Drivers of obesity?

Characterising biopsychological and environmental factors associated with overeating

by

Christle Coxon

MSc Sports & Exercise Science

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

Doctor of Philosophy

Department of Psychology

University of Roehampton

2020

Keywords

Appetite; Behaviour; Body composition; Eating behaviours; Eating styles; Emotional eating; Energy density; Energy intake; Environment; Fat free mass; Fat mass; Food choice; Genetic traits; Impulsivity; Obesity; Overeating; Overweight; Palatability; Personality traits; Physical activity; Portion sizes; Resting metabolic rate; Restrained eating; Sensitivity to reward; Socioeconomic status; Uncontrolled eating; Weight gain

Abstract

The prevalence of obesity has increased globally over the past four decades, and one of the primary factors implicated is the increased availability of highly processed, inexpensive, energy-dense foods that offer palatability but little nutritional value. However, not all humans are obese, suggesting that the individual variation in several physiological, psychological, biological and social-economic factors play a role in moderating eating behaviour in response to palatable food cues. The purpose of this thesis is to investigate (a) how the increased availability of highly palatable energy-dense foods, served in large portions yet containing little nutritional value, impacts appetite and amounts eaten; (b) how the individual variation in psychological, physiological, biological and socio-economic characteristics increases the susceptibility to overeating these foods.

The first two studies investigated the appetite and eating behaviour responses to consuming foods containing high levels of fat and sugar (Chapter 3 and 4). In a sample of twenty-five adults, the addition of sweetness to a high-fat food significantly enhanced the palatability and desire to eat on initial tasting. During the early stages of the meal, sweetness sustained feelings of hunger and the motivation to eat. These responses were associated with a higher intake of food (Chapter 3).In a second study, in ten participants, sweetness sustained prandial acyl-ghrelin levels, but these responses were not associated with increased food intake (Chapter 4).

The third study investigated how the individual variation in lifestyle factors (level of physical activity), dietary protein requirements and physiological characteristics influenced the appetite and eating behavioural responses to low protein intake (Chapter 5). Level of physical activity did not influence the response to a low protein meal as marginal differences were observed between the active (n =

9), moderately active (*n* = 9), and sedentary group (*n* = 8). However, across the group it was found that body composition and resting metabolic rate was strongly associated with energy and protein intake.

The fourth study investigated the biological, psychological, anthropometric and socio-economic factors associated with obesity-related eating behaviours and attitudes toward food in a large community-based sample (*n* = 560, 240 men, 320 women). In this sample, overweight individuals or individuals with obesity were more likely to have the at-risk AA/AT *FTO* allele and be of lower socioeconomic status; in addition, they showed a greater motivation to eat energy-dense foods and reported eating these foods more often. Furthermore, when viewing images of fixed portions of food, overweight and obese individuals reported lower anticipated satiation for energy-dense foods (Chapter 6).

The final study investigated the relationship between estimated portion size chosen for lunch and obesity in a large community-based sample (*n* = 555, 235 men, 306 women). The maximum food portion size for five foods was predicted by resting metabolic rate, body fat (waist-to-height ratio), age and sex. Body mass index and fat mass did not significantly predict portion size. Individuals with a higher resting metabolic rate chose larger food portions, while a higher waist-to-height ratio predicted smaller portion sizes. Across the sample, the maximum portion size of high energy-dense foods chosen provided more energy than the energy provided by portions of low energy-dense foods (Chapter 7).

Taken together, these results suggest a Western-style diet of energy-dense foods, rich in fat and sugar, influences appetite and eating behaviour. These foods heighten the sensory experience and reward response when eating, consequently encouraging a higher food intake. The individual variation in protein need and level of physical activity may modify the response to a low protein diet or meal; however further research is needed to investigate this research question. This thesis observed the individual variation in body composition and metabolic rate may direct food intake, macronutrient balance, and decisions about food portion size. The individual variation in restrained eating, weight status and inheritable traits may also increase the susceptibility to palatable food cues, meaning that these individuals are at risk of overeating. Individuals of lower socioeconomic status may also be vulnerable to overeating energy-dense foods as they demonstrate a heightened reward response and preference for high energy-dense foods yet find these foods to be less satisfying. These findings provide an informative insight into the factors that influence overeating and the development of obesity. Importantly, these insights should advance the development of research in this area.

Contents

List of Figures

List of Tables

List of abbreviations

AA or AT	Polymorphisms of *FTO* A allele variant
BAS	Behavioural Approach System
BIS	Behavioural Inhibition System
BIS-15	Barratt Impulsiveness Scale
BMI	Body Mass Index
BW	Body Weight
CHO	Carbohydrate
CI	Confidence Interval
CR	Cognitive Restraint
EE	Energy Expenditure
EE	Emotional Eating
EI	Energy Intake
FFM	Fat Free Mass
FM	Fat Mass
FTO	Fat Mass and Obesity Associated gene
g	gram
GFN	Geometric Framework for Nutrition
HP	High Protein
kcal	kilocalorie
kg	kilogram
kJ	kilojoule
LP	Low Protein
MET	Metabolic Equivalent
NHS	National Health Service

PANAS	Positive and Negative Affect Scale
PE	Percentage of energy obtained from protein
PFS	Power of Food Scale
PLH	Protein Leverage Hypothesis
RMR	Resting Metabolic Rate
SEM	Standard Error of Mean
SES	Socioeconomic Status
SIPM	Sussex Ingestion Pattern Monitor
TDEE	Total Daily Energy Expenditure
TFEQ	Three-Factor Eating Questionnaire
UE	Uncontrolled Eating
UK	United Kingdom
VAS	Visual Analog Scale

Statement of Original Authorship

To the best of my knowledge, the thesis contains no material previously published or written by another person, except where due reference is made. The work contained in this thesis has not been previously submitted to meet requirements for an award at this or any other higher education institution.

Signed

Dated 4th October 2019

Acknowledgements

This thesis represents one of the most challenging tasks that I have ever undertaken, and I could not have completed the journey without the support of supervisors, colleagues, study participants, clients, friends and family.

Firstly, I would like to thank my supervisory team, Dr E. Leigh Gibson and Professor Lewis Halsey, for their constant support and guidance throughout the doctoral programme. Their expertise has helped to steer my work in the right direction, and their encouragement instilled the confidence I needed to write the thesis.

My Director of Studies, Dr Leigh Gibson, provided invaluable insight, direction and support. Through Leigh's many connections, I had the opportunity to be involved in several research projects; including a research collaboration with Dr Tony Goldstone at Imperial College London investigating appetite changes following weight-loss surgery; a science feature experiment for a BBC's children's television documentary on childhood obesity; an educational documentary; and involvement in the 'Live Science' research project held at the Science Museum, London, in 2015. Leigh encouraged me to attend numerous conference and meetings, and present research at the annual meetings of the British Feeding and Drinking Group and International Society for Behavioral Nutrition and Physical Activity (ISBNPA). I'm incredibly grateful to Leigh for his expertise and ongoing support throughout the PhD term, especially when the journey involved some unexpected challenges.

My associate supervisor Professor Lewis Halsey also was invaluable in the PhD process. Lewis helped to maintain the Lifesciences aspect of the PhD but also continually challenged the status quo. Through our discussions, he helped to build my confidence in scientific reasoning, writing and understanding. Our meetings would always stray to discussing current views on British politics, gender inequality or problems with the social welfare system. These conversations were stimulating and thought-provoking, nonetheless.

I want to thank Dr Michael Patterson for teaching me the laboratory techniques required for my studies, and for his assistance in the data collection and laboratory work during my pregnancy.

The funding for this research came in the form of a research studentship, provided by the Department of Psychology at the University of Roehampton. I want to thank the University for providing this funding as it allowed me to research in a full-time capacity. I also want to thank the research participants who willingly committed their time (and taste buds) to participate in my studies.

I want to thank my parents-in-law, Trevor and Pat Coxon, who looked after our son Joshua intermittently during the write-up phase of the thesis.

I want to thank my parents, Gavin and Carla Johnson, for their steadfast love and support. Although they considered that a PhD might be stressful while trying to balance family and work life, they were incredibly supportive of my research topic and encouraged me to move forward in the area. Over the years, they were a source of emotional and spiritual guidance and strength, and they never cessed to provide this support despite my father being terminally ill with neuroendocrine tumours. Following the submission of this thesis, my father passed away on 20th October 2019.

I am deeply grateful to my husband, Gary, who has been a pillar of strength throughout the journey. His patience, love and support were invaluable to me over the years, and he encouraged me to move forward and keep going. I am deeply appreciative of his commitment to our family particularly during the write-up phase of the PhD. Over this time, Gary gave up the best part of two years of weekends to look after our son and in doing so, provided Joshua with a stable home environment needed for the many days when ‘Mommy gone to work’. I could not have completed this journey without you, Gary – thank-you.

Lastly, I would like to give glory, praise and honour to my Heavenly Father, who has been my source of faith, strength, peace, and hope. For ‘The LORD is my rock, my fortress, and my deliverer…in whom I take refuge, my shield, and the horn of my salvation, my stronghold’, Psalm 18 v 2.

Chapter 1: Introduction

1.1 Outline of the research question in context: The global prevalence of obesity and socioeconomic burden

The prevalence of obesity has increased globally over the past four decades, and it is now estimated that over 1.9 billion adults (aged 18 years or older) are overweight or obese (overweight: Body Mass Index (BMI) of 25 to 29.9 $kg.m^{-2}$, or obese: BMI >30 $kg.m^{-2}$ (World Health Organisation, 2019). The worldwide increase in obesity has risen from 3.2 to 10.8% in men, and from 6.4 to 14.9% in women since the 1970s (NCD Risk Factor Collaboration (NCD-RisC), 2016). These trends have also been reported in children and adolescents where prevalence has increased from 0.7 to 5.6% in boys and 0.9 to 7.8% in girls (NCD Risk Factor Collaboration (NCD-RisC), 2017). Notably, the rates of increase in developed countries have begun to plateau (Blüher, 2019; Jebb, 2017). However, obesity rates have continued to rise in low to middle-income developing countries in south and south-east Asia, the Caribbean and southern Latin America (Blüher, 2019; Jaacks et al., 2019). In England, it is estimated that approximately 64% of adults are overweight, of whom 26% men and 27% of women are now classed as obese (Health Survey for England, 2018). The prevalence of childhood obesity has remained stable over the past decade for children aged 4-5 years (10%); however there is a deprivation gap observed as rates decreased amongst children living in the least deprived regions, but increased in children living in the most deprived areas (The Health and Social Care Information Centre (NHS Digital), 2018a). In children aged 10-11 years, obesity prevalence increased to 20% and highest increase was observed for children living in the most deprived areas (The Health and Social Care Information Centre (NHS Digital), 2018b).

Obesity is associated with many comorbidities and disabilities. Diseases such as type 2 diabetes (Pedersen, 2013), cardiovascular disease and hypertension (Chrostowska et al., 2013), renal, breast, endometrial, adenocarcinoma and colon cancer (Arnold et al., 2016; Boeing, 2013), osteoarthritis and mobility disability (Forhan & Gill, 2013), and depression (Taylor et al., 2013) are all significantly associated with being overweight or obese. Furthermore, the estimated death rate attributed to obesity is approximately 5% of deaths each year (Dobbs et al., 2014), and the life expectancy from the age 40 years and older is estimated to be 4.2 years shorter in obese men and 3.5 years shorter in obese women (Bhaskaran et al., 2018). The associated health costs to the national health services in both the United States and the UK represent a substantial financial burden (Lehnert et al., 2013). In the US, the most expensive obesity-related comorbidities are hypertensive diseases, dyslipidaemia and osteoarthritis which are projected to cost over $18 million per annum to treat 100 000 patients (Li et al., 2015). In the UK, healthcare costs attributed to the states of being overweight or obese are estimated to increase annually by 12 and 36%, respectively (Kent et al., 2017). As such, the estimated financial burden associated with obesity is expected to increase by $48 - 66 billion per year in the US and £1.9 - 2 billion in the UK by 2030 (Wang et al., 2011). Since the associated health, medical and economical concerns are vast, there is an urgent need to understand the forces driving the obesity epidemic and to prioritise obesity treatment and prevention strategies.

1.2 The Obesity System Map

The increasing prevalence of obesity has prompted extensive research into understanding why individuals are becoming obese. Accordingly, over the past four decades, research studies have identified numerous physiological, biological, psychological, environmental and socio-economic factors that are involved in the development of the obese condition. To summarise the relevant research, the Foresight Programme of the UK Government Office for Science published the Obesity System Map in 2007 which conceptualises the relevant factors and their interactions on obesity (Please refer to figure 1, Foresight, 2007). The map identifies 108 variables arranged into seven clusters, including 'food production', 'food consumption', 'social psychology', 'individual psychology', 'physical activity environment', 'individual physical activity' and 'individual physiology'. At the centre of the map is energy balance, characterised by the balance between energy intake and expenditure and the map models how these variables interact (either positively or negatively) to influence energy balance. By its very nature, the map is detailed and complex (Finegood et al., 2010), and many of the areas have been developed further by research published since its release in 2007. However, there are two messages that I have drawn from this publication. First, despite its complexity, the map describes how the obese condition develops from a chronic imbalance between intake and expenditure, although there may be many pathways that lead to these imbalances. The predominant view is that obesity develops because individuals are consuming too much food relative to their energy requirements, yet not expending enough energy to match their intake (Crino et al., 2015; Schwartz et al., 2017). If energy intake exceeds energy expended, a positive energy balance occurs, and over time leads to increased body weight, excess adiposity and eventually obesity (Hill et al., 2012; Hill, 2006).

The second observation is that these variables and their clusters broadly fit into two categories: those that are intrinsic to the individual (referring to the clusters of 'biology', 'individual activity' and 'individual psychology') and those that are extrinsic to the individual and describe the environmental impact on individuals behaviour ('societal influences', 'food production', 'food consumption', 'activity environment'). Many recognise that the modern, Western environment has played a crucial role in driving the increased prevalence of obesity. There have been many socio-economic, agricultural and technological developments that have changed the way humans now move and eat, notably with the increased availability of processed, energy-dense, palatable foods, and reduced need to participate in energetically demanding activities (Jaacks et al., 2019; Popkin, 2006; Swinburn et al., 2011). Evidently, a large proportion of the global and local population are not obese, which indicates that some individuals are more susceptible to these environmental factors than others. This observation is not only relevant for our understanding of the aetiology of obesity, but also in the development of effective interventions and strategies that will reverse obesity trends. The environmental drivers and interaction of the individual are the two predominant themes explored in my thesis.

1.3 Environmental drivers of the obesity epidemic

Over the past century, there have been dramatic developments in economic, technological, agricultural, and social sectors that have fundamentally changed how humans live. The advances in technology and the expansion of global trade systems have improved the manufacturing, production and distribution of food, yet these changes have led to the increased production of processed, highly palatable, energy-dense foods (James, 2008; Popkin, 2015; Swinburn et al., 2011). This has shifted dietary intake away from traditional diets containing unprocessed or minimally processed, native foods, to Western diets consisting of heavily processed (ultra-processed) foods rich in fat and sugars, a phenomenon described as "Nutrition transition" (Monteiro et al., 2017; Popkin, 2017). There is also improved accessibility to these foods, as increases in population density has increased the proportion of fast food outlets, restaurants, supermarkets and retailers found in urban areas (Burgoine et al., 2016; Maguire et al., 2015). The current environment is no longer food scarce, but abundant in food supply (Penney et al., 2018). Moreover, the use of smart marketing tactics through television, social media and print media, further entices the consumer to purchase these foods, making food tempting and difficult to resist (Moodie et al., 2013), therefore increasing the likelihood to eat.

There has also been a dramatic shift in activity levels over this period. Improvements in technology and increased automation has reduced the need to participate in energy-demanding activities that previously formed part of occupational work, domestic work and travel, consequently increasing sedentary behaviour (Church et al., 2011; Ng & Popkin, 2012). It is noted, however, that activity levels declined rapidly between the 1970s and 2000s yet have remained relatively stable over the last two decades while obesity levels have dramatically increased (Westerterp & Speakman, 2008). This suggests that the energy imbalance causing obesity is driven predominantly by increased energy intake (Swinburn et al., 2011). Indeed, population-level studies reveal a substantial increase in daily energy intake over the past four decades (Austin & Krueger, 2013; Austin et al., 2011; Yancy et al., 2013), in particular, an increased intake of energy from fat, sugar and alcohol (Austin & Krueger, 2013), and the profile of macronutrient supply has changed to favour fat and carbohydrate (Martinez-Cordero et al., 2012; Simpson & Raubenheimer, 2005). Adults are less active and participate in more sedentary activities, such as watching television, playing video games, listening to music and engaging in sedentary occupations, than manual labour and domestic duties (Chaput et al., 2009; Church et al., 2011; Giallonardo & Warburton, 2016; Ng & Popkin, 2012). These changes to modern, Western society have led to the environment aptly described as obesogenic.

The view that obesity develops from chronic overconsumption of food suggests that the current food environment has a profound impact on human eating behaviour. Accordingly, studies have identified that environmental factors such as food palatability, energy density and portion size, strongly encourage overeating in humans (Ledikwe et al., 2005; McCrory et al., 2006; Rolls, 2018). There are, however, several gaps in the scientific literature that remain unexplored. The combination of high levels of fat and tastants (sweet or salty) greatly enhances food palatability and is an essential feature of Western cuisine. Palatability has an appetising effect on ingestive behaviour by stimulating hunger in the early stages of eating and encouraging the consumption of more food (Yeomans, 2000; Yeomans et al., 2004). However, less is known about how palatable combinations of fat and sweetness in food influence appetite and eating behaviour, and whether these foods elicit a more pleasurable and rewarding eating experience that then encourages overeating. An understanding of underlying appetite mechanisms is vital as previous research has focused on the hypothesis that individuals with obesity have a ‘sweet-tooth’ and overeat sweet foods. Yet, on the contrary, the preference for sweetness is inversely related to obesity, and obese demonstrate a higher preference for high-fat foods, particularly those containing high levels of salt or sugar (Bartoshuk et al., 2006; Cox et al., 1999; Lampuré et al., 2016). Moreover, there is evidence that the obese may be more sensitive to detection of sweetness, showing higher intensity ratings and lower detection thresholds in psychophysical tests using a range of sucrose concentrations in water (Hardikar et al., 2017; Pasquet et al., 2007). Notably other studies have not confirmed this finding or report a reduced taste sensitivity in obese individuals (Bertoli, 2014; Pepino et al., 2010; Simchen et al., 2006). However, there may be an interaction between fat content and tastant such that the high-fat content masks sensory perception and allows for a greater intake of food. Therefore, understanding the link between gustatory perception and obesity is crucial to determine how high-fat, sweetened foods influences appetite and eating behaviour.

Secondly, a primary feature of the Western diet is that the highly processed foods on offer have low nutritional value, with a lower proportion of micronutrients, dietary fibre and protein (Martínez Steele et al., 2017). The Protein Leverage Hypothesis proposes that human appetite is strongly regulated by the intake of dietary protein, such that if intake is insufficient, appetite is stimulated and food intake is encouraged until a sufficient protein intake is achieved (Simpson & Raubenheimer, 2005). This phenomenon is widely observed in non-human animals (Booth & Toates, 1974; Gibson & Booth, 1986; Gietzen & Aja, 2012; Gietzen & Rogers, 2006). Epidemiological data provide support for the Protein Leverage Hypothesis as a model for obesity development (Hall, 2018, 2019; Martinez-Cordero et al., 2012; Martínez Steele et al., 2018). However, experimental studies have failed consistently to demonstrate the effect of protein restriction on appetite and eating behaviour in human beings. There is a need to explore whether dietary and lifestyle factors, such as dietary protein requirements and level of physical activity, play a role in directing responses to protein restriction.

Thirdly, the increased prevalence of obesity is attributed to the increased availability of larger-sized food portions over the past 25 years (Young & Nestle, 2002, 2012). Serving larger-sized food portions encourages a higher energy intake and weight gain (Rolls, 2014), yet evidence that individuals with a higher body mass index (BMI) consistently choose larger portions of food is inconsistent (Brunstrom et al., 2008; Brunstrom & Shakeshaft, 2009; Fay et al., 2011; Reily et al., 2016; Wilkinson et al., 2012) . There is a need to explore the relationship between excess adiposity and portion size, because in addition to understanding its contribution to the development of obesity, it also represents a key area for industry regulation and the rationale for the reformulation and downsizing of food products (Dobbs et al., 2014; Hetherington et al., 2018).

1.3 Individual variation in response to an obesogenic environment

Despite the potency of an obesogenic environment, these factors do not influence all human beings in the same way. Across the world, a sizeable proportion of individuals remain lean, as it is estimated that 39% of adults are overweight, of which 13% are obese (World Health Organisation, 2019). In England, there is a smaller proportion of the population who remain lean (approximately 30% of the UK population, The Health and Social Care Information Centre (NHS Digital), 2018b), meaning that these individuals are resistant to the environmental factors influencing eating behaviour and energy expenditure. In contrast, a higher proportion of the population (nearly 70% of the UK population) is overweight or obese, indicating that these individuals are more susceptible to these environmental factors and so gain weight (Blundell et al., 2005). Consequently, there has been an increased effort made to understand how variation in genetic, biological, physiological and psychological factors can render an individual prone to the obesogenic nature of the food environment.

The interaction between the individual and the environment is modelled in The Obesity System Map (Foresight, 2007) where variables relating to the external environment act through the individual physiological, biological and psychological traits (or state) to influence energy balance. One example is within the ‘Individual Psychology’ cluster: the map models how the variable ‘stress’ is influenced by environmental variables like ‘perceived lack of time’, which consequently influences ‘demand for indulgence or compensation’ and impacts the ‘Forces of dietary habits’ (this interaction is highlighted in pathway A in Figure 1). In other words, an individual who perceives they have little time may experience more stress and permit themselves to eat palatable food, which may be influenced by habitual eating behaviour (Wardle & Gibson, 2016).

Since the publication of the map, however, there has been substantial progress in understanding of the role of the individual. Several psychometric tools have been developed or revised to assess obesity-related eating behaviours, eating styles or personality traits. These include new tools such as the Power of Food Scale which assesses how the food environment influences the individual (Lowe et al., 2009), or revised tools to measure eating styles (Three-factor eating questionnaire Rv18, Cappelleri et al., 2009) and trait impulsivity (Barratt Impulsiveness Scale, BIS-15, Spinella, 2007), or the assessment of behavioural components of food reward (Epstein et al., 2007; Finlayson et al., 2007). Neuroimaging technology, for instance functional magnetic resonance imaging (fMRI), has allowed for the assessment of the neurobiological functioning that underpins aberrant eating behaviour, specifically in reward-based eating (Stice & Burger, 2019). These studies have enhanced our understanding of how the individual variation in these traits or behaviours may lead to more susceptibility to palatable food cues.

Studies have also characterised population groups that are at greater risk of weight gain. Genome-wide association studies have more than 900 near independent single nucleotide polymorphisms associated with BMI (Van Der Klaauw & Farooqi, 2015; Yengo et al., 2018), and more specifically identified that polymorphisms of the Fat Mass and Obesity associated gene (*FTO*) are associated with a preference for high energy-dense foods and altered appetite responses with eating (Den Hoed et al., 2009; Dougkas et al., 2013; Rutters et al., 2010; Wardle et al., 2008). Similarly, the relationship between socioeconomic status and obesity has gained considerable attention, as the prevalence of obesity is increasing at a faster rate in developing countries amongst those with lower socioeconomic status (Monteiro et al., 2004; Popkin et al., 2012). The increased obesity rates observed for these countries is attributed to the rapid economic changes, increased urbanisation and, importantly, the increased availability of cheap, processed, energy-dense foods (Ford & Mokdad, 2008; Monteiro et al., 2004; Popkin et al., 2012). The increased availability and accessibility of highly processed foods may also impact prospective food choices and attitudes towards food; however comparatively little is known about how an individual's socioeconomic status influences responses to an obesogenic food environment.

1.4 Thesis outline

My thesis draws from a full body of research to investigate specific areas of interest. High-fat, sweet foods are associated with food reward, yet individuals who exhibit a heightened sensitivity to reward or trait impulsivity may be more susceptible to the rewarding properties of these foods (Davis, 2009; Mobbs et al., 2010; Stice & Burger, 2019). Individuals who practise certain eating styles, such as cognitive restraint, disinhibition or emotional eating, may find high-fat, sweet foods more appealing and difficult to resist (Davis, 2009; Gibson, 2012; Mobbs et al., 2010). Moreover, the individual variation in appetite-related hormones in response to consuming high-fat, sweet food may predict a higher food intake.

The thesis also investigates whether the individual variation in physiological and physical activity (lifestyle) factors predicts the response to consuming a protein-restricted diet and may explain the inconsistency observed in the experimental studies investigating protein leverage. Individuals who participate in regular physical activity arguably have higher dietary protein needs (Beals & Mitchell, 2015). Furthermore, physical activity not only increases energy and macronutrient requirements but also improves appetite control allowing the individual more accurately to match their food intake to their energy needs (Beaulieu et al., 2018). Therefore, these factors may influence how individuals respond to consuming a low-protein diet.

Lastly, eating behaviour is influenced by the motivation to obtain food, and the appetite responses with food ingestion that cause the meal to end (defined as satiation) and delay the onset of the next meal (defined as satiety) (Blundell, 2001; Dalton, Finlayson, et al., 2013; Tremblay & Bellisle, 2015). Aspects of eating behaviour, such as a heightened motivation to obtain food and weaker satiety response with eating, are observed in individuals with obesity (Drapeau et al., 2013; Epstein et al., 2012). Studies have largely investigated these behaviours at an experimental, laboratory-based level, yet further investigations are needed to determine whether individuals exhibit these tendencies or behaviours at a population-based level.

The next chapter (Chapter 2) will review the biopsychological and environmental factors associated with overeating and identify research questions that will be addressed within the thesis. This chapter will outline the specific aims of each experimental study. Chapters 3 to 7 will describe the rationale, methods, results and conclusions of each experimental study. The findings of the experimental studies are discussed in Chapter 8, where I summarise and conclude on some biopsychological factors influencing appetite, eating behaviour and the development of the obese condition. Chapter 8 also considers the contribution of the thesis to the wider body of knowledge, direction for future research and implications for understanding the aetiology of obesity.

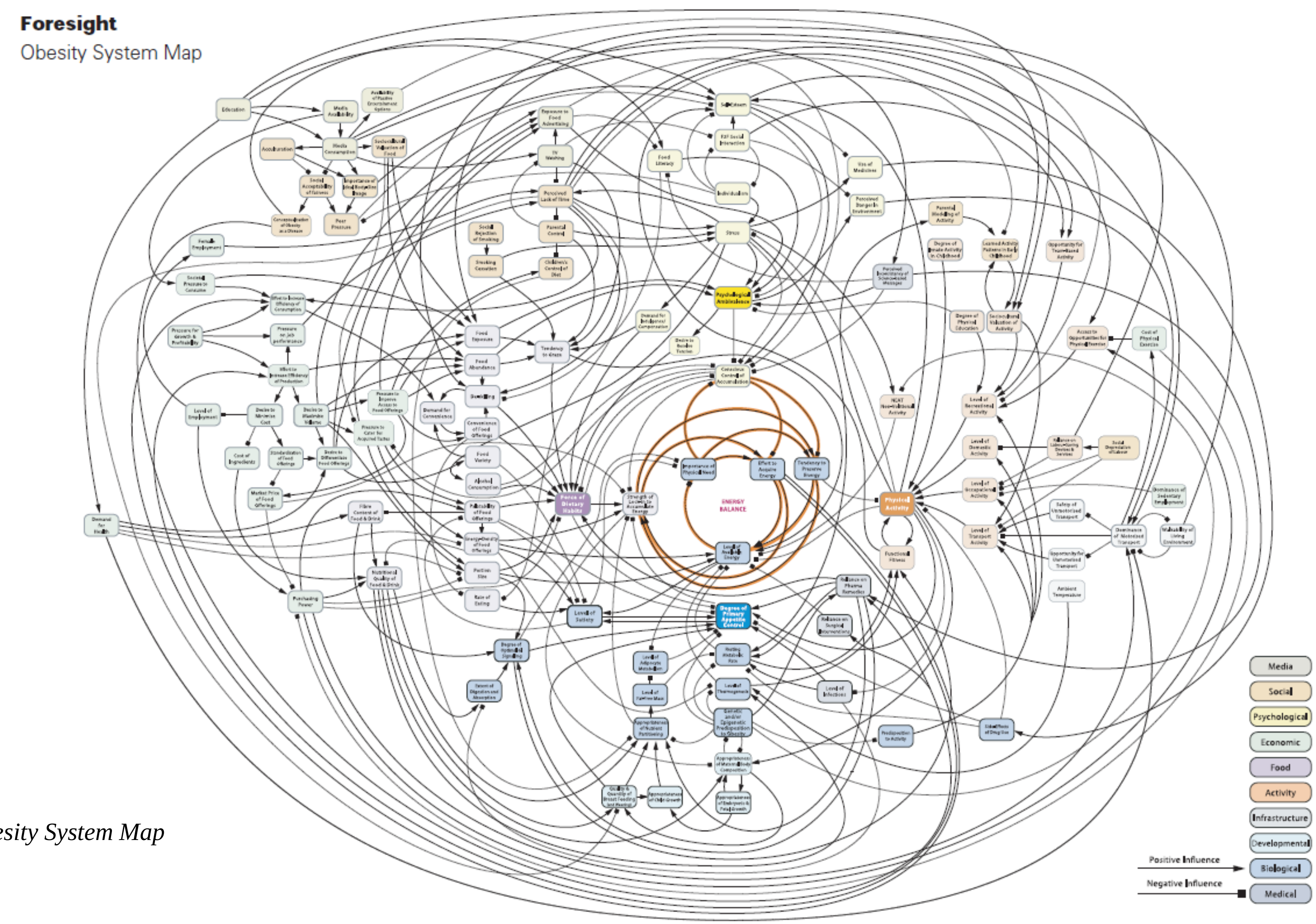


Figure 1: Obesity System Map

Chapter 2: Literature review: Characterising the biopsychological factors associated with overeating

2.1 Overview

The thesis explores the biopsychological factors that cause overeating under two common themes: 1) The obesogenic nature of the food environment; 2) The individual characteristics that increase susceptibility to a palatable food environment and cause overeating. An overview of the biopsychological and environmental factors explored in my thesis is presented in Figure 2.1. This chapter reviews the body of knowledge of the environmental, biological, physiological, psychological, socio-economic and genetic factors that are associated with overeating and identifies gaps for further investigation. The review is not exhaustive but provides a background to the research investigations undertaken in the proceeding experimental chapters. Additional information is presented in the introduction to each experimental chapter.

This chapter explores how environmental factors such as food palatability, low protein availability and larger-sized food portions influence appetite to provoke overeating. This chapter reviews how high-fat, sweet foods may alter the normal processes of appetite regulation that control taste perception, hunger, satiety and food reward, thereby provoking overeating. In this context, the chapter explores the Protein Leverage Hypothesis and evidence supporting a protein-regulated appetite that argues for one specific mechanism contributing to overeating (Steele et al., 2018; Simpson & Raubenheimer, 2005). The empirical evidence supporting protein leverage in humans is inconsistent, and there is a need to understand how individual variation in physical characteristics and lifestyle factors predicts the response to a low-protein diet. The chapter explores the association between portion size and adiposity (Rolls, 2018). A higher BMI is not consistently associated with choosing larger portion sizes. The thesis posits that another index of obesity, for instance waist-to-height ratio may be a better predictor of food portion size.

Throughout the chapter, I explore how the variation in biological, physiological, psychological, socioeconomic factors that may render an individual more susceptible to overeating, particularly when exposed to palatable food cues. Specifically, whether individuals who exhibit obesity-related personality traits, eating styles or eating behaviours find high-fat, sweet foods more pleasurable and rewarding to eat, and therefore at risk of overeating these foods. Additionally, whether the individual susceptibility to palatable food cues may be reflected in variations in appetite hormones, such as the hunger-related hormone ghrelin.

The chapter also explores how the individual variation in body composition, metabolism and activity levels may predict appetite and eating responses to a protein-restricted (Blundell, 2018).

Last, obesity is associated with eating behaviour traits, such as a heightened motivation to eat or reduced satiety response to food. In large sample populations the individual variation in genetic traits, age, sex and socio-economic status may be associated with these eating behaviours traits and predict the response to palatable food cues.

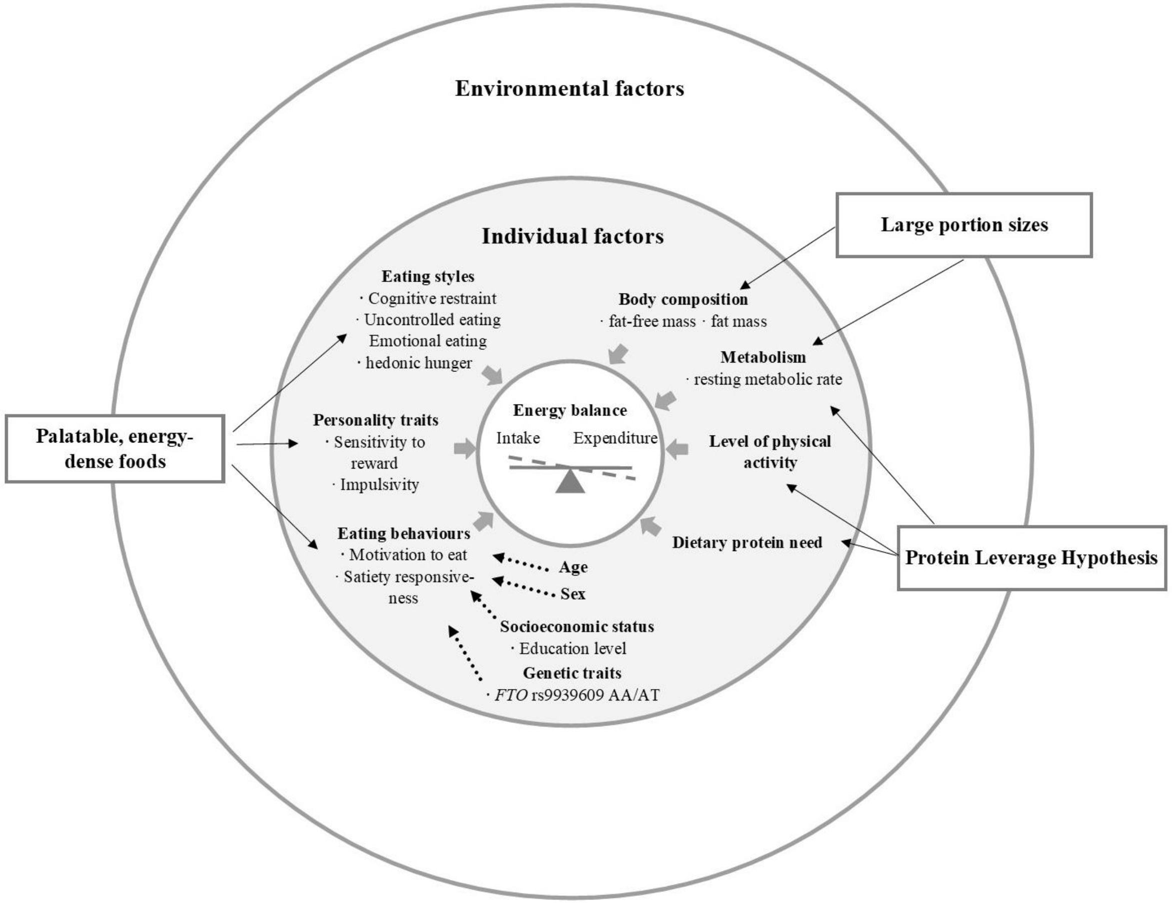


Figure 2.1: Overview of the factors associated with overeating explored in this thesis. These factors are discussed under two themes: 1) Obesogenic food environment: highly palatable, energy-dense, high-fat, sweet foods; foods (Palatable energy-dense foods) providing low protein nutritional value (Protein Leverage Hypothesis); foods served in larger-sized portions (Larger-portion sizes). These factors significantly influence appetite and food intake. 2) self-regulation: individual variation in physiological (body composition, metabolic rate, dietary protein need), psychological (personality traits, eating behaviours, eating styles), lifestyle (physical activity) and age, sex, socio-economic factors (education level), and genetic traits (FTO AA/AT allele) moderating responses to the obesogenic food environment (black arrows), rendering an individual prone to overeating and incurring a positive energy balance (grey arrows).

2.2 The physiological responses to consuming high fat, sweetened foods: do these foods heighten reward processes and undermine appetite control?

Many of the foods found in a Western diet are appetising, yet are heavily processed, containing high amounts of fat, refined starch, sugar and or salt, with minimal amounts of protein, dietary fibre and micronutrients (Monteiro et al., 2017). The macronutrient and flavour combinations in these foods, such as high levels of fat and sugar, enhance palatability and the appeal of these foods. However, high levels of fat and sugar are rarely found in naturally occurring foods. Since the taste of sweetness and of fat is generally acceptable to humans, foods that combine high levels of fat and sugar (sweetness) may exploit the liking for these tastes, and evoke a more rewarding experience when eating that then encourages food intake (Drewnowski & Almiron-roig, 2010; Small & DiFeliceantonio, 2019). Furthermore, the heightened responses may alter the physiological processes that regulate appetite, yet further work is needed to understand the specific mechanisms that influence short term eating behaviour.

2.2.1 Do high-fat, sweet foods exploit natural liking for sweet and fat flavours?

Taste plays an important role in the ingestive processes. The mechanisms of taste allow for the sensory evaluation of food to determine its nutrient content, and whether it provides a good source of energy and is safe to eat. The taste of sweetness and fat provide an indication of a source of energy, while bitter or sour tastes may indicate that the food is poisonous or contaminated (Besnard et al., 2015; Keast & Costanzo, 2015; Low et al., 2014).

In humans, sweet and fat are tastes that are commonly accepted and liked. The taste of sweetness evokes pleasurable sensation and acceptance is observed from birth and continues throughout life (Beauchamp & Mennella, 2009; Beauchamp, 2016; Drewnowski et al., 2012). The taste of sweetness is associated with, and may have evolved to indicate, an immediate source of energy (Beauchamp, 2016; Drewnowski et al., 2012). Sweetness is an important component of breastmilk, as it encourages infants to latch and feed, ensuring growth and development (Beauchamp & Mennella, 2009; Mennella & Bobowski, 2015). More recently the idea that human liking for sweetness is universal has been challenged (Iatridi et al., 2019; Yeomans et al., 2007), instead there appears to be several sweet liking phenotypes including individuals who dislike the taste of sweetness (Drewnowski et al., 1997; Yoon et al., 2014; Methven et al., 2016).

The taste of fat in food is also a commonly liked and accepted taste. Fat enhances the taste, odour and textural qualities in food and gives a favourable sensory profile, for instance the creamy, smooth, crispy or thick oral sensations that individuals attribute to high-fat foods (Drewnowski, 1998; Drewnowski & Almiron-roig, 2010). The taste and textural properties of fat are encoded as pleasant sensations (de Araujo & Rolls, 2004; Grabenhorst et al., 2010; Rolls, 2011).

The liking for fat may indicate a learned appetite preference for high-energy foods. It is proposed that consumption of high-energy (high fat) foods and post-ingestive consequences, confers a positive sensation to the individual. The associated stimuli (such as the sight, smell, and taste of the food) are used to reinforce learning of a novel food. The learnt appetite for fat in food is evident from a young age where early experimental studies demonstrated that children learn to prefer flavours associated with high-energy versions of the food (Gibson & Brunstrom, 2007; Gibson & Wardle, 2003; Ventura & Worobey, 2013).

The pleasant sensation and learnt appetite responses when consuming high-fat foods may direct an individual’s food intake and preferences. Experimental studies have demonstrated that Experimental studies have shown that humans are able to detect the presence of sweetness in food. Increasing the sugar concentration in solution increases the perceived sweetness intensity in a linear manner (Calviño et al., 1993; Choi & Chung, 2015; Graaf & Frijters, 1989; Peng et al., 2016). However, the pattern of hedonic responses with increasing sweetness varies between individuals. Some studies have identified two distinct phenotypes; sweet likers, whose hedonic ratings increase with increasing concentrations of sucrose; and sweet dislikers, whose hedonic ratings decrease with above a concentration threshold (Methven et al., 2016; Yeomans et al., 2007; Yeomans et al., 2009). Others have identified three or more distinct sweet liking groups (Garneau et al., 2018; Kim et al., 2014, 2017). However, there is no agreement on the best protocol to identify different sweet-liking phenotypes, making it difficult to accurately classify individuals accordingly (Iatridi et al., 2019).

The sensory perception of fat, however, appears to be more complex. Fat contributes to the texture, odour and taste sensory properties of food (Drewnowski & Almiron-roig, 2010), and there are a wide variety of fat sources (animal fats such as lard, butter, cream or vegetable sources such as sunflower oil) that influence the physiochemical properties and the sensory quality of the food (Bou et al., 2014). Humans are able to detect increasing concentrations of fatty acid in simple liquid solutions (Haryono et al., 2014; Mattes, 2009), however they find it more difficult to assess the fat content of more complex mixtures that are typically found in food, for instance fat found in solid food (Bolhuis et al., 2018; Drewnowski & Schwartz, 1990; Drewnowski et al., 1989; Urbano et al., 2016).

In a natural food environment, taste processes direct the individual to consume foods that have a positive effect on nutritional status (Rolls, 2016). Foods that occur naturally, for instance, foods that are not manufactured or processed, rarely contain high levels of both fat and sugar. The exception is mammalian breastmilk, which is both high in fat and sugars and serves to encourage feeding and weight gain (Ballard & Morrow, 2013). However, even breastmilk only contains roughly 3.5% fat and 7% sugar (Ballard & Morrow, 2013), while processed foods of a western-style diet contain much higher levels of fat and refined carbohydrates (DiFeliceantonio et al., 2018). An example is a chocolate bar, which contains 30% fat and 57% carbohydrate, of which 56% is sugars (Cadbury, 2020). Foods that contain unnaturally high levels of fat and sweetness may override the sensory evaluation of these flavours and encourage overeating.

Combinations of sweetness and fat in food have an effect on sensory perception. Experimental studies have shown that participants tasting foods containing either sweetness or fat generally rate these foods as pleasant and acceptable, however tasting foods that combine high levels of fat and sweetness elicits greater hedonic responses compared to tasting either macronutrient alone (Bolhuis et al., 2018; Drewnowski & Greenwood, 1983; Drewnowski et al., 1992, 1989; Drewnowski & Schwartz, 1990; Hayes & Duffy, 2008; Urbano et al., 2016). Moreover, the fat-sweet combination appears to enhance pleasantness in the early stages of eating, as ratings increase when eating the first few spoonful’s of a high-fat, sweet yoghurt (Gibson et al., 2008). Importantly, additional sweetness interferes with the ability to sensorially evaluate the fat content in a high-fat food and allows for an acceptance of a higher level of fat (Bolhuis et al., 2018; Drewnowski & Schwartz, 1990; Drewnowski et al., 1989). Additionally, increasing the fat content shifts the optimal level of preferred sweetness so that individuals prefer a higher level of sweetness in high-fat food compared to a low-fat food (Bolhuis et al., 2018). Consequently, individuals consume more of a high-fat sweet food compared to a bland, equicaloric version (Gibson et al., 2008; Valkauskaite & Gibson, 2010). It is noted that salt-fat flavour combinations also heightened hedonic responses and encourage overconsumption (Bolhuis et al., 2016; Bolhuis et al., 2016), however fat-sweet combinations allow for the acceptance of a much greater level of fat in comparison to fat-salty combinations, suggesting that sweet-fat stimuli offer a distinctive effect on sensory perception. In other words, high fat, sweet stimuli alter sensory perception to a greater degree than high fat, salty stimuli (Bolhuis et al., 2018).

