (2008), defined as eating episodes interpreted by individuals as involving more food than they should have eaten, accompanied by a sense of a lack of control when eating. Accordingly, participants were asked the questions: "Do you think you overate today in any single sitting?" and, "If yes, did you experience a loss of control during this episode?"

Likert scale questions: A scale from 1 – 5 (not at all; very little; a little; a lot; completely) was used to assess the participant's regulation of their eating behaviour and any disruption to it. Accordingly participants were asked “Using the scale above please indicate how much you achieved the following today: 1) “Eating an appropriate amount of food?” 2) “A balance across the types of food (e.g. starches, proteins, fats) you ate?” 3) “Taking an appropriate level of physical activity” 4) “To what extent did any significant events today affect or disrupt your eating habits?” 5) “How satisfied are you with your eating behaviour today?” Open-ended questions: Participants were asked: 1) To explain any significant events that affected or disrupted their eating habits; and, 2) reflecting on their eating behaviour today is there anything they would change about it?

Standard diary: Detailed instructions for completion of the food diary were set out on the cover page. Participants were reminded to include: between-meal snacks; food eaten whilst cooking; all drinks including water; take-away meals; and sugar added to drinks or meals. When describing food participants were asked for as much detail as possible including: the type of food (e.g. frozen, fresh, tinned); the method of cooking (e.g. grilled, fried, boiled, baked, raw); and brand names wherever possible. As regards the amount of food consumed, participants were asked to: record this in household measures (e.g. spoonfuls for solid quantities; cups or glasses for fluids) or packet sizes; compare amounts to the size of everyday standard objects (e.g. a portion of cheddar cheese the size of a matchbox); detail the number of food items (e.g. for slices of bread, biscuits, eggs); and for ready-prepared or packaged
foods to record the weight as stated on the package (e.g. fruit yoghurt 125g tub). The diaries were laid out in a table format with columns for: time; description of food / drink; and amount consumed. From this information the dietary team at the weight management clinic conducted a food pyramid analysis of intake, detailing the number of portions of carbohydrates, fruit and vegetables, dairy, protein, and top-shelf foods consumed daily. Portion sizes were based on the Irish Government's National Department of Health and Children guidelines (Harrington et al., 2008) and are summarised in Appendix 6.

5.2.2.2 Anthropometric measurements

BMI (weight in kilograms/height in meters squared) measurements were taken from the official records of the Weight Management Clinic, and calculated to the nearest 0.1Kg and 0.01m.

5.2.3 Data analysis

Norm comparisons of the theories of overeating and significant intercorrelations between the variables under study were examined. Standard multiple regression (with all variables entered simultaneously) was used to examine the first research question, that is, the relationships between self-reported eating style and both overall intake and intake by food type. In terms of the number of portions consumed from each food type, participants were removed from the analysis if they were greater than three standard deviations away from the mean in either direction (n = 2 participants removed for top-shelf foods). Standard regression was also used to assess the relationship between mood and self-reported eating style. Hierarchical regression (with variables entered in a predetermined stepwise format) was used to examine the second research question, that is, the relationship between emotional eating and intake when experiencing emotion (both PA and NA). Regarding the perceived regulation of eating behaviour (e.g. amount of food consumed, balance across food groups, satisfaction with eating behaviour) standard multiple regression was similarly used to investigate the relationship between self-reported eating style and the regulation of eating behaviour. A frequency analysis of responses was conducted on the types of significant events affecting eating behaviour and attributions for change in eating behaviour. Separate multiple regression analyses were conducted examining the relationship between eating style and intake (overall and by food type) for participant-identified bingers and non-bingers. T-tests
were used to compare the differences in portions consumed pre and post participant-identified binging episodes.

5.2.3.1 Missing data

Psychometric measures: For the I-PANAS-SF subscales were prorated where appropriate. For the DEBQ missing data in each subscale were replaced with the average of items that individual answered. For missing data on open-ended questions, participants were not included in those analyses.

5.3 Results

5.3.1 Sample characteristics

The clinical demographic profile is summarised in Table 5-1. There were no significant differences between males and females on age, education or BMI. Participant BMIs ranged from 33.47 – 75.85; 87.7% with class III obesity and 3.5% with class II obesity (WHO, 2000). BMI data was only available on 52 participants, as the remaining five participants (8.8%) did not present for treatment at the weight management clinic.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Male n (%)</th>
<th>Female n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 20 (35.1%)</td>
<td>n = 37 (64.9%)</td>
</tr>
<tr>
<td>M (SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (Years)</td>
<td>43.85 (14.53)</td>
<td>43.76 (12.68)</td>
</tr>
<tr>
<td>Education (Years)</td>
<td>13.65 (2.83)</td>
<td>13.24 (3.18)</td>
</tr>
<tr>
<td>BMI (Kg / m²)*</td>
<td>52.88 (8.88)</td>
<td>51.30 (8.63)</td>
</tr>
</tbody>
</table>

BMI = Body Mass Index, *based on n=52 participants.

5.3.2 Theories of overeating and variables under study

Average sample means for males and females on the restraint, emotional and external eating scales from the DEBQ are presented in Table 5-2, as well as their classification into the seven class norm structure (very high, high, above mean, mean, below mean, low, very low).
Table 5-2. Sample means on Restraint, Emotional, and External Eating DEBQ subscales and classification as per scale norms.

<table>
<thead>
<tr>
<th></th>
<th>Restraint M (SD)</th>
<th>Emotion M (SD)</th>
<th>External M (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obese norm</td>
<td>Above Mean</td>
<td>High</td>
<td>Mean</td>
</tr>
<tr>
<td></td>
<td>2.78 (1.03)</td>
<td>2.60 (0.83)</td>
<td>2.62 (0.81)</td>
</tr>
<tr>
<td>Females</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obese norm</td>
<td>Below Mean</td>
<td>High</td>
<td>Mean</td>
</tr>
<tr>
<td></td>
<td>2.62 (0.99)</td>
<td>2.97 (0.85)</td>
<td>2.92 (0.77)</td>
</tr>
</tbody>
</table>

Table 5-3 shows the intercorrelations of the variables under study. Apart from BMI, the average of participant entries (based on a minimum of three days and maximum of five days depending on diary completion) were reported.

Table 5-3. Intercorrelations between variables under study.

<table>
<thead>
<tr>
<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
<th>(4)</th>
<th>(5)</th>
<th>(6)</th>
<th>(7)</th>
<th>(8)</th>
<th>(9)</th>
<th>(10)</th>
<th>(11)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint (1)</td>
<td>x</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotion (2)</td>
<td>-.05</td>
<td>x</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>External (3)</td>
<td>.12</td>
<td>.43**</td>
<td>x</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI (4)</td>
<td>-.16</td>
<td>.08</td>
<td>-.10</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NA (5)</td>
<td>.05</td>
<td>.34*</td>
<td>.11</td>
<td>.33*</td>
<td>x</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PA (6)</td>
<td>.29*</td>
<td>-.13</td>
<td>.00</td>
<td>-.30*</td>
<td>-.32*</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amount eaten (7)</td>
<td>.66**</td>
<td>-.06</td>
<td>-.08</td>
<td>-.23</td>
<td>-.21</td>
<td>.35**</td>
<td>x</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Balance - food groups (8)</td>
<td>.46**</td>
<td>-.13</td>
<td>.00</td>
<td>-.33*</td>
<td>-.13</td>
<td>.15</td>
<td>.68**</td>
<td>x</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical activity (9)</td>
<td>.16</td>
<td>.03</td>
<td>-.06</td>
<td>-.17</td>
<td>-.14</td>
<td>.34*</td>
<td>.31*</td>
<td>.40**</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Significant events (10)</td>
<td>-.15</td>
<td>.13</td>
<td>-.06</td>
<td>-.17</td>
<td>-.05</td>
<td>.02</td>
<td>.01</td>
<td>.07</td>
<td>.19</td>
<td>X</td>
</tr>
<tr>
<td>Eating behaviour (11)</td>
<td>.50**</td>
<td>-.11</td>
<td>-.07</td>
<td>-.32*</td>
<td>-.34*</td>
<td>.29*</td>
<td>.76**</td>
<td>.55**</td>
<td>.29*</td>
<td>.01</td>
</tr>
</tbody>
</table>

**p<.01; *p<.05

With regards to the theories of overeating, significant positive correlations were found between restraint eating and PA, eating an appropriate amount of food, achieving balance across the food groups and overall satisfaction with eating behaviour. By contrast, emotional eating was significantly positively associated with NA and also with external eating. In terms of BMI, significant positive associations were found with NA, and similarly negative associations with PA, balance across food groups and overall satisfaction with eating
behaviour. The relationship between mood and the regulation of eating behaviour was evident in the significant positive correlations between PA and amount eaten, physical activity and satisfaction with eating behaviour and conversely the negative correlation between NA and the latter. Finally, physical activity was significantly positively associated with overall satisfaction with eating behaviour.

5.3.3 Eating style and overall intake

Multiple regression was performed to examine the influence of average self-reported restraint, emotional and external eating on actual food intake over the five day period. Table 5-4 shows that restraint eating was a significant (negative) predictor of overall intake, accounting for 13% of the total variance in intake. External (accounting for 5% of variance in intake) and emotional eating were not significant predictors. Examining males (n = 17) and females (n = 31) separately, self-reported eating style was not significantly related to intake in males, while restraint (B = -.54; p=.002) and external eating (B = .39; p=.036) were significant for females.

Table 5-4. Hierarchical multiple regression analyses for theories and overall food intake.

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>SE B</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>18.81</td>
<td>4.33</td>
<td>-</td>
</tr>
<tr>
<td>Restraint</td>
<td>-2.48</td>
<td>0.89</td>
<td>-.37*</td>
</tr>
<tr>
<td>Emotion</td>
<td>1.05</td>
<td>1.13</td>
<td>.14</td>
</tr>
<tr>
<td>External</td>
<td>2.29</td>
<td>1.31</td>
<td>.26</td>
</tr>
</tbody>
</table>

Note: R² = .23; *p<.01. n = 49.

When examining each day individually, the above pattern held for Saturday (p<.01) and Monday (p<.05) accounting for 21% and 10% of variance respectively. On Friday, external eating (p<.01) was significant explaining 18% of the variance in total intake, and restraint eating was marginally non-significant (p=.05) accounting for 6% of the variance. None of the theories were significant predictors of intake on Sunday, while on Tuesday Emotion was significant (p<.05) accounting for 10% of the variance in intake.

5.3.4 Eating style and food type

Five multiple regression analyses were conducted to examine the relationships between the three eating styles (self-reported restraint, emotional and external eating) and intake as per the food pyramid analysis of intake (carbohydrates, fruit and vegetables, dairy, protein and top-shelf foods). Significant relationships were found for top-shelf foods only with
restraint eating and external eating as significant predictors, as detailed in Table 5-5. Examining the relationship between self-reported eating style and top-shelf foods for males (n = 17) and females (n = 31) separately, self-reported eating style was not significantly related to intake in males, while restraint (B = -.43; p=.017) and external eating (B = .40; p=.04) were significant for females.

Table 5-5. Hierarchical multiple regression analyses for top-shelf foods.

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>SE B</th>
<th>(\beta)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>5.14</td>
<td>2.29</td>
<td></td>
</tr>
<tr>
<td>Restraint</td>
<td>-1.22</td>
<td>0.48</td>
<td>-.35*</td>
</tr>
<tr>
<td>Emotion</td>
<td>0.29</td>
<td>0.61</td>
<td>.07</td>
</tr>
<tr>
<td>External</td>
<td>1.44</td>
<td>0.70</td>
<td>.31*</td>
</tr>
</tbody>
</table>

Note: \(R^2 = .21; *p<.05. n = 47.\)

5.3.5 Mood and self-reported eating style

In terms of mood, aggregate scores for the five days revealed that higher NA (M = 8.62, SD = 2.98) was positively associated with self-reported emotional eating, while PA (M = 11.39, SD = 3.98) was positively associated with restraint eating as shown in Table 5-6. Mood was not associated with self-reported external eating.

Table 5-6. Hierarchical multiple regression analyses for emotional and restraint eating.

<table>
<thead>
<tr>
<th></th>
<th>Emotion</th>
<th>Restraint</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>SE B</td>
</tr>
<tr>
<td>Constant</td>
<td>2.00</td>
<td>0.62</td>
</tr>
<tr>
<td>NA</td>
<td>0.10</td>
<td>0.04</td>
</tr>
<tr>
<td>PA</td>
<td>-0.01</td>
<td>0.03</td>
</tr>
</tbody>
</table>

Note: \(R^2 = .12; *p<.05. n = 46.\) Note: \(R^2 = .33; *p<.05. n = 53.\)

5.3.6 Eating style and intake when emotional

Using aggregate scores for the five-day period, hierarchical regression analyses were performed to test whether self-reported emotional eaters showed greater increases in food intake when experiencing emotion than self-reported non-emotional eaters. Food intake was regressed onto: emotional experience (NA and PA) (step 1); self-reported emotional eating as per the emotional eater subscale of the DEBQ (step 2); and the interaction term between emotional experience and the emotional eater scale (step 3) (see Table 5-7 and Table 5-8). The interaction term for both PA and NA was non-significant indicating emotional eater status
had no effect on food intake when emotional. This result did not change when males and females were examined separately.

Table 5-7. Hierarchical multiple regression analyses for NA and overall food intake.

<table>
<thead>
<tr>
<th>Step 1</th>
<th>B</th>
<th>SE B</th>
<th>B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>20.78</td>
<td>3.21</td>
<td>0.11</td>
</tr>
<tr>
<td>NA</td>
<td>0.11</td>
<td>0.34</td>
<td>0.05</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Step 2</th>
<th>B</th>
<th>SE B</th>
<th>B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>16.11</td>
<td>3.82</td>
<td>-0.14</td>
</tr>
<tr>
<td>NA</td>
<td>-0.14</td>
<td>0.35</td>
<td>-0.06</td>
</tr>
<tr>
<td>Emotion</td>
<td>2.46</td>
<td>1.18</td>
<td>0.08</td>
</tr>
</tbody>
</table>

Step 3

| Constant        | 16.15  | 3.89  | -0.15  | 0.35  | -0.06  |
| NA              | -0.15  | 0.35  | -0.06  |
| Emotion         | 2.45   | 1.2   | 0.08   | 0.91  | .32*   |
| Emotion x NA   | 0.08   | 0.91  | 0.01   |

Note: $R^2 = .00$ for Step 1; $\Delta R^2 = .09$* for Step 2. $\Delta R^2 = .00$ for Step 3. *p<.05. n = 47.

Table 5-8. Hierarchical multiple regression analyses for PA and overall food intake.

<table>
<thead>
<tr>
<th>Step 1</th>
<th>B</th>
<th>SE B</th>
<th>B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>22.94</td>
<td>2.89</td>
<td>-0.12</td>
</tr>
<tr>
<td>PA</td>
<td>-0.12</td>
<td>0.24</td>
<td>-0.07</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Step 2</th>
<th>B</th>
<th>SE B</th>
<th>B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>15.87</td>
<td>4.46</td>
<td>-0.05</td>
</tr>
<tr>
<td>PA</td>
<td>-0.05</td>
<td>0.24</td>
<td>-0.03</td>
</tr>
<tr>
<td>Emotion</td>
<td>2.27</td>
<td>1.12</td>
<td>0.04</td>
</tr>
</tbody>
</table>

Step 3

| Constant        | 15.85  | 4.53  | -0.04  | 0.24  | -.03   |
| PA              | -0.04  | 0.24  | -.03   |
| Emotion         | 2.28   | 1.13  | .30    |
| Emotion x PA   | 0.04   | 0.89  | .01    |

Note: $R^2 = .00$ for Step 1; $\Delta R^2 = .09$* for Step 2. $\Delta R^2 = .00$ for Step 3. *p<.05. n = 47.

