



UNIVERSITY OF
LIVERPOOL

Psychological determinants and consequences
of self-perceived food addiction

Thesis submitted in accordance with the requirements of

The University of Liverpool

For the degree of Doctor of Philosophy

By

Helen Kate Ruddock

December 2016

Author's Declaration

This thesis is the result of my own work. The material contained in this thesis has not been presented, nor is currently being presented, either in part or wholly for any other degree qualification.

I designed this research in conjunction with my supervisors and was responsible for data collection, analysis and write-up.

Abstract

Introduction: Many people believe that obesity is caused by an addiction to food. However, within the scientific community, there is ongoing debate surrounding the validity of the food addiction concept, and an operational definition of food addiction is yet to be established. An aim of the current thesis was to identify behaviours and cognitions which characterise addictive patterns of eating. To do so, Chapters 2-4 explored the characteristics of individuals who *perceive* themselves to be addicted to food (i.e. self-perceived food addicts). Based on these findings, Chapter 5 presents the development of a novel assessment tool for addictive-like eating. A second aim of the thesis was to explore food addiction beliefs from a psychosocial perspective. Specifically, Chapter 6 examined the consequences of food addiction beliefs on subsequent eating, and Chapter 7 explored whether the concept of food addiction may be used to alleviate eating-related guilt by implying that eating is beyond personal control. **Methods:** A combination of qualitative and experimental techniques were used to establish the cognitive and behavioural features of self-perceived food addiction. Chapter 2 consisted of a brief questionnaire which inductively explored beliefs about the manifestations of food addiction within the lay public. These findings were extended in Chapters 3 and 4 which experimentally tested whether self-perceived food addicts would demonstrate increased food reward and attention to high-fat food cues (using an eye-tracking paradigm), compared to those who do not perceive themselves as food addicts. Chapter 5 used exploratory and confirmatory factor analyses to develop a novel tool for the assessment of addictive eating (i.e. the Addictive Eating Behaviour Scale, AEBS). To address the second aim of the thesis, experimental techniques were used to manipulate participants' beliefs about their levels of food addiction (Chapter 6) and feelings of eating-related guilt (Chapter 7). The effects of these beliefs on subsequent food intake (Chapter 6) and food addiction attributions (Chapter 7) were then examined. **Results:** Findings from Chapter 2 suggested that self-perceived food addicts find food particularly rewarding and may be particularly likely to overeat. Consistent with these findings, Chapter 3 found that self-perceived food addicts demonstrated increased desire-to-eat for a range of food, and consumed more of a high-fat food during *ad libitum* access, compared to self-perceived non-addicts. However, self-perceived food addicts did not show any increased attentional bias to food cues compared to non-addicts (Chapter 4). The AEBS (Chapter 5) consisted of two subscales: 1) unhealthy eating/low self-control, and 2) overeating/weight gain. This scale predicted variance in BMI beyond that accounted for by an existing measure of food addiction. With regard to the second aim of the thesis, Chapter 6 found that those who were told they had high levels of 'food addiction' consumed fewer calories compared to those who were told they had 'low' or 'average' food addiction. This was mediated by increased dietary concern and a reduction in the amount of time spent tasting high-fat foods. Finally, Chapter 7 found no effect of manipulating eating-related guilt on food addiction beliefs; however, across the whole sample, higher levels of guilt correlated with an increased tendency to attribute eating to the foods' addictiveness. **Conclusions:** Overall, these findings suggest that self-perceived food addiction is characterised by several core behaviours, and that perceiving oneself to be a food addict may be helpful for those attempting to reduce their intake of certain foods, in the short-term at least. Future research should establish whether the AEBS captures food reward and calorie intake beyond that accounted for by established measures of aberrant dietary behaviour. Research should also examine the effects of food addiction beliefs on longer-term patterns of eating.

Acknowledgements

Thank you to my primary supervisor, Dr Charlotte Hardman, for your invaluable help and support over the past 3 years. I feel so lucky to have received supervision from such an inspirational researcher, and I really hope I get the opportunity to work with you again in the future. Thank you also to Professor Jeff Brunstrom for putting Charlotte in touch with me in the first place – without this, I might never have had the opportunity to conduct this research. I am so grateful. I am also incredibly grateful to my secondary supervisor, Professor Matt Field, whose advice has played an imperative role in the development of this thesis. I would also like to thank Dr Paul Christiansen, Dr Eric Robinson, Dr Joanne Dickson, Dr Andrew Jones, and Professor Jason Halford, for their advice and input on the published (or soon to be published) chapters of this thesis, and thank you to Dr Pawel Jedras for getting me to grips with the eye-tracker.

I would also like to thank everyone who gave up their time to participate in the studies included in this thesis; without their input, none of this research would have been possible.

Thank you to everyone who kept me entertained during hours spent in the lab - Una, Cat, Graeme, Stefano and Hannah (et al.) - testing was never dull with you around.

Finally, I would like to thank my family and friends for their unwavering support. In particular, I would like to thank my partner, Tammy Thiele, for her patience and encouragement, especially throughout the past few stressful months of thesis writing!

Thank you all so much.

Table of Contents

Author's Declaration	2
Abstract.....	3
Acknowledgements	4
List of Tables	10
List of Figures.....	12
List of abbreviations	15
Chapter 1: Introduction	16
1.1. The role of physiological, hedonic, and cognitive mechanisms in appetite control 17	
1.1.1. Physiological control.....	17
1.1.2. Hedonic control and food reward.....	20
1.1.3. Cognitive control of food intake.....	25
1.2. The case for food addiction.....	28
1.2.1. The Yale Food Addiction Scale	29
1.2.2. Prevalence of food addiction.....	30
1.2.3. Distinctions between FA and binge eating.....	31
1.2.4. Food addiction: Weighing up the evidence.....	32
1.2.5. Food addiction as a behavioural addiction.....	41
1.2.6. Food addiction as a self-serving attribution: psychosocial account.....	42
1.2.7. The consequences of food addiction beliefs	44
1.3. The current thesis	45
Aim 1: To establish a cognitive and behavioural profile of self-perceived 'food addiction' (Chapters 2-5).	46
Aim 2. To examine the causes and consequences of food addiction beliefs	47
Chapter 2: Exploring the causal attributions of self-perceived food addiction. 49	
2.1. Overview.....	49
2.2. Abstract	49
2.3. Introduction	50
2.4. Method.....	52
2.4.1. Participants.....	52
2.4.2. Materials and Procedure.....	52
2.4.3. Data analysis	53

2.5. Results	55
2.5.1. Quantitative data	55
2.5.2. Qualitative data	56
2.6. Discussion	62
Chapter 3: Food reward and calorie intake in self-perceived food addicts	67
3.1. Overview	67
3.2. Abstract	67
3.3. Introduction	68
3.4. Method	71
3.4.1. Participants	71
3.4.2. Materials and measures	72
3.4.3. Procedure.....	76
3.4.4. Data analysis	77
3.5. Results	78
3.5.1. Participant characteristics.....	78
3.5.2. Measures of food reward.....	79
3.5.3. Calorie intake	81
3.5.4. Food liking and hunger	81
3.5.5. Regression analyses	82
3.6. Discussion	84
Chapter 4: Attentional bias to food cues in self-perceived food addicts	89
4.1. Overview	89
4.2. Abstract	89
4.3. Introduction	90
4.4. Method	93
4.4.1. Participants	93
4.4.2. Measures and Materials.....	94
4.4.3. Procedure.....	97
4.4.4. Data analysis	98
4.5. Results	100
4.5.1. Participant characteristics.....	100
4.5.2. Appetite ratings	101
4.5.3. Attentional bias	102
4.5.4. Exploratory analyses: Desire-to-eat	103
4.5.5. Predictors of chocolate intake	105
4.6. Discussion	105

Chapter 5: The Development and Validation of the Addictive Eating Behaviour

Scale.....	110
5.1. Overview.....	110
5.2. Abstract	110
5.3. Introduction	111
5.4. Method.....	115
5.4.1. Participants.....	115
5.4.2. Measures	115
5.4.3. Procedure.....	117
5.4.4. Data analysis	118
5.5. Results.....	120
5.5.1. Pre-analysis checks and participant characteristics.....	120
5.5.2. Exploratory Factor Analysis (group 1)	120
5.5.3. Internal consistency and descriptives (group 1).....	121
5.5.4. Internal consistency and descriptives (group 2).....	122
5.5.5. Confirmatory factor analysis (group 2).....	122
5.5.6. Convergent and Divergent validity (groups 1 and 2).....	124
5.5.7. Incremental validity (groups 1 and 2).....	125
5.6. Discussion	126
Chapter 6: The effects of believing in food addiction on eating behaviour.....	130
6.1. Overview.....	130
6.2. Abstract	130
6.3. Introduction	131
6.4. Study 1 Method.....	133
6.4.1. Overview of the study method	133
6.4.2. Participants.....	134
6.4.3. Measures and materials	134
6.4.4. Procedure.....	137
6.4.5. Data analysis	138
6.5. Study 1 Results.....	139
6.5.1. Participant characteristics.....	139
6.5.2. Manipulation check	140
6.5.3. Food intake.....	140
6.5.4. Supplementary analyses	141
6.6. Interim Discussion	141
6.7. Study 2 Method.....	142

6.7.1. Overview of method.....	142
6.7.2. Participants.....	143
6.7.3. Measures.....	143
6.7.4. Procedure.....	143
6.7.5. Data analysis.....	144
6.8. Study 2 Results.....	147
6.8.1. Participant characteristics.....	147
6.8.2. Manipulation check.....	148
6.8.3. Food intake.....	148
6.8.4. Dietary concern and time taken.....	149
6.8.5. Mediation analyses.....	151
6.8.6. Self-control and Dieting Intentions.....	152
6.8.7. Supplementary analyses.....	152
6.9. Discussion.....	154
Chapter 7: The effect of eating-related guilt on food addiction attributions and snack choice.....	157
7.1. Overview.....	157
7.2. Abstract.....	157
7.3. Introduction.....	158
7.4. Method.....	160
7.4.1. Overview of study method.....	160
7.4.2. Participants.....	161
7.4.3. Measures and materials.....	162
7.4.4. Procedure.....	166
7.4.5. Data analysis.....	168
7.5. Results.....	169
7.5.1. Manipulation check.....	170
7.5.2. Self-perceived food addiction (hypothesis 1).....	171
7.5.3. Addictiveness attribution ranking (hypothesis 2).....	172
7.5.4. Snack selection (hypothesis 3).....	172
7.6. Discussion.....	174
Chapter 8: General Discussion.....	179
8.1. Overview of aims.....	179
8.2. Establishing a cognitive and behavioural profile of ‘food addiction’.....	179
8.2.1. Food reward, attentional bias, and calorie consumption in self-perceived food addicts.....	180

8.2.2. A novel approach to the assessment of addictive eating.....	184
8.3. What are the causes and consequences of food addiction beliefs?.....	185
8.3.1. Food addiction as a helpful label	185
8.3.2. Food addiction as a self-serving attribution	187
8.4. Theoretical implications and directions for future research.....	189
8.5. Limitations	192
8.6. Overall conclusion	193
References.....	194
Appendix A: Addictive eating behaviour scale (Chapter 5)	233
Appendix B: Mood and taste ratings (Chapter 6)	235
Appendix C: Supplementary results (Chapter 7)	236

List of Tables

Table 1.1. *Criterion for substance-dependence (DSM-IV) and substance-related and addictive disorders (DSM-V).*

Table 2.1. *Frequency (percentages in parentheses) of self-perceived food addicts in the information and no information conditions.*

Table 2.2. *Participant characteristics for self-perceived food addicts (n=59) and self-perceived non-addicts (n=125). Values are means (SDs) unless otherwise stated*

Table 3.1. *Descriptive statistics of sample by food addiction group. Values are means with standard deviations in parentheses.*

Table 3.2. *Means (standard deviations) for the three measures of food reward, for self-perceived food addicts and non-addicts, before and after consumption of the lunch meal. NA = not applicable*

Table 3.3. *Results of regression analysis with measures of dietary restraint and disinhibition in step 1 and self-perceived food addiction in step 2. The dependent variable was total calories consumed.*

Table 3.4. *Results of regression analysis with measures of dietary restraint and disinhibition in step 1 and self-perceived food addiction in step 2. The dependent variable was mean overall DtE ratings (collapsed across conditions and foods).*

Table 4.1. *Participant characteristics in the hungry and satiated conditions. Unless otherwise stated, values are means with standard deviations in parentheses.*

Table 4.2. *Correlation coefficients between dependent variables. Values were collapsed across conditions (hungry and satiated). Hunger and DtE ratings were taken at T2 (i.e. just prior to the eye-tracking task) *p<.05, **p<.001*

Table 4.3. *Output from linear regression model of variables predicting chocolate intake(g). Values for hunger, fullness, and DtE were taken at T3 (i.e. just prior to ad libitum intake). **Significant at p<.001.*

Table 5.1. *Characteristics of participants in each group. Values in parentheses represent the standard deviation (SD) of the mean.*

Table 5.2. *Factors, items, and factor loadings*

Table 5.3. *AEBS total and subscale scores for each of the three groups. Values are means with standard deviations in parentheses*

Table 5.4. *Descriptive statistics and correlations with AEBS (n = 513)*

Table 5.5. *Results of regression analysis with YFAS symptom count step 1 and AEBS in step 2. The dependent variable was BMI*

Table 6.1. *Participant characteristics and appetite ratings in each condition. Values are means with standard deviations in parentheses.*

Table 6.2. *Participant characteristics and appetite ratings in each condition. Values are means with standard deviations in parentheses.*

Table 6.3. *Indirect effects of condition on calorie intake via dietary concern and time taken to complete the taste task.*

Table 7.1. *A worked example of the bogus calorie feedback provided to participants, in each condition, based on a participant's estimated intake of 500Kcals.*

Table 7.2. *Participant characteristics, appetite ratings, and estimated and actual calorie intake, in each condition. Values are means with standard deviations in parentheses*

Table 7.3. *Mean (standard deviations) self-perceived food addiction rating, and rank assigned to the 'foods were addictive' attribution, in each of the three conditions.*

Table A1. *Results from MANOVA with eating attributions as dependent variables, and condition as the independent variable.*

List of Figures

Figure 1.1. Model of energy balancing and appetite, adapted from Rogers & Brunstrom (2016). The water in the saucepan represents recent meal consumption, which has a strong and acute effect on appetite (represented by the thick arrow). The water in the bathtub represents stored energy, which has a relatively weak and chronic on appetite (represented by the thinner arrow).

Figure 1.2. Homeostatic and non-homeostatic factors influencing food intake (adapted from Berthoud, 2004). Homeostatic mechanisms are thought to exert weak control over food intake, relative to non-homeostatic factors, when food is easily available and energy stores replete.

Figure 1.3. A continuum of eating with energy balance (or ‘homeostatic eating’) represented at one end, and food addiction (i.e. the most severe form of non-homeostatic eating) at the other. Adapted from Davis (2013) copyrighted©2013 Caroline Davis, Creative Commons Attribution License, <http://creativecommons.org/licenses/by/3.0/>.

Figure 1.4. Thesis overview. Thesis aims are presented in the orange boxes, chapter headings are in purple boxes, and the individual aims of each chapter are presented in the blue boxes.

Figure 2.1. Codes and sub-themes assigned to each theme for self-perceived food addicts. Themes are numbered and in bold, sub-themes are in non-italics, and codes are bullet-pointed and provided in italics.

Figure 2.2. Codes and sub-themes assigned to each theme for self-perceived non-addicts. Themes are numbered and in bold, sub-themes are in non-italics, and codes are bullet-pointed and provided in italics.

Figure 3.1. Flow chart of the study procedure

Figure 3.2. Number of calories consumed from chocolate, grapes, and total calories consumed, by self-perceived food addicts and non-addicts.

Figure 3.3. Ratings of liking (panel A), and hunger (panel B) for self-perceived food addicts and non-addicts before and after the lunch meal. Liking ratings were averaged across all four test.

Figure 4.1. Order and duration of screen presentation in the eye-tracking task during a single trial. The task consisted of 120 trials and each trial type (i.e. 100%, 50%, 0%) was presented 40 times.

Figure 4.2. Flow chart of the study procedure

Figure 4.3. Ratings of hunger, fullness, and desire-to-eat chocolate at each time-point for hungry (Panel A) and satiated (Panel B) conditions. Values are means and standard errors.

Figure 4.4. Mean duration bias as a function of expectancy information and desire-to-eat chocolate

Figure 5.1. Factor model of AEBS with standardized factor loadings (i.e. values corresponding to one-way arrows), error terms (circled values), and covariances (values corresponding to two-way arrows).

Figure 6.1. Flow-chart of the Study 1 procedure.

Figure 6.2. Mean calories consumed from chocolate and crisps as a function of condition. **Significant between-condition difference at $p < .01$. Error bars represent standard error of the mean.

Figure 6.3. Flow chart of the Study 2 procedure.

Figure 6.4. Schematic representation of the hypothesised indirect effect of condition on calorie intake via dietary concern and time-taken (pathway c). The model also calculated the effects of condition on calorie intake via: (a) dietary concern, and (b) time-taken to complete the taste-task.

Figure 6.5. Mean calories consumed as a function of condition (high- addiction, low-addiction, or average-addiction) and food type (chocolate and crisps). *Significant at $p < .05$. Error bars represent standard error of the mean.

Figure 6.6. Dietary concern ratings following the food addiction feedback as a function of condition. ** $p < .001$. * $p < .05$.

Figure 6.7. Time taken to complete the taste and rate task as a function of condition. * $p < .05$.

Figure 6.8. Serial mediation analysis with High- vs- Average condition comparison as the predictor variable, calorie intake as the outcome variable, and eating behaviour concern and time-taken as first and second mediators, respectively. Values are unstandardized regression coefficients (SEs) and associated p-values. *Significant at $p < .05$., **Significant at $p < .001$. Bracketed association = direct effect after controlling for dietary concern and time taken.

Figure 6.9. Mean hunger ratings before and after the taste task as a function of condition

Figure 7.1. Overview of study procedure in sessions 1 and 2

Figure 7.2. Actual bogus datasheet that was given to a participant in the high-guilt condition. In this instance, the participant had estimated she had eaten 700 Kcals. Based on this estimate, the participant was led to believe she had eaten 1069 Kcals, and that previous bogus participants had eaten an average of 331 Kcals. Details of previous bogus participants are provided in rows 1-12. Details of the current participant are shown in row 13.

Figure 7.3. Mean dietary concern and guilt ratings by condition. *significant at $p < .01$, **significant at $p < .001$

Figure 7.4. Mean temptingness rank (1=most tempting, 6=least tempting) of snack taken in each of the three conditions. Median (mdn) and range values are also provided for each condition. * $p < .01$.

Figure 8.1. A proposed self-perpetuating relationship between self-perceived food addiction and food intake. Belief that one is a food addict leads to an initial restriction of high-calorie or unhealthy foods. This restriction increases cravings and eventually leads to overconsumption of restricted foods. This may reinforce perceptions of oneself as a 'food addiction'.

List of abbreviations

AEBS – Addictive Eating Behaviour Scale

BED – Binge Eating Disorder

BES – Binge Eating Scale

BMI – Body Mass Index

DSM – Diagnostic Statistical Manual

EAT-26 – Eating Attitudes Trouble

EES – Emotional Eating Scale

IAT – Implicit Association Task

Kcal – Kilocalories

NAc – Nucleus Accumbens

RAPI – Rutgers Alcohol Problem Index

SD – Standard deviation

SST – Stop-signal task

TFEQ – Three Factor Eating Questionnaire

VAS – Visual Analogue Scale

YFAS – Yale Food Addiction Scale

Chapter 1: Introduction

Worldwide rates of obesity have more than doubled in the past three decades, with approximately 1.9 billion people classified as overweight (BMI > 25 kg/m²), and 600 million classified as obese (BMI > 30 kg/m²) (World Health Organisation, 2016). Overweight and obesity are associated with increased risk of several chronic diseases (e.g. type 2 diabetes, cancer, heart disease and hypertension) and, as such, *over-nourishment* now poses a greater concern to global public health than starvation and malnutrition. In England, rates of obesity are higher than the global average with around 37% of adults classified as overweight, and 26% as obese (NHS Information Centre, 2012), and treatment of obesity and obesity-related conditions cost the NHS around £15.8 billion per year (Butland et al., 2007).

The rise in rates of obesity has been attributed to changes to the food environment and the abundant availability of high-calorie foods. O’Dea (1992) describes the modern food environment as a ‘continuous feast’, and contrasts it from the ‘feast and famine’ that would have been experienced by our earlier human ancestors. Given that most of our evolution took place in an environment of food scarcity, the ‘thrifty gene hypothesis’ proposes that the propensity to overeat whenever the opportunity arose would have ensured our survival (Prentice et al., 2005). This innate predisposition to overeat, combined with the availability of low-cost high-calorie foods, is thought to present an ‘obesogenic environment’ in which, for many, weight gain is virtually inevitable.

However, the complex aetiology of obesity is reflected by the fact that many people, *despite* the obesogenic environment of the modern Western world, manage to remain slim. Indeed, the causes of obesity are thought to encompass a range of environmental, behavioural (e.g. diet and exercise), and biological (e.g. hormonal / genetic) factors. In section 1.1 of this chapter, I provide a brief overview of the physiological, hedonic, and cognitive controls of dietary behaviour, and identify the underlying factors that make some people particularly prone to overeating, weight gain, and obesity. In section 1.2, I present the popular theory that an ‘addiction’ to food may underlie certain cases of overeating. Specifically, I will examine the extent to which certain foods yield addictive properties analogous to those of drugs of abuse, and I will review the current conceptualisations of the concept.

1.1. The role of physiological, hedonic, and cognitive mechanisms in appetite control

1.1.1. Physiological control

1.1.1.1. The weak and chronic effect of energy storage

To maintain energy balance, such that the amount of energy consumed equates to the amount of energy expended, the UK Committee on Medical Aspects of Food Policy (COMA), recommends a daily caloric intake of 2500 (10,500kj) and 2000 (8,400kj) kilo-calories for men and women, respectively (Department of Health, 1991). However, even a lean individual has enough stored energy, in the form of glucose, glycogen, and protein, to sustain them for up to 55 days without food (Frayn, 2010). Furthermore, bodily energy reserves play an important role in regulating appetite, and research has demonstrated the elevating effects of adipose tissue on circulating levels of satiety hormones (i.e. leptin and insulin) (Montague et al., 1997; Woods et al., 1998).

However, compared with the effects of recent energy consumption, the effect of stored energy on appetite regulation is weaker. This is reflected by the relatively weak effect of depletion of energy stores (via physical activity) on appetite (Schubert et al., 2013), further Rogers and Brunstrom (2016) point out that the inhibiting effect of ingested nutrients on appetite diminishes as they become stored within the body (Almiron-Roig, et al., 2013). As such, Rogers and Brunstrom (2016) provide a model of energy balancing which contrasts the weak and chronic effects of stored energy reserves, with the strong and acute effects of recent meal consumption (Figure 1.1).

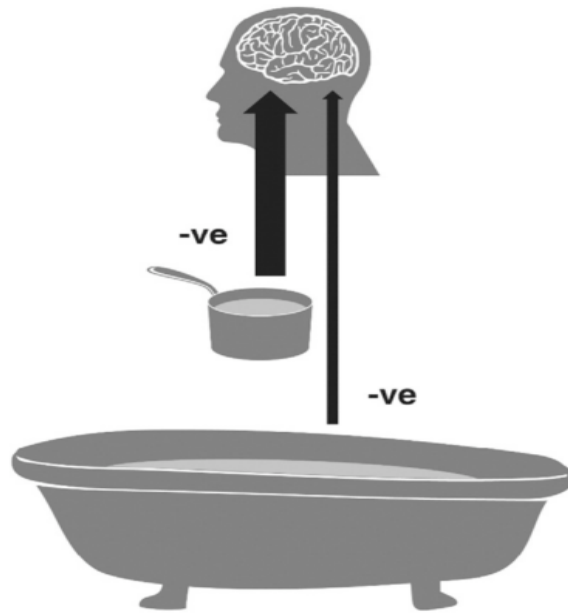


Figure 1.1. Model of energy balancing and appetite, adapted from Rogers & Brunstrom (2016). The water in the saucepan represents recent meal consumption, which has a strong and acute effect on appetite (represented by the thick arrow). The water in the bathtub represents stored energy, which has a relatively weak and chronic on appetite (represented by the thinner arrow).

1.1.1.2. The strong and acute effect of recent meal consumption

Drawing upon the weak effect of stored energy on appetite regulation, Rogers and Brunstrom (2016) define hunger as representing the ‘absence of fullness’, rather than an energy deficit *per se*. Sensations of hunger are mediated physiologically by the hormone ghrelin. Concentrations of ghrelin correlate positively with subjective hunger ratings, and decline following meal consumption (Cummings, 2006). Furthermore, long-term ghrelin administration leads to weight gain in rats (Tschop, Smiley, & Heiman, 2000), and increased circulating levels of ghrelin have been observed in individuals with Prada-Willi syndrome, in which symptoms include hyperphagia and weight gain (Cummings et al., 2002). Conversely, a lack of ghrelin function is associated with diminished risk of weight gain in mice (Zigman et al., 2005).

During the consumption of a meal, stomach distension, a rise in blood nutrient concentrations, and the release of regulatory hormones insulin and leptin, have an inhibiting effect on food intake (Arase et al., 1988; Figlewicz, Bennett, Naleid, Davis, & Grimm, 2006). Indeed, exogenous administration of leptin has been found to reduce food intake, while a state of leptin deficiency *increases* food intake

(Brunner et al., 1997). Furthermore, the administration of insulin has been found to decrease consumption of palatable snacks (Hallschmid et al., 2012). The main sites of action for ghrelin, leptin and insulin are within the hypothalamus, the brain's appetite control centre.

1.1.1.3. Leptin resistance

The mechanisms involved in satiety may be disrupted following the long-term consumption of a high-fat diet. In particular, despite having higher circulating levels of leptin, individuals with obesity may become resistant to its appetite suppressing effects (Hukshorn et al., 2000), and subsequent research has shown that this state of 'leptin resistance' is more closely associated with increased intake of dietary fat, than increased adiposity *per se* (Figlewicz et al., 2004, 2006). Furthermore, the diminished inhibitory effect of leptin may underlie an ability to consume larger portions of food over time in individuals with obesity or binge eating disorder (e.g. Cassin & von Ranson, 2007; Kenny, 2013).

However, individual differences in physiological appetite control (i.e. sensations of hunger and fullness) are thought to contribute minimally to the development of obesity (Berthoud, 2004). Rather, cognitive and environmental factors, such as the pleasurable and rewarding aspects of food, are thought to exert a stronger influence over intake in the modern environment, in which food is readily available (Figure 1.2. Berthoud, 2004).

Eating in response to external, rather than internal cues, represents a form of ‘reward-driven eating’, which has been defined as eating in response to a food’s hedonic qualities (Appelhans, 2009). The tendency to engage in reward-driven eating is thought to underlie much of the individual variation in body weight, and is driven both by perceptions of *food reward* (defined as the momentary value of a food, Rogers & Hardman, 2015) and the extent to which an individual exerts cognitive control over eating (Appelhans et al., 2011). Food reward is thought to be partly influenced by how much an individual ‘likes’ a food (i.e. the ‘pleasantness of the food’s taste’), and by their current level of physiological hunger (Rogers & Hardman, 2015).

The underlying rewarding value of a food can be indirectly assessed by examining the effect of a food-cue (i.e. a stimulus that has previously been associated with food receipt) on subjective (desire-to-eat/cravings), motivational (i.e. willingness to *work* for food/willingness to *pay* for food), physiological (e.g. salivation), and cognitive (e.g. attention to food cues) responses (Field, Werthmann, Franken, & Hofmann, 2016). Indeed, increased levels of hunger and liking (i.e. components of food reward) have been found to increase salivary responses to food-cues (e.g. Hodgson & Greene, 1980; Klajner, Herman, & Polivy et al., 1981), ‘desire-to-eat’ ratings (Rogers & Hardman, 2015), motivation to obtain food (Raynor & Epstein, 2003; Rogers & Hardman, 2015), and measures of attention to food, relative to non-food, cues (i.e. attentional bias) (Brignell, Griffiths, Bradley, & Mogg, 2009; Lavy & Van den Hout, 1993).

1.1.2.1. Incentive Salience Theory

It is important to note that variation in food reward is not entirely accounted for by measures of food liking and hunger (Rogers & Hardman, 2015). One possibility is that this unexplained variance may be accounted for by *implicit* processes which, according to ‘Incentive Sensitization Theory’ (IST) (Berridge & Robinson, 2003; Robinson & Berridge, 1993), may be conceptualised as ‘wanting’. Originally devised to explain the development of substance-dependence, IST proposes that repeated exposure to and consumption of a substance sensitises the release of dopamine within brain ‘reward’ areas. This sensitized dopaminergic response is thought to increase the ‘incentive value’, or motivational properties, of a substance and its associated cues (i.e. ‘wanting’). This model of substance

dependence has recently been extended to explain the development of obesity and overeating. In particular, Appelhans, French, Pagoto, and Sherwood (2016) proposed a ‘temptation magnet’ theory of obesity, which suggests that palatable foods may have motivational qualities which, for some, capture attention and trigger diet relapse.

Berridge (2009) distinguishes between ‘wanting’, which occurs outside of introspective awareness, and consciously accessible perceptions of ‘liking’. Wanting and liking are thought to represent distinct and separable components of reward and this has been supported by evidence from animal models. For example, administration of a dopamine antagonist, or lesioning to the nucleus accumbens (NAc) (which receive dopaminergic input from the ventral tegmental area), decreases behaviours that are associated with obtaining food, but do not affect feeding behaviour (Aberman & Salamone, 1999; Bakshi & Kelley, 1993; Koob, Riley, Smith, & Robbins, 1978; Salamone, Cousins, & Bucher, 1994). Conversely, increased dopamine concentrations within the NAc have been found to incentivise the motivational properties of food (Kiyatkin & Gratton, 1994). Liking reactions are primarily mediated by opioid transmission within the NAc, ventral pallidum, and parabrachial nucleus of the pons (Levine & Billington, 2004; Peciña & Berridge, 2005; Smith & Berridge, 2005). In particular, Berridge (2009) refers to the ‘hedonic hotspot’ within the medial shell of the NAc in which stimulation of opioid and endocannabinoid receptors increase liking reactions in rats (Peciña, Smith, & Berridge, 2006; Peciña, 2008; Smith, Tindell, Aldridge, & Berridge, 2009).

With regards to obesity and weight gain, two predictions can be derived from IST. Firstly, individuals who have experienced more frequent associations between food cues and food receipt (i.e. increased learning history), such as those with a propensity to overeat, will be expected to demonstrate increased ‘wanting’ in response to food cues. Secondly, IST predicts that this increased ‘wanting’ may occur in the absence of any increased ‘liking’.

Importantly, however, the extent to which it is possible to successfully capture implicit ‘wanting’ processes in humans is debated. Previous research has equated wanting to performance on implicit measures of reaction time (e.g. (Finlayson, King, & Blundell, 2008, 2007) and memory tasks (Lemmens et al.,

2009). However, Havermans (2011) argues that these measures are inevitably contaminated by explicit ‘liking’ judgements. As such assessments of ‘wanting’ may be best thought of as capturing the overall ‘reward’ value of a food. Indeed, in a series of studies, Rogers and Hardman (2015) found that measures of food reward (i.e. desire-to-eat ratings, willingness to pay, and an operant response task) were independently predicted by participants’ current levels of physiological hunger, and by food ‘liking’ ratings. Based on these findings, Rogers and Hardman (2015) propose that hunger influences food reward via effects on ‘wanting’ and, consistent with Havermans (2011), posit the inherent difficulty in obtaining a measure of implicit wanting that is distinct from the overall reward value of the food. Thus, in the proceeding sections I will use the term ‘food reward’, rather than ‘wanting’, to discuss evidence related to the two aforementioned predictions of IST.

In support of the first prediction of IST, there is evidence to suggest that individuals who have a propensity to overeat (i.e. those with an increased learning history) experience food as more rewarding (Mela, 2006). For example, increased salivary reactivity to the presence of environmental food cues has been observed in those with bulimia nervosa and obesity (LeGoff et al., 1988; Epstein, Paluch, & Coleman, 1996), and women with obesity demonstrated attenuated decline in their salivary responses, following repeated exposure to food cues, compared to non-obese women (Epstein et al., 1996). Furthermore, compared to normal weight participants, adults and children with obesity demonstrate increased motivation to work for food (Giesen, Havermans, Douven, Tekelenburg, & Jansen, 2010; Johnson, 1974; Temple, Legierski, Giacomelli, Salvy, & Epstein, 2008), and have elevated levels of desire-to-eat following food adverts (Kemps, Tiggemann, & Hollitt, 2014b). Conversely, individuals with anorexia nervosa (i.e. those with decreased learning history) demonstrated diminished salivary responses to food cues compared to controls (LeGoff et al., 1988).

In addition, individuals with a propensity to overeat may be less sensitive to the effects of physiological satiety on food reward. Nasser et al. (2008) found that, following consumption of a high-calorie preload, participants with binge eating disorder (BED) did not demonstrate a correlation between hunger ratings and motivation to obtain food; rather, this relationship was only observed in participants without BED. This is consistent with previous research which has examined the

relationship between hunger and food reward in patients with bulimia (Bulik & Brinded, 1994), and in animal models of binge eating (Boggiano et al., 2007). Furthermore, individual differences in food reward are thought to be most evident when participants are satiated rather than hungry (Field et al., 2016). Indeed, increased attentional bias to food cues in overweight, obese, or binge eating participants, relative to controls, have been observed under conditions of satiety but not when participants were hungry (Castellanos et al., 2009; Werthmann, Field, Roefs, Nederkoorn, & Jansen, 2014).

In support of the second prediction made by IST, there is evidence to suggest that increased food reward may occur in the absence of any increased liking. For example, Giesen et al. (2010) found that overweight participants demonstrated increased motivation to obtain snack foods, compared to fruits and vegetables, despite no differences in liking ratings for the two food categories. Similarly, Temple et al. (2008) observed a positive correlation between BMI and motivation to obtain food, but no correlation between BMI and food liking ratings. In a subsequent study, Temple et al. (2009) reported *increased* motivation to obtain food in obese participants, and *decreased* motivation in non-obese participants, following repeated consumption of a 300kcal portion of food. This was despite diminished liking ratings in both groups. According to IST, the dissociation between food reward and liking is thought to arise following repeated exposure to certain foods. In support of this, Zandstra, Graaf, Mela, and Staveren (2000) found that repeated consumption of a less preferred bread resulted in increased consumption and desire-to-eat in the absence of increased liking.

Contrary to previous suggestions (Dressler & Smith, 2013; Nasser, 2001; Rissanen et al., 2002), these findings suggest that overeating tendencies are driven by increased motivation to obtain food, rather than increased liking *per se*. Indeed, Salbe et al. (2004) points out that evidence of increased food liking in obese, relative to lean, individuals has not been consistently demonstrated, and de Graaf (2005) concludes that individuals with obesity do not have any abnormal taste function. An alternative perspective is that the inconsistent evidence of increased food-liking in those with overweight or obesity may be attributable to the inherent difficulty in separating aspects of liking from reward when providing subjective judgements (Rogers & Hardman, 2015). To address this methodological concern, Rogers and

Hardman (2015) suggest that measures of food liking should be obtained while the participant is tasting the food while ignoring what it would be like to actually ingest the food (i.e. pleasantness of taste of food in the mouth).

1.1.2.2. Food reward, food intake, and weight gain

Measures of food reward have been found to predict future weight gain. For example, responsivity to food cues, and motivation to obtain food, successfully predicted weight gain in children (Hill, Saxton, Webber, Blundell, & Wardle, 2009; Rodin & Slochower, 1976). Similarly, in adults, Calitri, Pothos, Tapper, Brunstrom, and Rogers (2010) found that an attentional bias to unhealthy food words predicted weight gain at 1-year follow-up, while weight loss was predicted by an attentional bias to healthy food words. Importantly, however, less than five per cent of variance in BMI change was accounted for by measures of food reward (Calitri et al., 2010). Similarly, while several studies have demonstrated a predictive ability of food reward measures on subsequent food intake (Epstein et al., 2004; Nijs, Muris, Euser, & Franken, 2010; Werthmann, Renner, et al., 2014; Werthmann, Roefs, Nederkoorn, & Jansen, 2013), this has not been consistently reported (Hardman, Scott, Field, & Jones, 2014; Nederkoorn, Smulders, & Jansen, 2000). Furthermore, Rogers and Hardman (2015) found that subjective measures of desire-to-eat only accounted for around one third of the variance in food intake. The limited predictive ability of food reward measures on subsequent food intake and longer-term weight gain, may be accounted for by the role of cognitive mechanisms on appetite control and food intake.

1.1.3. Cognitive control of food intake

1.1.3.1. Inhibitory control

Dual process models posit that overeating and weight gain are determined by an interplay of heightened food reward and diminished inhibitory control (Appelhans, 2009). This is supported by studies which found that measures of food reward significantly predicted food intake and weight gain at 1-year follow-up, but only for participants who demonstrated low levels of inhibitory control (Appelhans et al., 2011; Nederkoorn et al., 2010; Rollins, Dearing, & Epstein, 2010). Furthermore, neuroimaging studies of food choice have demonstrated an important role of the pre-frontal cortex (PFC), an area of the brain that is involved in planning and decision-making. For example, Hare et al. (2009) reported increased activity within the PFC

in individuals who selected healthy, rather than more palatable, foods. Similarly, DelParigi et al. (2007) demonstrated increased activity within the dorso-lateral area of the PFC (dlPFC) in response to food consumption in dieters, compared to non-dieters. Conversely, diminished dlPFC activation to food has been found in individuals with obesity (Le et al., 2006).

Low inhibitory control is one aspect of the personality trait of ‘impulsivity’, which is characterised by poor planning, and the tendency to make decisions without considering the consequences (Dalley et al., 2011). Increased trait impulsivity has been associated with obesity (Nederkoorn et al., 2006), increased food intake, future weight gain and lower diet success rate (van den Akker, Jansen, Frentz, & Havermans, 2013; Nederkoorn et al., 2007; Gauerrieri et al., 2007). Conversely, individuals with greater self-regulation and an increased ability to delay gratification demonstrate lower weight gain throughout adolescence and adulthood (Mischel et al., 1988; Schlam et al., 2013; Seeyave et al., 2009).

1.1.3.2. Dietary restraint

Restraint theory (Herman & Polivy, 1980) accounts for an individual’s capacity to restrict their food intake in order to achieve or maintain weight loss. This usually involves attempts to minimise ones intake of high calorie foods (Francis, Stewart, & Hounsell, 1997). To assess levels of dietary restraint, Herman and Polivy (1980) devised the ‘restraint scale’ which is comprised of 10-items related to ‘concern for diet’ and ‘weight fluctuation’. Using this scale, research has demonstrated that an increased sensitivity to external food cues may be more closely attributed to patterns of dietary restraint, than to obesity *per se*. Indeed, individuals who scored highly on the restraint scale have been found to consume *more* food following a preload, while those who scored low in dietary restraint consumed *less* (Federoff, Polivy, & Herman, 1997; Herman & Mack, 1975). Herman and Polivy (1983) attributed this to a ‘counter-regulatory’ response in which the violation of a ‘diet boundary’ resulted in highly restrained eaters ‘giving up’ control over their diet. Subsequent research has demonstrated that this counter-regulatory response can be elicited following a number of triggers such as emotional arousal, cognitive load, stress, and exposure to food cues (Hawks, Madanat, Smith, & De La Cruz, 2008; Polivy, Coleman, & Herman, 2005; Westerterp-Plantenga, Wouters, & ten Hoor, 1991).

Recent revisions of restraint theory distinguish between successful and unsuccessful restrained eaters (Westenhoefer, 1991). Specifically, scores on the restraint scale have been found to correlate with measures of dietary disinhibition (i.e. disrupted control over eating) and, as such, are thought to capture *unsuccessful* attempts to restrict eating. More recent assessments of dietary restraint, such as the Dutch Eating Behavior Scale (DEBQ) and the Three Factor Eating Scale (TFEQ), do not correlate with dietary disinhibition and are therefore thought to reflect successful dietary restraint. Indeed, Westenhoefer (1994) found that the counter-regulatory response observed following the consumption of a preload was observed only in unsuccessful, but not successful, restrained eaters.

Failed attempts at dietary restraint may be partly driven by the enhancing effects of chronic restriction on the reward value of certain foods. Indeed, several studies have demonstrated increased attention to food cues in restrained, compared to non-restrained eaters, (Francis et al., 1997; Green & Rogers, 1993; Hollitt, Kemps, Tiggemann, Smeets, & Mills, 2010; Overduin, Jansen, & Louwense, 1995; Perpina, Hemsley, Treasure, & de Silva, 1993; Stewart & Samoluk, 1997), particularly in overweight restrained eaters (Ouweland & Papies, 2010). Furthermore, one study found that, compared to non-restrained eaters, restrained eaters reported greater desire-to-eat, liking, and craving for a pizza, following exposure to food-cues (Federoff, Polivy, & Herman, 1997).

Taken together, these findings suggest that attempts to restrict one's intake of palatable foods may, in some individuals, elicit a self-perpetuating cycle of heightened food reward (i.e. cravings and desire-to-eat), increased food intake, and further attempts at dietary restriction. This inherent difficulty in exerting control over one's diet has prompted some to suggest that high-calorie foods may yield an addictive potential similar to that of drugs of abuse.

1.2. The case for food addiction

The concept of food addiction was first introduced within the scientific literature in 1956 by Theron Randolph who suggested that certain foods produce a “common pattern of symptoms descriptively similar to those of other addictive processes” (Randolph, 1956, p.221). Since then, this view has become widely accepted throughout Western society, and there now exist a plethora of websites, self-help books, and articles all dedicated to ‘curing’ people of their addiction to food. According to one survey, as many as 86% of Australians and Americans believed that certain foods are addictive, and 72% believed that food addiction could account for some cases of obesity (Lee et al., 2013). Furthermore, in a sample of 1000 Americans, food addiction was one of the most frequently given explanations for the increasing rates of obesity (Barry et al., 2009), and support for the concept appears to be most prevalent amongst individuals who are overweight or obese, and those who engage in pathological patterns of eating (Wilson et al., 2009).

As well as general support for the existence of food addiction, evidence suggests that up to 57% of individuals from community samples perceive themselves to be addicted to food (Hardman et al., 2015; Meadows & Higgs, 2013). Furthermore, Meadows and Higgs (2013) found that ‘self-perceived ‘food addicts’ had increased BMI and scored higher on measures of pathological eating, than those who did not perceive themselves to be food addicts. Similarly, self-diagnosed ‘chocolate addiction’ has been associated with feeling ‘out of control’ around chocolate, increased salivation and cravings in response to chocolate cues, and increased chocolate consumption (Hetherington & Macdiarmid, 1993; Tuomisto et al., 1999).

However, the extent to which food yields addictive properties similar to those of drugs of abuse has been the source of considerable controversy throughout the scientific community (e.g. Hebebrand, 2014; Ziauddeen, Farooqi, & Fletcher, 2012; Rogers & Smit, 2000), and there is some concern regarding the potential impact of ‘food addiction’ messages on health-related behaviour (Lee et al., 2012). In this section, I will discuss the prevalence and correlates of ‘food addiction’, defined using an existing substance-based criterion, and critically review the evidence that is frequently cited as providing support for the concept. Finally, I will review the psychosocial perspectives of ‘food addiction’, and explore the potential

consequences that food addiction messages and beliefs may have on dietary choices and eating behaviour.

1.2.1. The Yale Food Addiction Scale

The Yale Food Addiction Scale (Gearhardt et al., 2009) is the most widely used tool for the quantification and ‘diagnosis’ of food addiction within the scientific literature. Based upon the DSM-IV criteria for substance dependence (see Table 1.1), the 25-item scale provides a diagnosis of food addiction in cases where an individual demonstrates at least three, out of seven, symptoms, in conjunction with a clinically significant impairment as a result of their eating. A recent revision of the scale (YFAS 2.0., Gearhardt, Corbin, & Brownell, 2016) was developed in line with changes to the DSM-V classification of Substance-Related and Addictive Disorders (SRAD). The YFAS 2.0. thus includes a list of 11 symptoms (Table 1.1), and a diagnosis of food addiction is warranted in cases where an individual fulfils two or more of these symptoms. As well as providing a diagnostic assessment of food addiction, the YFAS and YFAS 2.0 provide a continuous ‘symptom count’ measure ranging from 0 to 7 symptoms (in the original YFAS), and 0 to 11 symptoms (in the revised YFAS 2.0).

Table 1.1. *Criterion for substance-dependence (DSM-IV) and substance-related and addictive disorders (DSM-V).*

Criterion	DSM-IV	DSM-V
Takes more than intended	✓	✓
Persistent desire/unsuccessful attempts to quit	✓	✓
Tolerance (i.e. requiring increasing amounts to achieve the same effect)	✓	✓
Withdrawal	✓	✓
Continued use despite negative consequences	✓	✓
Important activities given up because of use	✓	✓
Great deal of time taken to obtain/use/recover from the substance	✓	✓
Failure to fulfil role obligations because of use		✓
Recurrent use resulting in physically hazardous behaviour		✓
Continued use despite social problems		✓
Recurrent cravings		✓

1.2.2. Prevalence of food addiction

Within a community sample, the prevalence of ‘food addiction’ has been reported at around five per cent, using the original YFAS diagnostic criteria (Pedram et al., 2013), and 16 per cent, using the YFAS 2.0. (Gearhardt et al., 2016). In a systematic review of 23 studies which assessed food addiction in both clinical and non-clinical samples, Pursey et al. (2014) reported that YFAS-diagnosed food addiction was more common in females, compared to males, (12.2% vs. 6.4%), and in adults aged over 35 years, compared to those aged under 35 (22.2% vs. 17.0%) (Pursey, Stanwell, Gearhardt, Collins, & Burrows, 2014). Furthermore, Long et al. (2015) found that individuals with overweight and obesity were four to five times more likely to meet the YFAS diagnostic criteria for food addiction than the general population (33% vs. 6.8%), and studies have revealed positive associations between food addiction symptomology and BMI (Gearhardt et al., 2016; Pedram et al., 2013).

However, a linear relationship between food addiction and weight status has not been consistently demonstrated (Meule & Kübler, 2012; Gearhardt et al., 2011), and proponents of the food addiction concept point out that food addiction and obesity represent distinct and separable constructs. Indeed, many people with obesity do not fulfil the YFAS criteria for food addiction, and not all YFAS-diagnosed ‘food addicts’ have obesity (Davis et al., 2011).

As such, food addiction is thought to reflect certain patterns of eating, rather than general increased caloric intake *per se*. One particularly prominent view is that food addiction may be most closely characterised by a tendency to engage in binge eating (i.e. consuming a large amount of food within a short time period). Indeed, qualitative research has documented similarities between the experiences of individuals with binge eating disorder (BED) and the signs and symptoms of substance-dependence (Cassin & von Ranson, 2007; Curtis & Davis, 2014; Lyons, 1998). Furthermore, between 42%-57% of those with BED fulfil the YFAS criteria for food addiction (Bégin, 2012; Gearhardt & Brownell, 2013; Gearhardt, Treat, Hollingworth, & Corbin, 2012), and amongst overweight/obese participants with food addiction, 29%-72% demonstrated clinical-level binge eating (Caroline Davis et al., 2011; Imperatori et al., 2014).

1.2.3. Distinctions between FA and binge eating

The overlap between BED and food addiction raises important considerations regarding the characteristics which distinguish food addiction from other forms of pathological eating. Indeed, while studies have shown poorer outcomes following weight-loss interventions in those with YFAS-diagnosed food addiction (Clark & Saules, 2013), this may be largely attributable to increased binge eating tendencies (Burmeister, Hinman, Koball, Hoffmann, & Carels, 2013). As such, Long et al. (2015) suggest that the concept of food addiction may simply provide a novel term that is used to describe already-established patterns of eating. One possibility is that food addiction represents a *more severe* form of BED (Caroline Davis, 2013). In support of this view, studies have shown that obese binge eaters who fulfil the YFAS criteria for food addiction report more intense food cravings, and demonstrate an increased tendency to engage in hedonic and pathological eating patterns, compared to obese binge eaters without food addiction (Caroline Davis et al., 2013; Gearhardt, White, Masheb, & Grilo, 2013). Similarly, Gearhardt et al. (2013) reported that binge eating frequency was significantly predicted by YFAS symptomology, more than other measures of eating pathology.

Based on these findings, Davis (2013) proposed an ‘eating continuum’ in which food addiction represents the most severe form of compulsive overeating, while milder forms are represented by ‘occasional’ or ‘frequent’ overeating tendencies (see Figure 1.2). Similarly, Vainik, Neseliler, Konstabel, Fellows, and Dagher (2015) have recently shown that various assessments of eating behaviours reflect differing levels of severity of a single underlying construct, which they define as ‘uncontrolled eating’. While the researchers did not include a measure of food addiction, Vainik et al. (2015) posit that such measures may represent the most pathological form of non-homeostatic eating.

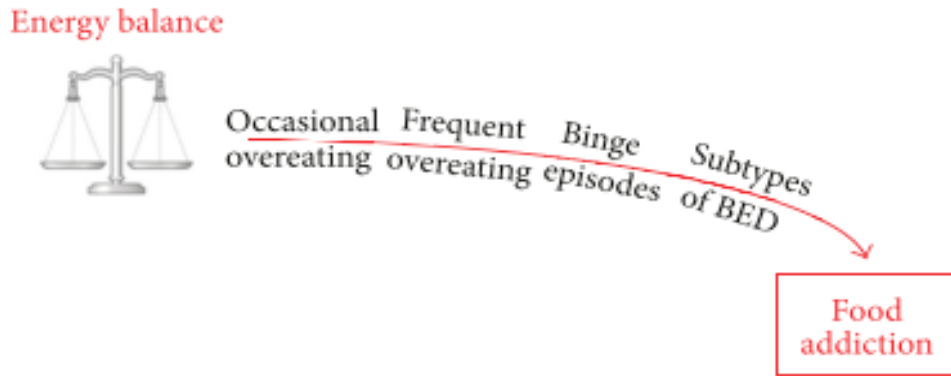


Figure 1.3. A continuum of eating with energy balance (or ‘homeostatic eating’) represented at one end, and food addiction (i.e. the most severe form of non-homeostatic eating) at the other. Adapted from Davis (2013) copyrighted©2013 Caroline Davis, Creative Commons Attribution License, <http://creativecommons.org/licenses/by/3.0/>.

Nonetheless, an agreed operational definition of food addiction, and its associated behaviours, is yet to be established, and there is on-going debate surrounding the applicability of a substance-based framework to the assessment of eating behaviours. In particular, Ziauddeen, Farooqi, and Fletcher (2012) argue that many of the symptoms of substance abuse may not be easily applied to overeating, and Hebebrand et al. (2014) contest the conceptualisation of food addiction as a substance-based addiction. These important limitations of the food addiction concept will now be discussed.

1.2.4. Food addiction: Weighing up the evidence

1.2.4.1. Clinical (symptom) overlap

Some of the clinical symptoms of substance abuse, outlined by the DSM-IV and DSM-V, appear to be directly applicable to eating behaviour. For example, people regularly consume ‘more than intended’, report a ‘persistent desire’ or ‘cravings’ for certain foods, and have ‘repeated unsuccessful attempts to quit’ consuming highly palatable and energy dense foods. Overeating, or the consumption of unhealthy foods, may also continue ‘despite negative consequences’ to one’s health, and these ‘symptoms’ are frequently reported by individuals with BED (Cassin & von Ranson, 2007). However, other core features of substance dependence cannot be so easily applied to eating behaviour. In particular, fundamental differences between the societal effects of drugs and food mean that problematic

eating behaviours may not necessitate any ‘impairment to daily functioning’, or the cessation of ‘important social, occupational, or recreational activities’. In addition, unlike drugs, there is little need to spend ‘much time obtaining’ food within the modern Western environment.

Reflective of ‘tolerance’ (i.e. requiring increased amounts over time to achieve the same effect), rats that were given intermittent access to a diet high in fat or sugar demonstrated escalating binge eating tendencies which corresponded to a rapid release of dopamine, and a delayed acetylcholine response (indicative of diminished satiety, Mark, Rada, Weinberg, Pothos, & Hoebel, 1992) (Avena, Rada, Moise, & Hoebel, 2006; Colantuoni et al., 2001). Furthermore, signs of withdrawal, analogous to those observed following opiate cessation (e.g. forepaw tremors, teeth chattering, and increased anxiety behaviours), were demonstrated following the administration of an opiate antagonist or food deprivation in rats that had been provided with intermittent access to a high-sugar diet (Colantuoni et al., 2002).

The extent to which physical symptoms of ‘tolerance’ and ‘withdrawal’ can be observed in humans in response to food is widely debated. Some studies have equated signs of food-related withdrawal to feelings of ‘irritability’, ‘anxiety’, ‘lethargy’, ‘poor concentration’, and ‘sleeplessness’ in response to the unobtainability of certain foods (Ifland et al., 2009; Cassin & von Ranson, 2007). Furthermore, ‘tolerance’ is thought to be reflected by the ‘need to increase frequency/intensity of binge eating’ and the escalating intake of certain foods over time that has been observed in binge eaters and self-perceived food addicts (Cassin & von Ranson, 2007; Geliebter et al., 2004; Ifland, 2009). However, the idea that these symptoms are reflective of drug-induced states of tolerance and withdrawal has been contested (Benton, 2010; Ziauddeen, Farooqi, & Fletcher, 2012), and in a review of studies which examined the effect of opioid antagonists in binge eaters, Benton (2010) concludes that there is little evidence to support the existence of a food-related ‘withdrawal’ syndrome in humans. Furthermore, the processes that underlie an ability to consume larger portion sizes over time may be distinct from those that facilitate a tolerance to commonly abused drugs. Indeed, Sullivan and Birch (1990) found that, while preference for sweet tastes increased following repeated consumption, this did not generalise across all foods; instead, individuals demonstrated preferences for tastes that had previously been associated with a

particular food. Drawing upon this evidence, Benton (2010) suggests that changes to food portion sizes and/or food preferences may arise following changes to one's *expectations* about a food's sensory qualities, rather than due to physiological changes reminiscent of those observed following repeated drug use.

Given the limited applicability of the clinical symptoms of substance-abuse to eating behaviour, it is important to consider the extent to which food addiction is best conceptualised as representing a substance-based addiction. To address this, a review of the similarities and differences between the effects of drugs and food is warranted.

1.2.4.2. Shared vulnerabilities

Proponents of the 'food addiction' model frequently cite evidence of shared vulnerabilities for the development of substance-dependence and compulsive overeating or obesity. For example, an increased risk of obesity and binge eating disorder has been observed in those with a family history of alcoholism and substance-dependence (Gruza et al., 2010; Lilenfeld et al., 2008). Furthermore, individuals who undergo bariatric surgery for obesity have been found to be at increased risk of developing alcoholism (Suzuki, Haimovici, & Chang, 2012), and this risk appears to be particularly pronounced in those who fulfil the YFAS criteria for food addiction (Clark & Saules, 2013). Conversely, former opiate addicts demonstrated increased cravings for sweet and salty foods (Cocores & Gold, 2009; Cowan & Devine, 2008; Morabia et al., 1989), and weight gain has been observed in rats and bulimic women following withdrawal from opiate use (Azar et al., 2004; Katzman, Greenberg, & Marcus, 1991). This overlap between obesity, overeating, and drug dependence suggests that these share similar underlying risk factors. These will now be briefly discussed.

A common genetic predisposition.

Certain genetic markers have been found to underlie an increased risk for obesity, binge eating, and substance-dependence. In particular, a specific genotype of the functional marker, A118G, of the opioid receptor gene, OPRM1, has been associated with an increased preference for high- sugar and high- fat foods, binge eating disorder, substance abuse, and alcoholism (Bart et al., 2005; Davis et al., 2011; Davis et al., 2009; Deb et al., 2010). Furthermore, polymorphisms on the DRD2 and DRD4 genes have been associated with cravings for food, nicotine,

heroin, and alcohol (McClernon et al., 2007; Filbey et al., 2008; Sobik et al., 2005; Shao et al., 2006), alcoholism (Munafò, Matheson, & Flint, 2007), increased BMI (Thomas et al., 2001; Kaplan et al., 2008; Spitz, et al. 2000), and future weight gain in those with weaker striatal and orbito-frontal cortex response to food cues (Eric Stice, Yokum, Bohon, Marti, & Smolen, 2010). These associations are thought to be mediated by the effect of DRD2 and DRD4 genes on the hypo-functioning of dopaminergic neural networks (Ritchie & Noble, 2003; Tupala et al., 2003). Furthermore, using the Multilocus Genetic Profile Score (MLGP) technique of genetic profiling (Nikolova et al., 2011), Davis et al. (2013) examined the ability of several genetic markers known to be associated with dopamine responsiveness to distinguish individuals with food addiction (as assessed using the YFAS). As predicted, greater MLGP scores were identified in those who fulfilled the criteria for food addiction. Further, MLGP scores correlated with measures of food cravings, emotional overeating, and binge eating, and these measures of hedonic eating mediated the relationship between MLGP scores and YFAS-diagnosed food addiction.

Importantly, however, evidence of an association between weight status and polymorphisms to dopamine and opioid receptor genes has not been consistently reported (Hardman, Rogers, Timpson, & Munafò, 2014; Jenkinson et al., 2000; Southon et al., 2003; Volkow et al., 2008). These discrepant findings may be due to sample size differences. Specifically, Hardman et al. (2014) point out that studies which have reported an association between BMI and OPRM1 and DRD2 polymorphisms were conducted using small sample sizes (e.g. Spitz et al., 2000; Davis, 2009, 2011), and that these findings have not been replicated in genome-wide association studies with large samples (e.g. Jenkinson et al., 2000).

Personality factors.

Certain personality traits may also represent shared vulnerabilities for overeating and substance dependence. For example, trait impulsivity (i.e. acting without thinking, seeking excitement, and an inability to complete tasks, Evenden, 1999) has been associated with an increased risk of obesity, binge eating disorder, YFAS-diagnosed food addiction, and substance abuse (Bégin, 2012; Guerrieri, Nederkoorn, & Jansen, 2008; Leehr et al., 2016; Murphy, Stojek, & MacKillop, 2013; Schoenmakers et al., 2006). Furthermore, the prevalence of substance

dependence, obesity, and YFAS-diagnosed food addiction, has been shown to be higher in those Attention Deficit Hyperactivity Disorder (ADHD), which is characterised by high levels of impulsivity (Altfas, 2002; Kessler et al., 2006; Davis et al., 2011). Additionally, low levels of ‘distress tolerance’ (i.e. one’s ability to tolerate negative states) have been associated with an increased risk of relapse following smoking cessation (Brandon, Herzog, Juliano, Irvin, & Lazev, 2003; Brown et al., 2002) and drug withdrawal (Daughters et al., 2005), and an increased tendency to engage in disinhibited eating, particularly in those with high levels of impulsivity (Anestis, Selby, Fink, & Joiner, 2007; Kozak & Fought, 2011).

Psychological factors.

Psychological factors such as life stressors and negative affect also present as shared vulnerabilities for the development of both substance abuse and obesity. Stress during childhood is associated with increased weight (Johnson et al., 2002) and drug addiction (Dube et al., 2003), and research has shown an increased propensity to overeat in animals that had experienced stress in early life (Babbs, Wojnicki, & Corwin, 2012; Jahng et al., 2013). Furthermore, high levels of anxiety and depression have been observed in people prior to the onset of binge eating disorder (Sawaoka, Barnes, Blomquist, Masheb, & Grilo, 2012; Svaldi, Griepenstroh, Tuschen-Caffier, & Ehring, 2012), and low mood has been identified as a trigger for episodes of binge eating (Schulz & Laessle, 2012; Wegner et al., 2002) and relapse in former drug addicts (Abrantes et al., 2008). Increased levels of depression and emotion dysregulation have been found in those with YFAS-diagnosed food addiction (Burmeister et al., 2013; Davis et al., 2013; Flint et al., 2014; Gearhardt et al., 2012), although this has not been consistently demonstrated (Bégin, 2012). These findings suggest that food (like drugs and alcohol), may be used as a means of coping with negative emotions. Indeed, research has demonstrated increased BMI and binge eating tendencies in those who use food as a coping mechanism (Boggiano et al., 2014; Burgess et al., 2014).

Taken together, there is evidence to suggest that substance dependence and overeating share similar fundamental risk factors (i.e. lifestyle, personality). However, as Ziauddeen et al. (2012) point out, such shared vulnerabilities do not necessitate that the same processes are involved in both. It is thus necessary to

consider the neurobiological processes which underlie motivation for substance-use and overeating.

1.2.4.3. Evidence of shared neural mechanisms

Cross sensitisation between drugs and food

There is evidence to suggest that repeated drug use sensitises the neural response to food, and vice versa. For example, Carroll, Anderson, and Morgan, (2007) demonstrated that rats bred for high saccharin intake, injected higher and more frequent doses of cocaine, than rats bred for low saccharin intake. Similarly, adult rats that had early post-natal exposure to nicotine demonstrated elevated preference for sugar and increased anxiety, compared to control rats. In humans, evidence for cross-sensitisation between nicotine and food has been demonstrated in a study which reported a 1.5 times higher prevalence of YFAS-diagnosed food addiction in former smokers, compared to current smokers (Flint et al., 2014).