The inability to accurately assess fat content in food is important, because it suggests that the heightened palatability offered by sweet-fat combinations overrides the ability to sensorially evaluate the nutrient content of the food. Experimental studies have reported that participants unable to subjectively evaluate the energy content of high energy-dense foods, particularly foods containing high levels of fat and carbohydrate (Brunstrom et al., 2018; DiFeliceantonio et al., 2018). Neuroimaging studies have revealed that increasing sweetness in a chocolate milkshake increased neural activity in the Rolandic operculum and thalamus - regions that process oral somatosensation and gustatory stimulation - confirming that sweetness greatly enhances the sensory experience when tasting foods containing fat (Stice, Burger, et al., 2013a). However, the inability to accurately assess the energy value of high-fat, sweet foods was associated with altered neural activity in the fusiform cortex and neural connectivity between the fusiform gyrus and ventral-medial prefrontal cortex, cingulate and cerebellum - regions responsible for evaluating the energetic properties and value of the food (DiFeliceantonio et al., 2018). Collectively, these studies suggest that high-fat, sweet foods exploit natural liking for sweet and fat flavours by altering the taste functionality and sensory perception when exposed to palatable food cues. Alterations in taste processing may contribute to appetite responses while eating, thereby encouraging a higher intake of food.

2.2.2 Do high fat, sweet foods elicit supra-normal reward responses?

Reward also plays a vital role in directing ingestive processes. The mechanisms of reward function to encode information gathered from sensory modalities with a value that guides food intake (Rolls, 2015). In a food-scarce environment, it is argued these biological mechanisms offer an advantage to humans as reward processes promote selection and intake of energy-dense foods and in doing so, defend body weight (Breslin, 2013; Kenny, 2011; Stice, Figlewicz, et al., 2013). However, in the current food-abundant environment, these mechanisms may be exploited to promote excess food intake. Overeating, therefore, may be driven by alterations in reward processing, if responses are enhanced, weakened or dissociated (Berthoud et al., 2011; Erlanson-Albertsson, 2005). Furthermore, alterations in reward processes may predict overeating tendencies and obesity risk (Dalton, Finlayson, et al., 2013).

Reward is characterised by three psychobiological components, namely, learning, ‘liking’ and ‘wanting’. The ‘liking’ and ‘wanting’ components reflect the pleasurable experience (‘liking’) and motivational drive to obtain food (‘wanting’) that define the qualitative aspects of eating behaviour (Berridge et al., 2009; Finlayson et al., 2007b, 2008). These components are underpinned by separate brain neuronal processes: ‘Liking’ is mediated by opioid neurotransmitters, and activation amplifies the hedonic impact of sweet taste to encourage eating (Berridge & Kringelbach, 2013; Kringelbach & Berridge, 2010; Peciña & Berridge, 2000; Peciña, 2008). 'Wanting' is encoded through the activation of the dopaminergic pathways found in the mesolimbic regions of the brain (Berridge, 2009; Zheng et al., 2009).

Neuroimaging studies have shown that tasting or viewing pictures of high-fat, sweet foods activates neural pathways associated with encoding a reward value to those stimuli. These include the activation of gustatory regions (primary taste cortex, anterior insula, frontal operculum) and the limbic system such as the caudate, amygdala, and orbitofrontal cortex (Burger & Stice, 2011, 2012; de Araujo et al., 2003; Killgore et al., 2003; Small et al., 2001; Stice, Burger, et al., 2013a). The ingestion of high-fat, sweet foods is also shown to stimulate opioid and dopaminergic networks. In rats, palatable food consumption activated opioid, GABAergic and dopaminergic pathways within the nucleus accumbens and also increased the motivational drive to obtain food (Hajnal et al., 2004; Wise, 2006; Zhang et al., 2003). Activation of these circuits not only induced food-seeking behaviour but also strengthened the environmental cues associated with food to reinforce feeding behaviour (Van Ree et al., 2000; Volkow, Wang, Baler, 2012). In humans, consuming a preferred meal induced a higher release of dopamine in the dorsal putamen and caudate nucleus in proportion to the subjective ratings of pleasantness (Small, Gregory, et al., 2003). More recently, Thanarajah et al. (2019) demonstrated that consuming a high-fat, sweet chocolate milkshake induced both an immediate and delayed release of dopamine in central neural regions, consistent with tasting the food and postingestive gut hormone signalling. Tasting the food elicited an immediate release of dopamine in orosensory neural pathways located in the nucleus of the solitary tract, thalamus and frontal insular cortex. This was followed with a second dopamine release about 15 - 20 minutes after food intake in regions within the amygdala, anterior and dorsomedial prefrontal cortex and caudate nucleus, demonstrating that high-fat, sweet foods elicit a dopaminergic reward response in the early and latter stages of eating.

Alterations of these reward processes may underlie overeating. The liking and wanting processes typically operate in unison yet can operate independently, i.e. ‘wanting’ to eat can occur even if the food is not ‘liked’ (Berridge & Kringelbach, 2013; Berridge et al., 2009; Finlayson et al., 2007). The Incentive Sensitisation Model proposes that with repeated consumption of high energy-dense foods, cues associated with the food are encoded with an enhanced motivational salience through a process of conditioning. Subsequent exposure to these cues then promotes food craving and intake. Consequently, overeating arises from a dissociation of reward processes, where cues associated with palatable food evoking an enhanced ‘wanting’ may occur independently of ‘liking’ in a similar manner to reward processing dysfunctions observed in drug and alcohol abuse (Berridge et al., 2010; Berridge & Robinson, 2016).

Certainly, behavioural and neuroimaging studies reveal that individuals demonstrate a greater preference and motivation to eat high-fat, sweet foods. There is a higher preference and desire to eat both high-fat, sweet and savoury foods before a meal (Finlayson et al., 2007a), in line with the observation that high-calorie foods are more appealing to people when they are hungry (Goldstone et al., 2009; Siep et al., 2009). However, with satiation there remains an elevated implicit desire to eat sweet foods (Finlayson et al., 2008), particularly high-fat, sweet foods (Griffioen-Roose et al., 2011) Furthermore, viewing images of high-fat, high carbohydrate (sweet) food elicited a greater neural response in the striatum compared to foods high in carbohydrate or fat. The fat and sweet stimuli appeared to act synergistically on neural regions to potentiate the reward response (DiFeliceantonio et al., 2018). Moreover, individuals demonstrated a greater motivational drive to obtain the high-fat, sweet foods independently of ratings for pleasantness (liking). The dissociation of reward processes further strengthens the hypothesis that foods containing unnaturally high levels of fat and sugar elicited unnatural reward responses in humans and encourages the motivational aspects of eating behaviour independently of the hedonic value (Berridge et al., 2010; Small & DiFeliceantonio, 2019).

Taken together, these studies suggest that combinations of fat and sugar act to powerfully influence processes regulating taste and reward, namely the sensory evaluation of food and alterations in reward processing, respectively. However, less is known about how these processes operate during the meal. It may be that the consumption of high fat, sweetened foods alters the sensory evaluation of food and sustains the pleasantness and desire to eat during the meal to encourage a higher intake. If combinations of fat and sweet flavours offer a supra-additive effect on reward processing, it may be that excess food intake is encouraged through an enhanced ‘wanting’ independently of ‘liking’, consequently leading to excessive food intake and a positive energy balance.

2.2.3 Do high-fat sweet foods undermine appetite control?

Ingestive behaviour is controlled by a complex, integrative network of neural and metabolic systems that act to maintain energy homeostasis and defend body weight (Berthoud et al., 2012; Berthoud, 2007; Berthoud et al., 2017; Rossi & Stuber, 2018).

During eating, there is a cascade of neural, metabolic and endocrine events that act to regulate food intake (Berthoud, 2006; Chaudhri et al., 2008; Cummings & Overduin, 2007; Kringelbach, 2007; Simpson et al., 2009; Wynne, 2005). These changes underlie the subjective appetite sensations of hunger and satiety, and contribute to meal initiation and termination (Blundell, 2001; Tremblay & Bellisle, 2015). For instance ghrelin, an orexigenic gut hormone, is released in response to fasting and acts to stimulate appetite (Cummings, 2006; Patterson, Gardiner, & Bloom, 2011). While other appetite hormones such as Glucagon-like peptide-1 (GLP-1), peptide YY (PYY_{3-36}), (De Silva & Bloom, 2012) pancreatic polypeptide (PP) and cholescystokinin (CCK) (Wren & Bloom, 2007) are satiety hormones released in response to feeding and act to increase feelings of fullness (Chaudhri et al., 2008).

The chronic consumption of a high fat and high sucrose diet is found to disrupt normal appetite functioning (Erlanson-Albertsson, 2005). In mice, high-fat feeding evokes changes in hunger-related hormones such as Agouti-peptide (Huang et al., 2003), increases in neuropeptide Y (Huang et al., 2004), increases in orexin (Wortley et al., 2003) and reduction in ghrelin (Moesgaard et al., 2004), while simultaneously altering the release of satiety related hormones such as Cholecystokinin (CCK) (Covasa et al., 2000) and Glucagon-like peptide 1 (GLP-1), and inducing leptin and insulin resistance in rats (Woods et al., 2003).

More recently experimental studies have demonstrated that consumption of a high-fat, refined sugar (HFS) diet changes hippocampal metabolism and function. This has been reflected as changes in brain glucose transport (GLUT1) and monocarboxylate transporter 1 (MCT1) expression (Sample et al., 2016), alterations in hippocampal and hypothalamic brain derived neurotrophic factor (BDNF) (Gan et al., 2015; Molteni et al., 2002) and evidence of neuroinflammation (Boitard et al., 2014; Sobesky et al., 2014). Furthermore, rats fed a HFS diet demonstrate a reduced ability to recognise internal appetite signals relating to nutritional status (Sample et al., 2016). Taken together these studies demonstrate that consumption of a fat-rich, refined sugar diet leads to acute changes in appetite function.

However, less is known about the changes that occur with acute consumption of high fat, sweet foods. For instance, it is not clear whether the taste of sweetness may increase hunger and the desire to eat, or whether the taste of fat influences energy intake (Sørensen et al., 2003). Palatable food induces an "appetiser effect", where the enhanced pleasantness increases or sustains hunger, delays the onset of satiation and encourages a higher intake of food (Gibson et al., 2008; Valkauskaite & Gibson, 2010; Yeomans, 2000; Yeomans, Blundell, et al., 2004). This suggests that these foods may stimulate the release of hunger-related hormones, such as ghrelin, or attenuate satiety-related hormones such as GLP-1, peptide YY (PYY), CCK or alter tonic appetite hormones insulin and leptin.

The high-fat content of the food may also influence the onset of satiation (feelings of fullness that leads to meal termination) and satiety (feelings of fullness that suppress hunger and food intake, (Blundell & Macdiarmid, 1997; Green et al., 1997; Rolls, 2000). High-fat foods offers the least satiation and allows individuals to eat more of these foods compared to high carbohydrate foods (Beaulieu et al., 2017; Green et al., 2000; Stubbs et al., 1995). High-fat foods are also anticipated to provide less satiety (Brunstrom, Shakeshaft, et al., 2008) and encourage the individual to initiate the next meal (Gibson et al., 2008). This indicates that, apart from the effect of palatability, fat weakens the ability to assess changes in appetite signals that occur with satiation. It may be that physiological responses to eating palatable combinations of fat and sugar override the signals controlling hunger and satiety. However, studies have not systematically characterised the effect of high-fat sweetened foods on changes in the profile of appetite hormones and the impact on meal termination.

2.3 Do personality traits, eating styles and eating behaviours predict susceptibility to energy-dense, palatable foods?

Palatable, high-fat, sweet foods may hold a general appeal to humans; however, some individuals may find these foods offer a more pleasant and rewarding experience when eating, and these heightened experiences encourage overconsumption. Individuals who exhibit personality traits such as sensitivity to reward or impulsivity may demonstrate a greater susceptibility and propensity to overeating palatable high-fat, sweet foods (Davis, 2009; Gerlach et al., 2015; Loxton, 2018). Individuals who exhibit eating styles, such as cognitive restraint, uncontrolled eating or emotional eating, may find high-fat, sweet foods more pleasurable and rewarding to eat, or use these foods as a method to abate feelings of negative affect (Gibson, 2012; Wang et al., 2016). Comparatively little is known about how these behavioural traits and eating styles alter appetite processes during eating to promote food intake.

2.3.1 Sensitivity to reward

The individual variation in reward sensitivity is a prominent psychobiological trait associated with the development of obesity. The basis of the trait draws upon J.A. Gray's Reinforcement Sensitivity Theory, a biological-based model of personality which proposes that individuals differ in their approach to reward and punishment related stimuli (Gray & Mcnaughton, 2000). The 'Behavioural Approach System' (BAS) describes the tendency to approach rewarding stimuli where the individual demonstrates a greater sensitivity towards conditioned cues that predict reward receipt, while the 'Behavioural Inhibition System' (BIS) describes the avoidance of punishing stimuli, where the individual demonstrates a sensitivity toward conditioned cues that predict punishment or cessation of reward (Pickering & Corr, 2008). A revised model proposes that a ‘Fight-Flight-Freeze System’ (FFFS) is associated with avoidance behaviours, while the behaviour inhibition system is proposed to balance the conflict between approach or avoidance behaviours (Gray & Mcnaughton, 2000; Van den Berg et al., 2010).

Reward sensitivity can be assessed using several psychometric tools, which assesses the degree to which an individual exhibits sensitivity to reward or punishing stimuli (Carver & White, 1994; Torrubia et al., 2001; Van den Berg et al., 2010). Accordingly, studies report that sensitivity to reward is associated with obesity. Using questionnaires, such as the BISBAS scale and the STR scale, self-reported STR was associated with a higher BMI (Davis & Fox, 2008; Davis et al., 2007, 2004; Franken & Muris, 2005; Mobbs et al., 2010; Van den Berg et al., 2010; Verbeken et al., 2012), and a higher preference and desire to eat high fat, sweet foods, even in the absence of hunger (Davis et al., 2007, 2004; Franken & Muris, 2005). Individuals sensitive to reward habitually eat more fat (Tapper et al., 2014) and exhibit heightened food cravings and attentional bias toward food (Li et al., 2015). In experimental studies, where sensitivity to reward is characterised by an enhanced motivational drive or willingness to invest effort to obtain food, studies have found that higher levels are associated with overeating (Epstein et al., 2012; Epstein et al., 2014, 2018; Epstein et al., 2007; Guerrieri et al., 2008; Rollins et al., 2010, 2014; Van den Berg et al., 2010) and obesity (Giesen et al., 2010; Hill et al., 2009; Kemps & Tiggemann, 2015; Saelens & Epstein, 1996; Temple et al., 2008; Temple et al., 2008).

Davis et al. (2013) suggests that individuals sensitive to reward may use food as a dysfunctional strategy to decrease the symptoms of negative affect. However, individuals who are sensitivity to punishing stimuli (BIS) may be equally susceptible to the rewarding properties of high fat sweet food, particular if they are sensitive to punishing stimuli and exhibit low levels of effort control, which refers to the ability to regulate emotional state and suppress habitual behaviours (Evans & Rothbart, 2007). In a similar manner to reward-sensitive individuals, punishment-sensitive individuals may seek to use food as a means to regulate or attenuate emotions (Rothbart et al., 2000). Recently, Mackey et al. (2019) reported in a sample of overweight and obese adults, BAS did not predict the liking or desire to eat high-fat sweet foods, yet in contrast BIS and effort control predicted a greater liking for high-fat, sweet foods, while lower levels of effort control predicted a greater desire to eat. Furthermore, BIS, effort control and liking collectively predicted tendency to overeat in these individuals. Collectively, this suggests that BIS sensitive individuals who exhibit lower effort control may be equally susceptible as BAS individuals to overeating, particularly foods high in fat and sugar.

2.3.2 Impulsivity

Impulsivity is broadly described as the tendency to act rapidly without thought or concern for future consequences of the action (Moeller et al., 2001). As a personality trait, impulsivity is seen as multidimensional construct because it encompasses a wide range of behavioural, cognitive, motor, and emotional behaviours (Mobini et al., 2007). Impulsive behaviours include the propensity to act without thought, lack of self-control, heightened sensitivity to rewarding stimuli, an inability to delay instant gratification and a lack of consideration of future consequences (Dawe et al., 2004; Dawe & Loxton, 2004; Logan et al., 1997; Whiteside et al., 2005). Consequently, impulsivity is seen to play a role in the development of obesity, as impulsive individuals may find it difficult to resist palatable foods and engage in spontaneous and uncontrolled food consumption, focusing on short-term gratification, while negating future consequences of their actions (Schag et al., 2013;Yeomans, 2017).

Impulsivity is measured using self-report questionnaires, such as the Barratt Impulsiveness Scale (Patton et al., 1995), and the UPPS Impulsive Behavior Scale (Whiteside & Lynam, 2001), or behavioural tasks such as the Stop Signal paradigm (Logan et al., 1997) or delayed discounting task (Baumann & Odum, 2012). These measures, however, are found to be poor correlated (Cyders & Coskunpinar, 2012; Meule, 2013; Reynolds et al., 2006) and it is proposed that the questionnaires and behavioural tasks assess different constructs of impulsivity (Reynolds et al. 2006; Cyders & Coskunpinar, 2012).

Studies have reported that measure of impulsivity are higher in individuals with obesity. In comparison with normal weight individuals, overweight individuals and individuals with obesity report higher levels of cognitive impulsiveness, motor impulsiveness and non-planning (Mobbs et al., 2010; Rydén et al., 2003; van Koningsbruggen et al., 2013). Individuals with obesity were found to demonstrate poor inhibitory control (Guerrieri et al., 2008; Guerrieri et al., 2007; Houben et al., 2014; Nederkoorn et al., 2006) and greater delayed discounting (Fields et al., 2013; Weller et al., 2008). Higher levels of impulsivity are also reported in individuals who present with binge eating disorder (BED) and food addiction (Davis et al., 2011; Meule & Kübler, 2014; Nasser et al., 2004; Schag et al., 2013), suggesting that impulsive behaviours may contribute to the development of disordered eating patterns.

Impulsivity has also been linked with measures of overeating, such as food craving (Meule & Kübler, 2014; Meule et al., 2014), reward sensitivity, external eating and attentional bias toward food cues (Hou et al., 2011), a heightened liking for energy dense foods (Nolan, 2012) and liking for sweet flavours (Weafer et al., 2014). Additionally, impulsive individuals were reported to consume more food in a bogus taste test (Guerrieri et al., 2008; Guerrieri, Nederkoorn, & Jansen, 2007; Guerrieri, Nederkoorn, Stankiewicz, et al., 2007).

In summary, since high, fat, sweet foods are typically associated with food reward, impulsive individuals may be at risk of overeating these foods as they may find these foods particularly appetising and difficult to resist.

2.3.3 Cognitive restraint, uncontrolled eating and emotional eating

Over the past few decades, research studies commonly report the association between eating styles, obesity and weight gain. These eating styles include dietary restraint, uncontrolled eating and emotional eating. Dietary restraint describes the attempt and practice of restricting food intake to achieve the desired body weight (Lowe, 2002). An individual employs cognitive strategies such as dieting, deliberately choosing smaller food portions or avoiding fattening foods, to restrict food intake (Polivy & Herman, 1985). However, these cognitive strategies can easily be undermined and lead to overeating (Johnson et al., 2012). Uncontrolled eating is also described as disinhibition, and characterises the episodic loss of control of eating (Bryant et al., 2008). The propensity to uncontrolled eating, however, may also represent a more general behavioural trait or phenotype (Vainik et al., 2019). Emotional eating describes the tendency to eat in response to negative emotions (Gibson, 2012). Emotional eaters may be susceptible to eating to regulate their emotional state and reduce feelings related to stress or negative affect (Macht, 2008). These eating styles may be associated with weight gain and a higher BMI.

Cross-sectional studies investigating the relationship between eating styles and BMI reported that both emotional eating (Anglé et al., 2009; Cappelleri et al., 2009; Konttinen et al., 2010; Lluch et al., 2000; van Strien et al., 2012) and uncontrolled eating (Bellisle & Dalix, 2001; Chaput et al., 2009; Dykes et al., 2004; Hays et al., 2002; Hays & Roberts, 2008; Lindroos et al., 1997; Provencher et al., 2004, 2003) are consistently associated with a higher BMI. Dietary restraint is not consistently associated with weight status as studies report that restraint is either positively (Anglé et al., 2009; De Lauzon-Guillain et al., 2006; Lluch et al., 2000; Price et al., 2015), negatively (van Strien et al., 2009; Williamson et al., 1995) or not associated with BMI (Chong et al., 2016). However, other studies reported that the relationship between restraint and BMI differed between normal weight and obese population groups; a positive association between restraint and BMI was found in normal weight individuals, while a negative association was found for the individuals with obesity (Bellisle et al., 2004; Cappelleri et al., 2009; De Lauzon-Guillain et al., 2006; Provencher et al., 2003). This suggests that the efficacy of restraint may differ between lean and overweight individuals or individuals with obesity. However, prospective studies report that high levels of restraint were associated with long-term weight gain (Chaput et al., 2009; Drapeau et al., 2003; Snoek et al., 2013; Stice et al., 1999), indicating that the restraint strategies used by individuals are not sustainable and individuals may succumb to overeating.

The susceptibility towards weight gain and a higher BMI amongst restrained, uncontrolled and emotional eaters indicates there are behaviours or attitudes towards food that increase the propensity to overeat. Restrained eaters, for instance, employ a variety of cognitive strategies to control food intake, yet experimental studies have demonstrated that these strategies can be easily undermined if the individual is exposed to stress (Lattimore & Caswell, 2004; Wallis & Hetherington, 2004; Weinstein et al., 1997) demanding cognitive activities (Ward & Mann, 2000), negative affect (Cardi et al., 2015) or distraction (Bellisle & Dalix, 2001). More recently, it has been shown that restrained eaters are more responsive to food cues - those associated with energy-dense, palatable food. Burger & Stice (2011) reported that dietary restraint was associated with greater activation of neural reward regions when tasting but not in anticipation of receiving a chocolate milkshake. Wang et al. (2016) reported that restrained eaters demonstrated a greater neural responsiveness to regions associated with reward, attention and visual processing, and responded more quickly to images of high energy-dense foods in comparison to unrestrained eaters. Interestingly, the study also reported a reduced response in regions associated with cognitive control (anterior cingulate), indicating that restrained eaters may find palatable food difficult to resist. Restrained eaters may find food more rewarding, yet more difficult to resist, and this may partly be acquired through repeated experience of eating too little to meet current energy needs.

Uncontrolled eaters show a tendency toward overeating as they are found to be more responsive to food cues, show a lower satiety response to food and report eating in the absence of hunger (French et al., 2012). Uncontrolled eaters also demonstrate greater responsiveness to palatable food cues and a higher motivational drive to eat these foods (Carr et al., 2014; Epstein et al., 2007). Uncontrolled eaters also exhibit reduced inhibitory control (less able to inhibit behavioural responses to stimuli in order to attain a higher-level goal) and consume more food when exposed to food cues (Houben et al., 2012), particularly if exercising a high degree of dietary restraint and strategies are challenged (Zhou et al., 2017). Moreover, disinhibition has been linked to impulsive behaviours, such as the tendency to respond hastily without prior reflection or deliberation (Kagan, 1966; Leitch et al., 2013; Yeomans et al., 2008), suggesting that uncontrolled eaters may be more responsiveness to food cues and act on impulse to eat, without consideration of future consequences.

Emotional eaters may turn to food to alleviate negative affect or feelings of stress. Food may be used as a coping mechanism (Dressler & Smith, 2013) or to buffer the effect of stressful events (Finch & Tomiyama, 2015). Emotional eating is also commonly observed in individuals who report depressive symptoms (Finch & Tomiyama, 2015; Konttinen et al., 2010; van Strien et al., 2016). Emotional eaters may exhibit overeating tendencies (Elfhag et al., 2007; Gibson, 2006, 2012; Macht, 2008; van Strien et al., 2009) if they perceive they are experiencing stress (O'Connor et al., 2008; Tan & Chow, 2014) or if stress or negative affect is induced experimentally (Van Strien et al., 2013). However, more recently, emotional eaters have been found to respond to both positive and negative mood induction (Bongers et al., 2016).

Individuals who exhibit high levels of restraint, or uncontrolled or emotional eating show a heightened preference for energy-dense, sweet and salty foods. Studies have reported that uncontrolled eaters demonstrated a preference for both high fat savoury or salty and sweet foods (Keskitalo et al., 2008; Lähteenmäki & Tuorila, 1995; Lampuré et al., 2015). Habnab et al. (2009) reported that restrained eaters show a preference for high-fat, sweet food, however this is finding has not consistently reported in other studies (Lampuré et al., 2015). Emotional eaters are commonly shown to have a preference for high-fat sweet foods (Camilleri et al., 2014; Keller & Siegrist, 2015; Konttinen et al., 2010), as it is thought that consumption of these foods is used to improve mood and lessen the effect of stress (Gibson, 2006).

2.4 Protein leverage hypothesis

Over the past two decades, attention has been drawn to the role that dietary protein intake may play in regulating appetite and eating behaviour including amount eaten. The Protein Leverage Hypothesis (PLH, Simpson et al., 2003; Simpson & Raubenheimer, 2005) proposes that because dietary protein provides a source of indispensable amino acids that cannot be synthesised by the body (Millward, 1997), its intake is more tightly regulated than that of non-protein energy sources - carbohydrate and fat. When protein intake is insufficient to meet nutrient requirements, it is proposed that appetite is stimulated to increase food consumption and reach a target protein intake (Simpson & Raubenheimer, 2012). These mechanisms act at the expense of regulating the intake of fat and carbohydrate, and the individual (or organism) will consume more food until protein intake is sufficient. As a result, the excess energy intake leads to a positive energy balance, increased adiposity and, eventually, obesity (Figure 2.2). The authors propose a Geometric Framework for Nutrition (GFN) that conceptualises how an individual or animal will achieve nutrient balance (Simpson et al., 2017). Using this framework, the PLH predicts that a small decrease in energy intake from protein, would result in a substantial increase in energy intake from carbohydrate and fat to achieve nutrient balance. This results in a substantial increase in total energy intake (Simpson et al., 2017; Simpson & Raubenheimer, 2005).

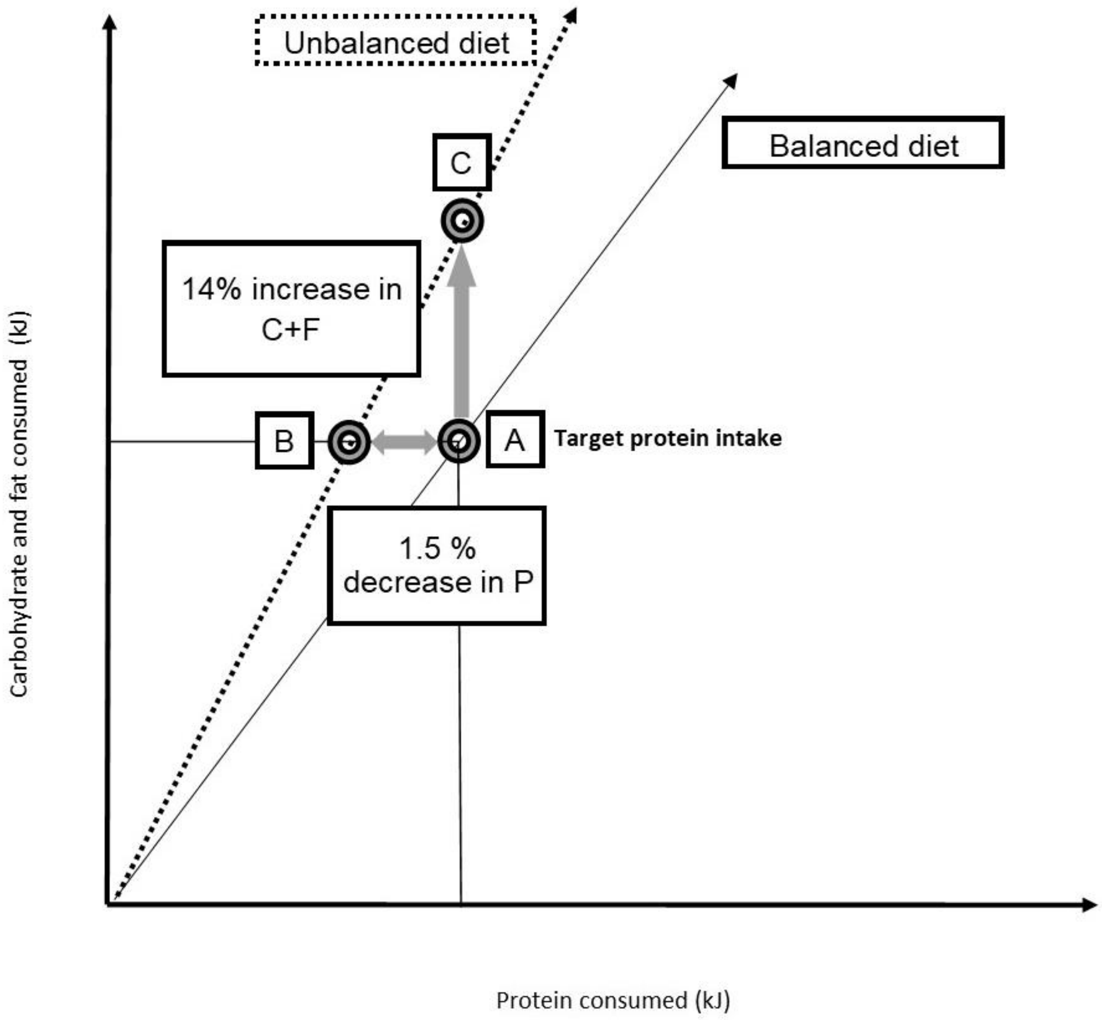


Figure 2.2: The Geometric Framework for Nutrition applied to the Protein Leverage Hypothesis. The target intake represents the optimal balance of protein to carbohydrate and fat intake. The radial lines represent the ratio of macronutrients in foods and describe how an individual will behave or 'move along the rail' to achieve nutrient balance. For a healthy, normal weight man, a balanced diet would contain 14% of energy from protein and 86% from carbohydrate and fat. If the individual consumes a diet balanced for a target protein intake (point A), they will achieve energy balance (solid line). If the individual consumes an unbalanced diet, they would have consumed enough energy from carbohydrate and fat, but not enough protein (point B). To achieve protein sufficiency (if the diet is restricted or unbalanced), the individual would need to consume an additional 14% energy from carbohydrate and fat to reach target protein intake (point C)(dotted line). Adapted from Simpson & Raubenheimer, 2005).

Evidence for the PLH is reported in a broad range of animal species (Simpson & Raubenheimer, 2012) including fruit flies (*Drosophila*, Lee et al., 2008), mice (Huang et al., 2013; Solon-Biet et al., 2015; Sørensen et al., 2008), rats

(Pezeshki et al., 2016; Simpson and Raubenheimer, 1997), cockroaches (*Batella germanica* (Raubenheimer & Jones, 2006), mink (Mayntz et al., 2009) and wild spider monkeys (Felton et al., 2008). In experimental studies, when protein is restricted by altering the protein to carbohydrate ratio of available foods, animals substantially increase their energy intake and over time show an increase in body weight and lipid storage (Huang et al., 2013; Pezeshki et al., 2016; Solon-Biet et al., 2014; Sørensen et al., 2008). With dietary variety, animals will seek to maintain a target protein intake, and in some species seek to achieve a target ratio of protein to carbohydrate, while allowing variation in energy intake (Felton et al., 2008; Lee et al., 2008; Mayntz et al., 2009; Raubenheimer & Jones, 2006; Simpson & Raubenheimer, 1997). Further evidence supporting the concept of a protein regulated appetite is found in experiments where rats rapidly learn to detect sufficient sources of protein following a protein-deficient meal (Gietzen & Aja, 2012; Gietzen & Rogers, 2006). Moreover, rats can avoid diets deficient in essential amino acids and fine-tune their food intake to achieve an adequate intake of protein (Booth, 1974). When acutely short of protein, rats also quickly learn to prefer flavours associated with good sources of protein over those paired with low amounts of protein, i.e. they acquire a protein-conditioned flavour preference (Baker et al., 1987; Gibson & Booth, 1986).

The evidence supporting a protein regulated appetite in humans has been derived from population-level data and experimental studies. Epidemiological data indicate that over the past four decades there has been a substantial increase in energy intake and particularly energy obtained from carbohydrates, fat and alcohol that has mirrored the increased prevalence in obesity (Austin & Krueger, 2013; Austin et al., 2011). The relative proportion of protein, however, has decreased or remained the same (Hall, 2019; Martinez-Cordero et al., 2012). These changes have also been associated with an increased intake of ultra-processed food (Martínez Steele et al., 2018)

The experimental evidence for PLH in humans, however, has not consistently demonstrated how protein leverage influences food intake. If appetite is tightly regulated by protein intake, studies should report the same effect when individuals consume either low or high levels of protein. However, only one study has demonstrated an increased energy intake when protein was restricted to just 10% of energy intake (Gosby et al., 2011), while other experiments providing a more restricted protein diet (5% PE) showed no effect on energy intake (Griffioen-Roose et al., 2012, 2014; Martens et al., 2013; Martens et al., 2014).

Conversely, a higher protein intake at 30% PE significantly reduced energy intake in several studies (Griffioen-Roose et al., 2012; Martens et al., 2013, 2014), while a diet of 25% PE did not (Gosby et al., 2011). The variation in study outcome may be due to differences in methodology and study design. More recently, a meta-analysis of 38 experimental studies confirmed that dietary protein intake was significantly negatively associated with total energy intake - as dietary protein intake decreased, energy intake from non-protein sources increased (Gosby et al., 2014). The inverse relationship between protein density and energy intake also revealed that this relationship was evident for a range of protein intakes (between 10-25% energy ingested). At levels above 20-25%, protein intake no longer appeared to influence energy intake strongly. For levels below 10%, the increase in the food intake required to achieve a target protein intake would be unachievable (D. Raubenheimer et al., 2015). It may be that the low protein diet offered in the experiments by Martens, Griffioen Roosen and colleagues may have been too low to elicit a compensatory response in energy intake. Supporting this suggestion, severely protein restricted mice (5% PE) reduce their total intake of food, whereas mildly protein restricted mice (10% PE) exhibited significant hyperphagia (Pezeshki et al., 2016).

It is also important to note that these studies observed similar changes in appetite (hunger, desire to eat, preference for savoury flavoured food) at both high and low protein dietary intakes that may be indicative of a learnt appetite behaviour for protein containing foods and an adaptive response to variations in protein intake. These responses will be discussed in detail in Chapter 5. Briefly, protein is found in a wide range of foods of varying tastes, for instance protein found in fruit, vegetables and cereals such as wheat, rice, sorghum and millet, (Fukagawa & Yu, 2009). Foods that contain the highest amounts of protein are meat, poultry, fish, beans and dairy products such as cheese, foods which are typically associated with having a salty, savoury, or 'umami' taste (Van Dongen et al., 2012). It is proposed that through a process of ’flavour-nutrient’ learning, individuals learn to discern which flavours provide the best sources of protein (Gibson et al., 1995; Gibson & Brunstrom, 2007; Sclafani, 1997). Therefore a heightened preference for savoury flavoured food may be indicative of an appetitive drive to eat more protein (Masic & Yeomans, 2014b, 2014a, 2017), possibly acquired through experience of flavour-nutrient learning (Baker et al., 1987; Gibson et al., 1995; Pérez et al., 1996).

These observations may provide further evidence for a protein-regulated appetite, yet an understanding of the underlying physiological mechanisms remains to be investigated. With the recent discovery of a liver-derived metabolic hormone, fibroblast growth factor 21 (FGF21) as a signal molecule for protein restriction, further work is being undertaken to understand how protein restriction influences metabolism and appetite control (Hill et al., 2018; Morrison & Laeger, 2015).

2.4.1 Does individual variation in protein need, body composition and physical activity influence responses to protein restriction?

2.4.1.1 Individual variation in body composition and metabolic rate.

Across a sample of healthy study participants, individuals will differ in their dietary protein needs. Although these differences may be small, the Protein Leverage Hypothesis applied through the Geometric Nutrition Framework predicts that relatively small variations in protein intake that will result in substantial variations in energy intake from non-protein sources (Simpson et al., 2017). Recently, Blundell and colleagues proposed that appetite and food intake are strongly regulated by energy expenditure (Blundell, 2018; Hopkins et al., 2017; MacLean et al., 2017). The Formula for Appetite Control proposes that components of energy expenditure, namely body composition, metabolic rate and physical activity, regulate appetite and food intake (Blundell, 2018). This contradicts the largely held view that body fat and circulating leptin levels play a primary (inhibitory) role in directing appetite and food intake (Woods & Ramsay, 2011). However, if energy intake is regulated by components of energy expenditure, these components may determine differences in protein need and the response to protein restriction.

Experimental and cross-sectional studies have reported that under conditions of energy balance, energy intake is strongly associated with fat-free body mass (FFM) - individuals with higher levels of FFM were found to consume more energy (Blundell, Caudwell, Gibbons, Hopkins, Naslund, et al., 2012; Blundell et al., 2015; Cameron et al., 2016; Caudwell et al., 2013; Lissner et al., 1989; Weise et al., 2014). Furthermore, FFM was also associated with higher levels of hunger and larger meal sizes, indicating that FFM exerts a stimulatory effect on eating behaviour (Blundell et al., 2012; Caudwell et al., 2013). In these studies, RMR was also associated with EI, meal size and hunger levels (Blundell, Caudwell, Gibbons, Hopkins, Naslund, et al., 2012; Blundell, Caudwell, Gibbons, Hopkins, Näslund, et al., 2012; Caudwell et al., 2013). More recently, it has been demonstrated that the relationship between FFM and EI is mediated by RMR (Hopkins et al., 2016). Fat-free mass is comprised of metabolically active tissue and organs, such as the heart, lungs and brain and is the principal determinant of RMR (Hall et al., 2012; Illner et al., 2000; Serra & Ryan, 2016; Sparti et al., 1997). The finding reported by Hopkins et al. indicates that FFM exerts its effects on energy intake through RMR, where RMR reflects the energy needs of metabolically active lean tissue. Therefore, the signals that arise from FFM provide an excitatory drive to stimulate food intake (Hopkins et al., 2016; Hopkins et al., 2017).

In contrast, these studies reported no association between fat mass, body mass index and energy intake. In lean individuals, however, fat mass (kg) predicts a lower energy intake, and fat mass index (height normalised indices of fat mass: kg per m^2) is negatively associated with EI in a large sample population (n = 184, (Weise, 2014). Furthermore, the relationship between energy intake and RMR is moderated by level of body fat. Energy intake is more closely matched with RMR in leaner individuals, while a weaker relationship was observed with a higher body fat percentage (Casanova et al., 2019). These findings support the view that adipose tissue provides an inhibitory signal to influence appetite, but with increasing levels of adiposity, the signal weakens and is less able to exert an inhibitory control on food intake (MacLean et al., 2017).

2.4.1.2 Individual variation in protein need

If RMR and FFM regulate energy intake, perhaps components of EE also influence macronutrient selection and intake. The protein-stat model proposes that appetite and food intake is driven by the metabolic demands of lean tissue (Millward, 1995). Under conditions of growth, maintenance or degeneration, the model proposes that an aminostatic appetite mechanism operates to regulate lean tissue mass and is responsible for detecting changes in protein need (Millward, 1998). If these mechanisms act to match protein intake with protein needs, then growth or degeneration of lean tissue should result in a shift in the preference for and intake of dietary protein (Morrison et al., 2012). Indeed, experimental studies indicate that protein restriction is sensed and acted upon (Gosby et al., 2011; Griffioen-Roose et al., 2012, 2014; Masic & Yeomans, 2017;Murphy et al., 2018; White et al., 2000b). However, the mechanisms underlying these responses are not well understood (Morrison & Laeger, 2015; Morrison et al., 2012).

Recent studies have demonstrated an association between FFM and fat-free mass index (FFMI kg/m2) and higher intakes of dietary protein (in grams) (Cameron et al., 2016; Weise et al., 2014), which suggests that a higher FFM may demand a greater protein need. Changes in muscle growth or degeneration may also be associated with changes in dietary protein intake. Conditions of tissue growth are evident in young animals, and experiments have demonstrated that young individuals show a greater preference for protein than do older animals (Jean et al., 2002; White et al., 2000a). Also, when muscle growth in artificially stimulated using somatotrophin, animals demonstrate a preference for protein (Phositlimpagul et al., 2002; Roberts & Azain, 1997). In humans, it is not known whether growing children and adolescents show a preference for protein over other macronutrients, although one study found that there was an inherited preference for protein-rich foods over the preference for fruit, vegetables and sweet foods, suggesting that the preference for protein may be genetically determined (Breen et al., 2006). However, nutritional guidelines emphasise the importance of sufficient protein intake to ensure proper growth and development (Das et al., 2017).

In contrast, elderly individuals reportedly consume less protein, that may be related to the loss of muscle (sarcopenia) that occurs with ageing (Beaudart et al., 2019; Hung et al., 2019). While the preference for protein appears to preserve FFM in older adults (Buckley et al., 2019), the lower intake of protein that occurs with sarcopenia may reflect an adaptative appetite response to a reduced need for protein. Taken together, these studies indicate that conditions for muscle growth or deterioration may drive an increased need for protein.

Physical activity also provides a stimulus for lean tissue growth. After a single bout of resistance or endurance exercise, whole body protein turnover is stimulated to initiate the anabolic and catabolic processes required for muscle tissue growth (Francaux & Deldicque, 2019). These responses are augmented when the individual ingests additional protein (Cermak et al., 2012). Chronic adaptations to exercise lead to significant increases in muscle mass, size and strength (Andersen, 2010; Kraemer & Ratamess, 2005; McGlory et al., 2019), including improved functioning of the cardiovascular, musculoskeletal, metabolic, endocrine and immune systems (Ruegsegger & Booth, 2018; U.S. Department of Health and Human Services, 1996).

Exercise will impose a higher demand for protein to support the metabolic processes involved in tissue growth and maintenance, including other metabolic processes such as cell signalling, regulation of gene expression, anabolism and catabolism of structural and functional proteins, and the production of neurotransmitters (Fernstrom, 2005; Wu, 2009). Protein is also a metabolised source of fuel, as amino acids such as alanine, asparagine, aspartate, glutamate, isoleucine, leucine, lysine, and valine are oxidised as a source of fuel, particularly during endurance-type activities (Tarnopolsky, 2004). As such, it is recommended that active individuals participating in regular endurance and strength activities increase protein intake to 1.2 – 1.7 g ·kg bw^{-1} · day^{-1} (Rodriguez et al., 2009). However, studies have not directly examined how responses to exercise training may increase the drive to eat protein in order to meet the increased needs.

2.4.1.3 Physical activity and appetite control

Physically active individuals may be more acutely aware of variations in protein intake in comparison to sedentary individuals. Participating in a physical activity regime enhances appetite awareness and the control of food intake (Beaulieu et al. 2018). If physically active individuals need more protein, are more sensitive to appetite sensations and aware of their energetic needs, they may demonstrate a heightened appetite response to protein restriction and regulate food intake accordingly.

Beaulieu et al. (2018) recently proposed a revised model of appetite regulation based on findings reported by Mayer et al. (1956) and Edholm et al. (1970). The model revisits the idea that appetite control occurs along a spectrum of physical activity: individuals who engage in low levels of physical activity and have higher levels of body fat show weaker appetite control and a greater propensity to overeat. In contrast, individuals who participate in high levels of physical activity and are leaner, demonstrate a greater ability to regulate appetite and food intake according to energy needs. Supporting this framework, a systematic review of 10 cross-sectional studies found that energy intake was more closely matched to energy expenditure in those individuals who engaged in higher levels of physical activity (Beaulieu et al., 2016). Furthermore, the relationship between fat mass and meal size is moderated by level of physical activity, where the association is strongest for individuals who participate in moderate to vigorous exercise (Beaulieu et al., 2018). Shook et al. (2015) reported that individuals who engaged in the highest levels of physical activity presented with the lowest risk for weight gain over one year.

Engaging in an exercise regime results in substantial changes in appetite. Individuals experience an increase in hunger before meals, but also greater sensations of fullness with eating (Guelfi et al., 2013; King et al., 2009, 2012; Martins et al., 2010). These changes are attributed to alterations in tonic and episodic hormones that regulate ingestive behaviour, including increases in plasma acyl-ghrelin and GLP-1, and reductions in fasting and postprandial insulin, leptin, glucose insulinotropic peptide (Martins et al., 2010, 2007). Regular exercise also enhances insulin and leptin sensitivity (Dyck, 2005; Goodyear & Kahn, 1998; Steinberg et al., 2004) and increases the rate of gastric emptying (Horner et al., 2015). Taken together, these studies suggest that physically active individuals can better regulate food intake to match their energy needs and demonstrate a heightened appetite control. Accordingly, physically active individuals may respond more acutely to periods of protein restriction and adjust protein intake accordingly.