Separate regression analyses controlling for external and restraint eating in step 2 of the equation revealed restraint as a significant covariate in step 2 (p<.01) and step 3 (p<.05) for NA, and similarly for step 2 (p<.01) and step 3 (p<.01) for PA, with emotional eating no longer significant. However, there was no overall influence on the interaction between emotional experience and emotional eater status. While experiencing both positive and negative emotion, self reported emotional eater status had no effect on actual intake. This result held when the regression equation was repeated for the different food types (carbohydrates, fruit and vegetables, dairy, protein and top-shelf foods) separately.
5.3.7 Regulation of intake

Consistent with the findings in respect of overall intake and top-shelf foods, standard multiple regression revealed restraint was the dominant factor with regards to the perceived regulation of food intake (as per the Likert scale questions), significantly predicting satisfaction with the amount of food consumed ($p<.001$), balance obtained across food groups ($p<.001$) and overall satisfaction with eating behaviour ($p<.001$). Major events were not found to impact significantly upon actual food intake. A frequency analysis of events reported to affect eating behaviour included: social occasions (27%; e.g. eating out, having guests or visiting others), work-related factors (19%; e.g. stress, use of convenience foods due to lack of time), illness (12%; e.g. pain, leg infections), stressful events (11%; e.g. bereavement, divorce, sick relative), low mood (11%), and having free time / being idle (7%). With regard to attributions for retrospective changes in eating behaviour, a frequency analysis of responses included: changing the types of food / drink consumed (17%), opting for balanced healthier meals (15%), cutting out unhealthy snacks (14%), smaller portions (13%), not overeating (9%), more activity (9%), better planning / scheduling of meals (7%) and controlling when and how much (7%).

5.3.8 Self-reported bingers and non-bingers

Of the fifty-seven participants 34 (60%) identified themselves as having a binge episode (overeating and experiencing a loss of control) and 23 (40%) as having no binge episode over the 5 day period. Multiple regression analyses for each group separately revealed no significant relationships between the three self-reported eating styles and overall intake. The Chow test using an online calculator (http://faculty.chass.ncsu.edu/garson/PA765 regress.htm#compare retrieved on 25/06/2010) was used to compare the regression results for bingers and non-bingers. Accordingly, the original independents were entered in block one, with the interaction term between the grouping variable and each independent variable in block two. No significant differences were found between the two groups. Similarly, no significant differences between the groups were found when comparing the Spearman correlation coefficients for restraint ($p = .9$), emotional ($p = .49$), and external eating ($p = .85$) and average total intake.

5.3.8.1 Portions during participant-identified binge episodes

An analysis of the mean number of overall portions consumed by bingers ($n = 34$) the day preceding ($M = 22.31, SD = 7.07$), of ($M = 22.96, SD = 5.34$) and after ($M = 20.07, SD = 8.25$)
a participant-identified binge, showed a significant decrease in intake the day after a participant-identified binge ($t (33) = 1.76, p<.05$). The increase in intake the day prior to, and day of, a participant-identified binge was non-significant.

5.4 Discussion

5.4.1 Eating style and overall food intake

All three eating styles were well-represented in the sample, with both male and female participants classified as high, above the mean, or at the mean in comparison to the obese norms for the DEBQ (Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986). The only exception to this was on the restraint subscale, where females fell below the obese norm. Despite this, using aggregate scores over the five-day period, the self-reported eating style of cognitive restraint was the strongest predictor of lower overall energy intake in the sample. By contrast, emotional eating was not associated with overall intake and external eating was predictive of higher intake in females only. When the relationship between self-reported eating style and intake was analysed by day, this pattern held for two out of the five days. These findings reflect previous research in this area using the food diary and FFQ methodologies in healthy weight populations in which restraint was strongly associated with lower overall energy intake in both adults (Anschutz et al., 2009; Lluch et al., 2000) and adolescents (De Lauzon et al., 2004; Wardle et al., 1992), external eating with higher intake (Anschutz et al., 2009; Wardle et al., 1992) and emotional eating was unrelated to intake. The lack of significance between eating style and intake when looking at male participants only is likely attributable to lower statistical power.

5.4.2 Eating style and food type

When looking at the relationship between self-reported eating style and food type, in terms of the consumption of carbohydrates, fruit and vegetables, dairy, protein and top-shelf foods, significant findings were found in the case of top-shelf foods only. Consistent with the results for overall intake, cognitive restraint was again the dominant factor significantly associated with lower intake of top-shelf foods, while external eating was significantly positively associated with such intake. Affective influences, as measured by emotional eating were again unrelated to intake. When looking at the gender groups separately this pattern held for females but not for males where as mentioned previously statistical power was likely an issue. These findings as a whole once more replicate previous research investigating the
relationship between eating style and food type in healthy weight populations where self-reported restraint eating has been negatively associated with snack (Adriaanse et al., 2010), fat and carbohydrate intake (Anschutz et al., 2009), French fries, sugar and confectionery (De Lauzon et al., 2004) and with decreases in fat and saturated fat intake over a four-year period (Van Strien & Van de Laar, 2008). Similarly, external eating has been positively related to snack (Newman et al., 2007), fat and carbohydrate intake (Anschutz et al., 2009) and the consumption of energy-dense foods (De Lauzon et al., 2004). Finally, the fact that emotional eating was unrelated to intake in terms of food type (inclusive of top-shelf foods which are akin to snack foods) is consistent with findings that emotional eater scales failed to predict snack (Adriaanse et al., 2010), fat or carbohydrate intake (Anschutz et al., 2009) in healthy participants, but inconsistent with population level research where emotional eating was related to higher snack intake (De Lauzon et al., 2004).

5.4.3 Emotional eater status and intake when emotional

The study’s second research question concerned whether, in an obese population, self-reported emotional eaters eat more when emotional? The results showed that for both positive and negative affective states, self-reported emotional eater status had no impact on overall energy intake, or on the consumption of specific food types (carbohydrates, fruit and vegetables, dairy, protein, and top-shelf foods). This finding is in line with previous work in healthy weight populations, in which emotional eater status did not predict caloric intake in response to negative emotion in a snack diary (Adriaanse et al., 2010) and both negative or positive emotion in lab-based experiments (Evers et al., 2009). It also corroborates those studies in which emotional eating did not moderate snack intake under stress (Conner et al., 1999; O'Connor & O'Connor, 2004). As such, these results lend support to the idea that people (both of healthy and unhealthy weight) lack the abilities to assess the extent to which they eat in reply to emotions, and that as a result, emotional eating scales are poor predictors of actual food intake (Adriaanse et al., 2010; Evers et al., 2009). Given the wide range in scores on the emotional eater scales over the five days (1.04 – 4.49) and wide range in average food intake (9.20 – 40.10) it is unlikely that a restriction in range constrained the ability to detect effects.

The fact that emotional eater scales seem to be poor predictors of actual food intake begs questions both of the assessment instruments and of the very construct of emotional eating. Regarding the assessment instrument, what is it that emotional eater scales measure?
Considering the DEBQ (Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986) used here as a measure of eating style, it is clear that the cognitive restraint subscale measures frequency of restraint (e.g. How often did you try not to eat between meals because you were watching your weight?), while the emotional eating subscale measures desire to eat (e.g. Did you get the desire to eat if you were anxious, worried, or tense?). As desire to eat does not necessarily equate with actual eating behaviour, the predictive validity of emotional eating scales, including both the DEBQ here and also the EES (Arnow et al., 1995), could benefit from measuring the frequency of eating in response to emotions (Masheb & Grilo, 2006) rather than only the desire to eat.

A second noteworthy issue regarding the assessment of emotional eating is the fact the emotional eating subscale here was significantly correlated with the external eating subscale of the DEBQ, a finding that has been frequently articulated in the literature (e.g. Rodin, 1981; Van Strien, Herman, & Verheijden, 2009; Van Strien et al., 1995). In addition, the research discussed earlier generally showed effects for external but not emotional eating and rarely both together. The notion that emotional eaters increase food intake when emotional has often been based on the fact that these individuals fail in attempts at weight control (Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986). However, as suggested by Evers et al. (2009) such positive associations between weight and emotional eater status may more parsimoniously reflect overeating in response to all cues, an interpretation supported by the observed correlations between emotional and external eater scales. The implication here that emotion and external eater subscales may not be theoretically different aspects of overeating has been defended by Van Strien et al. (1995). They found that emotional eating was significantly related to problems with emotional distress and relationships (anxiety, depression, phobias, suicidal acts or ideations, intimate relations and sexual contacts) but external eating was not. As such, they suggested that the two types of eating behaviour refer to independent constructs and thus the use of separate scales is warranted. However, while this shows that individuals scoring high on emotional eater scales clearly experience high levels of emotional distress, it does not necessarily follow that they will respond to this distress by (over)eating, as has been demonstrated both here and in previous research (Adriaanse et al., 2010; Evers et al., 2009). This leads us to question the construct of emotional eating itself. Why is it that self-reported emotional eaters do not increase food intake when emotional? And if emotional eating does not relate to increased food intake when emotional, what does the construct actually assess?
In addressing the first of these points, why self-reported emotional eating does not relate to increased food intake when emotional, the accuracy of people's understanding of their eating behaviour, and the role of emotion in driving that behaviour, is called into question. As highlighted by Evers et al. (2009), retrospective emotional ratings are highly sensitive for recall bias and are at risk for both under- or over-estimation of emotions (Ready, Weinberger, & Jones, 2007). Since both male and female obese participants in our sample were classified as high on emotional eating, as compared to the obese norm for the DEBQ (Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986), it is possible that they overestimated the influence of emotion on their eating behaviour; given emotional eater status was not associated with food intake. In addition to a bias for retrospective emotional ratings, people also have a tendency to under-report caloric intake (Stice, Fisher, & Lowe, 2004). As such, it has been argued that emotional eater scales potentially involve a 'triple recall bias' requiring individuals to recall their negative emotions, their food intake and the association between the two (Evers et al., 2009). With this in mind, perhaps it is unrealistic to expect healthy and obese individuals, to be accurate assessors of their emotional eating behaviour.

A related issue affecting people's ability to assess their emotional eating is the consideration of the impact of hot states on behaviour. When people are in hot states (e.g. emotional) they appreciate the influence of past or future hot states, while people in cold or neutral states chronically underestimate the impact of hot states (Van Boven & Loewenstein, 2003). Thus, in terms of assessing one's emotional eater status, this status may vary according to visceral state (Evers et al., 2009). For example, Bekker, Van de Meerendonk, and Mollerus (2004) showed that inducing negative affect substantially increases the level of self-reported emotional eating. Similarly, in this study, negative affect was significantly positively correlated with emotional eater status and positive affect with restraint. As such, if in a negative (hot) state when assessing their eating behaviour for the day, obese participants could have overestimated the influence of emotion on their food intake.

This brings us to our second question: if emotional eating does not relate to increased food intake when emotional, what does the construct actually assess? The positive association between emotional eater status and negative affect found here, ties in with an alternative interpretation of emotional eating proposed by Adriaanse et al. (2010). They examined the hypothesis that self-reported emotional eating (using the DEBQ; Van Strien, 2005; Van Strien,
Frijters, Bergers et al., 1986) is an expression of personal beliefs about the association between emotions and eating resulting from concerns about one’s eating behaviour. Accordingly they found that emotional eating was a significant predictor of increased worrying about one’s eating behaviour, a higher level of monitoring and lower perceived control over eating behaviour, and a stronger extrinsic motivation for healthy eating behaviour after controlling for BMI, self-regulatory skills and past snacking behaviour. Restraint was also a significant predictor of eating concerns, which corresponds with previous literature on restraint eating expressing a problematic attitude toward food intake. As such, they suggest that this association between emotional eating and eating concerns could point to a general preoccupation with eating and food in emotional eaters, which, in turn, might result in an inflated notion and subjective interpretation of the frequency of eating (or desire to eat) in response to emotion (Adriaanse et al., 2010). In other words, self-reported emotional eating may reflect an emotional attitude to eating or being emotional about eating.

This argument is in line with previous studies which have shown an association between feelings of guilt and the amount of food eaten (Rozin, Kabnick, Pete, Fischler, & Shields, 2003). In addition, research has shown adolescent emotional eaters felt fatter and often reported feeling upset about eating unhealthy foods, even though there was no association between their emotional eater scores and BMI (Wardle et al., 1992). In providing a broader framework for this argument, Evers et al. (2009) draw on Robinson and Clore’s emotional accessibility model (2002). The model argues that when people report emotions they are not currently experiencing, they shift to a semantic retrieval strategy whereby people access their generalised beliefs about their emotions rather than experiential emotion knowledge, particularly when reporting on emotional traits as in emotional eater scales. As such, people may have shaped beliefs about themselves that become dissociated from their actual behaviour in daily life (Evers et al., 2009).

A caveat on the lack of association between emotional eating and intake in our sample must be put forward at this point. The nature of assessment here was very much a general framework; addressing the relationship between an individual’s overall daily mood rating and their assessment of their daily emotional eating behaviour, over a five day period. However, it is possible that emotional eating occurs in a more episodic way, such that the individual may eat more when emotional, but then compensate for that intake by eating less the rest of the day, potentially obscuring the relationship between self-reported emotional eating and intake.
This sentiment was also expressed by Anschutz et al. (2009) who measured energy intake over a one-month period, but without assessing emotional states or life events. They too found emotional eating was not related to total energy intake, fat intake, or carbohydrate intake. However, they similarly suggested, that emotional eaters may only eat more if they are emotionally aroused, which might be observed shortly after the arousal. Consequently, an avenue for future research would be to assess this relationship in a more episodic way, examining the relationship between emotional eater status and the immediate emotional antecedents / life events to eating, perhaps addressing both overall intake as well as food type. This would also fit with Evers et al.’s (2009) proposal that it remains to be assessed if people are more accurate predictors of their own emotional eater behaviour when they fill out the scales while being emotional.

5.4.4 The dominance of restraint

Despite females scoring below the obese norm for cognitive restraint on the DEBQ, (Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986), restraint was the strongest predictor of lower overall intake and of top-shelf foods. However, unlike external eating which has been associated with higher intake, but lower BMI in healthy weight individuals (Snoek et al., 2007; Wardle et al., 1992), restrictive eating practices are consistently associated with higher BMI in the literature (French et al., 1994; Klesges et al., 1992; Lluch et al., 2000; Van Strien, Frijters, Van Staveren et al., 1986). This finding is hard to reconcile with the idea that restraint eating was associated with lower intake in our sample and in previous research of healthy populations. However, this discrepancy is perhaps explained in the concept of restraint itself, which does not necessarily involve successful dieting (Lluch et al., 2000). Accordingly, restraint theory argues that the restriction of food intake (e.g. through skipping meals) leads to irregular eating patterns and to counter-regulatory eating (see section 1.4.3. for more detail) at moments of disinhibition, thus bingeing, and eventually higher weight (Herman & Polivy, 1980). The use of such restrictive eating practices in our sample is supported by the fact that participant-identified bingers showed a significant reduction in overall intake the day after a binge episode. The possible rewarding nature of such restrictive practices was highlighted by the association between restraint and PA, and perceived regulation of eating behaviour in terms of eating an appropriate amount of food, achieving a balance across the food groups and overall satisfaction with eating behaviour.
While restraint showed the most robust associations with overall intake over the five-day period, it is interesting that this pattern varied by day, with external eating significant on Friday, none of theories playing a role on Sunday and emotion significant on Tuesday. These findings can perhaps be explained with reference to the open-ended question used in the study on significant events that may have disrupted one's eating behaviour. Within these events the majority of items (27%) concerned social occasions. Examples of such social occasions included eating out, having guests, visiting others. However, often these items focused on the significance of a particular day, for example “Sunday”, “Friday” or “treat night”, indicating a potential rationale used by people for lowering restraint or taking the brakes off the train (Swinburn & Egger, 2004). With regards to why emotional eating played a significant role on Tuesday, this could perhaps be attributable to work-related factors coming back into play, which were the second to social occasions in terms of their disruptive effect on eating behaviour (19%). These included quite negative, emotional items such as stress, intimidation, tiredness, or lack of time to eat properly. In addition, in a National Time Accounting (NTA) study using the U index as a single welfare measure of the percentage of moments spent in an unpleasant state (the proportion of time when rating of sad, stressed or pain exceeds happy), trends showed this index was higher Monday – Thursday, than at the weekend, although these differences were non-significant (Krueger, Kahneman, Schkade, Schwarz, & Stone, 2008). The U-index was lowest on Sundays and slightly higher on Mondays than on Tuesdays through Thursdays. Almost half of the weekend-weekday difference in the U index was accounted for by the by the different mix of activities that take place on the weekend.