Known as ‘cross-sensitisation’, these findings posit an overlap in the neural circuitry that regulates the consumption of drugs and food. Indeed, orexins and hormones involved in sensations of ‘hunger’ and ‘satiety’ (e.g. insulin, ghrelin, and leptin) have been implicated in cravings for drugs and alcohol (Borgland, Taha, Sarti, Fields, & Bonci, 2006; DiLeone, 2009; Harris, Wimmer, & Aston-Jones, 2005) and in drug seeking and relapse (Boutrel et al., 2005; Narita et al., 2006). Furthermore, the overlap between physiological appetite control mechanisms, dopaminergic and opioid ‘reward pathways’ (e.g. Aston-Jones, Smith, Moorman, & Richardson, 2009), and neural stress networks (Acerbo & Johnson, 2011; Erb & Brown, 2006) is thought to facilitate the cross-sensitisation between drugs and food.

The neural response to drugs and food

Neuroimaging research has documented similar neural-adaptations and patterns of activation to drugs and food within brain areas involved in reward and inhibitory control. In particular, using Positron Emission Topography (PET), studies have revealed a down-regulation of dopaminergic reward systems in substance users and participants with obesity (Volkow et al., 2001; Wang et al., 2001; Wang et al., 2004). This has led to a ‘reward-deficit hypothesis’, which suggests that overeating and drug-use represent attempts to compensate for a diminished reward response (Wang et al., 2002). Furthermore, this down-regulation of dopamine activity in obese subjects has been associated with the modulation of frontal brain areas (Volkow et

al., 2008), suggesting that the diminished reward response may be accompanied by an impaired inhibitory control, consistent with dual-system theories of overeating (Appelhans et al., 2009). However, subsequent research suggests that a down-regulation of dopamine binding is likely to be a *consequence*, rather than a *cause*, of overeating and substance use (Steele, Prokopowicz, Schweitzer, Brasic, & Wong, 2010; Eric Stice et al., 2010; Volkow et al., 2007). Furthermore, following a review of the literature, Stice and Yokum (2016) conclude that there is limited evidence to support the reward-deficit hypothesis of obesity.

An alternative perspective is that overeating and substance-use may be driven by a *hyper*-sensitive (i.e. reward surfeit), rather than *hypo*-sensitive (i.e. reward deficit), reward response. In support of this, substance-users and those with obesity demonstrate *heightened* activity within brain reward areas, and in areas associated with inhibitory-control, in response to drug- and food- cues, respectively (Batterink et al., 2010; Killgore & Yurgellon-Todd, 2005; Myrick et al., 2004; Rothemund et al., 2007; Stoeckel et al., 2008). Furthermore, the magnitude of activation within brain reward areas has been found to correlate with ratings of desire-to-eat (Killgore et al., 2013), drug cravings (Maas et al., 1998; Myrick et al., 2004), future weight gain (Yokum et al., 2011), and current BMI in those with low self-control (Lawrence, Hinton, Parkinson, & Lawrence, 2012).

In further support of the reward-surfeit account of overeating and substance-use, PET and microdialysis studies have shown increased dopaminergic release within brain reward areas in response to food and drug- related cues (V Bassareo & Chiara, 1999; Small, Jones-Gotman, & Dagher, 2003; Volkow, Wang, et al., 2002). Furthermore, PET studies have found that the amount of dopamine released correlates with the pleasure experienced (Barrett, Boileau, Okker, Pihl, & Dagher, 2004; Drevets et al., 2001; Small et al., 2003). There is evidence to suggest that this dopaminergic release in response to food cues is particularly pronounced in obesity (Figlewicz et al., 1998) and BED (Wang et al., 2011), and animal models of binge eating have shown an attenuated dopaminergic habituation in response to repeated presentation of food cues (Avena, Gold, Kroll, & Gold, 2012; Zilberter, 2012).

The apparent discrepancy between reward surfeit and reward deficiency accounts of overeating and substance-dependence may be reconciled by

distinguishing between the effects of reward *anticipation* and reward *receipt*. Specifically, neuroimaging research with individuals with increased weight, binge eating tendencies, or substance-dependence, have documented heightened reward activity during the *anticipation* of a reward (Balodis et al., 2013; Ng, Stice, Yokum, & Bohon, 2011), but blunted activity in response to reward *receipt* (Balodis et al., 2013; Field et al., 2016; Frank et al., 2011; Martinez et al., 2007; Stice, Spoor, Bohon, & Small, 2008). Furthermore, the blunted response to food receipt has been associated with future weight gain (Burger & Stice, 2014; Eric Stice et al., 2010; Yokum, Ng, & Stice, 2011). Similarly, compared to non-addicts, YFAS-diagnosed food addicts demonstrated increased activation of brain reward areas during the anticipation of food, while food receipt elicited diminished activation of brain areas associated with inhibitory control (Gearhardt et al., 2011).

Distinctions between brain responses to food and drugs

However, studies examining reward processing in participants with obesity or BED have yielded somewhat inconsistent findings (Ziauddeen et al., 2012). For example, some studies have failed to uncover any relationship between the striatal response to food cues and BMI (Killgore & Yurgelun-Todd, 2005; Batterink, Yokum, & Stice, 2010), and evidence for dopaminergic down-regulation in obese and binge eating participants has not been consistently observed (Dunn et al., 2010; Haltia et al., 2007; Volkow, Wang, & Baler, 2011; Wang et al., 2001; Wang et al., 2011). Furthermore, Stice, Yokum, and Burger (2013) found that reward area responsivity to a monetary reward successfully predicted future substance-dependence, but did not predict overweight or obesity onset.

Some important caveats should be considered when comparing the neural response to drugs and food. Firstly, the dopaminergic release in response to drugs is approximately 10 times greater than to food (Volkow & Wise, 2005) and does not diminish with increased satiety or following repeated consumption of a similar taste (Avena, Rada, & Hoebel, 2008; Epstein, Temple, Roemmich, & Bouton, 2009). Secondly, separate neuronal pathways within the NAc core and shell have been identified which specifically activate to food or drugs (Bassareo, Musio, & Di Chiara, 2011; Carelli, Ijames, & Crumling, 2000), and animal models have demonstrated the ability for deep brain stimulation to reduce responding for cocaine yet maintain responding for food (Pratt, Choi, & Guy, 2012). Finally, Stice,

Figlewicz, Gosnell, Levine, & Pratt (2012) point out that evidence for shared neural mechanisms for the consumption of drugs and food is not sufficient justification for an ‘addiction’ model of overeating. Indeed, DiLeone, Taylor, and Picciotto (2012) argue that brain reward networks have evolved to promote feeding and avoid starvation, and that these reward mechanisms are exploited by commonly abused drugs. Similarly, Wise (2013) points out that, given that the misuse of psycho-active chemicals is a relatively recent behaviour, it is unlikely that separate systems have evolved for the development of substance-dependence.

1.2.4.4. Which foods are addictive?

Finally, if we are to consider food addiction as a ‘substance based’ addiction, it is necessary to determine the specific substances in food that are ‘addictive’. One recent survey found that processed foods high in sugar, fat, and salt are most frequently reported as ‘problematic’ or ‘addictive’ by members of the lay public (Schulte, Avena, & Gearhardt, 2015), and this may be due to their ability to be readily absorbed into the bloodstream (Schulte et al., 2015). High-fructose corn syrup (HFCS) is a very sweet carbohydrate which is also thought to enhance the addictive potential of foods (Davis, 2013; Kenny, 2011) due to its ability to enhance foods’ palatability, and block insulin and leptin satiety signals (Haring & Harris, 2011; Sato et al., 1996).

Much of the evidence for the addictive potential of sugar, fat, and salt has been derived from animal models. For example, rats that were provided with intermittent access to foods high in fat and/or sugar engaged in binge eating and tolerated more aversive situations in order to obtain food (Avena & Gold, 2010; Avena et al., 2008; Teegarden et al., 2008; Teegarden & Bale, 2007). Further, bingeing on foods high in fat and/or sugar has been found to increase dopamine release in the NAc (Avena et al., 2006; Liang, Hajnal, & Norgren, 2006), although endogenous opioid release, and opiate-like withdrawal symptoms, occurs only following sugar (not fat) bingeing (Bocarsly, Berner, Hoebel, & Avena, 2011; Colantuoni et al., 2002). It is important to note, however, that animals fed an intermittent diet high in fat *or* sugar decreased their consumption of regular chow and thus did not gain weight (Avena & Gold, 2010; Avena et al., 2008; Dimitriou, Rice, & Corwin, 2000; Lomba et al., 2009). Weight gain was observed only in animals that were fed a diet high in fat *and* sugar, yet this combination of food did

not result in the characteristic withdrawal symptoms of addiction (Bocarsly et al., 2011).

In their ‘salted food hypothesis’, Cocores and Gold (2009) propose that foods high in salt act as opiate agonists in the brain. This is supported by findings of increased salt consumption following the administration of an opiate agonist within the NAc in rats (Zhang & Kelley, 2002). However, contrary to its addictive potential, salt consumption was unaffected following dopamine pathway lesions (Shibata et al., 2009), and no taste preference for salt was observed in sodium depleted rats (Clark & Bernstein, 2006). As such, Hebebrand et al. (2014) suggest that there is little evidence that salt is, in itself, rewarding. Rather, a preference for high-salt foods may arise following a conditioned learning process, and not an innate hedonic drive.

Thus, while certain palatable foods may be rewarding for both animals and humans, the nutritional complexity of such foods makes it difficult to identify any specific addictive substance. Indeed, overeating is usually facilitated by the availability of a *range* of foods (Rolls, Van Duijvenvoorde, & Rolls, 1984), and the conceptualisation of a substance-based food addiction is limited by the fact that the consumption of a single ingredient (e.g. sugar), is rarely observed.

1.2.5. Food addiction as a behavioural addiction

An alternative perspective is that fat and sugar are not addictive *per se*, but addictive patterns of eating may arise following intermittent access to these highly palatable foods (Corwin & Grigson, 2009). Indeed, Hebebrand et al. (2014) suggest that certain patterns of overeating may be best conceptualised as a behavioural ‘*eating* addiction’ rather than a substance-based ‘*food* addiction’, and point out that ‘addiction’ does not necessarily require exposure to exogenous chemicals. Rather, Hebebrand et al. (2014) suggests that the endogenous release of dopamine and opioids in response to eating may, in themselves, generate compulsive eating. Distinct from binge eating, Hebebrand et al. (2014) suggest that an eating addiction is not necessarily characterised by isolated bouts of overeating, but may manifest itself as constant snacking or ‘grazing’. Indeed, grazing behaviour has been associated with increased mental health problems and poorer weight-loss outcomes following bariatric surgery (Nicalau et al., 2015; Robinson et al., 2014; Sheets et al., 2015).

The conceptualisation of food addiction as a behavioural, rather than substance-based, addiction is consistent with commonly held beliefs amongst members of the lay public (Malika, Hayman, Miller, Lee, & Lumeng, 2015). Specifically, using semi-structured interviews to investigate beliefs about food addiction amongst low-income women, Malika et al. (2015) reported that food addiction was commonly characterised by a compulsive need to always have food available, and a tendency to go out of one's way to obtain food. A behavioural view of food addiction also yields implications for its inclusion within future editions of the DSM, which now recognises the existence of non-substance based addictions, such as gambling.

1.2.6. Food addiction as a self-serving attribution: psychosocial account

Given the limited evidence in support of a 'food addiction' model, an alternative possibility is that the concept may be used as a self-serving attribution for overeating and weight gain. In their psychosocial account, Rogers and Smit (2000) propose that labelling oneself a 'food addict' may arise following failed attempts to restrict one's intake of highly palatable but unhealthy ('naughty but nice') foods. By insinuating that such lapses in self-control are the result of a physiological inevitability, Rogers and Smit (2000) suggest that the concept of food addiction may help to alleviate feelings of guilt and personal responsibility.

This perspective is in accordance with the tenets of Attribution Theory (Weiner, 1971; 1974) which provides a framework by which to conceptualise individuals' explanations for positive and negative outcomes. According to this theory, explanations (or *attributions*) for outcomes can be classified along the following three domains: 1) *Locus*: attributions may be either external (i.e. outside of the individual) or internal (i.e. intrinsic to the individual), 2) *Controllability*: attributions can be either *controllable* or *uncontrollable*, and 3) *Stability*: attributions may be regarded as *permanent* or *temporary*. Attribution theory accounts for a phenomenon known as the self-serving bias, whereby undesirable outcomes are attributed to *external* and *uncontrollable* causes, such as biological or environmental influences, while the contribution of *internal* and *controllable* factors, such as personal choice, are minimised (Sedikides & Strube, 1995).

Using this framework, Davies (2013) suggest that the concept of 'addiction' may be used as a self-serving explanation which helps to portray the drug user or

overeater as a ‘helpless victim of disease’. Indeed, with regards to obesity and weight gain, there is some evidence to suggest that biological and addiction based explanations may be effective in reducing stigmatization and perceptions of blame (Crandall, et al., 2014; Latner, Puhl, Murakami, & O’Brien, 2014; Pearl & Lebowitz, 2014). In particular, Latner et al. (2014) found that a ‘food addiction’ based explanation for obesity led to less stigma and blame towards obese individuals, compared to a non-addiction based explanation that emphasised the role of personal control. Conversely, causal attributions for obesity that emphasise the role of behavioural choice (i.e. lack of exercise and an unhealthy diet) have been found to increase blame and obesity-related stigma, relative to environmental and biological explanations (Pearl & Lebowitz, 2014).

There is also evidence to suggest that attributions which emphasise the role of uncontrollable influences may be used to minimize feelings of guilt and negative affect following overeating. For example, one study found that self-reported emotional eaters, who were led to believe they had overeaten, were more likely to attribute their eating to negative emotions compared to those who did not identify as ‘emotional eaters’ (Adriaanse et al., 2016). In another study (Adriaanse, Weijers, De Ridder, De Witt Huberts, & Evers, 2014), participants were primed with food enjoyment words (to induce overeating), or neutral words, during a lexical decision task, and subsequently took part in an *ad libitum* taste test. Participants then read an article which provided an external reason for eating (i.e. that concentrating on a task can increase sugar cravings). The researchers found that participants who had high dieting standards, and who were exposed to the food enjoyment prime, experienced greater negative affect following the taste test and were more likely to confabulate reasons for eating using the information provided in the article (i.e. that their eating was caused by concentrating on the task).

Taken together, these findings support the possibility that feelings of eating-related guilt may provide a stronger antecedent to food addiction attributions, than *actual* eating behaviours *per se*. In particular, individuals who feel guilty following eating may be more likely to perceive themselves as food addicts and/or attribute their eating to the addictive qualities of food. However, research is yet to investigate this possibility.

1.2.7. The consequences of food addiction beliefs

Finally, as Carter et al. (2016) suggest, it is important to consider the way in which beliefs about food addiction may affect food intake and health behaviours. From one perspective, by implying that food intake is beyond personal control, perceiving oneself to be a food addict may have detrimental effects on dietary regulation. Indeed, self-control beliefs have consistently been found to be important in the initiation and maintenance of variety of healthy behaviours (e.g. Steptoe & Wardle, 2001), and Hoyt et al. (2014) found that leading people to believe that ‘obesity is a disease’ caused people to make more unhealthy food choices. Similarly, Dar-Nimrod et al. (2014) reported increased food intake in individuals who had been led to believe that genetics played a predominant role in weight gain and obesity. However, the concept of food addiction does not necessarily imply a lack of control over food intake. Indeed, one survey found that food addiction was perceived as more of a disease than smoking, but less of a disease than alcoholism (DePierre et al., 2014). These results suggest that food addiction may be regarded as being somewhat under personal control.

Another possibility is that the concept of food addiction may encourage people to abstain from foods that are deemed as particularly problematic. This has been supported by qualitative explorations of the experiences of members of overeating self-help groups. In particular, members of ‘Overeaters Anonymous’ are encouraged to recognise themselves as ‘food addicts’ and to avoid exposing themselves to their ‘problem foods’, in much the same way as members of ‘Alcoholics Anonymous’ are encouraged to abstain from alcohol. Studies have reported that ‘food addiction’ beliefs are crucial to members’ recovery from problematic or binge eating tendencies (Ronel & Libman, 2003; Russell-Mayhew et al., 2010).

The idea of food addiction as a helpful concept is also consistent with ‘restraint bias’ theory (Nordgren, van Harreveld, & van der Pligt, 2009) which proposes that holding unrealistic expectations of our ability for self-control may have detrimental effects on health behaviours. In one study (Nordgren et al. 2009), cigarette smokers were led to believe that they had either a high- or low- ability for self-control (i.e. high self-control/low self-control conditions). Participants were then offered a financial incentive to refrain from smoking, and could choose the level of

temptation to which they would be exposed. The more tempting the scenario, the more money the participants could win. Nordgren et al. (2009) found that participants in the high self-control condition exposed themselves to more tempting scenarios than those in the low-self-control condition, and were more likely to smoke as a result. Similar effects of self-control beliefs have been observed for snack selection, and alcohol consumption (Nordgren et al., 2009, Jones, Cole, Goudie, & Field, 2012).

Drawing on the above, it remains unclear whether beliefs about food addiction would exert a helpful or counter-productive effect on eating behaviour. This is an important consideration given the popularity of the concept throughout the lay public (Lee et al., 2013), and yields important implications for the implementation of ‘food addiction’ messages within dietary interventions.

1.3. The current thesis

As discussed, there is currently a lack of scientific consensus regarding the characteristics and definitions of addictive eating. To address this, the first aim of the current thesis was to identify the eating related cognitions and behaviours which characterise those who perceive themselves to be addicted to food (i.e. self-perceived food addicts). The decision to focus on self-perceived food addicts, rather than those who fulfil a diagnostic measure of food addiction (i.e. the YFAS), is consistent with the recommendation that an exploration of the food addiction concept should go beyond existing models of substance dependence (Ziauddeen et al., 2012). Indeed, as previously discussed, the use of the YFAS for the assessment of ‘food addiction’ is limited by fundamental differences between the neurobiological and societal effects of drugs and food, and its validity is heavily dependent upon the validity of the food addiction construct itself. Thus, by establishing the core behavioural features of self-perceived food addiction, the current thesis provides insight into the food addiction concept in such a way that is not limited by the applicability of the substance-dependence framework to eating behaviour.

Aim 1: To establish a cognitive and behavioural profile of self-perceived ‘food addiction’ (Chapters 2-5).

Chapter 2

The first aim of the thesis was addressed in Chapters 2-5. Firstly, Chapter 2 presents a qualitative exploration of the characteristics of self-perceived food addicts. In this study, participants completed a brief online questionnaire in which they were asked to indicate *why* they did or did not perceive themselves to be ‘food addicts’. In doing so, the study described in Chapter 2 identified several core behaviours which were believed to characterise ‘food addiction’ amongst members of the lay public.

Chapter 3

Drawing upon findings from Chapter 2, Chapter 3 presents a study that empirically explored food reward (using desire-to-eat, and willingness-to-pay ratings, and an operant response task) and calorie intake, in self-perceived food addicts and non-addicts. It was predicted that self-perceived food addicts would demonstrate increased food reward, particularly when satiated, compared to non-addicts, and would consume more calories during *ad libitum* access. These between-group differences were expected to be most pronounced towards high-fat, rather than low-fat, foods.

Chapter 4

As previously discussed (section 1.1.2), the underlying reward value of a food can be indirectly assessed by examining the extent to which individuals demonstrate elevated attention towards cues which signify its availability (e.g. food pictures) (Field et al., 2016). Thus, based on findings from Chapters 2 and 3, Chapter 4 incorporated an eye-tracking procedure to explore whether self-perceived food addicts would demonstrate increased attentional bias to chocolate pictures, relative to neutral stimuli, compared to self-perceived non-addicts. Chapter 4 also explored whether self-perceived food addiction would moderate the effect of two established state determinants of attentional bias to food cues; hunger (e.g. Castellanos et al., 2009) and the perceived availability food (Jones et al., 2012).

Chapter 5

Findings from Chapters 2 to 4 were used to inform the development of a novel tool for the assessment of addictive eating (i.e. the Addictive Eating Behaviour

Scale, AEBS) which is presented in Chapter 5. The AEBS quantifies the presence of behaviours and cognitions that are commonly associated with addictive-like eating . Through using an inductive approach to identify scale items, the AEBS overcomes some of the limitations of an existing measure of food addiction (i.e. the YFAS, Gearhardt et al., 2009). Specifically, unlike the YFAS, the validity of the AEBS is not constrained by the limited applicability of the substance-dependence criteria to eating behaviour.

Aim 2. To examine the causes and consequences of food addiction beliefs

Drawing upon psychosocial perspectives (discussed in section 1.2.6.), a second aim of the current thesis was to examine the causes and consequences of food addiction *beliefs*. This was addressed in Chapters 6 and 7.

Chapter 6

Chapter 6 presents two studies which aimed to establish whether self-perceived food addiction would have a helpful or counterproductive effect on subsequent eating behaviour. From one perspective, believing oneself to be a food addict may be expected to *increase* food consumption due to reduced personal responsibility for eating. Alternatively, self-perceived food addiction beliefs may cause a person to be concerned about their eating behaviour and consume *less* snack food as a result.

Chapter 7

Chapter 7 then addressed suggestions that the concept of food addiction may be used to provide a more socially and personally acceptable attribution for eating-related guilt (Rogers & Smit, 2000). Following *ad libitum* consumption of a buffet lunch, levels of eating-related guilt were manipulated. It was predicted that individuals who were led to feel guilty following eating (high-guilt condition) would be more likely to perceive themselves to be food addicts, and to attribute their eating to the ‘addictive qualities of the foods’, than those in low-guilt and control conditions.

Chapter 7 also examined the effect of the guilt condition on snack selection. Drawing upon findings from Chapter 6, it was predicted that those in the high-guilt condition would choose less tempting snacks, than those in the low-guilt and control conditions, when given a monetary incentive to return the snack, uneaten, one week

later. The relationship between self-perceived food addiction, dietary concern, and snack selection was also explored.

To summarise, the current thesis encompassed two primary aims: 1) To establish a cognitive and behavioural profile of self-perceived ‘food addiction’, and 2) to examine the causes and potential consequences of food addiction *beliefs*. See Figure 1.4. for an overview of the structure and aims of the thesis.

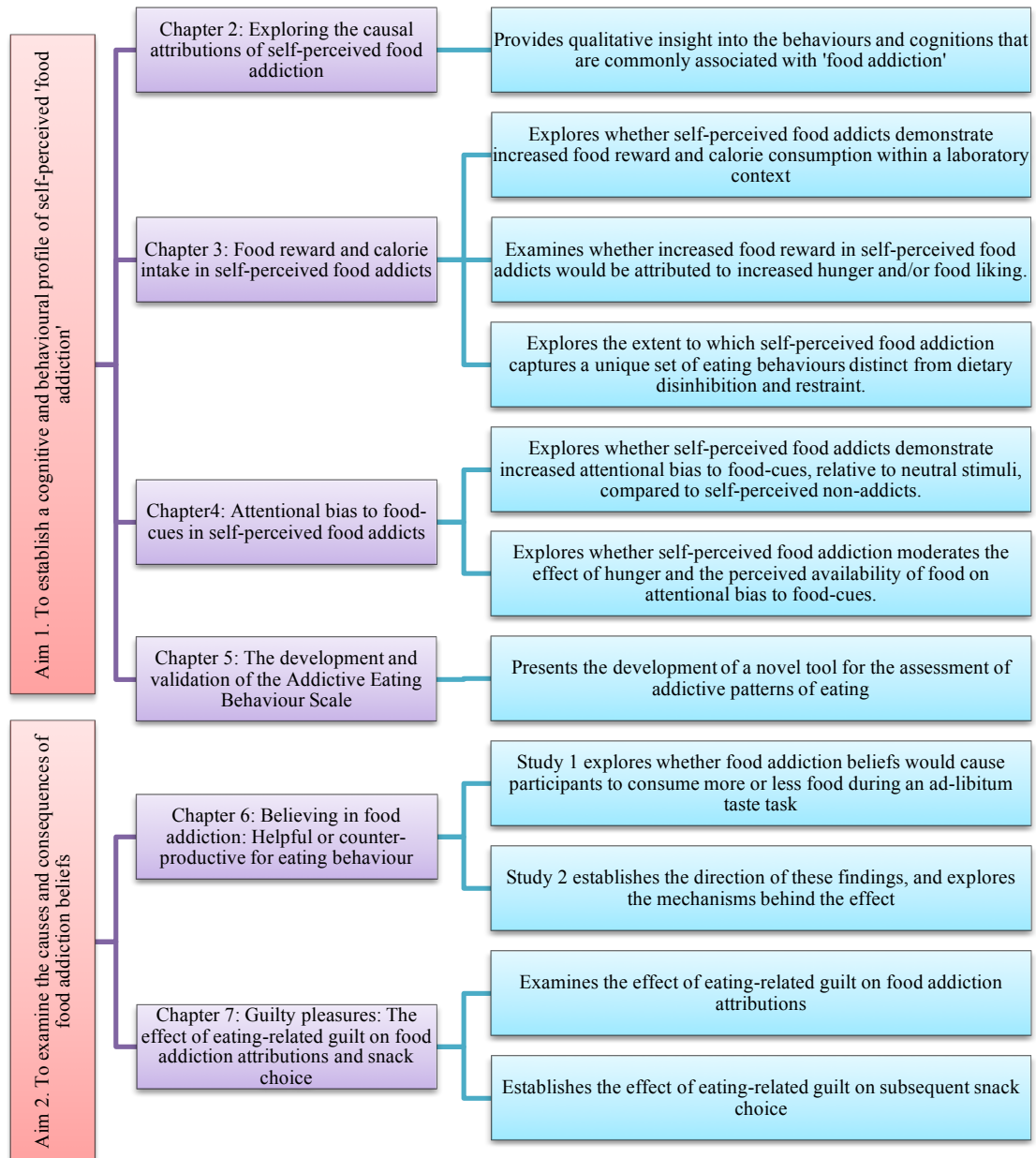


Figure 1.4. Thesis overview. Thesis aims are presented in the orange boxes, chapter headings are in purple boxes, and the individual aims of each chapter are presented in the blue boxes.

Chapter 2: Exploring the causal attributions of self-perceived food addiction.

2.1. Overview

Current conceptualisations of food addiction are dependent upon the DSM criteria for substance dependence (Gearhardt et al., 2009). However, the extent to which symptoms of substance dependence can be appropriately applied to the assessment of eating behaviour is somewhat limited (Ziauddeen et al., 2012). As such, Ziauddeen et al. (2012) posit the need to develop an operational definition of food addiction that is not based on current conceptualisations of addictive behaviour. This was addressed in Chapter 2 by qualitatively examining commonly held beliefs about the manifestations of food addiction within a community sample.

The study reported in this chapter has been published as:

Ruddock, H.K., Dickson, J.M., Field, M., & Hardman CA. (2015). Eating to live or living to eat? Exploring the causal attributions of self-perceived food addiction. *Appetite*, 95, 262–268.

2.2. Abstract

Previous studies indicate that many people perceive themselves to be addicted to food. However, little is known about how the concept of ‘food addiction’ is defined amongst members of the lay public. The current study examined beliefs about the cognitive and behavioural manifestations of food addiction. Participants ($N = 210$) completed an internet-delivered questionnaire in which they indicated whether or not they perceived themselves to be a food addict and they provided a brief explanation for their response. Over a quarter of participants (28%) perceived themselves to be food addicts and self-diagnosis was predicted by increased BMI and younger age, but not by gender. Thematic analysis was conducted to explore the causal attributions provided by self-perceived food addicts and non-addicts. Six characteristics were identified: 1) Reward-driven eating (*i.e.*, eating for psychological rather than physiological reasons), 2) A functional or psychological preoccupation with food, 3) A perceived lack of self-control around food, 4) Frequent food cravings, 5) Increased weight or an unhealthy diet, and 6) A problem with a specific type of food. The emergent themes, and their frequency, did not differ

between self-perceived food addicts and non-addicts. However, self-perceived food addicts and non-addicts reported divergent cognitions, behaviours and attitudes within each common theme. This study is the first to provide qualitative insight into beliefs about food addiction in both self-perceived food addicts and non-addicts. The findings appear to reflect a view of food addiction that is identifiable through several core behaviours.

2.3. Introduction

The notion of ‘food addiction’ has gained widespread media attention and public support for its existence appears to be strong (Barry, Brescoll, Brownell, & Schlesinger, 2009; Bird, Murphy, Bake, Albayrak, & Mercer, 2013; Lee et al., 2013; Wilson et al., 2009). In one study, 86% of Australians and Americans endorsed the idea that some foods have addictive properties and 72 percent believed that food addiction could account for some cases of obesity (Lee et al., 2013). More recently, it has been shown that a substantial proportion (42%-52%) of people from community samples perceive themselves to be addicted to food (Hardman et al., 2015; Meadows & Higgs, 2013). In these studies, this ‘self-perceived’ food addiction was assessed simply by asking participants to indicate whether or not they believe themselves to be addicted to food. It has also been shown that self-perceived food addiction is associated with eating pathology, weight concerns, dieting behaviour, and internalised weight stigma (Meadows & Higgs, 2013).

In contrast, the extent to which compulsive overeating is akin to a substance-based addiction remains heavily debated within the scientific community (Rogers & Smit, 2000; Ziauddeen, Farooqi, & Fletcher, 2012; Ziauddeen & Fletcher, 2013;). Nonetheless, the Yale Food Addiction Scale (YFAS; Gearhardt, Corbin, & Brownell, 2009) enables quantification and diagnosis of ‘food dependence’ based on the DSM-IV criteria for substance dependence (e.g., substance taken in larger amount than intended, persistent desire to quit, tolerance). Using this measure, the prevalence of food addiction was found to be around 15% for adults seeking weight loss treatment (Eichen, Lent, Goldbacher, & Foster, 2013), and between 5% to 7% within non-clinical populations (Meadows & Higgs, 2013; Pedram et al., 2013). Interestingly, previous studies indicate a substantial mismatch between the number of participants who are classified as food dependent on the YFAS (7%-8%) and those who self-

diagnose (42%-52%) (Hardman et al., 2015; Meadows & Higgs, 2013). This finding implies that, for many people, their own interpretation and experience of food addiction is not consistent with the substance dependence model proposed by the YFAS.

On this basis, an important avenue for research concerns the identification of specific eating behaviours that are attributed to self-perceived food addiction amongst members of the lay public. In a qualitative study by Ifland et al. (2009), interview questions from the DSM-IV criteria for substance dependence were adapted to explore eating behaviours in a group of self-perceived food addicts. These participants reported requiring greater amounts of food over time, a tendency to engage in emotional eating, consuming more food than intended, unsuccessful attempts to cut down on certain foods, and spending a lot of time obtaining food, eating, or recovering from the effects of overeating. Ifland et al. (2009) interpret these findings as demonstrating an overlap between the eating patterns of self-perceived food addicts and the clinical criteria for substance dependence. However, applying a substance dependence model to over-eating in this way may be premature given the lack of concrete evidence for any specific addictive substance in food. Indeed, it has been suggested that many of the DSM-IV substance dependence criteria are not easily applicable to eating behaviour given the availability and necessity of food (Ziauddeen et al., 2012). Furthermore, as noted above self-perceived food addiction and the YFAS diagnosis of food dependence often do not coincide, and the reason for this discrepancy is not clear.

To explore the manifestations of self-perceived food addiction, it may therefore be more appropriate to use a qualitative framework that is not guided by any prior theory of food addiction. To our knowledge, very few studies have employed this inductive approach in the current context. Hetherington and Macdiarmid (1993) found that the majority of self-perceived chocolate addicts attributed their addiction to an inability to resist chocolate. Participants also made causal attributions regarding the amount of chocolate they consumed, or having been labeled as a 'chocolate addict' by others.

Given the current lack of knowledge around self-perceived food addiction, the primary aim of the current study was to examine beliefs about the cognitive and behavioural manifestations of food addiction amongst members of the lay public. We adopted a similar inductive approach to Hetherington and Macdiarmid (1993) in

order to build on and extend these initial findings. Participants completed a short internet-delivered questionnaire in which they were asked to indicate whether or not they perceived themselves to be a food addict. They were then asked to provide a brief explanation for their response. A potential issue was that participants may be unfamiliar with the term ‘food addiction’ and hence might find it difficult to respond. For this reason, a secondary aim was to determine whether it is necessary to provide a definition of food addiction prior to administering a measure of self-perceived food addiction. Thus, before indicating whether they perceived themselves to be a food addict, half of participants read a short definition of food addiction, and half of participants received no information.

2.4. Method

2.4.1. Participants

A total of 210 participants (males, $n = 65$; females, $n = 145$) completed an internet-delivered questionnaire. Participants were aged between 18 and 62 years (mean age = 29.0 ± 11.5 years) and ranged from underweight (15.8 kg/m^2) to obese (37.3 kg/m^2) (mean BMI = 23.5 ± 4.0). The questionnaire was advertised on an internal website at the University of Liverpool and was accessible to staff and students. The study was approved by the University of Liverpool Research Ethics Committee. Participants provided written informed consent prior to completing the questionnaire.

2.4.2. Materials and Procedure

An internet-based questionnaire was developed using the resources available at www.qualtrics.com. Once they had given their consent, participants were randomly allocated to one of two conditions; (1) a “no information” condition ($n=104$), in which no information about food addiction was provided, or (2) an “information” condition ($n=106$), in which participants read a brief paragraph which provided the following definition of food addiction:

“People sometimes have difficulty controlling their intake of certain foods. One theory of why people overeat is that foods high in fat and sugar are addictive. In support of this, research in animals and humans suggests that certain foods activate similar brain areas to drugs of abuse. It is believed that addiction to food can be

experienced by anyone, regardless of weight. For example, food addiction may be experienced as persistent craving for food as well as spending a lot of time thinking of, purchasing, preparing and eating food despite knowledge that this is unhealthy.”

The aim here was to test whether providing participants with this information would influence the subsequent measure of self-perceived food addiction. For example, participants may be unfamiliar with the term ‘food addiction’ and so require this clarification in order to effectively self-diagnose themselves. The information included in the paragraph was adapted from current expert consensus on food addiction provided by the Neurofast project (<http://www.neurofast.eu/consensus>).

To assess self-perceived food addiction, participants were then asked “Do you agree with the following statement: ‘I believe myself to be a food-addict?’” to which they could respond either “Yes”, “No”, or “I don’t know”. To provide insight into the causal attributions of food addiction, participants were then asked the following: “Please let us know why you do/do not perceive yourself to be a food addict. If you answered ‘I don’t know’, please let us know why you gave this response”. Participants were free to write as much as they wished in response to this question.

Finally, participants were asked to provide their age, gender, weight (in kg or stones), and height (in m or feet). On completion, participants were debriefed and thanked for their time.

2.4.3. Data analysis

2.4.3.1. Quantitative data

A chi-square test was used to compare the frequency of responses on the self-perceived food addiction measure (*i.e.*, “Yes”, “No” and “I don’t know”) between the information and no information conditions. In line with the study aims, we were particularly interested to see whether the frequency of indecisive (*i.e.*, “I don’t know”) responses would be reduced among participants who had read a definition of food addiction (*i.e.*, information condition) relative to participants who had received no information.

For subsequent analyses, participants who had indicated an indecisive (*i.e.*, “I don’t know”) response ($n = 26$) were excluded. This was so that we could directly compare the causal attributions made by self-perceived food addicts with those made by non-addicts. A chi-square test was conducted to explore whether self-perceived food addicts differed according to gender. Differences in age and BMI between self-perceived food addicts and non-addicts were explored using independent samples *t*-tests.

2.4.3.2. Qualitative data

Theme extraction

Thematic analysis was used to explore causal attributions given by self-perceived food addicts and non-addicts. Thematic analysis provides a flexible method of identifying recurring responses within qualitative datasets (Braun & Clarke, 2006). An inductive analytic approach was used which was not guided by any theoretical account of food addiction. Inductive methods of analysis generate themes that are closely related to the data (Patton, 1990). As such, this approach was selected to maintain an accurate reflection of participants’ causal attributions and the emergence of themes rather than relying on theoretically predefining themes. The data were read several times by one researcher (HKR) before identifying initial codes and/or subthemes for each line of text or meaningful textual units.

Next, codes and subthemes were clustered at a higher level of abstraction into overarching themes. Each theme captured both convergent and divergent views about food addiction. For example, attributions that related to both high- and low- self-control were categorised under the ‘self-control’ theme. Figures 2.1 and 2.2 illustrate the codes and sub-themes that were assigned to each overarching theme for self-perceived food addicts and non-addicts, respectively. To check the validity of the themes identified, a second researcher (CAH) independently reviewed the coding procedures from initial codes to overarching themes. A third researcher (JMD) reviewed the final thematic analysis. There was complete agreement among the researchers on the validity of the final themes identified.

Frequency analysis

The frequency of each theme was calculated separately for self-perceived food addicts and non-addicts by one researcher (HKR). As in previous research (Carnell, Cooke, Cheng, Robbins, & Wardle, 2011), an all-or-nothing approach was employed such that, for each participant, each theme was assigned a score of 0 if it was not mentioned, or 1 if it was mentioned at least once. A second researcher (CAH) independently coded a sub-sample (20%) of the responses. Agreement between researchers (HKR and CAH) was then assessed on a case-by-case basis. This produced a mean level of 95% agreement between raters ($K = .95$), which exceeded the 80% target inter-rater reliability score. Where discrepancies were identified, these were discussed and a consensus reached ($K=1$).

Next, we assessed whether the frequency of participants who reported each theme could be predicted by condition (*i.e.*, information *vs.* no information). We also assessed whether reference to each theme could be predicted by gender, BMI, or age. Logistic regression analyses were conducted for each theme in turn, where condition was entered into model 1, and gender, BMI, and age were entered into model 2. The presence or absence of each theme was represented by a score of 1 or 0, respectively, and the participants were collapsed across both self-perceived addicts and non-addicts.

Finally, an ANOVA was conducted to ensure that there was no effect of condition (*i.e.*, information *vs.* no information) on the length of participants' responses (*i.e.* word count) and the number of themes produced.

2.5. Results

2.5.1. Quantitative data

A chi-square test revealed that responses to the assessment of self-perceived food addiction did not differ between conditions, $X^2(2) = 1.72, p = .424$ (Table 2.1). This result indicates that providing participants with a definition of food addiction did not significantly influence self-diagnosis.

The remaining sample (with indecisive responses removed) consisted of 61 males and 123 females (BMI, Mean = 23.6 ± 4.1 kg/m²; Age, mean = 29.1 ± 11.9 years). Of these, 59 participants (32%) identified as a food addict. The likelihood of

identifying as a food addict did not differ between males and females, $X^2(1) = .39, p = .408$. BMI was significantly higher for those who identified as a food addict, $t(155.65) = 2.45, p = .015$, and age was significantly lower, $t(91.03) = -1.99, p = .05$ (see Table 2.2).

Table 2.1. Frequency (percentages in parentheses) of self-perceived food addicts in the information and no information conditions.

Condition	Self-perceived food addict			Total
	“Yes”	“No”	“I don't know”	
Information	31 (29%)	65 (61%)	10 (9%)	106
No information	28 (27%)	60 (58%)	16 (15%)	104
Total	59 (28%)	125 (60%)	26 (12%)	210

Table 2.2. Participant characteristics for self-perceived food addicts ($n=59$) and self-perceived non-addicts ($n=125$). Values are means \pm SDs unless otherwise stated.

Characteristic	Food addicts	Non-food addicts
Age (y)	26.5 \pm 9.0	30.4 \pm 12.8*
BMI (kg/m ²)	24.6 \pm 4.7	23.2 \pm 3.7*
Female (%)	34%	66%
Male (%)	28%	72%

*significantly different from food addicts, $p < .05$.

2.5.2. Qualitative data

Six non-addicts and three addicts failed to provide a reason for their response. Thus qualitative analyses were conducted on responses obtained from the remaining 175 participants (addicts = 56, non-addicts = 119). Of these, only one participant (non-addict) indicated that they did not agree “that someone can be addicted to food”.

2.5.2.1. Themes and definitions

Six key ‘themes’ were identified (see Figures 2.1 and 2.2 for an illustration of the codes and sub-themes that were assigned to each overarching theme for self-perceived food addicts and non-addicts, respectively):

1. **Reward-driven eating** (eating in response to psychological rather than physiological cues);

2. **Preoccupation** (reference to the amount of time spent thinking about, preparing or eating food or the significance of food in everyday life);
3. **Self-control** (one's perceived ability to control food intake);
4. **Cravings** (intense desire-to-eat a particular food or type of food);
5. **Health** (reference to health or weight status, or the healthiness of food consumed);
6. **Specific foods** (a problem with a particular food or food group).

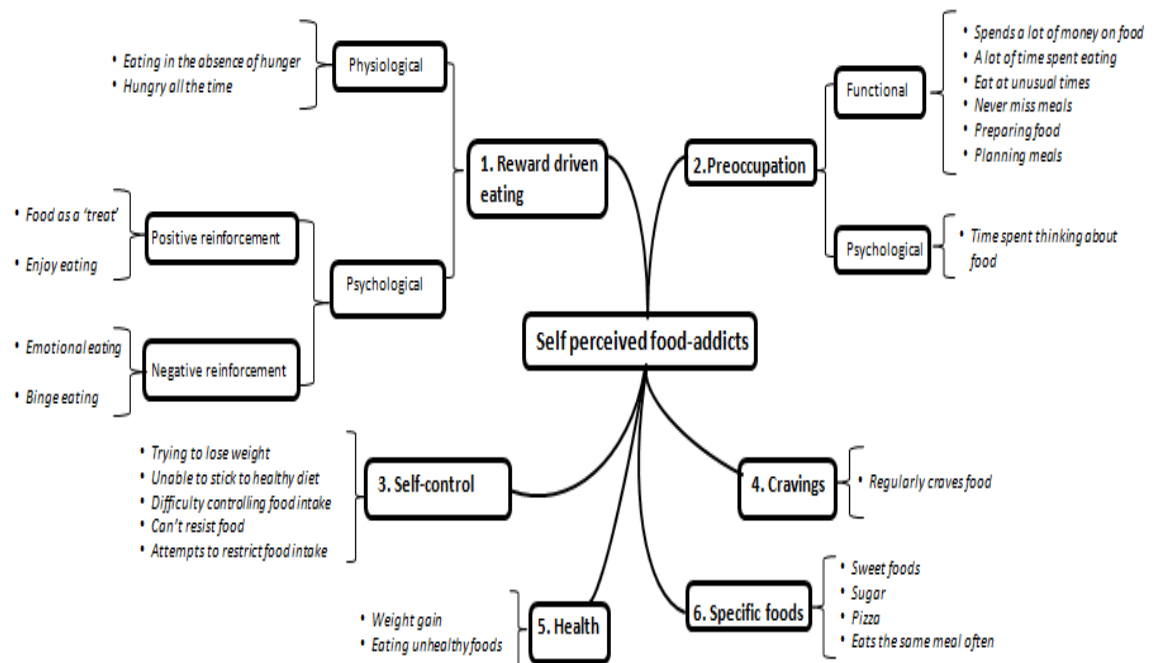


Figure 2.1. Codes and sub-themes assigned to each theme for self-perceived food addicts. Themes are numbered and in bold, sub-themes are in non-italics, and codes are bullet-pointed and provided in italics.

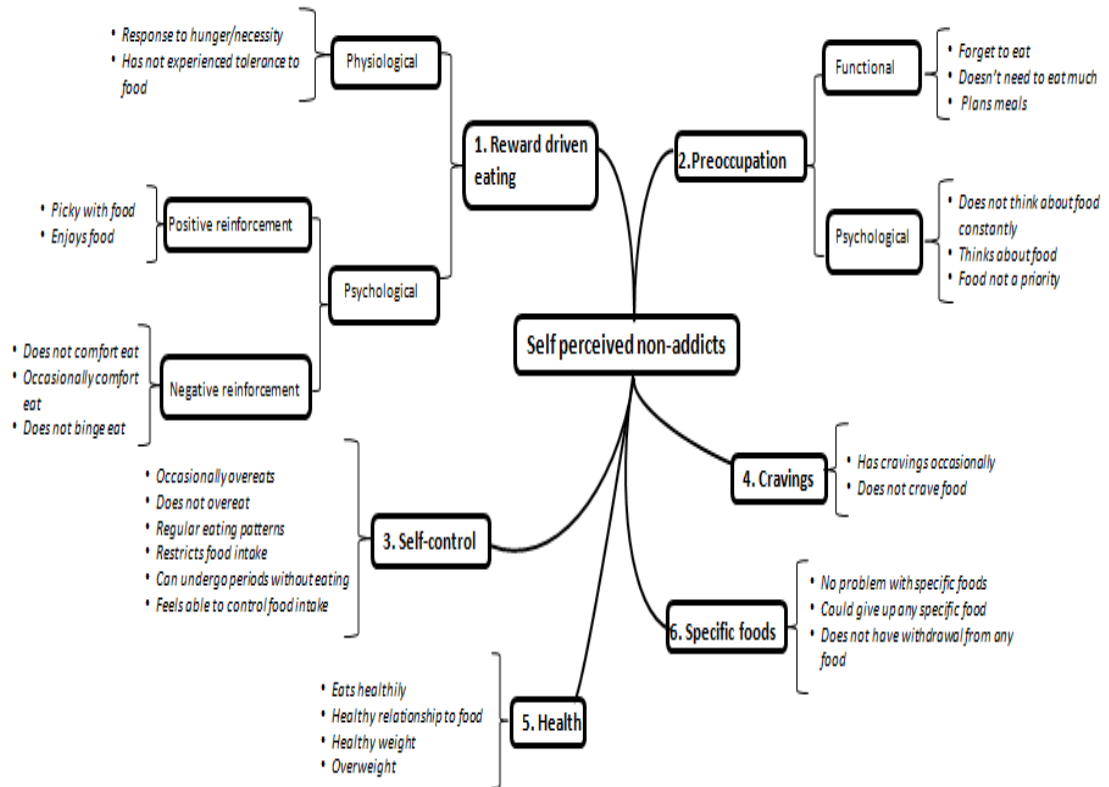


Figure 2.2. Codes and sub-themes assigned to each theme for self-perceived non-addicts. Themes are numbered and in bold, sub-themes are in non-italics, and codes are bullet-pointed and provided in italics.

2.5.2.2. Frequency analyses

The frequency of each theme for self-perceived food addicts and non-addicts is provided in the theme descriptions below. Regression analyses revealed that, compared to males, females were more likely to make causal attributions that referred to reward-driven eating ($B = -.83, SE = .34, p = .015, \text{Exp}(B) = .43$). Age, BMI, and condition (*i.e.*, information vs. no information) did not predict more frequent reference to any theme (all p -values $> .05$). ANOVA revealed no differences between the length of responses given in the two conditions (Mean word count (SD): information = 19.53 (14.08); no information = 16.25 (9.44), $F(1,173) = 3.21, p = .075$) nor the number of themes generated (mean theme count (SD): information = 1.71 (0.84); no information = 1.64 (0.81), $F(1,173) = .40, p = .529$). Thus, responses from each condition were grouped and analysed concurrently.

1. Reward-driven eating. Both self-perceived food addicts ($n = 23$, 41%) and non-addicts ($n = 55$, 46%) made causal attributions that referred to reward-driven eating. Responses within this theme were further categorised into ‘physiological’ or ‘psychological’ sub-themes.

Responses that referred to eating in response to hunger or physiological need were grouped under the ‘physiological’ sub-theme. A similar proportion of self-perceived food addicts ($n = 14$, 25%) and non-addicts ($n = 34$, 29%) made causal attributions that were categorised in this way. The majority of these responses referred to the tendency to eat in the absence of physiological hunger. Food addicts and non-addicts reported divergent eating behaviours; specifically, while food addicts reported eating “even when I’m not hungry”, non-addicts reported only eating in response to physiological need (“I eat to live, not live to eat”). In contrast, a minority ($n = 2$) of self-perceived food addicts indicated an increased physiological drive to eat (*e.g.*, “often hungry”; “always feel the need to eat”).

Responses within the ‘psychological’ sub-theme were categorised into two further sub-themes, ‘positive reinforcement’ (*i.e.*, eating for pleasure) and ‘negative reinforcement’ (*i.e.*, binge eating or comfort eating).

With regard to positive reinforcement, food addicts ($n = 9$, 16%) reported viewing food as a source of pleasure and enjoyment (*e.g.*, “I feel like I have to have something “treat” like all the time”). Non-addicts ($n = 24$, 20%) also indicated enjoyment towards food however, alongside this, they tended to report being “very picky with food”. While addicts tended to say that they enjoyed the activity of eating (“I like to eat”), non-addicts indicated a greater enjoyment towards the food itself (“I enjoy food”). This suggests that self-perceived food addicts may enjoy the behavioural aspect of eating, while non-addicts may demonstrate greater enjoyment to the sensory qualities of food.

With regard to negative reinforcement, both food addicts ($n = 5$, 9%) and non-addicts ($n = 8$, 7%) attributed food addiction to negatively reinforced eating habits such as emotional eating and binge-eating. Again, food addicts and non-addicts reported divergent eating behaviours; while food addicts reported eating in response to negative emotions (“I also go back to favourite foods when I am sad/happy”) and a tendency to “binge eat”, the majority of non-addicts said that they “don’t use food as a crutch”, and “do not compulsively overeat”.

2. Preoccupation. A similar proportion of addicts ($n = 25$, 45%) and non-addicts ($n = 46$, 39%) made causal attributions that referred to an excessive investment of time, money and effort into thinking about, preparing and eating food (“I perceive food addicts to be those who love to cook and explore different types of food and always have food on their mind” – non-addict).

Responses within this theme were further categorised as having either psychological (*i.e.*, thinking about food) or functional (*i.e.*, cooking, preparing, eating, or spending money on food) components. Again, self-perceived food addicts and non-addicts tended to report opposite experiences. With regards to psychological preoccupation, self-perceived food addicts ($n = 16$, 29%) indicated that they “think about food all the time”, while non-addicts ($n = 41$, 34%) reported having “little interest in food” and “don’t spend a large proportion of the day thinking about consuming food”. While a small number of non-addicts did report “think[ing] about food a lot”, they did not perceive themselves to be an addict as they “eat healthily in general” and “do not compulsively overeat”. Several non-addicts believed that food addiction involves an “obsession” or a tendency for food to dominate one’s life (“my life is not ruled by food”).

With regard to functional preoccupation, self-perceived food addicts ($n = 12$, 21%) indicated that “whenever I am unoccupied with other activities, I will probably either be preparing or consuming food” and “I am always eating, and spend quite a bit of money on food”. In contrast, non-addicts ($n = 11$, 9%) reported little functional preoccupation with food (*e.g.*, “Food: Most of the time I can take it or leave it”, “I don’t really feel like eating very often”, “I don’t have a particular interest in food or cooking” and “will sometimes forget to eat”).

3. Self-control. Both self-perceived food addicts ($n = 18$, 32%) and non-addicts ($n = 51$, 43%) made causal attributions regarding an inability to control food intake. Responses referred to an ability to adhere to self-imposed dietary restrictions, or to resist tempting foods. For example, food addicts indicated that they “have little self-control around food” and have “tried to diet but can’t”. Food addicts also reported a constant “need to curb [a] desire for food”, and one individual described how self-imposed dietary restrictions affect their mood (“my mood can change if I begin to think about foods I really love and want but don’t have”).

In contrast, non-addicts reported feeling “able to resist the desire-to-eat” and “find it easy to follow a strict diet”.

4. Cravings. A similar proportion of food addicts ($n = 10$, 18%) and non-addicts ($n = 18$, 15%) indicated causal attributions that referred to food cravings. Again, self-perceived food addicts and non-addicts tended to report opposite experiences with regard to cravings. For example, while food addicts reported experiencing “cravings for food...even when full”, the majority of non-addicts reported that they “don’t crave food”. A few non-addicts did report having occasional food cravings, however felt “easily able to control [these] urges”.

5. Health. Both food addicts ($n = 6$, 11%) and non-addicts ($n = 15$, 13%) made causal attributions with respect to their weight status or their ability to maintain a healthy diet. The general consensus among both groups was that food addiction involves weight gain (Addict: “I have put on a stone in the past year”; Non-addict: “I am not overweight”). This is reflective of the increased BMI observed in self-perceived food addicts. However, while self-perceived food addiction may be generally associated with weight, increased BMI was not perceived to be a necessary or sufficient characteristic of food addiction. This is illustrated by one non-addict who stated that “although I am overweight, my life is not ruled by food”. Further, one food addict believed that they have a sugar addiction despite being “relatively slim”.

Food addicts also indicated a tendency to continue eating unhealthy foods despite negative consequences (“even though I know exactly what [sugar] does to me I still eat ridiculous amounts of it”). In contrast, non-addicts reported having a “healthy and varied diet” and a “positive relationship with food”.

6. Specific foods. Eleven food addicts (20%) and 16 non-addicts (13%) made causal attributions that referred to a lack of control “towards a particular food/food group”. Responses indicated that both addicts and non-addicts regarded foods high in fat and sugar as being particularly addictive. While food addicts reported an addiction to “sweet foods”, sugar, chocolate and pizza, non-addicts stated that they do not tend to “eat a lot of sugary foods” or “buy fast or processed foods – I associate foods like that with ‘addiction’”.

Food addicts also indicated a tendency to eat the same meal often (“I will purchase and eat [pizza] 2-3 times a week”). In contrast, non-addicts stated that they

“do not eat an excessive amount of one food”. Further, non-addicts reported that they “could stop eating any type of food if needed”.

In summary, on the basis of these data, six key manifestations of self-perceived food addiction were identified. The most common of these were a tendency to eat for reward (*i.e.*, psychological rather than physiological reasons), a functional or psychological preoccupation with food, and a perceived lack of self-control around food. Participants also regarded frequent food cravings, increased weight or an unhealthy diet, and a problem with a specific type of food to be indicative of food addiction. Both self-perceived food addicts and non-addicts reported similar themes but divergent responses. Of the 175 responses analysed, 173 (99%) made attributions that referred to at least one of the above themes. Of the two participants who did not allude to any of the above themes, one (addict) regarded food addiction as “human nature”, while the other (non-addict) did not “agree that someone can be addicted to food”.

2.6. Discussion

The current study explored beliefs about the cognitive and behavioural manifestations of food addiction within the lay public. Notably, over a quarter (28%) of participants perceived themselves to be food addicts, 60% did not perceive themselves to be addicted to food, and 12% were undecided. Increased BMI and younger age predicted self-perceived food addiction, however males and females were equally likely to identify as food addicts.

The thematic analysis revealed six characteristics that were commonly attributed to food addiction. These were a tendency to eat for reward (*i.e.*, psychological rather than physiological reasons), a functional or psychological preoccupation with food, a perceived lack of control around food, persistent food craving, increased weight or an unhealthy diet, and a problem with a specific type of food. Only one participant (non-addict) disputed the notion of food addiction, reflecting the widely held acceptance of the concept throughout society. Attributions did not differ as a function of BMI or age, although females were more likely to refer to reward-driven eating than males. Importantly, the emergent themes, and their frequency, did not appear to differ between self-perceived food addicts and non-addicts. Rather, self-perceived food addicts and non-addicts tended to report

divergent cognitions, behaviours and attitudes within each common theme. For example, while non-addicts reported feeling in control of their eating habits, self-perceived food addicts reported experiencing little self-control around food. To our knowledge, the current study provides the first exploration of food addiction beliefs in both self-perceived food addicts and non-addicts.

The themes that emerged in the current study build upon previous qualitative findings. In particular, Hetherington and Macdiarmid (1993) reported that a perceived lack of self-control around chocolate was commonly attributed to an addiction to chocolate. In the current study, we extend these findings by showing that low self-control beliefs may also be attributed to a more general addiction to food. The current themes also appear consistent with Ifland et al.'s (2009) study in which self-perceived food addicts reported a tendency to eat more than intended, unsuccessful attempts to cut back, a lot of time spent obtaining food or eating, and a tendency to eat despite negative health consequences. Taken together, their findings were interpreted as providing support for an overlap between substance dependence and self-perceived food addiction. However, the responses obtained in this previous study were prompted by interview questions that had been adapted from the DSM-IV criteria for substance dependence. This theory-led approach may have generated data that were consistent with a substance dependence model of food addiction.

In the current study, we employed an inductive approach that was not guided by any prior theory of food addiction. Several of the themes that were identified in this study could be argued to overlap, or partially overlap, with the DSM-IV criteria for substance dependence that have been adapted for the YFAS (notably, all of these criteria were retained in the more recent DSM-V classification of substance use disorder). Firstly, frequent food cravings may reflect a 'persistent desire' (in the DSM-IV) and 'craving or strong desire/urge to use a substance' (in the DSM-V). Secondly, a lack of self-control around food may be likened to the substance being 'taken in larger amounts and for longer than intended' and 'repeated unsuccessful attempts to quit'. Thirdly, food addiction was associated with weight gain and a tendency to continue eating unhealthy foods despite negative consequences. This is reminiscent of the criterion in which 'use continues despite knowledge of adverse consequences'. Finally, many participants referred to a psychological or functional preoccupation with food, such as spending a lot of time planning, preparing, and thinking about food. This is partly consistent with the criterion in which there is

‘much time/activity to obtain, use, or recover from’ a substance. However, no participants made attributions that specifically related to the amount of time spent recovering from overeating.

The current study also highlighted reward-driven eating (eating in response to psychological rather than physiological cues) as another key characteristic of self-perceived food addiction. Of interest, participants’ responses could be further categorised as eating for pleasure (i.e., positive reinforcement) versus eating to alleviate negative emotional states (i.e., negative reinforcement). These characteristics appear to overlap with the reward-related dysfunction and emotional dysregulation observed in substance dependence (Ersche et al., 2012; Wilens, Martelon, Anderson, Shelley-Abrahamson, & Biederman, 2013).

Interestingly, several of our participants perceived food addiction to involve a problem with a specific type of food, and consistent with previous research (Ifland et al, 2009; Malika, Hayman, Miller, Lee, & Lumeng, 2015), these were exclusively high-fat and high-sugar foods such as pizza and chocolate. It has been suggested that an inability to control oneself around specific food types provides evidence for addictive properties of these foods (Schulte, Avena, & Gearhardt, 2015). However, an alternative possibility is that a perceived ‘addiction’ to highly palatable foods may arise from psychological associations that are perhaps formed during childhood (Michels et al., 2012; Troisi & Gabriel, 2011). Indeed, previous research has demonstrated a tendency for parents to use food to satisfy their child’s emotional needs which has been associated with comfort eating (Blissett, Haycraft, & Farrow, 2010; Carnell, Cooke, Cheng, Robbins, & Wardle, 2011). Further, it should be noted that, while foods reported as problematic generally tend to be high in fat and sugar, this is not always the case, for example, Malika et al. (2015) found that people reported an addiction to carrots and hot sauce. Therefore it appears that people’s specific ‘comfort’ or problematic foods are highly idiosyncratic.

According to the psychosocial perspective of Rogers and Smit (2000), self-perceived food addiction arises following unsuccessful attempts to restrict one’s intake of certain (*e.g.*, nice but naughty) foods, which is accompanied by a strong desire or ‘craving’ for these foods. Thus, a perceived addiction to specific foods may result from the perpetual restriction of such foods. Importantly, the psychosocial perspective is consistent with the elevated reward value, perceived lack of control around food and the persistent cravings that were key themes in the current study.

It is also important to consider that, while there is some overlap between the DSM-IV/V criteria for substance dependence/ substance use disorder and participants' perceptions of food addiction, the current study highlighted some important distinctions. Firstly, only two participants (non-addicts) in the current study made causal attributions with regards to tolerance (i.e., requiring increasing amounts of food over time). Secondly, only one participant referred to the experience of physical withdrawal from food. Thirdly, no participants made causal attributions with regards to 'important social, occupational, or recreational activities given up or reduced' as a result of their eating. Finally, participants did not refer to any 'significant distress' or 'impairment to daily functioning' caused by their eating behaviours. By highlighting qualitative differences between the substance dependence criteria and the characterisation of self-perceived food addiction, the current findings help to explain the previously observed discrepancy between the number of people who fulfil the YFAS criteria and those who self-diagnose (Hardman et al., 2015; Meadows & Higgs, 2013). Furthermore, consistent with other recent qualitative research (Malika et al., 2015), our findings indicate that food addiction is defined within the lay public by a set of core behaviours (as opposed to physical symptoms).

The secondary aim of the current study was to determine whether it is necessary to provide a definition of food addiction prior to administering a measure of self-perceived food addiction. It was found that providing participants with brief information about food addiction (relative to no information) did not influence either the frequency of self-perceived food addiction or the causal attributions that were made. This result is helpful with regard to future research endeavours that may seek to quantify self-perceived food addiction.

The brevity of the current questionnaire enabled inclusion of a larger sample relative to previous qualitative investigations. However, our study did not permit an in-depth exploration of the experiences of self-perceived food addicts and non-addicts. Furthermore, the methods used may not have sufficiently prompted individuals to report 'deeper' concerns such as psychological distress or daily impairment. In future research, it would be beneficial to obtain richer accounts of self-perceived food addiction using alternative qualitative methods such as interviews and focus groups. In particular, research should explore the extent to which self-perceived food addicts experience psychological distress and daily

impairment as a consequence of their overeating. Additionally, while beyond the scope of the current study, it would be interesting for future research to compare qualitative responses obtained from self-perceived food addiction with standardised measures of eating behaviour such as the YFAS. In particular, it would be informative to establish whether self-perceived food addicts and non-addicts differ on quantitative eating behaviour traits. Conversely, qualitative responses might also differ between those who do and do not fulfil the YFAS criteria for food dependence. Finally, the current study consisted predominantly of university staff and students and the extent to which the findings generalise to other populations is questionable. Future qualitative research should therefore explore beliefs about food addiction in participants from a range of backgrounds and in clinical samples, such as those with binge eating disorder.