2.5 Supersize me! Is obesity associated with choosing larger portions of food?

Over the past several decades, there has been a noticeable increase in the size of food portions served to consumers at restaurants and fast food outlets, and packaged food offered by retailers. These trends have been observed in both the United States and United Kingdom (Dobson et al., 2017; Economic & Social

Research Council, 2014; Nielsen & Popkin, 2003; Young & Nestle, 2003, 2012). The primary concern is that the increased availability of large or extra-large food portions may be directly related to the increasing rates of obesity. Young & Nestle (2012) reported a substantial increase in the number of large-size food portions offered by retailers to consumers over the past four decades, and these trends matched the increases of available energy in the food supply chain and the increased prevalence of obesity. In the United Kingdom, extra-large or supersize portions that are typically found in the United States are less common, however portion sizes of frequently consumed foods and readymade meals, such as bread and savoury pies, have increased substantially over the past 25 years (Figure 2.3, British Heart Foundation, 2013).

Experimental studies have demonstrated that serving larger-sized food portions substantially increases food intake and leads to weight-gain over time (Rolls, 2014). Consequently the availability of large portion sizes is seen as one of the primary environmental drivers of overeating and the development of obesity (Duffey & Popkin, 2011; Ledikwe et al., 2005; Marteau et al., 2015). However, it is not clear whether individuals who are overweight and obese habitually select larger portions of food. This is because experimental studies have not consistently demonstrated an association between large food portions and excess adiposity. Yet if increasing the size of food portions influence short-term eating behaviour, there is a need to understand the impact of portions sizes on long-term eating behaviour, specifically whether individuals with overweight or obesity habitually select larger portion sizes.

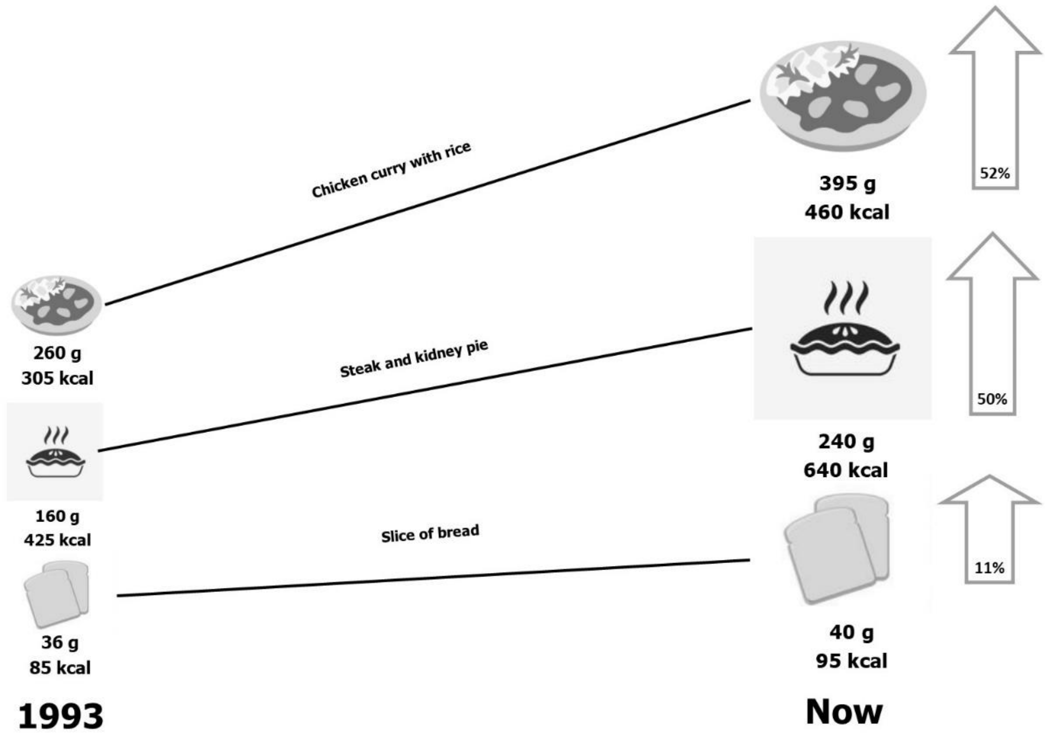


Figure 2.2: Increase in food portions served in the UK. Food portions have increased to provide 50% more energy (kcal) for ready-made meals such as steak and kidney pies and chicken curry with rice, and by 11% in a slice of bread. Adapted from British Heart Foundation (2013) *and Marteau et al.* (2015)

2.5.1 Do larger-sized food portions encourage overeating?

Serving larger portion sizes substantially increases food intake (Rolls 2014). The experimental studies where participants were provided with larger portions of food, they consumed significantly more food and, thus, increased energy intake. This is known as the portion size effect (English et al., 2015). It has been observed for a single meal or snack (Dilberti et al., 2004; Kral & Rolls, 2004; Rolls et al., 2002; Rolls, Roe, Kral, et al., 2004; Rolls, Roe, Meengs, et al., 2004; Wansink, 2000; Wansink & Kim, 2005; Zuraikat et al., 2018), over the short term (2 days, Kelly et al., 2009; Rolls et al., 2006; Rolls et al., 2007) and over longer periods (up to 6 months, French et al., 2014; Jeffery et al., 2007). A recent meta-analysis of 104 experimental studies demonstrated that larger portion sizes reliably increased food intake by an average of 35% (Zlatevska et al., 2014). A consistent observation from these studies is that individuals do not compensate for the increased energy intake with greater energy expenditure or by reducing subsequent food intake. For instance, Rolls et al., (2006) reported that doubling food portion sizes over two consecutive days resulted in a 26% increase in daily energy intake. The effect was observed in two other studies lasting 4 days (Kelly, Wallace, et al., 2009) and 11 days (Rolls et al., 2007). Interestingly appetite (hunger and fullness) did not change in response to consuming more food, indicating that the increased energy intake portion size did not influence appetite regulation. Furthermore, two longer term studies demonstrated that the effect of larger portion sizes was sustained for a period of two months (Jeffery et al., 2007) and six months (French et al., 2014) where participants significantly increased daily energy intake and gained an average of 0.2 kg per month over the six month testing period (French et al., 2014) . Collectively these studies suggest food portion sizes influence eating behaviour over a short and longer period and individuals do not compensate for the increased energy intake with regulatory behaviours (such as an increased energy expenditure or reducing energy intake) and consequently gain weight.

2.5.2 Is BMI associated with consuming larger portions of food?

While it is evident that serving larger-sized food portions increases energy intake and promotes weight gain, it is less clear whether weight status predicts choosing larger-sized food portions. In other words, do overweight individuals or individuals with obesity self-select larger portions of food? An understanding of this association will not only enhance our understanding of the factors influencing eating behaviour, but also represents a key area for obesity treatment and intervention (Hetherington et al., 2018). However, studies have not consistently demonstrated a relationship between a higher body mass index (BMI) and portion sizes.

Several experimental studies have used a method where participants are asked to choose their ideal portion size from an array of images of different portion sizes. Using this method, individuals with a higher BMI did not choose significantly larger portions of food (Brunstrom et al., 2008; Brunstrom & Shakeshaft, 2009; Fay et al., 2011; Reily et al., 2016; Wilkinson et al., 2012). Studies including larger sample population groups did find associations between BMI and ideal portion size (Labbe et al., 2017; Lewis et al., 2015; Spence et al., 2016). However, the relationship between BMI and portion size was relatively weak (Labbe et al., 2017) or only found in a one population group (Spence et al., 2016). Collectively, the evidence from these studies would suggest that weight status does not influence decisions about food portion sizes.

On the other hand, evidence from other research studies indicates that overweight individuals or individuals with obesity may choose larger food portions. Several large-scale cross-sectional studies report that overweight individuals or individuals with obesity habitually select larger portions of food (Albar et al., 2014; Berg et al., 2009; Gouvea et al., 2012; Liebman et al., 2003) and that individuals with obesity have a higher daily energy intake (Howarth et al., 2007; Lindroos et al., 1997).

An important observation from these studies is that BMI is commonly used as an index of adiposity. However, BMI does not differentiate between excess adiposity or musculature (Ashwell & Lejeune, 2011; Ashwell, 2005). In larger population-based studies, BMI is likely to reflect excess adiposity relative to excess musculature, which may explain why studies of a smaller sample sizes did not consistently report a relationship between weight status and larger portion sizes. Several studies have found that BMI is not associated with energy intake (Bellissimo et al., 2008; Blundell et al., 2012; Hopkins & Blundell, 2016; Piaggi et al., 2015; Stubbs et al., 2018; Weise et al., 2014). While it is important to consider that underreporting by individuals may significantly influence these findings (Karelis et al., 2010; Kelly, Rennie, et al., 2009), BMI could be considered a relatively weak or inconsistent predictor of energy intake and portion size.

Taking this into consideration, excess adiposity may still be associated with choosing larger portions of food. Body composition and metabolic rate, namely fat-free body mass and resting metabolic rate, are consistently associated with energy intake (Bellissimo et al., 2008; Blundell et al., 2012; Hopkins & Blundell, 2016; Piaggi et al., 2015; Stubbs et al., 2018; Weise et al., 2014) and predict meal size in a sample of overweight individuals and individuals with obesity (Caudwell et al., 2013). Comparatively, individuals with obesity have higher daily energy requirements compared to lean individuals (Blundell, Caudwell, Gibbons, Hopkins, Naslund, et al., 2012; Blundell, Caudwell, Gibbons, Hopkins, Näslund, et al., 2012; Caudwell et al., 2013; Ravussin et al., 1986). Individuals with obesity also demonstrate a reduced satiety with eating (Gautier et al., 2000; Gautier et al., 2001) and report needing a larger volume of food to reach satiation (Delgado-Aros et al., 2004; Meyer-Gerspach et al., 2014). Arguably, the increased energy needs and reduced satiety would drive a higher energy intake, either from consuming larger portions of food or eating more frequently (Mattes, 2014).

This suggests that indices of body composition, for instance waist circumference, waist-to-height ratio, fat-free body mass and resting metabolic rate, may be more strongly associated with food portion sizes. These indices, therefore, may describe the relationship between weight status and portion sizes more accurately than does BMI. Since few studies use these indices, there is a need to investigate the association between indices of body composition (as a measure of excess adiposity) and portions sizes.

2.6 Are variations in biological, anthropometric, genetic, and socio-economic factors associated with obesity-related eating behaviours and attitudes toward food?

Research studies have identified specific eating behaviours that are associated with the development of obesity. These behaviours include a greater motivational drive to eat, and a weaker satiety response with eating (Dalton, Finlayson, et al., 2013; Dalton et al., 2015; Epstein et al., 2012; French et al., 2014; French et al., 2012) and a fasting eating rate (Ohkuma et al., 2015). Typically, these behaviours have been measured in smaller laboratory-based studies and in overweight or obese population groups. However, these behaviours may predict susceptibility to palatable food cues and the tendency to overeat in other population groups (Carter and Jansen, 2012). Across a large sample population there are several biological, genetic and socio-economic factors that are also associated with obesity, such as age, sex, inheritable traits and a lower socioeconomic status, therefore, obesity-related eating behaviours may be more prominent in these groups and predict susceptibility to overeating palatable foods.

2.6.1 Obesity-related eating behaviours

2.6.1.1 Motivation to eat

Overweight individuals and individuals with obesity show a heightened preference for energy-dense foods, particularly those high in fat and sugar or salt (Blundell et al., 2005; Cox et al., 1999; Dressler & Smith, 2013; Lampuré et al., 2014; Mela, 2001; Wardle et al., 2001).Experimental studies have demonstrated that in comparison to lean individuals, individuals with obesity demonstrate a greater willingness to work for high energy-dense foods, and these foods have a greater reinforcing effect on eating behaviour, i.e. these individuals will choose to eat food rather than engage in a non-food related activity (Epstein et al., 2011; Epstein & Leddy, 2006; Finlayson et al., 2008; Giesen et al., 2010; Saelens & Epstein, 1996). Furthermore, individuals with obesity demonstrate a greater attentional and approach bias toward food than do lean individuals (Kemps & Tiggemann, 2015; Tetley et al., 2009; Werthmann et al., 2011), which indicates that these individuals may be more responsive to an abundant food environment and may initiate eating. More recent studies have identified that high food reinforcement may interact with other behavioural traits, such as disinhibition or the ability to delay gratification (delayed discounting), to predict a greater weight gain and higher BMI (Carr et al., 2014; Epstein et al., 2014).

2.6.1.2 Weak satiety responsiveness

Individuals with obesity show an impaired appetite control that is attributed, in part, to a weaker satiety response to food (Dalton, Finlayson, et al., 2013; Dalton et al., 2015). Several studies have reported that with food consumption, individuals with obesity have an altered postprandial release of gut hormones in comparison with lean individuals. Individuals with obesity demonstrate a reduced postprandial release of satiety-related appetite hormones Peptide YY (PYY, Batterham et al., 2006; Le Roux et al., 2006; Mittelman et al., 2010) and Glucagon-like peptide-1 (GLP-1, Adam & Westerterp-Plantenga, 2005; Devoto et al., 2018; Verdich et al., 2001). Also, suppression of the hunger-related hormone ghrelin was attenuated in individuals with obesity with food ingestion (English et al., 2002; Meyer-Gerspach et al., 2014; Mittelman et al., 2010; Tentolouris et al., 2004). Moreover, Gautier et al., (2000; 2001) demonstrated that satiation induced a differential brain neural response in obese men and women compared to lean individuals, indicating that these responses may reflect weaker satiety signalling with food ingestion. Alongside the physiological responses, obese people also report needing a greater volume of food to feel satisfied and have delayed gastric emptying (Delgado-Aros et al., 2004; Meyer-Gerspach et al., 2014). The subjective assessment of appetite also differs in individuals with obesity: a proportion of obese people report that they are unable to detect feelings hunger and fullness in response to daily eating patterns (Barkeling et al., 2007). These sensations may also be related to attenuated cortisol responses with eating and increased perceived levels of stress and anxiety (Drapeau et al., 2013a).

The weakened satiety response may lead to individuals eating for reasons unrelated to hunger (hedonic hunger).

These studies suggest that identifying obesity-associated eating behaviours may explain individual responses to the food environment and how these behaviours lead to overeating, weight gain and excess adiposity.

2.6.2 Socioeconomic status and obesity

A higher prevalence of obesity is observed amongst individuals of a lower socioeconomic status (Ball & Crawford, 2005; Booth et al., 2017; Davillas & Benzeval, 2016; Miech et al., 2006; Stafford et al., 2007; Stamatakis et al., 2005; Wardle et al., 2002). Typically, educational level is used as an indicator of SES (Booth et al., 2017; Wardle et al., 2002), however household income (Stamatakis et al., 2005), wealth indices or occupation level (Ball & Crawford, 2005; Davillas & Benzeval, 2016; Wardle et al., 2002) have also been used.

The regional or national economic status moderates the relationship between SES and obesity - as gross national product increases, there is an increased prevalence of obesity in individuals with a lower SES (Jaacks et al., 2019; Monteiro et al., 2004; Pampel et al., 2012). The further disparity is observed between genders, where the risk of obesity amongst lower SES individuals is greater in women than men (Monteiro et al., 2004; Wardle et al., 2002). In the UK, it is estimated that a greater proportion of women living in the most deprived areas are obese (38%), compared to women living in the least deprived areas (20%, The Health and Social Care Information Centre, NHS Digital, 2018b). However, Davillias et al. (2016) observed that increased adiposity was positively associated with socioeconomic inequalities in both men and women. Moreover, this study reported that SES status was not associated with obesity when using BMI as an indicator of adiposity. This finding adds to the suggestion that BMI may be a weak predictor of eating behaviour.

The prevalence of obesity is increasing at a faster rate amongst those with lower SES, particularly in developing countries (Monteiro et al., 2004; Popkin et al., 2012). These changes are attributed to the increasing availability of energy dense, processed foods that are cheap and affordable (Drewnowski & Darmon, 2005b; Ford & Mokdad, 2008). A lower SES is associated with consuming a less healthy diet (Méjean et al., 2011), lower intake of fruit and vegetables, but higher intake of processed foods (Fraser et al., 2000; Kearney et al., 1999; Stephens et al., 2018). Furthermore, lower SES is associated with an increased liking for high-fat foods (Lampuré et al., 2014). The relationship between obesity and SES may be mediated by specific behaviours, such as smoking, alcohol intake and income (Borodulin et al., 2012). These studies suggest that at a population level, socioeconomic status may predict eating behaviour and the appetitive responses to food cues, including a frequent intake and a greater motivation to eat processed, energy-dense foods.

2.6.3 Genetic risk factors associated with obesity

Inheritable characteristics may also influence eating behaviour. Genome-wide association studies have identified more than 900 near independent single nucleotide polymorphisms associated with BMI (Van Der Klaauw & Farooqi, 2015; Yengo et al., 2018). Locke et al. (2015) estimated that approximately 21% of the variation in BMI could be attributed to variations in common genes. These genes encode molecular pathways that influence appetite and eating behaviour, such as synaptic plasticity, glutamate receptor activity that is mediated by Brain-Derived Neurotrophic factor (BDNF) and Melanocortin 4 receptor (MC4R), and hypothalamic pathways involved in body weight regulation (Locke et al., 2015; Speakman, 2015).

The fat mass and obesity-associated gene (*FTO*) was the first gene found to be associated with obesity (Dina et al., 2007; Frayling et al., 2007; Scuteri et al., 2007). FTO protein and mRNA is widely expressed in tissues throughout the body, but more highly expressed in the brain, particularly regions within the hypothalamus (Fredriksson et al., 2008; McTaggart et al., 2011). *FTO* encodes 2-oxoglutarate-dependent nucleic acid demethylase, which plays an important role in regulating energy homeostasis and adipose cell lipolysis (Claussnitzer et al., 2015; Peng et al., 2011; Wåhlén et al., 2008) and is also suggested to be involved in nutrient sensing (Yeo & O'Rahilly, 2012). Five common *FTO* polymorphisms are associated with obesity risk: rs9939609, rs1421085, rs8050136, rs17817449 and rs1121980, Peng et al., 2011). These polymorphisms are associated with an increased body mass index (Ningombam et al., 2018), waist circumference (Andreasen et al., 2008; Liu et al., 2010a) body weight, hip circumference and fat mass (Dina et al., 2007; Hinney et al., 2007; Hunt et al., 2008; Scuteri et al., 2007), raised levels of serum leptin (Andreasen et al., 2008) and weight gain, specifically in females (Roswall et al., 2014).

Furthermore, studies have reported that *FTO* SNP's are associated with an increased energy intake in both adults and children (McCaffery et al., 2012; Melhorn et al., 2018; Speakman et al., 2008; Wardle et al., 2009), higher dietary fat intake (Harbron et al., 2014; Park et al., 2013; Timpson et al., 2008), refined carbohydrate intake (Harbron et al., 2014) and higher protein intake (Qi et al., 2014; Tanaka et al., 2013). Furthermore, *FTO* SNP's are associated with alterations in appetite. The at-risk A allele carriers of the *FTO* SNP rs9939609 report experiencing greater hunger and reduced satiety after eating (Den Hoed et al., 2009; Dougkas et al., 2013; Rutters et al., 2010; Wardle et al., 2008). A-allele individuals report an increased loss of eating control and preference for high-fat foods (Tanofsky-Kraff et al., 2009). A-allele individuals may find food more rewarding as several studies have demonstrated greater activation of reward-related brain regions in response to viewing pictures of food (Cecil et al., 2008; Karra et al., 2013) or in a food reward task (Scheid et al., 2014). Moreover, at-risk children and adolescents are more responsive to food cues, and report emotional eating and a greater enjoyment of food (Obregón Rivas et al., 2018). Studies have also reported that physical activity moderates the relationship between BMI and *FTO* genotype (Ahmad et al., 2011; Andreasen et al., 2008; Celis-Morales et al., 2016; Liu et al., 2010a), where individuals who participate in high levels of physical activity significantly reduce the risk of becoming overweight or obese (Kilpeläinen et al., 2011). These studies suggest that at a population level, inheritable traits associated with obesity risk may enhance appetitive responses to food cues, and that at-risk individuals may exhibit a heightened motivation to eat and reduced satiety towards energy dense, palatable foods.

2.6.4 Age

In the UK population, overweight and obesity is more prevalent in older individuals; specifically higher levels are found for individuals aged 45 – 74 years for men and 45 – 84 years for women (The Health and Social Care Information Centre, NHS Digital, 2018b). Excess adiposity in older adults is attributed to changes in muscle mass, metabolic rate and decreasing levels of physical activity that occur with ageing (Elia et al., 2000; Hughes et al., 2002; Klausen et al., 1997; Villareal et al., 2005). However, weight gain may be caused by an increased consumption of high energy-dense foods reported in older individuals, including a more frequent intake of full-fat dairy, fat spreads, pastries and rich dressings (Ax et al., 2016; Bamia et al., 2007; Beck et al., 2018; Bertin et al., 2016; Fraser et al., 2000; Gazan et al., 2016; Knudsen et al., 2014; Markussen et al., 2016; Mikkilä et al., 2005; Mishra et al., 2002).

Younger individuals, however, may be equally susceptible to weight gain and obesity as are older individuals; several studies have found that the young are more likely to practice unhealthy food choices and eating patterns. Younger individuals are found to consume more processed or fast-foods (Beck et al., 2018; Gazan et al., 2016; Knudsen et al., 2014; Whichelow & Prevost, 1996) and consume less fresh fruit and vegetables than older individuals (De Silva et al., 2011; Fraser et al., 2000; Nicklett & Kadell, 2013), and are more likely to snack and consume food between meal times (Zizza et al., 2001).

2.6.5 Sex

There are differences in eating behaviour between men and women. A higher proportion of men compared to women are classed as overweight (40% of men compared to 30% of women), although the proportion of individuals with obesity is similar between men and women (26% for men and 27% for women, The Health and Social Care Information Centre, NHS Digital, 2018b). This suggests that men may be more likely to overeat than are women. Men have higher energy requirements; on average, total daily energy expenditure is approximately 20% higher for men than women (Redman et al., 2014). These differences are partially accounted for by sex differences in body mass and composition (Klausen et al., 1997). Consequently, men have a higher energy intake and consume more food than do women (Cornier et al., 2010), and also select larger portions of food to serve themselves (Brunstrom, Rogers, et al., 2008; Lewis et al., 2015). This variation in dietary intake may be related to differences in how body weight and energy homeostasis affect appetite regulation in men and women (Shi & Clegg, 2009; Shi et al., 2009).

Neuroimaging studies have also demonstrated variation in neural responses to food cues between men and women; women demonstrated a heightened response to visual cues in both the fasted and fed state (Cornier et al., 2010; Uher et al., 2006). Fasted women also rated higher pleasantness for food cues compared to fed women (Del Parigi et al., 2002) .

There are differences between men and women in dietary intake and food choices. Men show a greater preference for savoury flavours, particularly high-fat, salty food, while women show a preference for sweet foods (Arganini & Saba, 2012; Drewnowski, Kurth, et al., 1992; Lampuré et al., 2014). Men eat foods such as full-fat dairy, eggs, fat spreads, bread, meat and wine more frequently than do women (Beck et al., 2018; Bertin et al., 2016; Togo et al., 2004). Women consume a greater variety of foods compared to men (De Silva et al., 2011) and report eating fruit, vegetables, cereals, legumes and potatoes more frequently (Baker & Wardle, 2003; Dibsdall et al., 2003; European Food Information Council, 2012; Gille et al., 2016). Alongside dietary intake, women show a greater concern for health and body weight, and tend to diet more frequently than men (Knudsen et al., 2014; Mikkilä et al., 2005; Mishra et al., 2002; Pinto de Souza Fernandes et al., 2017; Wardle et al., 2004). This means some women may exhibit greater level of cognitive restraint in attempting to restrict food intake (Cornier et al., 2010).

Collectively, these studies indicate that the individual variation in age and sex may contribute to variations in eating behaviours and attitudes toward food that predict susceptibility to overeating palatable foods.

2.7 Conclusion and Summary of gaps in knowledge

The overconsumption of energy dense, palatable foods is one of the primary factors implicated in the development of obesity. It is established that combinations of high levels of fat and sugar are more palatable and more rewarding to taste than either nutrient alone. However, less is known about the sensory, hedonic, physiological and appetitive responses to these macronutrient flavour combinations that occur during consumption. Closer examination of alterations in the appetite responses is needed to understand the mechanisms that cause overeating, and to characterise the obesity-related behavioural, psychological and personality traits that may increase susceptibility to overeating these foods.

The protein leverage hypothesis proposes that inadequate protein intake is the primary driver of food hyperphagia and the development of obesity. However, individual variation in protein need may influence the response to a restricted protein diet. A closer examination is needed to understand how the individual variation in body composition, metabolic rate and physical activity influences the response to low protein intake. This will further an understanding of the mechanisms of a protein-regulated appetite.

It has been shown that eating behaviours such as sensitivity to reward, motivation to eat and weakened satiety responsiveness are associated with weight gain and obesity. More specifically, these traits may predict the responses to food cues and be associated with increased obesity risk. There is a need, however, to investigate how the individual variation in eating behaviour traits is associated with the responses to food cues in individuals grouped by age, gender, socioeconomic status, obesity-associated genetic polymorphisms (FTO).

In Western societies, the increased prevalence of obesity is attributed, in part, to the increased availability of larger portions of food. However, an increased body mass index is not reliably associated with choosing larger portions of food. Eating behaviour may be predicted by measures of body composition, rather than BMI. A closer examination of the relationship between excess adiposity, body composition and metabolic rate is needed to determine the impact of large portion sizes on the development of obesity.

2.8 Research questions identified

- By how much does the unique combination of fat and sweet flavours in foods provoke overconsumption during a meal? How well do psychological and personality traits predict the risk of palatability-dependent overeating? (Chapter 3)
- Are the changes induced by palatability-dependent eating caused by alterations in appetite hormones? (Chapter 4)
- Does body composition, metabolic rate and physical activity moderate the appetite, eating behaviour and nutritional intake responses to acute low-protein intake? (Chapter 5)
- Does gender, genetic risk for obesity, age and socio-economic status influence appetitive responses to viewing images of food in a large sample size? (Chapter 6).
- Does body composition and resting metabolic rate predict the relationship between excess adiposity and food portion size? (Chapter 7)

Chapter 3: Microstructural analysis of a sweetened, fat-rich meal in relation to the individual variation in eating behaviours

3.1 Introduction

The overconsumption of processed, energy-dense foods is a primary factor implicated in the development of obesity (Mendoza et al., 2007; Monteiro & Cannon, 2019; Scarborough et al., 2011; Swinburn et al., 2011). A high intake of these foods is associated with overeating (Crino et al., 2015; Stinson et al., 2018), weight gain (Crino et al., 2015; Fogelholm et al., 2012; Salbe et al., 2004) and being overweight or obese (Lampuré et al., 2016; Mesas et al., 2012). However, it is not understood why individuals are overeating these foods. A characteristic feature of these foods is that they are extremely pleasant to eat, yet commonly found to have high levels of fat, sugar and/or salt. While the combination of high levels of fat and tastes (sweetness or saltiness) significantly improves food palatability, high-fat, sweet combinations are rarely found in naturally occurring foods, and therefore may provoke overeating (Breslin, 2013; Mela, 2006; Schulte et al., 2015). The scientific understanding of the underlying mechanisms is limited, as is whether some individuals may be more susceptible to appetising combinations of fat and sugar.

Food palatability plays a vital role in guiding ingestive behaviour, as individuals make food choices based primarily on taste (Dressler & Smith, 2013; Glanz et al., 1998; Sørensen et al., 2003). Experimental studies have demonstrated that enhancing the palatability by adding seasoning or sauce to pasta or sweetness to an oatmeal dish significantly increased food intake (Yeomans, 1996; Yeomans et al., 1997, 2001, 2005). An in-depth analysis of the eating processes that occur during the meal has demonstrated that palatable food has an early appetising effect; during consumption hunger levels increase in the early stages of eating, resulting in a delay in the onset of satiation, a longer meal duration and a higher intake of food (De Graaf et al., 1999; Yeomans, 1996; Yeomans et al., 1997). This indicates that the palatability increases food intake by stimulating orosensory reward processes to positively stimulate appetite, sustaining hunger levels, delaying satiation and decreasing satiety. However, few studies have investigated how palatable combinations of fat and sweetness influence appetite and food intake.

3.1.1 High-fat, sweet foods elicit greater sensory and reward-related responses

Combinations of fat and sweetness may elicit a distinctive appetising effect on eating behaviour because humans like the taste of sweetness and the taste of fat. Studies have demonstrated that most humans like the taste of fat and of sweetness; however when fat and sweetness is combined in a food, it greatly improves palatability and individuals show a preference for these combinations over foods high in fat or sweetness alone (Bolhuis et al., 2018; Drewnowski & Almiron-roig, 2010; Drewnowski & Greenwood, 1983; Drewnowski & Schwartz, 1990; Drewnowski et al., 1989; Salbe et al., 2004). The addition of sweetness to a high-fat food, however, hinders the ability to assess the fat content of food, allows acceptance of a higher level of fat (Drewnowski et al., 1992, 1989; Drewnowski & Schwartz, 1990) and the acceptance of a high level of sweetness (Bolhuis et al., 2018). In two recent studies, the addition of sweetness to a high-fat, plain yoghurt enhanced the pleasantness with the first few spoonful's of food and reduced anticipated satiety (defined as the amount of food a participant anticipated they would eat following consumption of the test meal (Gibson et al., 2008). Sweetness also attenuated the early reduction in hunger and encouraged a higher intake of food (Valkauskaite &

Gibson, 2010). This suggests that palatable combinations of fat and sweetness enhance sensory and appetite responses both on first tasting and during the meal.

High fat, sweetened foods are not only palatable but are also foods associated with reward, perhaps because they represent 'superstimuli' versions of breastmilk, which sensory properties must be both innately liked and wanted by mammalian neonates (Gibson, 2011). The pleasant taste experienced when eating palatable foods activates regions in the brain that are associated with reward, motivation, learning and reinforcement (Nolan-Pouparta et al., 2013; Small, Jones-Gotman, et al., 2003; Stice, Burger, et al., 2013a, 2013b; Thanarajah et al., 2019). However, in comparison with a high-fat chocolate milkshake, a high-sugar milkshake elicited a greater activation of neural regions involved in reward and motivation, and oral somatosensory perception (Stice, Burger, et al., 2013a). Moreover, the taste of a high-fat, sweet milkshake also elicited the release of dopamine in orosensory pathways such as the nucleus of the solitary tract, lateral ventral anterior nucleus of the thalamus and the anterior insular cortex. (Thanarajah et al., 2019).

Alongside the heightened reward responses, fat-sweetness combinations have been found to have a synergistic effect on reward processes. A recent study demonstrated that images of high-fat, sweet foods elicited a greater neural response in reward-related neural regions such as the dorsal striatum and mediodorsal thalamus compared to high fat or high-sweet foods. Furthermore, participants demonstrated a greater motivation to obtain the high-fat, sweet foods, indicating that fat and sweetness sensory stimuli act to potentiate the reward signal (DiFeliceantonio et al., 2018).

The heightened motivation to eat high-fat, sweet foods is also supported by studies investigating the behavioural components of reward, i.e. the pleasantness or 'liking' and desire to eat or 'wanting' for food stimuli. Participants show a greater preference, desire to eat and motivation to work for high-fat sweet foods even when satiated or after consuming a meal of sweet taste (Epstein et al., 2011; Griffioen-Roose et al., 2010). Moreover, a heightened motivation to eat high-fat sweet foods is implicated in the development of obesity and eating disorders (Dalton, Finlayson, et al., 2013; Epstein et al., 2010; Finlayson et al., 2011; Finlayson & Dalton, 2012b). These data suggest that high-fat, sweet foods elicit a more rewarding taste and ingestive experience that may heighten the motivation to eat and encourage overeating.

3.1.2 Individual variation in response to high-fat, sweet stimuli

Individuals vary in their response to rewarding stimuli, and a heightened sensitivity to reward has been shown to predict a higher BMI, a preference for and increased intake of high fat, sweetened foods and the desire to eat even in the absence of hunger (Davis, Strachan, & Berkson, 2004; Davis et al., 2007; Davis & Fox, 2008; Franken & Muris, 2005; Tapper, Baker, Jiga-Boy, Haddock, & Maio, 2015). Furthermore a heightened sensitivity to reward may interact with other personality traits such as impulsivity that results in an individual responding to palatable food cues and overeating without thought or consideration of the consequences (Dawe, Gullo, & Loxton, 2004; Guerrieri et al., 2007; Meule & Platte, 2015).

Alongside personality traits, there are several eating behaviours and attitudes towards food that may render an individual susceptible to overeating. Dietary restraint is an attempt to restrict food intake in order to control body weight (Johnson, Pratt, & Wardle, 2012). Disinhibition describes the loss of control and the tendency to overeat (Bryant, King, & Blundell, 2008). High levels of disinhibition are associated with greater BMI in cross-sectional studies (Epstein, Katelyn, Carr, & Fletcher, 2012; French, Mitchell, Finlayson, & Blundell, 2014; Tapper et al., 2015) and intervention studies (Nurkkala et al., 2015).

Dietary restraint is not consistently associated with weight status, however studies have demonstrated that dietary restraint is associated with weight gain over time (Chaput et al., 2009; Drapeau et al., 2003; Snoek et al., 2013; Stice et al., 1999). Dietary restraint may leave individuals feeling deprived and more susceptible to overeating palatable high fat, sweetened foods. Experimental studies have demonstrated that in some individuals, cognitive restraint may interact with disinhibition to encourage food consumption, particularly under conditions of stress and negative affect (Haynes, Lee, & Yeomans, 2003). Moreover, palatability appears to have a strong effect in high disinhibited individuals. Yeomans et al. (2004) demonstrated that high disinhibited, low restraint individuals consumed more palatable food compared to a bland food, while high restraint, low disinhibited individuals were unresponsive palatable condition. Interestingly palatability had influenced appetite in high disinhibited individuals, regardless of whether they also reported high or low scores for dietary restraint. These studies demonstrate that there is an interaction between eating styles that may render an individual prone to overeating, particularly energy dense, palatable foods.

Similarly, emotional eating, describing the tendency to eat in response to negative emotion, which is more prevalent in those with higher BMI (Gibson, 2012), may also interact with dietary restraint to increase eating (Macht, 2008). High levels of both dietary restraint and emotional eating and the tendency to overeat have been reported in individuals with a higher BMI (Anglé et al., 2009; Elfhag, Tynelius, & Rasmussen, 2007; van Strien, Herman, & Verheijden, 2009). Individuals who exhibit these behaviours are likely to overeat foods that are palatable and energy-dense, and therefore it is essential to understand how these behaviours may moderate the response to delicious combinations of fat and sugar (Gerlach et al., 2015).

Experimental studies performed in our laboratory used a high-fat, greek yoghurt as a test food; however the amount of test food eaten was relatively small and more representative of snack food intake (Gibson et al., 2008; Valkauskaite & Gibson, 2010). Therefore, to investigate changes throughout a meal, this study used a rice dish served at lunchtime to be more representative of the calorie intake in a meal. This method also served to provide a potential paradigm for investigating intrameal hormonal changes during and after eating the meal. The purpose of this study was to investigate whether (i) Sweetness in a high-fat food will encourage greater intake compared to an isocaloric non-sweet high-fat food (ii) Sweetness will alter sensory evaluation of food, and (iii) sustain hunger and the desire to eat (iv) Behavioural traits will be associated with appetite for and consumption of a high fat, sweetened food.

3.2 Methods

3.2.1 Participants:

Twenty-five volunteers (13 men and 12 women) were recruited from staff and student communities at the University of Roehampton. The age of participants ranged from 18 to 54 years (mean 26 ± 9 years), and body mass index (BMI) averaged 22.7 ± 2.5. Prospective participants were excluded from the study if they had any medical conditions, were pregnant, were allergic to or intolerant of milk products, or were restricting their diet or trying to lose weight. Participants were naïve to the purpose of the experiment and were told that the objective was to assess individual taste preferences for a rice meal. The experimental protocol was approved by the University of Roehampton Ethics committee PSY14_132.

3.2.2 Design:

The study employed a within-subject design whereby participants consumed either a high-fat sweet or high-fat non-sweet condition on two separate days. Participants were tested on two days, separated by a 7-day period. Both versions of the rice meal were nutritionally equivalent and presented to participants in a counterbalanced order.

3.2.3 Materials:

3.2.4 Procedure

Before each test day, participants were instructed to refrain from participating in vigorous exercise and eating or drinking (except water) from 22h00. On the first test day, participants reported to the laboratory between 08h30 and 10h30. The participants' heights (m) and weights (kg) were recorded and adiposity was measured using the 4-compartment body composition analyser TANITA-BC 418 MA (Amsterdam, Netherlands). Body mass index was calculated as weight/height^2

(kg/m^2). Total daily energy requirements (TDEE) were calculated using a new predicted equation derived from the CALERIE study (Redman et al., 2014) as follows: TDEE (kcal/d) = 1279+(18.3*weight (kg))+(2.3*age (years))-(338*sex (1 = female, 0 = male)).

The breakfast meal was calculated to provide approximately 20% of each participant's daily energy requirements. Breakfast consisted of cereal (Cornflakes, Kellogg Co.), semi-skimmed fresh milk, toasted white bread (Hovis soft white, Hovis), butter and jam or honey and tea (English breakfast tea, Twinings) or coffee (Original instant, Nescafé, Nestlé Ltd.).

Following breakfast, participants were asked to complete the psychometric questionnaires and then were free to carry out regular duties until lunchtime, but not to eat or drink anything except water. The participants returned to the laboratory three hours following breakfast and were instructed on the test meal procedure. They were provided with either a sweet or non-sweet version of the rice meal and asked to consume *ad libitum* at their own pace.

3.2.5 Measures:

3.2.5.1 Sussex Ingestion Pattern Monitor (SIPM)

Data were collected using the Sussex Ingestion Pattern Monitor (SIPM v2), a computer-based system modified from the University Eating Monitor (Yeomans, Weinberg, & James, 2005). For a description of SIPM please see Yeomans & Gray (1997) and Yeomans (2000). Food was weighed discreetly throughout the meal on a concealed digital balance (Sartorious BP 4100) connected through a serial line to an Apple Macintosh G3 computer. Custom-programmed software (SIPM v2) read the balance weight on stability to 0.1 g accuracy during the test meal.

The test meal began with the participants rating their appetite using computerised line (visual analogue) scales as described in Yeomans & Gray (1997). Participants were asked "How <word> do you feel right now?", where the word was 'hungry', 'full' or 'sick', and ratings were made automatically on a horizontal scale from 0 (Not at all) to 100 (As much as I can imagine) following Booth (2009) Participants were then provided with a small sample of rice meal and asked to taste and rate the intensity and preference for the meal sweetness, creaminess, estimated percentage fat content. In addition, ideal sweetness and creaminess levels were made on a horizontal scale from 0 (Too bland) through 50 (My ideal sweetness/creaminess) to 100 (Too sweet/creamy). The presentation order of ratings was fixed.

Following the tasting, participants were presented with 350 g of rice meal and instructed to eat at their normal eating rate, but to avoid leaving the spoon in the bowl or moving the bowl during eating. After every 30 g was consumed, the computer alerted the participant to complete a set of ratings for hunger ('how hungry do you feel right now?'), pleasantness ('how pleasant is the rice meal?') and desire to eat the rice meal ('how much would you like a spoonful right now?'). This pattern continued until 200 g was consumed. At this point, the computer alerted the researcher to provide a refill and an extra 200 g of meal was provided. This prevented the participant from terminating the meal based on sight of an empty bowl. Once the participant terminated the meal, they repeated the appetite ratings presented at the start of the meal.

3.2.5.2 Measures of personality traits and eating behaviour

3.2.5.2.1 Three-factor eating questionnaire (TFEQ-R18V2)

The Three-Factor Eating Questionnaire (TFEQ-R18V2; (Cappelleri, et al., 2009) is an 18-item questionnaire revised from the original 51-item TFEQ (Stunkard & Messick, 1985). The questionnaire assesses the cognitive and behavioural aspects of eating practices in three dimensions: cognitive restraint (CR) is the deliberate restriction of food intake to control body weight, uncontrolled eating (UE) describes the tendency to overeat and emotional eating (EE) describes eating due to a negative mood state, such as anxiety or depression (Cappelleri et al., 2009a). Each item is scored on a 4-point Likert scale; 1 = definitely true, 2 = mostly true, 3 = mostly false, 4 = definitely false. Items 1 – 16 are reverse coded as 1 = 4, 2 = 3, 3 = 2, 4 =1. Dimension subscale scores are represented as the mean scores of all items within the dimension (cognitive restraint, uncontrolled eating and emotional eating). The scale has been shown to have a good factor structure and internal reliability (Cappelleri et al., 2009a). In this experiment, Cronbach's alpha values were 0.86, 0.87 and 0.93 for cognitive restraint, uncontrolled eating and emotional eating respectively.

3.2.5.2.2 Power of food scale (PFS)

The Power of Food Scale (PFS) was used to measure how an individual respond to living in a food-abundant environment, more specifically, how the presence of or thought for food influences their appetite. The questionnaire comprises of 15-items which assess responsiveness to the food environment on three levels, namely (1) when the food is available, but not present; (2) when the food is present, but not tasted and (3) when the food is first tasted but not consumed. Examples of questions include, respectively, 'I get more pleasure from eating than I do from almost anything else'; 'When I know a delicious food is available, I can't help myself from thinking about having some.' And 'When I eat delicious food I focus a lot on how good it tastes'. The scale uses a 5-point Likert scale where responses are: 1 = I don't agree at all, 2 = I agree a little, 3 = I agree somewhat, 4 = I agree, 5 = I strongly agree. For each question, a higher score indicated greater responsiveness to the food environment. The mean score for each level was calculated and the aggregated score (Total PFS) comprised of the mean score for all three levels (Cappelleri et al., 2009b). The PFS has shown to have high reliability; Cronbach's α is 0.91. The corrected item-total correlations ranged from 0.50 to 0.73. Test-retest reliability has been shown to be good ($r = 0.77$, $p < 0.001$) (Lowe et al., 2009). The Cronbach alpha value here for total PFS was 0.88.

3.2.5.2.3 Behavioural Inhibition System and Behavioural Activation System (BIS-BAS)

The Behaviour Inhibition System (BIS) and Behavioural Activation System (BAS) (BIS-BAS) scales were used to measure individual differences in two behavioural systems that underlie behaviour and affect (Carver & White, 1994). The questionnaire comprises of 24-items with a 4-point scale (1 = Very true for me, 2 = Somewhat true for me, 3 = Somewhat false for me, 4 = Very false for me) with two BIS items scored in reverse. The BIS scale assesses sensitivity to punishment, conflict, and anxiety over the consequences of a bad event. Examples of questions include 'I worry about making mistakes', and 'Criticism or scolding hurts me quite a bit'. The BAS scale is a measure of reward sensitivity and consists of three subscales: reward responsiveness, measuring positive responses to the anticipation of reward ('When I get something I want, I feel excited and energized'); fun-seeking, reflecting the desire to seek out new rewards ('I'm always willing to try something new if I think it will be fun') and drive, describing the strong pursuit of rewarding goals ('I go out of my way to get things I want') (Poythress et al., 2008). The total score (total BAS) consists of a summed score of the mean score of each subscale. The BIS-BAS scale has been shown to have reliability. In this experiment, Cronbach alpha values were 0.76 for Total BAS scores and 0.56 for BAS fun-seeking, 0.70 for BAS drive and 0.64 for BAS reward responsiveness

3.2.5.2.4 Barratt Impulsiveness Scale (BIS-15)

The Barratt Impulsiveness Scale measures impulsivity, a trait that has been associated with overeating and obesity (Mobbs, Crépin, Thiéry, Golay, & van der Linden, 2010; Rydén et al., 2004). The BIS-15 is a shortened version of the original Barratt Impulsiveness Scale (Patton, Stanford, & Barratt, 1995) and contains 15 items assessing impulsivity in three domains; attentional (BISa), motor (BISm) and non-planning (BISnp). Examples of questions include 'I act on impulse' (BISm); 'I save regularly' (BISnp inverted item); 'I am restless at lecturers or talks' (BISa). Each item is rated on a 4-point Likert scale (1 = rarely/never to 4 = almost always). The BIS-15 has been shown to have good internal consistency (Meule & Platte, 2015) and high retest-reliability (Meule et al., 2015). The Cronbach alpha values for Total BIS-15 scores were 0.71.