5.4.5 Study limitations

The naturalistic approach to assessing decision making in an obese population, through use of the diary method to retain ecological validity brings costs. Although participants were asked to record intake throughout the day to minimise recall bias, there’s no guarantee that participants did not complete entries retrospectively on some occasions. Such retrospective methods can lead to a serious underreport of caloric intake (Stice et al., 2004). Estimates have shown that underreporting of caloric intake can be up to 27% of energy needs in men and 29% in women (Rennie, Coward, & Jebb, 2007). In addition, the underestimation of energy intake is thought to be greater in overweight and obese individuals than normal weight subjects (Lluch et al., 2000; Rennie et al., 2007). However, the alternatives to a food diary approach have also seen criticism, with FFQs seen to produce insufficient detail and to be overly reliant on memory (Stephen, 2007) and lab-based approaches assessing food intake
in a given moment, considered too short to be a valid method of dietary assessment (Stubbs et al., 1998). Indeed a recent review of methods of dietary assessment argued for the use of diet diaries, owing to their greater power to detect diet-disease relationships and in spite of the considerably higher costs in terms of coding time and expertise (Stephen, 2007).

The use of a paper diary could also be criticised on the grounds that compliance rates were found to be lower for paper rather than electronic diaries with compliance-enhancing features (Stone, Shiffman, Schwartz, Broderick, & Hufford, 2002). For example, in a study of chronic pain patients using paper diaries, reported compliance was 90%, but on 32% of days the paper diary binder was not opened, indicating a high level of faked compliance (Stone, Shiffman, Schwartz, Broderick, & Hufford, 2003). However, as implemented in this study, adequate communication with participants and a clear explanation of the utility or lack of utility of entries provided at the wrong time can dramatically reduce the risk of faked compliance (Green, Rafaeli, Bolger, Shrout, & Reis, 2006). The fact that 12 diaries (17%) were excluded from this study because participants left more than two of the five days blank, would indicate that people took this advice into consideration. Furthermore, the age and profile of our sample meant individuals were unfamiliar with computers or PDA devices, and so following recommendations in the literature the use of a paper-and-pencil method was seen as producing better data (Green et al., 2006).

Regarding the assessment of eating style, the study relied on the DEBQ only (Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986). While this scale is widely used, the inclusion of alternative measures of eating style such as the TFEQ (Stunkard & Messik, 1985) would have been desirable. In addition, by focusing specifically on the concepts of emotional, external and restraint eating through the use of closed response options derived from the DEBQ, the research primed these aspects of eating for participants and limited the study's ability to capture other potential influences on eating. The use of open-ended questions regarding eating style may be advantageous in future research, to investigate whether these widely cited constructs are naturally reported by participants. Indeed, while these self-reported eating styles explained up to 21% of the variance in intake in a given day, there was a considerable amount of unexplained variance in intake. The use of open-ended questions may capture other factors which affect intake, such as the role of habit strength, which has been found to be an important predictor of eating behaviour (Verplanken, 2006) and of snack intake (Adriaanse et al., 2010). In support, habitual disinhibition was found to be the strongest
correlate of adult weight gain in older women over a twenty-year period (Hays & Roberts, 2007). As the 'reflective diary' was completed retrospectively at the end of each day, recall bias may have affected the accuracy of people's assessments of their eating styles. This bias may have been particularly prevalent in the case of emotional eating, as discussed in section 5.4.3. As such, in addition to the use of open response options, future research may benefit from the assessment of eating style immediately after intake thus eliminating the potential for recall bias. Furthermore, research of this kind may capture the potential episodic relationship between emotion and eating as articulated previously.

The sample size in this study was moderate and low statistical power may account for the lack of significant results in male participants when looking at the gender groups separately. Participants were also a treatment seeking sample, limiting the generalisability of these results to the broader population of obese individuals. In addition, the degrees of freedom for some of the regressions analyses were smaller due to: selection of the 'not relevant' response category on the emotional eating items of the DEBQ on more than two of the five days (n = 7; 12%); failure to complete the PANAS (Thompson, 2007) on the minimum number of days leading to exclusion on analyses which required a mood rating; loss of data through incorrectly completing the questionnaires. With reference to the criteria for binge eating, data were only obtained on 'participant-identified binges' and not on objective binge episodes (Fairburn, 2008). As such, the finding that there were no significant differences between bingers and non-bingers in terms of eating style variables becomes less useful, as participants may have been 'binge' eaters using objective criteria, but either not reported it, or not had a binge episode during the five-day diet diary owed to potentially higher restrictive practices while completing the diary. More objective criteria include, for example: the consumption of >1000 Kcal in a single sitting and the experience of a loss of control (Fairburn, 2008) or eating episodes characterised by the consumption of a large amount of food in a discrete period of time and a feeling of being out of control while eating (APA, 2000). Finally, it should be acknowledged that the lack of support for emotional eating in this study, and external eating in males, could be related to a social desirability bias in our sample, whereby it could be socially desirable for obese individuals to endorse questions about being on a diet (restraint) and not to endorse those about emotional and external eating, due to both the stigma associated with being overweight in society (Van Strien & Ouwens, 2003) and the fact that this was a treatment-seeking obese population. However, the fact that females in this
study were below the obese norm for restraint eating (unlike the norms for external and emotional eating), would attenuate this concern.

5.4.6 Application of the findings

The findings here largely corroborate previous research in the food diary and FFQ domains in healthy populations, in an obese population, indicating analogous relationships between eating behaviour and intake in healthy and obese individuals. However, if the relationship between eating style and eating behaviour is similar in these populations, returning to our original question, why is it that in the same obesogenic environment, some individuals can maintain an appropriate weight while others can not? The answer may lie in the concept of restraint which was the strongest predictor of lower intake here and in previous research in healthy populations, yet evidently the morbidly obese population in the current study do not achieve successful restraint. Perhaps this indicates a failure in broader self-regulatory capacities, moderating the relationship between cognitive restraint and successful / unsuccessful dietary behaviours. This proposal ties in with the premises of TST (Hall & Fong, 2007), (mentioned in Chapter 1; section 1.5.6), a biopsychosocial model of human behaviour that considers temporal dynamics, behavioural prepotency, and most relevant here, the concept of a biologically based self-regulatory capacity (SRC). SRC refers to the capacity to exert top-down control over one’s actions, viewed as partially synonymous with executive function, and biologically based in the prefrontal cortex (Hall & Fong, 2010).

Individual differences in self-regulatory abilities – operationalised on tasks of executive function – have been found to predict intention-behaviour correspondence for both exercise behaviour and dietary choice; specifically those with stronger executive function show stronger intention-behaviour associations over time than their counterparts (Hall, Fong, Epp, & Elias, 2008). A similar process could exist for restraint and eating behaviour, whereby those with stronger executive function could show strong restraint – eating behaviour correspondence over time. This issue will be returned to in Chapter 6.

On a cautionary note, it must be acknowledged that the possibility that being obese leads to higher report of such restrictive practices cannot be ruled out. In addition, whether restrictive practices lead to long-term weight gain is an issue which remains to be resolved in the literature with some studies finding significant associations with weight gain (Schur, Heckbert, & Goldberg, 2010) and others no such associations in both younger (Delinsky & Wilson, 2008) and older women (Hays & Roberts, 2007). It is clear that more prospective
research is needed into eating styles, their corresponding intake patterns (measured by diet diaries), and BMI over time, to gain a better understanding and conceptualisation of normal and pathological eating behaviour and its relationship with weight regulation. This knowledge would inform the use of motivations to eat as potential targets for future interventions that promote healthy weight management (Hawks, Merrill, Gast, & Hawks, 2004) and would have the potential to contribute to both lifestyle (midstream) and health services (downstream) policy targets (Sacks et al., 2009) to control the obesity epidemic. With reference to midstream targets, such research could inform education and campaign-based programmes delivered in schools, workplaces and the community to promote healthier eating. Similarly with regards to downstream targets, such knowledge could provide new strategies for obesity prevention through health service delivery targeting children who are overweight or obese in an attempt to reduce the subsequent incidence of adult obesity and indeed through obesity management and the therapeutic treatment of existing obesity and its complications.

5.5 Conclusion

In a sample of morbidly obese individuals, cognitive restraint was the strongest predictor of lower intake for both overall intake and top-shelf foods. External eating was related to higher consumption of top-shelf foods in the sample as a whole and to higher overall intake in females only. With regards to affective influences on eating, self-reported emotional eating was unrelated to food intake. Similarly, self-reported emotional eater status did not moderate food intake in relation to positive and negative mood states. None of the theories played a significant role in the consumption of other food types, such as carbohydrates, protein, dairy and fruit and vegetable consumption. The results suggest that the relationships between self-reported eating style and intake are similar in healthy and unhealthy weight populations.
Chapter 6: General Discussion

6.1 Summary of empirical chapters

This thesis examined affective and cognitive influences on decision making in a morbidly obese population, to investigate factors within the individual that may have contributed to the rising obesity epidemic. In terms of the 'runaway weight gain train' (Swinburn & Egger, 2004), invoked as a model for understanding the relationship between the obesogenic environment, cycles or positive feedback loops which promote weight gain, and ineffectual brakes that fail to attenuate such factors, the thesis was seen to specifically address ineffectual cognitive and psychophysiological brakes. The focus on morbid obesity was timely, given the most rapid increases within the obesity epidemic have been seen in those considered extremely obese. The consequent high rate of disease comorbidity and reduced life expectancy renders this population a particularly vulnerable group, who place a high burden of care on public health systems. The focus on morbid obesity was also appropriate, given some of the methods used in this research were originally applied to understanding the decision making capacity of similarly extreme groups in addiction research. Finally, the focus on morbid obesity was deemed necessary in light of the relatively unsuccessful behavioural treatment of this group and the potential applicability of research in morbid obesity to overweight and moderately obese individuals.

Chapter 2 revealed impairments in decision making under uncertainty, as measured by the IGT, in a sample of morbidly obese individuals compared to a matched healthy weight comparison group. These deficits were independent of age, gender, education, eating pathology and severity of the condition as measured by BMI. It was suggested that the obese group's failure to learn on the task could be the expression of random choice behaviour, or could reflect a real inability to follow a specific strategy on the IGT with participants unable to maximise immediate reward or programme a delayed reward. In addition, the absence of a clear preference for either advantageous or disadvantageous decks was not indicative of impairments in impulse control in this group, further supported by the finding that a self-report measure of the weight given to future versus immediate consequences, the CFC, showed no relationship with IGT performance. These deficits were in line with previous investigations of disordered eating populations, indicating that the IGT is a useful tool in revealing decision making impairments in the inability to regulate food intake.
Extending on the parallels between decision making deficits in obesity and previous investigations of other disordered eating groups, Chapter 3 employed the IGT to demonstrate a shared profile of decision making deficits across AN, BN and obesity. The application of the task in this way was based on the premise that the pathological eating behaviour of the three groups can be conceptualised within a common framework; that is, the inability to forgo an immediate reward for a long-term better outcome. Accordingly, all three clinical groups were found to be significantly impaired on the IGT relative to the comparison group, but were not significantly different from each other, supporting a broad profile of shared decision making impairments among females in these disordered eating groups. As was the case in Chapter 2, the three clinical groups evidenced flat learning curves across the task. The learned helplessness model was proposed as a potential mechanism for this shared performance pattern, whereby an expectancy that the task was uncontrollable, with responding unrelated to reinforcement, together with an attribution of failure on the task to personal abilities, may have resulted in passive ‘giving up’ behaviour. Alternatively, common impairments in reversal learning, necessary for successful IGT performance, were proposed as an alternative mechanism underlying shared deficits. Indeed, it was highlighted how in these disordered eating groups, individuals must learn to reverse behaviours that may once have been highly rewarding.

Chapter 4 sought to further specify the decision making deficits in obesity revealed in Chapter 2, through the inclusion of psychophysiological measures and additional neuropsychological tasks. IGT deficits in obese participants, compared to a healthy and overweight comparison group, were replicated using the A'B'C'D' version of the task (Bechara, Tranel et al., 2000). However, intelligence was found to remove group differences in overall performance. Inconsistent with the SMH, affective anticipatory (and feedback) ‘somatic maker’ signals did not differentiate between the obese and comparison group. The SGT, which aimed to explore the relative contributions of EV and gain-loss frequency to decision making in obesity, revealed that both obese and comparison participants were significantly impaired on this task responding to immediate reinforcement, rather than to long-term EV. It was therefore concluded that both groups showed the normal tendency to underweight rare events and (or) to be loss averse. Consistent with results for the IGT, psychophysiological data did not discriminate between groups on the SGT. In assessing decision making under explicit conditions, obese participants were unimpaired on the GDT, and showed intact use of negative feedback after risky choices, with similarly no differences in psychophysiological
activation. Addressing the validity of the SMH, the ‘somatic maker’ profile of impaired vs. non-impaired participants on the IGT was examined. ‘Somatic marker’ activity failed to discriminate between these groups and was thus unsupportive of the SMH. However, the sample as a whole demonstrated higher anticipatory ‘somatic marker’ signals to the disadvantageous decks and higher autonomic activity after a win from a disadvantageous deck, leading to the conclusion that ‘somatic marker’ signals may only discriminate between good and bad decision making, reflecting the magnitude of gains and losses associated with the good and bad decks, but is independent of long-term consequences and fails to discriminate between good and bad decision makers.

Finally, Chapter 5 aimed to investigate decision making in a more naturalistic environment through the use of the food diary methodology in morbidly obese individuals. Invoking the concepts of emotional, external and restraint eating, the relationships between these self-reported eating styles which characterise affective, sensory, and cognitive influences on eating, respectively, and food intake were examined. Cognitive restraint was the strongest predictor of lower overall energy intake, external eating was related to higher intake in females only and emotional eating was unrelated to intake. With regards to food type, significant results were found in the case of top-shelf foods only where cognitive restraint was significantly associated with lower, and external eating with higher, intake of such foods. These findings largely reflected previous research in healthy weight populations. A second research question concerned whether obese self-reported emotional eaters ate more when emotional. The results showed that for both positive and negative affective states, self-reported emotional eater status had no impact on the amount of overall food intake or on the consumption of specific food types. As these findings were similar to those in healthy populations, both the predictive validity of emotional eater scales and indeed the construct of emotional eating itself were debated.