The current study successfully identified a number of behaviours that were perceived to characterise ‘food addiction’ within the lay public. Many of these appeared to overlap with some of the symptoms and characteristics associated with substance dependence/substance use disorder in DSM-IV/V. However, in contrast to this model we found that members of the lay public may identify as food addicts without necessarily encountering any significant distress or impairment to daily functioning. Further, participants in the current study did not appear to regard social occlusion, or the presence of food-related tolerance and physical withdrawal symptoms, as necessary characteristics of food addiction. As such, our findings would appear to reflect a view of food addiction that is identifiable through its core behaviours. Future research should aim to develop an operational definition of self-perceived food addiction, and explore the extent to which it may be clinically meaningful.

Chapter 3: Food reward and calorie intake in self-perceived food addicts

3.1. Overview

Findings from Chapter 2 suggest that individuals who perceive themselves to be addicted to food (i.e. self-perceived food addicts) have a tendency to eat for reward, rather than physiological hunger, and may be particularly susceptible to overeating foods that are high in fat. Building upon these findings, Chapter 3 examined whether self-perceived food addicts would demonstrate increased food reward and calorie consumption within a laboratory context. Furthermore, drawing upon theoretical perspectives of food reward (Rogers & Hardman, 2015), Chapter 3 examined whether increased food reward in self-perceived food addicts would be attributed to increased hunger and/or liking for the sensory qualities of the test foods. Finally, Chapter 3 considers the extent to which the concept of food addiction captures a unique set of eating behaviours that are distinct from already-established patterns of dietary disinhibition and restraint.

The study reported in this chapter has been published as:

Ruddock, H.K., Field, M., & Hardman, C. (2016). Food reward and calorie intake in self-perceived food addicts. *Appetite*. DOI: [10.1016/j.appet.2016.12.003](https://doi.org/10.1016/j.appet.2016.12.003)

3.2. Abstract

Previous research indicates that many people perceive themselves to be addicted to food. These ‘self-perceived food addicts’ may demonstrate aberrant eating patterns which put them at greater risk of weight gain. However, this is yet to be empirically investigated. Accordingly, the current study investigated whether self-perceived food addicts would exhibit higher food reward and calorie intake in a laboratory context relative to self-perceived non-addicts. A secondary aim was to investigate whether self-perceived food addicts would demonstrate increased food and/or increased hunger ratings. Finally, we explored whether self-perceived food addicts demonstrate patterns of aberrant eating, beyond that predicted by measures of trait dietary disinhibition and restraint. Female participants (self-perceived food addicts $n=31$, non-addicts $n=29$) completed measures of hunger, food reward (desire-to-eat, willingness-to-pay ratings, and an operant response task) and liking for high- and

low-fat foods. Participants completed all measures when they were hungry, and again when they were satiated after consuming a fixed-lunch meal. Finally, participants were provided with *ad libitum* access to high-and low-fat foods. Results indicated that self-perceived food addicts consumed more calories from high-fat food compared to non-addicts, despite the absence of any between-group differences in hunger or overall liking ratings. Self-perceived food addicts also displayed higher desire-to-eat ratings across foods compared to non-addicts, but groups did not differ on other measures of food reward. However, the differences in calorie intake and desire-to-eat between self-perceived food addicts and non-addicts were no longer significant after controlling for dietary disinhibition and restraint. These findings suggest that self-perceived food addicts experience food as more rewarding and are at particular risk of overeating. Furthermore, this may be attributable to the dual influences of increased dietary disinhibition and decreased restraint.

3.3. Introduction

The idea that certain foods have addictive properties similar to drugs of abuse is widely debated within the scientific community. While similarities have been identified between the neuro-behavioural effects of drugs and palatable food (e.g. Davis et al., 2011; Gearhardt et al., 2011), the extent to which excessive food intake is analogous to a substance abuse model remains a point of contention (Ziauddeen, Farooqi, & Fletcher, 2012; Hebebrand et al., 2014). Despite this, support for the concept of food addiction appears to be strong amongst members of the lay public (Lee et al., 2013; Ruddock, Dickson, Field, & Hardman, 2015). In a recent study, 86% of Australians and Americans believed that certain foods are ‘addictive’, and 72% believed that food addiction causes some cases of obesity (Lee et al., 2013). Furthermore, between 28-52% of people from community samples believe that they are ‘addicted’ to food (Hardman et al., 2015; Meadows & Higgs, 2013; Ruddock et al., 2015), indicating that self-perceived food addiction is prevalent within the general population.

To date, we know very little about the characteristics of people who *perceive* themselves to be ‘food addicts’. To address this, in a previous qualitative study (i.e. Chapter 2 of the current thesis), we identified several core behaviours which characterise self-perceived food addicts (Ruddock et al., 2015). These included a

tendency to eat for reward, rather than physiological hunger, frequent food cravings, diminished self-control around food, a particular problem controlling consumption of foods high in fat, salt, and sugar, and a preoccupation with food and eating. Our study also suggested differences between self-perceived food addiction and the clinical definition of food addiction used by the Yale Food Addiction Scale (YFAS) (Gearhardt, Corbin, & Brownell, 2009), which is based upon the DSM-IV criteria for substance dependence. Specifically, contrary to the YFAS definition, self-perceived food addiction was not thought to be characterised by ‘significant distress’ or an ‘impairment to daily functioning’. Consistent with this, other studies indicate that the majority of self-perceived food addicts do not meet the YFAS diagnostic criteria for food addiction (Hardman et al., 2015; Meadows & Higgs, 2013).

Despite not necessarily fulfilling the YFAS criteria for food addiction, self-perceived food addicts may demonstrate eating behaviours which put them at greater risk of over-eating and weight gain. In a previous study, self-perceived food addicts scored significantly higher on measures of pathological eating compared to self-perceived non-addicts (Meadows & Higgs, 2013). Furthermore, a number of laboratory studies have shown increased desire for and greater intake of chocolate in self-diagnosed chocolate addicts compared to non-addicts (Hetherington & Macdiarmid, 1995; Macdiarmid & Hetherington, 1995; Tuomisto et al., 1999). This is of potential concern because these patterns of eating and overconsumption may go undetected by existing measures of food addiction such as the YFAS. The extent to which self-perceived food addiction predicts meaningful differences in problematic eating (relative to non-addicts) thus requires further investigation.

Building on these preliminary findings, the aim of the current study was to examine the behavioural characteristics of individuals who perceive themselves to be ‘food addicts’. Specifically, (and following on from Hetherington & Macdiarmid, 1995; Macdiarmid & Hetherington, 1995; Tuomisto et al., 1999) we sought to determine whether self-perceived food addicts would exhibit higher food reward and calorie intake in a laboratory context relative to non-addicts. We employed three measures as proxy indicators of the reward of food – desire-to-eat ratings, an operant task and by asking participants to indicate how much money they would be willing to pay for a portion of food. These measures have been validated by Rogers and Hardman (2015) and used in previous studies on food reward (Brunstrom & Rogers 2009; Hardman, Herbert, Brunstrom, Munafò, & Rogers, 2012). Previous studies

indicate that individual differences in food reward are most apparent when participants are satiated relative to in a hungry state (Castellanos et al., 2009; Dalton, Blundell, & Finlayson, 2013; Nasser et al., 2008). We therefore assessed participants in both hungry and satiated states and we expected to see a greater difference between self-perceived addicts and non-addicts in the latter state. We also expected self-perceived food addicts to find high-fat foods more rewarding relative to low-fat foods and to consume more of these foods *ad libitum*, compared to non-addicts. This is consistent with our previous findings in which self-perceived food addicts reported a tendency to overeat high-fat foods (Ruddock et al., 2015). Similarly, another study found that high-fat foods, such as chocolate and crisps, were regarded as more ‘addictive’ than low-fat foods, such as fruit and plain crackers (Schulte, Avena, & Gearhardt, 2015).

A secondary aim of this study was to investigate whether self-perceived food addicts would demonstrate increased food liking and/or increased hunger ratings. Hunger and food liking are thought to represent distinct measurable components of food reward (Berridge, Ho, Richard, & DiFeliceantonio, 2010; Rogers & Hardman, 2015), and so we may find that either, or both, of these are increased in those with heightened food reward. However, previous research has yielded inconsistent findings regarding this. In one study, self-diagnosed ‘chocolate addicts’ had increased levels of food reward (i.e. desire-to-eat) but did not differ from controls on measures of hunger and liking for chocolate, prior to chocolate consumption (Hetherington & Macdiarmid, 1995). In contrast, increased chocolate liking has been observed in self-reported ‘chocolate cravers’ (Gibson & Desmond, 1999), and Finlayson et al. (2011) demonstrated increased hunger perceptions in those with a propensity to overeat.

A further secondary aim was to establish the extent to which self-perceived food addicts demonstrate patterns of aberrant eating behaviour that are distinct from those captured by existing measures of dietary disinhibition (i.e. loss of control over intake) and restraint (i.e. attempts to restrict intake). This is important as food addiction is considered to be a distinct construct, which nonetheless overlaps with other forms of pathological eating such as binge eating (Davis, 2016). It is therefore necessary to establish the extent to which the concept of food addiction *uniquely* predicts patterns of overeating (Long, Blundell, & Finlayson, 2015). To address this, we explored the extent to which self-perceived food addiction predicts increases in

food reward and calorie intake over and above that accounted for by high dietary disinhibition and low restraint. Dietary disinhibition was measured using the Binge Eating Scale (Gormally, Black, Daston, & Rardin, 1982) and the disinhibition subscale of the Three Factor Eating Questionnaire (TFEQ; Stunkard & Messick, 1985), both of which are thought to reflect differing degrees of ‘uncontrolled’ or disinhibited eating (Vainik et al., 2015). Dietary restraint was assessed using the restraint subscale of the TFEQ which assesses successful restraint (Heatherton et al., 1988) and, accordingly, in our study we considered low dietary restraint as a risk factor for over-eating (Rollins, Loken, & Birch, 2011). These measures demonstrate good predictive validity for *ad libitum* food intake, eating psychopathology, and the tendency to engage in uncontrolled eating (Duarte, Pinto-Gouveia, & Ferreira, 2015; Ouwens, van Strien, & van der Staak, 2003; Rollins, Loken, & Birch, 2011).

To summarize, the aims of the current study were as follows; (1) To investigate whether self-perceived food addicts would demonstrate increased food reward (most notably when satiated), and would subsequently consume more calories when given *ad libitum* access to high- and low- fat foods compared to non-addicts. In particular, these differences were expected to be most pronounced towards the high-fat foods. (2) To test the hypothesis that increased food reward in self-perceived food addicts would be accounted for by increased liking for the test foods, and/or increased hunger, (3) To explore the extent to which self-perceived food addiction predicts food reward and calorie intake over and above existing measures of binge eating, dietary disinhibition and restraint.

3.4. Method

3.4.1. Participants

Participants ($N=64$) were recruited from the University of Liverpool via poster and online advertisements. As this was a preliminary study into self-perceived food addiction, we restricted the sample to females in order to minimize between-subject differences. Participants were purposefully recruited such that approximately half were self-perceived food addicts. To achieve this, after approximately 30 self-perceived non-addicts had been recruited, we restricted recruitment to self-perceived food addicts *only*. This was specified in the inclusion criteria displayed on study advertisement posters, and on the participant information sheet. Self-perceived food

addiction was assessed using a self-report measure (see Measures section for details). Participants were excluded from the study if they had any food allergies or intolerances, had ever been diagnosed with an eating disorder, were on any medication which may affect appetite, or if they smoked tobacco. Ethical approval was granted by the University Research Ethics Committee. In exchange for their time, participants received course credits or a £5 shopping voucher.

3.4.2. Materials and measures

3.4.2.1. Assessment of self-perceived 'food-addiction'.

As in previous research (Hardman et al., 2015; Ruddock et al., 2015, Chapter 2 of this thesis), to assess self-perceived food addiction, participants were asked 'Do you agree with the following statement: "I believe myself to be a food addict"?' . Participants were required to tick either 'yes' or 'no'. For the purposes of our analyses, participants who ticked 'yes' were classified as 'self-perceived food addicts', and participants who ticked 'no' were classified as non-addicts.

3.4.2.2. Appetite.

Hunger and fullness ratings were obtained using 100mm visual analogue scales (VAS). Each scale was marked by anchor points 'Not at all' on the left and 'Extremely' on the right. Consistent with previous recommendations (i.e. Rogers & Hardman, 2015), hunger and fullness ratings were completed without food being present.

3.4.2.3. Ratings task.

For the ratings task, participants were presented with four small plates each with a sample of chocolate (6 x Galaxy Minstrels, 16g, 83 kcals, 4g fat), crisps (6 x HP Hula Hoops, 4.9g, 25 kcals, 1g fat), grapes (6 x seedless green grapes, 38g, 27 kcals, 0g fat), and six pieces of Tesco lightly salted rice cake (6g, 22 kcals, 0g fat). These foods were specifically chosen to provide two high fat foods which are commonly reported as 'addictive' or 'problematic' (Schulte et al., 2015) (crisps and chocolate) and two low fat foods (grapes and rice cakes), which are not regarded as particularly addictive (Schulte et al., 2015). For each food, participants were instructed to place one piece in their mouth and complete the rating scales in the

following order: 'Liking', 'Desire-to-eat', and 'Willingness to pay' (following the procedure of Rogers & Hardman, 2015). The order in which each food was rated was counterbalanced across participants.

Liking

Liking ratings for each of the test foods were obtained using a 100mm VAS with end anchor points 'Not at all' and 'Extremely' to the left and right of the scale, respectively. The following instructions were given to encourage participants to focus on the taste of the food, as opposed to the pleasantness of actually ingesting it: *How much do you like the taste of this food? That is, how pleasant does it taste in your mouth RIGHT NOW? When making this judgement, IGNORE how much or little of the food you want to eat, and what it would be like to chew and swallow it – JUST FOCUS PURELY ON ITS TASTE IN YOUR MOUTH.*

Desire-to-eat (Food reward).

Having completed the liking measure, a measure of 'Desire-to-eat' (DtE) was obtained for the remaining amount of each of the test foods using a 100mm VAS with end anchor points 'Not at all' and 'Extremely' to the left and right of the scale, respectively. Importantly, participants were instructed to indicate how much they desired to eat each of the foods 'right now'. Desire-to-eat ratings have been shown to provide a valid measure of food reward (Rogers & Hardman, 2015).

Willingness to pay (Food reward).

Using a 100mm VAS, participants were asked to indicate how much money they would be 'willing to pay' (WtP) for the remaining amount of each of the test foods. The VAS ranged from 1p on the left to £2 on the right, and £1 marked the mid-point of the scale. This task has been used in previous research to reflect the rewarding value of food (e.g. Hardman et al., 2012).

3.4.2.4. Operant task (Food reward).

An operant response task was included to assess participants' motivation to obtain chocolate (i.e. high-fat sweet food) and grapes (i.e. low-fat sweet food) as further measure of food reward. The task was programmed using E-prime 2.0 (Psychology Software Tools, Inc. Sharpsburg, PA, USA). For chocolate and grapes only, participants were required to tap the spacebar on a computer keypad for 60

seconds. They were informed that the more they tapped the space bar during this time, the more of each food they would receive at the end of the session. Previous research has demonstrated the validity of this task as a measure of food reward (Rogers & Hardman, 2015). The order in which participants tapped for chocolate and grapes was counterbalanced across participants. This task was performed for two out of the four test foods in order to minimize the potential confounding effects of participant fatigue.

3.4.2.5. Lunch meal

To induce satiety, participants were provided with cheese sandwiches. Sandwiches were made using 3 pieces of Tesco ‘Stay Fresh’ medium sliced white bread (121g, 303kcal, 2g fat), 1.5 pieces of Tesco medium pre-sliced cheddar (38g, 152 kcal, 13g fat), and 15g butter (Tesco Butterpak, 95 kcal, 11g fat). These were then sliced into six small sandwiches. This meal size was based on previous research in which, during *ad libitum* consumption, participants consumed slightly over 10 bite-size cheese sandwiches (Rogers & Hardman, 2015). Participants were given 10 minutes in which they were instructed to consume the entire meal. All but four participants complied with this instruction. These four participants were within the healthy weight range (i.e. $18.5 \text{ kg/m}^2 < \text{BMI} < 24.9 \text{ kg/m}^2$), and one identified as a food addict.

3.4.2.6. Additional measures and eating trait questionnaires.

Familiarity ratings

Participants were asked to indicate how often they consumed each of the four test foods. The following response options were given: ‘Never’, ‘Monthly or less’, ‘2-4 times a month’, ‘2-3 times a week’, ‘4 or more times a week’, and ‘Every day’. Participants indicated how often they ate each food by ticking the appropriate box.

Three Factor Eating Questionnaire (TFEQ).

Participants completed the ‘Restraint’ (TFEQ-R) and ‘Disinhibition’ (TFEQ-D) sub-scales of the TFEQ (Stunkard & Messick, 1985). Dietary restraint refers to attempts to restrict food intake, while disinhibition refers to the general tendency to overeat. The TFEQ-R sub-scale comprises 21 items such as “I have a pretty good

idea of the number of calories in common foods”. The TFEQ-D sub-scale consists of 16 items such as “I usually eat too much at social occasions like parties and picnics”.

Binge Eating Scale (BES).

The BES (Gormally, Black, Daston, & Rardin, 1982) consists of 16 items which assess the severity of binge eating symptoms. Each item consists of three or four statements about eating behaviours or emotions associated with binge-eating. Instructions are given to mark the statement within each item which the participant most identifies with. Higher scores on the BES indicate more severe binge eating symptoms.

Yale Food Addiction Scale (YFAS).

The YFAS (Gearhardt et al., 2009) consists of 25 items designed to measure an addiction to foods high in fat and/or sugar. The scale is based on the DSM-IV criteria for substance dependence. For the first 16 items, a Likert scale is used in which the respondent indicates how often, in the past 12 months, they have engaged in a particular behaviour (for example “I eat to the point where I feel physically ill”). For the next 9 items, respondents indicate whether or not they agree with each statement by marking either ‘Yes’ or ‘No’ (for example, “I want to cut down or stop eating certain kinds of foods”). Respondents are asked to base their response on their experiences in past 12 months. In the final item, respondents are asked to indicate all foods that they have problems with. A diagnosis of food addiction is given when the individual demonstrates significant clinical impairment due to their eating behaviours, and fulfils at least three of the following symptoms: unsuccessful attempts to quit, giving up activities to eat, eating large portions, continuing to overeat despite negative consequences, tolerance to food, withdrawal from not eating, and spending a lot of time eating. The YFAS also provides a continuous measure of the number of food addiction symptoms exhibited by an individual (i.e. symptom count) which range from 0 to 7. The YFAS was included to provide descriptive information about the characteristics of our sample, and was not central to the aims and objectives of the study. In particular, we included this measure to confirm previous findings in which few self-perceived food addicts met the YFAS-criteria for food addiction (Hardman et al., 2015; Meadows & Higgs, 2013).

3.4.3. Procedure

All participants attended one testing session which took place at the Ingestive Behaviour Laboratory at the University of Liverpool. Figure 3.1 illustrates the study procedure. Prior to testing, participants were asked to eat their usual breakfast but then to refrain from consuming any food or calorie-containing drinks for 3 hours before the start of their session. All participants indicated that they had adhered to this instruction. Participants were tested individually, and all sessions took place between 12pm and 2pm. Upon arrival, participants were provided with information about the study and signed a consent form. Participants then completed a medical history questionnaire to ensure that they did not have any food allergies. Participants indicated their current level of hunger and fullness (T1). This was followed by the ratings task in which participants indicated their Liking, Desire-to-eat (DtE) and Willingness to Pay (WtP) for each of the four foods. Participants then completed the ‘tapping task’ for chocolate and grapes, and levels of hunger and fullness were reassessed (T2). Participants then consumed the lunch meal, after which they were given a 5-minute break. During the break, participants could either sit quietly or engage in some light reading. Hunger and fullness levels were reassessed at this stage (T3), followed by the post-lunch ratings task and tapping task. To provide a valid comparison of food reward between hungry and satiated states, it was important that participants believed that the outcome of the tapping task (i.e. the amount of food they would receive) would not be influenced by their previous performance on the task. Therefore, participants were told that their results from the earlier tapping task had failed to save on the computer and therefore would not affect how much food they would receive at the end of the session (as used in Rogers & Hardman, 2015). Levels of hunger and fullness were reassessed (T4). Participants were then given *ad libitum* access to 160g of chocolate (Galaxy Minstrels 805 kcals, 36g fat) and 200g of grapes (140 kcals, 0g fat) under the pretence that that they had ‘earned’ these foods during the tapping task. Participants were told that they could eat as much of the food as they wished and to let the experimenter know when they had had enough. Following this, participants were again required to indicate their levels of hunger and fullness (T5).

The remaining measures were administered in the following order: Familiarity ratings, TFEQ, BES, YFAS, self-perceived ‘food-addiction’.

Participants' height and weight were also assessed to provide a measure of body mass index (BMI).

Finally, to ensure the absence of demand characteristics, participants were asked to indicate what they thought the aims of the study were. No participants guessed correctly. Participants were then fully debriefed.

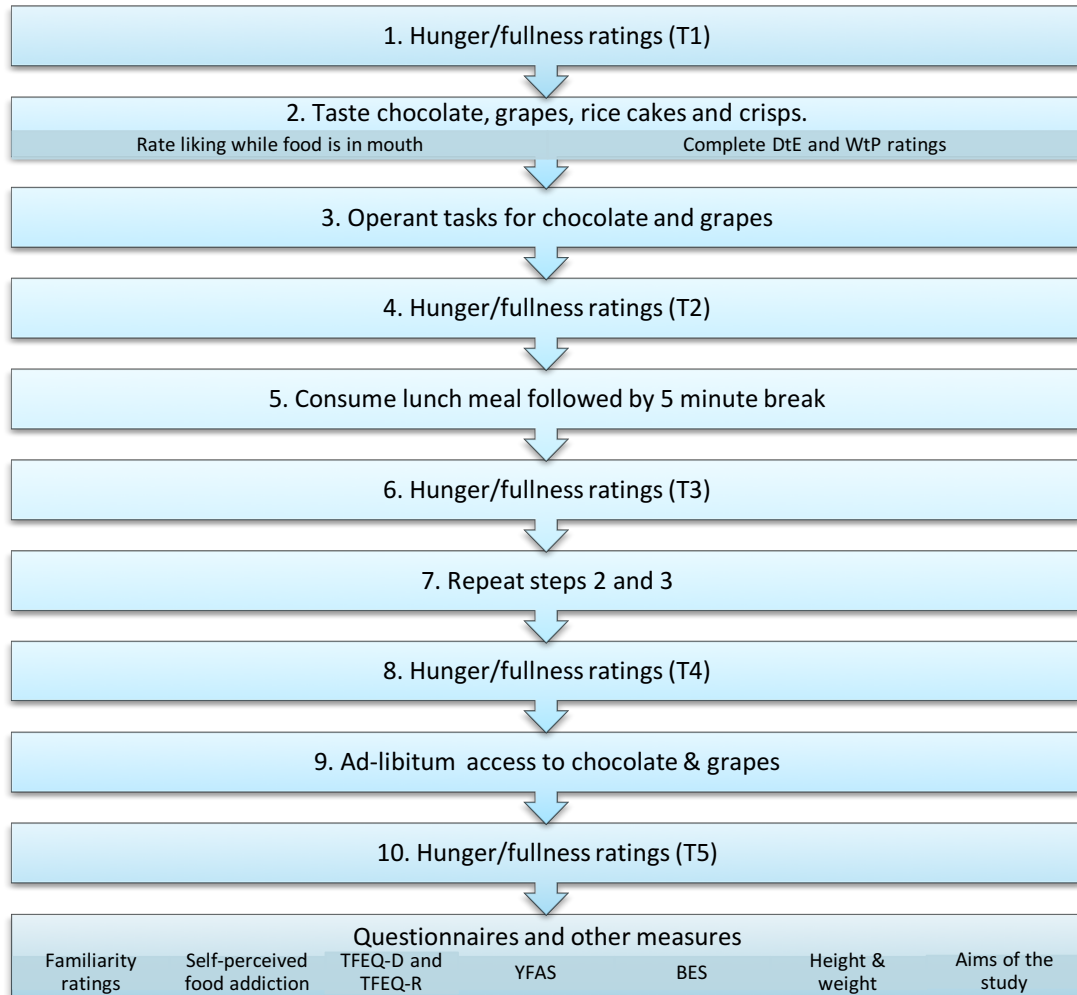


Figure 3.1. Flow chart of the study procedure

3.4.4. Data analysis

Liking, desire-to-eat (DtE), and willingness to pay (WtP) ratings were assessed using mixed design ANOVAs with a between-subject factors of Group (2: self-perceived food addicts/non-addicts) and within-subject factors of Time (2: before and after the lunch meal) and Food type (4: chocolate, crisps, rice cakes, grapes). Tapping frequency during the operant task was assessed using a 2(group) x 2(time) x 2(food type: chocolate/grapes) mixed-design ANOVA. For each analysis, food type and time were entered as within-subjects variables, and group was included

as a between-subjects variable. Calorie intake was analysed using a 2 (food type: chocolate/grapes) x 2(group) mixed-design ANOVA. Group differences in hunger ratings were explored using a 2 (group) x 5 (time) mixed-design ANOVA with time as a within-subjects variable, and group as a between-subjects variable.

Hierarchical regression analyses were conducted to examine the extent to which self-perceived food addiction could account for group differences in food reward and calorie intake, over and above that accounted for by dietary disinhibition and restraint. Scores from the BES and TFEQ disinhibition subscale were highly correlated, $r=.73$, $p<.001$. Therefore, to avoid problems arising from multicollinearity of predictor variables, a single ‘disinhibited eating index’ was calculated using the mean of the combined z-scores from these two measures (Thush et al., 2008). TFEQ-restraint subscale scores were also transformed to z-scores prior to analysis. Disinhibited eating index and TFEQ-restraint (z-scores) were then entered into the first step of the regression model, and group (i.e. self-perceived food addicts vs. non-addicts) was entered into the second step. Measures of food reward and calorie intake (where prior analyses revealed between-group differences) were entered as dependent variables.

3.5. Results

3.5.1. Participant characteristics

Participants who did not consume the entire set lunch were excluded from the analysis ($N=4$) leaving a total of 60 participants (self-perceived food addicts $n=31$; non-addicts $n=29$)¹. Post-hoc power analyses, using GPower 3.1, indicated that the current sample yielded 76% power to find significant interactions and differences between groups on measures of food reward and calorie intake, of medium effect sizes ($f=.35$, $\alpha=.05$). For the regression analyses, the sample size yielded 83% power to detect a medium effect size ($f^2=.15$) ($\alpha=.05$). Participants were aged between 18 and 54 years ($M=23.9 \pm 9.4$ y) and had a mean BMI of 23.7 kg/m^2 ($\pm 4.6 \text{ kg/m}^2$). Nine participants (15%) were classified as overweight ($\text{BMI}>25 \text{ kg/m}^2$) and seven (12%) were classified as obese ($\text{BMI} > 30\text{kg/m}^2$). Of the remaining 60 participants,

¹ Analyses were re-run with these four participants included. Results remained the same, however the main effect of group on DtE only approached significance, $F(1,62)=3.54$, $p=.065$.

31 identified as food addicts and 29 identified as non-food addicts. Self-perceived food addicts endorsed significantly more YFAS symptoms ($p < .001$), but were *not* more likely to fulfil the YFAS diagnosis for food addiction, relative to non-food addicts (see Table 3.1). Self-perceived food addicts also scored significantly higher on the BES and TFEQ-D sub-scale, compared to non-addicts. Importantly, groups did not differ on BMI or age (see Table 3.1). BMI did not correlate with any dependent variable and therefore was not included as a covariate in subsequent analyses.

Table 3.1. Descriptive statistics of sample by food addiction group. Values are means \pm standard deviations.

Variable	Self-perceived food-addict	Non-addict	F(df)	p
N	31	29		
Age (y)	24.2 \pm 9.8	23.6 \pm 9.0	.07(1,58)	.794
BMI (kg/m ²)	24.3 \pm 4.7	23.1 \pm 4.5	1.01(1,58)	.320
BES	16.7 \pm 6.7	9.7 \pm 5.0	20.97(1,58)	<.001
TFEQ				
Disinhibition	9.4 \pm 2.9	6.5 \pm 2.8	15.42(1,58)	<.001
Restraint	7.1 \pm 4.9	9.8 \pm 6.1	3.71(1,58)	.059
YFAS symptom count	3.19 \pm 1.89	1.45 \pm 0.87	20.68(1,58)	<.001
<i>Chi-Square</i>			X ²	
YFAS diagnosis (N)	4	1	1.75(1)	.355

3.5.2. Measures of food reward

The predicted 3-way time x food type x group interaction was not significant for any of the three reward measures (i.e. DtE, WtP, and tapping frequency –Table 3.2) ($ps > .206$). However, our primary hypothesis was partially supported by a main effect of group on overall DtE ratings, $F(1,58)=6.08$, $p=.017$, $\eta_p^2=.10$, such that self-perceived food addicts demonstrated increased overall DtE ratings compared to non-addicts. There was no main effect of group on WtP ratings, $F(1,58)=.35$, $p=.557$, $\eta_p^2=.01$, or tapping frequency $F(1,58)=1.13$, $p=.293$, $\eta_p^2=.02$. No 2-way interactions were observed between group x time ($ps > .081$), or group x food type ($ps > .237$) for any measure of food reward. Main effects of time revealed that all three measures of food reward decreased significantly following consumption of the lunch meal (Table 3.2) (DtE: $F(1,58)=124.75$, $p<.001$, $\eta_p^2=.69$; WtP: $F(1,58)=47.95$, $p<.001$, $\eta_p^2=.45$; Tapping frequency: $F(1,58)=40.35$, $p<.001$, $\eta_p^2=.41$).

Table 3.2. Means (\pm standard deviations) for the three measures of food reward, for self-perceived food addicts and non-addicts, before and after consumption of the lunch meal. NA = not applicable.

		DtE		WtP		Tapping (Operant task)	
		Before	After	Before	After	Before	After
Chocolate	Addicts	83 \pm 12	72 \pm 23	27 \pm 23	17 \pm 14	270 \pm 119	211 \pm 128
	Non-addicts	83 \pm 15	59 \pm 22	26 \pm 21	16 \pm 15	246 \pm 129	158 \pm 130
Crisps	Addicts	82 \pm 13	63 \pm 24	23 \pm 20	13 \pm 14	NA	NA
	Non-addicts	71 \pm 20	47 \pm 25	18 \pm 15	10 \pm 10	NA	NA
Rice cakes	Addicts	52 \pm 27	28 \pm 25	10 \pm 10	5 \pm 6	NA	NA
	Non-addicts	42 \pm 25	19 \pm 19	8 \pm 10	4 \pm 4	NA	NA
Grapes	Addicts	77 \pm 17	60 \pm 25	20 \pm 16	13 \pm 14	245 \pm 121	200 \pm 127
	Non-addicts	78 \pm 16	54 \pm 22	20 \pm 18	12 \pm 14	248 \pm 125	150 \pm 120

Note. Desire-to-eat (DtE) and willingness to pay (WtP) values represent scores (mm) provided on the corresponding 100mm Visual Analogue Scales. Tapping values represent the frequency of computer key taps within the allocated 1-minute time period in the operant response task.

3.5.3. Calorie intake

Consistent with our primary hypothesis, a main effect of group, $F(1,58)=8.65, p=.005, \eta_p^2=.13$, showed that food addicts consumed significantly more calories overall (Figure 3.2). There was also a main effect of food, $F(1,58)=65.40, p<.001, \eta_p^2=.53$, such that participants consumed significantly more calories from chocolate ($M=236 \pm 187$) than from grapes ($M=57 \pm 38$). These main effects were subsumed under the hypothesised 2-way food type x group interaction, $F(1,58)=6.64, p=.01, \eta_p^2=.103$. Follow-up univariate ANOVAs showed that food addicts consumed more chocolate, $F(1,58)=7.98, p=.006, \eta_p^2=.121$, but not more grapes, $F(1,58)=2.83, p=.098, \eta^2=.05$, than non-addicts (Figure 3.2). The between-group effect on chocolate consumption remained significant when using a Bonferroni adjustment for multiple comparisons.

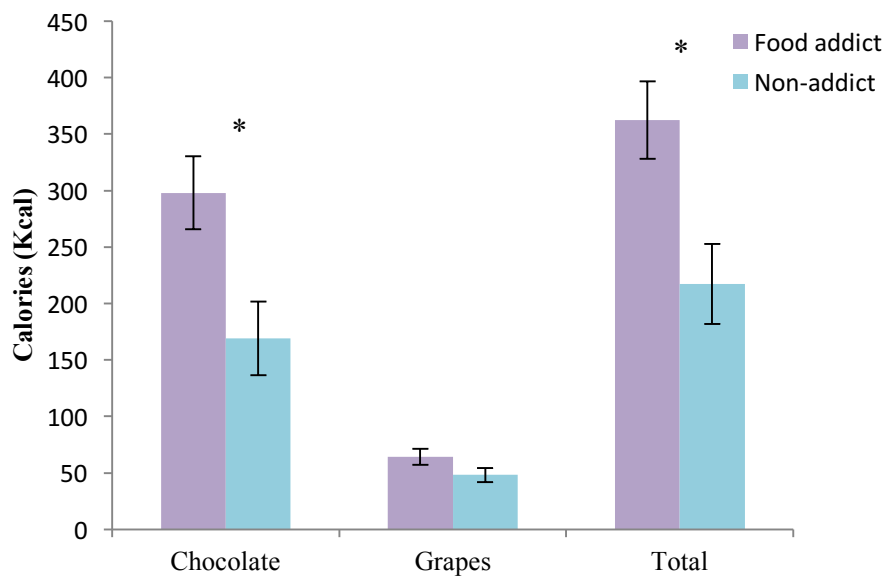


Figure 3.2. Number of calories consumed from chocolate, grapes, and total calories consumed, by self-perceived food addicts and non-addicts. * $p<.01$.

3.5.4. Food liking and hunger

There was no group x time interaction, $F(1,58)=.07, p=.799, \eta_p^2=.00$, and no main effect of group on hunger ratings, $F(1,58)=.30, p=.589, \eta_p^2=.01$. Furthermore, there was no main effect of group on overall liking ratings for the test foods, $F(1,58)=.31, p=.583, \eta_p^2=.01$. However, a group x time interaction for liking ratings

was observed, $F(1,58)=5.43$, $p=.023$, $\eta_p^2=.09$. To explore this further, we calculated the decline in liking ratings for each participant (collapsed across all test foods) by subtracting average liking ratings when satiated, from average liking when hungry. This ‘liking decline’ value was then entered into an independent t-test which revealed that self-perceived food addicts demonstrated less of a decline in ‘liking’ ratings for the test foods following the lunch meal compared to non-addicts, $t(58)=2.33$, $p=.023$ (Figure 3.3, panel A).

A main effect of time was observed on hunger ratings, $F(1,58)=412.26$, $p<.001$, $\eta_p^2=.88$. Specifically, hunger ratings were significantly greater at both T1 and T2 (i.e. prior to the lunch meal) compared to at T3 and T4 (i.e. following the lunch meal). Hunger ratings at T5 (i.e. following *ad libitum* food intake) were significantly lower than at all other time-points (Figure 3.3., panel B).

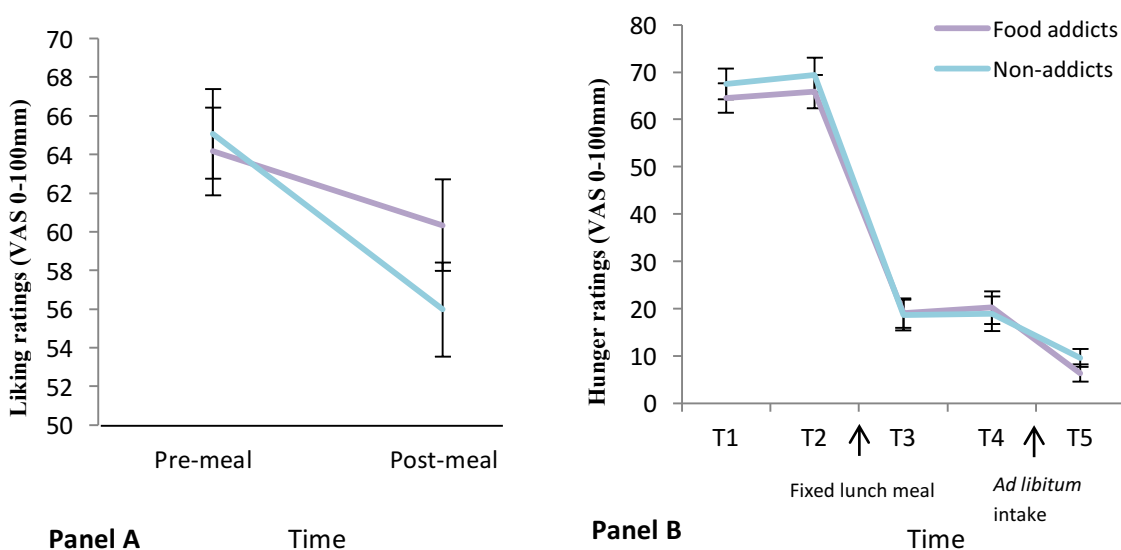


Figure 3.3. Ratings of liking (panel A), and hunger (panel B) for self-perceived food addicts and non-addicts before and after the lunch meal. Liking ratings were averaged across all four test foods.

3.5.5. Regression analyses

The results of the regression analyses revealed that group (i.e. self-perceived food addicts vs. non-addicts) failed to account for variance in total calories consumed (Table 3.3), or overall DtE ratings (Table 3.4), over and above that predicted by the disinhibited eating index and TFEQ-restraint (z-scores). Disinhibition was a significant positive predictor and restraint a significant negative predictor of calorie intake; however, these relationships became non-significant

when self-perceived food addiction was added to the model. For desire-to-eat ratings, disinhibition was the only significant predictor at both stages in the model. Tolerance (.67) and VIF (1.50) values indicated no problems with multi-collinearity between predictor variables (i.e. disinhibition, TFEQ-restraint, and group) in either regression model (Menard, 1995; Myers, 1990).

Table 3.3. Results of regression analysis with measures of dietary restraint and disinhibition in step 1 and self-perceived food addiction in step 2. The dependent variable was total calories consumed.

	<i>F-change</i>	<i>r² change</i>	<i>B</i>	<i>Std. Error</i>	<i>SR²</i>	<i>p</i>
Step 1	<i>F(2,57)=4.40*</i>	.13				
Disinhibition			55.03*	26.67	.07	.044
Restraint			-52.18*	24.43	.07	.037
Step 2	<i>F(1,56)=2.67</i>	.04				
Self-perceived food addiction			97.91	59.91	.05	.108

Note. SR^2 is the squared semi-partial correlation. * $p < .05$. Variance accounted for by the full regression model: $r^2 = .17$, $F(3,56) = 3.91$, $p = .013$.

Table 3.4. Results of regression analysis with measures of dietary restraint and disinhibition in step 1 and self-perceived food addiction in step 2. The dependent variable was mean overall DtE ratings (collapsed across conditions and foods).

	<i>F-change</i>	<i>r² change</i>	<i>B</i>	<i>Std. Error</i>	<i>SR²</i>	<i>p</i>
Step 1	<i>F(2,57)=6.30*</i>	.18				
Disinhibition			6.25*	1.77	.18	.001
Restraint			-.63	1.62	.00	.700
Step 2	<i>F(1,56)=0.58</i>	.01				
Self-perceived food addiction			3.09	4.05	.01	.449

Note. SR^2 is the squared semi-partial correlation. * $p < .05$. Variance accounted for by the full regression model: $r^2 = .19$, $F(3,56) = 4.37$, $p = .008$.

3.6. Discussion

According to recent studies, between 28-52 per cent of community samples perceive themselves to be addicted to food (Hardman et al., 2015; Meadows & Higgs, 2013; Ruddock et al., 2015). While the majority of self-perceived food addicts do not fulfil the diagnostic criteria for food addiction established by the YFAS (Gearhardt et al., 2009), previous research suggests that these individuals may demonstrate increased patterns of pathological eating (Meadows & Higgs, 2015; Ruddock et al., 2015). As such, self-perceived food addicts may represent a group of individuals who are at particular risk of weight gain and obesity.

To address this possibility, the current study investigated whether self-perceived food addicts would demonstrate increased food reward, particularly when satiated, and would consume more calories when provided with *ad libitum* access to high- and low- fat foods, compared to those who did not identify as food addicts. In particular, we expected to observe individual differences in reward and intake for foods that were high in fat. Food reward for high- and low-fat foods was assessed using desire-to-eat ratings, willingness to pay ratings, and an operant response task, consistent with methods used in previous research (Brunstrom & Rogers, 2009; Hardman et al., 2012; Rogers & Hardman, 2015). All measures of reward were taken when participants were hungry, and again when they were satiated after consuming a fixed sandwich-lunch meal.

Consistent with our hypothesis, self-perceived food addicts consumed more calories *ad libitum* from the high-fat food (i.e. chocolate), and more calories overall, compared to non-addicts. As predicted, groups did not differ in their intake of the low-fat food (i.e. grapes). Furthermore, self-perceived food addicts demonstrated increased *overall* desire-to-eat ratings for the test foods compared to non-addicts. However, contrary to our hypothesis that individual differences in food reward would be most pronounced in the satiated condition and towards the high-fat food, this effect was apparent in *both* the hungry and satiated states and across high-fat and low-fat food types. Also contrary to our hypothesis, the groups did not differ on the other measures of food reward (i.e. tapping frequency and willingness-to-pay measures),

Together, the current findings are partially consistent with previous research in which self-perceived food addicts and ‘chocolate addicts’ reported increased

desire for food and showed a propensity to overeat (Hetherington & Macdiarmid, 1995, Macdiarmid & Hetherington, 1995; Ruddock et al., 2015; Tuomisto et al., 1999). The current study extends these findings by demonstrating increased food reward in self-perceived food addicts, for a *range* of foods, when hungry and satiated. These differences in eating behaviour were observed despite the fact that very few (four participants out of 31; 13%) self-perceived food addicts fulfilled the YFAS diagnostic criteria. This is important as, consistent with previous findings (Meadows & Higgs, 2013), it suggests that self-perceived food addicts represent a population of individuals who have an increased tendency to overeat, and may go undetected by an existing measure of addictive eating. Importantly, while no weight differences were observed between self-perceived food addicts and non-addicts, this may be attributable to the young age of the sample. Indeed, in our previous research, which consisted of a slightly older demographic (i.e. mean age = 29 years), we found increased incidences of self-perceived food addiction amongst those with higher BMI (Ruddock et al., 2015).

Hunger and liking for the taste of a food are thought to represent measurable components of food reward (Berridge et al., 2010; Rogers & Hardman, 2015). On this basis, a further aim of the current study was to explore whether increased food reward in self-perceived food addicts was attributable to increased food liking and/or increased hunger ratings. There was no overall difference between the groups on liking for the test foods. This is consistent with previous research which found increased food reward in ‘chocolate addicts’, despite no differences in food *liking* (Hetherington & Macdiarmid, 1995). Similarly, we did not observe any between-group differences in hunger ratings at any point in the study, despite the fact that the self-perceived food addicts consumed significantly more chocolate between T4 and T5 than did non-addicts. This is important because it indicates that increased food reward and chocolate intake in the self-perceived food addicts relative to the non-addicts cannot be due to differences in hunger state. Notably, Hetherington and Macdiarmid (1995) also found that chocolate overeaters had higher desire to eat but were not hungrier or less full than controls at baseline (i.e. prior to consuming a chocolate snack).

Nonetheless, while *overall* liking ratings for the test foods did not differ between groups, self-perceived food addicts demonstrated an attenuated decline in

liking ratings following consumption of the fixed sandwich lunch meal relative to non-addicts. This was despite the fact that both groups demonstrated a similar decline in hunger ratings following the lunch meal. Future research should explore the possibility that self-perceived food addicts experience less of a reduction in the hedonic value of a food's taste following satiety *per se* or repeated consumption of a similar taste (i.e. sensory specific satiety). Indeed, Hetherington and Macdiarmid (1995) reported smaller changes in chocolate pleasantness ratings following chocolate consumption in chocolate overeaters, compared with control participants. Similarly, obese women demonstrated an attenuated decrease in the hedonic value of a sweet tasting solution over repeated trials compared to lean women (Pepino & Mennella, 2012).

A further secondary aim of the current study was to establish the extent to which self-perceived food addiction uniquely predicts overeating and increased food reward. This follows recent suggestions that food addiction may be a novel term that is used to describe already established patterns of over-eating (Long et al., 2015; Vainik et al., 2015). In the current study, self-perceived food addiction failed to predict a significant proportion of the variance in calorie intake and food reward (i.e. overall desire-to-eat ratings) beyond that accounted for by dietary disinhibition and restraint. This suggests that members of the lay public may use the term 'food addiction' as a means of conceptualizing patterns of over-eating that are already captured by established trait measures of dietary behaviour. Notably, in our study, food intake was predicted by both increased dietary disinhibition *and* reduced dietary restraint and this is consistent with dual system models of eating behaviour (Price, Higgs, & Lee, 2015).

The current study yields a number of limitations that should be addressed in future research. Firstly, while we specifically recruited non-smokers, we did not control for the use of other recreational drugs or alcohol. Given the association between aberrant eating behaviours and alcohol and drug use (e.g. Clark & Saules, 2013; Grucza et al., 2010; Lilenfeld et al., 2008), it is possible that those who identify as food addicts may have been more likely to use drugs and be heavy drinkers which may have affected our findings. Secondly, it is important to consider the choice of test foods used in the current study. Two high fat foods (chocolate and crisps) and two low fat foods (rice cakes and grapes) were selected to test the

hypothesis that individual differences in food reward and calorie intake would be specific to high-fat foods. However, with regards to food reward, no such group by food type interaction was observed. One possibility is that food reward may be particularly pronounced when self-perceived food addicts are presented with their particular ‘problem’ food. Thus future research into food reward may benefit from utilizing a more individualised approach in selecting test foods. Finally, it is important to consider the possibility that differences in food reward, pre- and post-meal consumption, may have been due to order-effects. This may be particularly the case for performance on the tapping task in which factors other than satiety (e.g. boredom) may have reduced performance on this task. However, as we were primarily interested in differences *between* groups (i.e. self-perceived food addicts versus non-addicts), this issue is unlikely to have affected our overall findings. Nonetheless, another important issue that should be addressed in future research concerns the order in which eating-related questionnaires are completed. In particular, it is possible that in the current study, completing the YFAS prior to the assessment of self-perceived food addiction may have influenced participants’ responses on the latter.

Despite these limitations, the current study provides preliminary insight into patterns of eating which characterise a self-perceived addiction to food, and highlights a number of avenues for future research. In particular, it would be informative to compare YFAS-diagnosed food addicts with self-perceived food addicts on the measures of food reward and calorie intake. This was beyond the scope of the current study due to the very small number of YFAS-diagnosed food addicts (as would be expected based on previous research on self-perceived food addicts; Hardman et al., 2015; Meadows & Higgs, 2013). It will also be important to replicate the current findings in male participants and in larger and more diverse samples. Finally, it would be interesting for future research to more specifically explore how food reward and calorie intake in self-perceived food addicts may be differentially affected by various macronutrient food profiles (e.g. high-fat, high carbohydrate vs. high-fat low carbohydrate).

To conclude, the current study provides evidence for increased calorie intake in self-perceived food addicts, despite no differences in hunger or overall liking. Furthermore, compared to non-addicts, self-perceived food addicts displayed higher

desire-to-eat ratings across foods, but did not differ on other measures of food reward (i.e. WtP and tapping frequency). However, differences in calorie intake and food reward between self-perceived food addicts and non-addicts were no longer significant after controlling for measures of dietary disinhibition and restraint. Taken together, these findings suggest that self-perceived food addicts experience food as more rewarding and are at particular risk of overeating. Furthermore, this may be attributable to the dual influences of increased dietary disinhibition and decreased restraint.

Chapter 4: Attentional bias to food cues in self-perceived food addicts

4.1. Overview

Findings from Chapters 2 and 3 suggest that self-perceived food addicts find food particularly rewarding and have an increased preoccupation with food and eating. According to Field et al. (2016), the reward value of a food may be reflected by the degree to which an individual allocates his or her attention to associated cues. As such, the primary aim of the Chapter 4 was to test whether self-perceived food addicts would demonstrate increased attentional bias to high-fat food cues (i.e. chocolate pictures) compared to self-perceived non-addicts. Previous research has demonstrated increased attentional bias to food cues when participants are hungry, relative to satiated (e.g. Castellanos et al., 2009), and when the food or reward is perceived to be imminently available (Field et al., 2011; Jones, Hogarth, et al., 2012). Additionally, the state effects of hunger and perceived availability tend to be moderated by trait characteristics (e.g. weight status, drinking frequency) (Castellanos et al., 2009; Field et al., 2011). Accordingly, a secondary aim of Chapter 4 was to examine whether self-perceived food addiction would moderate the effect hunger and perceived expectancy on attentional bias.

4.2. Abstract

Attentional bias (AB) to food cues is influenced by the extent to which a cue predicts imminent food receipt (i.e. expectancy), and an individual's current motivational state (i.e. hunger). Previous research suggests that the motivational salience of food may be particularly high in individuals who perceive themselves to be addicted to food (i.e. self-perceived food addicts). Therefore, trait self-perceived food addiction may also influence food-related AB and moderate the state effects of expectancy and hunger. To investigate this, 120 female participants completed an eye-tracking task which assessed AB to chocolate pictures. Participants' expectations of receiving chocolate (100%, 50%, 0%) were manipulated on a trial-by-trial basis. Half of these participants completed the task when hungry, and half were satiated. AB to chocolate pictures was greater on 100% and 50% trials, compared to 0% trials, indicating an effect of expectancy. However, there was no effect of hunger condition (hungry/satiated). Self-perceived food addiction did not influence AB, nor did it

moderate the effects of expectancy or hunger condition. Subsequent analyses revealed that desire to eat (DtE) chocolate was associated with increased AB. DtE chocolate also moderated the effect of expectancy on AB; participants with high DtE showed sensitivity to the expectancy information, while no effect of expectancy was observed in low-DtE participants. Finally, DtE correlated positively with self-perceived food addiction. Results suggest that AB to food cues is influenced by the current expectancy of food receipt, but only for those with higher DtE. Momentary levels of DtE may be more relevant in determining food-related AB than self-perceived food addiction per se.

4.3. Introduction

In the current obesogenic environment of Western society, foods that are high in fat and sugar are imminently available. According to the Incentive Sensitization theory of addiction and obesity (Berridge & Robinson, 1998; Robinson & Berridge, 1993; Robinson & Berridge, 2008), repeated consumption of these foods sensitises the release of dopamine in the brain. This, in turn, increases the rewarding value of the food, resulting in increased subjective craving or desire. Through a process of classical conditioning, cues that have previously been associated with the food, such as visual or orosensory stimuli, become sufficient at eliciting food-related conditioned responses including increased salivation, cravings, and an increased ‘attentional bias’ to food-related stimuli. Attentional bias is therefore thought to reflect the underlying motivational or ‘incentive value’ of a food, and thus varies both within (i.e. state influences) and between (i.e. trait influences) individuals (Field et al., 2016).

One trait factor which may affect attentional bias to food cues is the extent to which an individual demonstrates addictive patterns of eating. The Yale Food Addiction Scale (YFAS; Gearhardt et al., 2009) is currently the most widely used tool for the assessment of ‘food addiction’. The scale was adapted using the DSM criterion for substance-dependence, and provides both a continuous ‘symptom count’ measure, and a dichotomous diagnostic measure. Using this measure, women with increased food addiction symptomology have been found to demonstrate faster reaction times to food pictures, which is thought to indicate enhanced attentional processing towards food items (Meule, Lutz, Vögele, & Kübler, 2012). Similarly, in

an eye-tracking paradigm, Frayn, Sears, and von Ranson (2016) demonstrated increased attention to unhealthy food pictures (relative to healthy food and non-food images) in those who met the YFAS diagnostic criterion for food addiction, compared to those who did not meet this criterion.

However, the extent to which the YFAS provides a valid assessment of addictive-like eating is widely debated (e.g. Ziauddeen et al. 2012). In particular, the applicability of the DSM criterion for substance dependence to the assessment of eating behaviours is somewhat limited by the physiological and societal differences between drugs and food. As such, Ziauddeen et al. (2012) posit the need to develop an operational definition of food addiction that is not constrained by existing conceptualisations of substance dependence.

To address this suggestion, we have previously investigated the cognitions and behaviours of those who *perceive* themselves to be ‘food addicts’ (i.e. self-perceived food addicts). In a previous qualitative study, we found that self-perceived food addicts reported a particular ‘problem’ controlling their intake of foods high in fat and/or sugar, and a ‘preoccupation’ with food and eating (Ruddock, Dickson, Field, & Hardman, 2015; Chapter 2 in the current thesis). In a subsequent experimental paradigm, we found that self-perceived food addicts demonstrated increased food reward (assessed using a measure of ‘desire to eat’) and *ad libitum* calorie intake, compared to those who did not perceive themselves as food addicts (Ruddock et al., in press; Chapter 3 of the current thesis). Building upon these findings, the primary aim of the current study was to investigate whether self-perceived food addicts would demonstrate increased attentional bias to chocolate-pictures (vs. neutral pictures) compared to self-perceived non-addicts.

A secondary aim of the current study was to investigate whether self-perceived food addiction would moderate within-subject ‘state’ effects on attentional bias to chocolate-pictures. One factor that has been found to exert a *state* influence on attentional bias, is the perceived availability of the reward. Using an eye-tracking procedure, Jones et al. (2012) and Field et al. (2012) quantified attentional bias towards alcohol or chocolate pictures. Prior to each trial, participants’ expectations of receiving chocolate or alcohol were manipulated (100%, 50%, or 0% chance). Indicative of a state influence of expectancy, attentional bias towards chocolate or alcohol pictures was greater when participants believed they had 100% chance of

receiving the reward, compared to when they had 0% or 50% chance. It is thought that, by increasing anticipation for a reward, cues that signify the imminent availability of the reward serve to increase its incentive value and hence attentional bias (Field & Cox, 2008).

However, using a similar paradigm, Hardman et al. (2014) failed to uncover any effect of expectancy on attentional bias to food pictures in hungry participants. One possibility is that the presence of physiological hunger may have exerted a ceiling effect, such that the expectancy information was unable to further increase the incentive value of the food pictures. Indeed, hunger and satiety are thought to exert further influence over a food's incentive value (Field et al., 2016), and studies have demonstrated increased attentional bias to food cues in hungry, compared to satiated, participants (Channon & Hayward, 1990; Lavy & van den Hout, 1993; Mogg, Bradley, Hyare, & Lee, 1998; Placanica, Faunce, & Soames Job, 2001; Stockburger, Hamm, Weike, & Schupp, 2008; Stockburger, Schmalzle, Fleisch, Bublatzky, & Schupp, 2009).

The state effects of hunger and perceived expectancy have previously been found to moderate the effects of trait influences on attentional bias to food and reward cues. For example, previous research has demonstrated increased attentional bias to food cues in overweight, obese, or binge eating participants (Braet & Crombez, 2003; Castellanos et al., 2009; Graham, Hoover, Ceballos, & Komogortsev, 2011; Hendrikse et al., 2015; Kemps, Tiggemann, & Hollitt, 2014a; Nijs, Franken, & Muris, 2010; Nijs, Muris, et al., 2010; Schmitz, Naumann, Biehl, & Svaldi, 2015; Werthmann et al., 2011; Yokum et al., 2011). However, this was not observed in studies that failed to control for the effects of hunger (Loeber, Grosshans, Herpertz, Kiefer, & Herpertz, 2013; Phelan et al., 2011; Pothos, Tapper, & Calitri, 2009; Soetens & Braet, 2007). Similarly, Castellanos et al. (2009) found increased food-related attentional bias in obese compared to normal weight participants when satiated, but not when hungry. Furthermore, drinking frequency has been found to moderate the effect of perceived expectancy on attentional bias to alcohol pictures (Field et al., 2011). Specifically, the effect of expectancy information on attentional bias was only evident in less frequent drinkers; heavy drinkers allocated their attention towards the alcohol pictures regardless of the expectancy information.

Based on these findings, it was predicted that the trait influence of self-perceived food addiction on attentional bias to chocolate-pictures would be most pronounced when participants were satiated, rather than hungry. To investigate this, participants either completed the attentional bias task when they were hungry (hungry condition) or following the consumption of a lunch meal (satiated condition). Finally, it was hypothesised that self-perceived food addiction would moderate the effect of expectancy on attentional bias. Consistent with methods used in previous studies (Field et al., 2011; Hardman et al., 2014; Jones et al., 2012), participants' expectation of winning chocolate was manipulated prior to each trial. Drawing upon previous findings (Field et al., 2011), we might expect to observe a *diminished* effect of the expectancy information in self-perceived food addicts compared to non-addicts. Alternatively, due to an increased motivation to obtain chocolate, self-perceived food addicts may demonstrate *increased* sensitivity to the expectancy information.

To summarise, the current study tested the following three hypotheses: 1) Attentional bias to chocolate pictures (vs. neutral pictures) would be greater for self-perceived food addicts compared to non-addicts; 2) The effect of self-perceived food addiction on attentional bias to chocolate pictures would be most pronounced in the satiated condition, relative to the hungry condition; 3) The effect of the expectancy information on attentional bias to chocolate pictures would be either increased or decreased in self-perceived food addicts relative to non-addicts.

4.4. Method

4.4.1. Participants

Female participants ($N=120$) were recruited from the University of Liverpool via poster and online advertisements. Participants were informed that the aim of the study was to investigate the relationship between food reward and eating behaviour. Inclusion criteria required that participants were non-smokers, had no food allergies or intolerances, had never been diagnosed with an eating disorder, and were not on any medication known to affect appetite. Vegans, or anyone who would be unwilling to consume milk chocolate and cheese sandwiches, were also excluded. Finally, due to the eye-tracking technique used, glasses wearers were unable to take part. All participants completed a medical history questionnaire prior to testing to ensure that

they did not suffer from any food allergies. Participants were asked not to eat or consume any calorie-containing drinks for 3 hours before the study. All participants indicated that they had complied with this instruction. Ethical approval was granted by the Institute of Psychology, Health and Society at the University of Liverpool. Participants received course credits or were reimbursed with a £5 shopping voucher as compensation for their time and travel expenses.

4.4.2. Measures and Materials

4.4.2.1. Appetitive ratings.

Levels of hunger, fullness, and desire-to-eat chocolate were assessed using 100mm Visual Analogue Scales (VAS). Each scale was anchored by 'Not at all' on the left and 'Extremely' on the right.

4.4.2.2. Lunch meal.

To induce satiety, participants in the satiated condition were provided with cheese sandwiches. Sandwiches were made using 3 slices of Lidl Simply medium sliced white bread (255kcal, 3g fat), 1.5 pieces of Tesco medium pre-sliced cheddar (56g, 236kcal, 20g fat), and 15g butter (Tesco Butterpak, 95kcal, 11g fat). These were then sliced into six small sandwiches. Participants were given 10 minutes in which to consume the entire meal.

4.4.2.3. Self-perceived food addiction.

To assess self-perceived food addiction (FA), participants indicated the extent to which they agreed with the statement "I believe myself to be a food addict". Responses were provided on a 5-point Likert scale which ranged from 'Strongly disagree' to 'Strongly agree'.

4.4.2.4. Attentional bias task

Pictorial stimuli

All stimuli were presented using Inquisit (2.0) on a 15" computer screen. The pictorial stimuli used in the expectancy task consisted of 10 pairs of photographs. Each pair contained one chocolate-related photograph and one matching control photograph (i.e. stationery items). Picture pairs were matched as closely as possible for colour, complexity, brightness, shape, and size. Each picture was 100mm high

and 125mm wide. Four additional picture pairs depicting stationery items were used for the practice trials.

Expectancy task.

The task was similar to that used in previous research (Field et al., 2011; Jones et al., 2012; Hardman et al. 2014). Participants were led to believe that they were playing for ‘points’ which, following the task, would be exchanged for chocolate. During each trial, the expectancy of ‘winning’ a point was manipulated. Specifically, participants were instructed to pay attention to a percentage (100%, 50%, or 0%) that was presented in the center of the screen for 1000 milliseconds at the start of each trial. Participants were explicitly told that this percentage represented the probability that they would ‘win’ a point on that particular trial. The percentage was then followed by the presentation of a picture pair (i.e. chocolate image and control image) for 2000 milliseconds during which eye movements were recorded. Following picture offset, the instruction ‘press SPACE BAR to try and win!’ was presented in the center of the screen. Pressing the space bar triggered the feedback screen in which participants were informed whether or not they had ‘won’ a point. On all 100% trials, and half of the 50% trials, the feedback stated “You win a chocolate point”. On all 0% trials, and half of the 50% trials, the feedback stated “You win nothing”. The feedback screen was displayed for 1000 milliseconds. The order and duration of each screen presentation is shown in Figure 4.1. Four practice trials were presented prior to the start of the task (one 100% trial, one 0% trial, and two 50% trials). The main block consisted of 120 trials. Each trial type (i.e. 100%, 50%, or 0%) was presented 40 times. The positioning of chocolate pictures was such that they appeared on the left and right side of the screen with equal frequency for each trial type. Participants were seated approximately 23 inches away from the computer screen with their chin on a chin-rest. Eye movements were recorded using an Eye-Trac D6 desktop mounted camera (Applied Science Laboratories, Bedford, MA). The task lasted approximately 15 minutes.