3.2.6 Test food

The test meal consisted of two nutritionally equivalent high-fat sweet and non-sweet 'rice pudding' meals. The rice meal consisted of 600 g whole milk, 60 g meal rice, 35 g double cream, and 15 g unsalted butter and 5 g vegetable oil. For the sweet version, 35 g white sugar was added, while 35 g glucose polymer (maltodextrin; Glucidex®19, Roquette) was added to create the non-sweet version. The rice meal provided 141.0 kcal, 3.2 g protein, 8.0 g fat and 15.3 g carbohydrate per 100 g cooked weight. The rice meal was prepared the day before testing by heating the milk, rice, sugar or maltodextrin, cream, butter and oil together until 100 ^{o}C on an electric stove. The mixture was left to simmer for 35 minutes, frequently stirring to ensure a consistent mix. The mixture was then cooled and stored at 4 °C. On the test day, the meal was heated to 65 °C and served to the participant.

3.2.7 Data analysis:

Tests for normality and equal variance were conducted on all variables. To determine whether there were differences between men and women, a one-way analysis of variance (ANOVA) was conducted between sexes in body composition, energy expenditure, eating behaviour, and personality trait scores. Differences between sweet and non-sweet sensory ratings for sweetness, creaminess, estimated percentage fat, ideal sweetness and creaminess, pleasantness and desire to eat were analysed using Student's paired t-tests. Adjusted ideal sweetness and creaminess ratings were calculated by subtracting the raw score from the midpoint of a 100-mm horizontal line (50 mm). One-sided t-tests were used to determine whether ideal sweetness and creaminess ratings differed from ideal (0).

To assess changes in subjective appetite during the meal, the difference in ratings for hunger, pleasantness and desire to eat were calculated as the ratio of the difference from the first and fourth rating measurements to the first rating. The score was calculated as:

$$\textbf{Change in appetite} = \frac{\textbf{4th rating} - \textbf{1st rating}}{\textbf{1st rating}}$$

The general linear model (GLM) repeated measures ANOVA was used to assess differences between sweet and non-sweet food intake and change in appetite ratings (hunger, pleasantness, desire to eat). It was predicted that a smaller change in appetite would occur with high-fat, sweet food consumption (directional hypothesis). Therefore, a one-tailed *t*-test was conducted on changes in appetite (hunger, pleasantness and desire to eat), between sweet and non-sweet condition. Pearson's correlation coefficient was used to determine associations between sensory evaluation, changes in appetite, test meal food intake, eating styles and personality traits.

Intake data were excluded from two participants due to technical difficulties in recording intake. Technical difficulties also prevented the analysis of the change in appetite ratings for seven participants and obtaining psychometric scores for three participants. In the sub-analyses of the relationship between the desire to eat and intake, in two participants, the change in desire to eat was extremely high value, suggesting misunderstanding of the scale, and therefore excluded from analyses. All data were analysed using SPSS version 25.0 (IBM, Chicago). An alpha criterion of $p < 0.05$ was used to indicate statistical significance.

3.3 Results

3.3.1 Participant characteristics:

Participant characteristics are presented in Table 3.1. Male participants were older, heavier, but had less body fat and had higher estimated energy requirements compared to females ($p<0.05$). There were no differences in personality trait or eating behaviour ratings between males and females.

Table 3.1:

Age (years), body mass index (BMI), percentage body fat and total daily energy expenditure, personality traits and traits of eating behaviour for participants (n = 25)

	All	Men	Women	Sig
Age	26.1 ± 9.0 (18-54)	30.4 ± 10.5	21.0 ± 2.6	*p*<0.05
BMI	22.7 ± 2.5 (18.2-28.4)	23.4 ± 2.0	22.0 ± 2.9	*p*=0.175
%Body fat	20.7 ±7.1 (8.2 – 38.6)	18.1 ± 7.3	23.7 ± 5.8	*p*<0.05
TDEE (kcal)	2603.6 (1901.4–3332.4)	2859.4 ± 327.9	2326.4 ± 389.5	*p*<0.05
Restraint	1.94 ± 0.81 (1 - 3.67)	2.03 ± 0.78	1.83 ± 0.88	ns
Uncontrolled eating	2.25 ± 0.64 (1 - 3)	2.11 ± 0.64	2.43 ± 0.63	ns
Emotional eating	1.92 ± 0.77 (1 - 3.5)	1.93 ± 0.78	1.9 ± 0.79	ns
Total BAS	37.8 (26 - 47)	39.4 ± 4.8	35.8 ± 6.4	ns
BIS	20.5 (12 - 28)	20.5 ± 1.2	20.4 ± 4.3	ns
PFS	38.4 (23 - 58)	35.9 ± 10.4	41.4 ± 11.0	ns
Impulsivity (BIS-15)	32.0 (24 - 46)	31.8 ± 4.7	32.1 ± 5.4	ns

BMI: Body Mass Index; %Body Fat: percentage body fat; TDEE: Total Daily Energy Expenditure; Total BAS: Total Behavioural Activation score; BIS: Behavioural Inhibition Score; PFS: Power of Food Scale. Data expressed as Mean ± 1SEM and p value (Sig);

3.3.2 Comparison of initial sensory ratings between sweet and non-sweet conditions:

With the first taste of the sweet meal, ratings were higher for sweetness, $t(23) = 6.83$, $p<0.001$ and estimated percentage fat, $t(23) = 2.75$, $p=0.011$, while creaminess ratings were similar for both conditions, $p = 0.85$ (Table 3.2). Pleasantness and desire to eat ratings were higher for the sweet than the non-sweet condition: pleasantness $t(23) = 4.58$, $p<0.001$ and desire to eat $t(23) = 2.96$, $p=0.007$. Adjusted ideal sweetness and creaminess ratings for sweet condition were close to ideal, $p = 0.93$ and $p = 0.84$, respectively. For the non-sweet condition, participants rated the sweetness levels as less than ideal (or too bland), $t(23) = 7.46$, $p < 0.001$. Ideal creaminess ratings were close to ideal, $p = 0.96$ (Table 3.2).

When first tasting the sweet condition, a higher level of perceived fat content was associated with higher ratings for pleasantness, $r\ (24) = 0.45$, $p = 0.03$. Participants who were hungrier rated the meal as less creamy $r\ (24) = -0.49$, $p = 0.02$ and more desirable to eat $r\ (24) = 0.50$, $p = 0.013$, and higher ratings for pleasantness were associated with desire to eat, $r\ (24) = 0.54$, $p = 0.01$ and respectively. Higher ratings for creaminess were associated with a lower intake of both sweet and non-sweet condition, sweet $r\ (23) = -0.50$, $p = 0.015$; non-sweet $r\ (23) = -0.50$, $p = 0.013$

When tasting the non-sweet condition, higher levels of creaminess and perceived fat content were associated with a reduced desire to eat, $r\ (24) = -0.45$, $p = 0.03$ and $r\ (24) = -0.61$, $p = 0.002$.

Table 3.2:

Initial sensory ratings for sweetness, creaminess, estimated fat content (expressed as percentage fat; % Fat), pleasantness, desire to eat and ideal sweetness and creaminess for sweet and non-sweet condition.

	Sweet	**Non-sweet**	**Sig**
Sweetness	27.2 ± 1.8	11 ± 1.4	$p < 0.001$
Creaminess	30.4 ± 3.1	31.1 ± 3	$p = 0.849$
% Fat	49.4 ± 4.2	40.4 ± 3.4	$p = 0.011$
Pleasantness	68.7 ± 3.5	52.9 ± 2.3	$p < 0.001$
Desire to eat	63.4 ± 3.2	51.4 ± 3	$p = 0.007$
Ideal Sweet (adjusted)	-0.3 ± 13.5	21.7 ± 14.2	S: $p = 0.93$; NS: $p < 0.001$
Ideal Creaminess (adjusted)	0.5 ± 12.1	0.1 ± 10.9	S: $p = 0.84$; NS: $p = 0.95$

Mean ± 1SEM, Significance p value (Sig); S=sweet, NS=non-sweet. Adjusted score for ideal ratings represents raw score subtracted from midpoint of 100-mm scale (50), i.e. distance from ideal.

3.3.3 Intake

Participants ate significantly more of the sweet 312.08 ± 37.08 g than non-sweet 221.51 ± 23.41 g condition (Figure 3.1), F (21) = 7.71, $p = 0.011$, partial $\eta^2 = 0.27$. Eating rate (grammes consumed per minute) and time spent eating (minutes) were similar in both conditions, (p=0.177 and p=0.183 respectively). There were no sex differences in intake for either sweet or non-sweet conditions; Men 303.32 ± 36.43 vs Women 230.27 ± 36.43, F (20) = 2.01, $p = 0.17$.

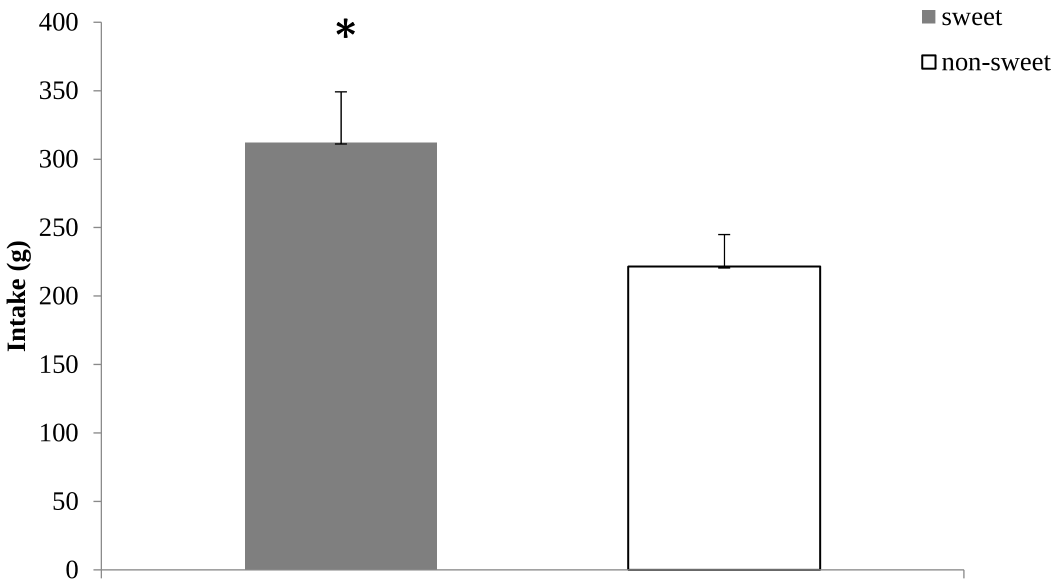


*Figure 3.1: Intake of sweet and non-sweet rice pudding condition. Mean ± SEM, *p<0.05*

3.3.4 Changes in appetite during the first part of the meal

The change in appetite ratings over the first four measurements while eating the sweet and non-sweet conditions are shown in Figure 3.2. Appetite was sustained in the early stages of eating the sweet condition. The change in hunger remained elevated in comparison to the non-sweet meal -0.21 ± 0.05 vs -0.36 ± 0.08, $t(18) = -1.63$, $p=0.06$ (1-tailed), CI [-0.043 – 0.34]. Similarly, the desire to eat was sustained in the sweet condition more than in the non-sweet condition, -0.26 ± 0.06 vs. -0.44 ± 0.09 respectively, $t(18) = 1.771$, $p=0.048$ (1-tailed), CI [-0.01 - 0.38]. By contrast, the reduction in pleasantness ratings did not differ between meal conditions ($p = 0.22$, 1-tailed).

Heightened appetite responses were associated with greater food intake in the sweet condition. Sustained feelings of hunger, pleasantness and desire to eat were associated with a higher intake of the sweet condition; hunger $r(18) = 0.50$, $p = 0.037$; pleasantness $r(18) = 0.56$, $p = 0.016$; desire to eat $r(16) = 0.49$, $p = 0.049$.

For the non-sweet condition, sustained pleasantness and desire to eat was associated with a higher intake, pleasantness *r* (19) = 0.58, *p* = 0.01; desire to eat *r* (19) = 0.72, *p* = 0.001.

On first tasting, higher ratings for pleasantness were associated with a sustained desire to eat in the sweet condition, *r* (18) = 0.63, *p* = 0.005. No other associations between initial sensory assessments and early appetite changes were observed for the sweet and non-sweet condition. There were no differences between males and females in changes in hunger, pleasantness and desire to eat ratings over the first four measurements.

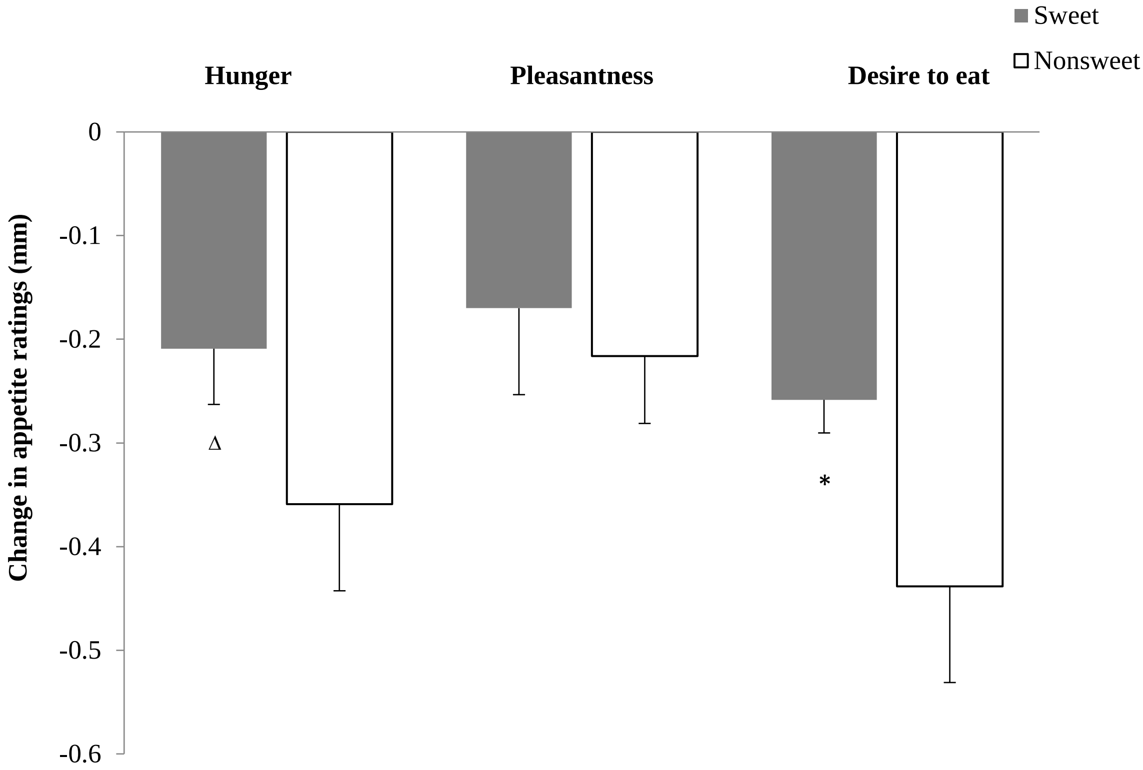


*Figure 3.2: Change in appetite ratings for hunger, pleasantness and desire to eat over the first four measurements of a sweet and non-sweet meal. Mean ±1SEM, Δ p<0.10, * p<0.05.*

3.3.5 Personality traits, eating behaviour and associations with initial sensory ratings, changes in appetite and intake:

Restrained eaters rated the sweet condition as closer to their ideal sweetness level, r (22) = -0.43, p = 0.045, while emotional eaters rated the non-sweet condition as closer to their ideal sweetness, r (22) = -0.46, p = 0.03, additionally emotional eaters found the sweet condition too sweet, r (22) = 0.043, p = 0.045. Restrained eaters demonstrated a sustained desire to eat, r (17) = 0.54, p = 0.03 and tended to be associated with sustained pleasantness in the first part of eating the sweet condition, r (17) = 0.44, p = 0.07, but not in the non-sweet condition (p = 0.80 for both), suggesting that the restrained eaters might be more susceptible to appetising effects of fat and sweetness.

In contrast to the study hypothesis, higher scores of BASRR and Total PFS were associated with a lower intake of the sweet condition. Higher scores for Total BAS were associated with a greater change in pleasantness scores for the sweet and non-sweet condition; sweet r (17) = -0.49, p = 0.046, non-sweet r (17) = -0.64, p = 0.006. However Total BAS and PFS scores were not associated with initial sensory ratings for either the sweet or non-sweet conditions.

Table 3.3:

Pearson's correlations coefficients of changes in hunger, pleasantness, desire to eat and intake for sweet (S) and non-sweet (NS) rice pudding over the first four measurements, and personality traits (total BAS, Impulsivity BIS15) and eating behaviours (cognitive restraint, uncontrolled eating, emotional eating, Total PFS, Impulsivity)

	Intake		**Change in hunger**		**Change in pleasantness**		**Change in desire to eat**	
	S	**NS**	**S**	**NS**	**S**	**NS**	**S**	**NS**
CR	0.26	0.30	0.34	0.04	**0.44†**	0.07	**0.54***	0.06
UE	-0.31	0.03	0.01	0.15	0.22	0.17	0.19	0.02
EE	-0.23	-0.07	-0.18	-0.45	0.26	-0.27	0.39	-0.23
BIS	0.12	0.14	0.44	-0.44	0.30	0.08	0.23	0.03
BASRR	**-0.46***	-0.11	-0.43	-0.32	-0.44	**-0.50***	-0.33	-0.24
BASD	-0.19	-0.14	0.06	0.07	-0.38	**-0.59***	-0.36	-0.23
BASFS	-0.32	-0.01	-0.17	0.06	-0.33	-0.40	-0.41	-0.13
Total BAS	-0.42	-0.11	-0.24	-0.08	**-0.49***	**-0.64****	-0.46	-0.25
Total PFS	**-0.54***	-0.19	-0.29	-0.03	-0.08	-0.17	0.00	-0.08
Impulsivity	-0.35	-0.42	-0.35	-0.32	-0.15	-0.48	-0.22	-0.41

*Change scores represent the difference ratings from baseline to fourth appetite rating. Significant values highlighted (bold typeface) †p < 0.10; *p < 0.05; **p < 0.001; CR: cognitive restraint; UE: uncontrolled eating; EE: emotional eating; BIS: Behavioural Inhibition System; BASRR: Reward Responsiveness; BASD: Drive; BA FS: Fun-seeking; Total BAS: Behavioural Activation System; Total PFS: Power of Food Scale.*

3.4 Discussion

This study investigated the sensory, appetitive and food intake responses when consuming a high-fat, sweet food compared to a nutritionally equivalent non-sweet version. The study also investigated whether obesity-related eating behaviours and personality traits would predict a greater appetite response when tasting and eating high-fat, sweet foods.

The results of the study showed that the addition of sweetness to a high-fat rice meal enhanced its palatability, by improving the sensory and appetitive assessments on first tasting and sustaining these responses during the early stages of the meal. Participants consumed more of the high-fat, sweet food in comparison to an isoenergetic non-sweet food. Furthermore, in the early stages of the meal, high cognitively restrained individuals demonstrated a sustained desire to eat and pleasantness when eating the sweet condition. These results suggest that the combination of sweet and fat tastes stimulate food intake by exerting a greater influence on appetite and eating behaviour than bland, high-fat foods alone. These effects may be particularly apparent in the early stages of eating for individuals who exhibit a high degree of dietary restraint.

This study hypothesised that combinations of high levels of fat and sweetness in food would not only enhance food palatability but would also provide a distinctive appetising effect by modifying appetite responses and stimulating a greater reward response during the meal compared to a high-fat, non-sweet version.

The results of this study showed that the addition of sweetness to a high-fat food improved its palatability and appeal. Participants rated the sweet condition as more pleasant and desirable to eat on first tasting than a nutritionally equivalent bland version. The level of sweetness and creaminess closely matched the participant's ideal taste preferences, while the non-sweet version was rated as too bland or contained a less than ideal level of sweetness, although the non-sweet version was rated as moderately pleasant. This suggests that sweetness enhances the palatability of high-fat food, but also elicits a higher reward response on first tasting.

It was also observed that appetite, notably hunger and the desire to eat, was sustained in the early stages of eating the sweet condition compared to the non-sweet condition, and this was directly associated with the enhanced pleasantness experienced with the first spoonful. The change in hunger and desire to eat was attenuated in the early stages of consuming the sweet condition, while these responses were not observed in the non-sweet condition. The higher pleasantness ratings on first tasting were associated with a sustained desire to eat in the early stages of the meal. Furthermore, the sustained hunger, pleasantness and desire to eat were associated with a higher intake of the sweet condition. These results suggest that the enhanced palatability offered by fat-sweet combinations influenced eating behaviour by heightening the sensory taste experience, eliciting a greater reward response and sustaining appetite in the early stages of the meal.

Food palatability is proposed to increase food intake by inducing an appetising effect on eating behaviour. The palatable cues associated with the food stimulate orosensory reward processes in a positive-feed forward manner to influence the processes regulating appetite, consequently encouraging a higher intake of food (Yeomans, 2000; Yeomans et al., 2004). Consistent with this proposal, the sustained hunger observed with the consumption of the sweet food, indicates that palatability modifies the physiological processes that regulate hunger and satiety, such as the release of gut hormones such as ghrelin and GLP-1. Gut hormone responses to palatable high-fat, sweet foods are further investigated in Chapter 4.

The appealing properties of fat-sweet have been reported in two studies using a high-fat, greek-style yoghurt: Gibson et al. (2008) demonstrated an increase in pleasantness from the first to the fifth spoonful of high-fat, sweet yoghurt, while Valkauskaite & Gibson (2010) observed attenuation in the early reduction of hunger observed in the first half of the meal of the high-fat, sweet condition. Participants also consumed more of the high-fat, sweet yoghurt. Since these responses were observed in the early stages of consumption when the absorption of nutrients from the gastrointestinal tract is unlikely to have played a contributory role, this further supports the role of orosensory reward stimulation in encouraging palatable food intake. However, it is important to note that post-hoc analyses of change in hunger rating demonstrated that they were underpowered (please see Appendix L). Although this work extends the previous work demonstrating a sustained hunger while eating a high-fat, sweetened yoghurt (Valkauskaite & Gibson, 2010), further studies will be required to confirm the findings of this study.

The addition of sweetness did not alter the sensory evaluation of fat content in this study. It was expected that sweetness might lower the perceived creaminess or fat content of the food. Instead, participants perceived the sweet condition to contain more fat, although creaminess ratings were similar for both conditions. As noted previously, the study may have been underpowered to test this association. In previous studies, the addition of sweetness to a high-fat food masked the participants' ability to accurately detect fat content (Drewnowski & Schwartz, 1990; Drewnowski et al., 1989) and allowed the acceptance of a higher level of sweetness (Bolhuis et al., 2018). Like the findings in this study, Valkausaite & Gibson (2010) reported no difference in the creaminess and percentage fat content ratings for sweet and non-sweet high-fat yoghurts, despite a greater intake of the sweet version. In this study, higher ratings for percentage fat content in the sweet condition were associated with higher pleasantness ratings, suggesting that the participants may have assumed that these hedonic sensory experiences indicate a higher fat content. Furthermore, the differential evaluation of fat content may be because the rice pudding meal is neither entirely liquid nor solid in texture, but a mixture of both. Since the sensory assessment of solid food is shown to be less accurate than for liquid (Drewnowski et al., 1989), it may be that the texture reduced the ability to assess fat content.

The addition of sweetness to the high-fat food heightened the affective evaluation and motivation to eat with the first taste. It was also found that in the sweet condition, the sustained desire to eat occurred independently of pleasantness. Pleasantness ratings declined for both conditions, while the desire to eat remained elevated in the early stages of eating the sweet condition. These findings suggest that high-fat, sweet food not only heighten reward responses with first tasting but also encouraged the separation of 'liking' and 'wanting' components of reward by increasing the appetitive drive to eat independently from the hedonic value of the food.

The dissociation of reward processes (i.e. a heightened 'wanting' or motivation to eat more than 'liking' or pleasantness) is consistent with the Incentive Sensitization Theory that proposes a heightened wanting may drive compulsive behaviours involving substance abuse (sex, pornography, gambling, food) (Berridge et al., 2010). Polk et al. (2017) observed higher craving scores for highly processed foods, particularly those containing high levels of fat and carbohydrate or sugar while liking ratings were not consistently elevated for these foods. A heightened wanting or craving for high-fat, sweet foods was also observed in normal weight and individuals with obesity who presented with higher binge-eating scores (Dalton, Blundell, et al., 2013; Dalton & Finlayson, 2014; Finlayson et al., 2011). High-fat, sweet foods were also perceived to be have addictive qualities and implicated in problematic eating behaviours, particularly those individuals who report higher scores of food addiction (Schulte et al., 2015). These appetite responses may be a supra-normal response to consuming levels of fat and sweetness that are not found in natural foods (Gibson et al., 2011); however, the heightened reward experience and dissociation of reward processes during eating need to be confirmed in future research investigations.

In this study, individuals with higher scores for cognitive restraint showed sustained pleasantness and desire to eat during the early stages of eating the sweet condition. The sustained appetite did not lead to a higher intake of food, perhaps because these participants were exerting restraint in the 'observed eating' experimental situation. However, the heightened appetite responses observed for restrained eaters suggests that the enhanced sensory properties for the sweet condition are more potent, and that restrained eaters experience greater reward response when eating high-fat, sweet foods. A greater neural reward response to palatable food in restrained eaters has been reported previously (Stice et al., 2010; Wang et al., 2016). However, cognitive restraint is not consistently associated with a heightened liking or craving for palatable foods (Gearhardt et al., 2014; Komatsu & Aoyama, 2014; Polivy et al., 2005; Polk et al., 2017). Furthermore, highly restrained women were found to be unresponsive to the effect of palatability in a pasta lunch meal, while high disinhibited women demonstrated a heightened appetite (hunger) and consumed more of the palatable food condition (Yeomans et al., 2004). However, the analyses did not control for multiple comparisons, therefore further research is need to confirm whether restrained individuals demonstrate a heightened reward response to food cues.

Since the cognitive strategies employed by restrained eaters are undermined by factors such as distraction, stress, negative affect and demanding cognitive activities (Bellisle & Dalix, 2001; Cardi et al., 2015; Lattimore & Caswell, 2004; Wallis & Hetherington, 2004; Ward & Mann, 2000; Weinstein et al., 1997), these factors may moderate the relationship between food palatability, food reward and cognitive restraint; thus it is important to understand how these factors may interact to drive food craving and overeating. Overall, the heightened appetite responses in restrained eaters suggest that these individuals are more susceptible to palatable combinations of fat and sweetness and provides insight into why people develop restrained eating. Thus, this approach could help understand possible processes that may interact with other traits or external influences to encourage food consumption.

This study found that there was a negative association between trait measures of reward sensitivity and the change in pleasantness and desire to eat, i.e. individuals more sensitive to reward showed a greater decline in pleasantness and desire to eat during the meal. This finding is in contrast with a previous study in which total BAS scores (reward sensitivity) were positively associated with intake, but negatively associated with perceived fat content of a non-sweet yoghurt (Valkauskaite & Gibson, 2010). The negative association observed in this study is surprising as it was expected that individuals more sensitive to rewarding stimuli would show sustained pleasantness and desire to eat while eating the sweet version (Davis, 2009; Davis et al., 2007). These responses may reflect an inconsistency between the self-report and behavioural measures of reward.

The BAS scale provides a measure of overall sensitivity to reward unrelated to food, yet it may be that it does not capture a more specific aspect of reward; a sensitivity to rewarding stimuli from appetizing combinations of fat and sugar. It has been argued that reward has been used to describe a number of behaviours relating to abuse and addiction, yet it is not known whether these behaviours have a common underlying process or whether different aspects of reward are related (Stephens et al., 2010). Although other studies have reported association between sensitivity to reward and preferences for high-fat sweetened foods (Davis et al., 2004; Davis & Fox, 2008), the population groups observed were predominantly female whereas this study included a heterogeneous sample of men and women. Further studies will need to investigate the specific aspects of food reward in relation to personality traits.

3.4.1 Limitations:

There are several limitations to the study. Firstly, the study design did not include a low-fat control (for instance a low-fat sweet and non-sweet condition). Comparisons of food intake and appetite responses across the high/low-fat, sweet/non-sweet conditions would have strengthened the study hypothesis. Importantly including a low-fat control would have determined the interaction effect of fat and sweetness on appetite and food intake.

Secondly, although there were findings that were statistically significant ($p<0.05$), these findings were underpowered, and it was determined that an additional 30 participants were needed to achieve statistical power $(1-\beta) > 80\%$. Although participants consumed more of the sweet rice pudding, which in real terms, amounted to an additional 82 g of sweet rice, 147 kcals of energy and 8 g of dietary fat, nevertheless the additional rice consumed does not imply overconsumption per se. Instead, overconsumption is more accurately defined as a higher energy intake relative to energy requirements captured over several meals or a longer period (Fay et al., 2012). However, participants were encouraged to consume as much as they would for a lunch meal, therefore relative to a non-sweet nutritionally equivalent alternative, the additional intake of the sweet condition may reflect a form of overeating.

Thirdly, the association between psychological traits and appetite responses were not corrected for multiple comparisons, increasing the likelihood of a type 1 error (Colquhoun, 2017) therefore further research is needed to investigate if restrained eating behaviour heightens the appetite and reward responses to the palatable tastes of fat and sweetness.

Fourthly, the study did not measure the habitual intake of sweet foods or drinks nor habitual preferences for sweetness, which may have influenced the taste and appetite evaluations. Habitual consumption of sweet foods and drinks may alter the reward value and subjective appetitive responses to such foods (Burger & Berner, 2014; Green & Murphy, 2012; Rudenga & Small, 2012).

Lastly, the within-meal appetite ratings were measured at 30-g intervals instead of 50 g used by Yeomans et al., (1996, 2000; 1997; Yeomans & Gray, 1997). The physical properties and overall size of the rice meals differ from the pasta meal used as a test food in those studies. It was decided that at 50-g intervals, a greater number of spoonfuls per volume of food was required before the participant was alerted to an appetite rating. This could have led to fewer appetite ratings per meal, thereby reducing the ability to detect subtle changes in appetite throughout the meal. However, the difference between interruptions taken at 30 g or 50 g was not tested, and these differences may have resulted in small, but noticeable changes in subjective evaluations.

3.4.2 Conclusion:

This study extends the previous work in this laboratory, demonstrating the appetising and synergistic effect of sweetness in high fat food. The addition of sweetness to a high-fat food enhanced the appetitive sensory experience on first tasting and provided a more rewarding ingestive experience than consuming a high-fat food alone. The heightened palatability may have influence appetite and reward-related processes to sustain hunger and the motivation to eat, which in turn encouraged a higher intake of food. This experiment also indicates that the heightened palatability may have stimulated appetite and provided a more rewarding taste experience for restrained eaters, which may indicate a possible mechanism by which cognitive restraint leads to disinhibited eating, or vice-versa – direction of causation cannot be determined here. These data indicate that unnaturally high levels of fat and sugar in foods may elicit a supra-normal sensory and reward response with eating. This mechanism may contribute to the overconsumption of energy-dense, palatable foods implicated in the development of obesity. However, further research is required to confirm the findings of this experiment.

Chapter 4: Changes in ghrelin, appetite and food intake with consumption of a high-fat, sweet versus non-sweet rice meal

4.1 Introduction

Over the past several decades, there has been a dramatic increase in the production and distribution of processed, energy-dense foods (Monteiro et al., 2017; Swinburn et al., 2011). These foods are engineered by food manufacturers to offer maximum palatability and appeal by cleverly combining high levels of fat, sugar, salt and other flavourings (Monteiro et al., 2017). However, overconsumption of these foods is associated with weight gain and excess adiposity (Crino et al., 2015; Steele et al., 2018; Swinburn et al., 2009), which indicates that the heightened palatability of the food may provoke overeating. The scientific understanding of the underlying physiological mechanisms and individual susceptibility is limited.

In the previous chapter, the addition of sweetness to a high-fat food greatly enhanced the palatability and desirability of the food and participants consumed more of the sweet condition compared to the non-sweet, equicaloric version. Importantly, in the early stages of the meal, sweetness sustained feelings of hunger, and the motivation to eat, and these changes were associated with higher food intake. Similar findings have also been observed with the consumption of a high-fat, sweet plain yoghurt, where sweetness increased pleasantness and sustained feelings of hunger in the early stage of eating (Gibson et al., 2008; Valkauskaite & Gibson, 2010). This suggests that the enhanced palatability (or some other effect of sweetness) modifies the intrameal appetite signals that control food intake, specifically the appetite hormones that regulate hunger and satiety (Chaudhri et al., 2008).

One appetite hormone that may be involved in an altered response to palatable food is ghrelin. Ghrelin, is a gut peptide shown to have an orexigenic effect on appetite and food intake (Chaudhri et al., 2008; Cummings, 2006; Valassi et al., 2008) and is also involved in several physiological processes including the regulation of body weight (Muller 2015; Stengel and Tache 2012, Perello and Zigman, 2012). Ghrelin is a 28-amino acid peptide released from 'A-X like' cells in the stomach (Date et al., 2000; Kojima et al., 1999). Ghrelin must be acylated by enzyme ghrelin-*O*-acyl transferase (GOAT) to form acyl-ghrelin to exert a biological effect via GHS-R1a receptors (Sun 2004; Muller 2015). Ghrelin receptors, GHS-R1a, are found in high concentrations in the pituitary gland and arcuate nucleus of the hypothalamus, consistent with ghrelin's role in regulating the release of growth hormone and influencing energy homeostasis, respectively (Camiña, 2006; Hou et al., 2006; Sun et al., 2004; Van Der Lely et al., 2004).

Circulating levels of ghrelin rise with fasting and decrease rapidly with food ingestion (Cummings et al., 2001, 2002). Preprandial ghrelin levels peak shortly before a meal, paralleled by the increase in subjective feelings of hunger (Cummings et al., 2004; Frecka & Mattes, 2008). Initially, ghrelin was thought to play a role primarily in meal initiation (Cummings, 2006). However, more recent studies have found that the preprandial rise in ghrelin varies with an individual's meal pattern, indicating that ghrelin may also be involved in the anticipation of feeding (Frecka & Mattes, 2008). The postprandial suppression of ghrelin occurs in proportion with the energy intake of the meal, that is a greater postprandial suppression is observed with higher energy intake (Cummings et al., 2004; Wren et al., 2001). The macronutrient content of the meal may influence the magnitude of ghrelin suppression as carbohydrates appeared to suppress levels more than fats and proteins (Bowen et al., 2006; Dit El Khoury et al., 2006; Foster-Schubert et al., 2008; Monteleone et al., 2003; Ouwens et al., 2003; Tentolouris et al., 2004). However other studies have reported that ghrelin responses are similar with the consumption of mixed macronutrient meals (Batterham et al., 2006; Brennan et al., 2006; Gibbons et al., 2013; Maffeis et al., 2010; Van Der Klaauw et al., 2013).

Ghrelin's role as an anticipatory signal for food intake is further supported by its involvement in the Cephalic Phase Responses (CPR) that occur before and during food ingestion. The CPR describes several physiological, endocrine and autonomic processes that prepare the gastro-intestinal tract to receive and process nutrients (Power & Schulkin, 2008; Smeets et al., 2010). The CPR also responds to food-related stimuli, such that the taste, smell and sight of food, for instance, triggers the release of salvia and gastric juice to aid digestion (Hsu et al., 2016).

Ghrelin forms part of the CPR and has also been found to respond to variations in food cues, such as the taste of the food. Using a modified sham-feeding model (a procedure where participants chew the test food in the mouth then spit it out rather than swallowing), studies have demonstrated that ghrelin levels are suppressed with sham-feeding similarly to that observed with the consumption of a meal (Arosio et al., 2004; Smeets et al., 2009). Two studies have reported an early rise in ghrelin within the first 15-minutes of sham-feeding either a bacon and cheese toastie (Simonian et al., 2005) or cheese (Zhu et al., 2014). The early rise in ghrelin observed in these latter studies may be related to food palatability, because the toastie and cheese were arguably more palatable than test foods provided in the previous studies (white bread, ham and boiled egg), although the palatability of the test foods was not reported. Other experiments have demonstrated an early rise in ghrelin when participants are exposed to palatable food cues (Monteleone et al., 2012, 2013). Furthermore, one study demonstrated a variation in ghrelin responses with the expectations of the healthfulness of a meal. When participants were presented with a milkshake that was labelled as 'indulgent' or 'sensible', yet contained the same amount of energy, ghrelin levels were significantly higher following the consumption of the 'indulgent' compared to the ‘sensible’ milkshake (Crum et al., 2011). Taken together, these studies demonstrate that the release of ghrelin is responsive to variations in food cues and expectations about the nutritional content of food.

Alongside the role in homeostatic feeding, ghrelin influences the hedonic processes regulating eating behaviour. GHS-R1a receptors have been located in reward-related regions of the brain, such as the Ventral Tegmental Area (VTA) and amygdala (Guan et al., 1997; Zigman et al., 2006). Ghrelin acts on the dopaminergic neurons in these areas to increase neural activity and dopamine turnover (Abizaid et al., 2006). More specifically, ghrelin acts to increase the reward value of foods (Perelló & Zigman, 2012) and the motivation to eat (Overduin et al., 2012). Studies in rats and mice have demonstrated that ghrelin administration increases the preference and intake of sweet and fat foods (Disse et al., 2010; Perello et al., 2010; Shimbara et al., 2004) and increases in reward-based feeding and hyperphagia (Naleid et al., 2005; Wei et al., 2015). In human studies, exogenous administration of ghrelin has been found to stimulate neural activity in central regions associated with reward and motivational behaviour; in regions such as the amygdala, orbitofrontal cortex, anterior insula and striatum (Malik et al., 2008). Furthermore, both fasting (endogenous) or administration (exogenous) of ghrelin increased bias toward energy-dense, high-fat foods indicating that ghrelin plays a role in modulating the rewarding value of food, thereby increasing the incentive to eat (Goldstone et al., 2014).

A further important consideration is the individual variation in ghrelin responses that may render some individuals more susceptible to palatable combinations of fat and sugar. The individual variation in ghrelin responses has been reported in people with amphetamine dependence (Suchankova et al., 2013), alcohol dependence (Landgren et al., 2010) and hyperphagic and hypophagic depression (Cerit et al., 2019), suggesting a disruption in the ghrelinergic regulation in these behaviours. In non-clinical individuals, higher fasting ghrelin levels are associated with higher levels of reward sensitivity and trait impulsivity (Ralevski et al., 2018), and increased preference for palatable, energy-dense foods (Beaver et al., 2006; Kroemer et al., 2013). Therefore, the individual variation in personality traits and appetite behaviours may predict an altered ghrelin response with consumption of high-fat, sweet food.

The aims of this study were to determine whether (i) sweetness in a high-fat food would alter early prandial and postprandial ghrelin responses and encourage a higher food intake, (ii) prandial and postprandial responses would be associated with appetite and sensory evaluations of food (iii) behavioural traits associated with eating behaviour would predict early and postprandial ghrelin responses to consuming a high-fat sweetened food.

I hypothesised that:

1. The addition of sweetness to a high-fat rice meal modifies the prandial and postprandial acyl ghrelin responses in comparison to a non-sweet, equicaloric alternative.
2. Variation in prandial and postprandial acyl ghrelin responses with consumption of a sweet, high-fat food is associated a heightened sensory experience on first tasting, changes in appetite during the meal or increased food intake compared to a non-sweet condition.
3. Individual variability in eating behaviours and personality traits predict a heightened ghrelin response with consumption a high-fat, sweet rice meal.

4.2 Methods

4.2.1 Participants:

Fifteen volunteers (4 men and 11 women) were recruited from staff and student communities at the University of Roehampton to participate in the study. From this group, blood samples from ten participants (8 females, 2 males) were analysed for plasma acyl ghrelin responses. Prospective participants were excluded from the study if they were allergic to or intolerant of milk products or were restricting their diet, trying to lose weight or had a dietary restraint score > 3.5 (Tatjana van Strien et al., 2009). Four prospective participants were excluded based on these criteria. Participants were naïve to the hypotheses of the experiment and were told only that the objective was to assess individual taste preferences for a rice meal. The experimental protocol was approved by the University of Roehampton Ethics committee PSY14_132.

4.2.2 Design:

The study employed the same study design as described in Chapter 3, with additional appetite assessments and measurement of plasma acyl ghrelin. Blood samples were obtained for the measurement of acyl ghrelin premeal (baseline – T0), at 5 minutes (T5), 15 minutes (T15), 30 minutes (T30) and 60 minutes (T60) from the start of eating the meal.

4.2.3 Test meal

The test meal consisted of two nutritionally equivalent high-fat sweet and non-sweet rice meals as described in Chapter 3.

4.2.4 Procedure

Testing followed procedures described in Chapter 3. Height (m), weight (kg), and body composition was measured using the 4-compartment body composition analyser TANITA-BC 418 MA (Amsterdam, Netherlands). Total daily energy requirements were calculated using a new predicted equation derived from the CALERIE study (Redman et al., 2014). A standardised breakfast meal was provided to participants and calculated to provide approximately 20% of each participant's daily energy requirements. Breakfast consisted of cereal (Kelloggs TM), semi-skimmed fresh milk, toasted white bread (Hovis soft white, Hovis), butter and jam or honey and tea (English breakfast tea, Twinings and Company, LTD) or coffee (Nescafé, Nestlé).

Following breakfast, participants were asked to carry out normal duties until the trial session, but not to eat or drink anything except water. The participants returned to the laboratory three hours following breakfast and were instructed on the test procedures. The participants were then placed in a semi-supine position, and a catheter was inserted into the forearm vein. A baseline blood sample was drawn, and the line was flushed with sterile physiological saline to maintain vascular access during the test period. The participants were moved to an upright seat in front of a computer screen where the test meal was consumed. The participants completed an appetite assessment to assess levels of hunger, fullness and sickness. Following the assessment, participants were provided with either a sweet or non-sweet version of the meal and asked to "Please consume as much or as little food as you like and please eat at your own pace". Blood samples and appetite scores were obtained at T5, T15, T30 and T60 following the start of the meal. After the meal, the participants returned to a semi-supine position and completed the psychometric questionnaires. Test procedures were repeated approximately one week later.

4.2.5 Measures:

4.2.5.1 Sussex Ingestion Pattern Monitor (SIPM)

Data were collected using the Sussex Ingestion Pattern Monitor (SIPM v2) as described in Chapter 3

4.2.5.2 Appetite assessments

In addition to appetite assessment taken before, during and after the meal, appetite assessments of hunger, fullness and sickness were made using an electronic version of the 100-point visual analogue scales (VAS) presented on a handheld computer (iPad, version 4; Apple, Inc). The iPad was for ease of use and to ensure minimal movement of the participant during assessment. Measurements were taken at baseline (T0), T5, T15, T30, T60 shortly before blood sampling.

4.2.5.3 Measures of personality traits and eating behaviour

Descriptions for each questionnaire used in this chapter can be found in Chapter 3.

4.2.5.3.1 Three-factor eating questionnaire (TFEQ-R18V2)

The Cronbach's alpha values for this chapter were 0.88, 0.71 and 0.91 for cognitive restraint, uncontrolled eating and emotional eating respectively.

4.2.5.3.2 Power of food scale (PFS)

The Cronbach alpha value for total PFS in this study was 0.88.

4.2.5.3.3 Behavioural Inhibition System and Behavioural Activation System (BIS-BAS)

Cronbach alpha values were 0.76 for Total BAS scores and 0.56 for BAS fun-seeking, 0.70 for BAS drive and 0.64 for BAS reward responsiveness

4.2.5.3.4 Barratt Impulsiveness Scale (BIS-15)

The Cronbach alpha values for Total BIS-15 scores were 0.71.