6.1.1 Affective decision making in obesity

Taken together, what do these findings reveal about affective decision making in obesity? In terms of the neuropsychological approach, the IGT was employed as the main test of affective decision making. Chapters 2 – 4 robustly showed that obese individuals were impaired on this task. However, the specific role of affect, and thus the SMH, as the source of such deficits was not confirmed as obese individuals developed anticipatory, and appraisal ‘somatic marker’ signals of similar magnitude to control participants. According to Bechara
and Damasio (2002), the profile of obese individuals here was that of 'high risk-takers', choosing to override somatic marker signals with conscious deliberation. This high risk-taker view was not supported by behavioural data on the GDT where obese participants were found to be unimpaired on this task. As such, perhaps it is the case that obese participants are high risk-takers in uncertain or ambiguous situations only, in contrast to explicit conditions. Findings from the WAIS-III indicated that intelligence played a significant role in relation to the IGT, removing overall performance differences between the obese and comparison group. However, as the interaction term between group and block remained significant, this indicated that the obese group did learn at a lower rate that the comparison group. Indeed, in Chapter 2 it was suggested that the relatively flat learning curve of the obese group (which tended to centre around zero in all tests of the IGT in this thesis) could be the expression of random choice behaviour, or could be the product of a real inability to develop a specific strategy on the IGT, with participants unable to maximise both an immediate reward, or programme a delayed reward. The findings from the SGT, which revealed that both groups responded to immediate gain-loss frequency (maximised an immediate reward) and not long-term expected value, would seem to point to those explanations concerning random choice behaviour or the ability to programme a delayed reward. In terms of the Expectancy Valence model, it would suggest that deficits may lie in the learning rate or choice inconsistency parameters (Yechiam, Busemeyer, Stout, & Bechara, 2005). Accordingly, large values of $\alpha$, characterised by insufficient updating of expectations due to strong recency effects and rapid discounting of past outcomes may lead to selection of the disadvantageous decks. Similarly, low values of $\delta$, whereby choices are inconsistent, random and independent of expectancies over time, may account for an inability to learn on the task.

6.1.2 Obesity and addiction

The original application of the IGT, widely used within the conventional addiction literature, to an overweight and obese population was based on the parallels between obesity and conventional addiction (Davis et al., 2004) as articulated in Chapter 2. While obese individuals showed similar performance to these addictive groups, application of the Expectancy Valence model (Busemeyer & Stout, 2002) would be a useful means of discriminating decision making deficits in obesity from conventional addiction. Yechiam et al. (2005), who used this model, identified a cluster of clinical groups characterised as high either in attention to gains or in recency, the most extreme of which included cocaine and cannabis abusers. By contrast, a second cluster of clinical groups was identified whose decision making
style was characterised by high in attention to loss (e.g. Parkinson's patients), in some cases coupled with erratic choices as demonstrated by a low sensitivity parameter (e.g. Asperger's syndrome). The 'normal' appraisal physiological profile of obese participants, combined with the fact that their behavioural profile on the IGT centred on zero (and not the high reward disadvantageous decks), and that they showed the normal tendency to be loss averse on the SGT, suggests that obese participants would not be characterised as high in attention to gains like the addictive groups above. However, the flat learning curves of obese participants demonstrated throughout the thesis, whereby participants tended to survive on a net score of zero, may indicate they adopted a simple 'win-stay' or 'lose-shift' strategy in all decks, producing a tendency to oscillate between alternatives and thus failing to learn to stick with the better decks. This profile is suggestive of an erratic or random choice pattern, and thus, as previously articulated, is suggestive of a potentially low sensitivity parameter (choice consistency) (Yechiam et al., 2005) on the Expectancy Valence model.

Davis and Carter (2009), in delineating the parallels between BED and conventional addiction, highlighted how presenting BED patients with an addiction model of compulsive overeating, with the implicit message that they may be fighting a strong neurobiological drive to overeat in an environment that exploits these urges, may help foster a therapeutic sense of self-empathy. In addition, it may promote an understanding that treatment is likely to involve learning effective strategies and enduring life-long efforts to resist overeating and prevent relapse. Successful behavioural interventions which have been applied from addiction to obesity include incentive motivation, cognitive-behavioural therapy and 12-step programs (Volkow & Wise, 2005). In addition, motivational interviewing (MI), a style of behaviour counselling designed to help increase one's motivation to change, was initially developed for treatment with alcohol abusers but has shown to be effective for obesity (Hettema, Steele, & Miller, 2005; Rubak, Sandbaek, Lauritzen, & Christensen, 2005). The overlap between the fields in terms of decision making processes is similarly evident in the recent Motivation to Eat Scale, which was developed based on a four-category model of motivations for alcohol use (Cooper, 1994), including to cope with negative affect, to be social, to comply with others' expectations, and to enhance pleasure (Jackson et al., 2003), in order to understand healthy and disordered eating. A better understanding of the way in which decision making in obesity is similar too, and differentiated from, conventional addiction, would allow for greater specification and tailoring in applying treatment models from conventional addiction to obesity.
6.1.3 Affective decision making across disordered eating groups

Parallels between obesity, AN and BN have previously been drawn in the cognitive orientation (CO) of eating disorders. Accordingly, this paradigm identified major themes which make up a 'general eating disorders core' including, for example, avoiding emotional expression of all kinds, avoiding negative emotions such as anger and hostility, not being in control of one's life, and the absence of enjoyment (Kreitler, 2010). This thesis revealed that using the IGT, behavioural decision making deficits in obesity were shared by other disordered eating groups such as AN and BN. Both a model of learned helplessness and deficits in reversal learning were proffered as potential mechanisms to account for these deficits. The profiles can be differentiated in terms of the role of affective biasing signals in decision making and therefore in their ability to support SMH. Accordingly, deficits in AN (Tchanturia et al., 2007) but not in BN (Liao et al., 2009) or in obesity, were related to the impaired activation of (both anticipatory and appraisal) 'somatic marker' states. However, as 'somatic marker' deficits were only revealed in currently ill AN patients, and were not present in recovered-AN (Tchanturia et al., 2007) or in the other disordered eating groups of BN and obesity where participants were on average overweight or severely obese, respectively, these results may be attributable to the effects of starvation itself. Thus, while it is evident that these disordered eating groups share common decision making impairments in terms of the ability to forego an immediate reward for a longer term better outcome, this does not appear to be due to common impairments in affective biasing signals.

Consistent with the previous conclusions based on the parallels between obesity and conventional addiction, further decomposition and differentiation of IGT performance in these groups, using for example the Expectancy Valence model (Busemeyer & Stout, 2002) or the variant IGT (Bechara et al., 2002), would be useful. Therapeutic intervention in the CO approach is derived from the tools for predicting eating disorders and differentiating eating disorder groups from each other. Accordingly, support is mobilised in clients for themes indicative of healthy behaviour so that a stable motivational disposition towards healthy behaviour can be formed. Clients are then provided with a behavioural programme that implements the health-oriented motivational disposition (Kreitler, 2010). As per suggestions in this thesis that future research in decision making should move to the level of intervention, such an approach could be applied with regards to decision making in ED's, whereby the specific type of decision making deficit underlying a particular ED (e.g. attention to gains vs.
losses parameter, choice consistency, reversal learning), becomes the target for intervention. Similar to the way in which the CO approach does not focus on strengthening beliefs like 'I want to lose weight' in obesity or 'I want to gain weight' in AN, the focus is on changing the underlying motivational disposition for healthful behavioural and the provision of an adequate behavioural program for the implementation of the newly formed disposition (Kreitler, 2010).

6.1.4 Affective decision making and food intake

With regards to the naturalistic approach to affective decision making used in this thesis, decision making around energy intake and food choice, was assessed through the construct of emotional eating, derived from the psychosomatic theories of overeating. In using the food diary method, this aspect of the thesis included the environmental context, namely the obesogenic environment, in which decision making occurs. In contrast to the concepts of cognitive restraint and external eating, self-reported emotional eater scales were not predictive of overall intake or food type in the current study, largely replicating previous findings in studies of healthy populations. Similarly, the finding that emotional eater status had no impact on overall energy intake or food choice, when in positive or negative mood states, replicates previous findings in both lab and food diary studies in healthy populations. In many ways linking findings using the neuropsychological approach with this food diary study, Davis et al. (2004) found the relationship between poor decision making and increased BMI was not mediated by the construct of emotional eating. These findings cast doubt, not on the association between negative emotions and food intake, an association which is widely supported in the literature (Conner et al., 1999; Macht et al., 2005; Newman et al., 2007; O'Connor et al., 2008; O'Connor & O'Connor, 2004), but specifically on the ability of emotional eater scales to capture the tendency to eat when emotional, in both healthy and obese populations. Questions were raised over the predictive validity of these scales, which measure desire to eat, as opposed to frequency of eating in response to negative emotions, as well as their heavy correlation with external eating scales. Regarding the construct of emotional eating itself, the accuracy of people’s understanding of their eating behaviour and the role of emotion in driving that behaviour was addressed, with specific focus on the ‘triple recall bias’ in emotional eater scales and the impact of hot states on behaviour. An alternative view, that emotional eater scales may be an expression of concerns about one’s eating behaviour or an emotional attitude to eating was also expressed. In summary, affective decision making, as assessed by emotional eater scales, was not predictive of food intake or food type in obesity.
6.1.5 Psychophysiological brakes

As part of addressing affective decision making in morbid obesity, this thesis sought to evaluate ineffective brakes at the cognitive and psychophysiological levels of the 'runaway weight gain train'. Using the neuropsychological approach, deficits were found on the IGT. However, impaired IGT performance was not associated with impaired 'somatic marker' signals, and so was not attributable to faulty psychophysiological brakes. Indeed, on all three tasks, the psychophysiological profile of the obese participants was in line with that of the comparison group. The pattern of performance on both the IGT and SGT showed that all participants showed heightened SCR to the disadvantageous decks of the former, and advantageous (likely perceived as disadvantageous) decks of the latter, while no effects were observed for anticipatory heart rate. Similarly, for response SCR there was a deck x reinforcement interaction on both tasks, for wins on the IGT and losses on the SGT. In addition, there was a main effect of reinforcement on the SGT with greater SCR and heart rate slowing to losses than wins. As such, it seems that psychophysiological activation was sensitive to the overall pattern of decision making including the type of decision (good or bad) and the reinforcement given to that decision (win or loss), but did not discriminate between obese and comparison participants.

6.1.6 Cognitive influences on decision making

What of the cognitive brakes of the train? The food diary study addressed personal factors, including restraint, external and emotional eating, which determine how a person responds to the obesogenic environment (Snoek et al., 2007). Restraint, as a concept, can be seen to most closely represent a cognitive brake, in that it invokes cognitive effort in the conscious restriction of food intake. In the sample, restraint was the dominant factor, most strongly associated with lower overall energy intake, and top-shelf foods, reflecting previous research in healthy populations. Therefore, it seems that analogous relationships between self-reported eating style and intake exist in healthy and obese populations, though clearly the former achieve regulation of dietary intake while the latter do not. However, the very concept of restraint does not necessarily involve 'successful restraint' and includes the issue of counterregulatory eating. In this way, restraint relates to the dieting positive feedback loop of the 'runaway weight gain train'. Although restraint is generally perceived as an anti-dieting paradigm, recent research has argued that dietary restraint and dieting are not equivalent (Rideout & Barr, 2009). In a large sample of postmenopausal women, restraint eating (when considered independently of dieting), was associated with lower BMI leading to conclusions
that restraint eating, rather than dieting, may contribute to successful weight management (Rideout & Barr, 2009). As such, it would seem that more knowledge on the factors which promote successful restraint independent of dieting is required. Similarly, as suggested in Chapter 5, future research should address those factors which disrupt successful restraint, that is, those potential self-regulatory capacities, which moderate the relationship between cognitive restraint and successful / unsuccessful dietary behaviours.

With regards to other elements of the thesis which tapped cognitive processes, no differences between explicit or risky decision making in the obese and comparison group were evident as measured by the GDT. Thus, unlike the ambiguous IGT, obese individuals were unimpaired on a task where the rules were explicit and reward / punishment contingencies were stable. This finding supports results of self-reported decision making in Chapter 2 as measured by the CFC, which explicitly asks people about the weight given to immediate versus distant consequences of behaviour. Accordingly, there were no differences between the groups on this measure and CFC scores did not relate to IGT performance. In addition, use of feedback in decision making was impaired on IGT but not the GDT, similarly supporting a clear distinction in cognitive decision making impairments in obesity, present under ambiguous but not explicit conditions.

6.1.7 Individual difference variables and decision making

Cognitive abilities were found to have a significant effect on decision making performance on the IGT, but not on the other decision making tasks. Accordingly, in Chapter 4, comparison participants were found to have significantly higher WAIS-III scores than obese participants. Similar to previous findings on the role of education in an obese population (Davis et al., 2010), WAIS–III scores removed group differences in net and block net scores. However, education level did not have the same effect, indicating WAIS-III scores may be more sensitive in discriminating differences in IGT performance. These findings corroborate previous studies which found cognitive intelligence was predictive of IGT performance (Demaree et al., 2010) but contrasts a review by Toplak et al. (2010) which generally found little role for cognitive abilities in IGT performance. With regards to eating pathology, Chapter 2 found obese participants scored significantly higher on measures of restraint eating, as well as the anger / frustration and depression subscales of the emotional eating scales. These measures of eating pathology did not uniquely predict IGT performance, both for the sample as a whole and for obese and comparison participants separately. Such findings were in line
with previous results in individuals with BED (Svaldi et al., 2010) but contrasted findings in BN where IGT performance negatively correlated with bulimic symptomatology (Boeka & Lokkenz, 2006). As such, in the context of obese populations, eating pathology does not seem to significantly impact upon IGT decision making. Similarly, severity of illness as assessed by BMI was unrelated to IGT performance, replicating previous findings in AN (Cavedini, Bassi, Ubbiali et al., 2004). With reference to personality factors which may impact decision making in obesity, impulsivity, as assessed by the CFC in Chapter 2, and BIS in Chapter 4, was not uniquely related to decision making on any of the decision making tasks used here, which reflects previous findings in BN (Liao et al., 2009) but contrasts findings in healthy populations where impaired performers on the IGT were found to be significantly more impulsive (Davis, Patte, Tweed et al., 2007). Differences between the groups were observed on the BIS-NP only, which may once more imply deficits on the choice consistency parameter of the Expectancy Valence model (Busemeyer & Stout, 2002). These results suggest that impulsivity is not uniquely related to decision making in obesity. Alternatively, the self-reported measures used here may not be sufficient to capture potential influences of impulsivity on decision making and as such, perhaps future research should move towards also incorporating behavioural measures of impulsivity. Finally, there were no significant differences between the groups on the GHQ, a measure of current psychological distress, which was unrelated to decision making capacity in obese individuals.

6.1.8 Integrating neuropsychological and naturalistic approaches

The pattern of findings, from both the IGT and the food diary study in this thesis, can perhaps be best integrated in terms of their common implication of executive functioning deficits in morbid obesity. Accordingly, in terms of IGT deficits, these deficits were unrelated to affective biasing signals as specified by the SMH, and measures of personality, eating pathology, psychological distress and severity of illness. In addition, the interaction between group and task learning over time on the IGT was found to persist after controlling for intelligence. As such, these results point to the possibility that broader executive functioning deficits underlie decision making in morbid obesity. Similarly, in the food diary study analogous relationships between cognitive restraint and lower overall energy intake and healthier food choices were found for morbidly obese participants and healthy populations. However, as obese individuals evidently do not achieve successful restraint it was suggested that a failure in broader self-regulatory capacities may moderate the relationship between cognitive restraint and dietary behaviour. In support of these inferences, recent research has
found that extremely obese individuals show executive functioning deficits in areas such as planning, problem solving and mental flexibility, independent of medial co-morbidities (Boeka & Lokken, 2008). In addition, in a study which used the IGT as representative of dieting, restraint eating was related to worse IGT decision making when dispositional self-control was low but to better decision making when self-control was high (Kuijer et al., 2008). Similarly, in a sample of obese patients seeking weight-loss treatment, successful weight loss was associated with increased dietary restraint and reduced disinhibition (Grave, Calugi, Corica, Domizio, & Marchesini, 2009).