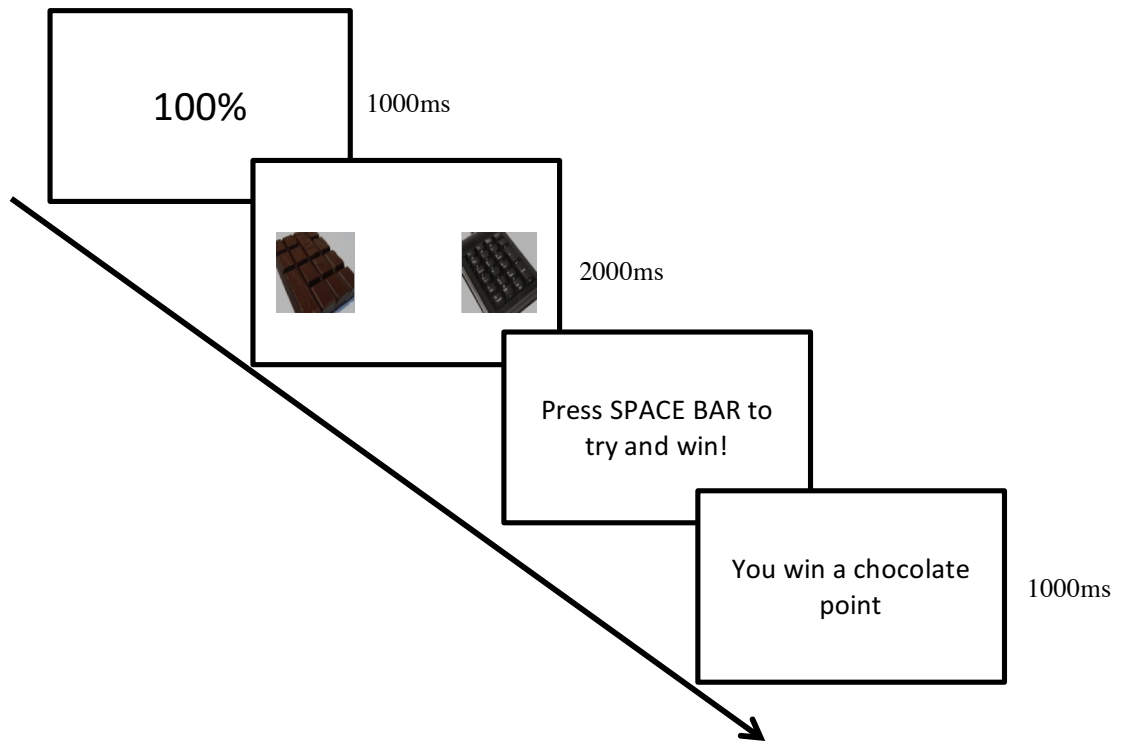


Figure 4.1. Order and duration of screen presentation in the eye-tracking task during a single trial. The task consisted of 120 trials and each trial type (i.e. 100%, 50%, 0%) was presented 40 times.

4.4.2.5. Additional measures and eating trait questionnaires.

The Yale Food Addiction Scale (YFAS; Gearhardt et al., 2009), Three Factor Eating Questionnaire (TFEQ, Stunkard & Messick, 1985), and Binge Eating Scale (BES; Gormally, Black, Daston, & Rardin, 1982) were used to provide descriptive information about the sample (see Chapter 3, section 3.4.2. for a full description of these measures).

Familiarity ratings.

Participants were asked to indicate how often they ate chocolate. The following response options were given: ‘Never’, ‘Monthly or less’, ‘2-4 times a month’, ‘2-3 times a week’, ‘4 or more times a week’, ‘Every day’. Participants indicated how often they ate each food by ticking the appropriate box.

4.4.3. Procedure

All sessions were conducted between 12pm and 6pm and took approximately 1 hour to complete. Prior to each session, participants were randomly allocated to either hungry or satiated conditions. Upon arrival, participants provided written informed consent and completed a medical history questionnaire to ensure the absence of any food allergies. To ensure compliance with the study procedure, participants were asked to confirm that they had not eaten for at least 3 hours prior to the study. Participants indicated their current levels of hunger, fullness, and desire-to-eat chocolate. Those in the satiated condition then ate the cheese sandwiches, while those in the hungry condition read a magazine for 10 minutes. Levels of hunger, fullness, and desire-to-eat chocolate were then reassessed. Participants then completed the eye-tracking task in which they were led to believe that they were playing for ‘chocolate points’. Levels of hunger, fullness, and desire-to-eat chocolate were assessed again at this stage. Participants were then given a bowl containing 100g of chocolate (galaxy counters: 528 kcal, 28.9g fat) under the pretence that this was what they had ‘won’ during the task. Participants were invited to consume as much as they wished. Chocolate intake was measured by covertly weighing the bowl before and after consumption. Following this, participants’ levels of hunger, fullness, and desire-to-eat chocolate were assessed again, and participants completed the chocolate familiarity scale. To ensure the absence of demand characteristics, participants were asked to indicate what they thought the aims of the study were. Finally, participants completed the measure of self-perceived food addiction, TFEQ, YFAS, and BES, and measures of height and weight were taken to calculate BMI. Participants were fully debriefed and thanked for their time. Figure 4.2 presents a flow-chart of the study procedure.

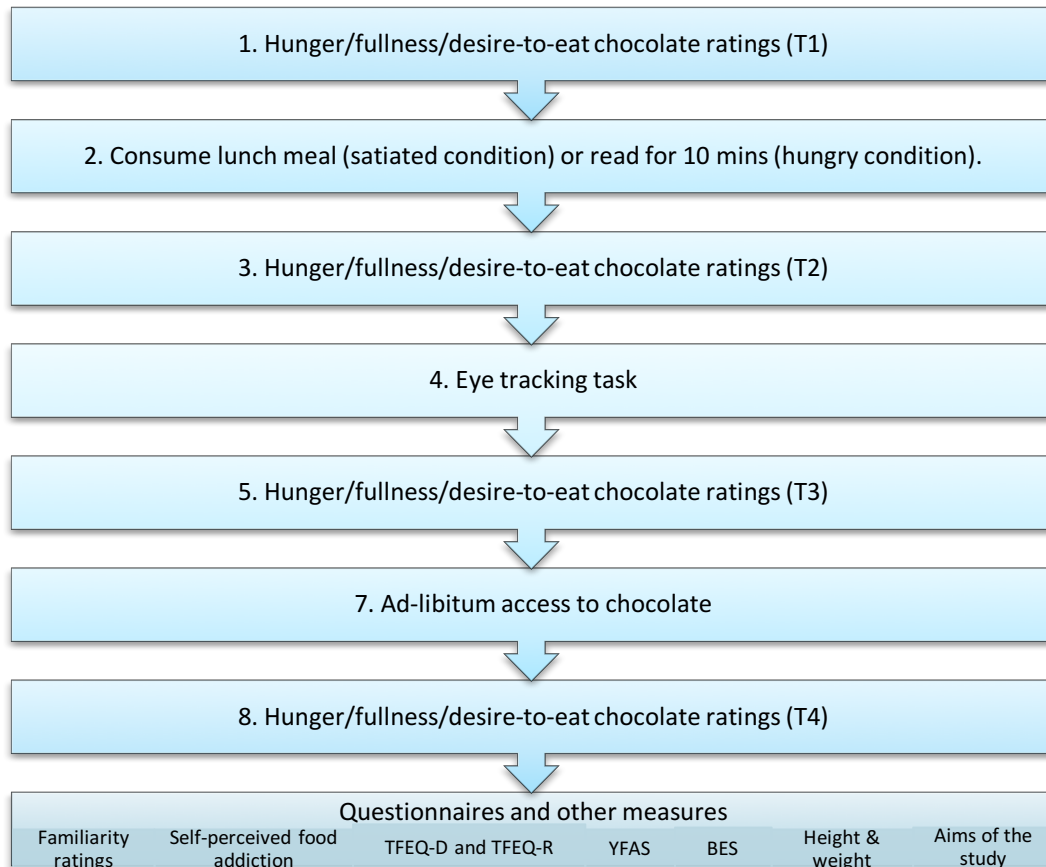


Figure 4.2. Flow chart of the study procedure

4.4.4. Data analysis

4.4.4.1. *Self-perceived food addiction*

Prior to data analysis, self-perceived food addicts and non-addicts were identified based on participants' responses to the assessment of self-perceived food addiction. Those who ticked 'Agree' or 'Strongly agree' to the assessment of self-perceived food addiction were grouped as 'food addicts', while those who ticked 'Disagree' or 'Strongly disagree' were grouped as 'non-addicts'. Those who indicated that they 'Neither agree nor disagree' were classed as 'Undecided'. A chi-square analysis was conducted to ensure that the number of self-perceived food addicts, non-addicts and 'undecided' participants were evenly distributed across hungry and satiated conditions.

4.4.4.2. Appetite ratings

Initial mixed design ANOVAs were conducted to explore differences in desire-to-eat, hunger, and fullness between time-points 1 (T1; i.e. upon arrival to the lab), time-point 2 (T2; i.e. following consumption of the sandwich or after 10 minutes of reading), time-point 3 (T3; i.e. following the attentional bias task), and time-point 4 (T4; i.e. following ad libitum chocolate intake). As self-perceived FA may have moderated the effect of condition (i.e. hungry/satiated) on appetite ratings, this was included in the ANOVA as a between-subjects factor. Each ANOVA therefore comprised a 2 (condition: hungry/satiated) x 3 (group: self-perceived food addicts/non-addicts/undecided) x 2 (time-point: T1/T2) design.

4.4.4.3. Attentional bias

For each participant, mean gaze duration (i.e. the amount of time spent looking at each picture) to chocolate and neutral pictures was calculated for each trial type (i.e. 0%, 50%, 100%). To check for the presence of attentional bias to chocolate pictures, gaze duration was analysed using a 3 (expectancy: 100%, 50%, 0%) x 2 (picture type: chocolate/neutral) repeated measures ANOVA. Attentional bias scores were then calculated by subtracting gaze duration to neutral pictures from gaze duration to chocolate pictures. A positive score indicated a attentional bias towards the chocolate pictures, while a negative score indicated a attentional bias towards the neutral pictures. The effect of expectancy, condition, and group on attentional bias scores was explored using a 3 (expectancy: 100%, 50%, 0%) x 2 (condition: Hungry/Satiated) x 3 (group: self-perceived food addicts/non-addicts/undecided) mixed ANOVA.²

Hypothesis 1 predicted a main effect of group, such that attentional bias to chocolate pictures (vs. neutral pictures) would be most pronounced in self-perceived food addicts compared to non-addicts. Hypothesis 2 predicted a group (self-

² Analyses of attentional bias were repeated using YFAS symptomology (instead of self-perceived food addiction) as a between-subjects factor. For this, participants were grouped into either high ($n=62$) or low ($n=56$) YFAS groups based on a median split of YFAS symptom scores. Those in the high YFAS group met the criteria for 2 or more symptoms, while those in the low YFAS group met the criteria for 0-1 symptoms. The number of participants in each YFAS group was evenly distributed across hungry (low: $n=31$; high: $n=28$) and satiated (low: $n=25$; high: $n=34$) conditions, $X^2(1)=1.22$, $p=.357$.

perceived food addicts vs. non-addicts) x condition (hungry vs. satiated) interaction, such that increased attentional bias to chocolate-pictures, in self-perceived food addicts, was expected to be most pronounced in the satiated condition, relative to the hungry condition. Hypothesis 3 predicted a group (self-perceived food addicts vs. non-addicts) x expectancy (100%, 50%, 0%) interaction. Specifically, the effect of expectancy on attentional bias to chocolate-pictures was expected to be either increased or decreased in self-perceived food addicts, relative to non-addicts.

4.5. Results

4.5.1. Participant characteristics

Female participants ($N=120$) took part in the study. Due to technical problems with the eye-tracker, data from two participants were lost. Data analysis was therefore conducted on 118 complete datasets (hungry condition: $n=59$; satiated condition: $n=59$). Participant characteristics are provided in Table 4.1. A MANOVA confirmed that participants did not differ, between conditions, with regards to any of these characteristics, $F(9,105)=1.04$, $p=.412$. Furthermore, a chi-squared test showed that the number of self-perceived food addicts, non-addicts, and undecided participants did not differ between hungry and satiated conditions, $X^2(2)=.83$, $p=.659$. All participants indicated that they consumed chocolate at least 2-3 times a week.

Table 4.1. Participant characteristics in the hungry and satiated conditions. Unless otherwise stated, values are means \pm standard deviations.

Characteristic	Hungry ($n=59$)	Satiated ($n=59$)	Total ($n=118$)
Age (years)	25.6 \pm 8.3	25.0 \pm 10.2	25.3 \pm 9.2
BMI (kg/m^2)	23.4 \pm 5.1	23.9 \pm 5.1	23.7 \pm 4.9
TFEQ-D	7.5 \pm 3.4	7.5 \pm 3.1	7.5 \pm 3.3
TFEQ-R	9.2 \pm 4.9	7.5 \pm 4.3	8.3 \pm 4.7
BES	10.1 \pm 6.6	10.6 \pm 7.3	10.4 \pm 6.9
YFAS symptom count	1.81 \pm 1.38	2.14 \pm 2.14	1.97 \pm 1.39
Chocolate familiarity	3.32 \pm .84	3.46 \pm .97	3.39 \pm .91
Chocolate liking	73 \pm 80	80 \pm 16	77 \pm 19
Self-perceived FA, non-addicts, undecided (n)	16,27,15	20,27,12	36,54,27
YFAS diagnosis (n)	3	4	7

4.5.2. Appetite ratings

Ratings of hunger, fullness, and desire-to-eat chocolate over each of the four time-points are depicted in Figure 4.3. There was a main effect of time on hunger, fullness, and desire-to-eat chocolate ratings ($ps < .001$), and a main effect of condition on hunger and fullness ratings ($ps < .001$). However, there was no main effect of condition on desire-to-eat chocolate ratings, $F(1,112)=2.94$, $p=.089$, $\eta p^2=.03$. Significant condition x time interactions were observed for desire-to-eat, hunger, and fullness ratings ($ps < .001$). These interactions were followed-up using paired-samples t-tests conducted for each condition. In particular, differences between time-points 1 and 2 (i.e. before and after the lunch meal) were investigated to ensure that the satiated condition had the desired effect of reducing appetite. In the satiated condition, hunger and desire-to-eat ratings decreased, while fullness ratings increased, significantly, between T1 and T2 (all $ps < .001$). Appetite ratings did not change in the hungry condition between T1 and T2 ($ps > .137$). This confirms that the lunch meal was effective in reducing appetite and increasing fullness in the satiated condition, in the absence of any changes in the hungry condition. There was no 3-way interaction of time x condition x group (self-perceived food addicts/non-addicts/undecided) on any appetite measure (all $ps > .233$). Exploratory analyses were also conducted to compare the decline in hunger and desire-to-eat ratings between T1 and T2 in the satiated condition. Hunger and desire-to-eat rating decline was calculated by subtracting ratings obtained at T2, from those obtained at T1. A paired-samples t-test revealed that the decline in hunger ratings ($M=45 \pm 24$) was significantly greater than the decline in desire-to-eat ratings ($M=18 \pm 24$), $t(58)=7.79$, $p < .001$.

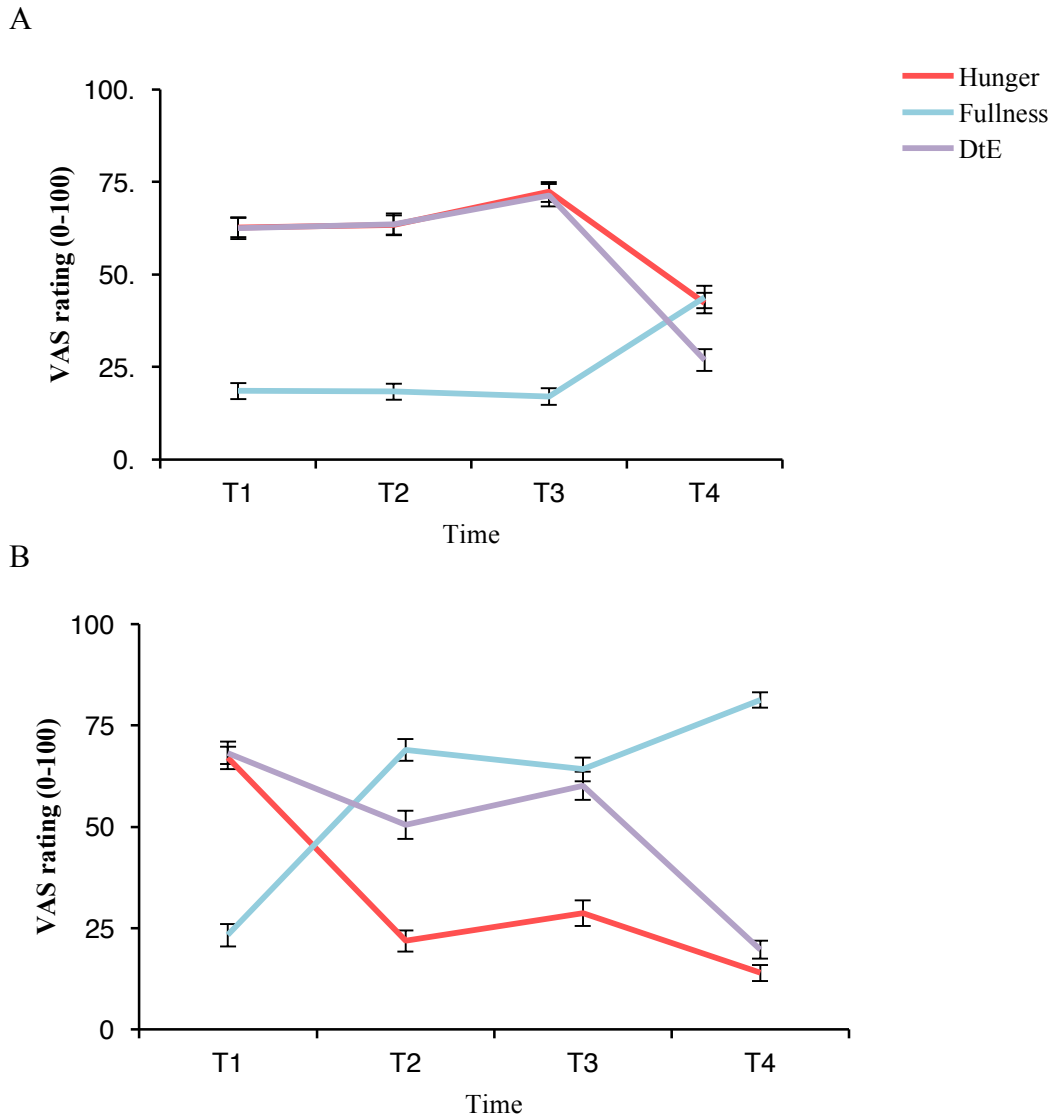


Figure 4.3. Ratings of hunger, fullness, and desire-to-eat chocolate at each time-point for hungry (Panel A) and satiated (Panel B) conditions. Values are means and standard errors.

4.5.3. Attentional bias

Analyses revealed a main effect of picture type, $F(1,117)=75.88, p<.001, \eta p^2=.39$, such that participants demonstrated increased overall gaze duration towards the chocolate ($M=719\text{ms} \pm 259$) compared to neutral pictures ($M=490\text{ms} \pm 191$) indicating an attentional bias to chocolate-related cues. Contrary to our first hypothesis, there was no main effect of group (i.e. self-perceived food addicts vs. non-addicts) on attentional bias to chocolate-pictures, $F(2,112)=.06, p=.945, \eta p^2=.00$. There was also no group x condition interaction, $F(2,112)=.51, p=.600, \eta p^2=.01$

(hypothesis 2), and no group x expectancy interaction, $F(3.53, 197.90)=.88, p=.465, \eta^2=.02$ (hypothesis 3).³

There was, however, a main effect of expectancy on attentional bias scores, $F(1.77,197.90)=11.01, p<.001, \eta^2=.09$. Pairwise comparisons revealed that participants demonstrated greater attentional bias towards the chocolate pictures when they had 100% ($M=255\text{ms} \pm 328$)($p=.001$) or 50% ($M=249\text{ms} \pm 307$)($p<.001$) chance of winning, compared to when they had 0% chance ($M=182\text{ms} \pm 287$). Attentional bias scores did not differ significantly between 100% and 50% trials ($p=.657$). There was no main effect of hunger condition, $F(1,112)=.128, p=.722, \eta^2=.001$, and no expectancy x condition interaction, $F(1.77,197.90)=1.21, p=.297, \eta^2=.011$, on attentional bias scores.

4.5.4. Exploratory analyses: Desire-to-eat

Exploratory correlational analyses were conducted to investigate relationships between the dependent variables (see Table 4.2). These revealed a significant positive correlation between desire-to-eat chocolate (DtE) and attentional bias on 50% and 100%, but not on 0% trials. Furthermore, desire-to-eat ratings correlated positively with self-perceived food addiction.

Table 4.2. Correlation coefficients between dependent variables. Values were collapsed across conditions (hungry and satiated). Hunger and DtE ratings were taken at T2 (i.e. just prior to the eye-tracking task) * $p<.05$, ** $p<.001$

	Expectancy	Hunger	DtE	Self-perceived FA	YFAS symptom count	Chocolate intake
Attentional bias	0%	.132	.145	-.021	.047	.000
	50%	.082	.237**	-.035	.125	.040
	100%	.044	.249**	-.056	.170	.042
Hunger			.501**	.069	-.035	.226*
DtE				.189*	.031	.365**
Self-perceived FA					.312**	.173
YFAs symptomology						.092

³ Similar null results were obtained when using YFAS symptom group (i.e. high vs. low symptom count) as a proxy for addictive eating ($ps > .125$).

To investigate the possibility that DtE chocolate may have moderated the effect of expectancy on attentional bias, an ANCOVA was conducted with expectancy as a within-subjects variable, and DtE as a covariate. There was an expectancy x DtE interaction which approached significance, $F(1.77, 205.24)=2.62$, $p=.082$, $\eta p^2=.02$. To investigate this further, participants were divided into either ‘high DtE’ ($n=60$) or ‘low DtE’ ($n=58$) groups based on a median split of DtE ratings at T2 (i.e. just prior to the eye-tracking task). The mean (\pm SD) DtE VAS rating was 77mm (\pm 11) and 37mm (\pm 19) for the high and low DtE groups, respectively. This was entered into a 3 (expectancy) x 2 (DtE chocolate) mixed ANOVA with attentional bias scores as the dependent variable. There was a main effect of DtE chocolate, $F(1,114)=5.55$, $p=.020$, $\eta p^2=.05$, such that those in the high DtE group demonstrated greater attentional bias towards the chocolate ($M=288\text{ms} \pm 275$) than those in the low DtE group ($M=166\text{ms} \pm 275$). There was also an interaction between DtE and expectancy, $F(1.79, 203.96)=5.54$, $p=.006$, $\eta p^2=.05$ (see Figure 4.4). Paired samples t-tests, conducted separately for low and high DtE groups revealed that, for those in the low DtE group, attentional bias did not differ between 0%, 50%, or 100% trials (all $p_s >.341$). However, for those with high DtE, attentional bias was significantly higher on 50% trials, $t(59)-4.02$, $p<.001$, $d=.37$, and 100% trials, $t(59)=-4.11$, $p<.001$, $d=.42$, compared to 0% trials. Attentional bias did not differ between 50% and 100% trials in the high DtE group, $t(59)=-.90$, $p=.373$.

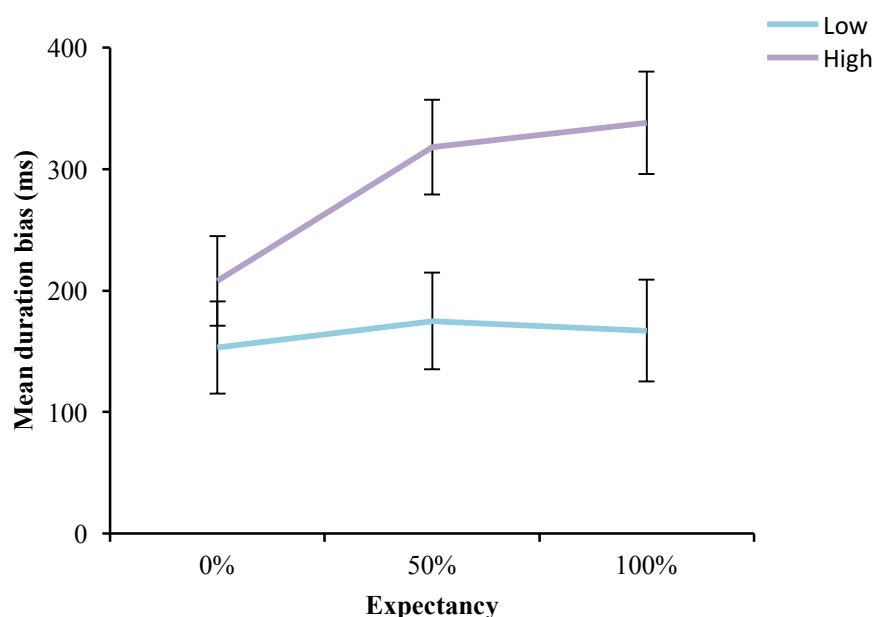


Figure 4.4. Mean duration bias as a function of expectancy information and desire-to-eat chocolate

4.5.5. Predictors of chocolate intake

An exploratory multiple linear regression analysis was conducted to examine the extent to which *ad libitum* chocolate intake could be predicted from appetitive measures (i.e. hunger, fullness, and desire-to-eat), self-perceived food addiction, YFAS symptom count, and attentional bias. Hunger, fullness, and desire-to-eat ratings from time-point 3 (T3; i.e. just prior to *ad libitum* chocolate intake) were included in the model. Attentional bias scores were collapsed across all 3 trial types (i.e. 0%, 50%, 100%) to provide an overall attentional bias score. Desire-to-eat ratings provided the only significant predictor of subsequent chocolate intake (Table 4.3).

Table 4.3. Output from linear regression model of variables predicting chocolate intake(g). Values for hunger, fullness, and DtE were taken at T3 (i.e. just prior to *ad libitum* intake). **Significant at $p < .001$.

	<i>B</i>	<i>Std. Error B</i>	<i>p</i>
Constant	-.08	13.96	.996
Hunger	.15	.15	.318
Fullness	.12	.14	.407
DtE	.30	.09	.001**
Self-perceived FA	2.26	2.00	.255
YFAS symptomology	.89	1.55	.564
Attentional bias	-2.20	7.23	.761

4.6. Discussion

Our previous research suggests that self-perceived food addicts find high-fat/sugar foods particularly rewarding, and have a ‘preoccupation’ with food and eating (Ruddock et al., 2015; Ruddock et al., in press; Chapters 2 and 3 of the current thesis). Drawing upon these findings, the primary aim of the current study was to investigate whether self-perceived food addicts would demonstrate increased attentional bias to chocolate-pictures (vs. neutral pictures), relative to self-perceived non-addicts (hypothesis 1). A secondary aim was to explore whether the effect of self-perceived food addiction would be moderated by condition (i.e. hungry vs. satiated), and perceived expectancy. Specifically, it was predicted that the effect of self-perceived food addiction would be most pronounced when participants were

satiated, relative to hungry (hypothesis 2). Finally, the effect of perceived expectancy on attentional bias was expected to be either increased or decreased in self-perceived food addicts, relative to non-addicts (hypothesis 3).

Contrary to the hypotheses, results revealed no main effect of group (i.e. self-perceived food addicts vs. non-addicts) on attentional bias to chocolate-pictures. There was also no moderating effect of condition or perceived expectancy on the effect of self-perceived food addiction, contrary to our second and third hypotheses respectively. The lack of effect of group may be attributable to the absence of an effect of condition on attentional bias to chocolate cues (as discussed later). Specifically, while self-perceived food addicts were expected to have higher levels of attentional bias in the *satiated* condition, but not the hungry condition, the lack of between-condition differences may have obscured this effect.

Nonetheless, consistent with previous findings (Jones et al., 2012; Field et al., 2011), participants demonstrated greater attentional bias towards chocolate pictures when they were led to believe they had 100% chance of receiving chocolate compared to when they had 0% chance. These findings lend further support to the suggestion that attentional bias develops towards stimuli that predict imminent receipt of a reward (Field & Cox, 2008). It is also important to note that, compared to 0% trials, attentional bias increased when the chances of receiving chocolate were uncertain (i.e. 50% trials). These findings differ from previous research in which attentional bias to alcohol pictures did not differ significantly between 0% and 50% trials (Field et al., 2011). While these findings are *partly* consistent with the suggestion that increased attentional bias should be observed in situations in which the outcome is uncertain (Pearce & Hall, 1980), this was not fully supported by the current findings as attentional bias was greater on 100% trials, compared to 50% trials, albeit not significantly. Similar linear relationships between expectancy and early attentional bias to food and cigarette cues have previously been observed (Carter & Tiffany, 2001; Hardman et al., 2014).

Contrary to previous findings (Channon & Hayward, 1990; Lavy & van den Hout, 1993; Mogg, Bradley, Hyare, & Lee, 1998; Placanica, Faunce, & Soames Job, 2001; Stockburger, Hamm, Weike, & Schupp, 2008; Stockburger, Schmalzle, Fleisch, Bublatzky, & Schupp, 2009), participants in the hungry condition did not

demonstrate any increased attentional bias towards chocolate pictures compared to those in the satiated condition. One possibility is that the consumption of cheese sandwiches in the satiated condition did not sufficiently reduce the reward value of chocolate. Indeed, previous research has demonstrated the role of sensory specific satiety on the modification of attentional bias to food pictures. Specifically, di Pellegrino, Magarelli, & Mengarelli (2011) reported diminished attentional bias towards an *eaten* food, but not towards an *uneaten* food. In further support of this suggestion, the current study found that desire-to-eat chocolate ratings did not diminish to the same extent as physiological hunger following consumption of the cheese sandwich which had different sensory properties. This suggests that chocolate may have continued to function as an effective reinforcer despite recent eating.

In the relation to the above point, post-hoc analyses revealed that desire-to-eat chocolate played a key role in determining attentional bias to chocolate pictures. Firstly, participants with higher levels of desire-to-eat chocolate demonstrated greater overall attentional bias towards chocolate pictures than participants with lower levels of desire-to-eat. Second, consistent with our previous findings (Ruddock et al., in press; Chapter 3), self-perceived food addicts had higher desire-to-eat chocolate ratings. Thus, while a direct relationship between self-perceived food addiction and attentional bias was not observed, these findings suggest that individuals who perceive themselves to be addicted to food may be more likely to develop attentional bias to food cues due to an increased subjective ‘desire-to-eat’. These findings provide support for the suggestion that within-subject state factors, such as desire-to-eat, exert stronger influence on attentional bias than trait characteristics (Field et al., 2016).

Post-hoc analyses also revealed a desire-to-eat by expectancy interaction such that only participants with high momentary levels of desire-to-eat chocolate demonstrated sensitivity to the expectancy information. These findings extend Field & Cox’s (2008) model of attentional bias by suggesting that the imminent availability of a reward may increase attentional bias, but only for individuals who have a pre-existing ‘desire’ for the reward. In the current study, it is possible that individuals with high levels of desire-to-eat chocolate would have been more interested in, and thus more likely to pay attention to, the expectancy information. To

address this, future research should examine whether those with higher levels of desire-to-eat demonstrate increased attentional bias to expectancy information, relative to neutral information, compared to those with lower levels of desire-to-eat.

Findings from the current study also contribute to a body of research examining the extent to which attentional bias predicts subsequent food intake. Contrary to previous findings (Nijs, et al., 2010; Werthmann, Renner, et al., 2014; Werthmann, Roefs, Nederkoorn, & Jansen, 2013), there was no positive association between attentional bias to chocolate pictures and chocolate consumption. Rather, ‘desire-to-eat’ ratings provided the only significant predictor of chocolate intake. These findings are consistent with Hardman et al. (2014) in which desire-to-eat ratings, and not attentional bias, positively predicted pizza consumption, and support the suggestion that desire-to-eat, attentional bias, and consumption represent distinct subjective and behavioural outputs of a food’s incentive value (Field et al., 2016; Rogers & Hardman, 2015). As such, while attentional bias may often predict subsequent consumption, we suggest that this relationship is reflective of a food’s underlying reward value. Future research should explore the extent to which desire-to-eat ratings, which are thought to provide a subjective measure of a food’s reward value (Rogers & Hardman, 2015), underlie positive relationships between attentional bias and subsequent intake.

The current study yields a number of limitations which should be considered in future research. Firstly, the use of a single food cue (i.e. chocolate pictures) for the assessment of attentional bias may have precluded the observation of individual differences between self-perceived food addicts and non-addicts. The use of chocolate cues was based on previous research which suggest that chocolate is perceived to be a particularly ‘addictive’ food (i.e. Ruddock et al., 2015, Schulte et al., 2015). However, evidence suggests that individuals’ ‘problem’ foods are highly idiosyncratic (e.g. Schulte et al., 2015), and therefore the stimuli used in the current study may not have been sufficient to capture differences in attentional bias to food-cues in self-perceived food addicts and non-addicts. Future research may therefore benefit from using personalised food stimuli to assess trait differences in attentional bias to food-cues. Secondly, the current study used an all-female sample, and thus it is not possible to generalize our findings to a male population. Nonetheless, as this was a preliminary study, it was necessary to minimize between-subject variability.

Future research is now required to explore state and trait influences on attentional bias to food-cues within a male sample.

It is also important to consider the possibility that individuals who fulfill an established measure of food addiction (i.e. the YFAS, Gearhardt et al., 2009) would demonstrate increased attentional bias to food-cues. Indeed, previous research has shown increased attentional allocation to food-cues in those who fulfil the YFAS diagnostic criterion, or have increased food addiction symptomology (Frayn et al., 2016; Meule et al., 2012). As only seven participants in the current study met the YFAS criteria, it was beyond the scope of the current study to explore this possibility. Importantly, however, in the current study, the YFAS symptom count measure was not associated with attentional bias to chocolate pictures or with desire-to-eat ratings for chocolate. This suggests that individual variation in food reward (i.e. reflected by assessments of desire-to-eat) may be more closely associated with an individual's perception of themselves as a food addict, than the number of YFAS 'symptoms' that they exhibit. Indeed, as previously discussed (Chapter 1, section 1.2.), the validity of the YFAS for the assessment of addictive eating is limited, and the scale may fail to account for many 'at-risk' individuals (as shown in Chapter 3).

In summary, the current study did not uncover any main effect of self-perceived food addiction on attentional bias to chocolate pictures. Self-perceived food addiction also did not moderate the effect of condition (i.e. hunger vs. satiety) or perceived expectancy on attentional bias to chocolate-pictures. Instead, our findings implicate a key role of 'desire-to-eat'. Firstly, individuals with high desire-to-eat chocolate demonstrated increased attentional bias to chocolate-pictures compared to those with low desire-to-eat chocolate. Secondly, desire-to-eat ratings correlated positively with self-perceived food addiction. Thirdly, desire-to-eat moderated the effect of expectancy on attentional bias, such that only those with high levels of desire-to-eat demonstrated sensitivity to the expectancy information. Finally, desire-to-eat ratings provided the only significant predictor of subsequent chocolate intake. Overall, these findings support the suggestion that ratings of desire-to-eat provide a subjective measure of a food's reward value which, in turn, influences cognitive and behavioural outputs such as attentional bias and subsequent consumption (Field et al., 2016; Rogers & Hardman, 2015).

Chapter 5: The Development and Validation of the Addictive Eating Behaviour Scale

5.1. Overview

Findings from Chapters 2-4 indicate that individuals who perceive themselves to be addicted to food (i.e. self-perceived food addicts) find food particularly rewarding and may have a particular propensity to overeat. Furthermore, these individuals appear to demonstrate patterns of aberrant eating that may go undetected by an existing measure of food addiction (i.e. YFAS; Gearhardt et al., 2009). This may be attributable to the limited applicability of the DSM substance-dependence criteria (upon which the YFAS is based) to the assessment of eating behaviours. As Ziauddeen et al. (2012) suggest, it is therefore necessary to develop an operational definition of food addiction that is not reliant upon existing conceptualisations of addiction. This would provide a starting point from which the concept of food addiction may be validated. Drawing upon this suggestion, Chapter 5 presents the development of a novel tool (the Addictive Eating Behaviour Scale) which assesses the presence of behaviours which are commonly attributed to addictive patterns of eating. Scale items were developed using participants' responses obtained in the qualitative study in Chapter 2.

The study reported in this chapter is currently under review as:

Ruddock, H.K., Christiansen, P., Halford, J.C.G., & Hardman, C.A (under review).
The Development and Validation of the Addiction-like Eating Behaviour Scale.

5.2. Abstract

Over-eating and obesity are frequently attributed to an addiction to food. However, there is currently a lack of evidence to support the existence of a substance-based food addiction. An alternative approach is to focus on dimensions of observable behaviour which may underpin a behavioural addiction to eating. It is therefore necessary to develop a tool to quantify addictive eating behaviour that is not based on the clinical criteria for substance dependence. To achieve this, the current study provides initial validation of the Addictive Eating Behaviour Scale (AEBS). English speaking male and female participants ($N=513$) from a community sample completed the AEBS, alongside a range of other health- and eating- related

questionnaires such as the Yale Food Addiction Scale (YFAS) and Binge Eating Scale (BES). Participants also provided their height and weight (to calculate body mass index, BMI). Principle components analysis revealed that a two-factor structure best accounted for the data. Factor 1 consisted of items which referred to weight gain and a tendency to overeat, while factor two consisted of items which referred to unhealthy eating practices and a perceived lack of dietary self-control. Both subscales demonstrated good internal reliability, and a confirmatory factor analysis confirmed the two-factor scale structure. AEBS scores correlated positively with BMI and other self-report measures of over-eating. Importantly, the AEBS significantly predicted variance in BMI above that accounted for by the YFAS and BES. The AEBS provides a valid and reliable tool to quantify addictive eating behaviours that is distinct from existing measures of food addiction. Further research is required to validate the scale in clinical samples and weight management contexts.

5.3. Introduction

Worldwide rates of obesity have more than doubled in the past three decades, with approximately 1.9 billion people classified as overweight (BMI > 25 kg/m²), and 600 million classified as obese (BMI > 30 kg/m²) (World Health Organisation, 2016). This recent rise in obesity is often attributed to the ‘addictive’ qualities of certain foods, and a popular theory holds that some people may develop an ‘addiction’ to food and eating (e.g. Kenny, 2013). However, while reward mechanisms common to addiction are, to an extent, also associated with control of eating behaviour, the validity of the ‘food addiction’ concept, and the way in which it should be defined and assessed, continues to be widely debated throughout the scientific community (e.g. Carter et al., 2016; Hebebrand et al., 2014; Ziauddeen et al., 2012).

Previous definitions and assessments of food addiction have relied upon the DSM-IV and DSM-V criteria for substance dependence (Gearhardt et al., 2009; Gearhardt et al., 2016). Specifically, the Yale Food Addiction Scale (YFAS; Gearhardt et al., 2009) provides a diagnosis of food addiction in cases where at least three of the following symptoms are met: 1. Food is consumed in larger amounts and for longer periods than intended. 2. There is persistent desire or unsuccessful attempts to quit. 3. The individual engages in much time or activity to obtain, use, or

recover from the effects of overeating. 4. Important social, occupational, or recreational activities are given up or reduced. 5. Use continues despite knowledge of adverse consequences. 6. Tolerance (i.e. Marked increase in amount, decrease in effect). 7. Characteristic withdrawal symptoms. In addition, to fulfil the YFAS diagnostic criteria for food addiction, an individual must demonstrate an impairment to daily functioning, or a clinically significant level of distress, as a result of their eating behaviour. A recent revision of the YFAS (YFAS 2.0; Gearhardt et al., 2016) also includes the assessment of ‘craving’, ‘failure in role obligation’, and ‘use despite interpersonal/social consequences’, in line with the DSM-V classification of Substance-Related and Addictive Disorders (SRAD).

However, the applicability of the clinical criteria for substance-dependence to the assessment of eating behaviours has been challenged. In particular, Ziauddeen et al. (2012) suggest that symptoms such as ‘withdrawal’ and ‘tolerance’ (i.e. requiring increasing amounts of a substance over time), which characterise frequent drug use, have not been reliably observed in humans in response to food. Furthermore, fundamental differences between the societal effects of drugs and food mean that problematic eating behaviours may not necessitate any ‘*impairment to daily functioning*’, or the cessation of ‘*important social, occupational, or recreational activities*’. In addition, unlike drugs, there is little need to spend ‘*much time obtaining*’ food within the modern Western environment.

The applicability of a substance-based framework to the assessment of addictive eating is further limited by neurobiological differences between the effects of drugs and food. For example, while brain opioid and dopaminergic networks have been implicated in the consumption and reward evaluation of both drugs and food (Colantuoni et al., 2001; Colantuoni et al., 2002; Drewnowski, Krahn, Demitrack, Nairn, & Gosnell, 1995; O’Malley et al., 2002; Volkow, Fowler, Wang, & Goldstein, 2002), it is important to consider that the dopaminergic response to drugs is approximately 10 times greater than to food (Volkow & Wise, 2005). Furthermore, the ‘reward centre’ of the brain, the nucleus accumbens, is differentially affected by drugs and food (Bassareo, Musio, & Di Chiara, 2011), and unlike drugs, the reward response to food diminishes with increased satiety or following repeated consumption of a similar taste (Avena et al., 2008; Epstein et al., 2009).

Finally, a substance-based account of food addiction necessitates the identification of a specific addictive agent contained in food. One suggestion is that highly processed foods, which contain large amounts of fat and/or sugar, may have addictive properties (Schulte et al., 2015). Indeed, rats that were given intermittent access to a high sugar or fat diet developed binge-eating tendencies, and opiate-like withdrawal symptoms (e.g. teeth chattering and tremors; Colantouni et al., 2001; Colantouni et al., 2002; Dimitriou et al., 2000). However, the extent to which animal models provide a valid reflection of eating in humans is limited by several caveats. In particular, animal models do not account for social and psychological influences of human eating, such as emotional eating, dietary restraint, and the stigmatisation that is associated with weight gain. Similarly, animal models which use intermittent dietary restriction, and provide access to single-nutrient diets (e.g. fat or sugar), are unrepresentative of the modern Western environment in which access to a wide-range of nutrients and energy dense foods is virtually unlimited.

The limited comparability between drugs and food poses a significant threat to the validity of the YFAS, which is largely dependent on a substance-based model of food addiction (Ziauddeen, Alonso-alonso, Hill, Kelley, & Khan, 2015). As such, several authors have suggested the need to develop a more precise operational definition of food addiction that is not reliant upon existing conceptualisations of substance-based addictions (Carter et al., 2016; Hebebrand et al., 2014; Ziauddeen et al., 2012). This may then be used to provide further validation of the food addiction concept, for example by establishing whether certain eating patterns resemble core features of addiction, such as habitual food-seeking in response to environmental food-cues (Ziauddeen et al., 2012).

In order to develop a novel framework for ‘food addiction’, one approach therefore is to focus on dimensions of observable behaviour which may underpin a behavioural addiction to eating (Hebebrand et al., 2014). Indeed, the view that ‘food addiction’ may be best conceptualised as a behavioural, rather than substance-based, ‘*eating* addiction’ represents the consensus opinion of a number of researchers in this area (e.g. NeuroFAST, 2013). This approach circumvents the assumption that certain foods contain specific ‘addictive’ substances, and yields important implications for the inclusion of ‘addictive eating’ within future editions of the DSM, which now provides a category for non-substance based addictions. While gambling is the only

behavioural addiction currently recognised within this category, there is scope for the inclusion of other deleterious behaviours. It is therefore necessary to identify exactly which behaviours and cognitions may underlie addictive patterns of eating, and to develop a method of assessing their severity.

One possibility is that addictive-like eating is characterised by increased reward responsivity and diminished inhibitory control. This would be consistent with dual-process theories which suggest that appetitive/reward systems compete with inhibitory systems for control over behaviour. Specifically, there is evidence to suggest that an increased reward response, combined with a diminished ability to control these responses, provides an underlying risk factor for the development of addictive behaviours (Wiers et al. 2007). Further, in a prospective study, Nederkoorn et al. (2010) reported greater weight gain, over a 1-year period, in those with an increased preference for snack foods *and* a lower capacity for inhibitory control, compared to those with higher inhibitory control. There is also evidence to suggest that a variety of eating behaviour trait questionnaires can be reduced to underlying dual-process components of food reward responsivity and inhibitory control (Price et al., 2015; Vainik et al., 2015), and these processes have been found to account for a significant proportion of variance in BMI (Price et al., 2015). Nonetheless, Vainik et al. (2015) found that measures of eating behaviour tap into similar underlying constructs but at *differing levels* of severity. Drawing upon this, and consistent with previous suggestions (Davis, 2013), Vainik et al. (2015) propose that addictive-like eating patterns represent a more *severe* stage of problematic eating beyond that captured by existing trait measures of eating behaviour.

The aim of the current research was therefore to develop a tool to quantify addictive-like eating behaviours. Previously, we used an inductive approach to identify behaviours that are commonly associated with food addiction amongst members of the lay public (Ruddock et al., 2015). These included: 1. A tendency to eat for reward rather than physiological need, 2. persistent food cravings, 3. an inability to control oneself around food, 4. a preoccupation with food and eating, 5. increased weight or an unhealthy diet, and 6. a particular problem controlling one's intake of foods high in fat, salt, and/or sugar. Using these commonly held beliefs about the manifestation of food addiction, the current study developed and provided preliminary validation for the Addictive Eating Behaviour Scale (AEBS).

Specifically, we conducted psychometric evaluation of the factor structure, and examined the scale's internal reliability. Finally, we explored the extent to which the AEBS could predict BMI and weight classification over and above that predicted by previously established measures of disinhibited and addictive eating.

5.4. Method

5.4.1. Participants

Participants ($N=513$) were recruited via public advertisements that were displayed on various social media websites (e.g. Facebook and Twitter) and on the University of Liverpool's intranet webpage. In exchange for taking part, participants were given the chance to enter a prize draw to win £50, and/or were allocated course credits. All participants who were over the age of 18 and fluent in English were eligible to take part. Prior to analysis, participants were randomly allocated into one of two groups (group 1 or group 2). Initial exploratory factor analysis and internal reliability analyses were performed using responses from group 1 ($n=308$). Responses from group 2 ($n=205$) were used to confirm the factor structure. Further analyses of the scale's convergent, divergent, and incremental validity were performed using responses from both groups. Ethical approval was obtained from the University of Liverpool Research Ethics Committee.

5.4.2. Measures

5.4.2.1. Addictive eating behaviour questionnaire.

The original pool of 62-items that were assessed for inclusion in the AEBS were derived from qualitative responses obtained from a previous study (Ruddock et al., 2015; Chapter 2 of the current thesis). To ensure that items adequately captured a range of addictive eating behaviours, we included at least 5 items for each 'theme' that was identified in the previous study. Specifically, items referred to either: 1. A tendency to eat for reward rather than physiological need (e.g. 'I continue to eat despite feeling full'), 2. persistent food cravings (e.g. 'I crave certain foods'), 3. an inability to control oneself around food (e.g. 'I find it difficult to limit what/how much I eat'), 4. a preoccupation with food and eating (e.g. 'I spend lots of time planning my meals'), 5. increased weight or an unhealthy diet (e.g. 'I have gained weight as a result of my overeating'), and 6. a particular problem controlling ones intake of foods high in fat, salt, and/or sugar (e.g. 'I have a particular problem

controlling myself around foods that are high in fat, sugar, and/or salt'). For each item, participants indicated the extent to which they *agreed* with the statement, or the frequency by which they engaged in the given behaviour. Responses were provided using 5-point Likert scales which ranged from 'Strongly Disagree' to 'Strongly Agree', or from 'Never' to 'Always'.

5.4.2.2. Assessments of convergent validity

The following scales were included to assess the convergent validity of the AEBS, and were therefore expected to correlate positively with the scale:

Yale Food Addiction Scale (YFAS; Gearhardt et al., 2009).

See section 3.4.2. for a description of this measure.

Binge eating scale (BES; Gormally, Black, Daston, & Rardin, 1982).

See section 3.4.2. for a description of this measure. The BES was selected on the basis that it has previously been used to assess the convergent validity of an existing measure of food addiction (i.e. the YFAS, Gearhardt et al., 2009).

Emotional Eating Scale (EES; Arnow, Kenardy, & Agras, 1995).

The EES consists of 25-items which assess tendencies to engage in emotionally-driven overeating. A list of 25 mood states is provided (e.g. lonely, bored), and respondents are required to indicate the extent to which each mood would initiate overeating (ranging from 'no desire' to 'an overwhelming desire'). This measure has previously been used to assess the convergent validity of the YFAS (Gearhardt et al., 2009).

Eating Troubles Module (EAT-26; Garner, Olmsted, Bohr, & Garfinkle, 1982).

The EAT-26 is based on the Eating Attitudes Test and provides a measure of the signs and symptoms associated with eating disorders. A high risk of disordered eating is indicated by scores equal to or greater than 20. This measure was selected on the basis that it has previously been used to assess the convergent validity of the YFAS, (Gearhardt et al., 2009).

Assessment of self-perceived 'food-addiction'.

As in Chapter 3, to assess self-perceived food addiction, participants were asked 'Do you agree with the following statement: "I believe myself to be a food addict"?'. Participants were required to tick either 'yes' or 'no'. Previously, we

found that individuals who perceived themselves to be addicted to food demonstrated increased food reward, and consumed more calories from a high-fat food, compared to those who did not perceive themselves as ‘food addicts’ (Ruddock et al., in press; Chapter 3).

5.4.2.3. Assessments of divergent validity

The following measures were included to assess the scale’s divergent validity, and thus were *not* expected to correlate with AEBS scores:

Rutgers Alcohol Problem Index (RAPI; White & Labouvie, 1989).

The RAPI consists of 23-items which assess drinking problems within young adults. This measure was selected on the basis that it has previously been used to assess the divergent validity of the YFAS (Gearhardt et al., 2009).

Behavioural Inhibition System/Behavioural Approach System Reactivity

(BIS/BAS; Carver & White, 1994).

The BIS/BAS questionnaire consists of 20-items which assess the Behavioural Inhibition (BIS) and Behavioural Approach Systems proposed by Gray (1987). The instrument provides a total BIS score and three BAS-related subscale scores: Drive (DRV), Fun Seeking (FS), and Reward Responsiveness (RR). This measure has previously been used to assess the divergent validity of the YFAS (Gearhardt et al., 2009).

5.4.3. Procedure

Groups 1 and 2 completed the questionnaires online at www.qualtrics.com. After providing informed consent, questionnaires were completed in the following order: AEBS, the assessment of self-perceived ‘food addiction’, BES, EAT-26, YFAS, EES, RAPI, and BIS/BAS. Participants then provided demographic information including their age, gender, weight (in kilograms, pounds, or stones), and height (in centimetres, inches, or feet). Finally, participants who wished to be entered into the prize draw provided their e-mail address. Participants were fully debriefed following the study.

5.4.4. Data analysis

5.4.4.1. Pre-analysis checks and data preparation

Prior to analysis, participants' responses on each of the AEBS items were assigned a value of 1 to 5 (1=Strongly disagree/Never, 2=Disagree/Rarely, 3=Neither agree or disagree/Sometimes, 4=Agree/Most of the time, 5=Strongly agree/Always). As higher scores indicated greater addictive eating tendencies, some items were reverse scored so that inter-correlations with other items remained positive. AEBS items were assessed for skewness and kurtosis, and sampling adequacy was checked using the Kaiser-Meyer-Olkin (KMO) statistic. Bartlett's test of sphericity was used to assess whether correlations between items were sufficiently large for principle components analysis (PCA) (i.e. significant values [$p < .05$] are indicative of large inter-item correlations).

5.4.4.2. Exploratory factor analysis (group 1)

A parallel analysis (using the Monte-Carlo simulation method, Glorfeld, 1995), and a scree-plot (Cattell 1966) were used to identify an initial factor solution. A Principle Components Analysis (PCA) with oblique rotation was then conducted, and items were removed if they had factor loadings of less than .50 (Nunnally & Bernstein, 1994), or had loadings of more than .35 on more than one factor (Kiffin-Petersen & Cordery, 2003). Items that had low item-total correlation ($< .40$; Gliem & Gliem, 2003) or did not share a conceptual meaning with the remaining items in a scale (O'Rourke & Hatcher, 2013) were also removed following reliability analysis (Cronbach's alpha).

5.4.4.3. Internal consistency and descriptives (groups 1 and 2).

Cronbach's alpha was used to assess the internal consistency of each AEBS subscale. Nunnally and Bernstein (1994) suggests $\alpha = .70$ as a lower acceptable bound for alpha. AEBS total and subscale scores were computed by summing values (i.e. 1 to 5) that corresponded to participants' responses to each item. Independent t-tests assessed whether AEBS total or subscale scores differed between males or females, and Pearson's correlations were used to examine whether scores were associated with age and BMI. All analyses were conducted for groups 1 and 2 separately.

5.4.4.4. Confirmatory factor analysis (group 2).

Using AMOS 22 (Arbuckle, 2013), confirmatory Factor Analysis was performed on the solution with best fit. Items were free to load onto their corresponding latent factors, and latent factors were free to correlate with each other. Model fit was assessed by examining the Normed X^2 statistic (X^2/df) (Ullman, 2001), Goodness of Fit Index (GFI; Bentler, 1990), Comparative Fit Index (Kelloway, 1998), the Root Mean Square Error of Approximation (RMSEA; MacCallum et al., 1996), and Standardized Root Mean Square Residual (SRMR; Hu & Bentler, 1999). Normed X^2/df ratios of less than 2 (Ullman, 2001), and GFI and CFI, values of above .90 (Bentler, 1990; Kelloway, 1998), are deemed acceptable. RMSEA values indicate either good fit (<0.05), fair fit ($0.05 < 0.08$), mediocre fit ($0.08 < 0.10$), or poor fit (>0.10) (MacCallum et al. 1996), and SRMR values of less than .08 are considered good fit (Hu & Bentler, 1999). Where appropriate, model fit was improved by adding covariance pathways between error terms. These were determined following inspection of the modification indices.

5.4.4.5. Convergent and Divergent validity (groups 1 and 2).

Correlational analyses were conducted to assess the convergent validity of the AEBS compared to other eating behaviour scales (i.e. YFAS, EES, BES, EAT-26) and BMI. A logistic regression was used to determine the extent to which AEBS scores could predict whether or not respondents perceived themselves to be food addicts. Divergent validity was assessed by comparing correlations between the AEBS total score and problematic alcohol use (assessed using the RAPI), and behavioural inhibition/activation (BIS/BAS).

5.4.4.6. Incremental validity (groups 1 and 2).

A hierarchical linear regression was conducted to assess whether the AEBS could account for additional variance in BMI beyond that predicted by the YFAS symptom count and BES. A hierarchical logistic regression was also conducted to explore whether the AEBS could predict self-perceived food addiction over and above YFAS symptom count and BES scores. In both models, YFAS symptom count and BES scores were included in step 1, while total AEBS scores were entered into step 2. Finally, an ordinal regression was conducted to evaluate the scale's

ability to predict weight classification. Participants were grouped as either underweight (BMI<.18.49), normal weight (18.50<BMI<24.99), overweight (25.00<BMI<29.99), or obese (30<BMI). Weight classification was entered as the dependent variable (with ‘underweight’ as the reference category), and BES, YFAS symptom count, and AEBS scores were entered as co-variates.

5.5. Results

5.5.1. Pre-analysis checks and participant characteristics

Values of skewness and kurtosis ranged between the acceptable levels of -2 and 2, thus no transformations were necessary (Lewis-Beck et al., 2004). The Kaiser–Meyer–Olkin statistic for the model was above the acceptable level of .05 (KMO=.91) and Bartlett’s test of sphericity was significant ($p<.001$). Participant characteristics for each of the two groups are shown in Table 5.1.

Table 5.1. *Characteristics of participants in each group.*

	Group 1 ($n=308$)	Group 2($n=205$)
Females/males(no.)	271/37	171/34
Age(yrs): mean \pm SD*	24.3 \pm 10.7	24.0 \pm 11.2
Age(yrs): range	18-67	17-66
BMI (kg/m ²): mean \pm SD	23.6 \pm 5.1	23.3 \pm 5.1
BMI (kg/m ²): range	13.9-53.1	15.2-60.3
Overweight/obese(no.)	45/30	29/17

*SD = Standard deviation

5.5.2. Exploratory Factor Analysis (group 1)

The parallel analysis and scree-plot initially identified a five-factor solution. However, subsequent Principle Components Analysis (PCA) with oblique rotation revealed no clear 5-factor solution. Following removal of items (using the procedure outlined in the data analysis section), a two-factor solution was derived from the remaining 16 items, with eigenvalues 7.00 and 2.04 for factors one and two, respectively. Factor one comprised of 10 items that referred to overeating and weight gain (e.g. I eat continue to eat despite feeling full), and accounted for 43.77%. Factor 2 comprised of 6 items that referred to unhealthy eating practices and perceived low

self-control around food (e.g. Despite trying to eat healthily, I end up eating 'naughty' foods) and accounted for 12.76%, of the total variance. Factors 1 and 2 were moderately positively correlated with each other ($r = .515, p < .001$). Item-factor loadings are provided in Table 5.2. The full AEBS and scoring instructions are provided in Appendix A.

Table 5.2. *Factors, items, and factor loadings*

Factor	Factor items	Factor loadings
Overeating/weight gain	I continue to eat despite feeling full	.812
	I serve myself overly large portions	.804
	I find it difficult to limit what/how much I eat	.797
	I have gained weight as a result of my overeating	.764
	Once I start eating certain foods, I can't stop until there's nothing left	.748
	When it comes to food, I tend to overindulge.	.721
	I don't tend to overeat*	.717
	I feel unable to control my weight	.667
	I binge eat	.614
	I eat until I feel sick	.604
Unhealthy eating/low self-control	I tend not to buy processed foods that are high in fat and/or sugar*	.851
	I don't eat a lot of high fat/sugar foods*	.850
	I believe I have a healthy diet*	.810
	I am easily able to make healthy food choices*	.756
	Despite trying to eat healthily, I end up eating 'naughty' foods	.647
	I continue to eat certain unhealthy foods despite being aware of its effect on my health	.620

Note. Items were reverse scored prior to analyses.

5.5.3. Internal consistency and descriptives (group 1)

Mean AEBS and subscale scores for group 1 are shown in Table 5.3. There were no differences between males and females on either subscale or on AEBS total scores ($ps > .246$), and age did not correlate with scores on the overeating subscale ($r = -.023, p = .687$) or with the AEBS total score ($r = -.109, p = .057$). However, there was a small but significant negative correlation between age and scores on the unhealthy eating/low self-control subscale ($r = -.218, p < .001$). Cronbach's alpha revealed high internal consistency for overeating/weight gain ($\alpha = .902$) and unhealthy eating/low self-control scales ($\alpha = .847$).

5.5.4. Internal consistency and descriptives (group 2)

Mean AEBS scores for group 2 are displayed in Table 5.3. AEBS total and subscale scores did not differ between groups 1 and 2 ($ps > .481$). There was no effect of gender on AEBS subscale or total scores in group 2 ($ps > .512$). Age was negatively associated with scores on the overeating/weight gain subscale ($r = -.150$, $p = .032$), unhealthy eating/low self-control subscale ($r = -.227$, $p = .001$), and total AEBS scores ($r = -.204$, $p = .003$). As in group 1, reliability estimates revealed high internal consistency for overeating/weight gain ($\alpha = .853$) and unhealthy eating/low self-control subscales ($\alpha = .822$).

Table 5.3. AEBS total and subscale scores for each of the three groups. Values are means \pm standard deviations.

	Group 1	Group 2
AEBS total ¹	44.3 \pm 10.6	43.8 \pm 9.6
AEBS (overeating/weight gain) ²	26.4 \pm 7.6	26.0 \pm 6.5
AEBS (unhealthy eating/low self-control) ³	17.9 \pm 4.5	17.9 \pm 4.4

¹The minimum possible score for AEBS total was 16, and the maximum was 80.

²The minimum possible score for the overeating/weight gain subscale was 10 and the maximum was 50

³The minimum possible score for the unhealthy eating/low self-control subscale was 6 and the maximum was 30.

5.5.5. Confirmatory factor analysis (group 2)

Ten items were free to load onto the latent factor overeating/weight gain, and 6 items were free to load onto the latent factor unhealthy eating/low self-control. The initial iteration indicated a poor fit model [Normed X^2 (X^2/df) = 2.28, GFI = .870, RMSEA (90% CI) = .079 (.066 – .093), CFI = .893, SRMR = .067]. However, following the addition of covariance pathways (see Figure 5.1) the two-factor model provided a good fit to the data [Normed X^2 (X^2/df) = 1.68, GFI = .909, RMSEA (90% CI) = .058 (.042 – .073), CFI = .945, SRMR = .0593]. Standardized regression weights indicated that all items appropriately reflected their underlying latent variable ($ps < .001$) (Figure 5.1).