4.2.6 Hormone analysis

4.2.6.1 Procedure

Blood was drawn into 5-ml sterile syringes and transferred to two 1-ml EDTA-lined polypropylene tubes and placed on ice. To prevent the degradation of acylated ghrelin, 4-(2-Aminoethyl) benzenesulfonyl fluoride hydrochloride (AEBSF) was added to the polypropylene tubes at concentration of 1 $mg.ml^{-1}$. Samples were centrifuged at 1500 *g* for 10 minutes, then plasma was separated into four aliquots. Plasma samples were acidified with 1M HCL. All samples were stored at -20°C until further analysis.

Plasma active (acyl) ghrelin is considered a more informative measure of ghrelin activity (Cummings et al., 2005; Mackelvie et al., 2007). Active ghrelin levels were measured in duplicate using a sandwich enzyme-linked immunosorbent assay (ELISA) produced by Merck Millipore (now Sigma-Aldrich) (Human Ghrelin (active) 96-well plate Cat. #EZGRA-88K). All laboratory work was carried out in the clinical laboratory at the University of Roehampton. The lowest level of total ghrelin detected by the ELISA kit was 15 pg/ml. The appropriate range of the assay was 25 pg/ml to 2,000 pg/ml active ghrelin in a 20-µl sample. The intra-assay coefficient of variation for this assay is 3.9% and the inter-assay variation for this assay is 9.9%.

4.2.7 Data analysis:

Tests for normality and equal variance were conducted on all variables and adjustments to scores or tests were made if data were non-normal or of unequal variances. Appetite assessments (initial taste ratings, intake and change in appetite ratings in the early stages of the meal), and association of BMI, eating styles, eating behaviours, test meal intake and sensory ratings were conducted on all 15 participants. Plasma acyl ghrelin responses (change in ghrelin, AUCg, AUCi, AUCi/kcal) and associations of early plasma acyl ghrelin responses with sensory evaluation, appetite ratings, test meal intake were conducted on ten participants.

Sweetness, creaminess ratings for the sweet condition and initial hunger and adjusted ideal creaminess ratings for non-sweet condition were not normally distributed (please refer to Table F1 in Appendix F). Therefore, a Wilcoxon signed-rank test was used to determine difference between test conditions. To determine if there were differences between the sexes in body composition, energy expenditure, eating behaviour, and personality trait scores were assessed using one-way analysis of variance (ANOVA).

Overall differences between sweet and non-sweet meals in intake, initial sensory ratings for sweetness, creaminess, estimated percentage fat, ideal sweetness and creaminess, pleasantness and desire to eat, acyl ghrelin responses (change in ghrelin, AUCg, AUCi, AUCi/kcal) were analysed using general linear model (GLM) repeated measures ANOVA. The Greenhouse-Geisser (Geisser & Greenhouse, 1959) correction was applied if Mauchly's test of sphericity was violated. Difference from ideal' sweetness and creaminess ratings were calculated by subtracting the raw score from the 'ideal' midpoint of a 100-mm horizontal line (50 mm). A one-sided t-test was used to determine whether 'difference from ideal' sweetness and creaminess ratings differed significantly from ideal (0). A two-way repeated measures ANOVA was conducted to determine the differences in appetite ratings for hunger or fullness between the sweet and non-sweet condition over the 60-minute test period.

Pearson's correlations were used to determine associations between anthropometric measurements, psychometric scores, sensory appetite ratings, prandial and post-prandial ghrelin and appetite responses. Spearman's correlation analysis was used to determine associations using non-parametric data (BMI, body fat, initial ratings for sweetness, creaminess, and adjusted ideal creaminess).

All appetite values of zero were given a value of 1 to allow for further calculations. To assess changes in subjective appetite (hunger, pleasantness, desire to eat) and plasma acyl ghrelin during the first five minutes of the meal, calculations were made by subtracting the measurement obtained at T5 (5 minutes) from the baseline measurement and dividing the difference by the baseline rating as follows:

$$\boldsymbol{Change\ in\ appetite} = \frac{\boldsymbol{T5\ measurement - T0\ (baseline)\ measurement}}{\boldsymbol{T0\ (baseline)\ measurement}}$$

To determine changes in acyl ghrelin and appetite (hunger, sickness, fullness) over the test period, the area under the curve (AUC) was presented in two ways, 1) the change from baseline 0 (AUCg) 2) the increase from baseline values (AUCi) (Khoury et al., 2015; Pruessner et al., 2003). Since changes in plasma acyl-ghrelin are associated with total energy intake (Callahan et al., 2004), a calculation was made to determine the acyl-ghrelin, and appetite, responses in proportion to the total energy consumed as follows:

$$\boldsymbol{Energy\ intake\ adjusted\ Ghrelin\ or\ Appetite\ response} = \frac{\boldsymbol{AUCi\ ghrelin\ or\ appetite}}{\boldsymbol{Energy\ intake\ (kcal)}}$$

Several blood samples were severely haemolysed, making accurate measurement of acyl-ghrelin difficult for a total of five values across the participant group. Similarly, fifteen appetite scores were missing due to technical error during data collection. To replace these values, a linear interpolation method was used as follows:

$$\boldsymbol{y\ missing = y2 + (x\ missing - x1) * \left(\frac{y2 - y1}{x2 - x1}\right)}$$

Where **x missing =** time interval of missing value**; y missing =** missing value; **y1 =** value before missing value; **y2 =** value subsequent to missing value; **x1 =** time interval before missing value; **x2 =** time interval subsequent missing value.

All data were analysed using SPSS version 25.0 (IBM, Chicago). An alpha criterion of $p < 0.05$ was used to determine statistical significance.

4.3 Results

4.3.1 Participant characteristics

Participant characteristics are listed in Table 4.1. In the full sample (N = 15), the age of participants ranged from 20 – 39 years, mean ± 1SD 27.7 ± 1.4 years, and body mass index (BMI) was 23.5 ± 3.4. There were some sex differences; females had a lower BMI yet had more body fat compared to males. Males had a higher total energy expenditure (TDEE). Females also had higher BIS and Total BAS scores. No other differences were observed between participants.

Table 4.1:

Age (years), body mass index (BMI), percentage body fat and total daily energy expenditure (TDEE), personality traits and traits of eating behaviour for participants (n = 15)

	Total (*N* = 15)	Male (*N* = 4)	Female (*N* = 11)
Age (years)	27.7 ± 5.5	30.8 ± 7.9	26.5 ± 4.3
	(20 - 39)	(20 - 39)	(20 - 32)
BMI (kg.m^{-2})	23.5 ± 3.4	24.7 ± 1.8	23.1 ± 3.8
	(19.2 - 33.4)	(23.2 - 27.2)	(19.2 - 33.4)
Percentage body fat (%)	30.4 ± 8.1	20.4 ± 2.2[b]	34.1 ± 5.9
	(18.6 - 48.6)	(18.6 - 23.5)	(27.2 - 48.6)
TDEE (kcal.day^{-1})	2408.8 ± 465.1	3074.3 ± 377.7[a]	2166.8 ± 136.1
	(1971.7 - 3541.2)	(2643.2 - 3541.2)	(1971.7 - 2368.7)
Cognitive restraint	2.3 ± 0.8	1.8 ± 0.7	2.5 ± 0.9
	(1 - 4)	(1 – 2.7)	(1 - 4)
Uncontrolled eating	2.4 ± 0.4	2.0 ± 0.4	2.5 ± 0.4
	(1.6 – 3.0)	(1.6 – 2.6)	(1.6 – 3.0)
Emotional eating	1.9 ± 0.7	1.5 ± 0.5	2.1 ± 0.8
	(1 – 3.7)	(1 - 2)	(1 – 3.7)
BIS	23.1 ± 2.7	19.5 ± 1.9[b]	24.4 ± 1.4
	(17 - 26)	(17 - 21)	(22 - 26)
Total BAS	40.4 ± 4.5	38.3 ± 5.9	41.2 ± 3.9
	(32 - 47)	(32 - 46)	(35 - 47)
Total PFS	43.2 ± 10	38.8 ± 10.8	44.8 ± 9.7
	(23 - 56)	(25 - 50)	(23 - 56)
Total BIS	29.4 ± 6.7	31.5 ± 10.5	28.6 ± 5.2
	(18 - 43)	(18 - 43)	(22 - 37)

Mean ± 1 standard deviation (minimum to maximum values), BIS: Behavioural Inhibition system, Total BAS: Total score for Behavioural Activation System, Total PFS: total score for Power of Food Scale, BIStotal: Total score for Impulsivity; [a] $p < 0.05$ Significantly higher than females, [b] $p < 0.001$ Significantly lower than females.

4.3.2 Initial sensory and appetite ratings

The appetite and sensory ratings on first tasting the sweet and non-sweet rice meal are described in Table 4.2. As expected, sweetness, pleasantness and desire to eat ratings were greater for the sweet compared to non-sweet meal: Sweetness, median z = -3.18, $p = 0.001$; Pleasantness, $t(14)$ 3.48, $p = 0.004$; Desire to eat , t (14) = 2.95, $p = 0.011$. Difference from ideal sweetness and creaminess ratings were closer to ideal for the sweet than non-sweet rice meal. Ideal sweetness for the non-sweet condition was significantly lower compared to ideal $t(14) = -3.37$, $p = 0.005$) Baseline hunger, fullness and sickness scores were similar on both test days ($p > 0.05$).

Table 4.2:

Initial sensory ratings for sweetness, creaminess, estimated fat content (expressed as percentage fat; % Fat), pleasantness, desire to eat and 'difference from ideal' sweetness and creaminess for sweet and non-sweet rice meal.

	S	NS	
Sweetness	26.5 ± 3.7 (median: 21)	7.4 ± 1.3 (median: 6)	$p<0.001$[a]
Creaminess	33.6 ± 3.5 (median: 35)	37.9 ± 4 (median: 37)	NS[b]
% Fat	50.1 ± 6.2	45.1 ± 5.6	NS
Pleasantness	73 ± 4.5	58.1 ± 5	$p < 0.05$
Desire to eat	74.3 ± 4.1	60.4 ± 5.1	$p < 0.05$
Ideal Sweet (adjusted)	-0.4 ± 3	-15.3 ± 4.5	S: non sig; NS $p=0.005$
Ideal Creaminess (adjusted)	-0.4 ± 2.5 (median: 0)	0.9 ± 5.1 (median: 0)	S: non sig; NS: non sig[c]

Mean ± 1SD; S = sweet condition; NS = non-sweet condition; non sig = non-significant; [a] Wilcoxon signed-rank test median z-score = -3.18, [b] median z-score = 0.94, [c] median z-score = -0.82

4.3.3 Intake

Participants did not consume significantly more of the sweet rice meal compared to the non-sweet condition, despite a 13% greater group mean intake for the sweet vs. non-sweet meal. Values are mean ± 1SEM (Sweet 315.11 ± 40.88 vs Non-sweet 279.57 ± 38.30, $p = 0.332$).

4.3.4 Change in hunger, desire to eat and pleasantness ratings during the first part of the meal

The change in appetite scores during the first five minutes of the meal was similar between the sweet and non-sweet conditions.: Hunger: Sweet -0.17 ± 0.10 vs Non-sweet -0.23 ± 0.045, t (13) = 0.59, $p = 0.56$; Desire to eat: Sweet -0.30 ± 0.11 vs Non-sweet -0.21 ± 0.084, t (13) = -1.08, $p = 0.30$; Pleasantness Sweet -0.18 ± 0.072 vs Non-sweet -0.15 ± 0.093, t (13) = -0.42, $p = 0.69$.

4.3.5 Percentage change in plasma acyl ghrelin over 60 minutes

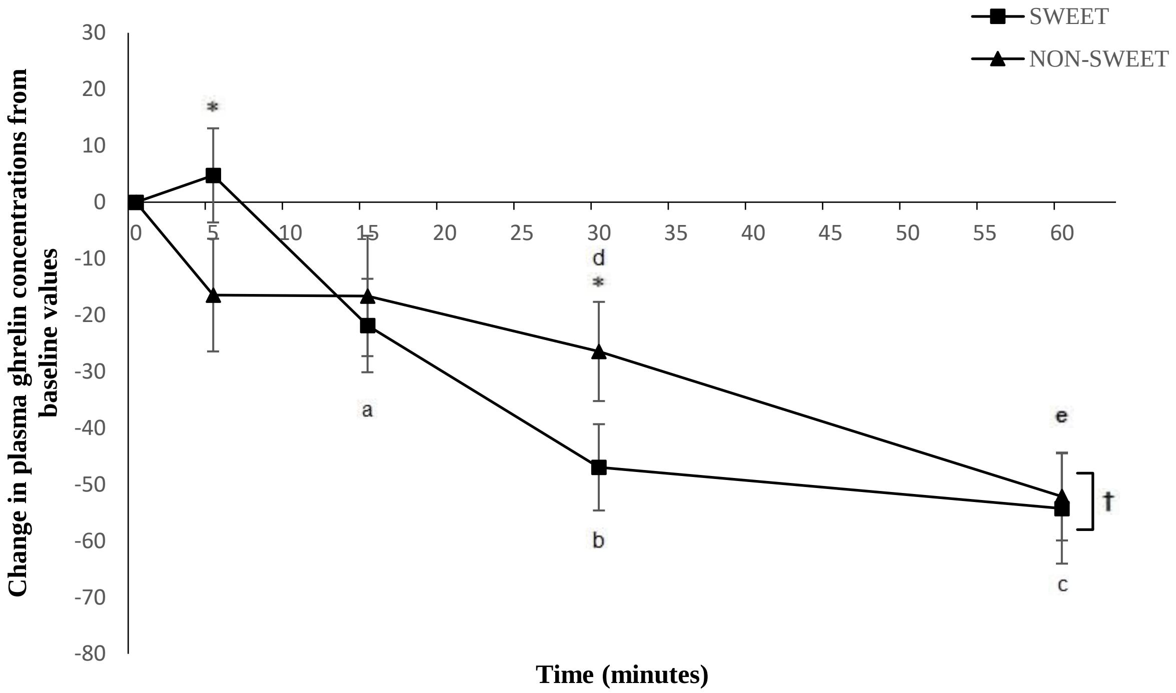


*Figure 4.1: Change in plasma acyl ghrelin values (pg.ml^{-1}) over 60 minutes during and following consumption of a sweet or non-sweet rice meal. Error ± 1 SEM mean is (square) or (triangle). *Sweet different to non-sweet, [a] Sweet T15 different from baseline; [b] Sweet T30 different from baseline, [c] Sweet T60 different from baseline, [d] Non-sweet T30 different from baseline, [e] Non-sweet T60 different from baseline †Main effect of time.*

The change in acyl ghrelin over the test period differed between the sweet and non-sweet condition and over time, time*condition, F (4, 36) = 15.22, p < 0.001, partial η^2 = 0.63. There was a simple effect of time where the acyl ghrelin levels showed a significant decrease from baseline at T15 -5.85 ± 8.70, p = 0.033; T30 -19.23 ± 7.63 and T60 -53.19 ± 7.83, p < 0.001, F (4, 6) = 8.41, p = 0.012, partial η^2 = 0.85. Multiple comparisons supported the following interpretations of the interaction: at T5, acyl ghrelin fell from baseline after the non-sweet condition but not after the Sweet condition, mean difference ± 1SEM: 21.20 ± 5.92 [7.76 to 34.64], p = 0.006; whereas by T30 mean acyl ghrelin levels had fallen further below baseline for the Sweet condition compared to the Non-sweet condition, -20.50 ± 8.94

[-40.73 to -0.31], *p* = 0.047. By T60, acyl ghrelin had fallen to same levels below baseline for both sensory conditions (see Figure 4.1).

4.3.6 Appetite changes over 60-minutes

The change in hunger from baseline did not differ between the sweet and non-sweet condition, and did not inteact with time, time*condition effect, Greenhouse-Geisser correction factor was applied, Epsilon (ε) = 0.38, F (1.5, 13.5) = 0.12, p =0.97. There was no main effect for condition; F (1, 9) = 0.22, p =0.65. There was a main effect of time, Greenhouse-Geisser correction Epsilon (ε) = 0.43, F (1.7, 15.5) = 4.68, p =0.004, partial η^2 = 0.34. Hunger was suppressed at T5 mean difference ± 1SEM [95%CI] 0.25 ± 0.07 [0.1 to 0.40], p = 0.005, T15 0.46 ± 0.10 [0.23 to 0.69], p = 0.002, T30 0.43 ± 0.13 [0.14 to 0.72], p = 0.008 but resumed close to baseline values at T60 0.25 ± 0.18 [-0.15 to 0.65], p = 0.18. (see Figure 4.2).

There was no interaction of condition and time on subjective feelings of fullness; time*condition effect, F (1,9) = 0.00 = 0.99. There was a main effect for time, F (4, 36) = 4.18, p =0.007, partial η^2 = 0.32. Multiple comparisons revealed that fullness tended to be elevated from baseline at T5 -13.14 ± 6.96 [-28.88 to 2.60], p = 0.092, T15 -17.19 ± 8,28 [-35.92 to 1.55], p = 0.068, T30 -17.90 ± 8.66 [-37.49 to 1.70], p = 0.069 and T60 -16.42 ± 7.71 [--33.87 to 1.02], p = 0.062

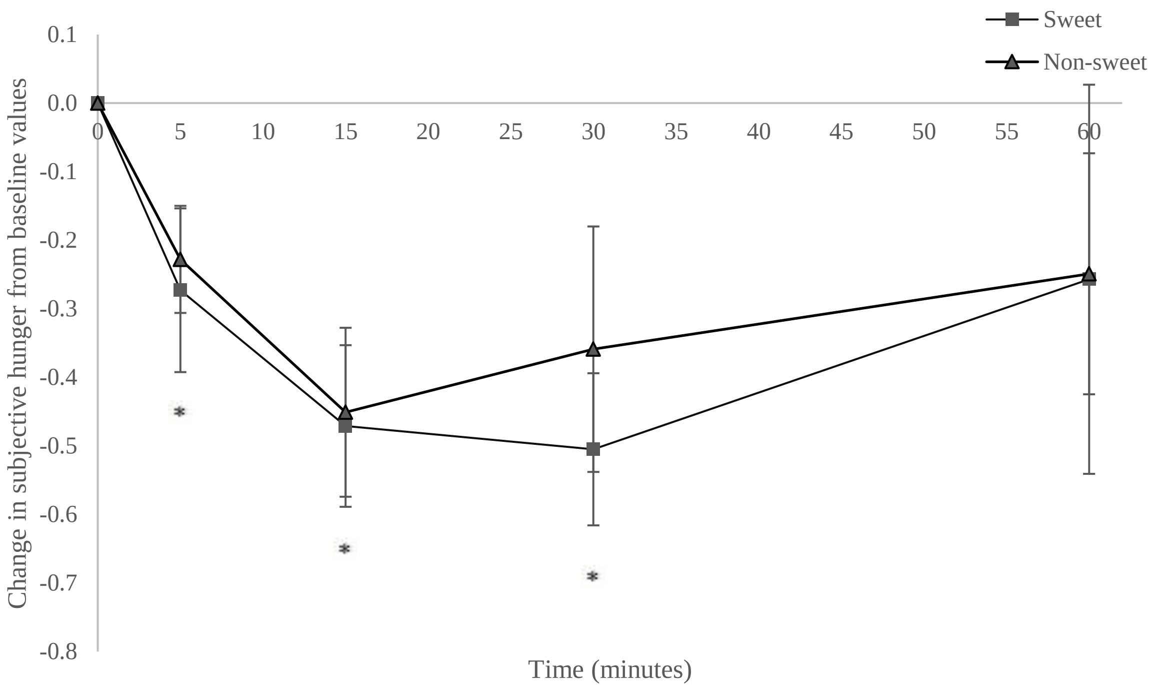


*Figure 4.2 Mean change in subjective hunger scores from baseline values over 60 minutes test period during and following consumption of a sweet or non-sweet rice meal. Error ± 1 SEM mean is (square) or (triangle). *p<0.05 Hunger suppressed at T5, T15 and T30 from baseline*

4.3.7 Area under the curve (with respect to increase: AUCi)

The mean ± SE values are presented in Table 4.3 and Table 4.4 below. There was no significant differences in the acyl ghrelin responses for AUCg, AUCi, and AUCi.kcal^{-1}(area under the curve as a proportion of energy intake, kcal), between the sweet and non-sweet conditions (AUCg, p = 0.61, AUCi.kcal^{1}, p = 0.54). Similarly, there were no differences in appetite responses (AUCg and AUCi) between sweet and non-sweet conditions.

Table 4.3

Area under the curve with respect to ground (AUCg) or increase (AUCi) and AUCi as a proportion of energy intake (kcal) for changes in plasma acyl ghrelin ($pg.ml^{-1}$)over the 60-minute period following consumption of a Sweet or Non-sweet high fat rice meal.

	Sweet	Non-sweet
AUCg	30426 ± 5145	32561 ± 5973
AUCi	-24404 ± 8482	-22301 ± 8804
$AUCi.kcal^{-1}$	-53 ± 16	-63 ± 22

Values are Mean ± 1 SEM

Table 4.4

Area under the curve with respect to ground (AUCg) or increase (AUCi) and AUCi as a proportion of energy intake (kcal) for changes in appetite (hunger, fullness, sickness) over the 60-minute period following consumption of a Sweet or Non-sweet high fat rice meal.

	Hunger		Sickness		Fullness	
	S	NS	S	NS	S	NS
AUCg	2171 ± 391	2160 ± 311	1512 ± 427	1005 ± 25	3407 ± 299	3705 ± 228
AUCi	-1675 ± 441	-1351 ± 409	-96 ± 367	105 ± 248	1877 ± 432	2037 ± 527
AUCi.kcal^{-1}	-3.32 ± 3.7	-5.87 ± 7.33	3.97 ± 0.94	9.2 ± 3.37	3.97 ± 2.98	9.2 ± 10.67

Values are Mean ± 1 SEM

4.3.8 Association of BMI, eating styles, eating behaviours and test meal intake and sensory ratings

A higher BMI was associated with greater fat mass (kg) r (14) = 0.79, p = 0.001. Emotional eaters and uncontrolled eaters tended to report higher total PFS scores, emotional eating: scores r (14) = 0.47, p = 0.089, uncontrolled eating: r (14) = 0.51, p = 0.064 yet consumed less of the sweet condition, emotional eaters r (13) = -0.63, p = 0.019, uncontrolled eaters r (13) = -0.73, p = 0.005. Restrained eaters demonstrated a sustained level of pleasantness when eating the sweet condition in the early stages of the meal, r (14) = 0.60, p = 0.022. Restrained eaters also demonstrated sustained desire to eat, but this was observed for both sweet, r (14) = 0.49, p = 0.076 and non-sweet, r(14) = 0.48, p = 0.08.

High scores of reward sensitivity (total BAS) were associated with less intense sweetness ratings, *rho* = -0.63, p = 0.015

4.3.9 Associations of early plasma acyl ghrelin responses with sensory evaluation, appetite ratings, test meal intake

The early change in acyl ghrelin was not significantly associated with a greater intake of sweet or non-sweet rice meal. However, higher ratings for creaminess of the non-sweet rice were associated with a greater change in acyl ghrelin in the early stages of the meal , r (10) = 0.71, p = 0.023, and similarly, a higher perceived fat content of the sweet condition tended to be associated with a greater change in acyl ghrelin in the early stages of eating, r (10) = 0.61, p = 0.064.

Higher intake of non-sweet rice was associated with a greater change in pleasantness r (12) = 0.68, p = 0.014 and tendency for a greater change in desire to eat, r (12) = 0.57, p = 0.052 in the early stages of the meal.

4.4 Discussion

The purpose of this study was to investigate whether consumption of a high-fat, sweet food would alter prandial and postprandial acyl ghrelin responses in comparison to a non-sweet, equicaloric food. The study also investigated whether these responses were associated with a heightened sensory experience on first tasting, changes in appetite during the meal or increased food intake. Lastly, the study investigated whether individual differences in personality traits and eating behaviour would predict a heightened postprandial acyl ghrelin response with consumption of high-fat, sweet food.

In this study, consumption of a high-fat sweet food did appear to modify prandial ghrelin responses in comparison with the non-sweet condition. In the sweet condition, ghrelin levels remained elevated during the first five minutes of the meal, while falling below baseline values for the non-sweet condition. The early attenuation of acyl ghrelin was not associated with preprandial appetite assessments, sensory evaluations, or with food consumption or energy intake. However, these results should be interpreted with caution as the sample size was small, analyses were not protected for multiple contrasts and findings were largely under-powered.

4.4.1 Early prandial plasma acyl ghrelin responses to consuming high-fat, sweet food

The study found that the addition of sweetness in a high-fat food enhanced its palatability. As with the findings reported in chapter 3, the participants rated the sweet condition as more pleasant and rewarding to eat when first tasting the food. In the early stages of eating, acyl ghrelin levels were sustained in the sweet condition but declined in the non-sweet condition. This suggests that the heightened palatability provided by high-fat, sweet food stimulated the release of ghrelin in the cephalic phase of eating. The ghrelin responses, however, were not associated with increased food intake, or sensory and appetite assessments with first tasting and during the meal. The study did find that participants who rated the non-sweet condition as creamier and tended to perceive the meal to contain a higher level of fat demonstrated a rise (or less of a decline) in acyl ghrelin levels in the early part of the meal. These data suggest that the processes that regulate ghrelin secretion in the cephalic phase of eating may be responsive to both external palatable food cues and internal signals that reflect cognitive, appetitive and sensory perceptions that may depend on experience and individual expectations (Woods et al., 2018).

Although overall changes in ghrelin levels were not related with sensory or appetite evaluations, however it is likely that the study was underpowered to test this association. It is plausible that the heightened ghrelin responses observed in the earlier stages of the meal may be associated with the palatable taste of the food rather than with nutrients absorbed from the gastrointestinal tract. If foods combining the taste of sweetness and fat enhance the palatability of the food and provide a more pleasant sensory experience, this may stimulate orosensory reward processes involved in food perception, providing a positive feedback to central regions regulating feeding behaviour (Yeomans, 2000). The positive feedback may also act on regions regulating the release of appetite hormones, more specifically the release of ghrelin (Møller, 2014). Perhaps the positive orosensory stimulation then acts on these regions to stimulate ghrelin secretion and so encourage food intake.

Ghrelin responses are regulated by vagal afferent and efferent activity via the dorsal vagal complex (Masuda et al., 2000). This area of the hindbrain receives input from sensory modalities (responding to external food cues) and visceral afferent signals that reflect the status of the internal milieu (Powley, 2000). Therefore, the ghrelin responses observed in this study suggest that the positive sensory stimulation from high-fat, sweet stimuli, or less pleasant appetite perceptions that arise with eating, act on neurones in the dorsal vagal complex to modulate the activity of the vagus nerve and regulate ghrelin responses accordingly (Powley, 2000).

Several experimental studies have demonstrated that palatable foods evoke a heightened gut hormone response in the cephalic phase of eating; however, less consistent results have been observed with ghrelin. Monteleone et al. (2015; 2012, 2013) demonstrated an early rise in plasma total ghrelin levels in response to the sight and smell of palatable food (either Italian confectionery served with chocolate or the individual's choice of palatable food) compared to a non-palatable food (bread and butter), although the effect of tasting or sham feeding the food was not investigated in these studies.

Tasting palatable foods have been demonstrated to elicit heightened pancreatic polypeptide (PP) responses when sham feeding high-fat pound cake (Crystal & Teff, 2006) and a sweetened cream cheese cracker (Teff, 2010). Similarly, early increases in plasma insulin levels were observed when sham feeding apple pie (Teff et al., 1995). In contrast, Mennella et al. (2015) reported similar acyl ghrelin response when sham feeding a sweet dessert (palatable condition), a bitter dessert (unpalatable condition) or tasteless dessert condition. Moreover, Lasschuijt et al., (2018) reported no differences in ghrelin and pancreatic polypeptide levels with sham feeding of hard and soft, sweet and non-sweet foods in comparison with fasting. The investigators acknowledged that the moderate palatability ratings for the test foods might have influenced the study outcome. It is noted that the test food used in the Mennella et al. study had a lower fat content (3%) in comparison to the test food used in this study (~50%). Since foods combining high levels of fat and sweetness are more palatable than sweetness (or fat) alone (Drewnowski & Greenwood, 1983; Gibson et al., 2008; Valkauskaite & Gibson, 2010), this further suggests that a higher level of sensory-stimulation offered by fat-sweet stimuli is required to elicit a modified ghrelin response.

Taken together, these studies support the observations in this chapter: that the enhanced palatability provided by high-fat, sweet foods acts to sustain ghrelin in the early stages of eating. These data also suggest that the ghrelin is responsive to unfavourable/less pleasant appetite sensations (such as creaminess sensation) and the perceived health value of a food, perhaps to increase satiation and discourage further eating (Crum et al., 2011; Smeets et al., 2010).

In contrast to the results from the previous chapter, consumption of a high-fat, sweet food did not sustain feelings of hunger or the desire to eat in the early stages of eating. Since the rise in preprandial ghrelin is associated with an increase in hunger (Cummings et al., 2004), and the release of ghrelin is associated with reward-based eating (Goldstone et al., 2014; Naleid et al., 2005; Perelló & Zigman, 2012), it was expected that the heightened ghrelin responses observed in this study might be associated with sustained feelings of hunger and motivation to eat in the early stages of the meal. However, also unlike the previous study, in this group of participants, the sweet rice condition did not result in a significantly greater total meal intake compared to the non-sweet condition, which may have reduced the likelihood of finding differences in appetite ratings and overall ghrelin levels. This study only tested 15 participants (10 participants for prandial ghrelin responses), while 25 participants were included in the previous study; therefore, this study may have been unpowered to examine the association between changes in appetite perceptions and prandial ghrelin responses.

4.4.2 Overall plasma acyl ghrelin responses to eating a high-fat, sweet food

The profile of postprandial acyl ghrelin responses over the 60-minute experimental period was similar for both the sweet or non-sweet conditions. Regardless of whether these responses were expressed as the overall change over time (area under the curve), the overall change as a proportion of energy intake (pg.ml.min.kcal-1) or comparison between conditions over time (two-way ANOVA), there were no differences observed between sweet and non-sweet conditions. Acyl ghrelin levels were significantly lower in the sweet condition at 15-minutes after the start of the meal. However, these responses may reflect a more significant ghrelin suppression with higher food intake, as participants consumed fractionally more of the sweet condition (although this was not statistically significant). As discussed previously, it is likely that the study was underpowered to test these comparisons.

4.4.3 Individual variation in ghrelin response

In this study, individual variation in personality traits and eating behaviour were not associated with early prandial or postprandial ghrelin responses. Emotional eaters and uncontrolled eaters consumed less of the sweet rice meal. This was surprising given that these individuals tended to show a propensity for hedonic hunger (i.e. higher PFS scores). These responses were unrelated to sensory perception or appetite assessments. Similar to the findings reported in Chapter 3, restrained eaters demonstrated a greater reward response in the early stages of eating the rice meals, although, in this study, they appear to derived a rewarding experience from both the sweet and non-sweet condition, suggesting that they are susceptible to high-fat foods *per se*. In the previous chapter (Chapter 3), high BAS individuals demonstrated a greater change in pleasantness in the early stages of the meal. While this finding was not replicated in this study, high BAS individuals rated the sweet condition less intense compared to low BAS individuals. Taken together, these findings suggest that individuals sensitive to reward may differ in their perception of sweetness in food which may influence appetite and food intake. However, no direct association was observed between sweetness and appetite assessments in these individuals, this finding will need to be confirmed in future studies.

The individual variation in ghrelin responses may still predict susceptibility to palatable food cues. Kroemer et al. (2013) reported that higher levels of ghrelin predicted a greater neural reward response to viewing palatable food images, and higher fasting levels were associated with stronger appetite sensations. As stated previously, the small sample size of this study may have been underpowered to examine the individual variation in ghrelin responses to the palatable taste of fat-sweet combinations. Studies of larger sample sizes are required to investigate whether personality traits or eating behaviour predict psychobiological responses to overeating high-fat, sweet foods.

4.4.4 Limitations

There are several limitations to this study. As with the study reported in chapter 3, the design did not include a low-fat control (discussed under section 3.4.1). The sample size was small (n=15 for total sample, n=10 for measurement of plasma acyl-ghrelin levels) and the plasma acyl ghrelin group was unbalanced for sex. The findings in this study were underpowered and it was determined that an additional 20 participants would have needed to be tested to achieve statistical power power $(1-\beta) > 80\%$ (see refer to Appendix L). While other studies report significant differences for small samples sizes (Monteleone et al., 2012, 2013), these studies employed a fixed meal study design, whereas the present study provided the rice meal to be consumed *ad libitum*. This may have contributed to the high degree of variability observed in the data set. Similarly, the non-significant differences observed in energy intake, change in appetite scores or postprandial responses (notably area under the curve) may be due to the small sample size.

Lastly, my study only measured one gut hormone that impacts hunger and food intake, whereas a number of satiety hormones, such as insulin, CCK, GLP-1, PYY and PP have been shown to influence satiation and satiety following food intake (Gibson et al., 2008; Rizi et al., 2018; Van Der Klaauw et al., 2013). Future research should endeavour to consider these factors when planning for study design.

4.4.5 Conclusion

The findings of this study contribute to our knowledge of the role of palatability in regulating appetite and food intake. Specifically, palatable combinations of sweetness and fat may stimulate appetite during the cephalic phase responses in part by attenuating the reduction in ghrelin. Further studies are required to determine how early ghrelin responses influence appetite and food intake during food consumption and whether responses predict overconsumption and reduced satiation, and furthermore, whether individual personality traits and eating behaviour are associated with postprandial ghrelin responses to palatable combinations of sugar and fat.

Chapter 5: Investigating the effect of a low protein meal on appetite, mood, food intake and flavour preferences in individuals whose lifestyles include high, moderate or low levels of physical activity

5.1 Introduction

One of the key factors driving the obesity epidemic is the overconsumption of energy-dense foods, high in fat and sugar or salt (Crino et al., 2015; Scarborough et al., 2011), which has prompted extensive research into understanding why individuals are consuming more food than they need (Berthoud, Münzberg, & Morrison, 2017). The Protein Leverage Hypothesis (PLH) proposes that human appetite is strongly regulated by dietary protein intake; if protein intake is insufficient to meet daily requirements, appetite regulatory mechanisms act to stimulate food intake to reach protein sufficiency at the expense of regulating intake from non-protein sources, i.e. from energy from carbohydrate and fat (Simpson et al., 2003; Simpson & Raubenheimer, 2005, 2012). The authors propose a Geometric Framework for Nutrition (GFN) that conceptualises how an individual or animal will achieve nutrient balance (Simpson et al., 2017).

Using this framework, the PLH predicts that a small decrease in protein intake, for example 1.5% reduction energy from protein, would act to drive appetite and increase carbohydrate and fat intake by 14% to achieve a target protein intake, resulting in a substantial increase in energy intake (Simpson et al., 2017; Simpson & Raubenheimer, 2005). Although extensive evidence has been observed and reported experimentally in animals (Morrison, Reed, & Henagan, 2012; Raubenheimer, Machovsky-Capuska, Gosby, & Simpson, 2015; Raubenheimer & Simpson, 2018), the experimental studies in humans have yielded inconsistent results. It may be that the individual variation in physical characteristics and lifestyle factors, such as differences in protein need, body composition and physical activity, play a small but significant role in directing appetite and food intake in response to mild protein restriction.

Since the proposal of the PLH, several experimental studies have examined the effect of protein restriction on appetite and food intake. These studies involved either consuming food *ad libitum* from a diet providing fixed proportions of energy from protein (%PE), 10%, 15% and 25% PE, for a short duration (four days) (Gosby et al., 2011); or a diet providing 5%, 15% or 30% PE for up to 2 weeks (Martens 2013, Martens 2014, 2014), or a fixed diet containing 5%,15% and 30% PE for 2 weeks followed by an *ad libitum* phase where participants could consume a wide variety of foods (Griffioen-Roose et al., 2012, 2014). Of these studies, only Gosby et al. (2011) demonstrated a leverage effect where participants consumed more energy in the low protein condition (10% energy from protein) compared to the medium (15%) or high (25%) condition. Other studies, however, showed no increase in energy intake in the low (5%) protein condition (Griffioen-Roose, Mars, et al., 2012; Martens, Lemmens, & Westerterp-Plantenga, 2013; Martens, Tan, Mattes, & Westerterp-Plantenga, 2014; Martens, Tan, Dunlop, Mattes, & Westerterp-Plantenga, 2014).

In contrast, consumption of a high protein diet resulted in significant decreases in energy intake and reductions in hunger (Griffioen-Roose, Mars, et al., 2012; Martens et al., 2013; Martens et al., 2014; Martens et al., 2014). Similar findings have been reported in other experimental studies examining the effect of high protein diets or meals on energy intake (Brennan et al., 2006; Dit El Khoury, Obeid, Azar, & Hwalla, 2006; Latner & Schwartz, 1999; Long, Jeffcoat, & Millward, 2000; Weigle et al., 2005). However, not all studies have observed these appetite responses to high protein consumption (Blatt et al., 2011; Gosby et al., 2011; Griffioen-Roose et al., 2011).

Despite the inconsistent results reported in these studies, there is an indication that protein restriction did influence appetite in study participants. During the experiment in the *ad libitum* phase following the protein-restricted period, participants consuming a low protein diet (5%PE or 10% PE) reported greater sensations of hunger and a preference for savoury or salty flavoured foods (Gosby et al., 2011; Griffioen-Roose, Mars, et al., 2012; Martens et al., 2013, 2014) and substantially increased their intake of protein particularly from savoury flavoured meals or snacks (Gosby et al., 2011; Griffioen-Roose et al., 2012, 2014). Functional brain imaging (fMRI) revealed that the low-protein status stimulated a greater response to odour and visual savoury food cues in reward-related areas, such as the orbital frontal cortex and striatum (Griffioen-Roose et al., 2014). In contrast, high protein diets have been found to induce substantial decreases in hunger and increases in satiety (Halton & Hu, 2004; Martens & Westerterp-Plantenga, 2014).

Furthermore, following protein restriction, several studies reported that participants showed a preference for savoury or salty flavours (Gosby et al., 2011; Griffioen-Roose et al., 2012, 2014), which may be an indication of a learned appetitive behaviour.. Dietary protein is not consistently associated with a specific flavour, yet savoury or ‘umami’ flavoured foods typically contain higher levels of protein than do sweet or starchy foods (Van Dongen et al., 2012). It is proposed that humans and animals learn which foods provide an adequate protein supply by learning to associate the sensory qualities of the food with its post-ingestive consequences, and therefore learn to associate savoury or 'umami' flavours with foods that contain a high/good source of protein (Gibson & Brunstrom, 2007;

Sclafani, 1997). The dietary learning for flavours associated with protein has been demonstrated both in rats (Baker et al., 1987) and humans (Gibson et al., 1995).

In humans, individuals who are able to detect lower thresholds of monosodium glutamate (MSG) in solution demonstrate a greater liking and preference for high protein foods (Luscombe-Marsh et al., 2008, 2009). With acute protein deprivation, individuals who habitually consume a high protein diet demonstrated a preference for higher concentrations of MSG (Masic & Yeomans, 2017). These findings indicate that protein content in food is sensed and acted upon, although the physiological mechanisms are not understood, (Morrison & Laeger, 2015; Morrison et al., 2012), and flavour-nutrient learning could still contribute to such findings. Therefore, if acutely short of protein, individuals may indicate a preference for and choose to consume more savoury or salty flavoured foods.

One key consideration for experimental investigations of PLH is that across a sample population, there will be individual variations in physical characteristics and lifestyle factors that may influence the appetite responses to protein restriction. Firstly, individuals vary in their dietary protein needs, and although this represents small differences across a sample population, the PLH predicts that, under conditions of protein restriction, these differences will elicit substantial variations in food intake, particularly in energy obtained from non-protein sources. One sub-population are individuals who exercise regularly. Currently it is recommended that adults aged between 18 – 65 years consume 0.8 g protein per kg body weight per day ($g \cdot kg\ BW^{-1} \cdot day^{-1}$, Institute of Medicine, 2005; Martens & Westerterp-Plantenga, 2014). However, for individuals who engage in regular physical activity, it is argued that their protein requirements are increased due to increased amino acid oxidation during exercise and the support of muscle tissue growth, maintenance and repair

(Genton et al., 2010). For these individuals it is recommended that protein intake be increased to 1.2 – 1.7 g ·kg BW^{-1} · day^{-1} (Rodriguez et al., 2009). Although the appetitive protein mechanisms are poorly understood, it may be that a greater protein need drives the appetite for protein foods, such that is observed in young, growing animals (Jean et al., 2002; Morrison et al., 2012; White et al., 2000a).

Furthermore, recent developments within appetite research have revealed that body composition, namely fat-free body mass and resting metabolic rate, plays a primary role in directing appetite and food intake (Blundell, 2018; Hopkins et al., 2017). Both fat-free body mass (FFM) and resting metabolic rate (RMR) is found to be positively associated with energy intake, appetite sensations and meal size (Blundell, Caudwell, Gibbons, Hopkins, Naslund, et al., 2012; Blundell, Caudwell, Gibbons, Hopkins, Näslund, et al., 2012; Blundell et al., 2015; Cameron et al., 2016; Caudwell et al., 2013; Weise et al., 2014).This finding occurs across the spectrum of adiposity (whether the individual is lean, overweight or obese); however fat mass (FM) has been found to correlate negatively with EI but only in leaner individuals (Blundell et al., 2015; Cugini et al., 1998; Weise et al., 2014).

This indicates that a signal from FFM may exert a strong influence over appetite and food intake as a need to maintain lean tissue mass, whereas the signal that arises from adipose tissue is mediated by the level of adiposity and is therefore relatively weaker with greater fat mass (Blundell, 2018). If FFM and RMR drive energy needs, it may be that these factors drive protein need to ensure an adequate supply for growth, development and maintenance of lean tissue, consistent with the protein-stat model proposed by Millward (1995).

Recent studies have observed a positive association between FFM and protein intake (Cameron et al., 2016; Weise et al., 2014). This follows that individuals who present with a higher FFM, lower body fat and who participate in a high volume of physical activity (Rodriguez et al., 2009), may be more responsive to a protein restricted diet. and exhibit a greater shift in dietary preference and intake.

Alongside body composition, physical activity is seen to play an essential role in influencing mechanisms controlling appetite. Exercise, particularly of high intensity, enhances the appetitive signals controlling hunger and satiety allowing the individual to more accurately respond to these signals (Beaulieu, Hopkins, Blundell, & Finlayson, 2016). Individuals who complete an exercise programme demonstrate a marked increase in hunger but also improved satiety following a meal. These changes are reflected in alterations in appetite hormones, particularly ghrelin and GLP-1 and increased insulin and leptin sensitivity (Dyck, 2005; Martins et al., 2010, 2007; Sim et al., 2015; Yaribeygi et al., 2019). Additionally, habitually active individuals showed a greater ability to regulate food intake at lunch after receiving a high or low energy preload (Long et al., 2002; Sim et al., 2015). On the other hand, inactivity (or sedentariness) appears to weaken appetite control and allows for an imbalance between energy intake and expenditure (Stubbs et al., 2004). Taken together, the research indicates that physically active individuals may be more sensitive to changes in protein intake and exhibit a greater appetitive response when intake is insufficient; and that active individuals are hungrier yet are more sensitive to the satiating qualities of food and are able to regulate food intake to ensure a target protein intake is achieved.

Therefore, the purpose of this study was to determine: (i) the changes in appetite, food intake, and mood and food preferences at lunch in response to eating a low protein breakfast and compare these responses in active and sedentary individuals; (ii) examine how these responses may be associated with body composition, resting metabolic rate and energy expenditure.

I hypothesise that:

1. Compared to an equicaloric high-protein breakfast, a low protein breakfast will increase hunger, the desire for savoury or salty flavoured food, alter mood and increase food intake at a subsequent lunch meal in all participants.
2. Compared to Sedentary individuals, physically active individuals will show a greater response in appetite, energy intake, food choices and food flavour preferences, and mood, following a low protein breakfast.
3. Physically active individuals will increase protein intake at lunch in response to a low protein intake at breakfast, whereas Sedentary individuals will increase carbohydrate and fat intake.
4. Fat-free mass, fat mass, resting metabolic rate and energy expenditure will be associated with an increased energy and protein intake and increased appetite and desire for savoury flavoured food following a low protein meal, and these variables will be more strongly associated in the active participants.