TST (Hall & Fong, 2007), discussed previously in Chapters 1 and 5, provides a useful way of bridging these disparate literatures in a comprehensive framework for decision making deficits in morbid obesity. Accordingly, the proposal that findings from the neuropsychological and naturalistic approaches are indicative of broader executive functioning deficits strongly fits with this biopsychosocial model of human behaviour, particularly the concept of a biologically based SRC, which refers to the capacity to exert top-down control over one's actions and is synonymous with executive function (Hall & Fong, 2010). TST argues for the potential for SRC to increase the influence of intention on actual behaviour. As such, increasing SRC in obese individuals could possibly extend the intact ability to make advantageous decisions in explicit situations, to those of an implicit or uncertain nature, with potentially tangible impacts on diet and weight regulation. In a similar manner, increasing SRC could arguably maintain (i.e., eliminate disruption to) and potentially improve the cognitive restraint – eating behaviour association in morbid obesity. In addition, in line with the behavioural prepotency element of TST, which argues that past behaviour is important to explaining health behaviour tendencies, both objectively and subjectively (Hall & Fong, 2010), future research could address the concept of habit strength, which has been found to be an important predictor of eating behaviour.

A move towards research of this kind is particularly significant in view of newly emerging technologies which aim to retrain executive functions and measure subsequent impact on target behavioural outcomes. For example, Neurosynergy games have both an IQ and EQ training model. The IQ training module has a plurality of independently adjustable neurocognitive variables, each of which is used to exercise a specific sub-component of executive function, the EQ training module allows conditioning / deconditioning and an IQ-EQ

6.1.9 The validity of the SMH

As demonstrated in Chapter 4, IGT deficits in obesity were not associated with impaired 'somatic marker' signals and so failed to support the SMH. Consistent with these findings, in comparing impaired vs. non-impaired performers on the IGT, in order to further test the SMH, autonomic activity did not discriminate between these decision making groups and failed to support the SMH as the mechanism underlying impaired decision making. These findings were similar to those of Suzuki et al. (2003) who found anticipatory SCR, and Jenkinson et al. (2008) who found both anticipatory and appraisal SCR, failed to discriminate between impaired and non-impaired decision makers in healthy populations. However, they contrast those of Crone et al. (2004) who found anticipatory SCR to disadvantageous choices discriminated bad from both moderate and good performers, while anticipatory heart rate discriminated good performers only. In terms of the SMH, the pattern of findings supported an alternative hypothesis in the literature, that 'somatic markers' distinguish between good vs. bad decision making, but not between good vs. bad decision makers. As such, 'somatic markers' were argued to influence immediate but not long-term decision making on the IGT. In addition, it was argued that the observed pattern of physiological activity reflected the magnitude of immediate rewards or punishments resulting from the chosen deck, which are larger for the disadvantageous decks of the IGT. As highlighted by Jenkinson et al. (2008) this alternative hypothesis may provide some explanation for the conflicting evidence in support of the SMH. Thus, regarding the debate around the occurrence of abnormal IGT performance in healthy individuals in the presence of normal 'somatic markers' (Bechara & Damasio, 2002; Bechara et al., 1999; Bechara et al., 2001), or similarly the occurrence of deficient IGT performance in obese individuals in the present thesis despite normal 'somatic markers', such findings can be accounted for if somatic markers reflect the immediate decision of choosing a good or bad deck, but are independent of overall IGT performance. As for the debate around whether anticipatory or feedback 'somatic marker' activity is more important for successful IGT performance, both were found to influence decision making in the current study, but as per previous findings, neither discriminated between impaired vs. non-impaired IGT performance. As such, in terms of the current proposal both can be considered important for guiding immediate decision making but fail to distinguish between decision makers in the long term. Finally, in terms of the cognitive penetrability of the IGT and therefore whether
participants need to rely on 'somatic marker' signals to guide decision making instead of conscious knowledge, Chapter 4 showed that all participants seemed to gain a clear understanding that they earned the least on the disadvantageous decks. However, only 61% of the comparison group and 38% of the obese group, correctly identified that they earned the most on the advantageous decks. Taken together, 50% of participants did not intuit the positive EV of the advantageous decks. These findings support the opaqueness of the reward / punish schedule of the IGT (Tranel et al., 2000). In terms of the current conceptualisation of SMH, it indicates that 'somatic markers' can be associated with immediate good or bad decision making, even if conscious knowledge of the task has not been acquired.

While the pattern of findings here fitted with those of Jenkinson et al. (2008), supporting their alternative hypothesis that 'somatic markers' distinguish between good vs. bad decision making, but not between good vs. bad decision makers, the limitations of our data to test the SMH must be acknowledged. Firstly, our claims rest upon a small heterogonous sample which included clinically obese participants with comorbid psychiatric and somatic conditions. In addition, a range of technical and measurement issues arose in the use of psychophysiological data as a measure of 'somatic marker' biasing signals, as outlined in section 6.1.10 below. Perhaps most importantly, our test of the SMH focused solely on one specific component of SMH neural circuitry, namely the role of peripheral body signals in decision making. However, such visceral responses are just one element of a broader emotional response system that includes changes in the endocrine, skeletomotor systems, and changes in the brain that alter the perceptual processing of biologically relevant stimuli (Reimann & Bechara, 2010). As such, the data presented here is limited both in its generalisability to all obese individuals, and in its ability to adequately assess the veracity of the SMH.

6.1.10 Limitations of the research

Sample sizes in Chapters 2 and 5 were moderate, while those in Chapters 3 and 4 were relatively small. In addition, with the exception of some participants in Chapter 4, the majority of participants in this thesis were either treatment-seeking or in treatment for their weight problems, a consequence of the specific focus on morbid obesity and indeed disordered eating groups. The use of such samples limits the generalisability of the findings from this thesis to the general population of obese individuals. In terms of measuring obesity, BMI was the only method employed in this study. While BMI is the most widely used indicator for weight
classification, it does not allow for differences in weight between muscle and fat. As such, other methods of measuring obesity which may be more useful, particularly on an individual as opposed to population level, include percentage body fat and waist-circumference and waist-hip-ratio in the measurement of abdominal obesity (Seidell & Flegal, 1997). However, due to the severity of obesity in this sample, with the average BMI > 40, it is unlikely that participants were miscategorised as obese using BMI. With regards to age, often the obese group was significantly older than comparison or other disordered eating groups and as a result age was controlled for statistically. Chapter 3, unlike the other empirical chapters, included female participants only. In addition, in Chapters 2, 3, and 5 the samples were not screened for comorbid somatic or psychiatric disorders, history of drug abuse, illness duration, medication use or IQ, factors which may influence decision making capacity. Similarly, executive functioning was not assessed in this thesis. While the focus of this thesis has been on elucidating potential decision making deficits in morbid obesity, it must be highlighted that the fact that these individuals were seeking or presenting for treatment is a good decision. This point perhaps highlights the micro-level quality of this research, addressing impairments in discrete aspects of decision making only, the cumulative influence of which may have a deleterious affect on health. Additionally, it echoes the conclusions of the thesis that impairments existed in ambiguous but not explicit situations, with the decision to seek treatment or engage in research arguably residing in the latter category.

With reference to behavioural measures used in this thesis, as per previous studies, there was substantial variation in control participant performance on the IGT, with 38% of participants in Chapter 2 and 50% of participants in Chapter 4, impaired on the task. Such findings question the validity of the IGT paradigm if such individuals are impaired on the task, but presumably function normally in terms of everyday decision making. They also support the suggestion by Dunn et al. (2006) that measures of the quality of everyday decision making be included as an ecological check to assess if control participants who are impaired on the IGT, are similarly impaired in everyday decision making. Conclusions from the SGT that obese individuals show the normal tendency to underweight rare events and / or to be loss averse, in line with comparison participants, are constrained by the argument that this task may have been more explicit than the uncertain IGT, due to the presentation of outcomes in the form of a net score and the enlargement of the differences in EV between the decks. As such, both sensitivity to gain-loss frequency, but also the potentially explicit nature of the task, may have contributed to performance.
The issue of sample size becomes more important with regards to the acquisition of physiological data in this thesis. Accordingly, as indicated by the error bars for SCR and heart rate data, there was substantial individual variation on these measures. There was also loss of data, particularly heart rate data, due to technical issues, which became most apparent on the GDT due to the smaller number of trials on this task. The acquisition of heart rate data, though moving away from the over-reliance on SCR data as the sole measurement of 'somatic marker' activity, proved less sensitive than SCR as a measure of autonomic activation relating to feedback responses on the SGT only. In addition, the impact that 'being obese' had on these measures is unclear. On a more general level, the matching of an individual's choice on a decision making task to specific SCRs or IBI responses is regarded as imprecise and is an association only with no implications of causality. Further, it is difficult to separate physiological arousal to the very act of doing the task, from the task itself. Finally, the ability to compare results across studies using physiological measures is constrained by substantial variability in the measurement (e.g. time windows in which data is acquired) and form of analysis of particularly SCR data. The relatively brief time windows adopted by researchers may also result in overlap of anticipatory and appraisal physiological activity, leading to a failure to clearly distinguish these events types.

With reference to the food diary study, the possibility that participants completed some entries retrospectively, potentially leading to an underreport of caloric intake (which research indicates is greater in overweight and obese individuals) cannot be ruled out. The paper and pencil method used here, although deemed most appropriate for the population under consideration, has been associated with lower compliance rates than electronic diaries with compliance-enhancing features. There was also loss of data here due to poorly completed diaries. In the assessment of restraint, external and emotional influences on intake, the study relied on the DEBQ (Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986) only as a measure of self-reported eating style. In addition, outside of self-reported eating style, there was a substantial amount of unexplained variance in intake. Data were obtained on participant-identified binges only and not using more objective criteria. Finally, the lack of support for emotional eating in the study could be related to a social desirability bias in this treatment-seeking sample, whereby it is socially desirable to endorse questions about being on a diet and not to endorse those about emotional and external eating.
6.1.11 Future research

A prevailing theme throughout Chapters 4 and 6 of this thesis is that future research should move to apply the Expectancy Valence model (Busemeyer & Stout, 2002) to IGT data. This would discriminate IGT deficits in obesity from those in conventional addiction. Similarly, the application of this model to the IGT data of other disordered eating groups (e.g. AN, BN) would further elucidate the shared profile of decision making deficits revealed in AN, BN and obesity in Chapter 3, and indeed ways in which individual profiles may differ. A move towards research of this kind would also address the criticism that the IGT, due to its complex nature, lacks specificity with a majority of patient groups showing deficits on this task (Dunn et al., 2006). It would also inform of the potential usefulness of applying common treatment interventions or part thereof, to both obesity and other conventional addictions and obesity and other disordered eating groups.

With regards to the role of SMH as a mechanism for explaining decision making deficits in obesity, it is recommend that future research replicate the work of Chapter 4 using the IGT together with psychophysiological measures (SCR, heart rate), due to the small sample in this study and the inherently large amount of individual variation on these measures. As the SMH was not supported as the mechanism for IGT decision making deficits in obese individuals, further investigation of potential alternative mechanisms, such as deficits in reversal learning or a model of learned helplessness as suggested in Chapters 2 and 3 is warranted. In addition, use of the variant E’F’G’H’ IGT in obesity would identify if deficits may be in part due to hypersensitivity to reward / insensitivity to punishment, or alternatively a general ‘myopia’ for future consequences with behaviour guided by immediate prospects. Overall, data from the thesis for obese individuals (e.g. flat IGT learning curves; SGT scores which evidenced the normal tendency to be loss averse; ‘normal’ somatic marker signals which did not differ significantly from the comparison group) are not suggestive of hypersensitivity to reward / insensitivity to punishment and are perhaps more fitting with the latter interpretation, and similarly a random or erratic decision making style as per the choice consistency parameter of the Expectancy Valence model (Busemeyer & Stout, 2002).

With regards to decision making in a more naturalistic context as assessed in the food diary study, it is recommended that future research evaluate the influence of emotional eating on intake (overall and by food type) using a more episodic approach to assess whether affective influences on eating, as assessed by emotional eater scales, may be better captured
in this way. In addition, it is recommended that prospective studies investigate the relationships between eating styles, intake patterns and BMI over time, to gain a better conceptualisation of normal and pathological eating behaviour and its relationships with weight regulation. Use of alternative measures to the DEBQ (Van Strien, 2005; Van Strien, Frijters, Bergers et al., 1986) is also recommended.

Integrating findings from the neuropsychological and naturalistic perspectives, it was suggested that broader deficits in executive functioning and SRC may underpin decision making impairments in morbid obesity. Throughout this thesis it was articulated that the challenge for future researchers is to bring research of this kind to the level of intervention, to change the ability to modulate reward and punishment in a long-term perspective and measure subsequent impact on psychological and physical outcome, in both obesity and indeed other disordered eating groups. As such, executive functioning and SRC may represent particularly important targets in this regard. Similarly, it was argued that future research should determine the executive functioning and SRC factors which modulate the cognitive restraint – eating behaviour relationship, with a view to sustaining and improving this relationship in morbid obesity.

6.2 Conclusion

A series of studies was employed to investigate affective and cognitive influences on decision making in morbid obesity. While obese participants were impaired on the IGT, the role of affective biasing signals and thus the SMH as the mechanism underlying these deficits was not supported. Similarly, the role of affective influences, as measured by emotional-eater scales, in predicting energy intake and food choice was not confirmed. By contrast, cognitive restraint was strongly related to the above factors. The specificity of decision making deficits to decision making under uncertain conditions was indicated by intact performance on explicit tests of decision making. The overall psychophysiological, neuropsychological and psychometric profile was not suggestive of a decision making style dominated by hypersensitivity to reward or insensitivity to punishment in obesity, but rather a random or erratic decision making style and a general myopia for future consequences. The robust findings with regard to IGT deficits and the inability to specify the source of such deficits, combined with the dominant presence but evident failure to achieve successful cognitive restraint in dietary behaviour, were suggestive of broader deficits in executive functioning and self-regulatory processes in morbid obesity.


162


168


172


174


Appendix 1: Psychophysiology and the IGT

1.1 SCR: Definitions

-SC: SC is a form of electrodermal activity, which refers to the degree to which the skin permits the flow of current and is related to the degree of eccrine sweating, that is, the secretion of electrolyte solution by sweat glands. It is measured by applying a small voltage across two electrodes and measuring the resulting current that flows between them. The amplitude of this current is proportional to the skin conductance. The unit of skin conductance is the microsiemen (μS). SC is typically measured from the palms of the hands but can also be measured from the soles of the feet (see image below for the measurement of SC in the current study). These areas, referred to as 'volar surfaces' are unique in that eccrine sweating is related to mental processes as opposed to thermoregulation. SC is based in the primitive 'fight or flight' response, whereby the body prepares itself for the exertion needed to deal with a perceived threat by increasing sweating to cool itself. It is seen as a highly sensitive index of autonomic responsivity especially with regards to emotional stimuli. SC is also known as Galvanic Skin Response (GSR) or Electrodermal activity (EDA) but SC is the currently used term.