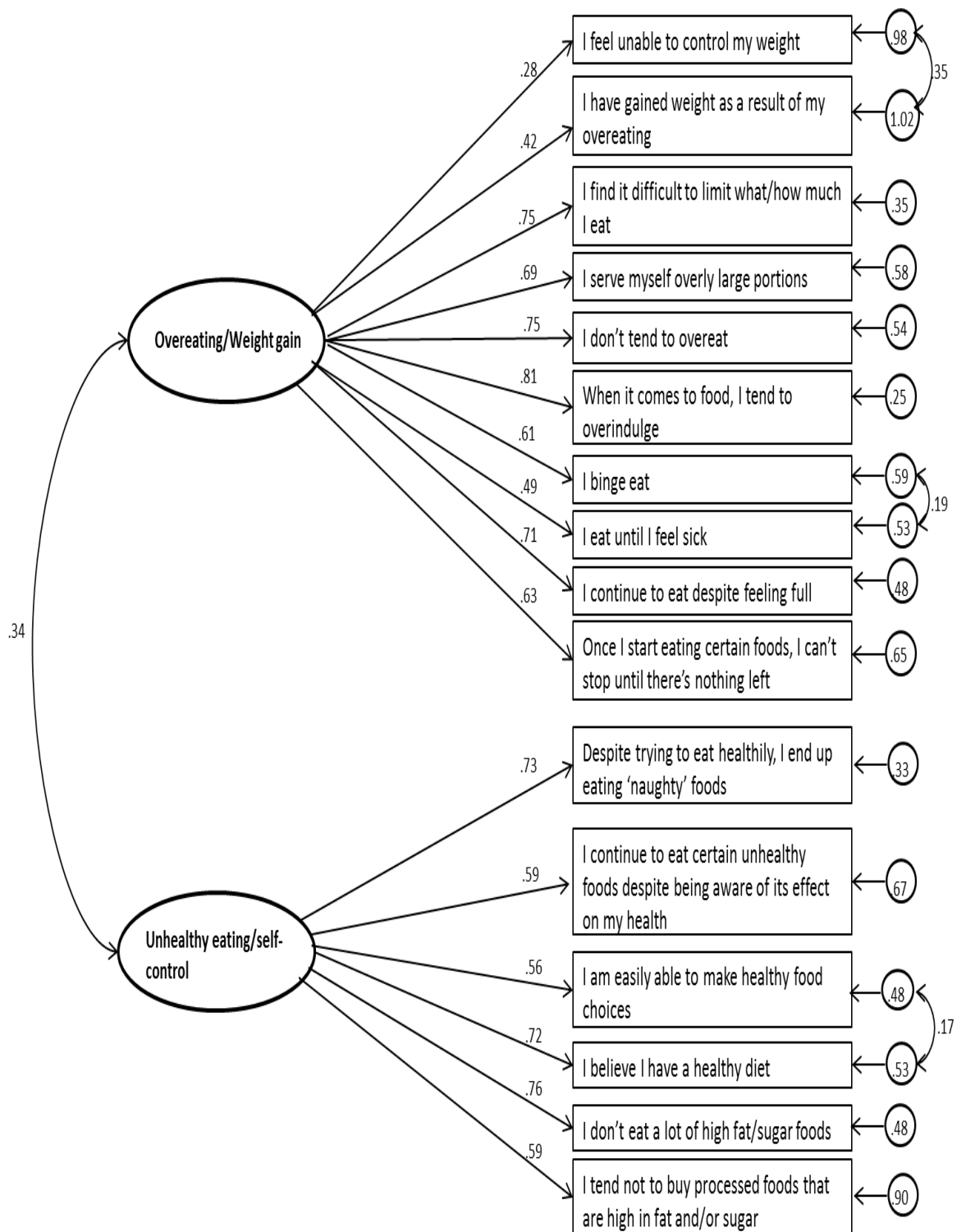


Figure 5.1. Factor model of AEBS with standardized factor loadings (i.e. values corresponding to one-way arrows), error terms (circled values), and covariances (values corresponding to two-way arrows).

5.5.6. Convergent and Divergent validity (groups 1 and 2)

The AEBS total score correlated positively with all but the EAT-26 scale (Table 5.4), indicating good convergent validity. Furthermore, AEBS scores successfully predicted whether or not respondents perceived themselves to be food addicts, $B=.12$, $SE=.01$, $\text{Exp}(B)=1.12$, $p<.001$. Total AEBS scores did not correlate with scores on the BAS scale, indicative of good divergent validity. However small but significant correlations were observed between AEBS scores and the RAPI and Behavioural Inhibition Scale (BIS) (Table 5.4).

Table 5.4. Descriptive statistics and correlations with AEBS ($n = 513$)

Variable	M \pm SD	Cronbach's α	Correlation with AEBS	p
Binge eating scale	10.8 \pm 8.0	.91	.69	<.001
YFAS(symptoms)*	2.08 \pm 1.51	.90	.56	<.001
EES	52.8 \pm 18.1	.94	.48	<.001
EAT-26	8.3 \pm 8.0	.89	.06	.164
BMI (kg/m ²)	23.4 \pm 5.2		.29	<.001
RAPI	7.6 \pm 9.5	.92	.22	<.001
BIS	13.7 \pm 3.2	.79	-.18	<.001
BAS	27.4 \pm 5.1	.84	-.05	.236

*46 (9%) participants from groups 1 and 2 fulfilled the YFAS criteria for food addiction

5.5.7. Incremental validity (groups 1 and 2)

After controlling for the variance accounted for by YFAS symptom count and BES scores, AEBS scores explained a significant proportion of variance in BMI (Table 5.5). AEBS and BES scores independently and significantly predicted BMI⁴.

Table 5.5 Hierarchical multiple regression showing the YFAS and BES symptom count (step 1) and AEBS (step 2) as predictors of BMI.

	Cumulative		Simultaneous			
	F-change	R ² -change	β	SR ²	p	95% Confidence interval
<i>Step 1</i>	<i>F</i> (2,502)=24.02**	.09				
YFAS(symptoms)			-.08	-.11	.201	-.64-.14
BES			.34**	.06	<.001	.14-.29
<i>Step 2</i>	<i>F</i> (1,501)=11.08*	.02				
AEBS			.20**	.02	<.001	.04-.16

Note. SR² is the squared semi-partial correlation. *p<.05 **p<.001. Variance accounted for by the full regression model: R²=.11, F(3,501)=20.02, p<.001.

For the ordinal regression analysis, the test of parallel lines confirmed that the assumption of proportionality had been met ($p=.244$). Results revealed that the likelihood of being overweight or obese increased with AEBS scores⁵, independent of BES and YFAS scores (logit regression coefficient=.05, 95% confidence intervals (CI)=.02, .07, Wald X²= 11.61, $df=1$, $p=.001$). The odds ratio indicated that the chances of an individual being classified as overweight or obese increased by 1.05 for every unit increase in AEBS scores. Weight classification was also significantly predicted by BES scores (logit regression coefficient=.04, 95% CI=.01, .08, Wald X²= 5.22, $df=1$, $p=.022$), but not by YFAS symptom count (logit regression coefficient=-.14, 95% CI=-.32, .03, Wald X²= 2.66, $df=1$, $p=.103$).

⁴ All Tolerance and VIF values were within the commonly accepted cut off criteria (i.e. tolerance >.20; VIF < 4.0), indicating no problems with multi-collinearity (O'Brien, 2007).

⁵ AEBS scores did not distinguish between underweight and normal weight participants (logit regression coefficient=-.53, 95%CI=-1.49, .42, Wald X²=1.19, $df=1$, $p=.275$).

5.6. Discussion

Existing measures of addictive eating such as the YFAS and YFAS 2.0 (Gearhardt et al., 2009, 2016) rely upon the clinical criteria for substance dependence to assess and diagnose food addiction. However, given the limited comparability of drugs and food, such measures may fail to account for many individuals with problematic eating behaviours. To address this, we developed and validated a novel tool, the Addictive Eating Behaviour Scale (AEBS), to assess the presence of behaviours which may underpin addictive-like patterns of eating.

The AEBS comprised a two-factor scale structure which was corroborated by a confirmatory factor analysis. Items in factor 1 referred to a tendency to overeat and gain weight, while items in factor 2 referred to a lack of self-control around food, and an unhealthy diet. Both subscales demonstrated good internal consistency. Mean scores on each subscale did not differ between males and females, however older age was associated with lower scores on the unhealthy eating/low self-control sub-scale in both groups 1 and 2.

The two-factor structure of the AEBS is consistent with dual-process accounts of overeating and weight gain (Appelhans, 2009). Specifically, an enhanced reward responsivity is reflected by the ‘Overeating/weight-gain’ subscale, while the ‘low-self-control/unhealthy diet’ is reflective of diminished inhibitory control. Furthermore, the additional variance in BMI that was captured by the AEBS beyond an existing measure of disinhibited eating (i.e. the Binge Eating Scale), supports the idea that addictive-like eating represents a more severe stage of ‘uncontrolled eating’ (Davis, 2013; Vainik et al., 2015).

Indicative of good convergent validity, total AEBS scores correlated positively with the Emotional Eating Scale, Binge Eating Scale, YFAS symptom count, and BMI, and significantly predicted whether or not individuals perceived themselves as ‘food addicts’. However, the scale failed to converge with a measure of disordered eating (i.e. EAT-26). This is perhaps reflective of fundamental differences between the characteristics of traditional eating disorders (i.e. anorexia nervosa, bulimia nervosa), and addictive eating patterns. Indeed, in our previous qualitative research (Ruddock et al., 2015; Chapter 2), participants did not believe that food addiction was associated with weight and shape concern, periods of

excessive food restriction, or the tendency to engage in compensatory behaviours (e.g. purging).

Crucially, the AEBS was able to account for a significant proportion of variance in BMI above that accounted for by the BES and YFAS. This is important as both of these measures assess patterns of eating that are thought to reflect a ‘food addiction’ (Davis & Carter, 2009; Gearhardt et al., 2009). Future research is now required to establish the clinical utility of the AEBS over and above the newly revised YFAS 2.0 (Gearhardt et al., 2016), which assesses food addiction based upon the DSM-V criteria for substance-related and addictive disorders. However, given that BMI was similarly associated with both original and revised versions of the YFAS (Gearhardt et al., 2016), it is possible that the AEBS would demonstrate comparable incremental validity over the YFAS 2.0. Furthermore, given that the YFAS 2.0 was developed using the DSM-V criteria for substance-related and abuse disorders, it encompasses similar limitations with regards to its applicability to eating behaviour.

However, given the high correlation between AEBS scores and a measure of binge eating (BES), it is necessary to consider the extent to which manifestations of addictive eating, captured by the AEBS, are distinct from more general patterns of disinhibited or ‘binge’ eating. Importantly, AEBS scores were able to significantly predict BMI over and above measures of disinhibited and addictive eating (i.e. BES and YFAS symptom count), and BES and AEBS were significant independent predictors of BMI. One imperative difference between the manifestations of binge eating and addictive eating patterns may concern the timeframe in which overeating occurs. According to the DSM-V criteria, binge eating disorder is characterised by a tendency to consume a large amount of food within a short space of time. In contrast, it has recently been suggested that food addiction may involve a more general tendency to overeat, or consume unhealthy foods, throughout the day (e.g. Hebebrand et al., 2014). Indeed, increased snacking has been associated with eating pathology and poorer weight-loss outcomes following bariatric surgery (Nicalau et al., 2015; Robinson et al., 2014; Sheets et al., 2015). Furthermore, Ruddock et al (2015) (Chapter 2 in the current thesis) found that conceptualisations of food addiction, amongst members of the lay public, do not necessarily implicate the secretive and planned ‘binge’ episodes, and subsequent caloric restriction, that

characterise binge eating disorder (Palmberg et al., 2014; Phillips, Kelly-weeder, & Farrell, 2016; Woods, Racine, & Klump, 2010).

An important distinction between the AEBS and previous measures of addictive eating (i.e. YFAS and YFAS 2.0), is that the AEBS does not provide a dichotomous diagnostic criterion for eating addiction. As Ziauddeen et al. (2012) discuss, the limited consensus and understanding regarding exactly which behaviours (and their frequency/intensity) warrant a diagnosis of ‘eating addiction’, currently precludes the development of a diagnostic criteria. In addition, although psychometrics tools offer the opportunity for screening and perhaps preliminary assessments, we agree with suggestions that the diagnosis of any psychological disorder should be reserved for trained clinicians, rather than self-report questionnaires (Long et al., 2015). As such, we suggest that continuous, rather than dichotomous or diagnostic, measures provide the most appropriate self-report assessment of addictive eating tendencies. Further exploration of the characteristics of addictive eating behaviours is required to provide a diagnostic criterion that may be used within clinical settings. In particular, the scale may be used to diagnose addictive-like eating in individuals with obesity and pathological patterns of eating.

The divergent validity of the AEBS was assessed by comparing its relationship to other distinct constructs. Specifically, we included the behavioural activation and inhibition scales (BIS/BAS, Carver & White, 1994), which measure sensitivity to reward and punishment, respectively, and Rutger’s Alcohol Problem Index (RAPI, White & Labouvie, 1989), which assesses problematic drinking behaviour. The AEBS was unrelated to scores on the BAS, however the AEBS correlated negatively with the BIS, and positively with the RAPI. Similar negative associations between the BIS and eating behaviour have been reported previously (Dietrich et al., 2014), and perhaps reflect a diminished sensitivity to the longer-term negative effects of overeating, such as weight gain and poor health. Furthermore, the positive correlation between the AEBS and RAPI may be explained by common personality attributes (e.g. impulsivity) that have been associated with alcoholism, obesity, and eating disorders (e.g. Fischer, Smith, & Anderson, 2003; Fischer & Smith, 2008; Slane, Burt, & Klump, 2012).

The current study has several limitations that should be addressed in future research. Firstly, while we attempted to recruit a representative community sample, respondents were predominantly female. Given that males and females may differ with regards to their conceptualisation of food addiction (Ruddock et al., 2015; Chapter 2), further validation of the scale is required within a male population. Secondly, while 18 percent of the sample were overweight or obese, the majority were of a healthy-weight. It is therefore possible that the characteristics of addictive-like eating identified in the AEBS, may differ to those extant in overweight or clinical samples. Thirdly, the current study used a cross-sectional design, and thus we were unable to draw conclusions about the *causal* relationship between AEBS scores and BMI. Therefore, the extent to which the scale is predictive of prospective weight gain and weight loss success are important avenues for future research.

Despite these limitations, the AEBS represents a valid and reliable tool to assess addictive-like eating behaviours in community samples. Through using an inductive approach and focusing on dimensions of observable behaviour, the scale overcomes many of the limitations of applying a substance-based framework to the assessment of eating behaviour. In particular, while there is on-going debate regarding whether food-addiction is best conceptualised as a substance-based (Schulte, Potenza, & Gearhardt, 2016) or behavioural (Hebebrand et al., 2014) addiction, the AEBS focuses on identifying core behaviours without any prior assumptions about the addictive qualities of certain foods.

Future research is required to validate the AEBS within obese and clinical populations, and establish clinically meaningful cut-off points for the scale. Furthermore, to provide validation of the 'eating addiction' concept, future research should establish the extent to which the AEBS captures core features of addiction, such as habitual food seeking in the presence of environmental food-cues (Ziauddeen et al., 2012). In doing so, the AEBS has important implications for the identification, prevention, and treatment of those at risk of weight gain and obesity.

Chapter 6: The effects of believing in food addiction on eating behaviour

6.1. Overview

To date, there has been much research and debate surrounding the validity and definition of the food addiction concept (e.g. Davis et al., 2011; Ziauddeen & Fletcher, 2013) and this issue has been examined in the thesis thus far. However, as Carter et al. (2016) point out, it is also imperative to consider the potential impact that food addiction messages and beliefs may have on eating behaviour and this is in line with the secondary aim of this thesis. To address this, Chapter 6 presents two studies which explored whether perceiving oneself to be a food addict would influence subsequent eating behaviour. Specifically, the studies examined whether personal food addiction beliefs would cause people to consume more or less food in a subsequent *ad libitum* taste task.

The studies reported in this chapter have been published as:

Ruddock, H.K., Christiansen, P., Jones, A., Robinson, E., Field, M., & Hardman CA. (2015). Believing in food addiction: Helpful or counter-productive for eating behaviour? *Obesity*, 24(6), 1238–1243.

6.2. Abstract

Obesity is often attributed to an addiction to food and many people believe themselves to be “food addicts”. However little is known about how such beliefs may affect dietary control and weight management. The current research examined the impact of experimentally manipulating participants’ personal food addiction beliefs on eating behaviour. In two studies, female participants (Study 1: $N=64$; Study 2: $N=90$) completed food-related computerized tasks and were given bogus feedback on their performance which indicated that they had high-, low- or average- food addiction tendencies. Food intake was then assessed in an *ad libitum* taste test. Dietary concern, and time-taken to complete the taste test, were also recorded in Study 2. In Study 1, participants in the high-addiction condition consumed fewer calories than those in the low-addiction condition, $F(1,60)=7.61$, $p=.008$, $\eta_p^2=0.11$. Study 2 replicated and extended this finding by showing that the effect of the high-addiction condition on food intake was mediated by increased dietary concern, which

reduced the amount of time participants willingly spent exposed to the foods during the taste test, $b = -.06(.03)$, 95% confidence interval (CI) = $-.13, -.01$. Believing oneself to be a food addict is associated with short-term dietary restriction. The longer-term effects on weight management now warrant attention.

6.3. Introduction

Obesity continues to increase with more than half of adults worldwide now overweight or obese (World Health Organization, 2016). Over-eating and obesity are frequently attributed to a food-based addiction though this notion has been the source of considerable controversy within the scientific community (Schulte, Avena, & Gearhardt, 2015; Ziauddeen & Fletcher, 2012; Ziauddeen, Farooqi, & Fletcher, 2013). However, scientific understanding has not kept pace with the lay public's enthusiasm for the concept of "food addiction" (Barry et al., 2009; Bird et al., 2008; Lee et al., 2013; Wilson et al., 2009). Indeed, in a recent study, almost three quarters of participants believed that obesity is caused by an addiction to certain foods (Lee et al., 2013). Furthermore, as many as 50% of people believe themselves to be food addicts (Hardman et al., 2015; Ruddock et al., 2015 (i.e. Chapter 2 of current thesis). To date, little is known about the potential impact of believing oneself to be a food addict on eating behaviour.

An addiction-based explanation might imply that excessive eating is outside of personal control and, in this way, may help to remove individual responsibility for over-consumption (Rogers & Smit, 2000). However, there may be counter-productive effects on eating behaviour. It is well-established that feeling in control of one's behaviour is important for health and predicts engagement in a variety of health-promoting dietary behaviours (Folkman, 1984; Wallston, 1992; Steptoe & Wardle, 2001). Conversely, public health messages which imply a lack of personal control over behaviour (e.g., "obesity is a disease") have been associated with unhealthy food choices and greater food intake (Dar-Nimrod et al., 2014; Hoyt, Burnett, & Auster-Gussman, 2014). For example, participants who had been led to believe that obesity is caused by a genetic susceptibility, consumed more calories during a follow-up task, relative to those who were told that obesity is caused by psychosocial factors or who received no information (Dar-Nimrod et al., 2014).

Similarly, Hoyt et al. (2014) found that, compared to an information-based article, obese participants who read an ‘obesity is a disease’ message, reported less concern for weight and body dissatisfaction. In turn, this led to the selection of higher calorie foods.

An opposing idea is that food addiction may be *helpful* for the initiation of healthy dietary behaviours. Notably, members of Overeaters Anonymous reported an increased sense of responsibility after acknowledging their “addiction” to food (Ronel & Libman, 2003; Russell-Mayhew, von Ranson, & Masson, 2010). Furthermore, diminished self-control beliefs may lead people to avoid putting themselves in tempting situations in the first place. In one study, smokers who were told that they had a low capacity for self-control subsequently exposed themselves to fewer tempting smoking scenarios, and were thus less likely to smoke, than participants who were told they had a high capacity for self-control (Nordgren, van Harreveld, & van der Pligt, 2009). Consistently, participants who were told that they had low self-control consumed less alcohol in a subsequent ‘taste test’ than those who believed that they had high self-control (Jones et al., 2012).

To test these possibilities, the current research aimed to experimentally manipulate participants’ personal beliefs about food addiction – that is, the extent to which they believed themselves to be food addicts. In a previous study, beliefs about the existence of food addiction (*e.g.*, “Food addiction is real”) were found to be malleable though there was no clear effect of this belief manipulation on food consumption (Hardman et al., 2015). However, in the current study, we reasoned that leading people to believe that they are personally affected by food addiction would be more likely to influence subsequent eating behaviour. This is supported by evidence that personalized feedback is highly effective at invoking dietary behaviour change (Celis-Morales, Lara, & Mathers, 2015). In a two-tailed hypothesis, we predicted that believing oneself to be a food addict would *either* promote over-consumption due to reduced personal responsibility for eating *or* cause a person to be concerned about their eating behaviour and consume less snack food.

6.4. Study 1 Method

6.4.1. Overview of the study method

Figure 6.1. presents an overview of the study procedure. In a between-subjects design, participants were randomly allocated to either the high-addiction or low-addiction condition. To manipulate food addiction beliefs, participants completed the Implicit Association Task (IAT) and Stop-Signal Tasks (SST), which they were told would assess their level of food addiction. Those in the high- and low- addiction conditions were then led to believe that they had scored high and low on food addiction tendencies, respectively. Participants then completed a leading questionnaire, to further manipulate food addiction beliefs, followed by the manipulation check. In keeping with the cover story, participants then completed several mood ratings. Food intake was then covertly assessed in an ostensible ‘taste and rate’ task in which participants could eat as much of two high-fat foods as they wished. Finally, participants completed several trait measures of eating behaviour, and height and weight were assessed.

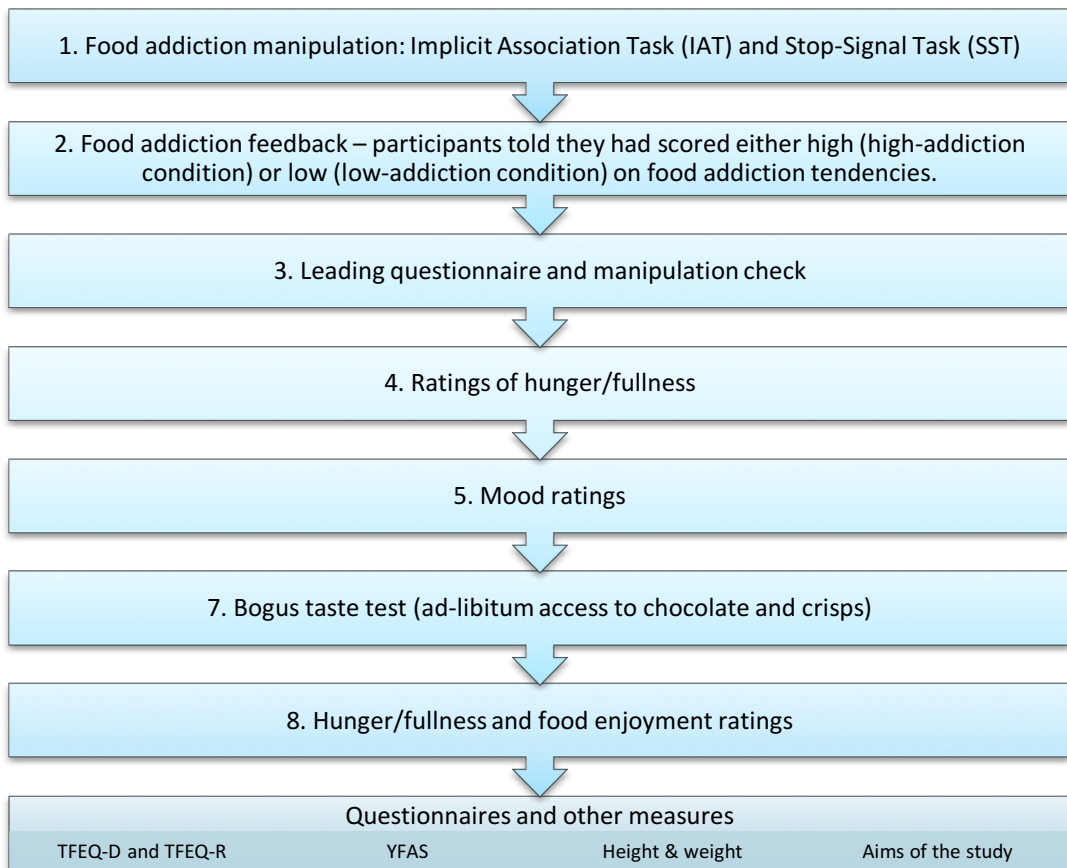


Figure 6.1. Flow-chart of the Study 1 procedure

6.4.2. Participants

Female participants ($N=64$) were recruited from the University of Liverpool via poster and online advertisements. Participants were informed that the aim of the study was to investigate the effect of mood on taste preferences. In a between-subjects design, participants were randomly allocated to either high-addiction or low-addiction conditions. Inclusion criteria required that participants were non-smokers, had no food allergies or intolerances, had never been diagnosed with an eating disorder, and were not on any medication known to affect appetite. All participants completed a medical history questionnaire prior to testing to ensure that they did not suffer from any food allergies. Participants were asked not to eat or consume any calorie-containing drinks for 3 hours before the study. All participants complied with this instruction. Ethical approval was granted by the Institute of Psychology, Health and Society at the University of Liverpool. Participants received course credits or were reimbursed with a £5 shopping voucher as compensation for their time and travel expenses.

6.4.3. Measures and materials

6.4.3.1. Food addiction beliefs manipulation

Implicit Association Task (IAT; Greenwald, McGhee, & Schwartz, 1998).

This task was programmed using Inquisit 2.0 (Millisecond Software, 2002). Participants were required to respond quickly to positive and negative words, high- and low-calorie food pictures, and neutral pictures. The task consisted of seven blocks. In the first block, participants were required to categorise words as either 'positive' or 'negative' using the computer keypad. In block 2, participants were required to categorise pictures as either 'food' or 'non-food'. In subsequent blocks, categories were combined. For example, participants were required to press the left key when they were presented with either positive words or food pictures, and the right key when presented with negative words or non-food pictures. In other blocks, these category combinations were reversed such that participants were required to press the left key in response to negative words and food pictures, and the right key when presented with positive words and non-food pictures. Blocks that presented single stimuli (i.e. either pictures or words) consisted of 8 trials, while blocks that presented combined stimuli (i.e. both pictures and words) consisted of 32 trials. Response keys were counterbalanced across blocks. A red cross was displayed if an

incorrect response was given. This task has previously been used to assess associations between positively and negatively valenced words and food and neutral stimuli (Kemps et al., 2013). The task took approximately 10 minutes to complete. At the end of the task, a bogus score was presented on the computer screen regardless of the participant's actual performance on the task. For those in the low-addiction condition, the IAT score was positive (142ms). For those in the high-addiction condition, the IAT score was negative (-169ms). To ensure that there were no pre-existing group differences on the IAT, participants' reaction times were recorded. These were used to calculate latency difference scores by subtracting response times to congruent trials (i.e. food and positive stimuli pairings) from response times to incongruent trials (i.e. food and negative stimuli pairings). Consistent with Palfai and Ostafin (2003), response times faster than 300ms, or slower than 3000ms, were considered outliers and excluded from analyses. Incorrect responses were also excluded.

Stop-Signal Task (SST; Logan & Cowan, 1984).

The stop-signal task was programmed in Inquisit 2.0 (Millisecond Software, 2002). Participants were required to respond to high- and low-calorie food pictures and neutral pictures but to inhibit their response when they heard an auditory 'stop' signal. The stop signal was presented on 50% of trials. The task consisted of 96 trials: 48 food pictures and 48 non-food pictures. Trials were terminated by either button press or by response times exceeding 2000ms. Feedback was displayed after each trial. The task took approximately 10 minutes to complete. Bogus feedback was then provided in which those in the low-addiction condition were provided with a higher score (76.6) than those in the high-addiction condition (39.8). In order to ensure that there were no pre-existing group differences on this task, participants' performance (i.e. the number of commission errors) on the SST was recorded.

Leading Questionnaire.

Participants completed one of two leading questionnaires which were designed to further enforce the manipulation. The questionnaires consisted of 5 leading questions with response options 'Agree' or 'Disagree'. For those in the 'high addiction' condition, the questionnaire was designed such that participants would tick 'Agree' to statements consistent with disinhibited eating (e.g. 'I sometimes eat

more of a food simply because it tastes nice, even when I'm full'). In the low-addiction condition, the questionnaire prompted participants to tick 'Agree' to statements consistent with good self-control around food (e.g. 'I usually feel in control of what and how much I eat'). The use of leading questionnaires as a means of changing beliefs has previously been demonstrated (Swann et al., 1988).

Manipulation check.

The success of the manipulation was assessed using the statement 'I believe myself to be a food addict' with five response options ranging from 'Strongly disagree' to 'Strongly agree'. Participants were also asked to tick 'agree' or 'disagree' to the statement 'I believe that some people are addicted to food'. This was included to ensure that participants believed in the concept of food addiction and thus would be susceptible to the manipulation.

6.4.3.2. Outcome measure: Ad libitum taste task

For the taste task, participants were provided with a 50g bowl of crisps (Tesco Ready salted crisps: 454kcal/100g, 33.2g fat/100g) and a 100g bowl of chocolate (Cadbury Dairy Milk Giant Buttons: 530kcal/100g, 30g fat/100g). Prior to tasting the foods, participants completed the following ratings using visual analogue scales: (1.) How much do you expect to like this food? (2.) How strong is your desire-to-eat this food right now? (3.) How strong is your craving for this food right now? (4.) How difficult is it to resist this food right now? Taste ratings were provided on a variety of visual analogue scales (e.g. salty, sweet). All scales were anchored with 'Not at all' on the left and 'Extremely' on the right. The full list of taste ratings can be found in Appendix B. To assess food intake (i.e. the primary outcome measure), bowls were covertly weighed before and after participants completed the taste task.

6.4.3.3. Additional measures and trait eating questionnaires

The Yale Food Addiction Scale (YFAS; Gearhardt et al., 2009) and Three Factor Eating Questionnaire (TFEQ, Stunkard & Messick, 1985) were used to provide descriptive information about the sample (see Chapter 3, section 3.4.2. for a full description of these measures).

Hunger/fullness ratings.

Levels of hunger and fullness were assessed using Visual Analogue Scales (VAS). Scales were 100mm in length with end anchor points ‘Not at all’ on the left and ‘Extremely’ on the right.

Mood ratings.

A variety of mood ratings (e.g. Tense, Alert) were provided for consistency with the cover story. Ratings were provided on visual analogue scales which were 100mm in length with end anchor points ‘Not at all’ on the left and ‘Extremely’ on the right. The full list of mood ratings is provided in Appendix B.

6.4.4. Procedure

All sessions were conducted between 12pm and 5pm and took approximately 1 hour to complete. Upon arrival, participants were randomly allocated to either the high- or low- addiction condition. Participants provided written informed consent and completed a medical history questionnaire to ensure the absence of any food allergies. To ensure compliance with the study procedure, participants were asked to confirm that they had not eaten for at least 3 hours prior to the study. Participants then completed the IAT and stop-signal tasks which they were led to believe would assess their addictive tendencies towards food. The experimenter noted down the bogus scores for each of the tasks and explained that these scores reflected a high (for those in the ‘high addiction’ group) or low (for those in the low-addiction group) tendency for food addiction. To further enforce the believability of the manipulation, participants were shown a bogus histogram which illustrated the distribution of food addiction scores within the general population. Those in the low-addiction condition were informed that they scored within the top-quartile of the distribution, while those in the high-addiction condition were told that they scored within the bottom-quartile. This procedure has previously been used to successfully manipulate beliefs about alcohol self-control (Jones et al., 2012). Participants then completed the leading questionnaire and manipulation check, followed by hunger and fullness ratings, and mood ratings. Participants then completed the bogus taste test, in which they were invited to eat as much of the food as they wished. Participants could end the taste task whenever they wanted.

Following the taste task, participants were asked to indicate, using visual analogue scales, their current level of hunger and fullness, and how much they enjoyed eating each food. To ensure the absence of demand characteristics, participants were asked to indicate what they thought the aims of the study were. Finally, participants completed the TFEQ and YFAS, and measures of height and weight were taken to calculate BMI. Participants were fully debriefed and were made aware of the element of deception used in the study. Figure 6.1. presents a flow-chart of the study procedure.

6.4.5. Data analysis

6.4.5.1. Participant characteristics.

Independent samples t-tests were conducted to identify group differences (i.e. high addiction group vs. low addiction group) in participants' BMI, age, TFEQ restraint, TFEQ disinhibition, and YFAS symptom count. One-way ANOVAs were also conducted to ensure that groups did not differ on actual SST and IAT performance.

6.4.5.2. Manipulation check.

To check that the manipulation had been successful, an independent samples t-test was conducted to compare levels of self-perceived food addiction in high- and low- addiction conditions.

6.4.5.3. Food intake.

The amount (in g) of chocolate and crisps consumed was converted into calories. A 2x2 mixed design ANOVA was conducted with food (chocolate, crisps) as the within subjects factor, and condition (high-addiction, low-addiction) as the between subjects factor.

6.4.5.4. Supplementary analyses

Food ratings.

Group differences in liking, craving, desire-to-eat, difficulty to resist, and post-consumption enjoyment ratings were explored using 2 x 2 mixed design ANOVAs. For each analysis, food (*i.e.*, crisps and chocolate) was entered as the within-subjects variable, and condition (*i.e.*, high- and low-addiction) was entered as the between subjects variable.

Hunger and fullness.

Hunger and fullness ratings were analysed using 2 x 2 mixed design factorial ANOVAs. Time (*i.e.*, before and after the taste task) was the within-subjects variable, and condition (*i.e.*, high- and low-addiction) was the between subjects variable.

Mood and taste.

Multivariate ANOVAs were conducted with condition (*i.e.* high- and low-addiction) as the independent variable, and mood and taste ratings entered as dependent variables.

6.5. Study 1 Results

6.5.1. Participant characteristics

Two participants did not believe in the concept of food addiction, one of whom also guessed the aims of the study. These two participants were removed from subsequent analyses⁶. The remaining sample consisted of $n=30$ in the low-addiction condition, and $n=32$ in the high-addiction condition. The average BMI was within the healthy weight range ($M=23.4 \pm 4.3\text{kg/m}^2$). Fourteen participants were classed as overweight ($\text{BMI}>25 \text{ kg/m}^2$), and four were classed as obese ($\text{BMI}>30 \text{ kg/m}^2$). There were no between-condition differences with regard to BMI, age, scores on trait measures of eating behaviour (*i.e.* YFAS symptom count, TFEQ-disinhibition or TFEQ-restraint), or performance on the SST or IAT tasks ($ps>.262$)(Table 6.1).

Table 6.1. Participant characteristics and appetite ratings in each condition. Values are means \pm standard deviation.

	Low-addiction ($n=29$)	High-addiction ($n=28$)
Age (years)	26.3 \pm 9.6	23.3 \pm 11.0
BMI (kg/m^2)	23.3 \pm 3.8	23.5 \pm 4.8
TFEQ-R	10.1 \pm 4.4	10.7 \pm 4.8
TFEQ-D	6.7 \pm 3.3	6.5 \pm 3.2
YFAS-symptoms	1.27 \pm .91	1.52 \pm 1.15
IAT score (ms)	400 \pm 187	426 \pm 243
SST (no. of commission errors)	3.64 \pm 4.62	3.97 \pm 3.47

⁶ The analyses were re-run to include the two participants who did not believe in food addiction and guessed the aims of the study, and the results did not change.

6.5.2. Manipulation check

Participants in the high-addiction condition believed more strongly that they were food addicts ($M=3.84 \pm 0.69$) than those in the low-addiction condition ($M=2.77 \pm 0.88$), $t(60)=5.29$, $p<.001$, $d=1.35$, thus indicating that the manipulation was successful.

6.5.3. Food intake

There was a main effect of condition on calorie intake, $F(1,60)=7.61$, $p=.008$, $\eta_p^2=0.11$, such that those in the high-addiction condition ($M=163 \pm 129$) consumed significantly fewer calories than those in the low-addiction condition ($M=261 \pm 148$). There was also a significant condition by food interaction, $F(1,60)=4.52$, $p=.038$, $\eta_p^2=0.07$, see Figure 6.2. Subsequent independent t-tests revealed that those in the high-addiction condition consumed fewer calories from chocolate than those in the low-addiction condition, $t(54)=-2.88$, $p=.006$, $d=0.73$. Participants also tended to eat fewer calories from crisps in the high addiction condition relative to the low addiction condition though this difference was not statistically significant $t(60)=-1.61$, $p=.113$, $d=0.41$.

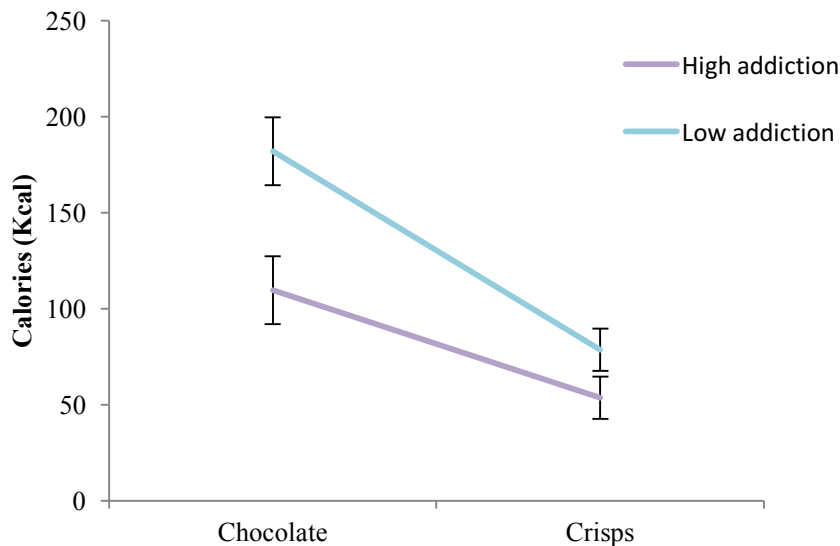


Figure 6.2. Mean calories consumed from chocolate and crisps as a function of condition.

**Significant between-condition difference at $p<.01$. Error bars represent standard error of the mean.

6.5.4. Supplementary analyses

6.5.4.1. Food ratings.

There was no main effect of condition ($ps > .250$), and no condition x food interactions ($ps > .215$), on any of the food ratings. There was a main effect of food such that chocolate was rated higher on all appetitive scales than crisps ($ps < .034$).

6.5.4.2. Hunger and fullness.

There was no main effect of condition ($ps > .250$), and no condition x time interaction ($ps > .229$), on hunger and fullness ratings. There was a main effect of time on hunger, $F(1,60)=56.36$, $p < .001$, $\eta_p^2 = .48$, and fullness, $F(1,60)=35.79$, $p < .001$, $\eta_p^2 = 0.37$. Specifically, prior to the taste task, hunger ratings were significantly greater ($M=62 \pm 38$ mm), and fullness ratings significantly lower ($M=25 \pm 20$ mm), than after the taste task (Hunger: $M=38 \pm 25$ mm; Fullness: $M=45 \pm 26$ mm).

6.5.4.3. Mood and taste.

There was no main effect of condition on mood, $F(21,40) = 1.00$, $p > .250$, $\eta_p^2 = .345$, or taste ratings, $F(14, 47) = .62$, $p > .250$, $\eta_p^2 = .16$.

6.6. Interim Discussion

Participants who were led to believe that they had scored highly on an ostensible measure of food addiction consumed less snack food than those who were led to believe that they had a low score. This is consistent with the notion that believing in food addiction may help people to limit their food intake. However, it is not possible to determine the direction of the results; calorie intake may have decreased in the high-addiction condition, increased in the low-addiction condition, or both. Accordingly, in Study 2, a control condition was included in which participants were led to believe that they had ‘average’ food addiction tendencies. Study 2 also included a direct test of the hypothesis that believing oneself to be a food addict would decrease eating because it generates concern about one’s eating behaviour. Specifically, it was predicted that those in the high-addiction condition would demonstrate higher levels of dietary concern than those in the low-addiction condition, and that this in turn would lead them to reduce the amount of time that they exposed themselves to the snack foods in the taste test. Finally, Study 2

examined whether the food addiction manipulation influenced participants' more general beliefs about food-related self-control and their future intentions to diet.

6.7. Study 2 Method

6.7.1. Overview of method

To investigate the direction of findings obtained in Study 1, Study 2 incorporated a third condition in which participants were led to believe they had average levels of food addiction (average-addiction condition). The overall procedure and materials used in Study 2 were similar to those used in Study 1 but with the following additions: Firstly, ratings of food-related self-control and dietary concern were obtained before and after the taste task, respectively. Secondly, the experimenter covertly recorded the amount of time participants spent completing the taste task. Finally, participants' longer-term 'intentions to diet' were assessed towards the end of the study using the Dietary Intention Scale (DIS). Figure 6.3. provides an overview of the study procedure.

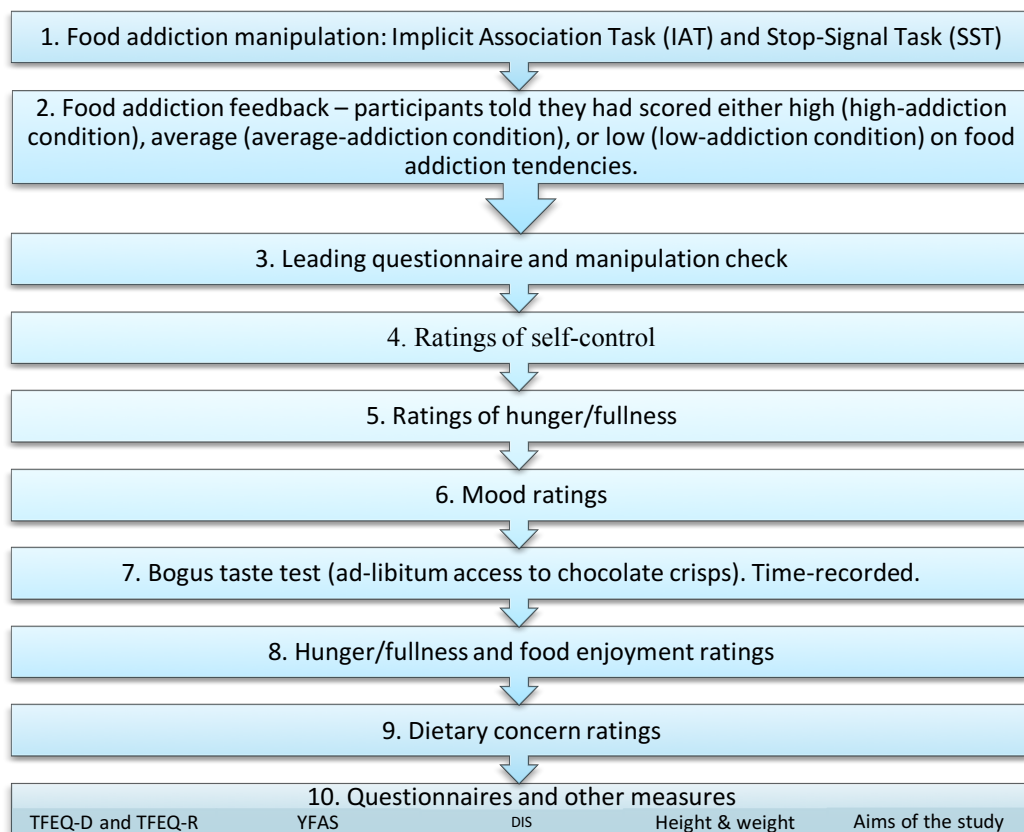


Figure 6.3. Flow chart of the Study 2 procedure

6.7.2. Participants

Ninety female participants were recruited using the same procedure as in Study 1. In a between-subjects design, participants were randomly allocated to high-, low-, or average-addiction conditions. We powered the study (80% power) using GPOWER 3.1 to detect a medium-large effect size ($f=.35$, on the basis of Study 1) at an alpha level of $p=.05$ and recruited slightly above the required sample ($N=84$) to account for any participants guessing the study aims.

6.7.3. Measures

Study 2 incorporated all the materials used in Study 1 but with the following additions:

6.7.3.1. Self-control beliefs

This was assessed by the following question; “On a scale of 0-8, how would you rate your ability to control your food intake?”. End anchor points “Extremely poor” and “Extremely good” were provided at the minimum (i.e. 0) and maximum (i.e. 8) points of the scale, respectively.

6.7.3.2. Dietary concern

This was assessed using the following question: “Earlier in the experiment, you were given some feedback on your performance on the computerised tasks. How concerned did this feedback make you feel about your eating behaviour?”. Participants indicated their response on a 100mm VAS scale. End anchor points ‘Not at all concerned’, and ‘Extremely concerned’, were provided to the left and right of the scale, respectively.

6.7.3.3. Dieting Intentions Scale (DIS). (Cruwys, Platow, Rieger, & Byrne, 2013).

The DIS is a 7-item questionnaire which assesses intentions to diet over the following three months (e.g. ‘In the next three months, I intend to go on a diet’). Scores were calculated by taking an average of participant responses to the seven items. Thus maximum intention to diet is represented by a score of 7, while the minimum score is 0.

6.7.4. Procedure

Participants were randomly assigned to high-, low-, or average (control) addiction conditions. In the average condition, participants were led to believe that they had average levels of food addiction, as indicated by scores of 45.245 and -24 in the SST and IAT, respectively. These values corresponded to the 50th percentile on

the bogus histogram. Furthermore, those in the average condition completed a leading questionnaire that consisted of two questions from the high addiction condition, and two questions from the low addiction condition. In the interest of maintaining consistency between conditions, those in the high- and low- addiction conditions also completed four leading questions, rather than the five used in Study 1. The overall procedure remained consistent with that used in Study 1 but with the following additions: Firstly, following the manipulation check, participants completed the self-control beliefs rating. Secondly, the amount of time that participants took to complete the *ad libitum* taste task was recorded. Thirdly, after completing the *ad libitum* taste task and subsequent hunger, fullness, and enjoyment rating scales, participants indicated their level of dietary concern. Finally, before completing the TFEQ-D, TFEQ-R and YFAS, participants completed the dieting intention scale. An overview of the study procedure is shown in Figure 6.3.

6.7.5. Data analysis

6.7.5.1. Participant characteristics.

One-way ANOVAs were conducted to identify group differences in participants' BMI, age, TFEQ restraint, TFEQ disinhibition, and YFAS symptom count. One-way ANOVAs were also conducted to ensure that groups did not differ on the SST and IAT.

6.7.5.2. Manipulation check.

To check that the manipulation had been successful, a univariate ANOVA was conducted to compare levels of self-perceived food addiction in high-, average- and low- addiction conditions.

6.7.5.3. Food intake.

The amount (in g) of chocolate and crisps consumed was converted into calories. A 2x3 mixed factorial ANOVA was conducted with food type (i.e. crisps and chocolate) as the within-subjects variable, and condition (i.e. high-, average- and low- food addiction) as the between subjects variable.

6.7.5.4. Dietary concern and time taken.

Univariate ANOVAs were conducted to examine the effect of condition on levels of dietary concern and time taken to complete the taste task. In each analysis,

condition was entered as the independent variable, and dietary concern or time taken were entered as dependent variables.

Mediation analyses.

Using PROCESS (Model 6) (Hayes, 2012; Hayes & Preacher, 2014), a serial multiple mediation analysis was conducted to examine whether the effect of condition on calorie intake would be mediated by levels of dietary concern which, in turn, would affect the amount of time participants willingly spent exposed to the food during the taste test (i.e. time taken) (Figure 6.4). The following procedure was used:

- 1) To explore the indirect effects of the low-addiction condition, this condition was dummy coded as 1, while average- and high-addiction conditions were dummy coded as 0.
- 2) To explore the indirect effects of the high-addiction condition, this was dummy coded as 1, while average- and low- addiction conditions were coded as 0.
- 3) The two proposed mediators (i.e. dietary concern and time-taken) and the dependent variable (i.e. calorie intake) were log-transformed prior to analysis. This was to ensure that the data met parametric assumptions, and to enable easier comparison of effects.
- 4) Using PROCESS (Hayes, 2012), the model was computed twice to provide coefficients for each between-condition comparison (i.e. low vs. average and high vs. average). The following specifications were applied for each computation: a) Between-condition comparisons (i.e. low vs average; high vs average) were included as independent variables. b) Calorie intake was entered as the dependent variable. c) Dietary concern and time taken were entered as the first and second mediators, respectively (as dietary concern was expected to influence calorie intake via its effect on time taken). d) When exploring the indirect effects of the high-addiction condition, the low addiction condition was entered as a covariate in the mediation model, and vice versa (as recommended by Hayes & Preacher, 2014). e) Due to the asymmetric distribution associated with mediation analyses, the total indirect effect was tested using a nonparametric bootstrapping procedure ($n=10,000$ samples). This procedure establishes 95% confidence intervals which can be used to deduce levels

of significance. Specifically, the total indirect effect of the model may be interpreted as significant ($p < .05$) if the resulting confidence intervals (CIs) do not contain zero (MacKinnon et al., 2002).

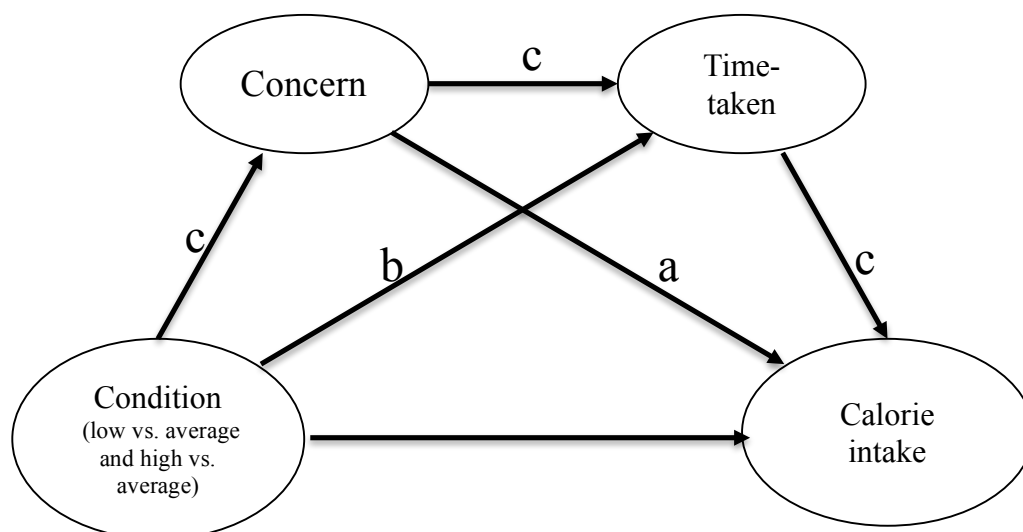


Figure 6.4. Schematic representation of the hypothesised indirect effect of condition on calorie intake via dietary concern and time-taken (pathway c). The model also calculated the effects of condition on calorie intake via dietary concern (pathway a) and time-taken to complete the taste-task (pathway b).

6.7.5.5. Self-control and dieting intentions.

Separate 3x2 univariate ANOVAs were conducted with self-control ratings or dieting intention scores (i.e. DIS scores) entered as dependent variables, and condition (high-, average-, low- addiction) entered as independent variables.

6.7.5.6. Supplementary analyses

Food ratings.

Group differences in liking, craving, desire-to-eat, difficulty to resist, and post-consumption enjoyment ratings were explored using 3 x 2 mixed design ANOVAs. For each analysis, food (i.e., crisps and chocolate) was entered as the within subjects variable, and condition (i.e., high-, average- and low-addiction) was entered as the between subjects variable.

Hunger and fullness.

Hunger and fullness ratings were analysed using 3 x 2 mixed design factorial ANOVAs. Time (i.e., before and after the taste task) was the within subjects

variable, and condition (*i.e.*, high-, average- and low-addiction) was the between subjects variable.

Mood and taste.

Multivariate ANOVAs were conducted with condition (*i.e.* high-, average- and low-addiction) as the independent variable, and mood and taste ratings entered as dependent variables.

6.8. Study 2 Results

6.8.1. Participant characteristics

Participants who guessed the aims of the study ($n=2$), or who did not believe in the concept of food addiction ($n=3$), were excluded from analyses⁷. The remaining sample consisted of $n=28$ in the high-addiction condition, $n=29$ in the low-addiction condition, and $n=28$ in the average-addiction condition. The average BMI was within the healthy weight range ($M=22.8 \pm 4.5\text{kg/m}^2$). Sixteen participants were classed as overweight ($\text{BMI} > 25\text{kg/m}^2$), and 3 were classed as obese ($\text{BMI} > 30\text{kg/m}^2$). There were no differences between conditions with regard age, BMI, performance on the computerized IAT and SST tasks, or scores on TFEQ-R, TFEQ-D or YFAS symptom count ($ps > .106$) (Table 6.2). A chi-square analysis confirmed that the number of people who fulfilled the YFAS diagnostic criteria for food addiction ($n=7$) did not differ between conditions, $\chi^2(2)=1.42, p=.536$.

Table 6.2. Participant characteristics and appetite ratings in each condition. Values are means with standard deviations in parentheses.

	Low-addiction ($n=29$)	Average-addiction ($n=28$)	High-addiction ($n=28$)
Age (years)	19.6 ± 1.8	19.1 ± 1.8	20.3 ± 3.9
BMI (kg/m²)	22.5 ± 2.9	21.9 ± 3.5	24.0 ± 6.4
TFEQ-R	10.0 ± 5.9	9.0 ± 5.2	11.5 ± 5.7
TFEQ-D	6.9 ± 3.4	7.6 ± 3.9	7.7 ± 3.4
YFAS-symptoms	1.86 ± 1.15	2.43 ± 1.60	2.39 ± 1.77
IAT score (ms)	359 ± 205	286 ± 151	275 ± 114
SST (no. of commission errors)	4.59 ± 3.42	4.61 ± 2.53	3.75 ± 3.95

⁷ All findings remained the same when the five participants who guessed the aims of the study, or who did not believe in the concept of food addiction, were included in the analyses.

6.8.2. Manipulation check

There was a main effect of condition on self-perceived food addiction, $F(2,82)=7.33, p=.001, \eta_p^2=0.15$. Specifically, those in the low-addiction condition believed less strongly on the 5-point Likert scale that they were food addicts ($M=2.10 \pm 0.72$) compared to those in the high-addiction condition ($M=3.00 \pm 1.05$), $p<.001, d=1.00$, and the average-addiction condition ($M=2.64 \pm 0.87$), $p=.025, d=0.68$. Self-perceived food addiction did not differ significantly between the high- and average-addiction conditions, $p=.138, d=0.37$.

6.8.3. Food intake

A 2 x 3 mixed ANOVA was conducted with food (crisps, chocolate) as a within subjects factor, and condition (high-, average-, low-addiction) as a between subjects factor. There was a main effect of condition on calorie intake, $F(2,82)=3.82, p=.026, \eta_p^2=.09$ (see Figure 6.5). Specifically, those in the high-addiction condition consumed significantly fewer total calories than those in the low-, $p=.024, d=0.58$, and average-addiction conditions, $p=.015, d=0.81$. Total calorie intake did not differ significantly between those in the low- and average-addiction conditions, $p=.837, d=.05$. The condition x food type interaction was not significant, $F(2,82)=1.30, p=.278, \eta_p^2=.03$.

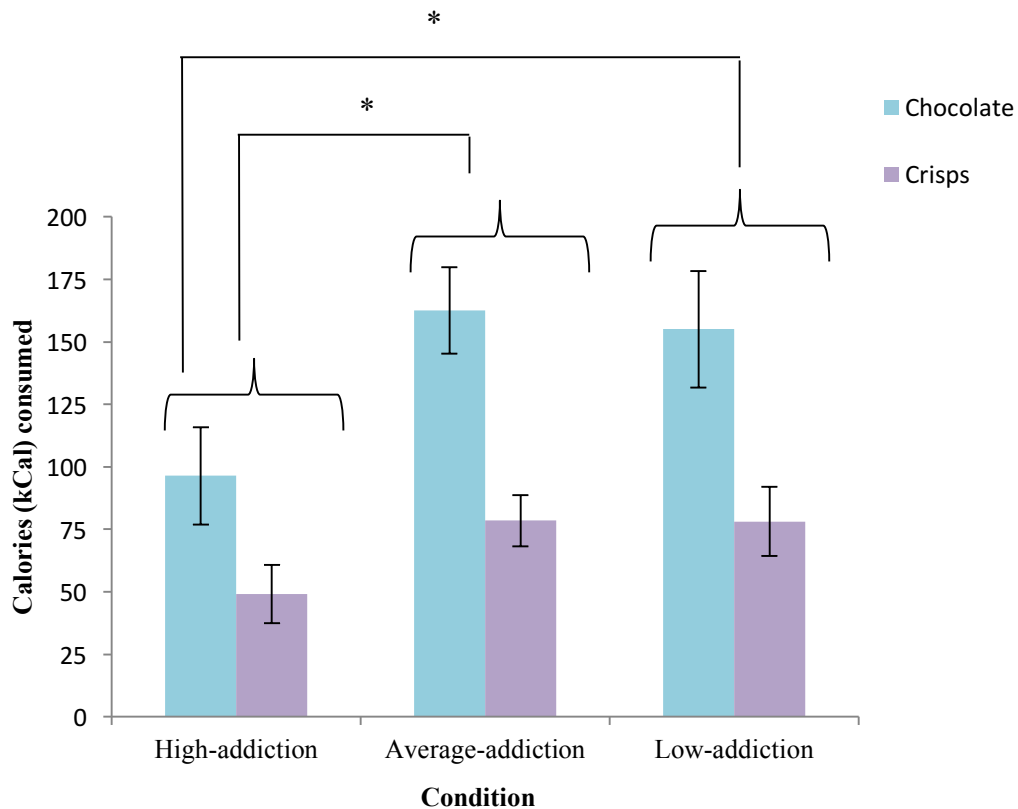


Figure 6.5. Mean calories consumed as a function of condition (high- addiction, low- addiction, or average-addiction) and food type (chocolate and crisps). *Significant at $p < .05$. Error bars represent standard error of the mean.

6.8.4. Dietary concern and time taken

There were main effects of condition on dietary concern, $F(2,82)=27.18$, $p < .001$, $\eta_p^2=.40$ (Figure 6.6.), and time taken to complete the taste task $F(2,82)=5.23$, $p=.007$, $\eta_p^2=.11$ (Figure 6.7.). With regard to dietary concern, those in the high-addiction condition had significantly greater levels of concern than those in the average-, $p < .001$, $d=1.11$, and low-, $p < .001$, $d=1.91$, addiction conditions. Those in the average-addiction condition demonstrated significantly more concern than those in the low-addiction condition, $p=.021$, $d=0.77$. With regard to time taken, those in the high-addiction condition took less time to complete the taste test than those in the low-, $p=.007$, $d=0.79$, and average-addiction conditions, $p=.006$, $d=0.83$. Time taken to complete the taste test did not differ between those in the low- and average-addiction conditions, $p=.940$, $d=.02$.

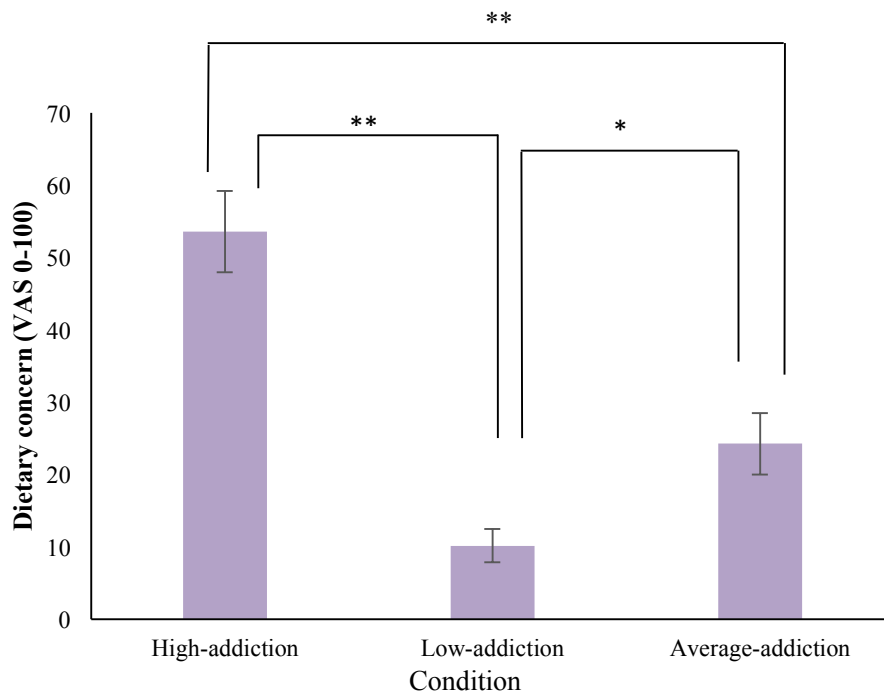


Figure 6.6. Dietary concern ratings following the food addiction feedback as a function of condition. ** $p < .001$. * $p < .05$.