5.2 Methods

5.2.1 Design

The study employed a randomized, single-blinded controlled design where participants consumed either a low-protein (<3 g) or high-protein (>20 g) breakfast on two separate days. Test days were separated by 7 days. A buffet meal was served approximately three hours after breakfast and participants could eat *ad libitum*. Appetite and mood scores were obtained before the breakfast and lunch meals. Food choice and preference for food flavours were assessed before the lunch to determine the effect of protein breakfast condition.

5.2.2 Participants

Twenty-five participants (8 men, 17 women), age 19-56 years participated in the study. Participants were recruited through advertisements posted on the University of Roehampton campus and through email. Potential subjects were directed to an online questionnaire which determined their eligibility to participate in the study (Qualtrics, Provo, UT). Exclusion criteria included restrained eating score > 3.6 from the Dutch Eating Behaviour Questionnaire (van Strien et al., 1986), following an energy-restricted diet, any health conditions or medication, pregnancy, food allergies or intolerances, vegetarian or veganism, dislike of foods offered in lunch buffet meal, or use of supplements, protein supplements or appetite suppressants. Qualified subjects were invited to participate in the study. Participants were unaware of the purpose of the experiment and told that the purpose of the study was to examine food preferences in individuals who participate in regular physical activity. Participants were grouped into tertiles based on total weekly levels of physical activity ($MET.mins^{-1}.week^{-1}$). The categories closely matched the low, moderate and high physical activity categories listed in the IPAQ Guidelines for

Data Processing (IPAQ Research Committee, 2005) . The low physically active group (Sedentary group) performed less than 665.8 MET.mins-1.week-1, the moderate physically active group (Moderate group) between 665.9 and 2701 MET.mins-1.week-1 and the high physically active group (Active group) performed more than 2701.1 MET.mins-1.week-1. The terms ‘Active’, ‘Moderate’ and ‘Sedentary’ were used to avoid confusion with the terms used to describe the protein condition (‘high’ vs ‘low’). Participants age (years), body mass index (BMI), body composition, metabolic rate, daily energy expenditure, physical activity levels and estimated protein requirements are listed in table 3.4 in the results section. There were 8 participants (all female) in the sedentary group, four males and five females in the moderately active group and four males and four females in the active exercise group. Protein requirements were estimated based on the activity level of each participant. Participants in the high and medium group regularly took part in several different modes of exercise, for instance individuals participated in both cardiovascular endurance activities (such as running, cycling, football) and resistance training (gym training, participating in group fitness classes etc.). Current recommendations suggest that endurance and strength athletes consume 1.2 g -1.7 g of protein per day based on training intensity, duration and exercise type (Rodriguez et al., 2009). Therefore, it was estimated that the moderately active exercise group would require 1.2 g per kg.day-1 while the active exercise group would require 1.6 g per kg.day-1. Participants were unaware of the aim of the study and were informed that the purpose of the trial was to assess appetite and food choices in Sedentary and Active individuals. The study was approved by the University of Roehampton Ethics Committee (PSYC 16 163).

5.2.3 Preliminary assessment

Participants arrived after a 10-h overnight fast and completed a physiological assessment to determine body composition and resting metabolic rate. Body composition was measured using BOD POD (Life Measurement, Inc. Concord, CA). Participants wore minimal tightly fitted clothing and a swimming cap to ensure accurate volume/mass measurements. Resting metabolic rate (RMR) (kcal.d^{-1}) was assessed and calculated indirectly using data from the BOD POD. Physical activity was assessed using the International Physical Activity Questionnaire (IPAQ) (Craig et al., 2003). Height and weight were measured, and together with fat and free-free mass measurements were used to calculate total daily energy requirements (TDDE) (kcal.day^{-1}) (Redman et al., 2014).

5.2.4 Procedure

Experimental sessions took place at the food laboratory, Whitelands College, University of Roehampton. Participants were asked to refrain from eating from 23:00 and from vigorous exercise on the evening before testing. Participants completed an appetite and mood questionnaire on arrival. Breakfast was served between 08:30 and 10:30 am, and participants were instructed to refrain from eating , however, they were permitted to drink plain water. Treatment conditions were randomised by randomly generating a number (1 or 2) using an algorithm-generated in excel and assigning number 1 to low condition and number 2 to the high condition.

Approximately three hours after breakfast, the participants arrived at the food laboratory and were instructed to complete an appetite and mood assessment. The participants were provided with a buffet lunch and instructed to consume ‘the same amount of food as they would at a normal/usual lunch meal’.

5.2.4 Test foods

Breakfast provided approximately 20% of TDEE and was comprised of a sweetened cereal (Ricicles, Kellogs), gluten-free bread (Genius Foods Ltd), low-protein milk, butter, jam and honey and a high or low protein chocolate milkshake. Low-Protein milk was prepared according to methods described elsewhere (Gibson et al., 1995). The chocolate milkshake was made by adding 16 g double cream, 10 g cocoa powder, 15 g white granulated sugar, 0.2 g xanthan gum, 0.2 g vanilla essence and either 25 g whey protein powder for the high protein version or 25 g maltodextrin for the low protein version. The total volume for the milkshake was 200 ml. Nutritional information for the milkshake is provided in table 5.1. Products were purchased at Sainsbury's™.

Table 5.1:

Nutritional information for breakfast chocolate milkshake (per portion 200 ml)

	Energy (kcal)	Carbohydrate (g)	Fat (g)	Protein (g)
Low protein	277.5	39.9	9.9	2.7 (3%PE)
High protein	280.8	17.9	11.6	23.2 (33%PE)

Values expressed as mean. Kcal: kilocalories, g: grams, %PE: percentage energy from protein

The aim of the buffet was to provide a choice from foods high in a single macronutrient, namely high carbohydrate or fat or protein (Latner & Schwartz, 1999). Lunch consisted of roasted chicken slices, meatballs, canned tuna, cheddar cheese, butter, ciabatta bread, cooked penne pasta, tomato pasta sauce, creamy pasta sauce, mayonnaise, tomato ketchup, plain salted crisps, salted peanuts, strawberry yoghurt and strawberry ice-cream. The nutritional information for the food items served at lunch is presented in table 5.2.

All lunch food items were bought in advance and stored. On the day of testing, meat, pasta, pasta sauces and the ciabatta bread rolls were heated to a temperature of 65°C and served to participants. All other lunch items were placed in large containers and placed on a table from which participants could serve themselves. Any heated food left at the end of the lunch session was discarded.

Table 5.2:
Nutritional information for food items served for lunch buffet (per 100 g and per portion)

Food item	Energy (kJ)	Energy (kcal)	Fat (g)	Saturated fat (g)	Carbohydrate (g)	Sugars (g)	Fibre (g)	Protein (g)	Salt (g)	portion (g)	protein (g)	PE%
Chicken	477.0	113.0	1.6	0.5	0.5	0.5	0.8	23.9	0.5	80.0	19.1	83%
Tuna	478.0	113.0	0.5	0.1	0.5	0.5	0.5	27.0	0.8	60.0	16.2	98%
Meatballs	1000.0	240.0	17.0	7.0	8.5	1.3	1.0	12.0	1.4	100.0	12.0	21%
Yoghurt	341.0	80.0	0.2	0.1	11.6	10.9	0.2	7.9	0.1	150.0	11.9	40%
Pasta	733.0	173.0	1.0	0.2	35.0	1.0	2.0	5.0	0.0	200.0	10.2	13%
Bread	1164.0	275.0	4.1	0.7	47.8	1.9	3.0	10.3	0.8	89.0	9.3	15%
Peanuts	2569.0	620.0	50.6	6.3	12.5	6.0	6.2	25.6	0.7	30.0	7.7	17%
Cheese	1619.0	370.0	32.0	20.8	0.5	0.5	0.5	25.5	1.8	30.0	7.6	25%
Ice-cream	684.0	163.0	5.6	4.6	24.4	22.6	1.0	3.2	0.1	56.0	1.8	8%
Crisps	2242.0	537.0	32.2	2.8	55.4	0.5	2.3	5.3	1.2	30.0	1.6	4%
Tomato sauce	216.0	51.0	1.0	0.1	8.1	7.0	1.6	1.7	0.7			
Ketchup	435.0	102.0	0.1	0.0	23.2	22.8	0.0	1.2	1.8			
Creamy sauce	422.0	102.0	8.1	3.2	5.8	2.3	0.2	1.1	0.8			
Mayonnaise	2749.0	668.0	73.2	6.1	1.4	1.2	0.0	1.0	1.5			

kJ = kilojoules; kcal = kilocalories; g = grams; PE% = percentage energy from protein

5.2.5 Measures

5.2.5.1 Appetite assessment and food flavour preferences

For the assessment of appetite, participants were instructed to assess their level of hunger, fullness, feelings of satisfaction, prospective food intake, and desire to consume something sweet, salty, fatty and savoury using visual analogue scales (VAS). The questionnaire was presented to participants using an online questionnaire (Qualtrics, Provo, UT) on an iPad (Apple Inc.). Each VAS was anchored with ‘Not at all’ on the left side and ‘As much as I can imagine’ on the right (Booth, 2009).

5.2.5.2 Mood assessment

Dietary protein intake may influence mood, as a high protein meal has been shown to increase positive affect (Firk & Markus, 2009; Gibson et al., 1999, 2014), therefore the participants completed an the assessment of mood, the Positive and Negative Affective Scale (PANAS) (Watson et al., 1988) was presented in the online format (as described above). The scale showed good internal consistency (Cronbach’s alpha values were as follows: positive affect scale range 0.88 – 0.93 and negative affect scale 0.86 – 0.93

5.2.6 Data analysis

Tests for normality and equal variance were conducted on all dependent and independent variables. A one-way analysis of variance (ANOVA) was used to determine whether there were significant differences in age, body mass index, body composition, resting metabolic rate and total energy expenditure between sexes and between activity groups. A p-value of < 0.05 was considered statistically significant for all analyses.

Food intake at lunch was expressed as energy (kcal) or weight (g). Physical activity was expressed as Metabolic Equivalent Units (MET) per minute per week (MET-mins.week^{-}1, Craig et al., 2003). Macronutrient intake was expressed in g and as a percentage of energy intake (%PE). Appetite and flavour preference scores were expressed as a score from 0 to 100. To determine the effect of protein condition on appetite and mood, percentage change scores were calculated from baseline (pre-breakfast) values using the following equation.

$$\textbf{Change in appetite or mood} = \left(\frac{\textbf{pre lunch score} - \textbf{pre breakfast score}}{\textbf{pre breakfast score}}\right) * \textbf{100}$$

The distribution of the change in appetite scores for each variable was analysed using a simple box plot. Outliers that were greater than 1.5 times the length of the box (interquartile range) were removed from the data set. The details of outliers and detailed in Table G1 in the Appendix G (Field, 2013).

To compare the choice of protein foods, the two foods items containing the highest and lowest amount of protein per serving were chosen to represent the choice for high or low protein foods. Chicken and tuna were chosen for high protein food, and ice-cream and crisps were chosen for low protein food category (details provided in Table 5.2). The proportion of high/low protein foods chosen (in grams) was calculated as follows:

$$\textbf{Proportion of high/low protein food} = \frac{\boldsymbol{intake\ of\ high\ or\ low\ protein\ food\ (g)}}{\boldsymbol{Total\ food\ intake\ (g)}}$$

Two-way repeated measures mixed ANOVA was conducted to determine differences in energy intake, macronutrient, appetite, mood and high or low protein food choice between breakfast protein levels and activity levels, with the main effect of breakfast protein condition and the second main effect of the activity group. Post-hoc analyses were performed to determine the simple effect of activity group (Active vs Moderate vs Sedentary), condition (High vs Low) or interaction between activity group and condition using pairwise comparisons. Associations between body composition, energy expenditure, energy intake and appetite scores were performed using Pearson's product-moment coefficient (r). Analyses were conducted across all participants and within activity groups.

5.3 Results

5.3.1 Participant characteristics

The descriptive statistics (mean ± 1 SD) for age, body composition, physical activity and total daily protein requirements are listed for each of the three activity groups in Table 5.3 below. The activity groups did not differ by age (Active vs Sedentary mean ± 1SEM difference: 8.0 ± 4.1 years, $p = 0.21$; Moderate vs Sedentary 5.4 ± 3.7 years, $p = 0.45$). There were no differences in BMI (Active vs Moderate -1.7 ± 0.9 $kg.m^{-2}$, $p = 0.50$; Active vs Sedentary -1.2 ± 1.7 $kg.m^{-2}$, $p = 0.71$. The Active group were leaner (lower percentage fat mass (FM)) and had more fat free mass (FFM)(kg) compared to the Sedentary (p =0.008 FM; $p = 0.033$ FFM). The Sedentary group had significantly lower RMR and TDEE compared to both the Moderate and Active group; RMR Sedentary vs Moderate -225.6 ± 103.9, $p = 0.041$, Sedentary vs Active -251.6 ± 106.9, $p = 0.028$; TDEE Sedentary vs Moderate -353.3 ± 148.7, $p = 0.027$, Sedentary vs Active -474.2 ± 153.1, $p = 0.005$. Levels of physical activity ($MET\text{-}mins.week^{-1}$) were significantly different between groups: Active vs Sedentary: 3729.64 ± 328.36 $MET\text{-}minss.week^{-1}$, $p < 0.001$; Moderate vs Sedentary: 1631.14 ± 319.11 $MET\text{-}minsMET\text{-}mins.week^{-1}$, $p <0.001$; Active vs Moderate: 2098.49 ± 319.10 $MET\text{-}minsMET\text{-}mins.week^{-1}$, $p < 0.001$. Estimated protein requirements (g or percentage of TDEE) were lowest in the Sedentary group ($p < 0.05$), highest in the Active group ($p<0.05$). For estimated protein requirements in grams, Active vs Sedentary: 54.32 ± 5.83 $g.kg^{-1}.day^{-1}$, $p < 0.001$; Moderate vs Sedentary: 32.74 ± 5.67 $g.kg^{-1}.day^{-1}$, $p <0.001$; Active vs Moderate: 21.59 ± 5.67 $g.kg^{-1}.day^{-1}$, $p < 0.001$. Please refer to table 5.3.

Table 5.3:

Age (years), body mass index (BMI), percentage body fat, resting metabolic rate (RMR), total daily energy expenditure (TDEE) and physical activity (IPAQ) and estimated protein requirements for Active, Moderate and Sedentary groups

	Sedentary (n=8)	Moderate (n=9)	Active (n=8)
Age	21.4 ± 2.7[a]	26.8 ± 10.2	29.4 ± 11.6
	(19-25)	(19-52)	(21-56)
Sex	0 M, 8 F	4 M, 5 F	4 M, 4 F
BMI ($kg.m^{-2}$)	23.9 ± 4.5	24.4 ± 2.4	22.7 ± 1.5
	(18.6-32)	(20.3-27.3)	(19.6-24.6)
Fat mass (%)	34.7 ± 8.3	28.2 ± 8.5	20.1 ± 9.6[b]
	(24.1-49.3)	(15.8-41)	(7.1-34.9)
Fat-free mass (%)	65.3 ± 8.3	71.8 ± 8.5	79.9 ± 9.6[c,d]
	(50.7-75.9)	(59-84.2)	(65.1-92.9)
Fat-free mass (kg)	41.2 ± 6.7	50.4 ± 9.1	52.5 ± 8.8
	(31.3-53.2)	(36.4-66.6)	(40.8-67)
RMR ($kcal.day^{-1}$)	1154.6 ± 188.6[e, f]	1380.2 ± 231	1406.3 ± 217
	(873-1496)	(1023-1817)	(1140-1790)
TDEE ($kcal.day^{-1}$)	2127.6 ± 241.3[f, g]	2480.9 ± 331	2601.8 ± 333.2
	(1793.3-2538.5)	(1955.8-3009.5)	(2138.3-3112.5)
IPAQ (MET-mins.$week^{-1}$)	443.8 ± 152.2[f,g]	2074.9 ± 736.8[h]	4173.4 ± 843.7
	(150-600)	(700-2630)	(2855-5540)
Estimated protein requirements ($g \cdot kg^{-1} \cdot day^{-1}$)	51.4 ± 10.4[f,g]	84.1 ± 11.8[h]	105.7 ± 12.6
	(38-67.1)	(68.6-109.4)	(91.7-131.9)
Estimated protein requirements (% of TDEE)	9.7 ± 1.6[f,g]	13.6 ± 1.4[h]	16.3 ± 1.5
	(7.9-12.5)	(11.8-16.3)	(14.6-18.9)

Means ± SD and (ranges); M: male, F:female; [a] p = 0.092 Sedentary tended to be younger than Active;[b] p < 0.01 Active less than Sedentary, [c] p<0.01 Active more than Sedentary; [d] p = 0.071 Tendency for Active to be more than Moderate; [e] p=0.06 Tendency for Sedentary to be lower than Moderate; [f] p<0.05 Sedentary lower than Active; [g] p<0.05 Sedentary lower than Moderate; [h]p<0.05 Moderate less than Active

5.3.2 Energy and macronutrient intake

Energy, macronutrient intake and food choices at lunch are listed in Table 5.4. Overall, collapsing across activity groups, there were no differences between the high and low protein conditions or between activity groups: simple effect for group, F (2,22) = 2.51, p = 0.10, partial η^2 = 0.19. However, the Sedentary consumed less energy compared to the Moderate group; mean difference ± 1SEM [95%CI] -477.92 ± 220.26 kcal [-934.70 to -21.14], p = 0.041.

The Sedentary also consumed significantly less protein (in g) than the Moderate Group, -31.33 ± 11.70 g [-55.59 to -7.08], p = 0.014, and the Active group, -22.13 ± 12.0 g [-47.09 to 2.82], p = 0.079; simple main effect for group, F (2, 22) = 3.73, p = 0.04, partial η^2 = 0.25. Please refer to figure 5.1

When protein intake was expressed as a percentage of overall energy intake, the patterns of protein intake differed across activity groups and between low and high protein conditions (significant two-way interaction; group x condition, F (2, 22) = 5.10, p = 0.015, partial η^2 = 0.32) Please refer to figure 5.2. The Active tended to consume similar proportions of protein (%PE) between conditions 0.72 ± 1.58 %PE [-2.46 to 3.91], p = 0.608), whereas the Moderate group tended to decrease percentage protein intake in the low protein condition (-3.45 ± 1.49 %PE [-7.64 to 0.75], p = 0.095) and the Sedentary group increased intake (3.45 ± 1.58 %PE [0.33 to 6.56], p = 0.035) in the low protein condition. Multiple comparisons revealed that in the high-protein condition, Sedentary consumed less than Moderate, -7.28 ± 2.78 %PE [-13.03 to -1.53], p = 0.016; and Active, -5.40 ± 2.86 %PE [-11.31 to 0.53], p = 0.072, while no differences between groups were observed for the low-protein condition.

The proportions of high protein foods chosen at lunch differed following consumption of a high or low breakfast and between activity groups, two-way interaction: Proportion High Protein food x Activity group; F (1,22) = 6.22, p = 0.007, partial $\eta^2 = 0.361$. There was no main effect for breakfast protein condition or activity group, however further analyses revealed that Sedentary chose a smaller proportion of high protein foods following a high protein breakfast compared to the Moderate and Active group, Sedentary vs Moderate mean difference ± 1SEM [95% CI]; -0.16 ± 0.05 [=0.26 to -0.05], p = 0.005; Sedentary vs Active: -0.11 ± 0.052 [-0.22 to 0.00], p = 0.051. Within-participant analyses revealed that the Sedentary increased the proportion of high protein foods following the low protein breakfast, high vs low: -0.067 ± 0.023 [-0.12 to -0.02], p = 0.007. The Moderate group tended to decrease the proportion of high protein foods following a low protein breakfast, 0.042 ± 0.022 [-0.003 to 0.087], p = 0.064.

The choice of low protein food did not differ in response to the breakfast protein conditions, nor between activity groups, however pair-wise comparisons revealed that the Sedentary choose less low protein foods following the low protein breakfast, 0.055 ± 0.024 [0.004 to 0.11], p = 0.035.

Table 5.4

Energy and macronutrient lunch intakes for Active, Moderate and Sedentary groups for high-protein and low-protein conditions.

Group:	ALL		Active (n = 8)		Moderate (n=9)		Sedentary (n=8)	
Protein condition	High	Low	High	Low	High	Low	High	Low
Energy (kcal)	967.3 ± 103.2	972.6 ± 94.9	1089.4 ± 204.7	1000.1 ± 167.9	1112.8 ± 194.3	1206.5 ± 182.6	681.5 ± 80.6[ab]	682.0 ± 66.5[ac]
CHO (g)	38.1 ± 4.9	38.6 ± 4.8	39.8 ± 9.1	38.9 ± 9.4	44.8 ± 10.6	48.7 ± 8.5	29.0 ± 3.7	27.0 ± 5.4
Fat (g)	101.6 ± 9.8	102.8 ± 9.8	118.8 ± 19.9	108.9 ± 16.2	110.9 ± 17.3	123.9 ± 20.5	73.9 ± 9.4	72.9 ± 4.6
Protein (g)	50.8 ± 5.9	50.4 ± 5.4	57.9 ± 10.4	50.99 ± 7.6	64.5 ± 10.8	62.8 ± 11.3	28.8 ± 3.2[a]	35.8 ± 5.8[a]
%Protein	21.3 ± 6.3	21.0 ± 6.1	22.4 ± 8.0	21.7 ± 9.6	24.3 ± 5.5	20.3 ± 4.5[*]	17.0 ± 2.1[d]	20.4 ± 3.9[*]
HP intake ratio	0.02 ± 0.06	0.02 ± 0.05	0.12 ± 0.04	0.11 ± 0.04	0.17 ± 0.03	0.13 ± 0.04[†]	0.01 ± 0.04[a]	0.08 ± 0.04[*]
LP intake ratio	0.02 ± 0.04	0.01 ± 0.04	0.07 ± 0.04	0.07 ± 0.03	0.06 ± 0.03	0.06 ± 0.02	0.12 ± 0.04	0.07 ± 0.03[**]

Mean ± standard error; Between-group differences: [a] $p<0.05$ Sedentary consumed less compared to Moderate; [b] $p<0.1$ Sedentary tended to consume less compared to Moderate in high condition; [c] $p=0.022$ Sedentary consumed less than Moderate in the low condition; [d] $p=0.016$ Sedentary consumed less than Moderate in the high condition. [e] Sedentary consumed less than Active, $p = 0.051$. Within-group protein condition differences: [*] $p<0.05$ Sedentary consumed more % protein and chose more high protein foods in low condition; [*] $p<0.05$ Moderate consumed less % protein in the low condition. [†] $p = 0.064$ Moderate tended to chose less in low protein condition; [**] $p = 0.035$ Sedentary chose less low protein foods in low protein condition

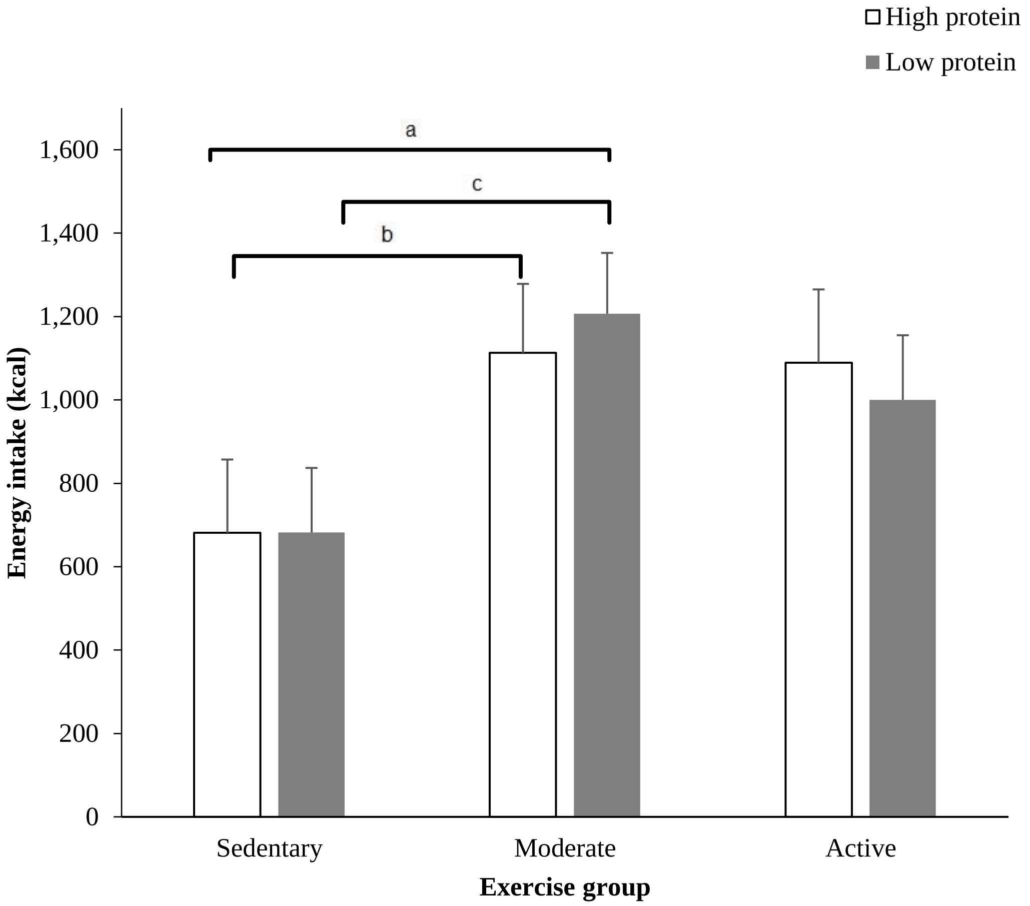


Figure 5.1: Comparison of energy intake consumed at a buffet lunch between Sedentary, Moderate and Active individuals. Mean ±1SEM. [a]Sedentary consumed less energy compared to Moderate, $p<0.05$; [b] $p<0.1$ Sedentary tended to consume less energy compared to Moderate in high condition; [c] $p=0.022$ Sedentary consumed less than Moderate in the low condition;

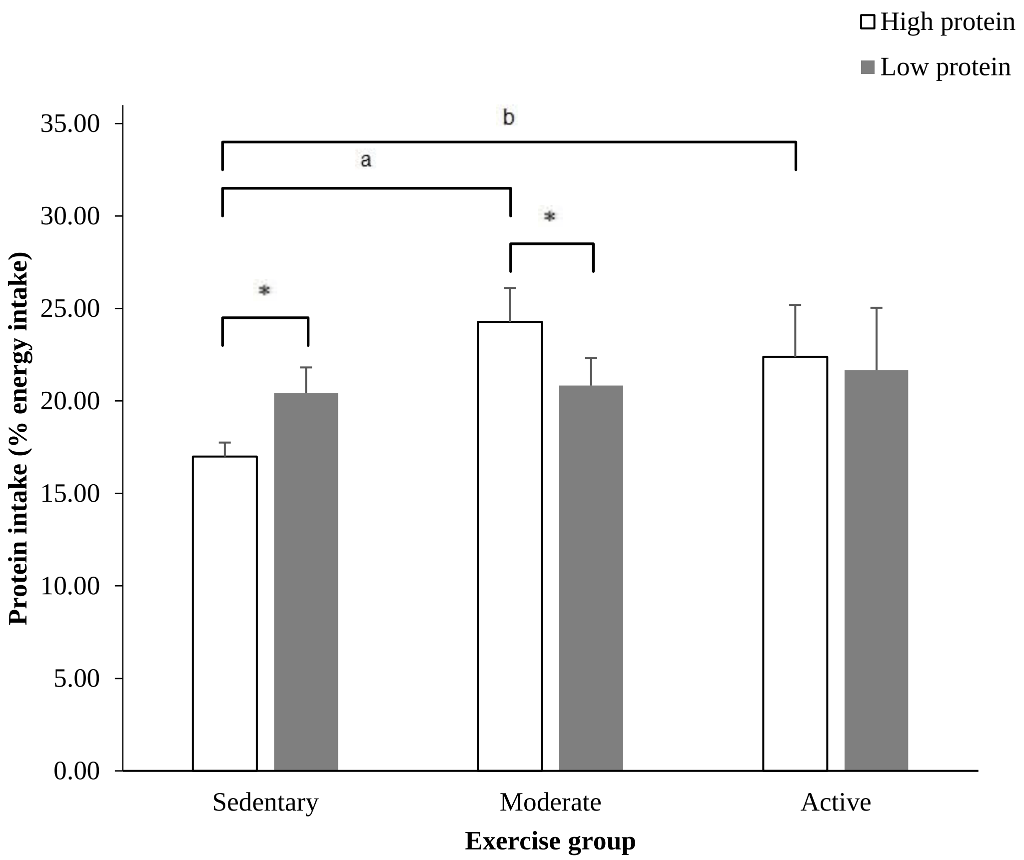


Figure 5.2: Comparison of percentage energy as protein intake (%PE) consumed at a buffet lunch between Sedentary, Moderate and Active individuals. Mean ±1SEM [*] Sedentary increased protein in low condition, p=0.040 ; [*] Moderate decreased protein in low condition, p= 0.030 , [a]Moderate consumed more protein compared to Sedentary in high condition, p= 0.016 ; [b]Active tended to consume more protein compared to Sedentary in high condition, p= 0.072

5.3.3 Change in appetite (hunger, fullness, satisfaction, estimated food intake) and flavour preferences (desire for sweet, salty, savoury and fatty flavours).

There were no significant changes in hunger, fullness, satisfaction or desire for salty and desire for fatty foods in response to the protein conditions. There was a main effect of the protein condition on the estimated food intake and desire for sweet and savoury foods. Please refer to figure 5.3 A - D and figure 5.4 E - H

Overall following the high protein breakfast, participants reported smaller estimations of prospective food intake, meaning that in comparison to the low protein meal they estimated needing to eat less food at lunch; High -33.07 ± 6.09 vs Low -0.27 ± 0.07, mean difference ± SE [95%CI]: -33.16 ± 5.91, [-45.45 to -20.88] p <0.001, partial η^2 =0.60. Pair-wise comparisons revealed all activity groups estimated eating less food in the high compared to low protein conditions; Sedentary mean difference ± SE [95% CI] -30.90 ± 10.77 [-53.37 to -8.44], p = 0.010, partial η^2 0.30; Moderate -40.88 ± 10.04 [-61.89 to -19.87], p = 0.001, partial η^2 = 0.47; Active -26.61 ± 10.73 [-49.07 to -4.15], p = 0.023. Please refer to figure 5.3 graph D.

The low protein condition caused an increase in the desire to eat sweet foods across all participants, High protein -6.21 ± 3.71 vs Low protein 26.84 ± 11.03, mean difference -33.05 ± 11.08 [-56.99 to -9.11], p = 0.011, partial η^2 =0.41. There were no differences between groups; however there were significant differences between activity groups in response to the low protein breakfast, F (2.22) = 3.50, p = 0.048, partial η^2 = 0.24. The pair-wise comparison revealed that the Moderate reported an increased desire for sweet compared to the Active group, 51.28 ± 19.79 [10.25 to 92.32], p = 0.017. Please refer to figure 5.4 graph E.

The change in desire to eat savoury foods differed between the activity groups in response to consuming a high or low protein breakfast, two-way interaction, F (2,17) = 5.17, $p = 0.018$, partial $\eta^2 = 0.38$. In the Sedentary and Active group, the low protein caused reduced desire to eat savoury foods, however these differences were not significant; Sedentary: mean difference ± SE [95%CI], 17.78 ± 15.35 [-14.61 to 50.17], $p = 0.26$; Active group: 18.97 ± 12.98 [-8.41 to 46.34], $p = 0.16$. The Moderate group increased their desire to eat savoury foods in the low protein condition -31.91 ± 12.14 [6.30 to 57.52], $p = 0.018$, partial $\eta^2 = 0.29$. In the high protein condition, the Sedentary and Active groups showed a reduced desired to eat savoury foods compared to Moderate group, Sedentary vs Moderate: 40.77 ± 14.71 [9.73 to 71.80], $p = 0.013$; Active vs Moderate: 29.42 ± 13.35 [1.25 to 57.60], $p = 0.042$. Please refer to figure 5.4 graph G.

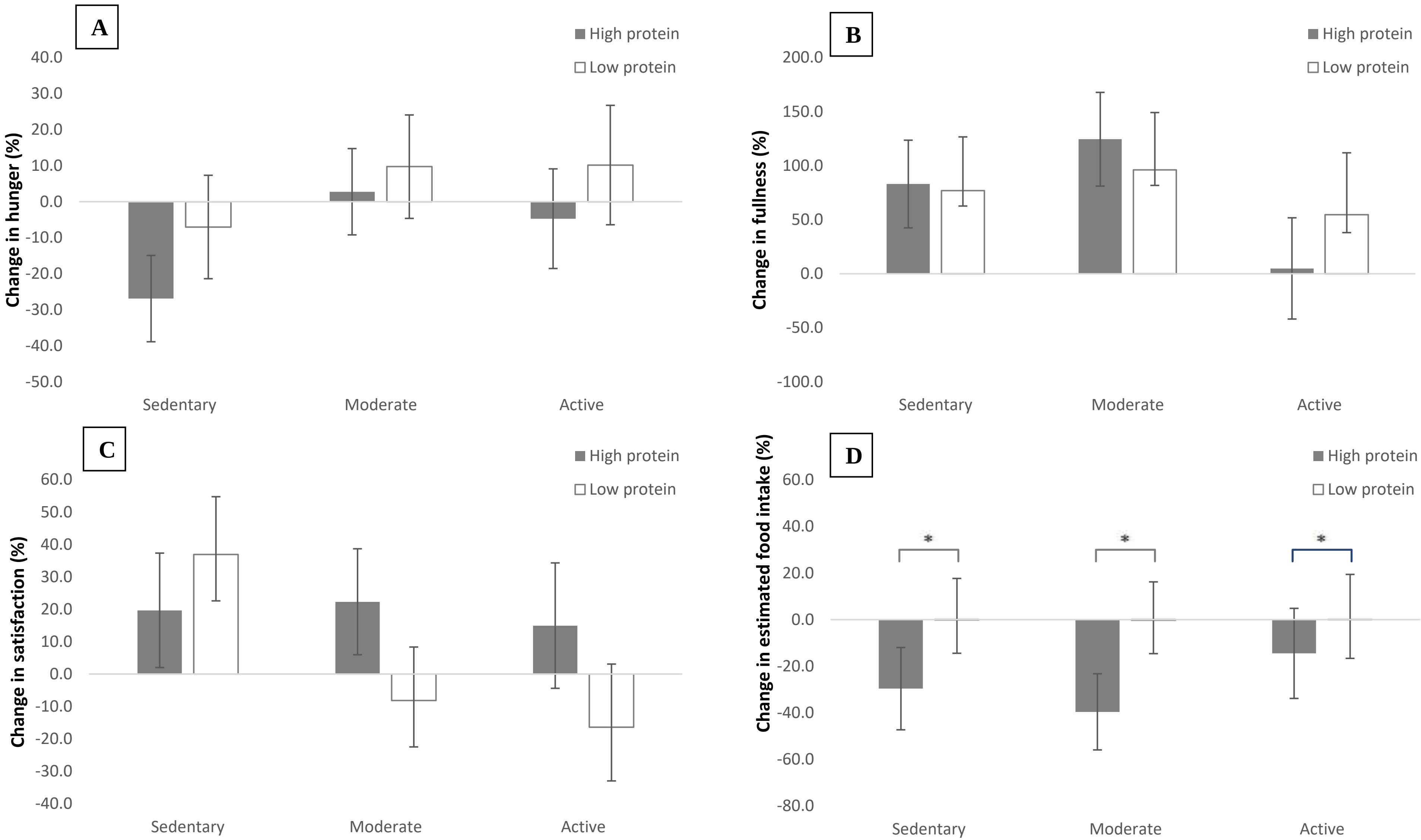


Figure 5.3: Change in appetite (pre breakfast to pre lunch) in response to consuming a high or low protein breakfast in Sedentary, Moderate and Active participants. Mean ± 1SEM. A: change in hunger; B: change in fullness; C: change in satisfaction; D: change in estimated food intake

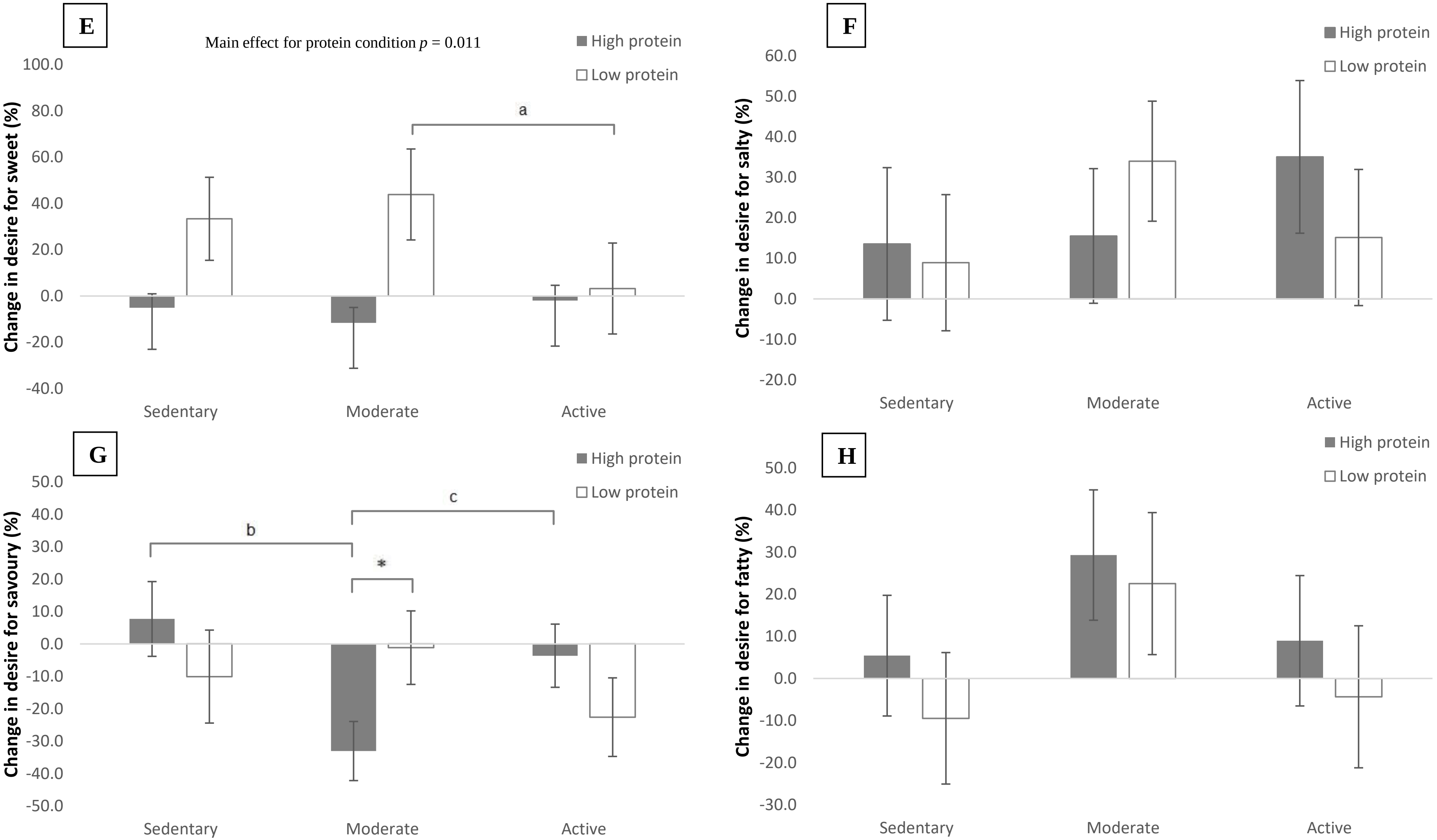


Figure 5.4: Change in food flavour preferences (pre breakfast to pre lunch) in response to consuming a high or low protein breakfast in Sedentary, Moderate and Active participants. Mean ± 1SEM. E: change in desire for sweet; F: change in desire for salty; G: change in desire for savoury; H: change in desire for fatty

5.3.4 Positive and Negative Affect (PANAS)

There were no significant changes in positive or negative affect following either the high or low protein breakfast, nor were there significant differences between the activity groups. The mean ± SEM of the changes in positive and negative affect scores from baseline (pre-breakfast) are presented in table 5.5 below:

Table 5.5

Change in positive and negative affect (PANAS) from baseline in response to consuming a high or low protein breakfast between Sedentary, Moderate and Active groups.

	Positive affect		Negative affect	
	High protein	Low protein	High protein	Low protein
Sedentary	0.5 ± 4.07	2.17 ± 5.81	6.05 ± 4.25	-15.46 ± 9.27
Moderate	5.35 ± 3.59	-5.72 ± 5.13	1.84 ± 3.17	-4.5 ± 6.91
Active	-9.3 ± 4.4	-3.72 ± 6.28	-10.06 ± 4.25	-0.32 ± 9.27

Mean ± 1SEM

5.3.5 Associations between energy and macronutrient intake, body composition and energy expenditure between activity groups for high and low protein conditions

To test the hypothesis that moderately active (Moderate) and active individuals (Active) would be more sensitive to energy and protein requirements and therefore better able to regulate food intake to meet those needs, associations between body composition and energy expenditure, and energy and macronutrient intake were examined in response to high or low protein intake. Pearson's correlation coefficients are reported in Table 5.6. For all participants, greater resting metabolic rate (RMR) and fat-free mass (FFM) were associated with higher energy intake, carbohydrate, fat, and protein intake (g) ($p < 0.05$), while a higher fat mass was associated with a lower protein intake ($p<0.05$).

Between each activity group, the correlation coefficients were strong and significant in the Active group, yet weaker and non-significant in the Moderate and Sedentary group; however no statistical analyses were performed to test this observed difference in coefficient strength. In the Active group, RMR, TDEE, FFM tended to be associated with higher energy intake (kcal) and fat intake (g) (r (8) = 0.63 to 0.67, $p < 0.10$) and were associated with higher protein intake (g) (r (8) = 0.75 to 0.78, $p < 0.05$). No significant correlations were observed for the Moderate and Sedentary groups; however in the Moderate group, a higher fat mass and was associated with a lower percentage carbohydrate and fat intake, r (8) = -0.69, $p < 0.05$, yet associated with a higher percentage protein intake in the low protein condition, r (8) = 0.72, $p < 0.05$.

Table 5.6:

Pearson's correlations between measures of body composition or metabolism and food intake (energy and macronutrients) for each activity group and high- or low-protein condition

	ALL (n=25)		Active (n=8)		Moderate (n=9)		Sedentary (n=8)	
	H	L	H	L	H	L	H	L
				Energy intake (kcal)				
RMR	0.53**	0.59**	0.63+	0.67+	0.41	0.52	0.01	0.12
FFM	0.56**	0.62**	0.64+	0.68+	0.44	0.57	0.06	0.19
FM	-0.37+	-0.37+	-0.37	-0.35	-0.37	-0.51	-0.24	-0.29
				Carbohydrate intake (g)				
RMR	0.49*	0.51**	0.58	0.54	0.32	0.37	0.02	0.27
FFM	0.52**	0.54**	0.60	0.56	0.35	0.42	0.05	0.22
FM	-0.39+	-0.34+	-0.44	-0.40	-0.31	-0.53	-0.12	0.36
				Fat intake (g)				
RMR	0.45*	0.53**	0.57	0.63+	0.38	0.55	0.02	0.27
FFM	0.48*	0.55**	0.58	0.63+	0.40	0.56	0.05	0.22
FM	-0.29	-0.26	-0.30	-0.23	-0.31	-0.16	-0.12	0.36
				Protein intake (g)				
RMR	0.62**	0.55**	0.71*	0.76*	0.49	0.43	-0.005	0.25
FFM	0.66**	0.60**	0.73*	0.78*	0.54	0.50	0.01	0.33
FM	-0.41*	-0.45*	-0.45	-0.46	-0.47	-0.74*	-0.091	-0.23
				Percentage protein (%)				
RMR	-0.35+	-0.24	-0.30	-0.23	-0.15	-0.07	0.25	-0.57
FFM	-0.35+	-0.26	-0.27	-0.23	-0.16	-0.15	0.33	-0.60
FM	0.11	0.23	-0.11	0.15	0.11	-0.69*	-0.27	-0.10

***p<0.01, p<0.05, +p<0.10; RMR: resting metabolic rate (kcal.day1), TDEE: total daily energy expenditure (kcal.day-)1; FFM: fat-free mass (kg), FM: fat mass (kg)*

5.3.6 Associations between body composition, metabolic rate and appetite ratings, following a high or low protein breakfast.