-Phasic skin conductance: changes that occur with a discrete time window (usually 1-5 seconds) following the presentation of a stimulus. This is the kind of skin conductance measured by the IGT. It contrasts tonic skin conductance, which generally refers to changes in skin conductance that are not casually related to an eliciting stimulus.
- **SCR**: An increase in skin conductance over a threshold value (0.01 μS in the current study) occurring within a given time interval after the onset of a stimulus, typically 5 seconds, beginning one second after stimulus onset.

- **Anticipatory SCR**: The SCR measured in the time widow before a participant selects a card. Anticipatory SCRs are always computed for the good and bad decks separately. Successful performance on the IGT is correlated with the development of anticipatory SCRs (i.e. 'somatic makers') which are *higher* for disadvantageous than advantageous decks. The absence of anticipatory SCRs has similarly been correlated with impaired task performance.

- **Reward SCR**: Feedback SCR to a card selection which leads to a win (no associated loss). Reward SCRs are usually computed for the good and bad decks separately.

- **Punishment SCR**: Feedback SCR to a card selection which leads to a loss (loss or a net loss). Punishment SCRs are also usually computed for the good and bad decks separately.

### 1.2 SCR Measurement

- **SCR amplitude**: SCR amplitude is the most commonly used phasic skin conductance measurement. It refers to the difference between the peak SC value within the measurement window (the high point where the tangent of the SC curve is zero) and the trough value (the low point where the tangent of the SC curve is zero) preceding this peak (i.e. the difference between the onset and peak value of an SCR wave). Studies may report the amplitude of the first SCR, the largest SCR, or the average of all SCR fluctuations within a measurement window. Other studies may not report the amplitude value but the *number* of SCRs within the time window.

- **'Area under the curve'**: This measurement calculates the total area between a waveform and a baseline value within the endpoints of a selected area. ‘Area under the curve’ measurement is similar to the function of an ‘integral’ except that, instead of using zero as a baseline for integration, a straight line is drawn between the endpoints of the selected area to function as the baseline. The area is expressed in terms of amplitude units (mS) per time interval (seconds). Unlike amplitude, ‘area under the curve’ measurement does not assume the onset of a discrete SC response (i.e. that SCR is casually related to a stimulus).
Inter-trial interval: The time window after the presentation of feedback, and before the next allowable response by the participant can be made.

1.3 Heart rate: Definitions

HRV: is a measure of the variability of the heart; i.e. variability in IBI.

IBI: The time in milliseconds (Ms) between consecutive R waves of an electrocardiogram (Ekg). IBI can be converted into BPM (beats per minute) values by taking the reciprocal of the IBI and multiplying it by 60. IBI generally fall within a range of 200 - 2000ms, corresponding to 300 - 30 BPM.

Anticipatory heart rate: The change in heart rate before making a card selection. Research has robustly shown that heart slows when an individual prepares for a voluntary response. Early results indicate that for good performances on the IGT, heart rate slowing (larger IBI intervals) is greater to the disadvantageous than advantageous decks.

Response heart rate: The change in heart rate after a card selection. This is calculated separately for choices which lead to wins (no associated loss) and losses (loss or a net loss). Early results indicate that for all performances heart rate slowing increases following loss relative to reward outcomes.

1.4 Measures of heart rate on the IGT

Anticipatory heart rate: Preliminary studies using heart rate with the IGT have computed anticipatory heart rate based on the difference between the IBI concurrent to the response (IBI 0) and the IBI preceding the response (IBI-1).

Response heart rate: The first IBI following the presentation of feedback (IBI+1) is referred to the 2nd IBI preceding the response (IBI -2). This is conducted separately for reward and punishment responses.

References


Appendix 2: Ethical approval letters

2.1 Ethical approval for studies in Chapters 2 and 3:

To Whom It May Concern:

I am writing to confirm that we support Amy Brogan’s project using cognitive assessments on patients at the Obesity Clinic in St Columcille’s Hospital in Loughlinstown. We confirm that the clinic has a sufficient number of clients to facilitate the successful completion of the project. The study has been reviewed by the clinic and ethical approval has been granted by the consultant.

We hope this proves satisfactory and we look forward to participating in the study.

Yours truly

Ruth Yoder, Clinical Psychologist

Dr. Donal O'Shea, Consultant Endocrinologist
F. A. O. Amy Brogan

School of Psychology Research Ethics Committee

19 February 2008

Dear Amy,

Following receipt of a satisfactory review from our medical expert, I am pleased to inform you that your application entitled "The Iowa Gambling Task (IGT) and obesity" has been approved by the School of Psychology Research Ethics Committee.

Yours sincerely,

Kevin Thomas, PhD
Chair,
School of Psychology Research Ethics Committee
2.2. Ethical approval for Chapter 4:

Dear Amy,

I am pleased to inform you that the amendments to your project entitled “The Iowa Gambling Task (IGT) and obesity” have been approved by the School of Psychology Research Ethics Committee.

Yours sincerely,

Dr. Tim Trimble
Chair
School of Psychology Ethics Committee

SCHOOL OF PSYCHOLOGY
Aras an Phaisceag
Trinity College
Dublin 2
2.3 Ethical approval for Chapter 5:

15/05/2009

The School of Psychology Research Ethics Committee

To Dr. Thomas,

I am writing to confirm that we, the Weight Management Service affiliated with St. Columcille’s Hospital in Loughlinstown, support Amy Brogan’s food diary project investigating the role of affect in regulating eating behaviour.

The study has been reviewed and approved by the clinic. As there is no Ethics Committee body for St. Columcille’s we will abide by the decision of the School of Psychology Research Ethics Committee, TCD.

We confirm that the clinic has a sufficient number of clients to facilitate the successful completion of the project.

We hope this proves satisfactory and we look forward to participating in the study.

Yours sincerely,

Ms. Ruth Yoder, Senior Clinical Psychologist

Prof. Dónal O’Shea, Consultant Endocrinologist
29 May 2009

F.O.A. Amy Brogan

School of Psychology Research Ethics Committee

Dear Amy,

Following receipt of specified amendments, I am pleased to inform you that your application entitled “Affect regulation and food diary” has been approved by the School of Psychology Research Ethics Committee.

Yours sincerely,

Kevin Thomas, PhD
Chair,
School of Psychology Research Ethics Committee
Appendix 3: Study protocol for Chapters 2 and 3

Information sheet: Eating habits and decision-making

My name is Amy Brogan and I am a post-graduate psychology student at Trinity College, Dublin. I request your consent to participate in a study entitled "Eating habits and decision-making". In this study you will be asked to provide some general background information on yourself and have your height and weight measured in order to calculate your Body Mass Index (BMI). You will then be asked to complete a short questionnaire on eating habits. Finally, you will perform a computerised card gambling task, which is designed to replicate real-life decision-making.

Participation in this study is voluntary and there are no consequences to not participating, as it will not affect your future medical care. All information provided by you will remain fully confidential. This information will be stored in accordance with the Data Protection Act. Under the Freedom of Information Act, 1997, you have the right to access your own data and can contact the researchers to get such information.

Once you have read this letter and have received satisfactory answers to any questions you may have about the study, if you decide that you would like to participate, then simply sign the consent form below. Please note that your consent sheet will be kept in a secure filing cabinet in my office in Trinity College Dublin. Finally, you are free to withdraw from the study at any time, without prejudice.

Should you require any additional information, please do not hesitate to contact myself or my supervisor Dr. David Hevey, as per the contact details below.

Amy Brogan B.A. (Hons.)
School of Psychology,
Trinity College Dublin,
Dublin 2.
Tel: 01 8962970
Email: broganam@tcd.ie

Dr. David Hevey, C Psychol, Reg Psychol PsSI
School of Psychology,
Trinity College Dublin,
Dublin 2.
Tel: 01 8962406
Email: heveydt@tcd.ie
Consent form:  
Eating habits and decision-making

Eating habits and decision-making

I consent to participate in this study. I am aware that participation is voluntary and that there are no consequences to non-participation. I understand that all information provided by me will remain fully confidential and that I may access this information in accordance with the Freedom of Information Act, 1997. I have been informed that I may withdraw from this study at any time without prejudice.

Print Name: ____________________________

Signature of Participant: ____________________________

Date: ____________________________

Amy Brogan B.A. (Hons.)  
School of Psychology,  
Trinity College Dublin,  
Dublin 2.  
Tel: 01 8962970  
Email: broganam@tcd.ie

Dr. David Hevey, C Psychol, Reg Psychol PsSI  
School of Psychology,  
Trinity College Dublin,  
Dublin 2.  
Tel: 01 8962406  
Email: heveydt@tcd.ie
GENERAL QUESTIONNAIRE

PARTICIPANT NUMBER

[PLEASE PRINT IN CAPITAL LETTERS]

NAME: □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ ...
HISTORY OF WEIGHT MANAGEMENT:

1(a). Are you currently participating in a weight management service? □ Yes □ No (if no skip to question 2)

1(b). If yes, what service are you currently in?
A national HSE weight management service □
A private organisation (e.g. Weight Watchers; Curves) □
Other, please provide details: ____________________________ □

2(a). Have you participated in a weight management service in the past? □ Yes □ No (if no skip to question 3)

2(b). If yes, what service(s) have you previously used?
A national HSE weight management service □
Private Organisation (e.g. Weight Watchers; Curves) □
Other, please provide details: ____________________________ □

2(c). If yes, how long did you use these services for?
Up to 6 months □ Up to 1 year □ 1-2 years □ 3-4 years □ 4+ years □

3. Have you ever taken prescription weight loss drugs? □ Yes □ No

4. Have you ever taken non-prescribed, over the counter, weight loss products? □ Yes □ No

5(a). Have you had bariatric (weight loss) surgery? □ Yes □ No

5(b). Is yes, when did this surgery take place? ____________________________

6. Are you satisfied with your current body weight? □ Yes □ No

Body Measurements
[Details of your height & weight will now be recorded by the researcher]

HEIGHT: ____________ Metres
WEIGHT: ____________ Kgs

193
We all respond to different emotions in different ways. Some types of feelings lead people to experience *an urge to eat*. Please indicate the extent to which the following *feelings* lead you to feel an urge to eat by checking the appropriate box.

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<td>Furious</td>
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<tr>
<td>On edge</td>
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<tr>
<td>Confused</td>
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<tr>
<td>Nervous</td>
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<td></td>
</tr>
<tr>
<td>Angry</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Guilty</td>
<td></td>
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</tr>
<tr>
<td>Bored</td>
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</tr>
<tr>
<td>Helpless</td>
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<tr>
<td>Upset</td>
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</tr>
</tbody>
</table>
The next series of questions ask you about your general eating habits. Please answer these questions using the scale above:

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. If you have put on weight, do you eat less than you usually do?</td>
<td>1. Never 2. Seldom 3. Sometimes 4. Often 5. Very often</td>
</tr>
<tr>
<td>3. How often do you refuse food or drink offered because you are concerned about your weight?</td>
<td>1. Never 2. Seldom 3. Sometimes 4. Often 5. Very often</td>
</tr>
<tr>
<td>6. When you have eaten too much, do you eat less than usual the following days?</td>
<td>1. Never 2. Seldom 3. Sometimes 4. Often 5. Very often</td>
</tr>
<tr>
<td>13. If you see or smell something delicious, do you have a desire to eat it?</td>
<td>1. Never 2. Seldom 3. Sometimes 4. Often 5. Very often</td>
</tr>
<tr>
<td>15. If you walk past the bakery do you have the desire to buy something delicious?</td>
<td>1. Never 2. Seldom 3. Sometimes 4. Often 5. Very often</td>
</tr>
<tr>
<td>16. If you walk past a snackbar or a café, do you have the desire to buy something delicious?</td>
<td>1. Never 2. Seldom 3. Sometimes 4. Often 5. Very often</td>
</tr>
<tr>
<td>17. If you see others eating, do you also have the desire to eat?</td>
<td>1. Never 2. Seldom 3. Sometimes 4. Often 5. Very often</td>
</tr>
</tbody>
</table>

Using the scale above, the next set of questions ask you to think about on how many days, out of the past 28 days, have you eaten an unusually large amount of food, given the circumstances, in response to feelings of:

<table>
<thead>
<tr>
<th>Feeling</th>
<th>Example Feeling</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANXIETY</td>
<td>e.g. worry, jittery, nervous</td>
</tr>
<tr>
<td>SADNESS</td>
<td>e.g. blue, down, depressed</td>
</tr>
<tr>
<td>LONELINESS</td>
<td>e.g. isolated, alone a lot</td>
</tr>
<tr>
<td>TIREDNESS</td>
<td>e.g. worn-out, fatigued</td>
</tr>
<tr>
<td>ANGER</td>
<td>e.g. upset, frustrated, furious</td>
</tr>
<tr>
<td>HAPPINESS</td>
<td>e.g. good, joyous, excited</td>
</tr>
</tbody>
</table>
Consideration of Future Consequences Scale

For each of the statements below, please indicate in the box after the statement whether or not the statement is characteristic of you. If the statement is extremely uncharacteristic of you (not at all like you) please fill-in a "1" on the answer sheet; if the statement is extremely characteristic of you (very much like you) please fill-in a "5" on the answer sheet. And, of course, use the numbers in the middle if you fall between the extremes.

Please keep the following scale in mind as you rate each of the statements below.

1=extremely uncharacteristic
2=somewhat uncharacteristic
3=uncertain
4=somewhat characteristic
5=extremely characteristic

<table>
<thead>
<tr>
<th>Statement</th>
<th>Extent to which statement is characteristic of you</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I consider how things might be in the future, and try to influence those things with my day to day behavior.</td>
<td></td>
</tr>
<tr>
<td>2. Often I engage in a particular behavior in order to achieve outcomes that may not result for many years.</td>
<td></td>
</tr>
<tr>
<td>3. I only act to satisfy immediate concerns, figuring the future will take care of itself.</td>
<td></td>
</tr>
<tr>
<td>4. My behavior is only influenced by the immediate (i.e., a matter of days or weeks) outcomes of my actions.</td>
<td></td>
</tr>
<tr>
<td>5. My convenience is a big factor in the decisions I make or the actions I take.</td>
<td></td>
</tr>
<tr>
<td>6. I am willing to sacrifice my immediate happiness or well-being in order to achieve future outcomes.</td>
<td></td>
</tr>
<tr>
<td>7. I think it is important to take warnings about negative outcomes seriously even if the negative outcome will not occur for many years.</td>
<td></td>
</tr>
<tr>
<td>8. I think it is more important to perform a behavior with important distant consequences than a behavior with less-important immediate consequences.</td>
<td></td>
</tr>
<tr>
<td>9. I generally ignore warnings about possible future problems because I think the problems will be resolved before they reach crisis level.</td>
<td></td>
</tr>
<tr>
<td>10. I think that sacrificing now is usually unnecessary since future outcomes can be dealt with at a later time.</td>
<td></td>
</tr>
<tr>
<td>11. I only act to satisfy immediate concerns, figuring that I will take care of future problems that may occur at a later date.</td>
<td></td>
</tr>
<tr>
<td>12. Since my day to day work has specific outcomes, it is more important to me than behavior that has distant outcomes.</td>
<td></td>
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</tbody>
</table>
Using the above scale (1 - 9), please rate your **preference** for each of the following food items (i.e. which of the following items you would **like** to eat daily):

### Breakfast:

<table>
<thead>
<tr>
<th>Item</th>
<th>Preference</th>
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</thead>
<tbody>
<tr>
<td>High-fibre cereal (e.g. all bran; shredded wheat) or hot cereal</td>
<td>5</td>
</tr>
<tr>
<td>Other cold cereal, such as corn flakes, rice krispies or pre-sweetened</td>
<td>2</td>
</tr>
<tr>
<td>Traditional Irish breakfast items (e.g. sausage, bacon, egg, pudding,</td>
<td>4</td>
</tr>
<tr>
<td>beans etc.)</td>
<td></td>
</tr>
<tr>
<td>Continental style breakfast (e.g. toast, bread, fruit)</td>
<td>1</td>
</tr>
</tbody>
</table>