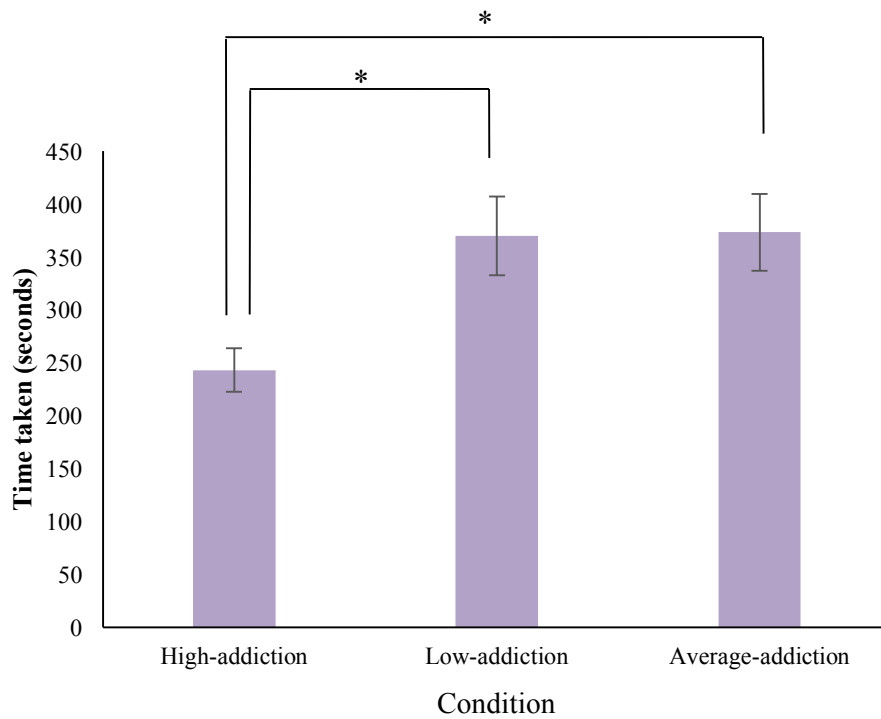


Figure 6.7. Time taken to complete the taste and rate task as a function of condition. * $p < .05$.

6.8.5. Mediation analyses

Mediation analyses revealed a significant total effect (Figure 6.8), and total indirect effect (Table 6.3), of the high- vs. average-addiction condition on calorie intake. As predicted, the high- vs. average-addiction condition affected calorie intake serially through dietary concern and time taken, $b=-.06(.03)$, 95% confidence interval (CI) = $-.13, -.01$. As the range of these CIs do not include zero, this indicates a significant indirect effect of condition on calorie intake. Specifically, the reduced calorie intake observed in the high-, relative to average-, addiction condition, was due to increased levels of dietary concern, which were subsequently associated with reduced time taken to complete the taste task. There was also a simple indirect effect of the high- vs. average-addiction condition on calorie intake through time-taken, $b=-.17(.08)$, 95%CI= $-.33,-.02$. After controlling for these indirect effects, the direct effect of the high- vs. average-addiction condition on calorie intake was no longer significant, $b=-.07(.08)$, $p=.355$ (Figure 6.8). There was no total effect, $b=-.12(.09)$, $p=.216$, or total indirect effect of the low- vs. average-addiction condition on calorie intake, and none of the direct or indirect pathways in this model were significant (Table 6.3).

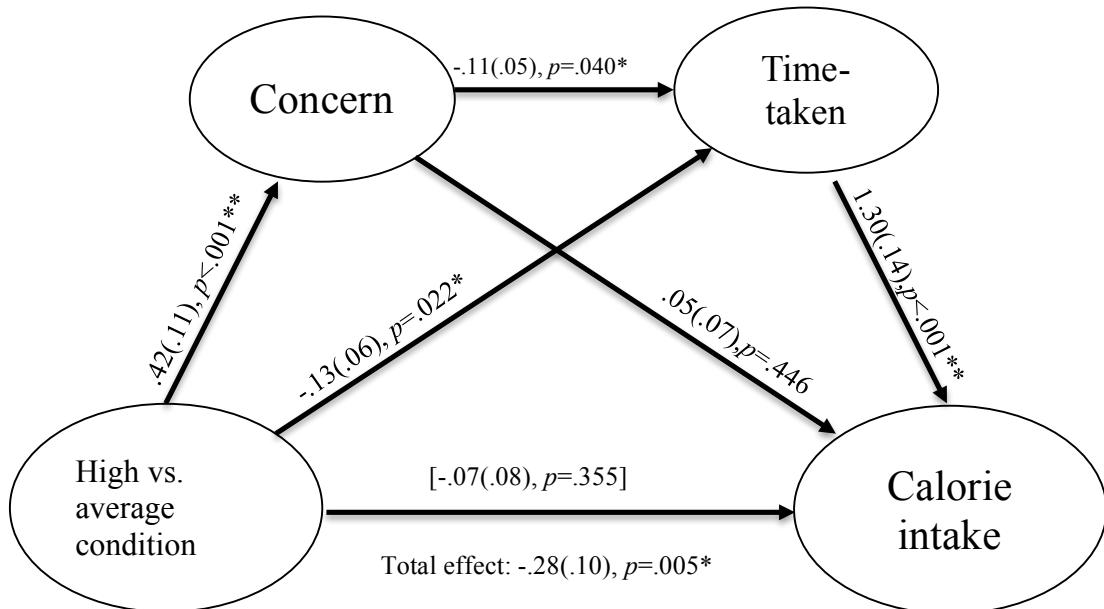


Figure 6.8. Serial mediation analysis with High- vs- Average condition comparison as the predictor variable, calorie intake as the outcome variable, and dietary concern and time-taken as first and second mediators, respectively. Values are unstandardized regression coefficients (SEs) and associated p-values. *Significant at $p < .05$., **Significant at $p < .001$. Bracketed association = direct effect after controlling for dietary concern and time taken.

Table 6.3. Indirect effects of condition on calorie intake via dietary concern and time taken to complete the taste task.

Indirect effect	Comparison	B (SE)	CL.95 (lower bound)	CL.95 (upper bound)
Total indirect effect	Low vs. Average	-0.02(.07)	-0.16	0.12
	High vs. Average	-.21(.08)*	-0.36	-0.07
Indirect effect 1 (pathway A)	Low vs. Average	-.02(.03)	-0.08	0.02
	High vs. Average	.02(.03)	-0.03	0.10
Indirect effect 2 (pathway B)	Low vs. Average	-0.05(.07)	-0.19	0.08
	High vs. Average	-.17(.08)*	-0.33	-0.02
Indirect effect 3 (pathway C)	Low vs. Average	.04(.03)	0.00	0.13
	High vs. Average	-.06(.03)*	-0.13	-0.01

*Significant effects at $p < .05$

Indirect effect 1 (pathway A) : Condition -> dietary concern -> intake

Indirect effect 2 (pathway B): Condition -> time taken -> intake

Indirect effect 3 (pathway C): Condition -> dietary concern -> Time taken -> intake

6.8.6. Self-control and Dieting Intentions.

There was no main effect of condition on self-control ratings, $F(2,82)=1.90$, $p=.158$, $\eta_p^2 = .04$, nor on the dieting intention scale (DIS) scores, $F(2,82)=0.99$, $p=.377$, $\eta_p^2=.02$. Thus, the effect of experimental condition on calorie consumption does not appear to be caused by changes in self-control ratings or future dieting intentions.

6.8.7. Supplementary analyses

6.8.7.1. Food ratings.

There was no main effect of condition ($ps > .086$), and no condition x food interaction ($ps > .250$), on liking, craving, desire-to-eat, difficulty to resist, or post-consumption enjoyment ratings. There was a main effect of food such that chocolate was rated higher than crisps on all scales ($ps < .001$) except for ‘difficulty to resist’ ($F(2,82)=0.48$, $p=.092$).

6.8.7.2. Hunger and fullness.

There were no differences, between conditions, with regards to hunger ratings obtained prior to the taste task (Figure 6.9) ($ps > .161$). Notably, hunger ratings were similar across Studies 1 and 2. There was also no main effect of condition on hunger or fullness ratings ($ps > .250$). There was a main effect of time

such that hunger ratings decreased, $F(1,82)=65.94, p<.001, \eta_p^2=.45$, and fullness ratings increased, $F(1,82)=65.93, p<.001, \eta_p^2=.45$, following the taste task. A condition x time interaction was observed for hunger ratings, $F(2,82)=5.31, p=.007, \eta_p^2=.12$ (Figure 6.9). To examine this further, changes in hunger ratings, before and after the taste task, were calculated for each participant. A univariate ANOVA revealed a main effect of condition on hunger change, $F(2,82)=5.31, p=.007, \eta_p^2=.12$. Pairwise comparisons revealed that hunger ratings declined significantly less, following the taste task, in the high-addiction condition ($M=9 \pm 19$ mm), compared to the low- ($M=25 \pm 30$ mm), $p=.016, d=.63$, and average-addiction ($M=29 \pm 22$ mm), $p=.003, d=.96$, conditions. Hunger change did not differ between average- and low- addiction conditions, $p=.526, d=.16$. There was no time x condition interaction on fullness ratings, $F(2,82)=2.56, p=.083, \eta_p^2=.06$.

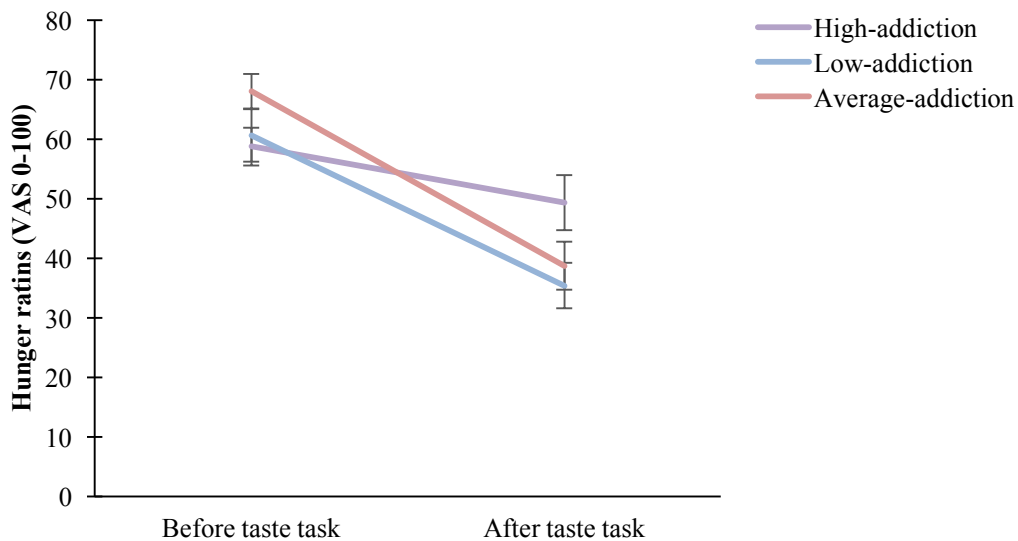


Figure 6.9. Mean hunger ratings before and after the taste task as a function of condition

6.8.7.3. Mood and taste.

Finally, multivariate ANOVAs revealed no main effect of condition on mood, $F(42,124)=1.05, p>.250, \eta_p^2=.26$, or taste ratings, $F(28,138)=0.66, p>.250, \eta_p^2=.12$.

6.9. Discussion

In Study 1, participants who were led to believe that they scored highly in food addiction consumed fewer calories than participants who were led to believe that they had scored low. Study 2 replicated and extended this finding by showing that the effect of the high-addiction condition on food intake was mediated by increased levels of dietary concern, which subsequently reduced the amount of time participants spent tasting and consuming the foods during the taste test.

Hoyt et al. (2014) recently demonstrated that an “obesity is a disease” message was associated with reduced concern for body weight and higher-calorie food choice in obese individuals. This public health message would appear to have similar connotations to the food addiction perspective in that both imply diminished personal control over eating behaviour and weight status. However, contrastingly, we found that leading people to believe themselves to be food addicts *increased* concern about eating and, in turn, *reduced* food intake. This may reflect differences between giving people general versus personalized information about health (Celis-Morales et al., 2015). There may also be different underlying conceptualizations of food addiction and disease. The notion that obesity is a disease implies that it is a physiological inevitability and therefore beyond personal control (Williamson, 2012). In contrast, in a recent survey, food addiction was regarded to be more a matter of personal choice, and less of a disease, than other addictions such as alcoholism (DePierre, Puhl, & Luedicke, 2014). Self-reported food addicts may therefore retain some sense of control over their “addiction”. Indeed, in the current study, participants’ perceptions of their ability to control food intake were not significantly influenced by the food addiction feedback. As such, believing oneself to be a food addict may not evoke the same deleterious effects on self-regulation as holding disease-based beliefs about one’s weight.

The inclusion of a control group in Study 2 clarified that food intake *decreased* in the high-addiction condition, and did not *increase* in the low-addiction condition. Such findings offer an alternative explanation to that provided by Nordgren et al.’s (2009) restraint bias theory, in which it is proposed that an over-confidence in one’s ability for self-control may cause people to over-expose themselves to tempting situations. Specifically, our findings showed that leading people to believe that they are food addicts reduced exposure to and intake of snack

foods, as opposed to a counter-productive effect of over-confidence in the group who were told they scored low in food addiction.

Few participants correctly guessed the aims of the research, suggesting that the food addiction feedback was a plausible manipulation. However, a potential limitation is that our findings may have been driven by participants' desire to prove the experimenter wrong. Specifically, upon receiving feedback that they had scored highly in food addiction, participants may have refrained from eating large amounts of food in an attempt to contradict their diagnosis as a 'food addict'. Similarly, given that food addiction may have negative connotations (DePierre et al., 2014), participants in the high-addiction condition may have been motivated to restrict their food intake in an attempt to minimise feelings of stigmatisation. Future studies should explore this possibility by measuring the extent to which participants feel they would be judged negatively after receiving the food addiction feedback. However, while such factors may have played a role, findings from Study 2 suggest that the effect of the feedback on food intake was primarily driven by increased dietary concern; indeed, the effect of condition was no longer observed after controlling for levels of dietary concern.

To our knowledge, the current study is the first to provide insight into the causal influence of personal food addiction beliefs on eating behaviour. Notably, we tested a non-clinical sample of female participants, most of whom were healthy weight, and it is now important to apply this approach to males and obese populations. Indeed, our belief manipulation may have an opposite effect on calorie intake in obese individuals, particularly if the feedback is congruent with pre-existing personal beliefs about eating behaviour. Specifically, given the association between perceptions of oneself as a food addict and increased weight (Ruddock et al., 2015; Chapter 2), it is likely that the majority of participants in the current study (i.e. who were of healthy weight) did not already perceive themselves to be addicted to food. As such, the food addiction feedback may have induced feelings of dissonance with participants' self-concepts (consistent with cognitive dissonance theory, Aronson, 1999), thus leading participants to modify their behaviour (i.e. to eat less). The moderating effect of weight status on food addiction beliefs should thus be investigated in future research. In particular, we may expect those with

obesity to have less dissonance between the food addiction feedback and self-concept; instead the feedback may confirm participants' pre-existing beliefs resulting in behaviour that is consistent with overeating. In contrast, in individuals with healthy weight, the food addiction feedback may be more dissonant with their self-concept, resulting in the need to reduce this by eating less. Indeed, Hoyt et al. (2014) reported a moderating effect of weight status, such that only participants with obesity demonstrated diminished concern for weight following an "obesity is a disease" message, relative to an information based message.

Future research should also consider the longevity of the effect. Notably, in Study 2 we did not uncover any significant effect of personal food addiction beliefs on longer term dieting intentions. Previous research has shown that attempts to restrict food intake over longer time periods can be futile by exacerbating cravings and promoting disinhibited eating patterns (Rogers & Smit, 2000; Keeler, Mattes, & Tan, 2015; Mann & Ward, 2001). On this basis, believing oneself to be a food addict might not be conducive to successful longer-term dietary control and weight management.

In conclusion, we found that believing oneself to be a food addict was associated with a subsequent reduction in calorie intake. By causing individuals to become more concerned about their eating behaviour, personal food addiction beliefs may help minimize the extent to which people expose themselves to food. Further research should establish the longer-term effects of personal food addiction beliefs and the potential implications for dietary control and obesity.

Chapter 7: The effect of eating-related guilt on food addiction attributions and snack choice

7.1. Overview

Chapters 2 to 5 suggest that individuals who perceive themselves to be addicted to food have increased levels of aberrant eating. It is possible that these individuals may use the concept of food addiction as an attribution for over-eating, perhaps as a way to alleviate feelings of eating-related guilt (Rogers & Smit, 2000). To test this possibility, Chapter 7 explored the effects of eating-related guilt on food addiction attributions.

A secondary aim of Chapter 7 was to extend findings from Chapter 6, in which participants who were led to believe they had scored highly on a measure of food addiction exposed themselves to tempting foods for less time than those who were told they had low or average levels of food addiction. Specifically, Chapter 7 explored whether eating-related guilt would increase participants' personal food addiction beliefs and, as a result, encourage them to choose less tempting snacks to take home with them (i.e. expose themselves to less temptation).

The study reported in this chapter is currently under review as:

Ruddock, H.K. & Hardman, C.A. (under review). Guilty pleasures: The effect of eating-related guilt on food addiction attributions and snack choice.

7.2. Abstract

The concept of food addiction is popular amongst members of the lay public and those with increased weight are particularly likely to perceive themselves to be 'food addicts'. One possibility is that attributing eating to a food addiction may help alleviate eating-related guilt by implying that overeating is outside of personal control. The current study tested this possibility by examining the effect of manipulating feelings of eating-related guilt on food addiction attributions. In order to manipulate eating-related guilt, female participants ($N=90$) were led to believe that they had eaten more than (high-guilt condition), less than (low-guilt condition), or roughly the same (control condition) amount of palatable foods in relation to

previous participants. It was predicted that participants in the high-guilt condition would be more likely to perceive themselves as ‘food addicts’ and to attribute their eating to the addictive qualities of the food. A secondary aim was to examine whether eating-related guilt and food addiction attributions would encourage people to avoid exposing themselves to tempting foods. To address this, participants were asked to choose a snack to take home with them and were given a monetary incentive to return the snack, uneaten, one week later. It was predicted that those in the high-guilt condition would choose less tempting snacks, than those in low-guilt and control conditions, and that self-perceived food addiction and increased dietary concern would be associated with less tempting snack selection. Findings revealed no effect of experimental condition on food addiction attributions. However, across the entire sample, participants with higher levels of guilt were more likely to attribute their eating to the addictiveness of the foods. Participants in the *low*-guilt condition selected *more* tempting snacks, than those in control and high-guilt conditions, however snack selection was unrelated to food addiction beliefs. These findings do not support suggestions that food addiction is used as a means of alleviating guilt following eating. However, future research should investigate whether food addiction beliefs may be used to reduce guilt associated with *longer-term* patterns of disinhibited eating.

7.3. Introduction

Within the scientific community, the extent to which foods yield an addictive potential analogous to that of drugs of abuse remains widely debated (Hebebrand et al., 2014; Carter et al., 2016; Ziauddeen et al., 2012). As discussed in Chapter 1 (section 1.2.6), an alternative perspective is that the concept of ‘food addiction’ may provide a more personally and socially acceptable attribution for overeating (Rogers & Smit, 2000). Specifically, it is thought that attributing lapses in self-control to a ‘food addiction’ implies that overeating is physiologically inevitable and beyond personal control. In doing so, Rogers and Smit (2000) propose that the concept of ‘food addiction’ may serve to alleviate feelings of personal responsibility and eating-related guilt. This perspective is in accordance with the tenets of Attribution Theory (Weiner, 1971; 1974) which accounts for the tendency to attribute undesirable outcomes to *external* and *uncontrollable* causes, such as biological or environmental

influences, and to downplay the role of *internal* and *controllable* factors, such as personal choice (Sedikides & Strube, 1995).

Indeed, previous research has demonstrated an ameliorating effect of biological and addiction-based explanations on stigmatization and perceptions of blame (Crandall & Reser, 2005; Hoyt et al., 2014; Latner et al., 2014; Pearl & Lebowitz, 2014). Conversely, causal attributions for obesity which emphasise the role of behavioural choice (i.e. lack of exercise and an unhealthy diet) have been found to increase blame and obesity-related stigma (e.g. Pearl & Lebowitz, 2014). Furthermore, there is evidence to suggest that attributions which emphasise the role of uncontrollable and external influences may be used to minimize feelings of guilt and negative affect following overeating (Adriaanse et al., 2011; Adriaanse et al., 2014; Adriaanse et al., 2016).

Drawing on the above, the primary aim of the current study was to investigate the extent to which ‘food addiction’ may be used as a self-serving attribution for eating. Specifically, it was predicted that individuals who were manipulated to have high levels of eating-related guilt (high-guilt condition), would be more likely to label themselves as food addicts (hypothesis 1) and to attribute their eating to the ‘addictiveness’ of the foods (hypothesis 2), than those in low-guilt and control conditions. Moreover, the effect of condition on food addiction attributions was expected to be mediated by levels of dietary concern and guilt.

A secondary aim was to examine the *consequences* of eating-related guilt and food addiction attributions on subsequent food choice. In Chapter 6 of this thesis, participants who were led to believe that they had scored highly on a measure of food addiction, exposed themselves to tempting foods for less time than those who were told they had low or average levels of food addiction. This was mediated by the effect of the food addiction feedback on dietary concern (Ruddock et al., 2016). A similar finding was reported by Nordgren et al. (2009), in which hungry participants, who believed they had a *low* capacity for self-control, selected less tempting snacks to take home with them (when given a monetary incentive to return the snack one week later), compared to satiated participants who believed they had a *high* capacity for self-control. Taken together, these findings suggest that individuals who perceive themselves as having low levels of self-control, or high levels of food addiction, may be inclined to minimise their exposure to tempting foods.

Using a similar paradigm to that of Nordgren et al. (2009), the current study explored the effects of eating-related guilt and self-perceived food addiction on snack selection. It was hypothesised that individuals who were led to feel guilty following eating would select less tempting snacks to take home with them, compared to those in control and low-guilt conditions. Furthermore, it was predicted that self-perceived food addiction and increased dietary concern (as a result of the guilt manipulation) would be associated with less tempting snack selection.

To summarise, the primary aim of the current study was to investigate whether food addiction beliefs may be used as a self-serving attribution for eating. It was hypothesised that individuals who were led to feel guilt following eating (high-guilt condition) would be more likely to attribute their eating to a self-perceived food addiction (hypothesis 1), and to the addictive qualities of foods (hypothesis 2), compared to those in low-guilt or control conditions. A secondary aim was to investigate the effect of eating-related guilt on subsequent snack choice. It was hypothesised that those in the high-guilt condition would select less tempting snacks to take home with them, compared to those in control and low-guilt conditions (hypothesis 3).

7.4. Method

7.4.1. Overview of study method

Figure 7.1. presents an overview of the study procedure. All participants were tested between 12pm and 2pm, to coincide with a typical lunch time. In a between-subjects design, participants were randomly allocated to one of three conditions (high-guilt, low-guilt, or control). After consuming a buffet lunch, participants were asked to estimate the number of calories they had consumed. To manipulate feelings of guilt, participants were then led to believe that they had consumed more than (in the high-guilt condition), less than (in the low-guilt condition), or roughly the same as (in the control condition) their estimate and compared to previous (bogus) participants. Food addiction attributions, and levels of guilt and dietary concern, were then assessed. To test whether eating related guilt would affect snack choice, participants were asked to rank six snacks from most to least tempting. They were then asked to choose a snack to take home with them and were given a monetary incentive to return the snack, uneaten, one week later. In the second session (i.e. one

week later), participants confirmed whether or not they had consumed the snack during the week, and completed several trait measures of eating behaviour. Finally, participants' height and weight were measured.

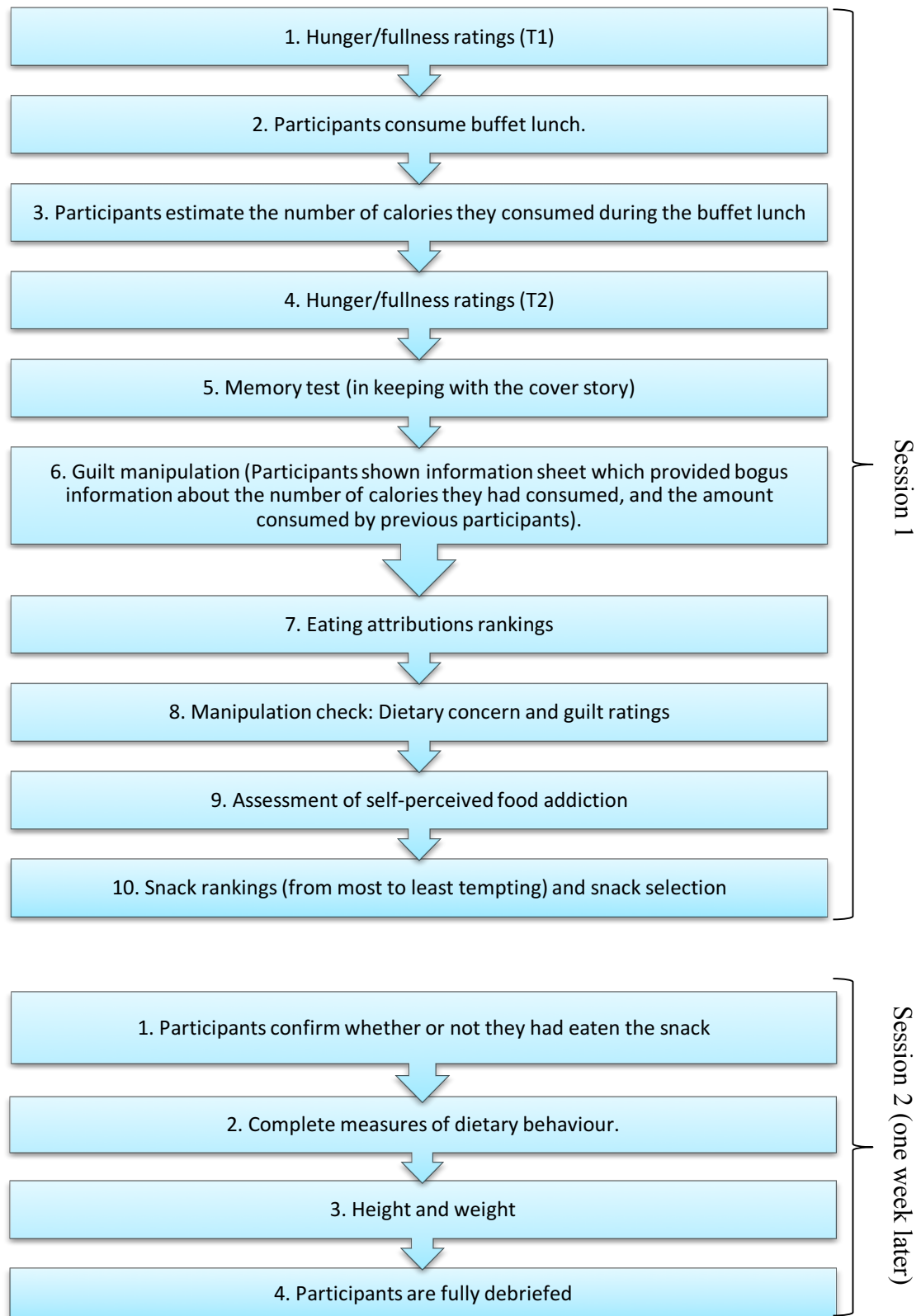


Figure 7.1. Overview of study procedure in sessions 1 and 2.

7.4.2. Participants

A power calculation was conducted using G*Power (Erdfelder, Faul, & Buchner, 1996). This determined a total sample size of 84 to detect a medium sized main effect ($f=0.35$) with a significance of .05. We slightly over-recruited to account for participants guessing the aims. Staff and students ($N=90$) from the University of Liverpool were invited to take part in a study which they were led to believe was about memory and food intake. As this was a preliminary study into food addiction attributions and consistent with the previous experimental studies in this thesis, only females were recruited in order to minimize between-subject differences.

Participants were excluded from the study if they were currently dieting, or had any food allergies or intolerances. Ethical approval was granted by the Institute of Psychology, Health and Society at the University of Liverpool.

7.4.3. Measures and materials

7.4.3.1. Ad libitum buffet lunch.

The buffet lunch consisted of 150g Tesco mini flapjacks (Per 100g: 458 Kcals, 21.9g fat), 115g Tesco mini brownies (Per 100g: 394 kcals, 15g fat), 2 x 22g packets of salt and vinegar Snack-a-Jacks (Per bag: 89 kcals, 1.6g fat), 1 x cheese and pickle sandwich (Tesco West Country Cheese and Pickle, Per pack: 572kcals, 28.8g fat), 2 x Tesco cheese and onion rolls (Per 60g roll: 173 kcals, 9.0g fat), 3 x 25g bags chocolate mini digestives (Per bag: 124 kcals, 5.8g fat). In total, the buffet lunch consisted of 2'608 calories and 117.5g fat. Previous research suggests that these foods may be perceived as particularly 'addictive' amongst members of the lay public (Schulte et al., 2015). Plates and bowls were covertly weighed before and after consumption to provide a measure of actual calorie intake.

7.4.3.2. Guilt and dietary concern manipulation: Bogus datasheet.

In order to manipulate levels of guilt and dietary concern, participants were exposed to bogus information about the amount of food eaten by ostensible previous "participants" via a bogus data sheet. Such techniques have previously been used to manipulate beliefs about the food consumption of previous 'bogus' participants (e.g. Robinson et al., 2014). The bogus participant datasheet consisted of four columns (Figure 7.2). The left hand column was headed 'Participant' and contained 27

numbered rows (i.e. 1-27). The following three columns, from left to right, were headed 'Gender', 'Age' and 'ACTUAL number of calories eaten', respectively. The first 12 rows in the table had been completed, thus providing bogus data for 12 participants. In the 'Gender' column, the first 12 rows were marked with an 'F' to indicate a female participant. In the 'Age' column, the first 12 rows indicated the ages of previous bogus participants, which ranged from 18 to 43 years. For each of the bogus participants, different handwriting and pen colours were used to create the illusion that these columns had been completed by different participants. The final column (i.e. 'ACTUAL number of calories eaten') contained the number of calories consumed by the previous (bogus) participants. These were arrived at based the current participant's estimated number of calories consumed (i.e. estimated intake). Specifically, in the 'high guilt' condition, the mean number of calories consumed by previous bogus participants was always 52.7% *less* than the current participant's own estimated intake. For those in the 'low-guilt' condition, the mean number of calories consumed by previous bogus participants was 52.7% *more* than the current participant's estimate. For those in the control condition, the mean intake of previous bogus participants was set at 7.2% more than the current participant's estimate (see column A of Table 7.1. for a worked example based on an estimated intake of 500 Kcals).

The bogus number of calories (i.e. bogus intake) consumed by the *current* participant was then provided in row 13 of the datasheet. For those in the high-guilt condition, participants were told that they had eaten 52.7% *more* than their estimate, while those in the low-guilt condition were led to believe they had consumed 52.7% *fewer* calories than they had estimated. Those in the control condition were told that they had consumed 7.2% more calories than their estimate, and thus did not differ from the mean number of calories consumed by previous bogus participants (see column B of Table 7.1. for a worked example based on an estimated intake of 500 Kcals).

The decision to vary the bogus calorie feedback in accordance with participants' estimated intake was taken to ensure that, in *all* cases, participants were led to believe that they had eaten more than (high-guilt condition), less than (low-guilt condition), or roughly the same as (Control condition) their estimated calorie intake.

Table 7.1. A worked example of the bogus calorie feedback provided to participants, in each condition, based on a participant's estimated intake of 500Kcals.

Condition	Participant's estimated intake	Column A Average intake of previous bogus participants	Column B Bogus intake*
Low-guilt	500 Kcals	(+52.7%)764 Kcals	(-52.7%) 237 Kcals
Control	500 Kcals	(+7.2%) 536 Kcals	(+7.2%) 536 Kcals
High-guilt	500 Kcals	(-52.7%) 236 Kcals	(+52.7%)764 Kcals

*i.e. the number of calories participants were led to believe they had eaten, in each condition.

Participant demographics

Participant	Gender	Age	ACTUAL number of calories eaten
1	F	28	364
2	F	34	315
3	F	23	338
4	F	35	381
5	F	21	295
6	F	19	324
7	F	22	354
8	F	23	287
9	F	18	298
10	F	36	348
11	F	27	308
12	F	26	364
13	F	18	1069
14			
15			
16			
17			
18			
19			
20			
21			
22			
23			
24			
25			
26			
27			

Figure 7.2. Actual bogus datasheet that was given to a participant in the high-guilt condition. In this instance, the participant had estimated she had eaten 700 Kcals. Based on this estimate, the participant was led to believe she had eaten 1069 Kcals, and that previous bogus participants had eaten an average of 331 Kcals. Details of previous bogus participants are provided in rows 1-12. Details of the current participant are shown in row 13.

7.4.3.3. Manipulation checks: Guilt and dietary concern ratings

To ensure that the manipulation had been successful, participants were asked to indicate their current level of guilt and dietary concern using two 100mm VAS scales. Each scale was presented on a computer screen with the following instruction: “Earlier in the experiment, you received some feedback on how many calories you consumed”. The Concern scale was then preceded with “How CONCERNED did this feedback make you feel?”, while the Guilt scale was preceded with “How GUILTY did this feedback make you feel?”. Both scales were anchored with ‘Not at all’ to the left, and ‘Extremely’ to the right. Dietary concern and guilt were presented, and responses recorded, on a laptop computer using Inquisit 3.0 software (Millisecond Software, 2012).

7.4.3.4. Self-perceived food addiction

To assess self-perceived food addiction, participants were asked to “Please indicate the extent to which you agree with the following statement: ‘I believe myself to be a food addict’”. Responses were provided on a 5-point Likert scale ranging from ‘Strongly Disagree’ to ‘Strongly Agree’. The assessment was presented, and responses recorded, on a laptop computer using Inquisit 3.0 software (Millisecond Software, 2012).

7.4.3.5. Eating attributions

An assessment of participants’ attributions for eating was presented using Inquisit 3.0. (Millisecond Software, 2012). The following instruction was displayed on the computer screen: “What was the most influential factor in determining how much of the buffet lunch you ate? Please indicate by assigning values ‘1’ (most influential) to ‘10’ (least influential) to the reasons provided below”. The following ten reasons were provided: ‘I felt hungry’, ‘The foods were really addictive’, ‘To relieve negative emotions (e.g. boredom, anxiety etc.)’, ‘I couldn’t control myself’, ‘I didn’t want to turn down free food’, ‘I was craving something sweet/salty’, ‘Because they were just there’, ‘I liked the taste of the foods’, ‘Out of habit - I eat when I’m watching TV’, and ‘I wanted to fill myself up’.

7.4.3.6. Temptingness ratings and snack selection

For the temptingness ratings and snack selection, participants were presented with the following six snacks: 70g pack Tesco chocolate rice cakes (336 kcals, 15g fat), 25g bag Walkers Baked Ready Salted crisps (102 kcals, 2g fat), 45g bar

Cadburys Dairy Milk (238 kcals, 14g fat), 36g Go Ahead Yoghurt break forest fruit flavour (146 kcals, 4g fat), 25g bag Tesco mini jelly beans (93 kcals, 0g fat), 160g pack Nairn's Gluten Free Oat cakes (774 kcal, 33g fat). Participants ranked the snacks in order of temptingness, ranging from 1 (most tempting) to 6 (least tempting), and selected one of the snacks to take home with them. All snacks were worth less than the monetary incentive offered to participants if they refrained from eating the snack (i.e. £2).

7.4.3.7. Food-related self-control ratings

Participants' perceived ability to control their food intake was assessed on a scale which ranged from 0 (extremely poor) to 8 (extremely good). The scale was presented on a computer screen, using Inquisit 3.0. (Millisecond software, 2012), alongside the following instruction: "On a scale of 0-8, how would you rate your ability to control your food intake?".

7.4.3.8. Additional measures and eating behaviour trait questionnaires

The Yale Food Addiction Scale (YFAS; Gearhardt et al., 2009) and Three Factor Eating Questionnaire (TFEQ, Stunkard & Messick, 1985) were used to provide descriptive information about the sample (see Chapter 3, section 3.4.2. for a full description of these measures). These measures were presented, and responses recorded, on a laptop computer using Inquisit 3.0. (Millisecond Software, 2012).

Hunger/fullness ratings.

Hunger and fullness ratings were provided on 100mm visual analogue scales (VAS) which were presented on a computer screen using Inquisit 3.0. (Millisecond Software, 2012). These were presented alongside the instruction to "rate how you feel right now". Each scale was anchored by 'Not at all' and 'Extremely' to the left and right of the scale, respectively.

7.4.4. Procedure

Figure 7.1. provides an overview of the study procedure. Participants were required to attend two study sessions, 1 week apart. The first session took place between 12pm and 2pm in order to coincide with usual lunch hours, and participants were asked to refrain from eating or consuming any calorie-containing drinks for 3 hours beforehand. Upon arrival, participants provided written consent, and completed a medical history questionnaire to ensure the absence of any food

allergies or intolerances. They then completed hunger and fullness VAS scales before being presented with the *ad libitum* buffet lunch to be consumed while watching a television (TV) programme. The experimenter gave the instructions to ‘eat as much of the food as you wish’ and to ‘pay attention to the TV programme as there would be a memory test afterwards’. The experimenter then started the TV programme (*Fawlty Towers*; episode ‘The Kipper and the Corpse’) which was approximately 30 minutes in duration. The decision to include a TV programme while eating the buffet lunch was two-fold. Firstly, it coincides with the cover story that the study was looking into food intake and memory. Secondly, watching television while eating has previously been shown to decrease one’s ability to monitor food intake (e.g. Moray, Fu, Brill, & Mayoral, 2007), thus maximizing the believability of our manipulation.

Once the programme had ended, participants were asked to estimate how many calories they had consumed during the buffet lunch. The participant wrote down their estimate which was then used by the experimenter to calculate the bogus calorie feedback. Participants then completed hunger and fullness VAS scales, and a memory test which consisted of 20 multiple-choice questions about the TV programme. While participants were completing these tasks, the experimenter covertly calculated, and wrote down on the bogus participant data sheet, the number of calories consumed by previous bogus participants (see section 7.4.3.2. and Table 7.1. for details about how these were calculated). The experimenter then returned with the completed bogus participant datasheet and informed the participant of the ‘actual’ number of calories they had consumed (i.e. bogus intake), and wrote this value onto the bogus datasheet (see section 7.4.3. and Table 7.1. for details of how bogus intake was computed based on the participant’s estimate). The participant was then asked to complete the gender and age columns on the datasheet (i.e. gender and age). Participants were left alone with the datasheet for 1 minute while they completed these columns. This was to provide participants with sufficient time to notice the number of calories consumed by previous bogus participants.

Participants then completed the eating attributions rankings, guilt and concern VAS scales, food-related self-control ratings, and the assessment of self-perceived food addiction. The experimenter then presented participants with 6 snack foods which they were asked to rank in order of ‘temptingness’. After they had done

this, participants were asked to choose one snack to take home with them. Participants were instructed to keep the snack with them at all times, and were told that if they returned the snack uneaten one week later, they would ‘win’ £2 and be able to keep the snack. The experimenter marked the selected snack with a sticker to ensure that the returned snack was the original.

During the second session, participants confirmed whether or not they had eaten the snack during the week and, if applicable, showed the experimenter the snack. Participants’ height and weight were taken and they completed the TFEQ-R, TFEQ-D and YFAS. Finally, participants were fully debriefed and informed of the aims of the study. Importantly, participants were told that the calorie feedback, and details of previous participants, that they had received in the previous session was bogus information designed to manipulate feelings of guilt.

7.4.5 Data analysis

A multivariate analysis of variance (MANOVA) was conducted to ensure that groups did not differ with regards to appetite ratings (i.e. hunger and fullness), estimated calorie intake, actual calorie intake, age, BMI, scores on the TFEQ-D, TFEQ-R, and YFAS symptom count.

7.4.5.1. Manipulation checks

A MANOVA was conducted to ensure that the three conditions (i.e. low guilt, control, and high guilt) had the expected effects on participants’ ratings of dietary concern and guilt. In particular, we expected that those in the high guilt condition would demonstrate greater levels of dietary concern and guilt compared to those in the control and low-guilt conditions. Those in the low-guilt condition were expected to demonstrate the lowest levels of dietary concern and guilt.

7.4.5.2. Self-perceived food addiction (hypothesis 1)

For our first hypothesis, it was predicted that self-perceived food addiction beliefs would be greatest in the high-guilt condition, compared to control and low-guilt conditions. To test this, a univariate ANOVA was conducted with condition (i.e. high-guilt, low-guilt, control) as the independent variable, and self-perceived food addiction as the dependent measure. In addition, correlational analyses were conducted to explore whether self-perceived food addiction beliefs were positively associated with levels of guilt and dietary concern.

7.4.5.3. Addictiveness attribution ranking (hypothesis 2)

It was hypothesised that participants in the high-guilt condition would assign a lower rank (i.e. indicating greater influence) to the addictiveness attribution (i.e. ‘foods were really addictive’) compared to those in the control or low-guilt conditions. To test this, a multivariate ANOVA was conducted in which condition (i.e. high-guilt, low-guilt, and control) was entered as a ‘fixed factor’ and the rankings for each of the 10 eating attributions were entered as dependent variables. Correlational analyses were also conducted to test whether the rank assigned to the addictiveness attribution would be negatively associated with levels of guilt and dietary concern.

7.4.5.4. Snack selection (hypothesis 3)

It was hypothesised that those in the high-guilt condition would choose a less tempting snack to take home with them, compared to those in control and low-guilt. Due to its non-parametric properties, snack selection was analysed using a Kruskal-Wallis test. The dependent variable was the temptingness rank that was assigned to the selected snack. Follow-up Mann-Whitney U tests were conducted to compare snack selection between each of the three conditions. Finally, Spearman’s correlation analyses were conducted to explore whether the selected snack rank was associated with self-perceived food addiction, self-control ratings, dietary concern and guilt.

7.5. Results

Preliminary analysis of the data revealed that BMI was positively skewed. Thus, using the outlier labelling rule defined by Hoaglin and Iglewicz (1987), one participant in the high-guilt condition (BMI = 40.2) was removed. Two participants (both in the high-guilt condition) indicated that they had guessed the aims of the study and were therefore also removed from subsequent analyses.⁸ Participant characteristics, appetite ratings, and estimated and actual calorie intake are provided in Table 7.2. Importantly, participants did not differ significantly between groups with regards to any of these characteristics ($ps > .13$). One participant (in the control condition) met the YFAS diagnostic criteria for food addiction.

⁸ The overall pattern of results remained the same when analyses were re-run with these three participants included.

Table 7.2. Participant characteristics, appetite ratings, and estimated and actual calorie intake, in each condition. Values are means with standard deviations in parentheses.

	Low guilt (n=30)	Control (n=30)	High guilt (n=27)
Age (years)	22.3 ± 7.3	22.7 ± 9.3	20.7 ± 5.9
BMI (kg/m ²)	23.1 ± 2.7	22.5 ± 1.7	23.6 ± 2.7
TFEQ-R	7.3 ± 4.4	8.6 ± 4.6	7.7 ± 4.0
TFEQ-D	7.5 ± 3.1	5.9 ± 3.2	7.1 ± 3.5
YFAS-symptoms	1.97 ± 1.16	2.00 ± 1.44	1.41 ± 0.93
Estimated intake (Kcal)*	566 ± 268	500 ± 282	623 ± 414
Bogus intake	268 ± 141	536 ± 146	951 ± 218
Actual intake (Kcal)	811 ± 260	793 ± 304	839 ± 354
Hunger (pre meal)	64 ± 18	69 ± 23	72 ± 14
Fullness (pre meal)	19 ± 19	15 ± 17	18 ± 18
Hunger (post meal)	8 ± 9	10 ± 18	13 ± 19
Fullness (post meal)	78 ± 19	79 ± 22	70 ± 28

*i.e. the number of calories participants estimated they had consumed during the buffet lunch, *prior to* the manipulation. N.B. All hunger and fullness ratings were taken before the guilt manipulation.

7.5.1. Manipulation check

Ratings of dietary concern and guilt differed significantly between groups, $F(4,168)=6.77, p<.001, \eta_p^2=.14$, (Figure 7.3). Pairwise comparisons revealed that levels of dietary concern were significantly greater in the high-guilt condition relative to both control ($p=.003$) and low-guilt conditions ($p<.001$). Control and low-guilt conditions did not differ with regards to dietary concern ($p=.100$). Levels of guilt were greatest in the high-guilt condition compared to low-guilt ($p<.001$) and control conditions ($p=.052$), although the latter comparison only approached significance. Guilt levels were also significantly lower in the low-guilt condition compared to the control condition ($p=.004$). These results indicate that our manipulation had been successful.

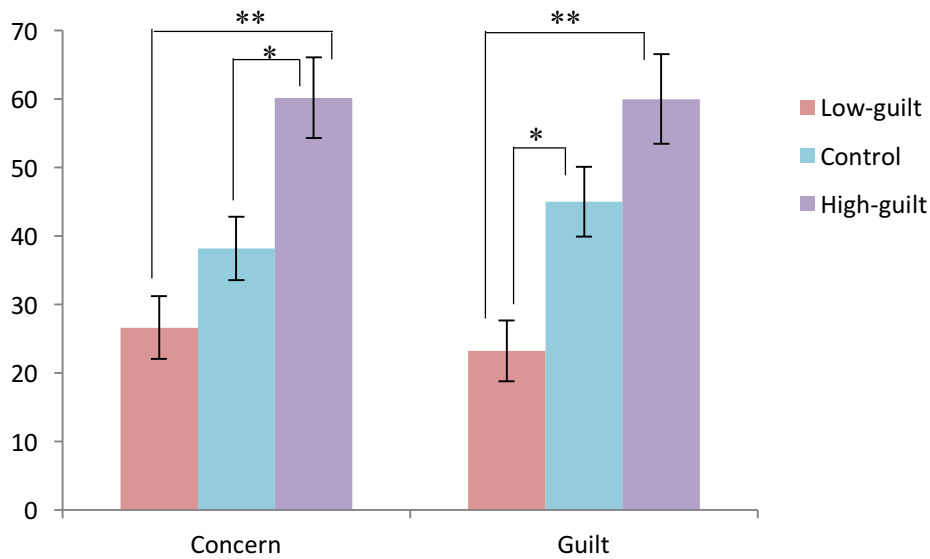


Figure 7.3. Mean dietary concern and guilt ratings by condition. *significant at $p < .01$, **significant at $p < .001$.

7.5.2. Self-perceived food addiction (hypothesis 1)

Contrary to our first hypothesis, there was no effect of condition on participants' responses to the assessment of self-perceived food addiction, $F(2,84) = .13, p = .878, \eta_p^2 = .00$, (Table 7.3). Self-perceived food addiction was also not significantly correlated with levels of guilt ($r = .088, p = .420$) or dietary concern ($r = .056, p = .606$). Rather, exploratory correlational analyses revealed that self-perceived food addiction correlated negatively with self-control ratings ($r = -.429, p < .001$), and positively with TFEQ-D ($r = .444, p < .001$), and YFAS symptom count ($r = .341, p = .002$). There was also a positive correlation between self-perceived food addiction and actual calorie intake which approached significance ($r = .209, p = .052$). Exploratory analysis also revealed no effect of condition on self-control ratings, $F(2,84) = 2.48, p = .090, \eta_p^2 = .06$.

Table 7.3. Mean (standard deviations) self-perceived food addiction rating, and rank assigned to the ‘foods were addictive’ attribution, in each of the three conditions.

	Low-guilt	Control	High-guilt
Self-perceived food addiction (Likert rating 1-5)	2.70(1.06)	2.63(1.19)	2.78(.93)
“Foods were addictive” attribution (rank, 1-10)*	6.50(2.45)	6.33(2.32)	6.51(2.38)

*Lower rank indicates more importance

7.5.3. Addictiveness attribution ranking (hypothesis 2)

The hunger attribution (“I was hungry”) was most frequently ranked as the first or second most influential reason for eating across all participants (78.2%), while the emotional eating attribution (“For emotional reasons”) was most frequently ranked as the least or second from least influential reason for eating (64.4%). Overall, there were no differences in attribution ranking between conditions, $F(20,152)=1.08, p=.377, \eta_p^2=.12$ (see Table A1 in Appendix C for descriptive and statistical data for each attribution). In particular, contrary to our second hypothesis, the rank assigned the addiction attribution (“foods are really addictive”) did not differ between groups, $F(2,84)=.05, p=.948, \eta_p^2=.00$, (Table 7.3). However, the rank assigned to the addiction attribution was negatively associated with levels of guilt and dietary concern (guilt: $r=-.334, p=.002$; concern: $r=-.249, p=.020$). This suggests a relationship between higher levels of guilt following eating and rating the ‘addictiveness of the foods’ as a more influential reason for eating. Exploratory analyses revealed that the rank assigned to the ‘addictiveness of the foods’ did not correlate with estimated calorie intake (i.e. prior to the manipulation) ($r=-.129, p=.235$) or with actual calorie intake ($r=-.016, p=.880$). This is important as it suggests that beliefs about the addictiveness of the foods were more closely related to feelings of guilt, than to perceived or actual calorie intake.

7.5.4. Snack selection (hypothesis 3)

The majority of participants (62.1%) selected their most tempting snack to take home with them. The Kruskal-Wallis test indicated that the temptingness of the snack selected differed significantly between conditions, $H(2)=7.16, p=.028$. As predicted, participants in the high-guilt condition selected significantly less tempting snacks than those in the low-guilt condition, $U=265.50, Z=-2.62, p=.009$. Those in

the low-guilt condition chose more tempting snacks than those in the control condition, $U=342.50$, $Z=-1.93$, $p=.053$ (although this only approached significance) (Figure 7.4). However, there was no significant difference between the snack selected in high-guilt and control conditions, $U=357.00$, $Z=-.834$, $p=.404$. There was no association between selected snack rank and self-perceived food addiction ($r_s=-.044$, $p=.682$). Finally, exploratory correlational analysis revealed no association between selected snack rank and self-control ratings ($r_s=-.011$, $p=.461$), dietary concern ($r_s=.137$, $p=.204$), or guilt ($r_s=.038$, $p=.725$).

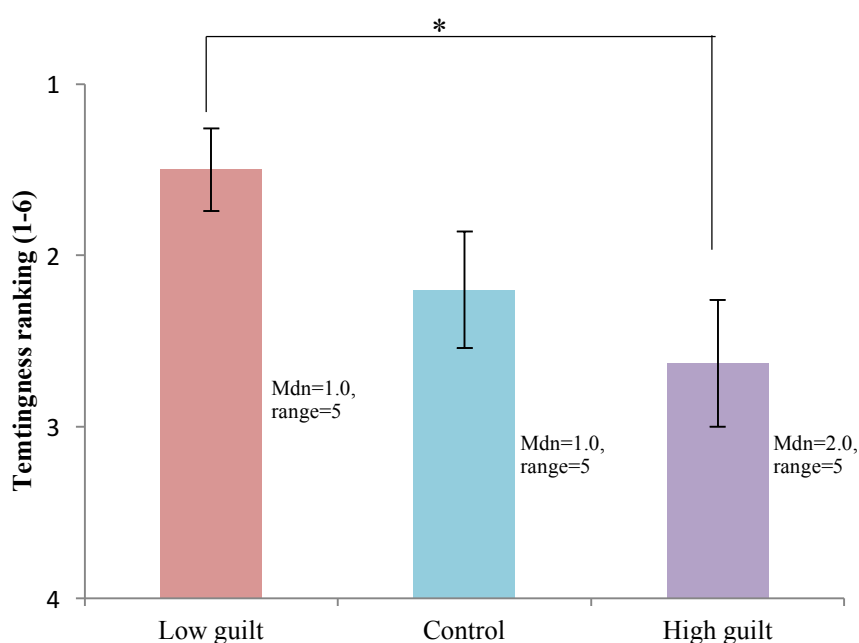


Figure 7.4. Mean temptingness rank (1=most tempting, 6=least tempting) of snack taken in each of the three conditions. Median (mdn) and range values are also provided for each condition. * $p<.01$.

All but three participants attended the follow-up session one week later ($n=84$). Of these, 76 (90.5%) did not consume the snack during the week. Of the 8 people who had consumed the snack, 4 were in the low-guilt condition, 3 were in the control condition, and 1 was in the high-guilt condition. Seven out of the 8 participants had selected their most tempting snack, and one had chosen their third most tempting snack. The majority ($n=5$) of those that had consumed the snack indicated that they ‘Disagree’ or ‘Strongly disagree’ that they were food addicts.

Statistical analyses of these data were not possible due to the low numbers of participants who consumed the snack.

7.6. Discussion

The primary aim of the current study was to investigate the extent to which ‘food addiction’ may be used as a self-serving attribution for eating. Specifically, it was predicted that individuals who were manipulated to have high levels of eating-related guilt (high-guilt condition), would be more likely to label themselves as food addicts (hypothesis 1) and to attribute their eating to the ‘addictiveness’ of the foods (hypothesis 2), than those in low-guilt and control conditions, and that this would be due to higher levels of dietary concern and guilt.

Levels of dietary concern and guilt were highest in the high-guilt condition, and lowest in the low-guilt condition indicating that our manipulation had been successful. However, contrary to our first hypothesis, there was no effect of condition on self-perceived food addiction. There were also no significant positive correlations between self-perceived food addiction and ratings of guilt or dietary concern. Instead, self-perceived food addiction correlated negatively with self-control ratings, and positively with actual calorie intake, and two trait measures of addictive and disinhibited eating (i.e. TFEQ-D and YFAS) that were obtained 1 week following the manipulation. This is consistent with findings from Chapter 3 of the current thesis, in which individuals who perceived themselves as food addicts consumed more food, and scored higher on trait measures of disinhibited eating, than those who did not identify as food addicts.

These findings indicate that a single episode of eating-related guilt is unlikely to prompt ‘food addiction’ attributions. Nonetheless, it remains plausible that the concept of food addiction may be used as a self-serving attribution following more regular and repeated patterns of disinhibited or ‘binge’ eating. Indeed, evidence suggests that binge eating is a highly stigmatized behaviour (Bannon, Hunter-Reel, Wilson, & Karlin, 2009), and thus individuals who regularly engage in disinhibited patterns of eating may be particularly inclined to use the concept of ‘food addiction’ as a means of minimizing perceptions of blame. Future research should investigate this possibility by examining the effects of food addiction explanations for

overeating on feelings of guilt and personal responsibility in those with a propensity for trait overeating, such as in obese or binge eating samples.

Contrary to our second hypothesis, we uncovered no effect of condition on the ‘foods were addictive’ attribution for eating. However, across the entire sample, this attribution was ranked as a more influential reason for eating in those with increased levels of guilt and dietary concern. Furthermore, the rank assigned to the addictiveness attribution was not associated with estimated or actual calorie intake. These findings suggest that external attributions about the addictive potential of certain foods may be more closely related to negative emotions elicited following consumption of these foods, rather than to actual food intake. This is consistent with previous research in which providing external attributions for eating (i.e. emotional eating) was related to increased dietary related concerns, rather than to an actual tendency to engage in emotionally driven eating (Adriaanse et al., 2011). However, as there was no effect of condition on the rank assigned to the ‘addictiveness’ attribution, we are unable to conclude a causal effect of eating-related guilt.

A secondary aim was to examine the *consequences* of eating-related guilt and food addiction attributions on subsequent food choice. Based on previous findings (Ruddock et al., 2016; Chapter 6 of the current this), it was predicted that individuals who were led to feel more guilty and concerned following eating would select less tempting snacks to take home with them, compared to those in low-guilt and control conditions. Further, we explored whether snack selection would be associated with self-perceived food addiction and dietary concern. Specifically, drawing upon our previous findings (Ruddock et al., 2016; Chapter 6), we expected self-perceived food addiction and increased dietary concern to be associated with less tempting snack selection (i.e., this may be because these participants felt concerned about their eating and tried to limit their exposure to tempting foods as a result). We found an effect of condition on snack selection, and this was due to those in the low-guilt condition choosing *more* tempting snacks than those in high-guilt conditions as predicted. However, snack selection did not differ significantly between those in the control and low-guilt conditions (though there was a non-significant trend), or between the control and high-guilt conditions. Furthermore, as there was no relationship between snack selection and self-perceived food addiction, self-control ratings, or measures of guilt and dietary concern, it is not possible to draw any

conclusions regarding the mechanisms behind this effect. This suggests that, in the current study, the effect of condition on snack selection was driven by an alternative mechanism.

One possible explanation for these findings is that perceptions of lower calorie intake in the low-guilt condition may have given participants a ‘license to over-eat’, thus leading to more tempting snack selection. This idea is consistent with recent findings in which participants who were led to believe they had expended more calories during exercise consumed more food during a subsequent ad libitum test meal, than those who were told they had expended fewer calories (McCaig, Hawkins, & Rogers, 2016).

The findings obtained in the current study are partially consistent with findings from Chapter 6, in that the guilt manipulation affected both dietary concern and snack choice. However, in contrast to findings from Chapter 6, snack selection was not associated with dietary concern, or self-perceived food addiction. These discrepant findings may be attributable to increased levels of satiety. In particular, participants in the current study selected a snack to take home with them after consuming a buffet lunch (i.e. when they were satiated). In contrast, in Chapter 6, participants were exposed to foods when they were hungry. Known as the ‘cold-to-hot empathy gap’ (Loewenstein, 1996), previous research has demonstrated that individuals who are satiated (i.e. in a ‘cold’ state) tend to overestimate their ability for self-control compared to when they are hungry (i.e. in a ‘hot’ state) (Nordgren et al., 2009). As such, one possibility is that satiated participants in the current study may have felt particularly confident about their ability to refrain from eating the snack during the week, and this may have exerted greater influence over snack choice than dietary concern or food addiction beliefs. Future research is required to investigate the effect of food addiction beliefs on snack choice under conditions of hunger and satiety.

The current study yields a number of limitations which should be addressed in future research. Firstly, very few participants consumed the snack during the subsequent week, and thus we were unable to explore the effect of condition on snack consumption. While participants were informed that they would be required to keep the snack with them at all times, adherence to this instruction was not

monitored. It is therefore possible that participants may have stored the snack out of reach during the subsequent week, thus minimizing temptation. Future research should aim to overcome this limitation by implementing methods of ensuring that participants adhere to this instruction.

Secondly, it is important to consider that participants in the current study were informed that they had consumed an amount that was relative to their estimated calorie intake. As such, the bogus calorie feedback may have generated between-*subject*, as well as between-*condition*, differences. Specifically, it is possible that feelings of guilt may have varied substantially between participants in the same condition as a result of receiving different calorie feedback. Nonetheless, the decision to provide participants with tailored (rather than consistent) calorie feedback, was taken to ensure that it was always less than (in the low-guilt condition), more than (in the high-guilt condition), or equal to (in the control condition) the amount of calories participants *believed* they had eaten. This may not have been achieved had we provided participants with consistent calorie feedback. As such, providing participants with tailored calorie feedback likely maximized the effectiveness of the manipulation on feelings of guilt. Indeed, no between-group differences were observed with regard to actual or estimated (pre-manipulation) calorie intake, suggesting that the observations made in the current study were due to the guilt manipulation.

Finally, the current study did not take into account participants' dieting goals. This may have been an important factor in the current study, as previous findings suggest that individuals are most likely to provide self-serving attributions for behaviours which are perceived to violate their own personal standards (Eiser & Sutton, 1957; Jellinek, 1960). As such, future research should investigate the possibility that individuals with strict dietary goals may be most likely to provide food addiction attributions following an eating-related guilt induction.

Despite the aforementioned limitations, to our knowledge the current study represents the first to investigate whether the concept of food addiction may be used as a self-serving attribution for eating. In doing so, we provide a novel methodological approach for inducing eating-related guilt that may be useful for future research. While our findings do not fully support the concept of food addiction

as an ‘attribution’ (Rogers & Smit, 2000), we provide correlational evidence to suggest that beliefs about the addictive potential of foods may be more closely related to feelings of guilt than to actual calorie intake. Future research is required to clarify the nature of this relationship (i.e. whether eating-related guilt *causes* attributions about the addictiveness of foods, or alternatively whether eating foods that are perceived as addictive causes guilt), and investigate the possibility that food addiction may be used as a self-serving attribution for those who experience repeated episodes of eating-related guilt.

Chapter 8: General Discussion

8.1. Overview of aims

The recent rise in worldwide rates of obesity is often attributed to an ‘addiction’ to high-calorie, processed foods. Current conceptualisations of food addiction (e.g. Gearhardt et al., 2009) rely upon the DSM criterion for substance dependence. However, the applicability of this criterion to eating behaviour is limited, and Ziauddeen et al. (2012) suggest the need to develop an operational definition of food addiction that is not based upon existing models of substance-dependence. The first aim of the current thesis was therefore to establish a cognitive and behavioural profile of self-perceived food addiction by examining the eating-related cognitions and behaviours of individuals who *perceive* themselves to be ‘addicted’ to food (i.e. self-perceived food addicts). This aim was addressed in Chapters 2-5.

A second aim of the current thesis was to examine the causes and consequences of food addiction *beliefs* from a psychosocial perspective. Specifically, over two studies, Chapter 6 examined whether believing oneself to be a food addict would have a helpful or counterproductive effect on eating habits. Additionally, drawing upon previous suggestions (Rogers & Smit, 2000), Chapter 7 examined whether the concept of food addiction may be used to provide a more socially and personally acceptable attribution for eating-related guilt.

8.2. Establishing a cognitive and behavioural profile of ‘food addiction’

Chapter 2 presents a study which aimed to provide initial insight into the prevalence and perceptions of self-perceived food addiction within a community sample. Of the 210 respondents to this survey, over a quarter ($n=59$) identified as food addicts, 60 per cent ($n=125$) did not perceive themselves to be addicted to food, and 12 per cent ($n=26$) were undecided. The prevalence of self-perceived food addiction did not differ between males and females, but was higher in those with increased BMI and younger age.