Across the sample population, there were no associations between body composition, metabolic rate and appetite ratings in response to the high or low protein condition. There were a few differences observed between activity groups. In the Moderate group, increases in feelings of fullness were negatively associated with FFM, $r(9) = -0.66$, $p = 0.05$ and RMR, $r(9) = -0.66$, $p = 0.055$. Similarly, in Active group increases estimated food intake were negatively associated with FFM, $r(8) = -0.80$, $p = 0.016$ and RMR, $r(8) = -0.81$, $p = 0.015$. However, no associations between FFM, RMR and appetite ratings were found in the Sedentary.

Following the low protein breakfast, in the Sedentary increase in fullness was associated with higher FFM, $r(8) = 0.73$, $p = 0.039$ and RMR, $r(8) = 0.71$, $p = 0.049$ increases in the desire for salty foods was associated with a higher FFM, $r(8) = 0.79$, $p = 0.019$ and RMR, $r(8) = 0.078$, $p = 0.023$. No associations were observed in the Moderate and Active groups.

5.4 Discussion

The objective of this study was to investigate the acute impact of low- versus high-protein breakfasts on appetite, food intake, food choices and mood, and to determine whether responses differed between individuals grouped by levels of physical activity, as a proxy for variation in protein need. Overall the results of the study did not confirm the study hypothesis: the active and moderately active groups did not demonstrate greater changes in appetite, food intake, food choices or mood following a low protein breakfast in comparison with the sedentary group. However, body composition, metabolic rate and energy and macronutrient intake was strongly associated with energy, carbohydrate and protein intake, indicating that body composition may play a role in directing food intake.

5.4.1 The response to mild protein restriction in Active, Moderately Active and Sedentary groups

In this investigation, I hypothesised that physically active individuals with greater dietary protein requirements would be more responsive to a low protein meal than those who are sedentary. I estimated that the active needed to consume an additional 6.6% energy from protein or 50 g of protein per day compared to the sedentary, to maintain nitrogen balance, representing a substantially greater protein need. Therefore, it was expected that following a low protein meal, the active and moderate would exhibit heightened appetite responses, such as increased feelings of hunger and the preference for savoury or salty foods and increase protein intake by choosing foods containing more protein. However, the active did not increase their protein intake nor did they demonstrate greater changes in appetite, mood or preferences for a specific food flavour in comparison to the sedentary. The moderate group decreased their protein intake following a low protein breakfast and reported a heightened appetite for sweet foods. Although the behaviours observed in the moderate group were contrary to the study hypothesis, Griffioen-Roose et al. (2012) reported an increase in preference for sweet foods following the consumption of a low protein diet (5%PE) in the first day of the dietary intervention. It may be that the preference for sweet foods reflects the initial appetite response to low protein intake, i.e. awareness of reduced satiety but not of a clear protein deficit.

By comparison, the sedentary increased protein intake following the low protein breakfast and chose a greater proportion of high protein foods. Furthermore, body composition and resting metabolic rate predicted a greater desire for salty foods in the low protein condition for this group. These findings indicate that the sedentary, rather than the active and moderate groups, were more responsive to mild protein restriction. However, it is important to note that the participants in the sedentary group were young (mean age 21.4 years) and female. By comparison, the moderate and active groups were older (Moderate mean age 26.8 years and active mean age 29.4 years) and included both men and women. Both sex and age may have influenced the response to protein restriction. Men generally have a higher resting metabolic rate and greater energy needs (Klausen et al., 1997), and this may account for the differences in energy intake and protein intake observed between the activity groups. Furthermore, men and women may differ in appetite responses, where men appear to be hungrier before and less full after consuming a meal (Gregersen et al., 2011),

Similarly, age may influence responses to protein restriction. Animal experimental studies have reported that younger animals exhibit a drive to consume higher protein foods compared to older animals, presumably because they have an increased protein need for growth and development (Jean et al., 2002; Morrison et al., 2012; White et al., 2000a). It may be that in humans, younger individuals exhibit an increased drive to eat protein in comparison to the older; however further studies are needed to determine whether age and sex influence the acute response to protein restriction.

5.4.2 The response to mild protein restriction across all participants

Overall, the main effect of protein condition did not result in a significant change in appetite and food intake. Energy and macronutrient intake and changes in appetite and food flavour preferences were similar in both protein conditions. However, the high protein breakfast did elicit a decrease in prospective food intake in comparison to the low protein condition. This finding is supported by other studies demonstrating that a single high protein meal or drink suppresses appetite and food intake (Leidy et al., 2015; Soenen & Westerterp-Plantenga, 2008; Westerterp-Plantenga et al., 2012). This indicates that despite differences in protein need between activity groups, a meal providing 20 g of protein influenced appetite and reduced prospective food intake in all participants.

It was expected that a low protein breakfast would cause an increased appetite and change energy and macronutrient intake in all study participants. The results of the study indicate that a single low protein meal may not have elicited a substantial change in appetite and food intake. Perhaps a greater deficit in protein intake, such that would occur following the intake of several low protein meals or consuming a low protein diet, is required to permit and assess learned appetitive behaviour. Indeed, many of the experimental studies investigating responses to a low protein diet took place over a minimum of four days and up to two weeks (Gosby et al., 2011; Griffioen-Roose et al., 2012, 2014; Martens et al., 2013, 2014).

Comparatively fewer studies have investigated acute appetite responses to a low protein meal. Blatt, Roe & Rolls (2011) reported no changes in appetite and food intake at dinner after consuming lunch meals that varied in protein content (from 10 to 30 % PE) which suggests that the lower protein meal (10 %PE) had no influence on appetite and food intake in this study. Similarly, Griffioen-Roose et al. (2011) reported that consuming a low (~7% PE) or high protein (~25% PE) preload did not influence appetite or energy intake at a subsequent buffet meal. Similarly, Masic et al., (2017) observed an increase in liking for all food flavours and an increased desire to eat savoury foods following a low-protein breakfast, but not a specific liking for umami or 'meaty' flavours. However, Gibson, Wainwright & Booth (1995) demonstrated that after four days of consuming a low protein breakfast, participants developed a preference for dessert flavours that were paired with high protein, which suggests that individuals are responsive to low protein meals, given the opportunity for differential flavour-nutrient learning and its expression, which other experimental designs did not allow (Gosby et al., 2011; Griffioen-Roose et al., 2012, 2014;. Martens et al., 2014).

In animals, experimental studies have demonstrated that protein restriction does cause an increase in energy intake and the selection for high protein foods (Huang et al., 2013; Pezeshkiet al., 2016; Solon-Biet et al., 2014; Sørensen, Mayntz, Raubenheimer, & Simpson, 2008; White 2000). Rats are shown rapidly to detect and avoid diets that are imbalanced for amino acid content, and these behaviours are observed within 20 minutes of food ingestion (Gietzen & Aja, 2012; Gietzen & Rogers, 2006; Hao et al., 2010). This speed of the observed responses indicates that diets that contain a poor quality and quantity of protein are sensed and acted upon; however the response is dependent on the degree of protein restriction and the physiological state of the animal (Berthoud, Münzberg, Richards, & Morrison, 2012; Morrison & Laeger, 2015). Given that appetite changes that occur with protein restriction were not observed across the group, this suggests that a greater degree of protein restriction should be applied in subsequent studies, although extrapolating this aspect from rodent studies is not straightforward.

5.4.3 Comparison of food intake and appetite between activity groups

The study observed associations between body composition, metabolism, and energy and macronutrient intake for all participants, specifically that energy, carbohydrate, protein and fat intake were positively associated with fat free mass and resting metabolic rate, while fat mass was negatively associated with protein intake and tended to be negatively associated with energy and carbohydrate intake. These findings are supported by the recent emerging theories of appetite control that propose that body composition, metabolism and physical activity play a primary role in directing appetite and food intake (Beaulieu, Hopkins, Blundell, & Finlayson, 2018; Blundell, 2018). It is proposed that tonic signals that arise from fat-free mass reflect the energetic demands of lean tissue and exert an excitatory signal to drive food intake. While the signals that arise from fat mass exerts an inhibitory effect on appetite (Blundell, 2018). The effect of fat-free mass on energy intake is mediated by resting metabolic rate (Hopkins et al., 2016) and 24-hour energy expenditure (Piaggi et al., 2015). These associations have been confirmed across the spectrum of lean, overweight and individuals with obesity (Blundell, Caudwell, Gibbons, Hopkins, Naslund, et al., 2012; Blundell et al., 2015; Cugini et al., 1999; Hopkins et al., 2016; McNeil et al., 2017; Piaggi et al., 2015).

When study participants were subdivided into activity groups, these associations remained in the active. In the active group, fat-free body mass and resting metabolic rate was positively associated with protein intake in both test conditions and tended to be associated with energy intake and fat intake. Fat mass did not predict energy or macronutrient intake. However, these results are interpreted with caution as the sample size was small. It may suggest that active individuals regulate energy and macronutrient intake more accurately than moderately active and sedentary individuals. More recently it has been reported that the level of physical activity moderates the relationship between body composition and energy intake. Beaulieu et al. (2018) reported that the strongest associations between meal size and fat mass were observed for individuals who spent the most time participating in moderate to vigorous exercise activities, while weaker, non-significant associations were found for the moderate and low activity groups.

Furthermore, following a high energy preload, moderately active individuals reduced food intake at a subsequent buffet meal, while this effect was not observed for the low activity group (Beaulieu, Hopkins, Long, Blundell, & Finlayson, 2017). This indicates that the level of physical activity (i.e. how active an individual is on a weekly basis) may influence the strength of episodic signals controlling appetite (Beaulieu et al., 2018). Indeed, studies have demonstrated that participation in a long-term exercise programme results in improvements in appetite control and changes in the appetite signals (gut hormones) that underlie these processes (Blundell, 2011; Caudwell et al., 2011; King et al., 2009; Martins et al., 2010; Martins, Morgan, & Truby, 2008). However, the mechanism underlying the influence of physical activity on appetite is not fully understood (Beaulieu et al., 2018).

It was also observed that in active individuals, fat-free mass and resting metabolic rate were positively associated with protein intake (g). This may indicate that fat-free mass influences both energy intake and macronutrient intake, and that signals arising from lean tissue reflect both the energy demands and the need for protein. Millward (1995) proposed that the protein-stat mechanism operates to ensure that the metabolic requirements of lean tissue are met. Since protein provides a source of essential amino acids (Millward, 1997), it may be that this mechanism operates to ensure an adequate supply of protein that is required for growth, repair and maintenance. It could be suggested that under conditions of acute protein restriction, the signals that arise from lean tissue exert a stronger influence on appetite regulatory processes to ensure food intake adequately matches the protein needs of the body. Further studies are needed to confirm this finding; however recent cross-sectional studies have confirmed the finding that fat-free mass is positively associated with protein intake (Cameron et al., 2016; Weise et al., 2014).

5.4.4 Limitations

This study had several limitations. The study design employed a single low protein meal to create an acute deficit in protein intake. However, the results suggest that a single meal did not create enough of a protein deficit to elicit a meaningful change in eating behaviour. Although changes in food intake and appetite were observed in activity groups, these findings were underpowered and may reflect the normal day to day variation in eating behaviour (Arvaniti et al., 2000; Gregersen et al., 2008). The overall coefficient of variation (CV) for energy and protein intake in this study was 13% and 19% respectively, however CV values of 18.9% have been reported for energy intake at a buffet meal (King et al., 2017). Additionally, two lunches were perhaps too few eating sessions to obtain an accurate representation of food intake, as typically better representation has been found with four or more eating sessions (Yeomans et al., 2009, 2005). Further studies should include multiple time-point assessments to examine if there is an exact condition effect.

The breakfast meals provided a set amount of protein (low: 3 g vs high: 33 g) and were not calculated according to individual protein needs. Although the low protein condition would have been low for all participants, the high condition would have provided a higher level of protein for some participants yet only a moderate amount to active individuals with comparatively greater daily protein requirements. This may have influenced the eating and appetite responses in the high protein condition. However, the finding that estimated food intake was reduced following the high protein breakfast suggests that the protein content was enough to elicit a small change in estimated food intake.

Alongside limitations in study design, the sample sizes for activity groups were small and unbalanced for sex, as there were no males in the sedentary activity group, as previously discussed. The group differences that were found were underpowered, and comparisons between activity groups were difficult to interpret accurately. Similar experimental studies have reported significant results using samples sizes of eleven participants or more (Beaulieu et al., 2018; Beaulieu, Hopkins, Long, et al., 2017). Future research should consider larger study samples and ensure activity groups are balanced for men and women.

Habitual diet was not assessed, however habitual dietary protein intake has been found to influence taste preferences following protein restriction (Masic & Yeomans, 2017). Obtaining an objective measure of physical activity would also strengthen the findings, for instance using data collected from accelerometers.

Therefore, future studies should include an assessment of dietary intake and level of physical activity.

5.4.5 Conclusion

The present study endeavoured to investigate how individual variations in physical characteristics and level of physical activity may impact the response to a low protein meal. Taking into consideration the study limitations, it was concluded that a single low protein meal may not be severe enough to elicit a consistent appetite response. However, across the study participants, strong associations observed between fat-free mass, resting metabolic rate and food intake indicate that body composition and metabolism and habitual physical activity play a key role in directing both energy and protein intake. Although the study does not provide conclusive evidence that individual differences influence the response to acute protein restriction, this area of research represents a key area for future studies. Further work is needed to advance our understanding of how dietary protein intake influences appetite and ingestive behaviour, and whether variations in physical characteristics, habitual dietary intake or level of physical activity moderates this relationship.

Chapter 6: Investigating associations between anthropometry, behavioural, socio-economic and genetic traits with eating behaviours and attitudes towards food in a community sample population

6.1 Introduction

Currently, it is estimated that over 60% of adults in the UK are classified as overweight or obese (The Health and Social Care Information Centre (NHS Digital), 2018b). This leaves 40% of the population classified as lean. This statistic indicates that despite the modern obesogenic environment, some individuals can avoid behaviours that promote the development of obesity, while others are more susceptible to them (Blundell et al., 2005). As such, research studies have characterised several eating behaviour traits associated with the development of obesity (Carter & Jansen, 2012; French et al., 2012; Mela, 2006; Mesas et al., 2012). The individual variation of these behaviour traits, therefore, may predict susceptibility to overeating, weight gain and excess adiposity. However, many of the studies have been conducted in experimental laboratories using small sample populations, while it is essential to understand whether these behaviours are associated with obesity at a broader population level.

There are several behaviour traits that have been observed in individuals with excess adiposity. These traits include an increased motivation to eat (Epstein & Leddy, 2006; Finlayson et al., 2007), reduced satiety responsiveness (Dalton, Finlayson, et al., 2013; Dalton et al., 2015), and a heightened sensitivity to reward (Davis et al., 2007, 2004). Studies have demonstrated that, in comparison to normal weight individuals, overweight individuals and individuals with obesity show a greater motivation to eat and are willing to exert more effort to obtain food, particularly foods that are palatable and energy-dense (Epstein & Leddy, 2006; Finlayson et al., 2008; Giesen et al., 2010; Saelens & Epstein, 1996).

Studies have also show that some individuals with obesity demonstrate impaired appetite regulation and are unable to accurately assess whether they are hungry or full (Drapeau et al., 2011). Delgardo-Argos et al., (2004) reported that an increased body mass index was associated with delayed satiation and individuals with obesity required approximately 225 kcal more energy from food to reach maximum satiation. Furthermore, the variation in appetite sensations in obese are reflected in an altered neural response to food cues (Gautier et al., 2000; Gautier et al., 2001) and modified postprandial gut hormone responses with food ingestion (Adam & Westerterp-Plantenga, 2005; Devoto et al., 2018; Le Roux et al., 2006; Mittelman et al., 2010; Le Roux et al., 2006; Verdich et al., 2001). These findings have important implications, because variations in appetite responses with ingestion may alter the expected satiation (the expected feelings of fullness) that foods may deliver (Brunstrom & Rogers, 2009).

Obesity is also associated with an altered neural reward response to food cues (Ng et al., 2011; Stice & Burger, 2019; Stice, Spoor, Ng, et al., 2009), suggesting that the individual variation in reward may increase the susceptibility to highly palatable foods. The individual variation in trait reward sensitivity, measured by Behaviour Activation Scale (BAS, Carver & White, 1994), may promote approach behaviours to palatable food cues and moderate the hedonic response to eating these foods (Beaver et al., 2006; Davis, 2009; Dawe & Loxton, 2004). These data indicate that at a population level, overweight and individuals with obesity may exhibit a heightened sensitivity to reward, greater motivation to eat and reduced satiety responsiveness toward palatable food cues.

An important consideration is that across a population group there are variations in food preferences and dietary intake, according to factors such as age, gender, socio-economic status, ethnicity, variations in genetic phenotypes and health behaviours such as exercise and smoking (Newby & Tucker, 2004). Accordingly, studies have identified common patterns of dietary intake in sub-population groups. For instance, a 'healthy' dietary pattern, consisting of a frequent intake of fruit, vegetables, legumes, grains, low-fat dairy products, fish and seafood, is found to be consumed by older individuals, who had attained a higher level of education and had greater wealth, lived in urban areas and were very often women. A 'traditional', 'continental' or 'Western' pattern dietary pattern, consisting of a high intake of full-fat dairy, meat, starchy vegetables, sweet pastries, fat spreads, sweet condiments, and dressings, is found to be consumed by individuals who are men, older, but those less well educated, less wealthy, and who lived in rural or more socially deprived areas (Ax et al., 2016; Bamia et al., 2007; Beck et al., 2018; Bertin et al., 2016; Fraser et al., 2000; Gazan et al., 2016; Knudsen et al., 2014; Markussen et al., 2016; Mikkilä et al., 2005; Mishra et al., 2002). Furthermore, individuals who consumed a 'healthy' dietary pattern had a lower body mass index (BMI) and waist circumference, while individuals who consumed 'traditional' dietary pattern, which typically contained higher amounts of saturated fatty acids, added sugars and less dietary fibre (Ax et al., 2016; Bertin et al., 2016; Knudsen et al., 2014), presented with a higher BMI (Bertin et al., 2016; Holmes et al., 2018) and waist circumference (Beck et al., 2018), higher levels of glycated haemoglobin (HbA1c) and fasting glucose - biomarkers of type 2 diabetes (Dekker et al., 2015).

Differences in dietary patterns between men and women are commonly reported. In several studies, men report eating foods typically of a 'traditional' dietary pattern (Ax et al., 2016; Beck et al., 2018; Bertin et al., 2016; De Silva et al., 2011; Fraser et al., 2000; Mikkilä et al., 2005; Sánchez-Villegas et al., 2003; Togo et al., 2004), while women commonly consumed a 'healthy' dietary pattern (Knudsen et al., 2014; Mikkilä et al., 2005; de Souza Fernandes et al., 2017; Wardle et al., 2004). Women are reported to consume a greater variety of foods compared to men (De Silva et al., 2011) and a more frequent intake of fruit, vegetables, cereals, legumes and potatoes (Baker & Wardle, 2003; Dibsdall et al., 2003; European Food Information Council, 2012; Gille et al., 2016), while men consume more meat, eggs, milk and sugary foods (Fraser et al., 2000; Gille et al., 2016). Similar differences in fruit and vegetable intake have also been reported between boys and girls (Bere et al., 2008; Krølner et al., 2011; Rasmussen et al., 2006) and it has been suggested that there are differences in liking or attitude toward fruit and vegetables between sexes, although this effect on intake is not apparent (Bere et al., 2008; Dibsdall et al., 2003; European Food Information Council, 2012). It may be that women have a tendency to follow a healthier eating pattern, to be aware of food or to be dieting, and have a higher intake of healthier foods (Knudsen et al., 2014; Mikkilä et al., 2005; Mishra et al., 2002; Pinto de Souza Fernandes et al., 2017).

Patterns of dietary intake also vary with age. Younger individuals are found to consume a 'processed' or 'fast-food' dietary pattern that includes a higher intake of processed foods and takeaway meals (Beck et al., 2018; Gazan et al., 2016; Knudsen et al., 2014; Whichelow & Prevost, 1996). In comparison, older individuals are found to consume more fruit and vegetables (De Silva et al., 2011; Fraser et al., 2000; Nicklett & Kadell, 2013). This observation has been found for populations in both the U.S. and Europe, although collectively, adults still fail to meet the current fruit and vegetable intake recommendations in these countries

(European Food Information Council, 2012; U.S. Department of Agriculture Center for Nutrition Policy and Promotion, 2007). The difference in dietary intake may reflect variations in the liking for specific flavours of food that changes with age (Lampuré et al., 2015; Padulo et al., 2017), cultural influences on food choices (Rozin, 2002), or a greater compliancy to meet nutritional recommendations (de Abreu et al., 2013; Webb et al., 1999).

Socio-economic status, such as education level, is seen to be an indicator of SES and shown to be inversely associated with weight gain over time (Atella & Kopinska, 2012; Ball & Crawford, 2005; Baum & Ruhm, 2009; Boylan et al., 2014), although McLaren (2007) highlights that this relationship depends on sex and the SES indicator (income, wealth index, education level). Studies have reported that individuals with lower levels of education have higher intakes of processed foods (Bertin et al., 2016; Fraser et al., 2000; Mishra et al., 2002), consume more meat, fat spreads (like butter and margarine), full-fat dairy products, added sugars, cakes and biscuits (Ax et al., 2016; Bamia et al., 2007; Beck et al., 2018; Bertin et al., 2016; Kesse-Guyot et al., 2009; Knudsen et al., 2014; Markussen et al., 2016), consume less fruit and vegetables (P. H. G. J. De Silva et al., 2011; Dibsdall et al., 2003), and have a more inferior quality of diet (Livingstone et al., 2016; Pinto de Souza Fernandes et al., 2017; Ribeiro et al., 2017). While individuals with a higher level of education were found to consume a more healthful diet composed of fruit, fruit juices vegetables, grains, cereals (Bamia et al., 2007; Deshmukh-Taskar et al., 2007; Krølner et al., 2011; Whichelow & Prevost, 1996) and more likely to comply to dietary recommendations (Abreu et al., 2013).

Inheritable characteristics may also influence eating behaviour. Specifically, polymorphisms of the ‘fat mass and obesity’ associated *FTO* gene are associated with overeating and obesity. Of the five most commonly reported polymorphisms of the *FTO* gene, single nucleotide polymorphisms of the A-allele variant (AA or AT) that occurs within rs 9939609, demonstrate a higher risk of obesity compared to individuals homozygous for the low-risk T allele (Frayling et al., 2007). Furthermore these polymorphisms are associated with increased body weight, hip circumference and fat mass (Dina et al., 2007; Hinney et al., 2007; Hunt et al., 2008; Scuteri et al., 2007). These associations have been found in both adults (Andreasen et al., 2008; Do et al., 2008; Frayling et al., 2007) and children (Cecil et al., 2008; Tanofsky-Kraff et al., 2009; Wardle et al., 2009). Studies including large sample populations report that the obesity-associated AA allele is associated with reduced satiety responsiveness, $n = 3337$ (Wardle et al., 2008), increased loss of control of eating, preference for high fat foods, $n = 299$ (Tanofsky-Kraff et al., 2009) and higher food intake, $n = 114$ (Melhorn et al., 2018). Specifically, individuals with the AA allele also find energy-dense foods more appealing and show greater activation of reward-related brain regions in response to viewing pictures of food.

Taken together, these studies suggest that there may be variations in eating behaviour and attitudes toward food between individuals grouped by age, sex, education level, and polymorphisms of the *FTO* gene. The variations in eating behaviour, such as a greater motivational drive to eat, a higher frequency of intake and reduced satiety postprandial response, may predict the susceptibility to overeating, particularly for foods that are energy-dense and high in fat and sugar or salt, and describe several pathways to the development of obesity. To this end, the objective of this study was to investigate the desire to eat, expected satiation and frequency of intake in a variety of food and compare responses across individuals grouped for age, sex, weight status, genetic risk for obesity (genetic variation of rs99393609 *FTO* gene), education level and sensitivity to reward.

I hypothesised that:

1. BMI and waist circumference will be higher in older individuals, men, individuals homozygous for the AA or AT allele of the *FTO* gene, and individuals with a lower level of education.
2. Overweight individuals and individuals with obesity and individuals homozygous for the AA or AT allele of *FTO* gene will have a greater desire to eat and their eating behaviour will involve an increased frequency of intake but reduced expected satiation when viewing images of energy-dense foods.
3. Individuals with a lower level of education will show an increased desire to eat and increased frequency of intake of processed, energy-dense foods, while individuals with a higher level of education will show a greater preference for healthier, lower energy-dense foods. Expected satiation will be reduced for healthier, lower energy-dense foods in individuals with lower level of education.
4. Men will show a greater desire to eat and increased frequency in intake of savoury foods and energy-dense foods, while women will show a preference for lower energy-dense, healthier foods.

6.2 Methods

6.2.1 Participants

Participants were recruited from an opportunity sample of visitors to the Science Museum, London, who took part in a study entitled "How much do you like to eat?" ('Live Science' public engagement, September-October 2015). A total of 560 participants (320 women, 240 men), aged between 18 to 85 years, took part. The predominant language spoken at home by the participants were English (73.4%). Details of the study population are listed in Table 6.2. Three participants were excluded from the analysis: of these participants, two individuals reported that they were pregnant at the time of testing and one participant reported as being a vegetarian. The study collected data from both children and adults; however, only data from the adults (participants 18 years old and older) were analysed.

6.2.2 Procedure

Members of the public that expressed interest in taking part were informed of the purpose of the study and provided written consent. Participants were then instructed on how to record their height, weight and waist circumference and then completed a series of computer-based (online) surveys (using online survey software, Qualtrics Inc., Provo, Utah, USA). Following the study, each participant was provided with a debriefing sheet which provided a detailed explanation of the study. The study was approved by the University of Roehampton Human Research Ethics Committee and Science Museum, London (PSYC 15/185)

6.2.3 Measures

6.2.3.1 Anthropometry

Under investigator supervision, participants measured their height, weight and waist circumferences according to given instructions. Height was measured to the nearest cm using a portable stadiometer (Tanita, TM). Weight was measured to the nearest 0.1 kg using a Tanita electronic scale. Waist circumference was measured using a standard plastic dressmaker's tape measure. Total daily energy requirements were calculated using a new predicted equation derived from the CALERIE study (Redman et al., 2014).

Participants were categorised into two groups according to BMI, lean (Lean) or overweight/obese (OWOB) similar to categories reported elsewhere (Tetley et al., 2009). The Lean group had BMIs ranging from 16.50 to 24.95 and OWOB group had BMIs > 25. Resting metabolic rate (RMR) was estimated using the Miffin-St Jeor (1990) equation using participant height (units), weight (units), age and sex and reported as megajoules per day (MJ.day^{-1}). Age was recorded to the nearest year.

6.2.3.2 FTO genotyping

Buccal (cheek) cell swabs were taken from each participant that agreed to donate anonymised DNA samples for *FTO* SNP genotyping. Participants were provided with nitrile/vinyl gloves and SK-1 Isohelix swab to take their sample, under guidance, by rubbing the swab for 30 seconds on the inside of each cheek. Each swab was then placed in a sealed tube (Isohelix Dri-capsules), stored in a locked container, until taken to Roehampton at the end of each week from where they were sent directly to DNA Genetics Ltd. (Norwich) for genotyping of DNA for the SNP rs9939609. After analysis, the remaining DNA samples were destroyed within three months of the end of the study.

There was a total of 68 AA individuals and 274 AT individuals, therefore these individuals were grouped together to form the AA/AT group (*n*= 342). There were 209 TT individuals.

6.2.3.3 Education level

Socio-economic status was assessed using the level of education participant had achieved. Education level has defined according to the National Qualification Framework as described UK government (Department of Education and Learning, UK government, 2020). Participants were grouped into four groups; A-level having achieved a high school diploma up to A-level (up to level 3); Diploma having achieved further education from (level 4 and 5); Bachelors (BA/BSc) (level 6) and Professional (level 7 and 8).

6.2.3.4 BAS scales: a measure of reward sensitivity

To provide a measure of reward sensitivity, participants completed the thirteen questions of the behavioural activation scale from the BIS/BAS questionnaire (Carver & White, 1994). The questions measure three subscales for behavioural activation: reward responsiveness (BAS RR, 5 items), determining positive responses to receiving of rewards ('When I get something I want, I feel excited and energized'); fun-seeking (BAS FS, 4 items), reflecting the desire to seek out new rewards ('I'm always willing to try something new if I think it will be fun') and drive (BAS D, 4 items), describing the strong pursuit of rewarding goals ('I go out of my way to get things I want'). Each question was responded to on a 4-point Likert scale, from "Very true for me" to "Very false for me", scored 4, 3, 2, 1. Each of the three scale scores was calculated by summing their item responses. The total score (Total BAS) consists of the total sum of each score of all subscales, thus ranging from 13 to 52. Reliability analyses for this sample showed that Cronbach's alpha values were 0.67 for Fun-seeking, 0.75 for drive and 0.64 for reward responsiveness. Overall Cronbach's alpha values were 0.81 for Total BAS.

6.2.3.5 Assessment of food satisfaction: online ranking questionnaire

Participants completed an online questionnaire to determine their preferences for ten food items (Table 6.1). The food images were obtained under licence from The Eating Behaviour Laboratory, University of Salzburg www.food-pics.sbg.ac.at, (Blechert et al., 2014). The size of each image was approximately 1.96 x 2.67 cm presented on a white background as a vertical list along with the other nine images (please refer to figure 6.1). Participants were asked to rank the food images from highest (1) to least (10) according to three questions: 1) “Which of these 10 foods would you most like to eat right now (in these portions)” [‘desire to eat’]; 2) “How filling are these 10 foods (in these portions)?” [‘expected satiation’]; 3) “How often do you eat each of these ten foods?” [‘frequency/familiarity’]. Participants were required to drag each image into a ranking box and order the images from 1 to 10 according to their preferences. For the question ‘How often do you eat each food?’, participants could assign foods to a “Never eaten before” box, and otherwise were asked to order them with the most often eaten food at the top. The order of presentation of food images was randomised for each participant. Each portion of food provided an approximately similar amount of energy, mean (SD) 238 (18) kcals, 974 (74) kJ (range: 213 to 267 kcal; 871 to 1094 kJ, please refer to Table 5.1). Participants were also asked to indicate the timing of their last meal: “When did you last eat?”. They chose from a drop-down menu offering time slots in minutes and hours (for example from ‘10 minutes’ to ‘over 6 hours ago’). An example of the questionnaire can be found in Appendix E1.

6.2.4 Data analysis

Tests for normality were conducted on all dependent variables. Age, waist, BMI, and timing of last meal were not normally distributed, and thus were natural-log-transformed. Frequency of intake scores was adjusted by excluding data for individuals who reported ‘never eating’ given food. Pearson’s correlations were used to determine the relationship between age and (separately) timing since the last meal, and rank scores for the desire to eat expected satiation and frequency of intake. Rank scores for foods were reversed coded to 10 as most preferred and 1 as the least preferred. To reduce the rank score data, a principal components analysis was performed separately for each eating behaviour (i.e. separately for desire to eat, filling and frequency). It was expected that food items could be grouped for fat content and flavour (e.g. high fat sweet or low-fat savoury food), or some other grouping following PCA analysis. However, the food items were poorly correlated and below the commonly used threshold of $r>0.3$ (Schwedhelm et al., 2018). This indicated that rank scores for similar food items (such as waffle, ice-cream, chocolate cake, and doughnut) were not positively correlated; refer to table A, B, and C in Appendix 1. Therefore, the rank scores for each food item were analysed separately for each eating behaviour. Chi-square analyses were used to test for any distribution bias between sex and BMI status (whether lean or overweight/obese), and between sex and the obesity-linked *FTO* gene (polymorphisms).

A one-way ANOVA was used to determine group differences in participant characteristic variables and reward sensitivity (sex, BMI, education level, genetic-linked obesity gene *FTO*). A two-way ANOVA examined the effects of sex and BMI status on rank scores, and, separately, the effects of sex and education level on rank scores. Analyses for education level used four levels: school education, post-school diploma level, bachelor’s degree level, and postgraduate degree level. Years since qualification was controlled for by excluding any participants younger than 24 years.

6.3 Results

6.3.1 Characteristics of sample population

The characteristics and statistical analyses of the study population are listed in Table 6.1. There were differences between men and women: BMI, waist circumference and RMR were all higher in men than women. The proportion of lean and OWOB participants differed between men and women, X^2 (1) = 20.98, $p <$ 0.001. There was a similar proportion of men and women in the AA/AT and TT categories for obesity linked *FTO* polymorphisms, X^2 (1) = 0.81, $p = 0.78$.

There were sex differences between scores for reward sensitivity; men tended to score higher in BAS fun seeking, F (1, 562) = 3.60, $p = 0.058$. Women scored higher in BAS reward responsiveness, F (1,561) = 8.73, $p = 0.003$. Proportions of men and women across levels of education did not vary significantly, X^2 (3) = 2.51, $p = 0.48$. Due to the high proportion of British, white participants (85.9%), no further analysis was conducted for Ethnicity.

6.3.2 Characteristics of different groups

6.3.2.1 FTO

AA/AT allele participants had a significantly higher BMI and waist circumference than did TT allele participants; Waist (mean ± SD): AA/AT = 84.78 ± 13.97 vs TT = 82.00 ± 11.73, F (1, 546) = 5.31, $p = 0.022$, partial $\eta^2 = 0.010$; BMI: AA/AT 26.68 ± 5.47 vs TT 24.68 ± 4.44, F (1, 546) = 10.86, $p = 0.001$, partial $\eta^2 =$ 0.020.

6.3.2.2 Age

Older individuals had a larger waist circumference and higher BMI compared to younger individuals, Waist r (555) = 0.25, $p < 0.001$; BMI r (555) = 0.30, $p <$ 0.001.

6.3.2.3 Education level

Age, waist circumference, BMI, RMR and reward sensitivity for levels of education are presented in table 5.2. There were differences in waist circumference and BMI between groups, BMI: F (3,347) = 5.26, $p = 0.001$, partial $\eta^2 = 0.044$), Waist: F (3,347) = 6.54, $p < 0.001$, partial $\eta^2 = 0.054$. Waist circumference was higher in A-level compared to BA/BSc participants, mean difference [95% CI] 6.36 [2.55 to 10.17], $p = 0.001$, and compared to Professionals, 5.72 [1.93 to 9.52], $p = 0.004$. BMI was higher in A-level compared to compared to BA/BSc participants, 2.63 [0.98 to 4.29], $p = 0.001$, and compared to Professionals, 2.26 [0.61 to 3.91] $p = 0.007$. Similarly, diploma educated participants had a higher waist circumference compared to BA/BSc, 6.00 [2.41 to 9.57], $p = 0.001$ and Professionals 5.35 [1.79 to 8.92], $p = 0.004$. BMI was higher in diploma educated participants compared to BA/BSc, 2.00 [0.44 to 3.56], $p = 0.006$ and Professionals 1.62 [0.073 to 3.17], $p = 0.031$. There was no interaction between education level and sex for both BMI and waist circumference (BMI: $p = 0.71$; Waist = 0.20). (please refer to figure 6.1)

Table 6.1
Descriptive information for sample population of visitors to the Science Museum, London.

	Total (n = 560)	Men (n = 240)	Women (n = 320)
Age (years)	31.77 (11.9)	31.15 (11.15)	32.24 (12.42)
BMI (kg.m^{-2})	25.57 (5.13)	26.2 (4.70)*	25.09 (5.39)
Waist (cm)	83.72 (13.19)	89.83 (11.97)**	79.13 (12.18)
RMR (MJ.day^{-1})	6564.45 (1106.92)	7564.94 (715.06)**	5818.8 (674.22)
BMI category			
Lean	305 [54.5%]	104 [18.6%]	201 [35.9%][a]
Overweight/Obese	255 [45.5%]	136 [24.3%]	119 [21.3%]
FTO			
AA/AT	339 [62.4%]	142 [26.2%]	197 [36.3%]
TT	204 [37.6]	88 [16.2%]	116 [21.4%]
Total BAS	41.4 (4.97)	41.45 (4.7)	41.37 (5.18)
BAS Drive	11.59 (2.2)	11.73 (2.08)	11.48 (2.28)
BAS fun seeking	12.35 (2.15)	12.54 (1.89)$^{+}$	12.21 (2.32)
BAS Reward Responsiveness	17.46 (1.96)	17.17 (2.04)	17.68 (1.88)***
Education			
Primary, Secondary, GCSE and A level	130 [23.2%]	63 [12.0%]	67 [11.3%]
Vocational diploma or Technikon	95 [17.0%]	41 [7.3%]	54 [9.6%]
BA/BSc's degree	193 [34.5%]	80 [14.3%]	113 [20.2%]

Postgraduate: Masters, Doctorate or Professional qualification	142 [25.4%]	56 [10.0%]	86 [15.4%]
Ethnicity			
White			
English, Welsh, Scottish, Northern Irish, British, Irish, Gypsy or Irish Traveller, Other	476 [85.9%]	199 [35.9%]	277 [50.0%]
Mixed White or Other			
White and Black Caribbean, White and Black African, White and Asian, other mixed	26 [4.7%]	11 [2.0%]	15 [2.7%]
Asian			
Indian, Pakistani, Bangladeshi, Chinese, other	33 [6.0%]	17 [3.1%]	16 [2.9%]
Black/African/Caribbean/Black British			
African, Caribbean, other black	5 [0.9%]	3 [0.5%]	2 [0.4%]
Middle Eastern & Other			
Arab, Jew, Other	14 [2.5%]	7 [1.3%]	7 [1.3%]

*BMI: Body Mass Index, RMR: Resting Metabolic Rate. Values are mean (Standard deviation) or frequency [percentage of total group]; * $p = 0.011$ higher in men compared to women; ** $p < 0.001$ higher in men compared to women; ***$p = 0.003$ Higher in women compared to men; $^{+}p = 0.073$ tendency to be higher in men compared to women. [a] A greater proportion of women than men were lean, X^2 (1) = 20.98, $p < 0.001$.*

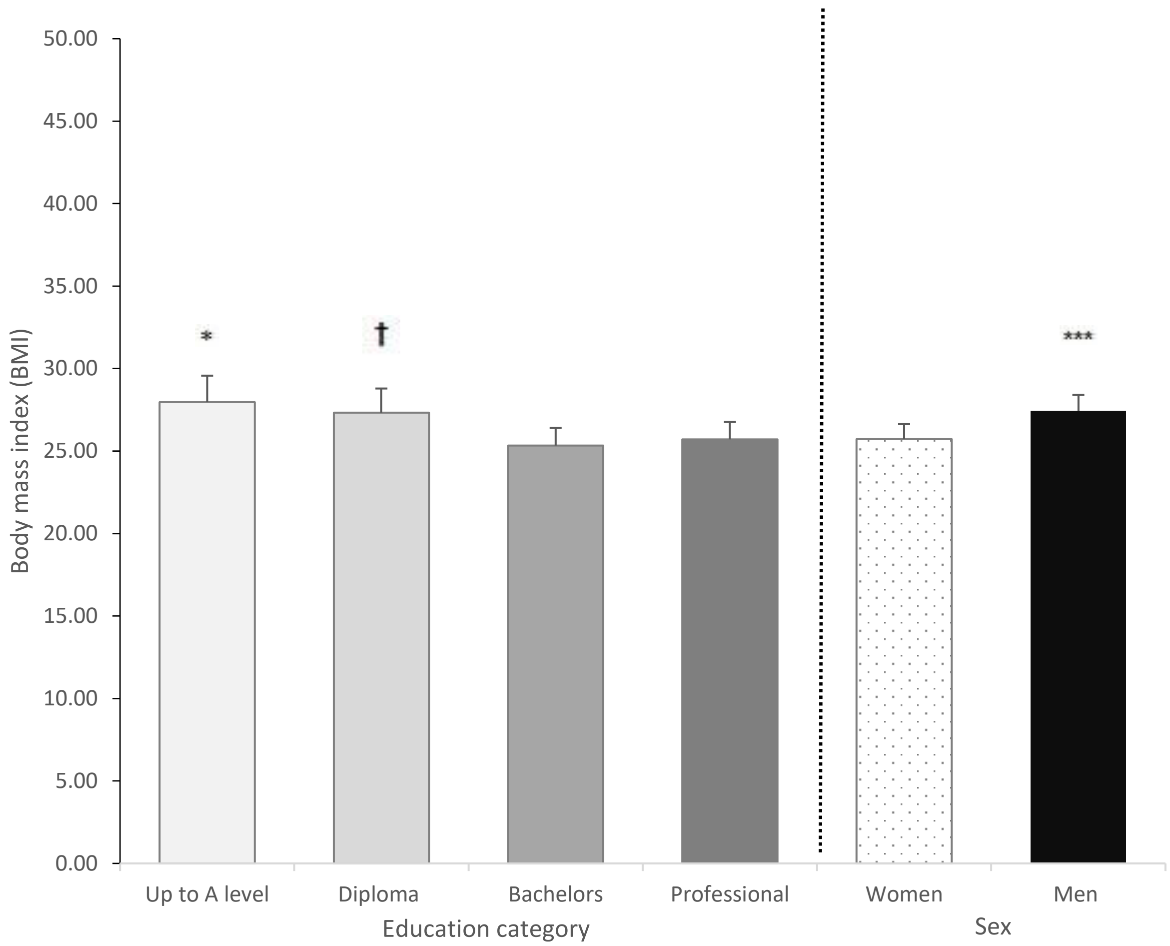


*Figure 6.1: Left bar graph: Comparison of BMI between education categories; up to A level (N = 52), Diploma or Technicon (N = 63), BA/BSc's degree (N=115), Professional (Masters, Doctorate or Professional qualification N = 125. Values are Mean ± 1SEM (accounting for difference between men and women), *p <0.05 significantly higher BMI in participants educated up to A level compared to BA/BSCs and Professional. † p = 0.072 tendency for higher BMI in participants educated up to diploma level compared to BA/BSCs and Professional. Right bar graph: Comparison of BMI between men and women. *** p < 0.001 Men had significantly higher BMI than women.*

6.3.3 Rank scores for desire to eat, expected satiation and frequency of intake:

6.3.3.1 Differences between men and women

A comparison of rank scores between men and women can be found in Table 6.3. Chocolate cake, red grapes, bread roll and waffle with whipped cream were ranked higher for desire to eat by women than men, chocolate F (1, 562) = 4.32, p = 0.038, grapes F (1, 562) = 17.05, p <0.001, bread roll F (1, 562) = 8.40, p = 0.004, waffle F (1, 562) = 12.04, p = 0.001 whereas beefsteak was ranked higher in desire to eat by men than women, beefsteak F (1, 562) = 35.38, p <0.001. Chocolate cake was ranked as more filling by men than women, F (1, 562) = 6.44, p = 0.011, bread roll was ranked more filling for women than men, F (1, 562) = 10.99., p = 0.001. Salmon fillet and grapes were consumed more frequently by women than men, salmon F (1, 562) = 5.317, p = 0.021, grapes F (1, 562) = 18.95, p < 0.001. Beefsteak was consumed more frequently by men than women, F (1, 562) = 10.89, p = 0.001 (Table 6.3).

6.3.3.2 Correlations between age and rank scores

Salmon, croissant and bread were ranked higher for desire to eat by older individuals, salmon, r (555) = 0.20, p < 0.001; croissant r (555) = 0.087, p = 0.041, and bread, r (555) = 0.16, p < 0.001. Salmon was ranked as more frequently consumed in the older, r (474) = 0.20, p <0.001. Hotdog, chocolate cake and doughnut were ranked lower for desire to eat by older individuals, hotdog r = -0.17, p < 0.001; chocolate cake, r (555) = -0.12, p = 0.005; and a doughnut, r (555) = -0.13, p = 0.002. Chocolate cake and doughnut were consumed less frequently by older individuals, chocolate cake, r (526) = -0.095, p = 0.029; and a doughnut, r (489) = -0.12, p 0.009. Doughnut was ranked as more filling, while waffle with whipped cream ranked as less filling by older individual's, doughnut, *r* (555) = 0.089, *p* = 0.036, waffle, *r* (555) = -0.13, *p* = 0.002.