### Lunch:

<table>
<thead>
<tr>
<th>Item</th>
<th>Preference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soup (e.g. vegetable, minestrone, tomato etc.)</td>
<td>6</td>
</tr>
<tr>
<td>Brown bread</td>
<td>3</td>
</tr>
<tr>
<td>White bread</td>
<td>1</td>
</tr>
<tr>
<td>Pizza</td>
<td>9</td>
</tr>
<tr>
<td>Sandwich (e.g. chicken / ham / tuna / egg and salad)</td>
<td>8</td>
</tr>
<tr>
<td>Hamburger / cheeseburger / sausage &amp; chips</td>
<td>6</td>
</tr>
<tr>
<td>Salad (e.g. chicken / ham / tuna / egg with lettuce, tomato, onion etc.)</td>
<td>2</td>
</tr>
<tr>
<td>Butter, margarine, salad dressing (e.g. Italian, 1000 island) or mayonnaise added to food</td>
<td>3</td>
</tr>
</tbody>
</table>

### Dinner:

<table>
<thead>
<tr>
<th>Item</th>
<th>Preference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fish (boiled, baked) or shellfish (shrimp, clams, mussels etc.)</td>
<td>1</td>
</tr>
<tr>
<td>Red meat (beef, pork or lamb as a main dish)</td>
<td>8</td>
</tr>
<tr>
<td>White meat (chicken, turkey as a main dish)</td>
<td>4</td>
</tr>
<tr>
<td>Fried chicken</td>
<td>2</td>
</tr>
<tr>
<td>Vegetables (e.g. carrots, broccoli, peas, corn, mixed vegetables)</td>
<td>9</td>
</tr>
<tr>
<td>Salad (lettuce, tomatoes, peppers, onions)</td>
<td>7</td>
</tr>
<tr>
<td>Chips and fried potatoes</td>
<td>5</td>
</tr>
<tr>
<td>Rice, pasta or other grain</td>
<td>7</td>
</tr>
<tr>
<td>Pizza</td>
<td>9</td>
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</tbody>
</table>

### Snacks:

<table>
<thead>
<tr>
<th>Item</th>
<th>Preference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fruit (apples, bananas, oranges, pears etc.)</td>
<td>3</td>
</tr>
<tr>
<td>Biscuits, muffins, pastry, cake etc.</td>
<td>2</td>
</tr>
<tr>
<td>Chocolate (e.g. Cadbury’s, M&amp;M’s) and chocolate bars (e.g. Mars, snickers) N</td>
<td>1</td>
</tr>
<tr>
<td>Salty snacks (potato crisps, tortillas etc.)</td>
<td>5</td>
</tr>
<tr>
<td>Salty snacks (popcorn, rice cakes etc.)</td>
<td>1</td>
</tr>
<tr>
<td>Dried fruit (popcorn, rice cakes etc.)</td>
<td>7</td>
</tr>
</tbody>
</table>

### Beverages:

1. Glasses of water
2. Whole milk and beverages with whole milk
<p>| | |</p>
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<tbody>
<tr>
<td>3.</td>
<td>Skim or low fat milk and beverages with skim or low fat milk</td>
</tr>
<tr>
<td>4.</td>
<td>Regular soft drinks (not diet)</td>
</tr>
<tr>
<td>5.</td>
<td>Diet soft drinks</td>
</tr>
<tr>
<td>6.</td>
<td>Tea, coffee</td>
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<tr>
<td>7.</td>
<td>Fruit juice drinks (e.g. orange, apple, cranberry, and Vitamin C enriched drinks)</td>
</tr>
<tr>
<td>8.</td>
<td>Beer, wine</td>
</tr>
<tr>
<td>9.</td>
<td>Hard liquor (tequila, gin, vodka, scotch, rum, whiskey, and liqueurs)</td>
</tr>
</tbody>
</table>
1. In front of you on the screen, there are four decks of cards, A, B, C, and D.
2. I want you to select one card at a time, by clicking on the card, from any deck you choose.
3. Each time you select a card, the colour of the card turns red or black, and the computer will tell you that you won some money. I don’t know how much money you will win. You will find out as we go along. Every time you win, the marker on the bar moves into the black.
4. Every so often, however, when you click on a card, the computer tells you that you won some money, but then it says that you lost some money too. I don’t know when you will lose, or how much you will lose. You will find out as we go along. Every time you lose, the marker on the bar moves into the red.
5. You are absolutely free to switch from one deck to the other at any time, and as often as you wish.
6. The goal of the game is to win as much money as possible, and if you can’t win, avoid losing money as much as possible.
7. You won’t know when the game will end. You must keep on playing until the computer stops.
8. I am going to give you this $2,000 credit, the black bar, to start the game. The black bar here is a reminder of how much money you borrowed to play the game, and how much money you have to pay back before we see how much you won or lost.
9. It is important to know that just like in a real card game, the computer does not change the order of the cards after the game starts. You may not be able to figure out exactly when you will lose money, but the game is fair. The computer does not make you lose money at random, or make you lose money based on the last card you picked.
10. Also, each deck contains an equal number of cards of each colour, so the colour of the cards does not tell you which decks are better in this game.
11. So you must not try to figure out what the computer is doing. All I can say is that some decks are worse than the others. You may find all of them bad, but some are worse than the others. No matter how much you find yourself losing, you can still win if you stay away from the worst decks.
12. Please treat the play money in this game as real money, and any decision on what to do with it should be made as if you were using your own money. The participant who wins the largest amount over the course of the study will win a real monetary bonus of €100.
Dear Participant,

Thank you for taking part in this study entitled “Eating habits and decision-making”. The aim of this research was to investigate the relationship between eating habits (motivations to eat & food preferences), decision-making processes and Body Mass Index (BMI).

The computer game you just completed is a measure of the ability to postpone immediate rewards in favour of long-term gain. Using this task, we hope to investigate the relationship between decision-making processes and eating habits. Such research may help in addressing barriers to weight management. Please be assured that all information provided by you will remain anonymous.

Should you have any queries relating to the research and / or your participation, please do not hesitate to contact myself, or my supervisor Dr. Hevey, as per the contact details below.

The following are contact details for psychological support, should you experience any distress over the issues raised in this project:

Ruth Yoder, Senior Clinical Psychologist, Loughlinstown Hospital, Co. Dublin.
Tel: (01) 211 5051.

Yours sincerely,

Amy Brogan
Amy Brogan B.A. (Hons.)
School of Psychology,
Trinity College Dublin,
Dublin 2.
Tel: 01 8962970
Email: broganam@tcd.ie

Dr. David Hevey, C Psychol, Reg Psychol PsSI
School of Psychology,
Trinity College Dublin,
Dublin 2.
Tel: 01 8962406
Email: heveydt@tcd.ie
Debriefing form [Control Group]:
Eating habits and decision-making

School of Psychology,
Trinity College Dublin,
Dublin 2.

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Should you have any queries relating to the research and / or your participation, please do not hesitate to contact Amy Brogan, or the supervisor of this research Dr. Hevey, as per the contact details below.

The following are contact details for psychological support, should you experience any distress over the issues raised in this project:

-For students of Trinity College: The Student Counselling Service. Tel: (01) 896 1407
-For participants outside of Trinity College: Samaritans, Tel: 1850 609 090; Aware, Tel: 1890 303 302.

Yours sincerely,

Amy Brogan

Amy Brogan B.A. (Hons.)
School of Psychology,
Trinity College Dublin,
Dublin 2.
Tel: 01 8962970
Email: broganam@tcd.ie

Dr. David Hevey, C Psychol, Reg Psychol PsSI
School of Psychology,
Trinity College Dublin,
Dublin 2.
Tel: 01 8962406
Email: heveydt@tcd.ie
Appendix 4: Gain-loss structure on the original IGT and SGT

The immediate net value of each trial and gain-loss structure in the original IGT and SGT (Chiu et al., 2008).

<table>
<thead>
<tr>
<th>IGT Serial Numbers</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>SGT</th>
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<th>B</th>
<th>C</th>
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<td>-100</td>
</tr>
<tr>
<td>28</td>
<td>-50</td>
<td>100</td>
<td>50</td>
<td>50</td>
<td>28</td>
<td>200</td>
<td>100</td>
<td>-200</td>
<td>-100</td>
</tr>
<tr>
<td>29</td>
<td>100</td>
<td>100</td>
<td>-25</td>
<td>-200</td>
<td>29</td>
<td>200</td>
<td>100</td>
<td>-200</td>
<td>-100</td>
</tr>
<tr>
<td>30</td>
<td>100</td>
<td>100</td>
<td>0</td>
<td>50</td>
<td>30</td>
<td>-1050</td>
<td>-650</td>
<td>1050</td>
<td>650</td>
</tr>
</tbody>
</table>
Note. (Left part: IGT) Deck A contains the relative high-frequency loss, while decks B, C, and D contain the high-frequency gain (net value within each trial). Decks A and B have negative net value (namely, the EV), -250 over an average of ten trials; moreover, C and D have a positive net value of +250 over an average of ten trials. (Right part: SGT) Five cumulative trials are repeated for each deck in the Soochow Gambling Task, decks A and B have high-frequency gain, while decks C and D exhibit a reversed gain-loss pattern. The task enlarges the difference between positive and negative EVs to make the difference more noticeable than in the Iowa Gambling Task. While playing this game, subjects only experienced a gain or a loss during each trial, and there was no reciprocal gain-loss within individual trials.

INFORMATION SHEET:

My name is Amy Brogan and I am a postgraduate psychology student at Trinity College, Dublin. I request your consent to participate in a study on health and decision-making. In this study you will be asked to provide some general background information on yourself (including details of your medical and weight management history) and to have your height and weight measured in order to calculate your Body Mass Index (BMI). You will be asked to complete some brief questionnaires and to perform 4 short tasks on the computer. These tasks are computerised card gambling tasks, which are designed to replicate real-life decision-making.

Participation in this study is voluntary and there are no consequences to not participating, as it will not affect your future medical care. All information provided by you will remain fully confidential. This information will be stored in accordance with the Data Protection Act. Under the Freedom of Information Act, 1997, you have the right to access your own data and can contact the researchers to get such information.

Once you have read this letter and have received satisfactory answers to any questions you may have about the study, if you decide that you would like to participate, then simply sign the consent form below. Please note that your consent sheet will be kept in a secure filing cabinet in my office in Trinity College Dublin. Finally, you are free to withdraw from the study at any time, without prejudice.

Should you require any additional information, please do not hesitate to contact myself or my supervisor Dr. David Hevey, as per the contact details below.

Yours sincerely,

Amy Brogan
Amy Brogan B.A. (Hons.)
School of Psychology,
Trinity College Dublin,
Dublin 2.
Tel: 01 8963904
Email: broganam@tcd.ie

Dr. David Hevey, C Psychol, Reg Psychol PsSI
School of Psychology,
Trinity College Dublin,
Dublin 2.
Tel: 01 8962406
Email: heveydt@tcd.ie
CONSENT FORM:

Health and decision-making

I consent to participate in this study. I am aware that participation is voluntary and that there are no consequences to non-participation. I understand that all information provided by me will remain fully confidential and that I may access this information in accordance with the Freedom of Information Act, 1997. I have been informed that I may withdraw from this study at any time without prejudice.

Print Name ____________________________

Signature of Participant: ____________________________

Date: ____________________________
GENERAL BACKGROUND INFORMATION:

PARTICIPANT NUMBER

[PLEASE PRINT IN CAPITAL LETTERS]

NAME: □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □
   First name
   □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □ □
   Surname

AGE: □ □

[PLEASE TICK BELOW]

SEX: □ Male □ Female

MARITAL STATUS: □ Married □ Never Married / Single □
   Widowed
   □ Separated / Divorced □ Cohabiting
   □ Other (please explain) ____________________________

MY EMPLOYMENT STATUS IS:

Full-time Employment □ Part-time Employment □ Student □
Unemployed □ Retired □ Other □ (Please explain) ______

THE HIGHEST EDUCATION I HAVE COMPLETED IS:

Primary □ Junior Cert □ Leaving Cert □
Diploma / Cert. Course □ Third level □

YOU WILL NOW COMPLETE A VERBAL TEST WITH THE RESEARCHER:

-WAIS: □
1. Picture Completion

**TIME LIMIT**
20 seconds each item

**REVERSE RULE**
Score of 0 on Item 6 or 7, administer Items 3-5 in reverse sequence until two consecutive perfect scores are obtained.

**DISCONTINUE RULE**
3 consecutive scores of 0

**Examinee Response to Item**
- Names object pictured rather than missing part
- Mentions part of the picture that is off the page (e.g., the legs of the man in Item 14)
- Mentions an unessential missing part

**Examiner Query**
(Say each query only once for the entire administration)
- Yes, but what is missing?
- Something is missing in the picture. What is it that is missing?
- Yes, but what is the most important part that is missing?

If the examinee responds correctly after any of the above queries, score 1 point for the response.

<table>
<thead>
<tr>
<th>Item</th>
<th>Response</th>
<th>Score (0 or 1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Comb</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Table</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Face</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Briefcase</td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Train</td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Door</td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Glasses</td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>Pitcher</td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>Pliers</td>
<td></td>
</tr>
<tr>
<td>10.</td>
<td>Leaf</td>
<td></td>
</tr>
<tr>
<td>11.</td>
<td>Pie</td>
<td></td>
</tr>
<tr>
<td>12.</td>
<td>Jogging</td>
<td></td>
</tr>
<tr>
<td>13.</td>
<td>Fireplace</td>
<td></td>
</tr>
<tr>
<td>14.</td>
<td>Mirror</td>
<td></td>
</tr>
<tr>
<td>15.</td>
<td>Chair</td>
<td></td>
</tr>
<tr>
<td>16.</td>
<td>Roses</td>
<td></td>
</tr>
<tr>
<td>17.</td>
<td>Knife</td>
<td></td>
</tr>
<tr>
<td>18.</td>
<td>Boot</td>
<td></td>
</tr>
<tr>
<td>19.</td>
<td>Basket</td>
<td></td>
</tr>
<tr>
<td>20.</td>
<td>Clothing</td>
<td></td>
</tr>
<tr>
<td>21.</td>
<td>Lockers</td>
<td></td>
</tr>
<tr>
<td>22.</td>
<td>Cow</td>
<td></td>
</tr>
<tr>
<td>23.</td>
<td>Tennis Shoes</td>
<td></td>
</tr>
<tr>
<td>24.</td>
<td>Woman</td>
<td></td>
</tr>
<tr>
<td>25.</td>
<td>Barn</td>
<td></td>
</tr>
</tbody>
</table>

Total Raw Score (Maximum = 25)

2. Vocabulary

**REVERSE RULE**
Score of 0 on Item 4 or 5, administer Items 1-3 in reverse sequence until two consecutive perfect scores are obtained.