Using a qualitative approach, Chapter 2 identified six core behavioural features of ‘self-perceived food addiction’. These included: 1) Reward-driven eating (i.e. eating for psychological factors, rather than physiological hunger), 2) A functional or psychological preoccupation with food, 3) A perceived lack of self-

control around food, 4) Frequent food cravings, 5) Increased weight or an unhealthy diet, and 6) A problem with a specific type of food. Gender differences were observed such that females were more likely than males to associate food addiction with reward-driven eating. Importantly, conceptualisations of food addiction did not differ between self-perceived food addicts and non-addicts; rather, they reported opposite patterns of behaviour (i.e. while self-perceived food addicts reported that they “think about food all the time”, non-addicts reported “little interest in food”). These findings suggest that the concept of ‘food addiction’ may be used, by members of the lay public, as an umbrella term for problematic patterns of eating behaviour.

8.2.1. Food reward, attentional bias, and calorie consumption in self-perceived food addicts

Findings from Chapter 2 suggest that self-perceived food addicts find high-fat foods particularly rewarding, and may consume more calories when satiated, compared to non-addicts. This was empirically explored in Chapter 3. In this study, food reward for two high-fat foods (chocolate and crisps) and two low-fat foods (rice cakes and grapes) was assessed using ratings of desire-to-eat and willingness-to-pay. Participants also completed an operant response task in which they were led to believe that the more they tapped a computer key during a 1-minute period, the more food (i.e. chocolate and grapes) they would receive at the end of the session. Participants completed these measures when hungry and again when satiated. *Ad libitum* intake of chocolate and grapes was also assessed when participants were satiated. Based on findings from Chapter 2, it was predicted that self-perceived food addicts would demonstrate increased food reward (most notably when satiated), and would subsequently consume more calories when given *ad libitum* access to high- and low- fat foods compared to non-addicts. In particular, these differences were expected to be most pronounced towards the high-fat foods.

In partial support of these hypotheses, self-perceived food addicts demonstrated increased food reward (as assessed using desire-to-eat ratings) for both high- and low-fat foods, and in hungry and satiated conditions, compared to non-addicts. Furthermore, as expected, increased *ad libitum* calorie intake was observed in self-perceived food addicts, compared to non-addicts, for the high-fat food (i.e. chocolate), but not for the low-fat food (i.e. grapes). These findings are consistent

with previous research which has demonstrated increased food reward and calorie intake in those who perceive themselves to be addicted to chocolate (Tuomisto et al., 1999; Hetherington & Macdiarmid, 1995; Macdiarmid & Hetherington, 1995). Importantly, these findings were observed despite the fact that very few self-perceived food addicts (12 per cent), fulfilled an established measure of food addiction (i.e. the YFAS, Gearhardt et al., 2009). This suggests that self-perceived food addicts have problematic patterns of eating that may go undetected by an existing measure of addictive eating. Notably, increased calorie intake in self-perceived food addicts was determined by increased dietary disinhibition and diminished restraint, consistent with dual-process theories of overeating and addiction (Appelhans et al., 2009; Weirs et al., 2007).

According to Field et al. (2016), the underlying reward value of a food can be indirectly assessed by examining the effect of a food-cue (i.e. a stimulus that has previously been associated with food receipt) on cognitive responses, such as attention to food cues. As such, Chapter 4 aimed to extend findings from Chapter 3 by examining whether self-perceived food addicts would demonstrate increased attention to chocolate-pictures, relative to neutral pictures (i.e. attentional bias), compared to self-perceived non-addicts. Chapter 4 also investigated whether self-perceived food addiction would moderate the effect of two previously established state determinants of attentional bias; hunger/satiety and the perceived availability of chocolate (Castellanos et al., 2009; Jones et al., 2012). Specifically, it was predicted that the effect of self-perceived food addiction on attentional bias to chocolate-pictures would be most pronounced when participants were satiated, compared to hungry. Furthermore, we hypothesised that the effect of perceived availability on attentional bias would be either increased or decreased in self-perceived food addicts, relative to non-addicts.

Contrary to prediction, self-perceived food addicts did not demonstrate any increased attentional bias to chocolate-pictures, relative to non-addicts. Furthermore, self-perceived food addiction did not moderate the effect of condition (i.e. hungry vs. satiated) or expectancy information on attentional bias to chocolate pictures. In line with previous findings (Field et al., 2011; Jones et al., 2012;), attentional bias to chocolate pictures was greater when participants expected to receive a chocolate

point (i.e. 100% trials), relative to 50% and 0% trials. However, contrary to previous research (e.g. Castellanos et al., 2009) there was no effect of hunger condition.

Instead, post-hoc analyses revealed a key role of desire-to-eat. Specifically, those who had high levels of desire-to-eat chocolate demonstrated increased attentional bias to chocolate pictures relative to those who had low levels of desire-to-eat. There was also an interaction between desire-to-eat and expectancy such that only those with high levels of desire-to-eat demonstrated sensitivity to the expectancy information. Desire-to-eat ratings also provided the only significant predictor of chocolate consumption; measures of attentional bias, hunger, and self-perceived food addiction failed to predict *ad libitum* chocolate intake.

Importantly, consistent with findings from Chapter 3, desire-to-eat ratings were greater in self-perceived food addicts, relative to non-addicts. Thus, while a direct relationship was not observed, these findings suggest that individuals who perceive themselves to be addicted to food may have an increased tendency to develop attentional bias to food cues due to an increased subjective desire-to-eat. Overall, these findings provide support for the suggestion that within-subject state factors, such as desire-to-eat, exert a stronger influence on attentional bias than trait characteristics (Field et al., 2016). They also support previous suggestions that desire-to-eat ratings provide a valid reflection of a food's rewarding value (Rogers & Hardman, 2015).

Finally, it is necessary to consider that, contrary to findings from Chapter 3, Chapter 4 uncovered no significant relationship between self-perceived food addiction and *ad libitum* intake of chocolate. One possibility is that the implementation of an attentional bias task in Chapter 4 (in which participants were presented with repeated exposure to chocolate pictures) may have increased the reward value of chocolate in both self-perceived food addicts and non-addicts, leading to similar intake. Indeed, previous studies have demonstrated increased food consumption following attention to food cues (e.g. Werthmann et al., 2014), albeit inconsistently (e.g. Hardman et al., 2014). Additionally, in Chapter 4, desire-to-eat chocolate ratings remained high following consumption of the savoury meal. This may have masked any relationship between self-perceived food addiction and *ad libitum* chocolate intake.

8.2.1.1. Food liking and hunger in self-perceived food addicts

Food reward is thought to be influenced by an individual's current level of physiological hunger and the extent to which they *like* a food (Berridge et al., 2010; Rogers & Hardman 2015). Based on this theoretical perspective, an aim of Chapter 3 was to investigate whether increased food reward, in self-perceived food addicts, was due to increased liking for the test foods and/or increased hunger. Self-perceived food addicts and non-addicts did not differ with regards to hunger ratings or liking for the test foods. This is analogous to previous findings in which self-identified 'chocolate overeaters' demonstrated increased food reward, despite no differences in liking or hunger (Hetherington & Macdiarmid, 1995). Nonetheless, consistent with findings from Hetherington and Macdiarmid (1995), self-perceived food addicts demonstrated an attenuated decline in liking ratings following consumption of the lunch meal relative to non-addicts. This suggests that satiety may have less of an effect on the hedonic value of a food's taste in self-perceived food addicts. Future research is required to investigate this possibility.

Given that self-perceived food addicts demonstrated no differences in liking or hunger, one possibility is that the increased food reward observed in these individuals may be attributable to other, perhaps *implicit*, processes. As discussed in Chapter 1, increased exposure to a rewarding stimulus, such as food, may increase individuals' 'wanting' for the reward, without increasing 'liking' perceptions (Berridge, 2009). While 'wanting' is difficult to measure directly (Havermans et al., 2011), it may be inferred from the variance in food reward that is unaccounted for by food liking ratings (Rogers & Hardman, 2015). Furthermore, Rogers and Hardman (2015) suggest that hunger influences the rewarding value of a food via a 'wanting' component. As such, given that individual differences in food reward were also not accounted for by hunger variation, it is possible that increased food reward in self-perceived food addicts may be attributable to differences in implicit 'wanting'.

8.2.1.2. Does food addiction reflect a 'unique' set of eating behaviours?

Findings from Chapters 2, 3 and 4 suggest that self-perceived food addicts find food particularly rewarding and may be particularly likely to overeat high-fat foods. However, it is important to consider the extent to which these behaviours reflect a *unique* set of aberrant eating patterns, or whether they simply reflect already-established patterns of eating such as dietary disinhibition or restraint (Long

et al., 2015). This was addressed in Chapter 3 which showed that self-perceived food addiction did *not* account for any additional variance in food reward and calorie intake beyond that captured by measures of dietary disinhibition and restraint. This suggests that members of the lay public may use the term ‘food addiction’ to refer to patterns of over-eating that are already captured by established trait measures of dietary behaviour. Notably, the combination of increased disinhibition and low dietary restraint suggest that self-perceived food addicts demonstrate patterns of overeating that are reflective of dual-process models of eating behaviour (Appelhans, 2009). Specifically, dual-process theories suggest that overeating and weight gain are caused by increased food reward and diminished self-control (Appelhans, 2009; Nederkoorn et al., 2010).

From a clinical standpoint, this raises questions regarding the extent to which ‘food addiction’ warrants a separate diagnostic criterion to that of ‘Binge Eating Disorder’, which is characterised by severe bouts of disinhibited eating, within future editions of the DSM. To address this, it is important to identify the key differences between binge eating disorder and behaviours which may characterise addictive eating patterns. Indeed, qualitative reports suggest that binge eating and food addiction represent two dissociable constructs such that individuals *without* binge eating disorder may experience their overeating tendencies as an ‘addiction’, and not all people with binge eating disorder report an ‘addiction’ to food (Cassin & von Ranson, 2007; Curtis & Davis, 2014). Findings from Chapter 2 provide further insight into this issue. Specifically, members of the lay public did not equate food addiction to the secretive and planned ‘binge’ episodes, and subsequent caloric restriction, that characterise binge eating disorder (Palmberg et al., 2014; Phillips et al., 2016; Woods et al., 2010). Furthermore, Hebebrand et al. (2014) suggest that addictive patterns of eating may take the form of continual ‘grazing’ throughout the day, rather than isolated binges. Indeed, increased snacking has been associated with eating pathology and poorer weight-loss outcomes following bariatric surgery (Nicalau et al., 2015; Robinson et al., 2014; Sheets et al., 2015).

8.2.2. A novel approach to the assessment of addictive eating

An alternative possibility is that food addiction may represent a more severe form of disinhibited or ‘uncontrolled’ eating to that observed in binge eating disorder (Vainik et al., 2015; Davis et al.). In support of this, Gearhardt et al. (2012)

documented more frequent binge eating episodes and increased psychopathology, in individuals who are co-morbid for binge eating disorder and food addiction (as assessed using the YFAS; Gearhardt et al., 2009), relative to those with binge eating disorder *without* food addiction. However, as previously discussed (Chapters 1 and 5), the validity of the YFAS for the assessment of food addiction is limited by its reliance upon the DSM criteria for substance dependence (Hebebrand et al., 2014; Ziauddeen et al., 2012). As such, in order to provide further validation of the food addiction concept, it is necessary to develop an assessment tool for addictive patterns of eating that is not constrained by existing conceptualisations of addictive behaviour.

This was addressed in Chapter 5 which presents the development of the Addictive Eating Behaviour Scale. This scale assesses the presence of behaviours that are commonly associated with addictive patterns of eating, and is comprised of two underlying factors which reflect overeating/weight gain, and low food-related self-control/an unhealthy diet. Crucially, the AEBS was able to account for a significant proportion of variance in BMI *above* that predicted by the Binge Eating Scale (BES; Gormally et al., 1982) and YFAS (Gearhardt et al., 2009). This is important given that both of these scales are thought to reflect patterns of eating that are indicative of a ‘food addiction’ (Davis et al., 2011; Gearhardt et al., 2009), and also suggests that the AEBS captures patterns of eating that are distinct from those assessed by existing measures. Further research is required to provide behavioural validation of the scale. For example, it will be important to establish the extent to which the AEBS is able to account for variation in food reward and *ad libitum* food intake over and above existing measures of disinhibited eating and dietary restraint.

8.3. What are the causes and consequences of food addiction beliefs?

8.3.1. Food addiction as a helpful label

The second aim of the current thesis was addressed in Chapters 6 and 7. Specifically, Chapter 6 presents two studies which explore the consequences of perceiving oneself to be a food addict on subsequent eating behaviour. In Study 1, participants were led to believe that they had scored either high (high-addiction condition) or low (low-addiction condition) on an ostensible measure of food addiction. They were then asked to taste and rate chocolate and crisps, and were told

that they could eat as much of the foods as they liked. Results revealed that those in the high-addiction condition consumed significantly less food than those in the low-addiction condition. These findings were replicated and extended in Study 2 which provided insight into the direction and mechanisms behind these effects.

Specifically, with the addition of a control group, who were led to believe they had 'average' levels of food addiction (i.e. average-addiction condition), Study 2 found that calorie intake was *decreased* in the high-addiction, relative to in the low- and average-addiction conditions. Furthermore, this was driven by increased dietary concern, in the high-addiction condition, and a subsequent reduction in the amount of time participants spent exposed to the foods during the *ad libitum* taste task. These findings suggest that perceiving oneself to be a food addict may help to encourage people to avoid exposing themselves to tempting foods, at least in the short term.

These findings extend Nordgren et al.'s (2009) 'restraint bias' theory, which proposes that holding unrealistically high expectations about one's capacity for self-control may be detrimental for health behaviours. In a series of experiments, Nordgren et al. (2009) showed that individuals who perceived themselves as having high levels of self-control selected more tempting snacks, and exposed themselves to more tempting smoking scenarios, compared to those who held low self-control beliefs. Similarly, Jones et al. (2012) found that individuals who were told that they had scored high on measures of self-control consumed more alcohol during an *ad libitum* taste task, than those who were led to believe that they had low levels of self-control. However, the interpretation of these previous findings is somewhat limited by the absence of a control group. In Chapter 6 (Study 2), we provide evidence that such effects may be attributable to a *helpful* effect of low self-control beliefs (rather than a *harmful* effect high self-control beliefs, as previously suggested, Nordgren et al., 2009).

Chapter 7 aimed to further extend these findings by examining the effect of eating-related guilt on food addiction attributions and snack choice. In this study, it was hypothesised that individuals who were led to feel guilty following eating (high-guilt condition) would be more likely to perceive themselves to be food addicts, and would select less tempting snacks to take home with them (when given a monetary incentive to return the snack one week later), relative to those in low-guilt and control conditions. Results revealed a main effect of condition on snack selection,

which was driven by those in the *low*-guilt condition selecting *more* tempting snacks compared to those in the high-guilt condition. However, contrary to findings from Chapter 6, snack selection was not associated with self-perceived food addiction or ratings of dietary concern.

One possibility is that perceptions of low calorie intake, in the low-guilt condition, may have led participants to feel that they had a ‘license to overeat’, thus prompting the selection of more tempting snacks. Furthermore, due to the effect of satiety, participants in Chapter 7 may have felt particularly confident in their ability to refrain from eating the snack during the week, and this may have exerted greater influence over snack choice than dietary concern or food addiction beliefs. Future research is required to investigate the effect of food addiction beliefs on snack choice under conditions of hunger and satiety.

8.3.2. Food addiction as a self-serving attribution

A final consideration addressed in the current thesis was whether ‘food addiction’ may be used as a self-serving attribution for overeating. This follows previous suggestions that failures to restrict one’s intake of highly palatable but unhealthy (i.e. naughty but nice) foods may be attributed to a ‘food addiction’ in an attempt to alleviate feelings of personal responsibility and eating-related guilt. Indeed, the concept of ‘addiction’ is thought to imply that one’s behaviours are the result of a physiological inevitability, and thus may help to portray the overeater or drug user as a ‘helpless victim of disease’ (Davies, 2013; Rogers & Smit, 2000).

To address this suggestion, Chapter 7 examined whether participants who were led to feel guilty following eating (high-guilt condition) would be more likely to identify as ‘food addicts’ and to attribute their eating to the ‘addictiveness of the foods’, compared to those in low-guilt and control conditions. To manipulate eating-related guilt, participants were asked to estimate how many calories they had consumed during a buffet lunch. Those in the high-guilt condition were then led to believe that they had consumed more calories than they had estimated, and more than previous (bogus) participants. Those in the low-guilt and control conditions were led to believe that they had eaten less than, or roughly the same as, their estimate and relative to previous participants, respectively. A subsequent manipulation check confirmed that the guilt manipulation had been successful (i.e.

levels of guilt and dietary concern were highest in the high-guilt condition, and lowest in the low-guilt condition). Self-perceived food addiction and beliefs about the addictiveness of the foods were then assessed.

Contrary to our first hypothesis, there was no effect of the guilt condition on self-perceived food addiction. There were also no significant positive correlations between self-perceived food addiction and ratings of guilt or dietary concern. Instead, self-perceived food addiction correlated positively with actual calorie intake, food-related self-control ratings, and two trait measures of addictive and disinhibited eating that were obtained one week following the manipulation. This is consistent with findings from Chapter 3 in which individuals who perceived themselves to be food addicts consumed more food during *ad libitum* access, and scored higher on trait measures of disinhibited eating, compared to those who did not identify as food addicts. Taken together, these findings do not support the suggestion that the concept of food addiction is used to minimise feelings of guilt following a single episode of overeating. Nonetheless, as discussed in Chapter 7, it remains plausible that the concept of food addiction may be used as a self-serving attribution following more regular and repeated patterns of disinhibited or binge eating and this should be investigated in future research.

Contrary to our second hypothesis, participants in the high-guilt condition were not more likely to attribute their eating to the foods' 'addictive' qualities than those in the low-guilt and control conditions. However, this attribution was associated with eating-related guilt ratings. Specifically, those with increased levels of guilt assigned a lower rank (indicative of greater influence) to the 'foods were really addictive' attribution. Importantly, the rank assigned to the addictiveness attribution was not associated with estimated or actual calorie intake. Taken together, these findings suggest that attributions about the addictive potential of certain foods may be more closely related to negative emotions elicited following consumption of these foods, than to actual food intake. This may reflect attempts to diminish feelings of guilt or diffuse personal responsibility. However, as there was no effect of condition (i.e. high-guilt, low-guilt, control) on the 'addictiveness of the foods' attribution, it is not possible to conclude a causal relationship from these findings. Indeed, an alternative explanation is that perceiving the foods as more addictive may

have led participants to experience higher levels of guilt than those who did not perceive the foods as addictive.

8.4. Theoretical implications and directions for future research

This thesis explored the manifestations of *self-perceived* food addiction, and was not constrained by the application of a substance-dependence framework to eating behaviours (see Chapter 1). Thus in doing so, this thesis yields several important theoretical and practical implications. In particular, findings suggest that food addiction within the lay public is identifiable through several core behaviours, and is not generally associated with physical symptoms of withdrawal or tolerance that are characteristic of substance dependence. Furthermore, unlike substance-dependence, food addiction does not necessarily entail a ‘giving up’ of important activities, or an ‘impairment to daily functioning’. These findings suggest that people’s beliefs coincide with the conceptualisation of food addiction as a behavioural, rather than substance-based, addiction (Hebebrand et al., 2014). Importantly, however, the results discussed in the current thesis were obtained from non-clinical populations, and thus future research is required to explore food addiction beliefs in obese and binge-eating samples.

Furthermore, in order for compulsive patterns of overeating to be recognised as an ‘addictive’ disorder, it is necessary for future research to provide further validation of the food addiction concept. One approach may be to assess the extent to which individuals who score highly on the AEBS (Chapter 5) demonstrate eating patterns that adhere to core features of addiction, such as habitual food-seeking in response to food cues. It would also be necessary to establish whether such patterns of eating are distinct from those incorporated within the ‘Eating Disorders’ category of the DSM, such as binge eating disorder. Further insight into this issue may be achieved by conducting a qualitative exploration of eating experiences in those who score high and low on the AEBS. Additionally, research should aim to validate the AEBS within clinical populations, such as those with morbid obesity and/or binge eating.

Findings from the current thesis yield important implications for dietary intervention strategies for those with addictive eating. In particular, findings from Chapters 2 and 3 suggest that self-perceived food addicts may be characterised by

increased food reward and diminished dietary restraint, consistent with dual-process models of overeating. As such, these individuals may benefit from a combination of dietary interventions which aim to strengthen self-control, and minimise temptation for high-calorie foods. This two-dimensional approach to dietary interventions is consistent with the framework proposed by Appelhans et al. (2016) which distinguishes between interventions which aim to increase self-control (i.e. temptation *resistance* strategies), and those which aim to minimise temptation (i.e. temptation *prevention* strategies). For example, ‘choice restriction’ interventions prevent food temptation by restricting the availability of foods to healthier alternatives (Hanks, Just, & Wansink, 2013), while ‘commitment by punishment’ interventions aim to increase self-control by enforcing negative outcomes (e.g. a financial penalty) following the consumption of unhealthy foods (Halpern et al., 2012). Findings from the current thesis suggest that a combination of these techniques may be particularly beneficial for those with addictive patterns of eating, and future research should investigate this possibility.

Findings from Chapter 6 raise the possibility that food addiction messages could be usefully incorporated within dietary interventions. In particular, perceiving oneself to be a food addict may encourage individuals to avoid exposing themselves to tempting situations, such as the supermarket confectionary aisle or the buffet table at a party. These findings support methods adopted by overeating self-help groups (e.g. Overeaters Anonymous) in which members are encouraged to recognise their ‘addiction’ to food and to avoid exposing themselves to problematic foods (Russell-Mayhew, 2010; Ronel & Libman, 2003). Indeed, this approach is thought to minimise feelings of blame, while empowering individuals to take responsibility for their recovery (Ronel & Libman, 2003).

However, before drawing conclusions about the beneficial effects of food addiction beliefs, it is necessary for future research to establish the *longer-term* effects on eating behaviour. Importantly, contrary to findings from Chapter 6, self-perceived food addiction was associated with *increased* calorie intake in Chapter 3 (i.e. participants who perceived themselves as food addicts consumed more calories from chocolate than self-perceived non-addicts). In order to reconcile these apparent disparate findings, Figure 8.1 proposes a cyclical relationship between self-perceived food addiction, attempts at restriction, and increased calorie consumption.

Specifically, it is proposed that perceiving oneself to be a food addict may initially encourage individuals to abstain from eating their ‘problem’ foods. However, as demonstrated by previous research (e.g. Warren & Cooper, 1988), such attempts at dietary restriction may strengthen cravings and eventually lead to the over consumption of the forbidden food. Indeed, Chapter 3 demonstrated increased desire-to-eat and calorie consumption in self-perceived food addicts, relative to non-addicts. In line with previous suggestions (Rogers & Smit, 2000), this may reinforce the perception of oneself as a ‘food addict’ thus prompting further attempts at dietary restriction. Future research is required to explore this potential self-perpetuating relationship between food addiction beliefs, restriction and high-calorie food consumption. In particular, it would be informative to extend findings from Chapter 6 by examining the longer-term effects of manipulating food addiction beliefs on intentions to restrict food, and the effects of this on food cravings and subsequent consumption.

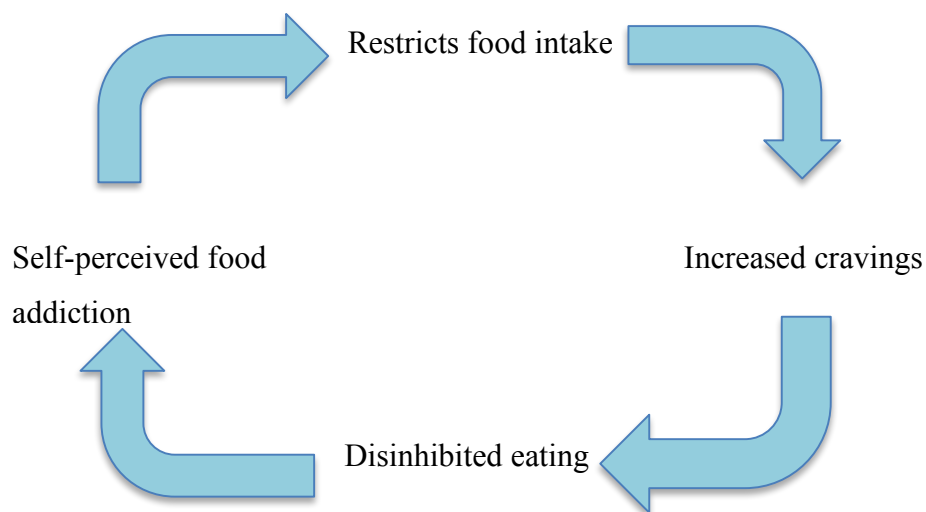


Figure 8.1. A proposed self-perpetuating relationship between self-perceived food addiction and food intake. Belief that one is a food addict leads to an initial restriction of high-calorie or unhealthy foods. This restriction increases cravings and eventually leads to overconsumption of restricted foods. This may reinforce perceptions of oneself as a ‘food addiction’.

8.5. Limitations

The current thesis yields a number of methodological limitations that should be addressed in future research. Firstly, the experimental studies described in Chapters 3,4,6 and 7, used a female-only sample, and thus findings provide limited applicability to male populations. Nonetheless, as these were preliminary studies, it was necessary to minimise the effects of individual variation. Indeed, gender differences have been observed with regards to the prioritisation of health behaviours (Wardle et al., 2004), and Chapter 2 documented different conceptualisations of food addiction in males and females, such that females were more likely than males to associate food addiction with an increased tendency to engage in reward-driven eating. As such, future research is required to examine patterns of addictive eating in males. Furthermore, given that the development of the AEBS was based upon a *predominantly* female sample (83 per cent), future research should aim to validate the scale within a male population.

Secondly, the studies described in the current thesis included participants who were derived from community samples. Furthermore, the majority of these participants were of normal weight. It is therefore possible that the behavioural profile of ‘addictive eaters’ in overweight or binge eating samples may differ from that established in the current thesis. Nonetheless, given that addictive eating is considered distinct from binge eating or obesity (Davis, 2013), and that many individuals in community samples perceive themselves as ‘food addicts’ (e.g. Hardman et al., 2015), it was necessary to explore the determinants and consequences of these beliefs within *non-clinical* samples. Future research is required to extend these findings to clinical samples. In particular, it will be important to provide validation of the AEBS within obese and binge eating populations, and establish the eating behaviours and personality traits of those who score high on this measure.

Finally, it is important to consider the choice of test foods used in the experiments described in the current thesis. In Chapter 3, food reward and *ad libitum* intake were compared between high-fat (chocolate and crisps) and low-fat foods (rice cakes and grapes). Similarly, in Chapter 4, attentional bias was examined towards a high-fat/sugar food (i.e. chocolate). These foods were selected on the basis

of findings obtained from Chapter 2, in which food addiction was associated with ‘problems’ controlling intake of foods high fat and/or sugar. Likewise, Schulte et al. (2015) found that low-fat foods, such as plain crackers and fruit, were considered less addictive than high-fat foods, such as chocolate and crisps. Nonetheless, it is possible that differential findings may have been obtained had we tailored test foods to each participant’s ‘problem’ food/s. This may have yielded the expected food type by group interaction on food reward that was expected in Chapter 3. Additionally, self-perceived food addicts may demonstrate increased attentional bias to pictures of their specific problem food (rather than to generic pictures of chocolate that were used in Chapter 4). Future research is required to explore these possibilities.

8.6. Overall conclusion

By exploring the eating-related behaviours and cognitions of ‘self-perceived food addicts’, the current thesis provided insight into the manifestations of addictive eating in a way that was not constrained by existing substance-dependence models of addiction. Findings suggest that food addiction may be identifiable through several core behaviours indicative of increased food reward and diminished food related self-control. The Addictive Eating Behaviour Scale (AEBS) provides a novel tool for the assessment of such behaviours, and thus should be incorporated within future research to assess the validity of the food addiction concept.

From a psychosocial perspective, findings from the current thesis do not support suggestions that the concept of ‘food addiction’ is used to provide a self-serving attribution for eating-related guilt. However, future research should examine the possibility that the concept of food addiction provides a more personally acceptable attribution for *longer*-term patterns of aberrant eating. In addition, it would be necessary to reconcile findings of increased calorie intake and dietary disinhibition in self-perceived food addicts (Chapters 3 and 7), with those which suggest that food addiction beliefs are *helpful* for those attempting to minimise intake of unhealthy but palatable foods (Chapter 6). To address this, future research should examine the longer-term consequences of food addiction beliefs on food cravings and subsequent consumption.

References

- Aberman, J. E., & Salamone, J. D. (1999). Nucleus accumbens dopamine depletions make rats more sensitive to high ratio requirements but do not impair primary food reinforcement. *Neuroscience*, *92*(2), 545–552.
- Abrantes, A. M., Strong, D. R., Lejuez, C. W., Kahler, C. W., Carpenter, L. L., Price, L. H., ... Brown, R. A. (2008). The role of negative affect in risk for early lapse among low distress tolerance smokers. *Addictive Behaviors*, *33*, 1394–1401.
- Acerbo, M. J., & Johnson, A. K. (2011). Behavioral cross-sensitization between DOCA-induced sodium appetite and cocaine-induced locomotor behavior. *Pharmacology, Biochemistry, and Behavior*, *98*(3), 440–448.
- Adriaanse, M.A., Prinsen, S., de Witt Hubeer, J.C., de Ridder, D.T.D., Evers, C. (2016). ‘I ate too much so I must have been sad’: Emotions as a confabulated reason for overeating. *Appetite*, *103*, 318-323.
- Adriaanse, M. A., Ridder, D. T. D. De, & Evers, C. (2011). Emotional eating: Eating when emotional or emotional about eating? *Psychology and health*, *26*(1), 23–39.
- Adriaanse, M. A., Weijers, J., De Ridder, D. T. D., De Witt Huberts, J., & Evers, C. (2014). Confabulating reasons for behaving bad: The psychological consequences of unconsciously activated behaviour that violates one’s standards. *European Journal of Social Psychology*, *44*(3), 255–266.
- Almiron-Roig, E., Palla, L., Guest, K., Ricchiuti, C., Vint, N., Jebb, S. A., et al. (2013). Factors that determine energy compensation. A systematic review of preload studies. *Nutrition Reviews*, *71*, 458–473.
- Altfas, J.R. (2002). Prevalence of Attention deficit/hyperactivity disorder among adults in obesity treatment. *BMC Psychiatry*, *2*, 1-8.
- Anestis, M. D., Selby, E. A., Fink, E. L., & Joiner, T. E. (2007). The Multifaceted Role of Distress Tolerance in Dysregulated Eating Behaviors. *International Journal of Eating Disorders*, *40*(8), 718–726.
- Appelhans, B.M. (2009). “Neurobehavioral inhibition of reward-driven feeding: implications for dieting and obesity.” *Obesity*, *17*(4), 622–624.

- Appelhans, B. M., French, S. A., Pagoto, S. L., & Sherwood, N. E. (2016). Managing temptation in obesity treatment: a neurobehavioral model of intervention strategies. *Appetite*, *96*, 268–279.
- Appelhans, B. M., Woolf, K., Pagoto, S. L., Schneider, K. L., Whited, M. C., & Liebman, R. (2011). Inhibiting food reward: delay discounting, food reward sensitivity, and palatable food intake in overweight and obese women. *Obesity*, *19*(11), 2175–2182.
- Arase, K., Fisler, J. S., Shargill, N. S., York, D. A., & Bray, G. A. (1988). Intracerebroventricular infusions of 3-OHB and insulin in a rat model of dietary obesity. *American Journal of Physiology*, *255*(6), 974–981.
- Arbuckle, J. L. (2013). *Amos 22 user's guide*. Chicago, IL: SPSS Inc.
- Arnou, B., Kenardy, J., & Agras, W. S. (1995). The Emotional Eating Scale: The Development of a Measure to Assess Coping with Negative Affect by Eating. *International Journal of Eating Disorder*, *18*(1), 79–90.
- Aston-Jones, G., Smith, R. J., Moorman, D. E., & Richardson, K.A. (2009). Role of lateral hypothalamic orexin neurons in reward processing and addiction. *Neuropharmacology*, *56*, 112–121.
- Avena, N. M., & Gold, M. S. (2010). Food and Addiction – Sugars, Fats and Hedonic Overeating. *Addiction*, *106*, 1214–1215.
- Avena, N. M., Gold, J. A., Kroll, C., & Gold, M. S. (2012). Further Developments in the Neurobiology of Food and Addiction: Update on the state of the science. *Nutrition*, *28*(4), 341–343.
- Avena, N. M., Rada, P., & Hoebel, B. G. (2008). Evidence for Sugar Addiction: Behavioral and neurochemical effects of intermittent, excessive sugar intake. *Neuroscience and Biobehavioral Reviews*, *32*, 20–39.
- Avena, N. M., Rada, P., Moise, N., & Hoebel, B. G. (2006). Sucrose Sham Feeding on a Binge Schedule Releases Accumbens Dopamine Repeatedly and Eliminates the Acetylcholine Satiety Response. *Neuroscience*, *139*, 813–820.
- Azar, M. R., Ahmed, S. H., Lintz, R., Gutierrez, T., Stinus, L., & Koob, G. F. (2004). A non-invasive gating device for continuous drug delivery that allows control over the timing and duration of spontaneous opiate withdrawal, *135*, 129–135.

- Babbs, R. K., Wojnicki, F. H. E., & Corwin, R. L. W. (2012). Assessing binge eating. An analysis of data previously collected in bingeing rats. *Appetite*, 59(2), 478–482.
- Bakshi, V., & Kelley, A. (1993). Feeding Induced by Opioid Stimulation of the Ventral Striatum: Role of Opiate Receptor Subtypes. *The Journal of Pharmacology and Experimental Therapeutics*, 265(3), 1253–1260.
- Balodis, I. M., Kober, H., Worhunsky, P. D., White, M. a, Stevens, M. C., Pearlson, G. D., ... Potenza, M. N. (2013). Monetary reward processing in obese individuals with and without binge eating disorder. *Biological Psychiatry*, 73(9), 877–886.
- Bannon, K. L., Hunter-Reel, D., Wilson, G. T., & Karlin, R. A. (2009). The effects of causal beliefs and binge eating on the stigmatization of obesity. *International Journal of Eating Disorders*, 42(2), 118–124.
- Barrett, S. P., Boileau, I., Okker, J., Pihl, R. O., & Dagher, A. (2004). The hedonic response to cigarette smoking is proportional to dopamine release in the human striatum as measured by positron emission tomography and [11C]raclopride. *Synapse*, 54(2), 65–71.
- Barry, C. L., Brescoll, V. L., Brownell, K. D., & Schlesinger, M. (2009). Obesity metaphors: How beliefs about the causes of obesity affect support for public policy. *Milbank Quarterly*, 87(1), 7–47.
- Bart, G., Kreek, M.J., Ott, J., LaForge, K.S., Proudnikov, D., Pollak, L. et al. (2005). Increased attributable risk related to a functional mu-opioid receptor gene polymorphism in association with alcohol dependence in central Sweden. *Neuropsychopharmacology*, 30, 417–422.
- Bassareo, V., & Di Chiara, G. (1999). Differential responsiveness of dopamine transmission to food-stiumli in nucleus accumbens shell/core compartments, *Neuroscience*, 89(3), 637–641.
- Bassareo, V., Musio, P., & Di Chiara, G. (2011). Reciprocal responsiveness of nucleus accumbens shell and core dopamine to food- and drug-conditioned stimuli. *Psychopharmacology*, 214(3), 687–697.
- Batterink, L., Yokum, S., and Stice, E. (2010). Body mass correlates inversely with inhibitory control in response to food among adolescent girls: an fMRI study. *Neuroimage*, 52, 1696–1703.

- Bégin, C. (2012). Does food addiction distinguish a specific subgroup of overweight/obese overeating women? *Health, 4*(12), 1492–1499.
- Bellisle, F. (2009). Assessing various aspects of the motivation to eat that can affect food intake and body weight control. *Encephale, 35*(2), 182-185.
- Bentler, P. M. (1990) Comparative fit indexes in structural models. *Psychological Bulletin, 107*, 238–246.
- Benton, D. (2010). The plausibility of sugar addiction and its role in obesity and eating disorders. *Clinical Nutrition, 29*(3), 288–303.
- Berridge, K. C. (2009). “Liking” and “wanting” food rewards: brain substrates and roles in eating disorders. *Physiology & Behavior, 97*(5), 537–550.
- Berridge, K. C., & Robinson, T. E. (2003). Parsing reward. *Trends in Neurosciences, 26*(9), 507–13.
- Berridge, K. C., Ho, C. Y., Richard, J. M., & DiFeliceantonio, A. G. (2010). The tempted brain eats: Pleasure and desire circuits in obesity and eating disorders. *Brain Research, 1350*, 43-64.
- Berthoud, H. R. (2004). Mind versus metabolism in the control of food intake and energy balance. *Physiology & Behavior, 81*(5), 781–793.
- Bird, S.P., Murphy, M., Bake, T., Albayrak, O., & Mercer, J.G. (2013). Getting science to the citizen – ‘food addiction’ at the British Science Festival as a case study of interactive public engagement with high profile scientific controversy. *Obesity Facts, 6*, 103–108.
- Blissett, J., Haycraft, E., & Farrow, C. (2010). Inducing preschool children’s emotional eating: Relations with parental feeding practices. *American Journal of Clinical Nutrition, 92*(2), 359–365.
- Bocarsly, M. E., Berner, L. A., Hoebel, B. G., & Avena, N. M. (2011). Rats that binge eat fat-rich food do not show somatic signs or anxiety associated with opiate-like withdrawal: Implications for nutrient-specific food addiction behaviors. *Physiology and Behavior, 104*(5), 865–872.
- Boggiano, M.M., Artiga, A.I., Pritchett, C.E., Chandler-Laney, P.C., Smith, M.L., et al. (2007). High intake of palatable food predicts binge-eating independent of susceptibility to obesity: an animal model of lean vs obese binge-eating and obesity with and without binge-eating. *International Journal of Obesity, 31*, 1357–1367.

- Boggiano, M. M., Burgess, E. E., Turan, B., Soleymani, T., Daniel, S., Vinson, L. D., ... Morse, A. (2014). Motives for eating tasty foods associated with binge-eating. Results from a student and a weight-loss seeking population. *Appetite, 83*, 160–166.
- Borgland, S. L., Taha, S. A., Sarti, F., Fields, H. L., & Bonci, A. (2006). Orexin A in the VTA is critical for the induction of synaptic plasticity and behavioral sensitization to cocaine. *Neuron, 49*(4), 589–601.
- Boutrel, B., Kenny, P. J., Specio, S. E., Martin-Fardon, R., Markou, A., Koob, G. F., & de Lecea, L. (2005). Role for hypocretin in mediating stress-induced reinstatement of cocaine-seeking behavior. *Proceedings of the National Academy of Sciences of the United States of America, 102*(52), 19168–19173.
- Braet, C., & Crombez, G. (2003). Cognitive interference due to food cues in childhood obesity. *Journal of Clinical Child and Adolescent Psychology, 32*(1), 32-39.
- Brandon, T. H., Herzog, T. A., Juliano, L. M., Irvin, J. E., Lazev, A. B., & Simmons, V. N. (2003). Pretreatment task persistence predicts smoking cessation outcome. *Journal of abnormal psychology, 112*(3), 448-456.
- Braun, V., & Clarke, V. (2006). Using thematic analysis in psychology. *Qualitative Research in Psychology, 3*, 77–101.
- Brignell, C., Griffiths, T., Bradley, B. P., & Mogg, K. (2009). Attentional and approach biases for pictorial food cues. Influence of external eating. *Appetite, 52*(2), 299–306.
- Brown, R. A., Lejuez, C.W., Kahler, C.W., & Strong, D. R. (2002). Distress tolerance and duration of past smoking cessation attempts. *Journal of Abnormal Psychology, 111*, 180–185.
- Brunner, L., Nick, H.P., Cumin, F., Chiesi, M., Baum, H-P., Whitebread, S., Stricker-Krongrad, A., & Levens, N. (1997). Leptin is a physiologically important regulator of food intake. *International Journal of Obesity, 21*, 1152-1160.
- Brunstrom, J. M., & Rogers, P. J. (2009). How many calories are on our plate? Expected fullness, not liking, determines meal-size selection. *Obesity, 17*(10), 1884–1890.

- Bulik, C. M., & Brinded, E. C. (1994). The effect of food deprivation on the reinforcing value of food and smoking in bulimic and control women. *Physiology and Behavior, 55*, 665–672.
- Burger, K. S., & Stice, E. (2014). Neural responsivity during soft drink intake, anticipation, and advertisement exposure in habitually consuming youth. *Obesity, 22*(2), 441–50.
- Burgess, E. E., Turan, B., Lokken, K. L., Morse, A., & Boggiano, M. M. (2014). Profiling motives behind hedonic eating. Preliminary validation of the Palatable Eating Motives Scale. *Appetite, 72*, 66–72.
- Burmeister, J. M., Hinman, N., Koball, A., Hoffmann, D. A., & Carels, R. A. (2013). Food addiction in adults seeking weight loss treatment. Implications for psychosocial health and weight loss. *Appetite, 60*, 103–110.
- Butland, B., Jebb, S., Kopelman, P., McPherson, K., Thomas, S., Mardell, J., & Parry, V. (2007). *Tackling obesities: future choices-project report* (Vol. 10, p. 17). London: Department of Innovation, Universities and Skills.
- Calitri, R., Pothos, E. M., Tapper, K., Brunstrom, J. M., & Rogers, P. J. (2010). Cognitive biases to healthy and unhealthy food words predict change in BMI. *Obesity, 18*(12), 2282–2287.
- Carelli, R. M., Ijames, S. G., & Crumling, A. J. (2000). Evidence that separate neural circuits in the nucleus accumbens encode cocaine versus “natural” (water and food) reward. *The Journal of Neuroscience, 20*(11), 4255–4266.
- Carnell, S., Cooke, L., Cheng, R., Robbins, A., & Wardle, J. (2011). Parental feeding behaviours and motivations. A qualitative study in mothers of UK pre-schoolers. *Appetite, 57*(3), 665–673.
- Carroll, M. E., Anderson, M. M., & Morgan, A. D. (2007). Regulation of intravenous cocaine self-administration in rats selectively bred for high (HiS) and low (LoS) saccharin intake. *Psychopharmacology, 190*, 331–341.
- Carter, A., Hendrikse, J., Lee, N., Yücel, M., Verdejo-Garcia, A., Andrews, Z., & Hall, W. (2016). The Neurobiology of “Food Addiction” and its Implications for Obesity Treatment and Policy. *Annual Reviews of Nutrition, 36*, 105–128.

- Carter, B. L., & Tiffany, S. T. (2001). The cue-availability paradigm. The effects of cigarette availability on cue reactivity in smokers. *Experimental and Clinical Psychopharmacology*, *9*(2), 183–190.
- Carver, C. S., & White, T. L. (1994). Behavioral Inhibition, Behavioral Activation, and Affective Responses to Impending Reward and Punishment: The BIS/BAS Scales. *Journal of Personality and Social Psychology*.
- Cassin, S. E., & von Ranson, K. M. (2007). Is binge eating experienced as an addiction? *Appetite*, *49*(3), 687–90.
- Castellanos, E. H., Charboneau, E., Dietrich, M. S., Park, S., Bradley, B. P., Mogg, K., & Cowan, R. L. (2009). Obese adults have visual attention bias for food cue images: Evidence for altered reward system function. *International Journal of Obesity*, *33*(9), 1063-1073.
- Castellanos, E. H., Charboneau, E., Dietrich, M. S., Park, S., Bradley, B. P., Mogg, K., & Cowan, R. L. (2009). Obese adults have visual attention bias for food cue images: Evidence for altered reward system function. *International Journal of Obesity*, *33*(9), 1063–1073.
- Cattell, R. B. (1966). The scree test for the number of factors. *Multivariate Behavioural Research*, *1*(2), 245–276.
- Celis-Morales, C., Lara, J., & Mathers, J. C. (2015). Personalising nutritional guidance for more effective behaviour change. *Proceedings of the Nutrition Society*, *74*(02), 130-138.
- Channon, S., & Hayward, A. (1990). The effect of short-term fasting on processing of food cues in normal subjects. *International Journal of Eating Disorders*, *9*, 447–452.
- Cicchetti, D.V. (1994). Guidelines, Criteria, and Rules of Thumb for Evaluating Normed and Standardized Assessment Instruments in Psychology. *Psychological Assessment*, *64*(4), 284-290.
- Clark, J.J., & Bernstein, I.L. (2006). Sensitization of salt appetite is associated with increased wanting “but not liking” of a salt reward in the sodium-deplete rat. *Behavioural Neuroscience*, *120*, 206–210.
- Clark, S. M., & Saules, K. K. (2013). Validation of the Yale Food Addiction Scale among a weight-loss surgery population. *Eating Behaviors*, *14*(2), 216–219.

- Cocores, J. A., & Gold, M. S. (2009). The Salted Food Addiction Hypothesis may explain overeating and the obesity epidemic. *Medical Hypotheses*, 73(6), 892–899.
- Colantuoni, C., Rada, P., McCarthy, J., Patten, C., Avena, N. M., Chadeayne, A., & Hoebel, B. G. (2002). Evidence That Intermittent, Excessive Sugar Intake Causes Endogenous Opioid Dependence. *Obesity Research*, 10(6), 478–488.
- Colantuoni, C., Schwenker, J., McCarthy, J., Rada, P., Ladenheim, B., Cadet, J., ... Hoebel, B. G. (2001). Excessive sugar intake alters binding to dopamine and mu-opioid receptors in the brain. *Neuroreport*, 12(16), 3549–3552.
- Corwin, R. L., & Grigson, P. S. (2009). Symposium Overview - Food Addiction: Fact or Fiction? *The Journal of Nutrition*, 139, 617–619.
- Cowan, J., & Devine, C. (2008). Food, eating, and weight concerns of men in recovery from substance addiction. *Appetite*, 50, 33–42.
- Crandall, C. S., & Reser, A. H. (2005). Attributions and weight-based prejudices. In K. D. Brownell, R. M. Puhl, M. B. Schwartz, & L. Rudd (Eds.), *Weight bias: Nature, consequences, and remedies* (pp. 83–96). New York, NY: Guilford Press
- Cruwys, T., Platow, M.J., Rieger, E., & Byrne, D.G. (2013). The development and validation of the Dieting Intentions Scale (DIS). *Psychological Assessment*, 25, 264–278.
- Cummings, D.E. (2006). Ghrelin and the short- and long-term regulation of appetite and body weight. *Physiology of Behaviour*, 89, 71–84.
- Cummings, D.E., Clement, K., Purnell, J.Q., Vaisse, C., Foster, K.E., Frayo, R.S., Schwartz, M.W., Basdevant, A., & Weigle, D.S. (2002). Elevated plasma ghrelin levels in Prader Willi syndrome. *Nature Medicine*, 8, 643–644.
- Curtis, C., & Davis, C. (2014). A qualitative study of binge eating and obesity from an addiction perspective. *Eating Disorders*, 22(1), 19–32.
- Dalley, J. W., Everitt, B. J., & Robbins, T. W. (2011). Impulsivity, compulsivity, and top-down cognitive control. *Neuron*, 69, 680–694.
- Dalton, M., Blundell, J., & Finlayson, G. (2013). Effect of BMI and binge eating on food reward and energy intake: further evidence for a binge eating subtype of obesity. *Obesity Facts*, 6(4), 348–359.

- Dar-Nimrod, I., Cheung, B.Y., Ruby, M.B., & Heine, S.J. (2014). Can merely learning about obesity genes affect eating behavior? *Appetite*, *81*, 269–276.
- Daughters, S. B., Lejuez, C. W., Bornovalova, M. A., Kahler, C. W., Strong, D. R., & Brown, R. A. (2005). Distress Tolerance as a Predictor of Early Treatment Dropout in a Residential Substance Abuse Treatment Facility, *114*(4), 729–734.
- Davies, J. B. (2013). *The myth of Addiction: Second Edition*. Taylor & Francis: Florence.
- Davis, C. A., Levitan, R. D., Reid, C., Carter, J. C., Kaplan, A. S., Patte, K. A., ... & Kennedy, J. L. (2009). Dopamine for “wanting” and opioids for “liking”: a comparison of obese adults with and without binge eating. *Obesity*, *17*(6), 1220-1225.
- Davis, C. (2013). From Passive Overeating to “Food Addiction”: A Spectrum of Compulsion and Severity. *ISRN Obesity*, 1–20.
- Davis, C. (2016). A commentary on the associations among ‘food addiction’, binge eating disorder, and obesity: Overlapping conditions with idiosyncratic clinical features. *Appetite*.
- Davis, C., & Carter, J. C. (2009). Compulsive overeating as an addiction disorder. A review of theory and evidence. *Appetite*, *53*(1), 1–8.
- Davis, C., Curtis, C., Levitan, R. D., Carter, J. C., Kaplan, A. S., & Kennedy, J. L. (2011). Evidence that “food addiction” is a valid phenotype of obesity. *Appetite*, *57*(3), 711–717.
- Davis, C., Loxton, N. J., Levitan, R. D., Kaplan, A. S., Carter, J. C., & Kennedy, J. L. (2013). “Food addiction” and its association with a dopaminergic multilocus genetic profile. *Physiology & Behavior*, *118*, 63–69.
- Davis, C., Zai, C., Levitan, R. D., Kaplan, a S., Carter, J. C., Reid-Westoby, C., ... Kennedy, J. L. (2011). Opiates, overeating and obesity: a psychogenetic analysis. *International Journal of Obesity (2005)*, *35*(10), 1347–54.
- Deb, I., Chakraborty, J., Gangopadhyay, P.K., Choudhury, S.R., & Das S. (2010). Single-nucleotide polymorphism (A118G) in exon 1 of OPRM1 gene causes alteration in downstream signaling by mu-opioid receptor and may contribute to the genetic risk for addiction. *Journal of Neurochemistry*, *112*, 486–496.

- De Graaf, C. (2005). Sensory responses, food intake and obesity. In D. Mela (Ed.), *Food, diet and obesity* (pp. 137–159). Cambridge, England: Woodhead Publishing Limited.
- DelParigi, A., Chen, K., Salbe, A.D., Hill, J.O., Wing, R.R., Reiman, E.M., & Tataranni, P.A. (2007). Successful dieters have increased neural activity in cortical areas involved in the control of behavior. *International Journal of Obesity*, *31*(3), 440–448.
- DePierre, J.A., Puhl, R.M., Luedicke, J. (2014). Public perceptions of food addiction: a comparison with alcohol and tobacco. *Journal of Substance Use*, *19*, 1–6.
- Department of Health (1991). *Dietary Reference Values for Food Energy and Nutrients for the United Kingdom* no. 41. London: HMSO.
- DiLeone, R. J. (2009). The influence of leptin on the dopamine system and implications for ingestive behavior. *International Journal of Obesity*, *33*, S25–S29.
- Dimitriou, S. G., Rice, H. B., & Corwin, R. L. (2000). Effects of limited access to a fat option on food intake and body composition in female rats. *The International Journal of Eating Disorders*, *28*(4), 436–445.
- di Pellegrino, G., Magarelli, S., & Mengarelli, F. (2011). Food pleasantness affects visual selective attention. *Quarterly Journal of Experimental Psychology*, *64*(3), 560–571.
- Dressler, H., & Smith, C. (2013). Food choice, eating behavior, and food liking differs between lean/normal and overweight/obese, low-income women. *Appetite*, *65*, 145–152.
- Drevets, W. C., Gautier, C., Price, J. C., Kupfer, D. J., Kinahan, P. E., Grace, A.A, ... Mathis, C. A. (2001). Amphetamine-induced dopamine release in human ventral striatum correlates with euphoria. *Biological Psychiatry*, *49*(2), 81–96.
- Drewnowski, A., Krahn, D. D., Demitrack, M. A, Nairn, K., & Gosnell, B. A. (1995). Naloxone, an opiate blocker, reduces the consumption of sweet high-fat foods in obese and lean female binge eaters. *The American Journal of Clinical Nutrition*, *61*(6), 1206–1212.

- Duarte, C., Pinto-Gouveia, J., & Ferreira, C. (2015). Expanding binge eating assessment: Validity and screening value of the Binge Eating Scale in women from the general population. *Eating Behaviors, 18*, 41-47.
- Dube, S. R., Felitti, V. J., Dong, M., Chapman, D. P., Giles, W. H., & Anda, R. F. (2003). Childhood Abuse, Neglect, and Household Dysfunction and the Risk of Illicit Drug Use: The Adverse Childhood Experiences Study. *Pediatrics, 111*(3), 564–572.
- Dunn, J. P., Cowan, R. L., Volkow, N. D., Feurer, I. D., Li, R., Williams, D. B., ... Abumrad, N. N. (2010). Decreased dopamine type 2 receptor availability after bariatric surgery: Preliminary findings. *Brain Research, 1350*, 123–130.
- Eichen, D. M., Lent, M. R., Goldbacher, E., & Foster, G. D. (2013). Exploration of “Food Addiction” in overweight and obese treatment-seeking adults. *Appetite, 67*, 22–24.
- Eiser, J. R., & Sutton, S. R. (1957). “Consonant” and “dissonant” and the self-attribution of addiction. *Addictive behaviors, 3*(2), 99-106.
- Epstein, L. H., Paluch, R., & Coleman, K. J. (1996). Differences in salivation to repeated food cues in obese and nonobese women. *Psychosomatic Medicine, 58*(2), 160–164.
- Epstein, L. H., Temple, J. L., Roemmich, J. N., & Bouton, M. E. (2009). Habituation as a Determinant of Human Food Intake, *116*(2), 384–407.
- Epstein, L. H., Wright, S. M., Paluch, R. A, Leddy, J., Hawk, L. W., Jaroni, J. L., ... Lerman, C. (2004). Food hedonics and reinforcement as determinants of laboratory food intake in smokers. *Physiology & Behavior, 81*(3), 511–517.
- Erb, S., & Brown, Z.J.A. (2006). A role for corticotropin-releasing factor in the long-term expression of behavioral sensitization to cocaine. *Behavioural Brain Research, 172*(2), 360–364.
- Erdfelder, E., Faul, F., & Buchner, A. (1996). GPOWER. A general power analysis program. *Behavior Research Methods, Instruments, & Computers, 28*, 1–11.
- Ersche, K. D., Jones, P. S., Williams, G. B., Turton, A. J., Robbins, T. W., & Bullmore, E. T. (2012). Abnormal brain structure implicated in stimulant drug addiction. *Science, 335*(6068), 601-604.

- Evenden, J. L. (1999). Varieties of impulsivity. *Psychopharmacology*, *146*, 348–361.
- Fedoroff, I.C., Polivy, J., & Herman, C.P. (1997). The effect of pre-exposure to food cues on the eating behavior of restrained and unrestrained eaters. *Appetite*, *28*, 33–47.
- Field, M., & Cox, W. M. (2008). Attentional bias in addictive behaviors: a review of its development, causes, and consequences. *Drug and Alcohol Dependence*, *97*(1), 1–20.
- Field, M., Hogarth, L., Bleasdale, D., Wright, P., Fernie, G., & Christiansen, P. (2011). Alcohol expectancy moderates attentional bias for alcohol cues in light drinkers. *Addiction*, *106*(6), 1097–1103.
- Field, M., Werthmann, J., Franken, I., & Hofmann, W. (2016). The role of attentional bias in obesity and addiction. *Health Psychology*, *28*, 1–45.
- Figlewicz, D.P., Patterson, T.A., Johnson, L.B., Zavosh, A., Israel, P.A., & Szot, P. (1998). Dopamine transporter mRNA is increased in the CNS of Zucker fatty (fa/fa) rats. *Brain Research Bulletin*, *46*, 199–202.
- Figlewicz, D. P., Bennett, J. L., Naleid, A. M., Davis, C., & Grimm, J. W. (2006). Intraventricular insulin and leptin decrease sucrose self-administration in rats. *Physiology & Behavior*, *89*, 611–6.
- Figlewicz, D. P., Bennett, J., Evans, S. B., Kaiyala, K., Sipols, A. J., & Benoit, S. C. (2004). Intraventricular insulin and leptin reverse place preference conditioned with high-fat diet in rats. *Behavioral Neuroscience*, *118*(3), 479–87.
- Filbey, F.M., Ray, L., Smolen, A., Claus, E.D., Audette, A., & Hutchison, K.E. (2008). Differential neural response to alcohol priming and alcohol taste cues is associated with DRD4 VNTR and OPRM1 genotypes. *Alcoholism: Clinical and Experimental Research*, *32*(7), 1113–1123.
- Finlayson, G., Arlotti, A., Dalton, M., King, N., & Blundell, J. E. (2011). Implicit wanting and explicit liking are markers for trait binge eating. A susceptible phenotype for overeating. *Appetite*, *57*(3), 722–728.
- Finlayson, G., King, N., & Blundell, J. (2008). The role of implicit wanting in relation to explicit liking and wanting for food: implications for appetite control. *Appetite*, *50*, 120–127.

- Finlayson, G., King, N., & Blundell, J. E. (2007). Liking vs. wanting food: importance for human appetite control and weight regulation. *Neuroscience and Biobehavioral Reviews*, *31*(7), 987–1002.
- Fischer, S., & Smith, G. T. (2008). Binge eating, problem drinking, and pathological gambling: Linking behavior to shared traits and social learning. *Personality and Individual Differences*, *44*(4), 789-800.
- Fischer, S., Smith, G. T., & Anderson, K. G. (2003). Clarifying the role of impulsivity in bulimia nervosa. *International Journal of Eating Disorders*, *33*(4), 406–411.
- Flint, A. J., Gearhardt, A. N., Corbin, W. R., Brownell, K. D., Field, A. E., & Rimm, E. B. (2014). Food-addiction scale measurement in 2 cohorts of middle-aged and older women. *The American Journal of Clinical Nutrition*, *99*, 578–586.
- Folkman S. (1984). Personal control and stress and coping processes. A theoretical analysis. *Journal of Personality and Social Psychology*, *46*, 839-852.
- Francis, J. A., Stewart, S. H., & Hounsell, S. (1997). Dietary Restraint and the Selective Processing of Forbidden and Nonforbidden Food Words. *Cognitive Therapy and Research*, *21*(6), 633-646.
- Frank, G. K., Reynolds, J. R., Shott, M. E., Jappe, L., Yang, T. T., Tregellas, J. R., & O'Reilly, R. C. (2012). Anorexia nervosa and obesity are associated with opposite brain reward response. *Neuropsychopharmacology*, *37*(9), 2031-2046.
- Frayn, K. N. (2010). *Metabolic regulation. A human perspective*. Chichester: Wiley- Blackwell.
- Frayn, M., Sears, C. R., & von Ranson, K. M. (2016). A sad mood increases attention to unhealthy food images in women with food addiction. *Appetite*, *100*, 55–63.
- Garner, D. M., Olmsted, M. P., Bohr, Y., & Garfinkel, P. E. (1982). The eating attitudes test: psychometric features and clinical correlates. *Psychological Medicine*, *12*, 871–878.
- Gearhardt, A. N., & Brownell, K. D. (2013). Can food and addiction change the game? *Biological Psychiatry*, *73*(9), 802–803.

- Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2009). Preliminary validation of the Yale Food Addiction Scale. *Appetite*, *52*(2), 430–436.
- Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2016). Development of the Yale Food Addiction Scale Version 2.0. *Psychology of Addictive Behaviors*, *30*(1), 113–121.
- Gearhardt, A. N., Treat, T. A., Hollingworth, A., & Corbin, W. R. (2012). The relationship between eating-related individual differences and visual attention to foods high in added fat and sugar. *Eating Behaviors*, *13*(4), 371–374.
- Gearhardt, A. N., White, M. A., Masheb, R. M., & Grilo, C. M. (2013). An examination of food addiction in a racially diverse sample of obese patients with binge eating disorder in primary care settings. *Comprehensive Psychiatry*, *54*(5), 500–505.
- Gearhardt, A. N., Yokum, S., Orr, P. T., Stice, E., Corbin, W. R., & Brownell, K. D. (2011). Neural Correlates of Food Addiction. *Archives of General Psychiatry*, *68*(8), 808–816.
- Geliebter, E.K., Yahav, M.E., Gluck, S.A., & Hashim, S.A. (2004). Gastric capacity, test meal intake, and appetitive hormones in binge eating disorder. *Physiology and Behavior*, *81*, 735–740.
- Gibson, E. L., & Desmond, E. (1999). Chocolate Craving and Hunger State: Implications for the Acquisition and Expression of Appetite and Food Choice. *Appetite*, *32*(2), 219–240.
- Giesen, J.C.A.H., Havermans, R. C., Douven, A., Tekelenburg, M., & Jansen, A. (2010). Will work for snack food: the association of BMI and snack reinforcement. *Obesity*, *18*(5), 966–970.
- Gleim, J. A. & Gleim, R. R. (2003). *Cronbach Alpha and Spearman-Brown coefficient of reliability explanations*. Paper presented at the Midwest research-to-practice conference in adult, continuing, and community education, The Ohio State University, Columbus, OH, October 8-10, 2003.
- Glorfeld, L. W. (1995). An improvement on Horn's parallel analysis methodology for selecting the correct number of factors to retain. *Educational and Psychological Measurement*, *55*, 377–393.

- Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors*, 7(1), 47–55.
- Graham, R., Hoover, A., Ceballos, N. A., & Komogortsev, O. (2011). Body mass index moderates gaze orienting biases and pupil diameter to high and low calorie food images. *Appetite*, 56(3), 577–586.
- Gray, J. A. (1987). *The psychology of fear and stress*. Cambridge: Cambridge University Press.
- Green, M. W., & Rogers, P. J. (1993). Selective Attention to Food and Body Shape Words in Dieters and Restrained Non-dieters. *International Journal of Eating Disorders*, 14(4), 515–517.
- Greenwald, A.G., McGhee, D.E., Schwartz, J.L.K. (1998). Measuring individual differences in implicit cognition: The implicit Association Test. *Journal of Personality and Social Psychology*, 74, 1464-1480.
- Grucza, R. A, Krueger, R. F., Racette, S. B., Norberg, K. E., Hipp, P. R., & Bierut, L. J. (2010). The emerging link between alcoholism risk and obesity in the United States. *Archives of General Psychiatry*, 67(12), 1301–1308.
- Guerrieri, R., Nederkoorn, C., & Jansen, A. (2007). How impulsiveness and variety influence food intake in a sample of healthy women. *Appetite*, 45, 119–122.
- Guerrieri, R., Nederkoorn, C., & Jansen, A. (2008). The Effect of an Impulsive Personality on Overeating and Obesity: Current State of Affairs. *Psychological Topics*, 17(2), 265–286.
- Hallschmid, M., Higgs, S., Thienel, M., Ott, V., & Lehnert, H. (2012). Postprandial administration of intranasal insulin intensifies satiety and reduces intake of palatable snacks in women. *Diabetes*, 61(4), 782-789.
- Halpern, S. D., Asch, D. A., & Volpp, K. G. (2012). Commitment contracts as a way to health. *BMJ*, 344, e522
- Haltia, L. T., Rinne, J. O., Merisaari, H., Maguire, R. P., Savontaus, E., Helin, S.,... Kaasinen, V. (2007). Effects of intravenous glucose on dopaminergic function in the human brain in vivo. *Synapse*, 61, 748– 756.
- Hanks, A. S., Just, D. R., & Wansink, B. (2013). Preordering school lunch encourages better food choices by children. *JAMA Pediatrics*, 167(7), 482e673.

- Hardman, C.A., Rogers, P.J., Dallas, R., Scott, J., Ruddock, H., & Robinson, E. (2015). "Food addiction is real": the effects of exposure to this message on self-diagnosed food addiction and eating behaviour. *Appetite*, *91*, 179–184.
- Hardman, C. A., Herbert, V. M. B., Brunstrom, J. M., Munafò, M. R., & Rogers, P. J. (2012). Dopamine and food reward: effects of acute tyrosine/phenylalanine depletion on appetite. *Physiology & Behavior*, *105*(5), 1202–1207.
- Hardman, C. A., Scott, J., Field, M., & Jones, A. (2014). To eat or not to eat. The effects of expectancy on reactivity to food cues. *Appetite*, *76*, 153–60.
- Hardman, C. A., Rogers, P. J., Dallas, R., Scott, J., Ruddock, H. K., & Robinson, E. (2015). "Food addiction is real": the effects of exposure to this message on self-diagnosed food addiction and eating behaviour. *Appetite*, *91*, 179–184.
- Hardman, C. A., Rogers, P. J., Timpson, N. J., & Munafò, M. R. (2014). Lack of association between DRD2 and OPRM1 genotypes and adiposity. *International Journal of Obesity*, *38*(5), 730–736.
- Hare, T.A., Camerer, C.F., Rangel, A. (2009). Self-control in decision-making involves modulation of the vmPFC valuation system. *Science*, *324*, 646–648.
- Haring, S. J., & Harris, R. B. S. (2011). The relation between dietary fructose, dietary fat and leptin responsiveness in rats. *Physiology & Behavior*, *104*(5), 914–922.
- Harris, G.C., Wimmer, M., & Aston-Jones, G. (2005). A role for lateral hypothalamic orexin neurons in reward seeking. *Nature*, *437*(7058), 556–559.
- Hashim, S. A., & Van Itallie, T. B. (1965). Cholestyramine resin therapy for hypercholesteremia: clinical and metabolic studies. *Jama*, *192*(4), 289–293.
- Havermans, R. C. (2011). "You Say it's Liking, I Say it's Wanting ...". On the difficulty of disentangling food reward in man. *Appetite*, *57*(1), 286–94.
- Hawks, S. R., Madanat, H., Smith, T., & De La Cruz, N. (2008). Classroom approach for managing dietary restraint, negative eating styles, and body image concerns among college women. *Journal of American college health*, *56*(4), 359–366.

- Hayes, A.F., & Preacher, K.J. (2014). Statistical mediation analysis with a multicategorical independent variable. *British Journal of Mathematical and Statistical Psychology*, *67*, 451–470.
- Hayes, A.F. (2012). PROCESS: A versatile computational tool for observed variable mediation, moderation, and conditional process modeling. Available from: <http://www.afhayes.com/public/process2012.pdf>
- Heatherton, T. F., Herman, C. P., Polivy, J., King, J. A., & McGree, S. T. (1988). The (mis)measurement of restraint: An analysis of conceptual and psychometric issues. *Journal of Abnormal Psychology*, *97*, 19-28.
- Hebebrand, J., Albayrak, Ö., Adan, R., Antel, J., Dieguez, C., de Jong, J., ... Dickson, S. L. (2014). “Eating addiction”, rather than “food addiction”, better captures addictive-like eating behavior. *Neuroscience & Biobehavioral Reviews*, *47*, 295–306.
- Hendrikse, J. J., Cachia, R. L., Kothe, E. J., McPhie, S., Skouteris, H., & Hayden, M. J. (2015). Attentional biases for food cues in overweight and individuals with obesity: a systematic review of the literature. *Obesity Reviews*, *16*(5), 424e432.
- Herman, C.P., & Polivy, J. (1983). A boundary model for the regulation of eating. *Psychiatric Annals*, *13*(12), 918-927.
- Herman, C. P., & Mack, D. (1975). Restrained and unrestrained eating. *Journal of Personality*, *43*, 647-660
- Herman, C. P., & Polivy, J. (1980). *Restrained eating*. In A. J. Stunkard (Ed.), *Obesity* (pp. 208–225). Philadelphia: Saunders.
- Hetherington, M. M., & Macdiarmid, J. I. (1993). “Chocolate Addiction”: a preliminary study of its description and its relationship to problem eating. *Appetite*, *21*, 233–246.
- Hetherington, M.M. & Macdiarmid, J.I. (1995). Pleasure and excess: Liking for and overconsumption of chocolate. *Physiology and Behavior*, *57*(1), 27-35.
- Hill, C., Saxton, J., Webber, L., Blundell, J., & Wardle, J. (2009). The relative reinforcing value of food predicts weight gain in a longitudinal study of 7–10y-old children. *The American Journal of Clinical Nutrition*, *90*, 276–281.

- Hoaglin, D. C., & Iglewicz, B. (1987). Fine-tuning some resistant rules for outlier labeling. *Journal of the American Statistical Association*, 82(400), 1147-1149.
- Hodgson, R.J., & Greene, J.B (1981). The saliva priming effect, eating speed and the measurement of hunger. *Behaviour Research and Therapy*, 18(4), 243-247.
- Hollitt, S., Kemps, E., Tiggemann, M., Smeets, E., & Mills, J. S. (2010). Components of attentional bias for food cues among restrained eaters. *Appetite*, 54(2), 309–313.
- Hoyt, C.L., Burnette, J.L., Auster-Gussman, L. (2014). “Obesity Is a Disease”: Examining the Self-Regulatory Impact of This Public-Health Message. *Psychological Science*, 25, 1–6.
- Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*, 6, 1–55.
- Hukshorn, C.J., Saris, W.H., Westerterp-Plantenga, M.S., Farid, A.R., Smith, F.J., & Campfield, L.A. (2000). Weekly subcutaneous pegylated recombinant native human leptin (PEG-OB) administration in obese men. *Journal of Clinical Endocrinology Metabolism*, 85, 4003–4009.
- Hutcheson, G. & Sofroniou, N. (1999). *The multivariate social scientist*. Sage, London.
- Ifland, J. R., Preuss, H. G., Marcus, M. T., Rourke, K. M., Taylor, W. C., Burau, K., et al. (2009). Refined food addiction: A classic substance use disorder. *Medical Hypotheses*, 72(5), 518–526.
- Imperatori, C., Innamorati, M., Contardi, A., Continisio, M., Tamburello, S., Lamis, D. A., ... Fabbriatore, M. (2014). The association among food addiction, binge eating severity and psychopathology in obese and overweight patients attending low-energy-diet therapy. *Comprehensive Psychiatry*, 55(6), 1358–1362.
- Jahng, J.W. (2013). “Stressful experiences in early life and subsequent food intake,” in *Animal Models of Eating Disorders*, N.M.Avena, Ed., vol. 74 of *Neuromethods*, pp. 127–153, Springer, Berlin, Germany.

- Jellinek, E. M. (1960). *The Disease Concept of Alcoholism*. Highland Park, N J: Hillhouse Press.
- Jenkinson, C.P., Hanson, R., Cray, K., Wiedrich, C., Knowler, W.C., Bogardus, C., et al. (2000). Association of dopamine D2 receptor polymorphisms Ser311Cys and TaqIA with obesity or type 2 diabetes mellitus in Pima Indians. *International Journal of Obesity and Related Metabolic Disorders*, 24, 1233–1238.
- Johnson, W. G. (1974). Effect of cue prominence and subject weight on human food-directed performance. *Journal of Personality and Social Psychology*, 29, 843–848.
- Jones, A., Cole, J., Goudie, A., & Field, M. (2012). The effect of restraint beliefs on alcohol-seeking behavior. *Psychology of Addictive Behaviors*, 26(2), 325–329.
- Jones, A., Hogarth, L., Christiansen, P., Rose, A. K., Martinovic, J., & Field, M. (2012). Reward expectancy promotes generalized increases in attentional bias for rewarding stimuli. *The Quarterly Journal of Experimental Psychology*, 65(12), 2333-2342.
- Kaplan, A.S., Levitan, R.D., Yilmaz, Z., Davis, C., Tharmalingam, S., Kennedy, J.L. (2008). A DRD4/BDNF gene–gene interaction associated with maximum BMI in women with bulimia nervosa. *International Journal of Eating Disorders*, 41, 22–28.
- Katzman, M. A., Greenberg, A., & Marcus, I. D. (1991). Bulimia in Opiate-Addicted Women: Developmental Cousin and Relapse Factor. *Journal of Substance Abuse Treatment*, 8, 107–112.
- Keeler, C.L., Mattes, R.D., & Tan, S-Y. (2015). Anticipatory and reactive responses to chocolate restriction in frequent chocolate consumers. *Obesity*, 23, 1130–1135.
- Kelloway, E. K. (1998). *Using LISREL for Structural Equation Modeling: a Researcher's Guide*. Thousand Oaks, CA: Sage Publications, Inc.
- Kemps, E., Tiggemann, M., & Hollitt, S. (2014a). Biased Attentional Processing of Food Cues and Modification in Obese Individuals. *Health Psychology*, 33(11), 1391–1401.

- Kemps, E., Tiggemann, M., & Hollitt, S. (2014b). Exposure to television food advertising primes food-related cognitions and triggers motivation to eat. *Psychology & Health, 29*(10), 1192–205.
- Kemps, E., Tiggemann, M., Martin, R., & Elliott, M. (2013). Implicit approach-avoidance associations for craved food cues. *Journal of Experimental Psychology: Applied, 19*, 30–8.
- Kenny, P. J. (2013). The Food Addiction. *Scientific American, 309*(3), 44–49.
- Kessler, R., Adler, L., R, B., Biederman, J., Conners, C., & Al., E. (2006). The Prevalence and Correlates of Adult ADHD in the United States: Results From the National Comorbidity Survey Replication. *The American Journal of Psychiatry, 163*(4), 716–723.
- Kiffin-Petersen, S., & Cordery, J. L. (2003). Trust, individualism and job characteristics as predictors of employee preference for teamwork. *International Journal Human Resource Management, 14*, 93–116.
- Killgore, W. & Yurgelun-Todd, D. (2005) Body mass predicts orbitofrontal activity during visual presentations of high-calorie foods. *Neuroreport, 31*, 859–863
- Killgore, W. D. S., Weber, M., Schwab, Z. J., Kipman, M., Deldonno, S. R., Webb, C. A., & Rauch, S. L. (2013). Cortico-limbic responsiveness to high-calorie food images predicts weight status among women. *International Journal of Obesity, 37*, 1435–1442.
- Kiyatkin, E.A, & Gratton, A. (1994). Electrochemical monitoring of extracellular dopamine in nucleus accumbens of rats lever-pressing for food. *Brain Research, 652*(2), 225–234.
- Klajner, F., Herman, C. P., Polivy, J., & Chhabra, R. (1981). Human obesity, dieting, and anticipatory salivation to food. *Physiology & Behavior, 27*, 195–198.
- Koob, G. F., Riley, S. J., Smith, S. C., & Robbins, T. W. (1978). Effects of 6-hydroxydopamine lesions of the nucleus accumbens septi and olfactory tubercle on feeding, locomotor activity, and amphetamine anorexia in the rat. *Journal of Comparative and Physiological Psychology, 92*(5), 917–27.

- Kozak, A. T., & Fought, A. (2011). Beyond alcohol and drug addiction. Does the negative trait of low distress tolerance have an association with overeating? *Appetite*, *57*(3), 578–581.
- Latner, J. D., Puhl, R. M., Murakami, J. M., & O'Brien, K. S. (2014). Food addiction as a causal model of obesity. Effects on stigma, blame, and perceived psychopathology. *Appetite*, *77*, 77–82.
- Lavy, E.H., & van den Hout, M.A. (1993). Attentional bias for appetitive cues: effects of fasting in normal subjects. *Behavioural and Cognitive Psychotherapy*, *21*(4), 297-310.
- Lawrence, N. S., Hinton, E. C., Parkinson, J. A., & Lawrence, A. D. (2012). Nucleus accumbens response to food cues predicts subsequent snack consumption in women and increased body mass index in those with reduced self-control. *NeuroImage*, *63*(1), 415–422.
- Le, D.S., Pannacciulli, N., Chen, K., Del Parigi, A., Salbe AD., et al. (2006). Less activation of the left dorsolateral prefrontal cortex in response to a meal: a feature of obesity. *American Journal of Clinical Nutrition*, *84*, 725–731.
- Lee, N.M., Lucke, J., Hall, W.D., Meurk, C., Boyle, F.M., & Carter, A. (2013). Public views on food addiction and obesity: implications for policy and treatment. *PloS ONE*, *8*, e74836.
- Leehr, E. J., Schag, K., Bruckmann, C., Plewnia, C., Zipfel, S., Nieratschker, V., et al. (2016) A putative association of COMT Val(108/158)Met with impulsivity in binge eating disorder. *European Eating Disorders Review*, *24*, 169-173.
- LeGoff, D.B., Leichner, P. & Spigelman, M.N. (1988). Salivary response to olfactory food stimuli in anorexics and bulimics. *Appetite*, *11*, 15-25.
- Lemmens, S. G. T., Schoffelen, P. F. M., Wouters, L., Born, J. M., Martens, M. J. I., Rutters, F., et al. (2009). Eating what you like induces a stronger decrease in ‘wanting’ to eat. *Physiology and Behavior*, *98*, 318–325.
- Lemmens, S. G., Rutters, F., Born, J. M., & Westerterp-Plantenga, M. S. (2011). Stress augments food “wanting” and energy intake in visceral overweight subjects in the absence of hunger. *Physiology & Behavior*, *103*(2), 157–163.

- Levine, A. S., & Billington, C. J. (2004). Opioids as agents of reward-related feeding: a consideration of the evidence. *Physiology & Behavior*, 82(1), 57–61.
- Lewis-Beck, M., Bryman, A. E., & Liao, T. F. (2003). *The Sage encyclopedia of social science research methods*. Sage Publications.
- Liang, N.-C., Hajnal, A., & Norgren, R. (2006). Sham feeding corn oil increases accumbens dopamine in the rat. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology*, 291(5), 1236–1239.
- Lilenfeld, L. R. R., Ringham, R., Kalarchian, M. A., & Marcus, M. D. (2008). A family history study of binge-eating disorder. *Comprehensive Psychiatry*, 49, 247–254.
- Loeber, S., Grosshans, M., Herpertz, S., Kiefer, F., & Herpertz, S. C. (2013). Hunger modulates behavioral disinhibition and attention allocation to food-associated cues in normal-weight controls. *Appetite*, 71, 32–39.
- Loewenstein, G. (1996). Out of control: Visceral influences on behavior. *Organizational Behavior and Human Decision Processes*, 65, 272–292.
- Logan, G., & Cowan, W. (1984). On the ability to inhibit thought and action: A theory of an act of control. *Psychological Review*, 91, 295–327.
- Lomba, A., Milagro, F. I., Garcia-Diaz, D. F., Campion, J., Marzo, F., and Martinez, J. A. (2009). A high-sucrose isocaloric pair-fed model induces obesity and impairs NDUFB6 gene function in rat adipose tissue. *Journal of nutrigenetics and nutrigenomics*, 2(6), 267–272.
- Long, C. G., Blundell, J. E., & Finlayson, G. (2015). A Systematic Review of the Application And Correlates of YFAS-Diagnosed “Food Addiction ” in Humans: Are Eating-Related “ Addictions ” a Cause for Concern or Empty Concepts? *Obesity Facts*, 8, 386–401.
- Lyons, M. A. (1998). The phenomenon of compulsive overeating in a selected group of professional women. *Journal of Advanced Nursing*, 27(6), 1158–64. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/9663866>
- Maas, L. C., Lukas, S. E., Kaufman, M. J., Weiss, R. D., Daniels, S. L., Rogers, V. W., ... & Renshaw, P. F. (1998). Functional magnetic resonance imaging of human brain activation during cue-induced cocaine craving. *American Journal of Psychiatry*, 155(1), 124–126.

- MacCallum, R. C., Browne, M. W. & Sugawara, H. M. (1996) Power analysis and determination of sample size for covariance structure modeling. *Psychological Methods, 1*, 130–149.
- Macdiarmid, J. L. & Hetherington, M. M. (1995). Mood modulation by food: An exploration of affect and cravings in “chocolate addicts”. *British Journal of Clinical Psychology, 34*, 129–138.
- MacKinnon, D.P., Lockwood, C.M., Hoffman, J.M., West, S.G., & Sheets, V.A. (2002). Comparison of methods to test mediation and other intervening variable effects. *Psychological Methods, 7*, 83–104.
- Malika, N. M., Hayman, L. W., Miller, A. L., Lee, H. J., & Lumeng, J. C. (2015). Low-income women’s conceptualizations of food craving and food addiction. *Eating Behaviors, 18*, 25–29.
- Mann, T., & Ward, A. (2001). Forbidden fruit: does thinking about a prohibited food lead to its consumption? *International Journal of Eating Disorders, 29*, 319–327
- Mark, G., Rada, P., Weinberg, J., Pothos, E., & Hoebel, B. G. (1992). Cholinergic activity in the nucleus accumbens may be inversely related to food intake. *Appetite, 19*, 201.
- Martinez, D., Narendran, R., Foltin, R., Slifstein, M., Hwang, D., Broft, A., et al., (2007). Amphetamine-induced dopamine release: markedly blunted in cocaine dependence and predictive of the choice to self-administer cocaine. *The American Journal of Psychiatry 164*, 622–629.
- McCaig, D. C., Hawkins, L. A., & Rogers, P. J. (2016). Licence to eat: Information on energy expended during exercise affects subsequent energy intake. *Appetite, 107*, 323-329.
- McClernon, F.J., Hutchison, K.E., Rose, J.E., Kozink, R.V., 2007. DRD4 VNTR polymorphism is associated with transient fMRI-BOLD responses to smoking cues. *Psychopharmacology 194*(4), 433–441
- Meadows, A., & Higgs, S. (2013). I think, therefore I am? Characteristics of a non-clinical population of self-perceived food addicts. *Appetite, 71*, 482.
- Mela, D. J. (2006). Eating for pleasure or just wanting to eat? Reconsidering sensory hedonic responses as a driver of obesity. *Appetite, 47*(1), 10–17.

- Menard, S. (1995). *Applied Logistic Regression Analysis: Sage University Series on Quantitative Applications in the Social Sciences*. Thousand Oaks, CA: Sage.
- Meule, A., & Kübler, A. (2012). Eating Behaviors Food cravings in food addiction: The distinct role of positive reinforcement. *Eating Behaviors*, *13*(3), 252–255.
- Meule, A., Lutz, A., Vögele, C., & Kübler, A. (2012). Eating Behaviors Women with elevated food addiction symptoms show accelerated reactions, but no impaired inhibitory control, in response to pictures of high-calorie food-cues. *Eating Behaviors*, *13*(4), 423–428.
- Michels, N., Sioen, I., Braet, C., Eiben, G., Hebestreit, A., Huybrechts, I., et al. (2012). Stress, emotional eating behaviour and dietary patterns in children. *Appetite*, *59*(3), 762–769.
- Mischel, W., Shoda, Y., & Rodriguez, M.I. (1989). Delay of gratification in children. *Science*, *244*, 933–938.
- Mogg, K., Bradley, B. P., Hyare, H., & Lee, S. (1998). Selective attention to food-related stimuli in hunger: are attentional biases specific to emotional and psychopathological states, or are they also found in normal drive states? *Behaviour Research and Therapy*, *36*, 227–237.
- Morabia, A., Fabre, J., Chee, E., Zeger, S., Orsat, E., & Robert, A. (1989). Diet and opiate addiction: a quantitative assessment of the diet of non-institutionalized opiate addicts. *British Journal of Addiction*, *84*, 173–180.
- Moray, J., Fu, A., Brill, K., & Mayoral, M. S. (2007). Viewing Television While Eating Impairs the Ability to Accurately Estimate Total Amount of Food Consumed. *Bariatric Nursing and Surgical Patient Care*, *2*(1), 71–76.
- Munafò, M., Matheson, I. J., & Flint, J. (2007). Association of the DRD2 gene Taq1A polymorphism and alcoholism: a meta-analysis of case-control studies and evidence of publication bias. *Molecular Psychiatry*, *12*, 454–461.
- Murphy, C. M., Stojek, M. K., & MacKillop, J. (2013). Interrelationships among Impulsive Personality Traits, Food Addiction, and Body Mass Index. *Appetite*, *73*, 45-50.

- Myers, R.H. (1990). *Classical and modern regression with applications*. Boston: PWS-Kent
- Myrick, H., Anton, R. F., Li, X., Henderson, S., Drobos, D., Voronin, K., & George, M. S. (2004). Differential brain activity in alcoholics and social drinkers to alcohol cues: relationship to craving. *Neuropsychopharmacology*, *29*(2), 393–402.
- Narita, M., Nagumo, Y., Hashimoto, S., Narita, M., Khotib, J., Miyatake, M., ... Suzuki, T. (2006). Direct involvement of orexinergic systems in the activation of the mesolimbic dopamine pathway and related behaviors induced by morphine. *The Journal of Neuroscience*, *26*(2), 398–405.
- Nasser, J. (2001). Taste, food intake and obesity. *Obesity Reviews*, *2*(10), 213–218.
- Nasser, J. A, Evans, S. M., Geliebter, A., Pi-Sunyer, F. X., & Foltin, R. W. (2008). Use of an operant task to estimate food reinforcement in adult humans with and without BED. *Obesity*, *16*(8), 1816–1820.
- Nederkoorn, C., Houben, K., Hofmann, W., Roefs, A., & Jansen, A. (2010). Control yourself or just eat what you like? Weight gain over a year is predicted by an interactive effect of response inhibition and implicit preference for snack foods. *Health Psychology*, *29*, 389–393.
- Nederkoorn, C., Jansen, E., Mulkens, S., & Jansen, A. (2007). Impulsivity predicts treatment outcome in obese children. *Behaviour Research and Therapy*, *45*, 1071-1075.
- Nederkoorn, C., Smulders, F. T. Y., Havermans, R. C., Roefs, A., and Jansen, A. (2006). Impulsivity in obese women. *Appetite*, *47*, 253–256.
- Nederkoorn, C., Smulders, F. T., & Jansen, A. (2000). Cephalic phase responses, craving and food intake in normal subjects. *Appetite*, *35*(1), 45–55.
- NeuroFAST (2013). *NeuroFAST consensus opinion on food addiction*. Available at: <http://www.neurofast.eu/consensus/> (accessed 12 August 2016).
- Ng, J., Stice, E., Yokum, S., & Bohon, C. (2011). An fMRI study of obesity, food reward, and perceived caloric density. Does a low-fat label make food less appealing? *Appetite*, *57*(1), 65–72.

- The NHS Information Centre, Lifestyles Statistics. *Statistics on obesity, physical activity and diet: England 2012*. London: The Health and Social Care Information Centre.
- Nicolau, J., Ayala, L., Rivera, R., Speranskaya, A., Sanchis, P., Julian, X., et al. (2015) Postoperative grazing as a risk factor for negative outcomes after bariatric surgery. *Eating Behaviors*, 18, 147-150.
- Nijs, I. M. T., Franken, I. H.A, & Muris, P. (2010). Food-related Stroop interference in obese and normal-weight individuals: behavioral and electrophysiological indices. *Eating Behaviors*, 11(4), 258–65.
- Nijs, I. M. T., Muris, P., Euser, A. S., & Franken, I. H. A. (2010). Differences in attention to food and food intake between overweight / obese and normal-weight females under conditions of hunger and satiety. *Appetite*, 54(2), 243–254.
- Nikolova, Y. S., Ferrell, R. E., Manuck, S. B., & Hariri, A. R. (2011). Multilocus genetic profile for dopamine signaling predicts ventral striatum reactivity. *Neuropsychopharmacology*, 36, 1940–1947.
- Nisbett, R.E. (1968). Determinants of food intake in obesity. *Science*, 159(3820), 1254-1255.
- Nordgren, L. F., van Harreveld, F., & van der Pligt, J. (2009). The Restraint Bias: How the Illusion of Self-Restraint Promotes Impulsive Behavior. *Psychological Science*, 20(12), 1523–1528.
- Nunnally, J. C., & Bernstein, I. H. (1994). *Psychometric theory* (3rd ed.). New York: McGraw-Hill.
- O'Brien, R.M. (2007). A caution regarding rules of thumb for variance inflation factors. *Quality & Quantity*, 41, 673–690.
- O'Rourke, N., & Hatcher, L. (2013). *A step-by-step approach to using SAS for factor analysis and structural equation modeling*. Sas Institute.
- O'Dea, K. (1992). Obesity and diabetes in the land of milk and honey. *Diabetes Metabolism Reviews*, 8, 373-388.
- O'Malley, S.S., Krishan-Sarin, S., Farren, C., Sinha, R., & Kreek, M.J. (2002). Naltrexone decreases craving and alcohol self-administration in alcohol-dependent subjects and activates the hypothalamo-pituitary-adrenocortical axis. *Psychopharmacology*, 160, 19–29.

- Ouwehand, C., & Papies, E. K. (2010). Eat it or beat it. The differential effects of food temptations on overweight and normal-weight restrained eaters. *Appetite*, *55*(1), 56–60.
- Ouwens, M.A., van Strien, T., & van der Staak, C.P.F. (2003). Tendency towards overeating and restraint as predictors of food consumption. *Appetite*, *40*(1), 291-298.
- Overduin, J., Jansen, A., & Louwse, E. (1995). Stroop interference and food intake. *International Journal of Eating Disorders*, *18*, 277–285.
- Palmberg, A. A., Stern, M., Kelly, N. R., Bulik, C., Belgrave, F. Z., Trapp, S. K., ... Mazzeo, S. E. (2014). Adolescent Girls and Their Mothers Talk About Experiences of Binge and Loss of Control Eating. *Journal of Child and Family Studies*, *23*(8), 1403–1416.
- Patton, M.Q. (1990). *Qualitative evaluation and research methods* (2nd ed.). Thousand Oaks, CA, US: Sage Publications.
- Pearce, J. M., & Hall, G. (1980). A model for Pavlovian learning. Variations in the effectiveness of conditioned but not of unconditioned stimuli. *Psychological Review*, *87*(6), 532–552
- Pearl, R. L., & Lebowitz, M. S. (2014). Beyond personal responsibility: effects of causal attributions for overweight and obesity on weight-related beliefs, stigma, and policy support. *Psychology & Health*, *29*(10), 1176–1191.
- Peciña, S. (2008). Opioid reward “liking” and “wanting” in the nucleus accumbens. *Physiology & Behavior*, *94*(5), 675–680.
- Peciña, S., & Berridge, K. C. (2005). Hedonic hot spot in nucleus accumbens shell: where do mu-opioids cause increased hedonic impact of sweetness? *The Journal of Neuroscience*, *25*(50), 11777–11786.
- Peciña, S., Smith, K. S., & Berridge, K. C. (2006). Hedonic hot spots in the brain. *The Neuroscientist*, *12*(6), 500–511.
- Pedram, P., Wadden, D., Amini, P., Gulliver, W., Randell, E., Cahill, F., et al. (2013). Food addiction: its prevalence and significant association with obesity in the general population. *PloS One*, *8*(9), e74832.
- Pepino, M. Y., & Mennella, J. A. (2012). Habituation to the pleasure elicited by sweetness in lean and obese women. *Appetite*, *58*(3), 800–805.

- Perpina, C., Hemsley, D., Treasure, J., & de Silva, P. (1993). Is selective information processing of food and body words specific to patients with eating disorders? *International Journal of Eating Disorders*, 14, 359–366.
- Phelan, S., Hassenstab, J., McCaffery, J. M., Sweet, L., Raynor, H. a, Cohen, R. a, & Wing, R. R. (2011). Cognitive interference from food cues in weight loss maintainers, normal weight, and obese individuals. *Obesity*, 19(1), 69–73.
- Phillips, K. E., Kelly-Weeder, S., & Farrell, K. (2016). Binge eating behavior in college students: What is a binge? *Applied Nursing Research*, 30, 7-11.
- Placanica, J. L., Faunce, G. J., & Soames Job, R. F. (2001). The effect of fasting on attentional biases for food and body shape/weight words in high and low Eating Disorder Inventory scorers. *International Journal of Eating Disorders*, 32, 79–90.
- Polivy, J., Coleman, J., & Herman, C. P. (2005). The effect of deprivation on food cravings and eating behavior in restrained and unrestrained eaters. *The International Journal of Eating Disorders*, 38(4), 301–309.
- Pothos, E. M., Tapper, K., & Calitri, R. (2009). Cognitive and behavioral correlates of BMI among male and female undergraduate students. *Appetite*, 52(3), 797–800.
- Pratt, W. E., Choi, E., & Guy, E. G. (2012). An examination of the effects of subthalamic nucleus inhibition or μ -opioid receptor stimulation on food-directed motivation in the non-deprived rat. *Behavioural Brain Research*, 230(2), 365–73.
- Prentice, A.M. (2005). Early influences on human energy regulation: Thrifty genotypes and thrifty phenotypes. *Physiology and Behavior*, 86, 640– 645.
- Price, M., Higgs, S., & Lee, M. (2015). Self-reported eating traits: Underlying components of food responsivity and dietary restriction are positively related to BMI. *Appetite*, 95, 203-210.
- Pudel, V., & Oetting, M. (1977). Eating in the laboratory: Behavioural aspects of the positive energy balance. *International Journal of Obesity*, 1, 369-386.
- Pursey, K., Stanwell, P., Gearhardt, A., Collins, C., & Burrows, T. (2014). The Prevalence of Food Addiction as Assessed by the Yale Food Addiction Scale: A Systematic Review. *Nutrients*, 6(10), 4552–4590.

- Randolph, T. G. (1956). The descriptive features of food addiction. Addictive eating and drinking. *Quarterly Journal of Studies on Alcohol*, *17*, 198–224.
- Raynor, H. A., & Epstein, L. H. (2003). The relative-reinforcing value of food under differing levels of food deprivation and restriction, *Appetite*, *40*(1), 15–24.
- Rissanen, A., Hakala, P., Lissner, L., Mattlar, C.-E., Koskenvuo, M., & Ronnema, T. (2002). Acquired preference especially for dietary fat and obesity: A study of weight-discordant monozygotic twin pairs. *International Journal of Obesity*, *26*(7), 973–977.
- Ritchie, T., & Noble, E.P. (2003). Association of seven polymorphisms of the D2 dopamine receptor gene with brain receptor-binding characteristics. *Neurochemistry Research*, *28*(1), 73–82.
- Robinson, E., Sharps, M., Price, N., & Dallas, R. (2014). Eating like you are overweight: The effect of overweight models on food intake in a remote confederate study. *Appetite*, *82*, 119-123.
- Robinson, A. H., Adler, S., Stevens, S. B., Darcy, A. M., Morton, J. M., & Safer, D. L. (2014) What variables are associated with successful weight loss outcomes for bariatric surgery after 1 year? *Surgery for Obesity and Related Diseases*, *10*, 697-704.
- Robinson, T. E., & Berridge, K. C. (1993). The Neural Basis of Drug Craving: An incentive-sensitization theory of addiction. *Brain Research Reviews*, *18*, 247–291.
- Robinson, T. E., & Berridge, K. C. (2008). Review. The incentive sensitization theory of addiction: some current issues. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *363*(1507), 3137–3146.
- Rodin, J. & Slochower, J. (1976). Externality in the non-obese: Effects of environmental responsiveness on weight gain. *Journal of Personality and Social Psychology*, *33*, 338-344.
- Rogers, P. J., & Brunstrom, J. M. (2016). Appetite and energy balancing. *Physiology and Behavior*, *164*, 465-471.
- Rogers, P. J., & Hardman, C. A. (2015). Food reward. What it is and how to measure it. *Appetite*, *90*, 1–15.

- Rogers, P. J., & Smit, H. J. (2000). Food Craving and Food “Addiction”: A Critical Review of the Evidence From a Biopsychosocial Perspective. *Pharmacology Biochemistry and Behavior*, 66(1), 3–14.
- Rollins, B. Y., Dearing, K. K., & Epstein, L. H. (2010). Delay discounting moderates the effect of food reinforcement on energy intake among non-obese women. *Appetite*, 55(3), 420–425.
- Rollins, B.Y., Loken, E., & Birch, L.L. (2011). Preferences predict food intake from 5 to 11 years, but not in girls with higher weight concerns, dietary restraint, and % body fat. *Obesity*, 19(11), 2190-2197.
- Rolls, B. J., Van Duijvenvoorde, P. M., & Rolls, E. T. (1984). Pleasantness changes and food intake in a varied four-course meal. *Appetite*, 5(4), 337–348.
- Ronel N, Libman G. (2003). Eating Disorders and Recovery: Lessons from Overeaters Anonymous. *Clinical Social Work Journal*, 31(2),155–171.
- Rothmund, Y., Preuschhof, C., Bohner, G., Bauknecht, H.-C., Klingebiel, R., Flor, H., & Klapp, B. F. (2007). Differential activation of the dorsal striatum by high-calorie visual food stimuli in obese individuals. *NeuroImage*, 37(2), 410–421.
- Ruddock, H.K., Christiansen, P., Jones, A., Robinson, E., Field, M., & Hardman CA. (2015). Believing in food addiction: Helpful or counter-productive for eating behaviour? *Obesity*,24(6), 1238–1243.
- Ruddock, H. K., Dickson, J. M., Field, M., & Hardman, C. A. (2015). Eating to live or living to eat? Exploring the causal attributions of self-perceived food addiction. *Appetite*, 95, 262-268.
- Ruddock, H.K., Field, M., & Hardman, C.A. (2016). Food reward and calorie intake in self-perceived food addicts. *Appetite*.
- Russell-Mayhew, S., von Ranson, K. M., & Masson, P. C. (2010). How does overeaters anonymous help its members? A qualitative analysis. *European Eating Disorders Review*, 18(1), 33-42
- Salamone, J. D., Cousins, M. S., & Bucher, S. (1994). Anhedonia or anergia? Effects of haloperidol and nucleus accumbens dopamine depletion on instrumental response selection in a T-maze cost/benefit procedure. *Behavioural Brain Research*, 65(2), 221–229.

- Salbe, A. D., DelParigi, A., Pratley, R. E., Drewnowski, A., & Tataranni, P. A. (2004). Taste preferences and body weight changes in an obesity- prone population. *American Journal of Clinical Nutrition*, 79, 372–378
- Sato, Y., Ito, T., Udaka, N., Kanisawa, M., Noguchi, Y., Cushman, S.W., Satoh, S., (1996). Immunohistochemical localization of facilitated- diffusion glucose transporters in rat pancreatic islets. *Tissue Cell*, 28, 637–643.
- Sawaoka, T., Barnes, R. D., Blomquist, K. K., Masheb, R. M., & Grilo, C. M. (2012). Social anxiety and self-consciousness in binge eating disorder: associations with eating disorder psychopathology. *Comprehensive Psychiatry*, 53(6), 740–745.
- Schachter, S. & Gross, L.P. (1968). Manipulated time and eating behavior. *Journal of Personality and social psychology*, 10(2), 98-106.
- Schachter, S. (1971). Some extraordinary facts about obese humans and rats. *American Psychology*, 26, 129–44.
- Schlam, T. R., Wilson, N. L., Shoda, Y., Mischel, W., & Ayduk, O. (2013). Preschoolers' delay of gratification predicts their body mass 30 years later. *The Journal of Pediatrics*, 162, 90–93.
- Schmitz, F., Naumann, E., Biehl, S., & Svaldi, J. (2015). Gating of attention towards food stimuli in binge eating disorder. *Appetite*, 95, 368–374.
- Schoenmakers, T., Wiers, R.W., Jones, B.T., Bruce, G., & Jansen, A.T.M. (2006). Attentional re-training decreases attentional bias in heavy drinkers without generalization. *Addiction*, 102, 399-405
- Schubert, M. M., Desbrow, B., Sabapathy, S., & Leveritt, M. (2013). Acute exercise and subsequent energy intake. A meta-analysis. *Appetite*, 63, 92–104.
- Schulte, E. M., Avena, N. M., & Gearhardt, A. N. (2015). Which foods may be addictive? The roles of processing, fat content, and glycemic load. *Plos One*, 10, e0117959.
- Schulte, E. M., Potenza, M. N., & Gearhardt, A. N. (2016). A commentary on the “eating addiction” versus “food addiction” perspectives on addictive-like food consumption. *Appetite*.
- Schulte, E.M., Avena, N.M., & Gearhardt, A.N. (2015). Which foods may be addictive? The roles of processing, fat content, and glycemic load. *PloS one*, 10(2), e0117959.

- Schulz, S., & Laessle, R. G. (2012). Stress-induced laboratory eating behavior in obese women with binge eating disorder. *Appetite*, *58*(2), 457–461.
- Sedikides, C., & Strube, M. (1995). The multiply motivated self. *Personality and Social Psychology Bulletin*, *21*, 1330–1335.
- Seeyave, D. M., Coleman, S., Appugliese, D., Corwyn, R. F., Bradley, R. H., Davidson, N. S., ... Lumeng, J. C. (2009). Ability to delay gratification at age 4 years and risk of overweight at age 11 years. *Archives of Pediatrics & Adolescent Medicine*, *163*, 303–308.
- Shao, C., Li, Y., Jiang, K., Zhang, D., Xu, Y., Lin, L., Wang, Q., Zhao, M., Jin, L., (2006). Dopamine D4 receptor polymorphism modulates cue-elicited heroin craving in Chinese. *Psychopharmacology*, *186*, 185–190.
- Sheets, C. S., Peat, C. M., Berg, K. C., White, E. K., Bocchieri-Ricciardi, L., Chen, E. Y., et al. (2015) Post-operative predictors of outcome in bariatric surgery. *Obesity Surgery*, *25*, 330-345.
- Shibata, R., Kameishi, M., Kondoh, T., & Torii, K. (2009). Bilateral dopaminergic lesions in the ventral tegmental area of rats influence sucrose intake, but not umami and amino acid intake. *Physiology and Behavior*, *96*, 667–674.
- Slane, J. D., Burt, S. A., & Klump, K. L. (2012). Bulimic behaviors and alcohol use: shared genetic influences. *Behavior Genetics*, *42*(4), 603–613.
- Small, D. M., Jones-Gotman, M., & Dagher, A. (2003). Feeding-induced dopamine release in dorsal striatum correlates with meal pleasantness ratings in healthy human volunteers. *NeuroImage*, *19*(4), 1709–1715.
- Smith, K. S., & Berridge, K. C. (2005). The ventral pallidum and hedonic reward: neurochemical maps of sucrose “liking” and food intake. *The Journal of Neuroscience : The Official Journal of the Society for Neuroscience*, *25*(38), 8637–8649.
- Smith, K. S., Tindell, A. J., Aldridge, J. W., & Berridge, K. C. (2009). Ventral pallidum roles in reward and motivation. *Behavioural Brain Research*, *196*(2), 155–167.
- Sobik, L., Hutchison, K., & Craighead, L. (2005). Cue-elicited craving for food: a fresh approach to the study of binge eating. *Appetite*, *44*(3), 253–261.
- Soetens, B., & Braet, C. (2007). Information processing of food cues in overweight and normal weight adolescents. *British Journal of Health Psychology*, *12*(2), 285–304.

- Southon, A., Walder, K., Sanigorski, A.M., Zimmet, P., Nicholson, G.C., Kotowicz, M.A., Collier, G. (2003). The TaqIA and Ser311 Cys polymorphisms in the dopamine D2 receptor gene and obesity. *Diabetes, Nutrition, & Metabolism*, 16(1), 72–76.
- Spitz, M.R., Detry, M.A., Pillow, P., Hu, Y., Amos, C.I., Hong, W.K. & Wu, X., (2000). Variant alleles of the D2 dopamine receptor gene and obesity. *Nutrition Research*, 20(3), 371–380.
- Steele, K. E., Prokopowicz, G. P., Schweitzer, M. A., Magunsuon, T. H., Lidor, A. O., Kuwabawa, H., ... & Wong, D. F. (2010). Alterations of central dopamine receptors before and after gastric bypass surgery. *Obesity surgery*, 20(3), 369-374.
- Stephoe, A., & Wardle J. (2001). Locus of control and health behaviour revisited. A multivariate analysis of young adults from 18 countries. *British Journal of Psychology*, 92, 659–672.
- Stewart, S. H., & Samoluk, S. B. (1997). Effects of Short-Term Food Deprivation and Chronic Dietary Restraint on the Selective Processing of Appetitive-Related Cues. *International Journal of Eating Disorders*, 21(2), 129–135.
- Stice, E., & Yokum, S. (2016). Neural Vulnerability Factors That Increase Risk for Future Weight Gain. *Psychological Bulletin*, 142(5), 447–471.
- Stice, E., Figlewicz, D. P., Gosnell, B. A, Levine, A. S., & Pratt, W. E. (2012). The contribution of brain reward circuits to the obesity epidemic. *Neuroscience and Biobehavioral Reviews*, 37(9), 2047-2058.
- Stice, E., Spoor, S., Bohon, C., & Small, D. M. (2008). Relation Between Obesity and Blunted Striatal Response to Food Is Moderated by TaqIA A1 Allele. *Science*, 322, 449–452.
- Stice, E., Yokum, S., & Burger, K. S. (2013). Elevated reward region responsivity predicts future substance use onset but not overweight/obesity onset. *Biological Psychiatry*, 73(9), 869–876.
- Stice, E., Yokum, S., Bohon, C., Marti, N., & Smolen, A. (2010). NeuroImage Reward circuitry responsivity to food predicts future increases in body mass: Moderating effects of DRD2 and DRD4. *NeuroImage*, 50(4), 1618–1625.

- Stockburger, J., Hamm, A. O., Weike, A. I., & Schupp, H. T. (2008). Deprivation selectively modulates brain potentials to food pictures. *Behavioral Neuroscience, 122*, 936–942.
- Stockburger, J., Schmälzle, R., Flaisch, T., Bublatzky, F., & Schupp, H. T. (2009). The impact of hunger on food cue processing: An event-related brain potential study. *Neuroimage, 47*, 1819–1829.
- Stoeckel, L. E., Weller, R. E., Cook, E. W., Twieg, D. B., Knowlton, R. C., & Cox, J. E. (2008). Widespread reward-system activation in obese women in response to pictures of high-calorie foods. *NeuroImage, 41*(2), 636–647.
- Stunkard, A. J., & Messick, S. (1985). The Three-Factor Eating Questionnaire to Measure Dietary Restraint, Disinhibition and Hunger. *Journal of Psychosomatic Research, 29*(1), 71–83.
- Sullivan, S. A., & Birch, L. L. (1990). Pass the sugar, pass the salt. Experience dictates preference. *Developmental Psychology, 26*, 546–555.
- Suzuki, J., Haimovici, F., & Chang, G. (2012). Alcohol use disorders after bariatric surgery. *Obesity Surgery, 22*(2), 201–207.
- Svaldi, J., Griepenstroh, J., Tuschen-Caffier, B., & Ehring, T. (2012). Emotion regulation deficits in eating disorders: a marker of eating pathology or general psychopathology? *Psychiatry Research, 197*, 103–111.
- Teegarden, S.L., & Bale, T.L. (2007). Decreases in dietary preference produce increased emotionality and risk for dietary relapse. *Biological Psychiatry, 61*, 1021–1029.
- Teegarden, S.L., Nestler, E.J., & Bale, T.L. (2008). Delta FosB-mediated alterations in dopamine signaling are normalized by a palatable high-fat diet. *Biological Psychiatry, 64*, 941–950.
- Temple, J. L., Bulkley, A. M., Badawy, R. L., Krause, N., Mccann, S., & Epstein, L. H. (2009). Differential effects of daily snack food intake on the reinforcing value of food in obese and nonobese women. *American Journal of Clinical Nutrition, 90*, 304–313.
- Temple, J. L., Legierski, C. M., Giacomelli, A. M., Salvy, S.-J., & Epstein, L. H. (2008). Overweight children find food more reinforcing and consume more energy than do nonoverweight children. *The American Journal of Clinical Nutrition, 87*, 1121–1127.

- Thomas, G.N., Critchley, J.A., Tomlinson, B., Cockram, C.S., & Chan, J.C. (2001). Relationships between the TaqI polymorphism of the dopamine D2 receptor and blood pressure in hyperglycaemic and normoglycaemic Chinese subjects. *Clinical Endocrinology*, *55*(5), 605–611.
- Thush, C., Wiers, R.W., Ames, S.L., Grenard, J.L., Sussman, S., & Stacy, A.W. (2008). Interactions between implicit and explicit cognition and working memory capacity in the prediction of alcohol use in at-risk adolescents. *Drug and Alcohol Dependence*, *94*, 116–124.
- Troisi, J. D., & Gabriel, S. (2011). Chicken soup really is good for the soul: “Comfort food” fulfills the need to belong. *Psychological Science*, *22*(6), 747–753.
- Tschop, M., Smiley, D.L., & Heiman, M.L. (2000). Ghrelin induces adiposity in rodents. *Nature*, *407*, 908–913.
- Tuomisto, T., Hetherington, M. M., Morris, M.-F., Tuomisto, M. T., Turjanmaa, V., & Lappalainen, R. (1999). Psychological and Physiological Characteristics of Sweet Food “Addiction.” *International Journal of Eating Disorders*, *25*(2), 169–175.
- Tupala, E., Hall, H., Bergström, K., Mantere, T., Räsänen, P., Särkioja, R., Tiihonen, J., 2003. Dopamine D2 receptors and transporters in type 1 and 2 alcoholics measured with human whole hemisphere autoradiography. *Hum. Brain Mapp.* *20* (2), 91–102.
- Ullman, J. B. (2001). Structural equation modeling. In B. G. Tabachnick & L. S. Fidell (2001). *Using Multivariate Statistics* (4th ed& pp 653- 771). Needham Heights, MA: Allyn & Bacon.
- Vainik, U., Neseliler, S., Konstabel, K., Fellows, L. K., & Dagher, A. (2015). Eating traits questionnaires as a continuum of a single concept. Uncontrolled eating. *Appetite*, *90*, 229–239.
- van den Akker, K., Jansen, A., Frentz, F., & Havermans, R. C. (2013). Impulsivity makes more susceptible to overeating after contextual appetitive conditioning. *Appetite*, *70*, 73–80.
- Volkow, N.D., Wang, G.J., Telang, F., Fowler, J.S., Thanos, P.K., Logan, J. et al. (2008). Low dopamine striatal D2 receptors are associated with prefrontal metabolism in obese subjects: Possible contributing factors. *NeuroImage*, *42*, 1537–1543.

- Volkow, N. D., & Wise, R. A. (2005). How can drug addiction help us understand obesity? *Nature Neuroscience*, 8(5), 555–560.
- Volkow, N. D., Chang, L., Wang, G.-J., Fowler, J. S., Ding, Y.-S., Sedler, M., et al. (2001). Low level of brain dopamine D(2) receptors in methamphetamine abusers: Association with metabolism in the orbitofrontal cortex. *American Journal of Psychiatry*, 158, 2015–2021.
- Volkow, N. D., Fowler, J. S., Wang, G., & Goldstein, R. Z. (2002). Role of Dopamine, the Frontal Cortex and Memory Circuits in Drug Addiction : Insight from Imaging Studies, 624, 610–624.
- Volkow, N. D., Wang, G.-J., & Baler, R. D. (2011). Reward, dopamine and the control of food intake: implications for obesity. *Trends in Cognitive Sciences*, 15(1), 37–46.
- Volkow, N. D., Wang, G.-J., Fowler, J. S., Logan, J., Jayne, M., Franceschi, D., ... Pappas, N. (2002). “Nonhedonic” food motivation in humans involves dopamine in the dorsal striatum and methylphenidate amplifies this effect. *Synapse (New York, N.Y.)*, 44(3), 175–180.
- Volkow, N. D., Wang, G.-J., Telang, F., Fowler, J. S., Thanos, P. K., Logan, J., ... Pradhan, K. (2008). Low dopamine striatal D2 receptors are associated with prefrontal metabolism in obese subjects: possible contributing factors. *NeuroImage*, 42(4), 1537–1543.
- Volkow, N. D., Wang, G., Telang, F., Fowler, J. S., Logan, J., Jayne, M., ... Wong, C. (2007). Profound Decreases in Dopamine Release in Striatum in Detoxified Alcoholics: Possible Orbitofrontal Involvement. *The Journal of Neuroscience*, 27(46), 12700–12706.
- Wallston K. (1992). Hocus-pocus, the focus isn't strictly on locus. Rotter's social learning theory modified for health. *Cognitive Therapy Research*, 16, 183–199.
- Wang, G. J., Volkow, N. D., & Fowler, J. S. (2002). The role of dopamine in motivation for food in humans: Implications for obesity. *Expert Opinion on Therapeutic Targets*, 6, 601–609.
- Wang, G.-J., Volkow, N. D., Telang, F., Jayne, M., Ma, J., Rao, M., ... Fowler, J. S. (2004). Exposure to appetitive food stimuli markedly activates the human brain. *NeuroImage*, 21(4), 1790–1797.

- Wang, G., Geliebter, A., Volkow, N. D., Telang, F. W., Logan, J., Jayne, M. C., ... Fowler, J. S. (2011). Enhanced striatal Dopamine Release During Food stimulation in Binge Eating Disorder. *Obesity, 19*(8), 1601–1608.
- Wang, G., Volkow, N. D., Logan, J., Pappas, N. R., Wong, C. T., Zhu, W., & Netusil, N. (2001). Brain dopamine and obesity. *The Lancet, 357*(9253), 354–357.
- Wardle, J., Haase, A. M., Steptoe, A., Nillapun, M., Jonwutiwes, K., & Bellisle, F. (2004). Gender differences in food choice: The contribution of health beliefs and dieting. *Annals of Behavioral Medicine, 27*(2), 107–116.
- Warren, C. & Cooper, P. J. (1988). Psychological effects of dieting. *British Journal of Clinical Psychology, 27*, 269-270.
- Wegner, K. E., Smyth, J. M., Crosby, R. D., Wittrock, D., Wonderlich, S.A, & Mitchell, J. E. (2002). An evaluation of the relationship between mood and binge eating in the natural environment using ecological momentary assessment. *The International Journal of Eating Disorders, 32*(3), 352–361.
- Weiner, B. (1974). *Achievement motivation and attribution theory*. Morristown, N.J.: General Learning Press.
- Weiner, B., Frieze, I. H., Kukla, A., Reed, L., Rest, S., & Rosenbaum, R. M. (1971). *Perceiving the causes of success and failure*. Morristown, NJ: General Learning Press.
- Werthmann, J., Field, M., Roefs, A., Nederkoorn, C., & Jansen, A. (2014). Attention bias for chocolate increases chocolate consumption - An attention bias modification study. *Journal of Behavior Therapy and Experimental Psychiatry, 45*(1), 136–143.
- Werthmann, J., Renner, F., Roefs, A., Huibers, M. J. H., Plumanns, L., Krott, N., et al. (2014). Looking at food in sad mood: do attention biases lead emotional eaters into overeating after a negative MI? *Eating Behaviours, 15*, 230e236.
- Werthmann, J., Roefs, A., Nederkoorn, C., & Jansen, A. (2013). Desire lies in the eyes: attention bias for chocolate is related to craving and self-endorsed eating permission. *Appetite, 70*, 81e89
- Werthmann, J., Roefs, A., Nederkoorn, C., Mogg, K., Bradley, B. P., & Jansen, A. (2011). Can(not) take my eyes off it: attention bias for food in overweight participants. *Health Psychology, 30*(5), 561–569.

- Westenhoefer, J., Broeckmann, P., Munch, A. K., & Pudel, V. (1994). Cognitive control of eating behaviour and the disinhibition effect. *Appetite*, *23*, 27-41.
- Westenhoefer, J., Pudel, V., and Maus, N. (1990). Some restrictions on dietary restraint. *Appetite*, *14*, 137–141.
- Westerterp-Plantenga, M.S., Wouters, L., & ten Hoor., F. (1990). Deceleration in cumulative food intake curves, changes in body temperature and diet-induced thermogenesis. *Physiology and Behavior*, *48*, 831-836.
- World Health Organization (2016). *Obesity and overweight. Fact sheet*. Available at: <http://www.who.int/mediacentre/factsheets/fs311/en>. (accessed 07 June 2016).
- White, H. R., & Labouvie, E. W. (1989). Toward the assessment of adolescent problem drinking. *Journal of Studies on Alcohol*, *50*, 30–37.
- Wiers, R.W., Bartholow, B.D., van den Wildenberg, E., Thush, C., Engels, R.C.M.E., Sher, K.J., Grenard, J., Ames, S.L., Stacy, A.W., 2007. Automatic and controlled processes and the development of addictive behaviors in adolescents: a review and a model. *Pharmacology, Biochemistry and Behavior*, *86*(82), 263–283.
- Wilens, T. E., Martelon, M., Anderson, J. P., Shelley-Abrahamson, R., & Biederman, J. (2013). Difficulties in emotional regulation and substance use disorders: A controlled family study of bipolar adolescents. *Drug and Alcohol Dependence*, *132*, 114–121.
- Williamson, L. (2012). Destigmatizing alcohol dependence: The requirement for an ethical (not only medical) remedy. *American Journal of Public Health*, *102*, 5–9.
- Wilson, G.T., Perrin, N.A., Rosselli, F., Striegel-Moore, R.H., Debar, L.L., & Kraemer, H.C. (2009). Beliefs about eating and eating disorders. *Eating Behaviour*, *10*, 157–160.
- Wise, R. A. (2013). Dual roles of dopamine in food and drug seeking: the drive-reward paradox. *Biological Psychiatry*, *73*(9), 819–826.
- Woods, A. M., Racine, S. E., & Klump, K. L. (2010). Examining the relationship between dietary restraint and binge eating: Differential effects of major and minor stressors. *Eating Behaviors*, *11*(4), 276–280.
- Yokum, S., Ng, J., & Stice, E. (2011). Attentional bias to food images associated with elevated weight and future weight gain: an fMRI study. *Obesity*, *19*(9), 1775–1783.

- Zandstra, E. H., Graaf, C. De, Mela, D. J., & Staveren, W. A. Van. (2000). Short- and long-term effects of changes in pleasantness on food intake. *Appetite*, *34*, 253–260.
- Zhang, M., & Kelley, A. E. (2002). Intake of saccharin, salt, and ethanol solutions is increased by infusion of a mu opioid agonist into the nucleus accumbens. *Psychopharmacology*, *159*(4), 415–423.
- Ziauddeen, H., & Fletcher, P. C. (2013). Is food addiction a valid and useful concept? *Obesity Reviews*, *14*(1), 19–28.
- Ziauddeen, H., Alonso-Alonso, M., Hill, J. O., Kelley, M., & Khan, N. A. (2015). Obesity and the Neurocognitive Basis of Food Reward and the Control of Intake. *Advances in Nutrition*, 3–5.
- Ziauddeen, H., Farooqi, I. S., & Fletcher, P. C. (2012). Obesity and the brain: how convincing is the addiction model? *Nature Reviews Neuroscience*, *13*, 279–286.
- Zigman, J.M., Nakano, Y., Coppari, R., Balthasar, N., Marcus, J.N., Lee, C.E., ...& Williams, T.D. (2005). Mice lacking ghrelin receptors resist the development of diet-induced obesity. *The Journal of Clinical Investigation*, *115*(12), 3564-3572.

Appendix A: Addictive eating behaviour scale (Chapter 5)

	Never	Rarely	Sometimes	Most of the time	Always
1. I continue to eat despite feeling full					
2. I serve myself overly large portions					
3. I find it difficult to limit what/how much I eat					
4. I binge eat					
5. When it comes to food, I tend to over-indulge					
6. I am easily able to make healthy food choices					
7. Once I start eating certain foods, I can't stop until there's nothing left					
8. Despite trying to eat healthy, I end up eating 'naughty' foods					
9. I eat until I feel sick					
10. I continue to eat certain unhealthy foods despite being aware of their effects on my health					
	Strongly disagree	Disagree	Neither agree/disagree	Agree	Strongly agree
11. I tend NOT to buy processed foods that are high in fat, salt, & sugar					
12. I don't eat a lot of high fat/sugar foods					
13. I have gained weight as a result of my overeating					
14. I believe I have a healthy diet					
15. I don't tend to overeat					
16. I feel unable to control my weight					

AEBS scoring Instructions:

Each item is given a score ranging from 1 ('Never' or 'Strongly disagree') to 5 ('Always' to 'Strongly Agree'). Items 6, 11, 12, 14, 15 are REVERSE scored (i.e. 'Always/Strongly agree' = 1; and 'Never/Strongly disagree' =5).

The scale provides total score (max score = 80), and 2 subscale scores:

Overeating/weight gain subscale (max score = 50): Items 1-5, 7, 9, 13, 15, and 16.

Unhealthy eating/low self-control subscale (max score = 30): Items 6, 8, 10, 11, 12, 14.

Appendix B: Mood and taste ratings (Chapter 6)

Participants provided ratings, using 100mm VAS scales, for the following moods:

I feel tense/anxious/nervous/on edge
I feel mentally alert/attentive/observant
I feel motivated
I feel hot/sweaty
I feel physically tired
I feel clearheaded
I feel miserable/dejected
I feel stressed
I feel friendly/sociable
I feel mentally fatigued/drained/worn out
I feel relaxed/calm/at ease
I feel strange/weird/not my usual self
I feel sleepy/drowsy/half awake
I feel energetic/active/lively
My head aches/I feel head-achy
I feel able to take on a physically demanding task
I feel able to concentrate/able to focus
I feel angry/cross/annoyed
I feel lethargic/sluggish
I feel cheerful/happy
My heart is pounding/racing

Participants completed the following taste ratings, for the chocolate and crisps, using 100mm VAS scales:

Sweet
Salty
Enjoyable
Fresh
Bland
Crunchy
Crumbly

Appendix C: Supplementary results (Chapter 7)

Table A1. Results from MANOVA with eating attributions as dependent variables, and condition as the independent variable.

Attribution	Condition			Significance testing
	Low-guilt	Control	High-guilt	
“I felt hungry”	2.53(2.00)	1.67(1.27)	2.07(2.35)	$F(2,84)=1.55, p=.219$
“The foods were really addictive”	6.50(2.45)	6.33(2.32)	6.52(2.38)	$F(2,84)=.05, p=.948$
“To relieve negative emotions”	8.63(2.08)	8.60(2.06)	6.52(2.38)	$F(2,84)=1.27, p=.286$
“I couldn’t control myself”	8.10(1.97)	7.77(2.29)	7.22(2.99)	$F(2,84)=.94, p=.396$
“I didn’t want to turn down free food”	5.07(2.41)	5.40(2.75)	5.00(2.63)	$F(2,84)=.20, p=.820$
“I was craving something sweet/salty”	4.70(2.15)	5.10(2.31)	5.85(2.43)	$F(2,84)=1.83, p=.167$
“Because they were just there”	4.27(2.21)	4.77(2.31)	4.41(2.31)	$F(2,84)=.38, p=.685$
“I liked the taste of the foods”	3.37(1.65)	3.53(1.91)	3.67(2.22)	$F(2,84)=.17, p=.841$
“Out of habit – I eat when I’m watching TV”	4.97(2.64)	6.60(2.44)	6.33(2.91)	$F(2,84)=3.22, p=.045$
“I wanted to fill myself up”	5.80(2.40)	4.70(2.73)	4.74(2.36)	$F(2,84)=1.83, p=.167$