**DISCONTINUE RULE**
6 consecutive scores of 0

**SCORING RULE**
All Items 0, 1, or 2 pts.

<table>
<thead>
<tr>
<th>Item</th>
<th>Response</th>
<th>Score (0, 1 or 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Bed</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Ship</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Penny</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Winter</td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Breakfast</td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Repair</td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Assemble</td>
<td></td>
</tr>
</tbody>
</table>

207
## 2. Vocabulary (continued)

<table>
<thead>
<tr>
<th>Item</th>
<th>Response</th>
<th>Score (0, 1, or 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>8. Yesterday</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Terminate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Consume</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Sentence</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Confide</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. Remorse</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14. Ponder</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15. Compassion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>16. Tranquil</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17. Sanctuary</td>
<td></td>
<td></td>
</tr>
<tr>
<td>18. Designate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>19. Reluctant</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20. Colony</td>
<td></td>
<td></td>
</tr>
<tr>
<td>21. Generate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>22. Ballad</td>
<td></td>
<td></td>
</tr>
<tr>
<td>23. Pout</td>
<td></td>
<td></td>
</tr>
<tr>
<td>24. Plagiarize</td>
<td></td>
<td></td>
</tr>
<tr>
<td>25. Diverse</td>
<td></td>
<td></td>
</tr>
<tr>
<td>26. Evolve</td>
<td></td>
<td></td>
</tr>
<tr>
<td>27. Turgible</td>
<td></td>
<td></td>
</tr>
<tr>
<td>28. Fortitude</td>
<td></td>
<td></td>
</tr>
<tr>
<td>29. Epic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30. Audacious</td>
<td></td>
<td></td>
</tr>
<tr>
<td>31. Ominous</td>
<td></td>
<td></td>
</tr>
<tr>
<td>32. Encumber</td>
<td></td>
<td></td>
</tr>
<tr>
<td>33. Trade</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Total Raw Score (Maximum = 66)
(Includes credit for items on previous page)
GENERAL HEALTH QUESTIONNAIRE
(GHQ-12)

Please read this carefully.

We should like to know if you have had any medical complaints and how your health has been in general, over the last few weeks. Please answer ALL the questions simply by underlining the answer which you think most nearly applies to you. Remember that we want to know about present and recent complaints, not those that you had in the past.

It is important that you try to answer ALL the questions.

Thank you very much for your co-operation.

Have you recently . . .

1. been able to concentrate on whatever you're doing?
   - Better than usual
   - Same as usual
   - Less than usual
   - Much less than usual

2. lost much sleep over worry?
   - Not at all
   - No more than usual
   - Rather more than usual
   - Much more than usual

3. felt that you are playing a useful part in things?
   - More so than usual
   - Same as usual
   - Less useful than usual
   - Much less useful than usual

4. felt capable of making decisions about things?
   - More so than usual
   - Same as usual
   - Less so than usual
   - Much less than usual

5. felt constantly under strain?
   - Not at all
   - No more than usual
   - Rather more than usual
   - Much more than usual

6. felt you couldn't overcome your difficulties?
   - Not at all
   - No more than usual
   - Rather more than usual
   - Much more than usual

7. been able to enjoy your normal day-to-day activities?
   - More so than usual
   - Same as usual
   - Less than usual
   - Much less than usual

8. been able to face up to your problems?
   - More so than usual
   - Same as usual
   - Less so than usual
   - Much less able

9. been feeling unhappy and depressed?
   - Not at all
   - No more than usual
   - Rather more than usual
   - Much more than usual

10. been losing confidence in yourself?
    - Not at all
    - No more than usual
    - Rather more than usual
    - Much more than usual

11. been thinking of yourself as a worthless person?
    - Not at all
    - No more than usual
    - Rather more than usual
    - Much more than usual

12. been feeling reasonably happy, all things considered?
    - More so than usual
    - About same as usual
    - Less so than usual
    - Much less than usual
**Barrett Impulsivity Scale (BIS-15)**

*For each statement, circle a number to the right to indicate how well it describes you.*

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Rarely/Neve</th>
<th>Occasionally</th>
<th>Often</th>
<th>Almost always</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>I plan tasks carefully.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>I do things without thinking.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>I don't &quot;pay attention.&quot;</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>I concentrate easily.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>I save money on a regular basis.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>I squirm at plays or lectures.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7</td>
<td>I am a careful thinker.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8</td>
<td>I plan for job security.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9</td>
<td>I say things without thinking.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10</td>
<td>I act &quot;on impulse.&quot;</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>11</td>
<td>I get easily bored when solving thought problems.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>12</td>
<td>I act on the spur of the moment.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>13</td>
<td>I buy things on impulse</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>14</td>
<td>I am restless at lectures or talks.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>15</td>
<td>I plan for the future</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
GENRAL MEDICAL HISTORY:

1. Do you currently have any of the following medical conditions?
   - Heart Disease □
   - Diabetes □
   - Arthritis □
   - Sleep Apnea □
   - Other □ (Please specify)___________________________
   - None □

   1.(b) If yes to any of the above, how long have you had this condition?

2. Are you currently being treated for depression?
   □ Yes    □ No

   2.(b) If yes, how long are you in treatment?

3. Are you currently being treated for any other psychological condition? (e.g. ADHD)
   □ Yes    □ No

   If yes, please specify

4. Have you been diagnosed with an eating disorder (e.g. bulimia nervosa, binge eating disorder, anorexia nervosa) in the past 6 months?
   □ Yes    □ No

   If yes, please specify

5. Are you currently on any medication?
   □ Yes    □ No

   If yes, please specify:

HISTORY OF WEIGHT MANAGEMENT:

1. Are you currently participating in a weight management service?
   □ Yes    □ No (if no skip to question 2)

   1.(b) If yes, what service are you currently in?
   - The National Weight Management Clinic, Loughlinstown □
   - Private Organisation (e.g. Weight Watchers; Curves) □
   - Other, please provide details: _____________________________

2. Are you currently taking prescription weight loss drugs?
   □ Yes    □ No

3. Are you satisfied with your current body weight?
   □ Yes    □ No

   Body Measurements:
   [Details of your height & weight will now be recorded by the researcher]
   HEIGHT: ________ Metres    WEIGHT: ________ Kgs

YOU WILL NOW COMPLETE A TASK ON THE COMPUTER

211
POST-TASK QUESTIONNAIRE

1. On which deck do you experience the highest **frequency gain**? (i.e. most often)
   
   A    B    C    D    [Please circle]

2. On which deck do you experience the highest **frequency loss**? (i.e. most often)
   
   A    B    C    D    [Please circle]

3. On which deck do you **earn** the **largest overall** amount of money?
   
   A    B    C    D    [Please circle]

4. On which deck do you **lose** the **largest overall** amount of money?
   
   A    B    C    D    [Please circle]

5. Which deck do you **prefer most** to choose?
   
   A    B    C    D    [Please circle]

6. Which deck do you **dislike most** to choose?
   
   A    B    C    D    [Please circle]
POST-TASK QUESTIONNAIRE

1. On which deck do you experience the highest frequency gain? (i.e. most often)
   A   B   C   D   [Please circle]

2. On which deck do experience the highest frequency loss? (i.e. most often)
   A   B   C   D   [Please circle]

3. On which deck do you earn the largest overall amount of money?
   A   B   C   D   [Please circle]

4. On which deck do you lose the largest overall amount of money?
   A   B   C   D   [Please circle]

5. Which deck do you prefer most to choose?
   A   B   C   D   [Please circle]

6. Which deck do you dislike most to choose?
   A   B   C   D   [Please circle]
Appendix 7: Protocol for food diary study

Feidhmeannacht na Seirbhise Slainte
Health Service Executive

INFORMATION BOOKLET & 5 DAY
REFLECTIVE DIARY ON YOUR EATING BEHAVIOUR

NAME: _____________________________

DATE OF BIRTH: ____________________
INFORMATION SHEET:

A Reflective Diary on your Eating Behaviour

My name is Amy Brogan and I am a postgraduate psychology student at Trinity College, Dublin. I am conducting research into eating behaviour and the factors which may influence eating across the day. I ask for your consent to complete this 5-day reflective diary together with the food diary you are already completing for the dietician at the weight management clinic. As you will see the diary starts on a Friday and finishes on a Tuesday (5 days inclusive), so you will finish it half-way through your food diary for the clinic.

You should try to record your food and drinks in your food diary as you eat/drink them, do not wait until the end of the day to write things down. The reflective diary is to be completed once at the end of each day, and will ask you to reflect on your eating throughout that day. It will focus on motivations for eating, and any significant events or factors associated with your food intake. You will be asked to comment on your overall mood each day and any physical activity you took. Finally, there will be an opportunity for you to rate your overall satisfaction with the day.

Participation in this diary is voluntary and there are no consequences to not participating, as it will not affect your future medical care. All information provided by you will remain fully confidential. This information will be stored in accordance with the Data Protection Act. Under the Freedom of Information Act, 1997, you have the right to access your own data and can contact the researchers to get such information.

Once you have read this letter, if you decide that you would like to participate, then simply sign the consent form on the next page and start your reflective diary on the same day as your food diary. You are free to withdraw from the study at any time, without prejudice.

If you decided not to participate, please return the unused diary with your food diary.

Should you require any additional information, please do not hesitate to contact myself or my supervisor Dr. David Hevey, as per the contact details below.

Yours sincerely,

Amy Brogan B.A. (Hons.)
School of Psychology,
Trinity College Dublin,
Dublin 2.
Tel: 01 8962970
Email: broganam@tcd.ie

Dr. David Hevey, C Psychol, Reg Psychol PsSI
School of Psychology,
Trinity College Dublin,
Dublin 2.
Tel: 01 8962406
Email: heveydt@tcd.ie
CONSENT FORM:

Eating behaviour diary

I consent to participate in this study which requests that I complete a 5-day eating behaviour diary. I am aware that participation is voluntary and that there are no consequences to non-participation. I understand that all information provided by me will remain anonymous and fully confidential and that I may access this information in accordance with the Freedom of Information Act, 1997. I have been informed that I may withdraw from this study at any time without prejudice.

Print Name ________________________________

Signature of Participant: ________________________________

Date: ________________________________

Amy Brogan B.A. (Hons.)
School of Psychology,
Trinity College Dublin,
Dublin 2.
Tel: 01 8962970
Email: broganam@tcd.ie

Dr. David Hevey, C Psychol, Reg Psychol PsSI
School of Psychology,
Trinity College Dublin,
Dublin 2.
Tel: 01 8962406
Email: heveydt@tcd.ie
PLEASE COMPLETE THIS SHORT QUESTIONNAIRE:

Name: ................................................................. .................................................................

First Name ___________________________ Last Name ______________________________

Sex: Male □ Female □ Age: □ □ □ □

Tel: Home: ___________________________ Mobile: ____________________________

My marital status is:

Married □ Never Married □ Widowed □ Cohabiting □

Separated/Divorced □ Other □

My employment status is:

Full-time Employment □ Part-time Employment □ Student □

Unemployed □ Retired □ Other □ (Please explain)__________________________

The highest education I have completed is:

Primary □ Junior Cert □ Leaving Cert □

Diploma / Cert. Course □ Third level □

My most recent weight is _________ Kg or ________ St _______ Lbs

DIARY INSTRUCTIONS

Please try to do the following:

1. Complete the diary every night, last thing before you go to bed.
2. Record the date and time of each entry, at the top of each page.
3. Fill in every box, and do not leave boxes blank.
4. Be as honest as possible in completing the diary, as accurate information is crucial in this research. Only complete the diary at the end of each day, while reflecting over your dietary behaviour that day.
5. As the diary is short (5 days) try not to miss a day. However, if you do miss a day, please leave that day blank and do not try to complete it at a later date.
1. YOUR MOOD TODAY...

This scale consists of a number of words that describe different feelings and emotions. Indicate to what extent you have felt this way today. Read each item and then mark the appropriate answer in the space next to that word. Use the following scale to record your answers.

| 1 = Very slightly or not at all | 2 = A little | 3 = Moderately | 4 = Quite a bit | 5 = Extremely |

Please fill in EVERY BOX e.g. Upset 3; Afraid 1

- Upset  □
- Hostile  □
- Alert  □
- Ashamed  □
- Inspired  □
- Nervous  □
- Determined  □
- Attentive  □
- Afraid  □
- Active  □

2. YOUR EATING BEHAVIOUR TODAY...

Please think about your eating behaviour today and answer the questions using the following scale:

| 1 = Never | 2 = Seldom | 3 = Sometimes | 4 = Often | 5 = Very often |

Please fill in every box: e.g. did you have a desire to eat if you were irritated? 3

*For questions 1–6, if you did not have that feeling today you may respond not applicable (N/a)

1) Did you have a desire to eat if you were irritated?

2) Did you have a desire to eat if you were depressed or discouraged?

3) Did you get the desire to eat if you were anxious, worried, or tense?

4) Did you have a desire to eat if things were going against you or if things had gone wrong?

5) Did you have a desire to eat if you were feeling lonely?

6) Did you have a desire to eat if you were bored or restless?

7) Did you try to eat less at mealtimes than you would like to eat?

8) How often did you refuse food or drink offered because you are concerned about your weight?

9) Did you watch exactly what you ate?

10) Did you deliberately eat less in order not to become heavier?

11) How often did you try not to eat between meals because you were watching your weight?
12) If food tasted good to you, did you eat more than usual? □

13) If you saw or smelled something delicious, did you have a desire to eat it? □

14) If you had something delicious to eat, did you eat it straight away? □

15) If you walked past a snackbar or a café, did you have the desire to buy something delicious? □

16) If you saw others eating, did you also have the desire to eat? □

3. YOUR ASSESSMENT OF TODAY...
Using the numbers below please indicate how much you achieved the following today:

<table>
<thead>
<tr>
<th>1 = not at all</th>
<th>2 = very little</th>
<th>3 = a little</th>
<th>4 = a lot</th>
<th>5 = completely</th>
</tr>
</thead>
</table>

1. Eating an appropriate amount of food? □ [Insert 1 – 5]

2. A balance across the types of food (e.g. starches; proteins, fats) you ate? □ [Insert 1–5]

3. Taking an appropriate level of physical activity? □ [Insert 1 – 5]

Please describe any activity and its duration:

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

4. To what extent did any significant events today affect or disrupt your eating habits? □
[Insert 1 – 5]. Please explain any such events:

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

5. Do you think you overate today in any single sitting? Yes / No [please circle]
If yes, did you experience a loss of control during this episode? Yes / No [please circle]

6. How satisfied are you with your eating behaviour today? □ [Insert 1 – 5]

7. Reflecting on your eating behaviour today, is there anything you would change about it?
Yes / No [please circle]
If yes, please explain:

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

THANK YOU FOR COMPLETING DAY 1

**This was repeated for Saturday – Tuesday**

220
CONTACT DETAILS FORM

We occasionally seek participants for our research projects. If you consent to be contacted to participate in future research please tick the box and provide us with your contact details below □

Name: .................................................................

Address Line 1: ............................................................
Address Line 2: ............................................................
Address Line 3: ............................................................
Address Line 4: ............................................................

Telephone [Home]: ............................................................
(Area Code)

Telephone [Mobile]: ............................................................

Email: .................................................................
Dear participant,

Thank you for taking part in this study entitled “emotion regulation & eating behaviour”. The overall aim of this research was to investigate your eating habits over a 5-day period, together with the information in your food diary which you completed for the weight management programme.

This research will examine the factors or decision making strategies which contributed to days when an appropriate amount of food was eaten, and factors which may have lead to an inappropriate or excessive amount of food being consumed. This will be achieved with reference to current psychological theory in this area. Particular focus will be give to the role of emotion or mood in regulating food intake.

It is hoped that this research will inform behavioural strategies for weight management and the barriers to achieving this. Once again, please be assured that all information provided by you will remain anonymous and confidential. Should you have any queries relating to the research and / or your participation, please do not hesitate to contact myself, or my supervisor Dr. Hevey, as per the contact details below.

The following are contact details for psychological support, should you experience any distress over the issues raised in this project:

- Ruth Yoder, Senior Clinical Psychologist, Loughlinstown Hospital, Co. Dublin; Tel: (01) 211 5051
- The Samaritans; Tel: 1850 609 090
- Aware; Tel: 1890 303 302

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