6.3.3.3 Correlation with timing of last meal and rank scores

Participants who reported more time since last eating, ranked the hotdog and bread as significantly higher in desire to eat, hotdog, *r* (547) = 0.087, *p* = 0.042, and bread, *r* (547) = 0.084, *p* = 0.049. No further associations were observed for desire to eat, expected satiation and frequency of intake.

6.3.3.4 Associations between sensitivity to reward and rank scores

Higher scores for Total BAS were weakly associated with an increased desire for hotdog, *r* (560) = 0.10, *p* = 0.014 and increased ranking for frequency of beef steak, r (508) = 0.092, *p* = 0.039. No further associations were observed for desire to eat, expected satiation and frequency of intake.

Table 6.2:
Average rank scores for the desire to eat, expected satiation and frequency of intake between men (N = 240) and women (N = 320) for ten food items.

	Desire		Filling		Frequency	
	Men	Women	Men	Women	Men	Women
	Mean (SE)	Mean (SE)	Mean (SE)	Mean (SE)	Mean (SE)	Mean (SE)
Chocolate cake	4.69 (0.17)	5.16 (0.15)[a]	7.08 (0.14)[d]	6.56 (0.14)	4.95 (0.14)	5.29 (0.12)
Salmon Fillet	6.1 (0.21)	5.99 (0.18)	5.56 (0.17)	5.57 (0.16)	6.43 (0.2)	6.95 (0.16)[a]
Strawberry ice-cream	5.27 (0.15)	5.11 (0.14)	3.06 (0.12)	3.09 (0.1)	5.19 (0.15)	5.24 (0.13)
Chocolate covered doughnut	4.28 (0.16)	4.27 (0.14)	4.90 (0.14)	4.77 (0.12)	4.06 (0.15)	3.86 (0.13)
Beef Steak	7.99 (0.18)[b]	6.47 (0.17)	8.55 (0.15)	8.29 (0.13)	7.62 (0.15)[b]	6.74 (0.15)
Red Grapes	6.47 (0.18)	7.41 (0.14)[c]	2.83 (0.15)	3.07 (0.14)	6.86 (0.17)	7.79 (0.13)[c]
Bread roll	4.98 (0.18)	5.66 (0.15)[c]	6.13 (0.17)	6.83 (0.14)[c]	8.23 (0.14)	8.56 (0.11)
Waffle with whipped cream	4.37 (0.15)	5.08 (0.14)[c]	5.32 (0.14)	5.59 (0.13)	2.96 (0.13)	3.29 (0.1)
Plain croissant	5.25 (0.16)	5.48 (0.14)	4.28 (0.14)	4.15 (0.12)	5.96 (0.15)[f]	5.82 (0.13)
Hot dog	5.60 (0.18)[b]	4.38 (0.16)	7.30 (0.13)	7.08 (0.12)	4.72 (0.17)	3.97 (0.14)

Reverse scoring 1 = least preferred, 10 = most preferred. Women higher rank score than men, [a]$p < 0.05$, [c] $p < 0.001$; Men greater rank score than women, [b] $p < 0.001$; [d] $p < 0.05$, [f] $p < 0.05$

6.3.3.5 Differences between lean and overweight/obese participants

6.3.3.5.1 Desire to eat

Ice-cream was ranked higher in desire to eat by OWOB participants compared with the lean, OWOB 5.38 ± 0.15 vs Lean 4.94 ± 0.15, mean difference [95% CI] 0.44 [0.018 to 0.85], simple main effect for BMI, F (1, 556) = 4.19, p = 0.041, partial $\eta^2 = 0.007$. There was no effect of sex, $p = 0.62$. Multiple comparisons revealed that ice-cream was ranked higher in desire to eat by OWOB men compared to Lean men, OWOB 5.64 ± 0.21 vs Lean 4.79 ± 0.24, -0.85 [0.23 to 1.48], p = 0.008. Also, ice-cream was ranked higher in desire to eat by OWOB men compared to OWOB women (OWOB men 5.64 ± 0.21 vs OWOB women 5.12 ± 0.22, 0.52 [-0.079 to 1.12] $p = 0.088$); Interaction: F (1, 556) = 3.85, $p = 0.05$, partial $\eta^2 = 0.007$. Please refer to Figure 6.2.

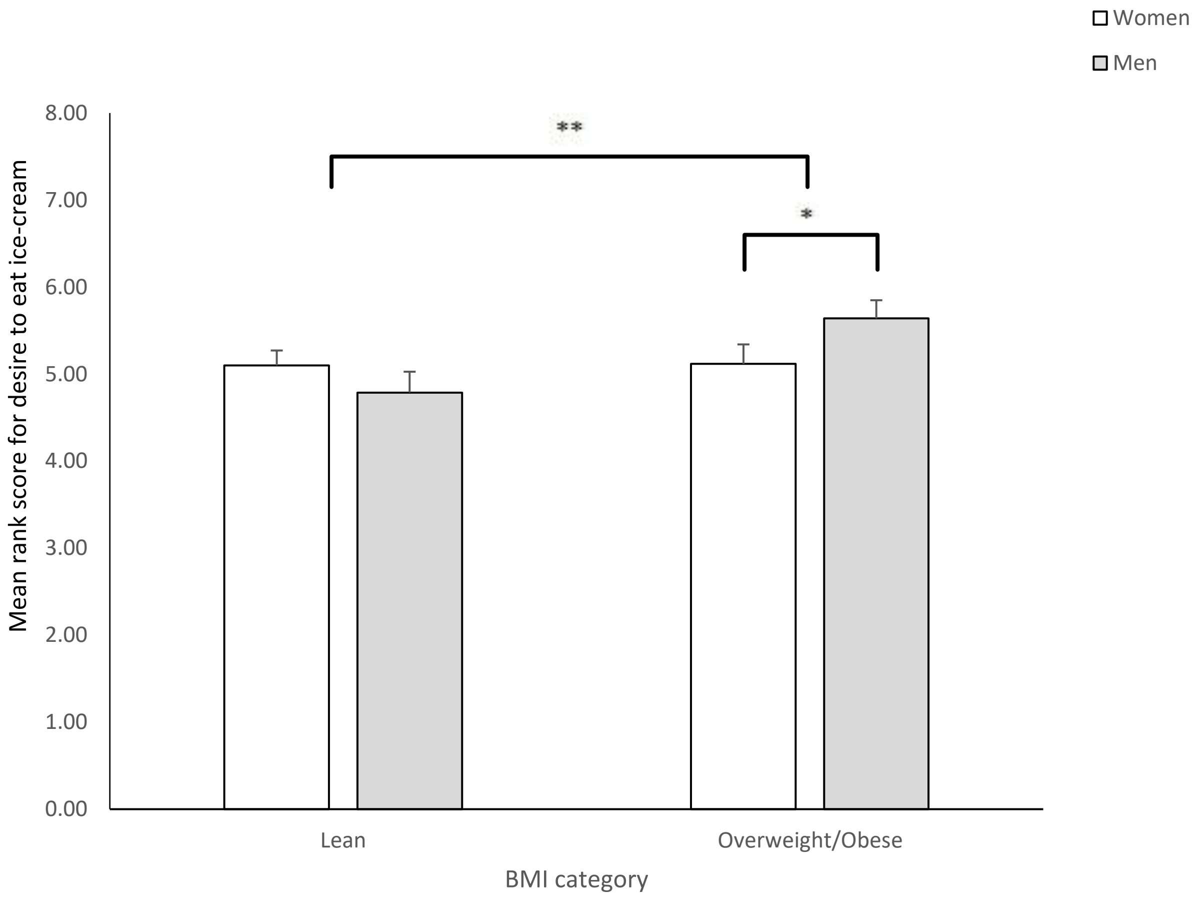


*Figure 6:2 Comparison of mean rank score for the desire to eat ice-cream between lean and overweight/obese men and women, *p = 0.008 higher mean rank score in overweight/obese men compared to overweight/obese women, **p = 0.041 higher mean rank score in overweight/obese compared to lean participants;*

6.3.3.5.2 Expected Satiation

'Filling' rank scores for grapes, waffle and hotdog differed between Lean and OWOB. Red grapes was ranked as more filling by OWOB compared to Lean (OWOB 3.37 ± 0.15 vs Lean 2.63 ± 0.14, 0.74 [0.34 to 1.15], F (1, 556) = 13.02, $p <$ 0.001, partial $\eta^2 = 0.023$). Waffle with whipped cream was ranked as less filling by OWOB compared with lean (OWOB 5.13 ± 0.14 vs Lean 5.71 ± 0.14, -0.58 [-0.96 to -0.20], F (1, 556) = 8.88, $p = 0.003$, partial $\eta^2 = 0.016$). Rank scores for hotdog tended to be lower for OWOB compared to lean (OWOB 7.00 ± 0.13 vs Lean 7.35 ± 0.13, -0.36 [-0.72 to 0.009], F (1, 556) = 3.66, $p = 0.056$, partial $\eta^2 = 0.007$). Please refer to Figure 6.3.

Further analyses using multiple comparisons revealed that there were differences between Lean and OWOB men and women in filling rank scores for chocolate cake, F (1, 556) = 3.11, $p = 0.078$, partial $\eta^2 = 0.006$; doughnut F (1, 556) = 2.90, $p = 0.089$, partial $\eta^2 = 0.005$, and beef steak F (1, 556) = 2.96, $p = 0.086$, partial $\eta^2 = 0.005$. Chocolate cake and doughnut was ranked as less filling by OWOB women compared to Lean women, OWOB women 6.21 ± 0.21 vs Lean 6.77 ± 0.16, -0.56 [-1.09 to -0.03], $p = 0.038$, doughnut, OWOB women 4.45 ± 0.20 vs Lean women 4.95 ± 0.66, -0.51 [-1.01 to -0.008], $p = 0.047$. Beefsteak ranked scores were lower for Lean women compared Lean men, Lean women 8.17 ± 0.17 vs Lean men 8.77 ± 0.23, -0.60 [-1.16 to -0.034], $p =0.038$.

6.3.3.5.3 Frequency

Hotdog, ice-cream and beefsteak was eaten more frequently by OWOB compared to Lean (OWOB 4.59 ± 0.16 vs Lean 4.20 ± 0.15, 0.39 [-0.05 to 0.82], F (1, 487) = 3.03, $p =0.082$, partial $\eta^2 = 0.005$); Ice-cream (OWOB 5.36 ± 0.14 vs Lean 5.03 ± 0.14, 0.33 [-0.059 to 0.72], F (1, 538) = 2.79, $p = 0.095$, partial η^2 =

0.005; and beef steak (OWOB 7.38 ± 0.16 vs Lean 7.03 ± 0.15, 0.36 [-0.066 to 0.78], F (1, 504) = 2.75, p = 0.098, partial η^2 = 0.005).

6.3.3.6 Differences between AA/AT and TT FTO polymorphisms linked with obesity

6.3.3.6.1 Desire to eat

Overall AA/AT participants ranked foods similarly to TT participants for all eating behaviours; however, differences were found for the desire to eat ice-cream. Ice-cream was ranked higher in desire to eat in AA/AT compared to TT participants; AA/AT 5.33 ± 0.13 vs TT 4.86 ± 0.17, mean difference [95% CI] 0.43 [0.00 to 0.86], F (1, 539) = 3.86, p = 0.05, partial η^2 = 0.007.

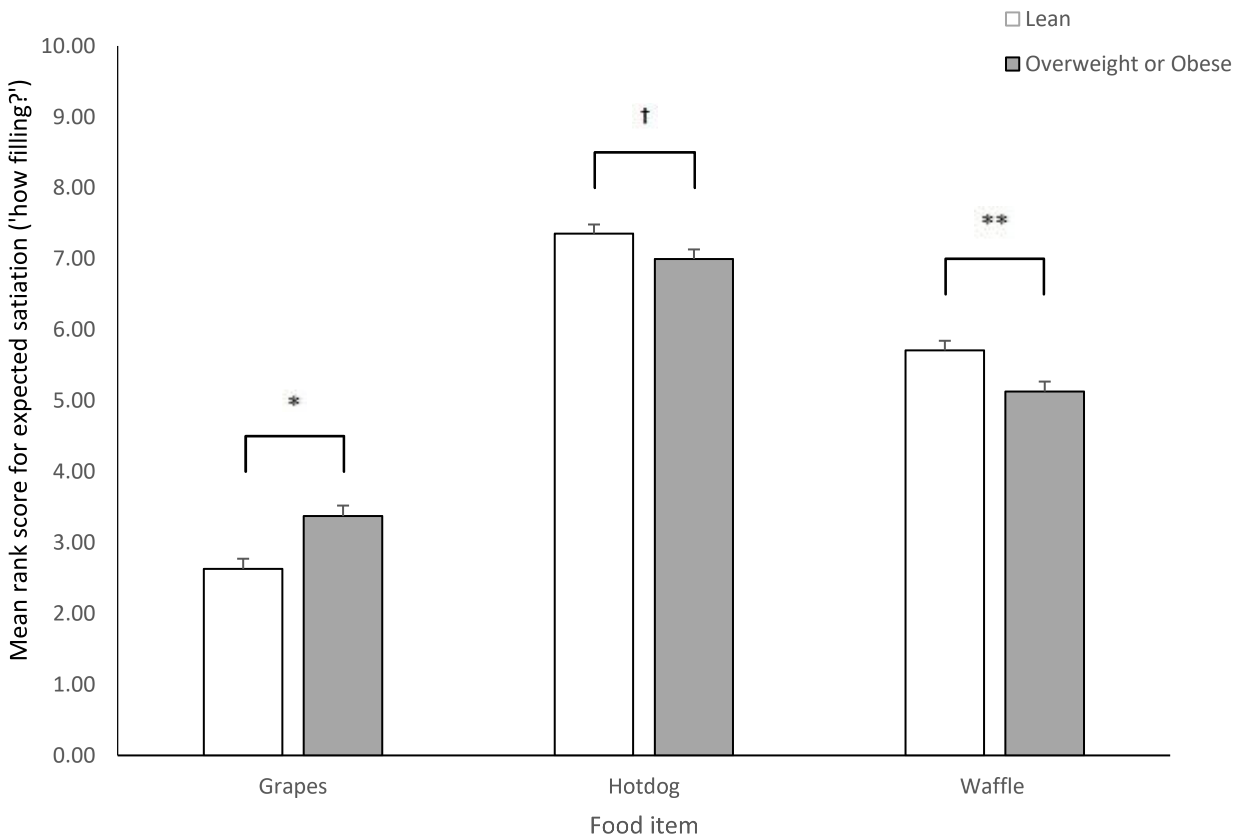


*Figure 6:3 Comparison of mean rank scores between lean and overweight/obese participants for expected satiation of grapes, hotdog and waffle with whipped cream, *p < 0.05, **p < 0.01, †p = 0.056.*

6.3.3.7 Education level

6.3.3.7.1 Desire to eat

6.3.3.7.1.1 Salmon

Salmon ranked higher in desire to eat in the BA/BSc and Professional educated participants compared to A level and Diploma group, *F* (3, 347) = 5.53, p = 0.001, partial η^2 = 0.046. The mean ± SE ranks scores were 5.60 ± 0.42, 5.48 ± 0.39, 6.93 ± 0.29, 7.00 ± 0.28 for the A level, Diploma, BA/BSc's and Professional group respectively. The BA/BSc group rank higher scores compared to A level, mean difference [95% CI] 1.33 [0.32 to 2.34], p = 0.010, and Diploma 1.46 [0.51 to 2.41], p = 0.003. Professionals rank higher scores compared to A level, 1.39 [0.38 to 2.39], p = 0.007 and Diploma group, 1.51 [0.57 to 2.46], p = 0.002. (please refer to figure 6.5)

6.3.3.7.1.2 Beef steak

Beefsteak was ranked higher in desire to eat by the A level and Diploma educated participants compared to BA/BSc's and Professionally educated participants, *F* (3, 347) = 3.60, p = 0.014, partial η^2 = 0.030. The mean ± SE ranks scores were 8.10 ± 0.41, 7.77 ± 0.38, 6.67 ± 0.28, 7.11 ± 0.28, respectively. The A levels ranked higher scores compared to BA/BSc 1.44 [0.46 to 2.42], p = 0.004, and Professional 0.99 [0.011 to 2.00] p = 0.047. The diploma educated participants ranked higher scored compared to BA/BSc 1.11 [0.18 to 2.03], p = 0.019.

6.3.3.7.1.3 Hotdog

Hotdog was ranked higher in desire to eat by A level educated participants compared to all other education groups, *F* (3,347) = 3.60, p = 0.014, partial η^2 = 0.030. The mean ± SE rank scores were 5.85 ± 0.34, 4.13 ± 0.36, 4.69 ± 0.26, 4.71 ± 0.26. A level vs Diploma 1.72 [0.67 to 2.76], p = 0.001; A level vs BA/BSc, 1.16 ±

[0.23 to 2.09], *p* = 0.015; A level vs Professional, 1.14 [0.21 to 2.07], *p* = 0.017. There was no interaction between education and sex both beef steak and hotdog (*p* = 0.75 and 0.78, respectively).

6.3.3.7.2 Expected Satiation

The rank scores for expected satiation for salmon was different between education groups, *F* (3, 347) = 5.70, *p* = 0.001, partial η^2 = 0.047. The mean rank scores were 4.67 ± 0.38, 4.87 ± 0.35, 5.84 ± 0.26, 6.22 ± 0.25 for the A level, Diploma, BA/BSc's and Professional group respectively. Salmon was ranked as less filling in the A level group compared to BA/BSc -1.17 [-2.07 to -0.26], *p* = 0.012, and Professionals -1.55 [-2.45 to -0.65], *p* = 0.001. The Diploma educated participants ranked lower scores compared to the BA/BSc -0.97 [-1.82 to -0.12], *p* = 0.026, and Professionals, -1.35 [-2.20 to -0.50], *p* = 0.002. There was no interaction between sex and education level, *p* = 0.54.

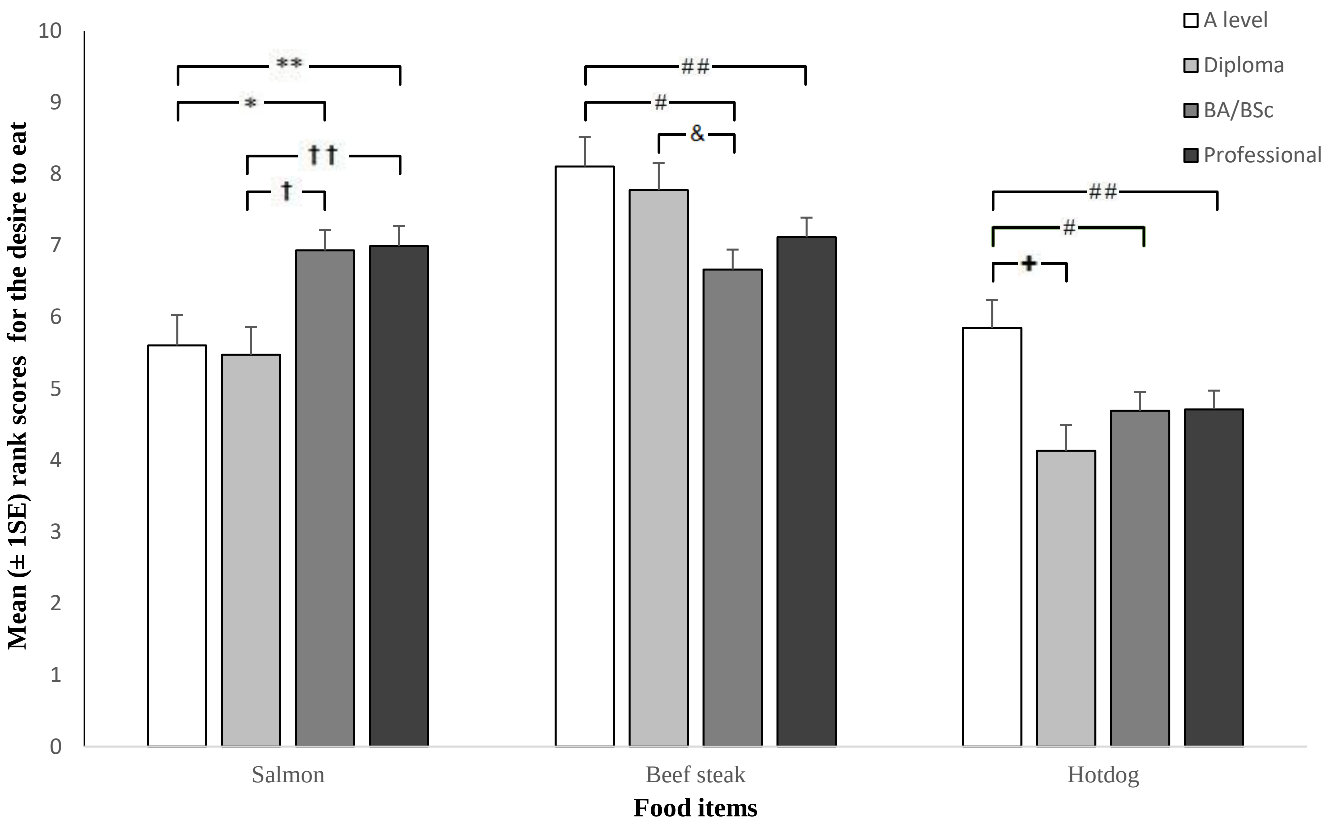


*Figure 6:5: Comparison of mean rank scores for the desire to eat salmon, beef steak and hotdog between participants educated to A level, Diploma, BA/BSc Degree or Professional level. Mean ± 1SEM †$p < 0.05$ Diploma lower than BA/BSc, ††$p < 0.05$ Diploma lower than Professional, *$p < 0.05$ A level lower than BA/BSc, **$p < 0.05$ A level lower than professional, $^{\#}p<0.05$ A level higher than BA/BSc, $^{\#\#}p<0.05$ A level higher than professionals, $^{\&}p<0.05$ Diploma higher than BA/BSc, $^{+}p<0.05$ A level higher than Diploma.*

6.3.3.7.3 Frequency

6.3.3.7.3.1 Salmon

There were differences ranking of frequency of salmon intake between men and women at different levels of education, F (3, 347) = 2.55, $p = 0.056$, partial η^2= 0.022. Salmon was eaten less frequently in diploma educated men compared to professional men, -1.88 [-3.56 to -0.21], $p = 0.018$. Salmon was eaten more frequently in professionally educated men compared to professionally educated women, 0.94 [-0.018 to 1.90], $p = 0.054$. There were no differences between sex (p = 0.34) or education level ($p = 0.14$) (simple main effects). Please refer to Figure 6.5

6.3.3.7.3.2 Doughnut

The simple main effects of education levels revealed that the rank scores for frequency of doughnut intake was different across education categories, F (3, 299) = 6.12, $p < 0.001$, partial $\eta^2 = 0.058$. The mean ± SE rank scores 4.90 ± 0.30, 4.09 ± 0.28, 3.66 ± 0.21, 3.40 ± 0.21 for A level, Diploma, BA/BSc's and Professional groups, respectively. Doughnuts were consumed more frequently by A-level participants compared to Diploma, 0.81 [0.003 to 1.62], $p = 0.049$, BA/BSc, 1.24 [0.52 to 1.92], $p = 0.001$ and Professional, 1.50 [0.78 to 2.23], $p < 0.001$. The diploma educated participants ranked higher scores compared to professionals, 0.69 [0.015 to 1.37], $p = 0.045$. Please refer to Figure 6.5

6.3.3.7.3.3 Hotdog

A simple main effect of education level on rank scores for hotdog F (3, 303) = 2.74, $p = 0.043$, partial $\eta^2 = 0.026$. The mean ranks scores were 5.16 ± 0.35, 4.28 ± 0.32, 4.39 ± 0.25, 3.94 ± 0.24 for A level, Diploma, BA/BSc's and Professional groups, respectively. Hotdog was consumed more frequently by A level educated participants compared to Professional, 1.27 [0.38 to 2.06], $p = 0.005$. Please refer to Figure 6.5

6.4 Discussion

This study provided an opportunity to measure eating behaviours and attitudes toward food in relation to behavioural and genetic traits in a community sample of visitors to the Science Museum, London. I observed a similar pattern of responses for individuals grouped according by BMI status, obesity-associated (*FTO*) gene, and education level; that individuals who ranked higher desire to eat and frequency of intake, yet lower satiation scores for energy-dense foods, were overweight or obese, had obesity-related ‘AA’ or ‘AT’ polymorphisms *FTO* gene, and had lower levels of education.

6.4.1 Eating differences between OWOB and lean individuals

In the study, individuals who were overweight or obese (OWOB) and those with the AA/AT allele, demonstrated a greater desire to eat ice-cream than lean, or low-risk TT genotype participants. OWOB individuals also reported consuming hotdog, ice-cream and beefsteak more frequently. Although beefsteak is considered a minimally processed, low energy dense food, and hotdog and ice-cream are medium energy-dense foods (energy density: 1.2, 2.3 and 2.6 $kcal.g^{-1}$ for beefsteak, hotdog, and ice-cream respectively, British Nutrition Foundation, 2018), these foods contain a high proportion of fat and sugar or salt. Beefsteak, ice-cream and hotdog have a lower nutrient quality, are processed and generally contain a high amount of energy, protein, alcohol, higher levels of saturated fatty acids, sodium and added sugars, but lower amounts of micronutrients and dietary fibre (Bertin et al., 2016; Gazan et al., 2016; Knudsen et al., 2014). Although I cannot infer causality, cross-sectional studies have shown that a higher intake of these foods is associated with a higher BMI and excess adiposity (Ax et al., 2016; Beck et al., 2018; Bertin et al., 2016; Gazan et al., 2016; Holmes et al., 2018; Knudsen et al., 2014). Two studies found that ice-cream intake was associated with a ‘sweet’ or ‘sweet-traditional’ dietary pattern and higher scores of these patterns were associated with increased BMI (Togo et al., 2004) and waist circumference (Newby et al., 2004). Furthermore, a higher intake of meat, refined grains, sweets and desserts (Fogelholm et al., 2012), fries, processed meats, butter (Mozaffarian et al., 2011) and alcohol (Lahti-Koski et al., 2002) was associated with long-term weight gain.

A heightened desire to eat palatable, energy-dense foods observed in the OWOB is supported by several experimental studies, where individuals with obesity show a greater motivation to eat food, particularly energy-dense, palatable foods (Epstein et al., 2012; Giesen et al., 2010). The finding from this study indicates that this behaviour trait is observable at a population level. Individuals with obesity are more motivated to receive immediate food or monetary reward, rather than delaying gratification for a relatively greater reward (Rasmussen et al., 2010). Epstein et al. (2014) reported that obese women were more motivated to eat and willing to discount a future reward for immediate gratification. Individuals with obesity are reportedly more drawn to food and will direct their attention to food more readily than lean individuals (Kemps & Tiggemann, 2015; Werthmann et al., 2011). Furthermore, studies in patients undergoing weight-loss surgery also demonstrated a specific reduction in desire to eat following the procedure, while the preference or liking for food remains the same (Miras et al., 2007; Ochner et al., 2012; Scholtz et al., 2014).

Similarly, ‘at risk’ AA/AT individuals had a higher desire to eat ice-cream, indicating that they may exhibit a greater motivation to consume these foods. These individuals also had a higher BMI and waist circumference compared to low-risk TT individuals, which is consistent with the findings from other studies (Hunt et al., 2008; Liu et al., 2010b; Scuteri et al., 2007). However, there was no difference in expected satiation or frequency of intake for any other foods, which was contrary to the study hypothesis.

The genetic influence on eating behaviour may to exert its effect through appetite. Specifically, AA/AT individuals report reduced satiety after eating (Grimm & Steinle, 2011; Wardle et al., 2008). Dougkas et al. (2013) reported that in overweight men, satiety was lower in the AA/AT genotype compared with the TT allele. The enhanced motivation to eat indicates that these individuals exhibit similar behavioural traits to the OWOB population; however, there may be specific traits of eating behaviour for which the AA/AT individual may be more susceptible. Although no differences in expected satiation were observed between at-risk AA/AT or low-risk TT individuals, a reduced satiety response may lead an individual to choose larger portion sizes or consuming foods more frequently. On the other hand, having a higher energy requirement, for instance due to greater fat-free mass in OWOB and AA/AT individuals (Dulloo et al., 2017), could lead to stronger learned appetites for energy-rich foods. Further studies are required to differentiate whether these individuals exhibit similar traits of eating behaviour that is associated with obesity development.

The OWOB men demonstrated a greater desire for ice-cream in comparison to OWOB women. This finding is perhaps not surprising because of the reported differences between men and women in attitudes toward foods. Generally, women may be more likely to be dieting (Wardle et al., 2004) or following a healthy eating pattern and therefore more likely to provide a socially desirable response (Arganini & Saba, 2012). Compared to overweight men, overweight women are likely to be exercising a higher level of dietary restraint to reduce body weight (Savage et al., 2009). Havermans et al. (2011) observed differences in approach tendency toward high-calorie foods between OW men and women, such that overweight women who presented with high dietary restraint demonstrated an avoidance of palatable food cues compared to overweight men. Thus, dietary restraint may be operating to influence food choice and eating behaviour.

Additionally, Knudsen (2014) reported no association between BMI and score of a ‘health-conscious’ eating pattern in women, while men with a higher BMI tended to score lower in this dietary pattern. They concluded that women might be more health-conscious than men irrespective of BMI status. Therefore, they will likely provide a more socially desirable response. Furthermore, Frankort et al. (2012) reported a reduced reward response to viewing high energy-dense foods compared to lean women that similarly indicates an avoidance of these foods as an attempt to restrain eating.

The OWOB individuals ranked divergent scores for the expected satiation of high and low energy-dense foods compared to lean individuals. The OWOB expected that high energy density foods, such as hotdog and waffle, would provide lower feelings of fullness, while lower energy density foods such as grapes were expected to provide greater feelings of fullness. Overall, energy-dense foods have been shown to offer less satiation than lower energy density foods (Brunstrom, Collingwood, et al., 2010; Brunstrom, Shakeshaft, et al., 2008), and high-fat food has been demonstrated to provide less satiation and satiety in comparison to high carbohydrate foods (Hopkins 2016). More specifically, overweight individuals and individuals with obesity show a reduced postprandial hormone response (Mittelman et al., 2010; Rizi et al., 2018; Tentolouris et al., 2004), and show an attenuated release of peptide YY (PYY) and impaired appetite response following ingestion of high fat foods (Batterham et al., 2006; Brennan et al., 2006). Moreover, compared to lean individuals, individuals with obesity required a higher volume of food to reach satiation (Delgado-Aros et al., 2004; Meyer-Gerspach et al., 2014).

The expected satiation value of food will be dependent on how familiar an individual is with that food. My study showed that OWOB individuals had higher expected satiation for grapes compared to lean individuals. While fruit and vegetables are reported to have a greater satiety value than high fat, processed foods (Buckland et al., 2015), OWOB frequently consumes a highly-processed diet, low in fibre, fruit and vegetables (Yu et al., 2018). OWOB will likely be less familiar with the satiation value of fruit and vegetables. Deglaire et al. (2015) reported that individuals with a higher BMI show a reduced liking for naturally occurring sweet flavours, like fruit, yet an increased liking for foods with processed sweet foods, like jams, sweets and soft drinks, and high-fat sweet foods, like doughnuts, croissants and chocolate cake, and this association was particularly strong in women. The study surmised that OWOB individuals might be less able to evaluate the sensory and post-ingestion qualities of the food because they are less accustomed to eating it. If an individual is not familiar with food, they will make a judgement based on portion size (Keenan et al., 2015a) and the image used in this study was a relatively large portion of grapes (300 g). The OWOB may have made judgements of the satiety value based on the presented portion size.

6.4.2 Differences in rank scores across education level

I observed differences in eating behaviour traits and attitudes toward food between individuals who were educated to an A level, Diploma/Technikon, university bachelor’s degree or professional/doctorate level. Individuals who were educated to a university degree and higher demonstrated a greater desire to eat salmon and reported eating it more frequently, while individuals educated to a diploma level or less demonstrated a higher preference and intake for foods like hotdog, beefsteak and doughnut. Furthermore, BMI and waist circumference were significantly higher in individuals with lower levels of education. In support of these findings, dietary intake studies have reported individuals who have achieved a higher level of education commonly consume healthier diets. Specifically, intake of fish or seafood was associated with dietary patterns such as 'healthy' (Ax et al., 2016), 'diversified' (Bertin et al., 2016), 'prudent' (Markussen et al., 2016; Perrin et al., 2005) and 'Green' (Togo et al., 2004), and higher educated individuals scored higher in these dietary patterns (or scores of these patterns are associated with higher education level). Furthermore, Deglaire (2015) reported that a decreased liking for salt was associated with a higher level of education. Educated individuals are found to consume fewer takeaway meals (Ax et al., 2016; Beck et al., 2018), consume more breads, cereals, fruit and vegetables (De Silva et al., 2011; Dibsdall et al., 2003; Fraser et al., 2000; Deshmukh-Taskar 2017, Fraser 2000, Dibsdall 2003, De Silva 2011). Educated individuals are also more likely to comply with nutritional recommendations (de Abreu et al., 2013; Webb et al., 1999).

In contrast lower levels of education were associated with dietary patterns such as 'traditional' (Ax et al., 2016; Beck et al., 2018; Bertin et al., 2016) 'basic' (Gazan et al., 2016), 'alcohol and meat' (Kesse-Guyot et al., 2009) 'Western' (Markussen et al., 2016). Lower levels of education were associated with poor quality of diet (Ribeiro et al., 2017), unhealthy snacking behaviour and unhealthy behaviours such as fast food consumption, smoking and sedentary behaviour, and snacking on energy-dense foods (Si Hassen et al., 2018; Wouters et al., 2017). The 'sandwiches' and 'burgers and sandwiches' dietary patterns were associated with lower levels of education (Bertin et al., 2016) or income (Deshmukh-Taskar et al., 2007). In contrast, Sánchez-Villegas et al. (2003) reported that higher educated individuals were more likely to follow a Western-style diet; however these findings may reflect a departure from consuming a traditional Mediterranean style diet in this population group.

I observed that less-educated individuals reported eating doughnut, beefsteak and hotdog more frequently; however, a high intake of these foods is not consistently associated with a dietary pattern. Beef is commonly found in 'traditional' dietary patterns (Ax et al., 2016; Bamia et al., 2007; Beck et al., 2018; Bertin et al., 2016; Kesse-Guyot et al., 2009; Knudsen et al., 2014). However, doughnuts are commonly represented under categories such as 'cakes' or 'pastries' and are not consistently associated with the commonly reported dietary patterns. For instance 'cakes', or 'pastries' were represented in 'traditional' (Ax et al., 2016; Knudsen et al., 2014; Lau et al., 2008), 'processed' (Bertin et al., 2016), 'Western' (Sánchez-Villegas et al., 2003), 'Sweet-fat dominated' (Bamia et al., 2007), 'Snacks and desserts' (Deshmukh-Taskar et al., 2007) and 'convenience' (Kesse-Guyot et al., 2009) dietary patterns; however, these dietary patterns, specifically the 'Western' 'snacks and desserts' and 'convenience' patterns, were not associated with level of education.

Similarly, the hotdog is often included in 'fast food' (Knudsen et al., 2014), 'burgers and sandwiches' (Deshmukh-Taskar et al., 2007), 'convenience' (Kesse-Guyot et al., 2009) and 'sandwiches' (Bertin et al., 2016) dietary patterns.

It should be noted that 'fast-food' and similar dietary patterns are more common in younger individuals (Beck et al., 2018; Gazan et al., 2016; Kearney et al., 1999; Knudsen et al., 2014), and also associated with unhealthy lifestyle behaviours such as smoking, low physical activity and high alcohol consumption (Mishra et al., 2002; Whichelow & Prevost, 1996).

Despite the contrasting results in these studies, an increased intake of fast foods is found to be associated with a higher BMI, particularly in lower educated individuals. Pieroni and Salmasi (2014) reported that higher BMI's were directly associated with availability and density of restaurants and fast-food outlets, and that BMI was also associated with comparatively lower-priced takeaway meals and snacks. Similarly Burgione et al. (2016) reported that higher exposure to fast food outlet and lower level of education level were significantly associated with higher odds of becoming obese. These findings have also been reported elsewhere (Burgoine et al., 2016; Murphy et al., 2018; Penney et al., 2018). Education is closely related to level of income (Specter & Drewnowski, 2004), and income is found to play a primary role in directing food choice (Dressler & Smith, 2013). Following a healthier diet is perceived as more expensive (Dibsdall et al., 2003; Dammann & Smith, 2009).

Monsivais (2009) demonstrated that lower energy-dense foods cost significantly more than higher energy-dense foods, and lower energy-dense foods were commonly consumed by individuals with a higher SES. Fast foods are found to be cheaper (Pieroni & Salmasi, 2014) and reportedly used more frequently in individuals with a lower income (French et al., 2000) . It may be that lower educated individuals have limited access to healthy food (Walker et al., 2010), are less likely to purchase healthier food (Turrell & Kavanagh, 2006), more likely to buy energy-dense foods that are comparatively lower in price and more affordable (Drewnowski, 2007). This means that they are more likely to consume fast-foods and take-away meals and snacks more frequently, and thus support more fast food outlets, as well as increasing their risk of obesity.

In this study, the variations in eating behaviour across a level of education may be a bit surprising. The lowest level of education in this study was a A-level education, which may be considered as relatively higher level of education attainment, while other studies observed associations in individuals who did not have secondary level education (Beck et al., 2018; Bertin et al., 2016; Markussen et al., 2016). Furthermore, visitors to the Science museum would have had an interest in education, and certainly a greater proportion of individuals were educated to a bachelor's degree level and above (59.9% of sample population). However, the results of the study further support the role of education level (as an extension of socioeconomic status), in influencing food choice and eating behaviour (Giskes et al., 2011).

6.4.3 The difference in rank scores between men and women:

In the study, men had a significantly higher BMI and waist circumference than women. This finding mirrors the population demographic reported for the UK; a higher proportion of men are classified as overweight compared to women, whereas the similar proportions of men and women are classed as obese (The Health and Social Care Information Centre, NHS Digital, 2018b). Men demonstrated greater desire to eat and consumed beefsteak more frequently and reported chocolate cake to be more filling. Women showed a greater desire to eat chocolate cake and reported eating it more frequently. Women also reported consuming grapes and salmon more frequently and inferred bread to be more filling compared to men. The results of the study indicate that men and women may differ in their motivational drive to eat, that men are more motivated to eat foods high in fat or protein, and salt, whereas women eat foods high in fat and sugar, although women also showed a preference for lower energy density foods. These findings are supported by other studies: women reportedly show a preference for sweet and fatty flavoured food, while men a preference for salty and fatty flavours (Lampuré et al., 2014); however, a greater preference for these flavours is also associated with obesity risk (Deglaire et al., 2015; Lampuré et al., 2016). Certainly, it has been reported that men find greater comfort in eating hot, savoury meals, where women find greater comfort in sweet snack food (Wansink et al., 2003). Women, especially those who score high in emotional eating, report a greater liking for sweet flavours (Lampuré et al., 2015) and women are more likely than men to eat for emotional reasons (Gibson, 2012).

The increased frequency of intake of foods such as bread and grapes are supported by the finding that women consume a healthier diet compared to men, and are more likely to be dieting or following nutritional recommendations (de Abreu et al., 2013; Deshmukh-Taskar et al., 2007; Friel et al., 2005;Wardle et al., 2004). Women also show a greater liking for naturally sweet flavours (Deglaire et al., 2015; Padulo et al., 2017) and eat more fruit and vegetables compared to men (Baker & Wardle, 2003; Padulo et al., 2017; Provencher et al., 2003).

6.4.4 The relationship between age and rank scores

Age was associated with a decreased desire to eat foods such as chocolate cake, doughnut and hotdog, yet increased for salmon, bread and croissant. In support of these findings, studies assessing food flavour preferences have reported that older individuals show a reduced preference for sweet foods (Lampuré et al., 2015; Padulo et al., 2017) and also a reduced liking for sweet and fat flavours (Lampuré et al., 2014). As discussed previously, hotdog is considered a ‘fast food’ or ‘takeaway’ food that would be more frequently consumed by younger individuals. Furthermore, the increase in snacking behaviour and particularly of foods high in fat and salt has been observed in younger individuals aged 19-29 years (Zizza et al., 2001), which suggests foods like 'hotdog' are consumed more prevalently by younger individuals. The study also observed that waist circumference and BMI were associated with age, which is a consistent finding in dietary intake studies. Both 'healthy' and 'traditional' diets are reported in older individuals; however these studies also show that smaller gains in BMI and waist circumferences are found in individuals who follow a healthier eating pattern (Beunza et al., 2010; Gazan et al., 2016; Newby et al., 2004).

6.4.5 Limitations

There were several limitations to this study. The study included a questionnaire that had not been validated. Although several validated research tools for the measurement of eating behaviour are available (Deglaire et al., 2012; Epstein & Leddy, 2006; Finlayson et al., 2007), the study took place in a public setting and participants were limited in how much time they could spend engaging in the 'Live Science' project. The questionnaire needed to assess traits and attitudes in a simple and quick manner; therefore, a rank order questionnaire using the ten food items was decided upon. Nevertheless, a limitation of ranked responses is the exclusivity or lack of independence of each rank, e.g. choosing to rank one food as highest means the ranks of all other foods must be lower, therefore only univariate analysis could be made from the data.

However, another limitation of the study is that the findings are based on univariate analyses, whereas multivariate analyses would have considered how several dependent variables influenced the outcome measure (for instance, age, sex, socioeconomic status influencing attitudes toward the ten chosen foods). Furthermore, the analyses were not corrected for multiple contrasts.

The results of this study largely agreed with findings from other studies, suggesting that the questionnaire did accurately assess traits and attitudes toward food. However, the poor correlation observed between rank scores for similar food items (i.e. waffle and doughnut, see table H1, H2 AND H3in Appendix H) suggests perhaps that some individuals may have misunderstood the nature of the question and answered incorrectly or that the food did not represent a food commonly consumed; for instance, waffle with cream was most frequently indicated as a food that participants never ate (18% of population data not shown).

As the study took place in a public setting, it was challenging to control extraneous influences on answers, particularly as individuals may deviate from the normal eating behaviours in comparison to those normally practised (i.e. in a naturalistic setting) (Robinson et al., 2014). The study did not control for restrained eating or dieting status. Furthermore, the results of the study are cross-sectional and cannot infer causality.

6.4.6 Conclusion

The results of the study showed that traits of eating behaviour and attitudes to food mirrored the reported variations in food intake and food preferences in individuals who differed by age, sex, adiposity, genetic risk for obesity and level of education. Individuals who expressed a greater desire to eat energy-dense foods and reported eating them more frequently were individuals who were overweight or obese, were carriers of the at-risk AA or AT alleles of *FTO* and had a lower level of education. Moreover, these individuals also demonstrated lower expected satiation for energy-dense foods. These findings provide important insight into the variation in eating practices in sub-population groups that may predict a susceptibility to overeating and weight gain. Although the results of this study cannot infer causality, the consistency with previous research indicates that these traits may underlie the observed variations in behaviour. Further research is needed to determine the direct or causal relationships between these traits and attitudes and dietary intake, to understand the pathways leading to the development of obesity.

Chapter 7: Does adiposity predict chosen portion sizes of commonly consumed foods as assessed by a food image task?

7.1 Introduction

One of the primary factors attributed to the development of obesity is the increased availability of large food portions offered to consumers. The portions sizes served at restaurants and fast-food outlets, and food packages provided by retailers to consumers have increased substantially, notably an increase in the availability of ‘supersize’ products, extra-large food packaging or food price promotions to encourage consumer sales (Dobson et al., 2017; Economic & Social Research Council, 2014; Nielsen & Popkin, 2003; Young & Nestle, 2003, 2012). Experimental studies have demonstrated that serving larger portions of food has a substantial effect on food intake and body weight (French et al., 2014; Jeffery et al., 2007; Rolls, 2014; Zlatevska et al., 2014), and therefore short-term appetite regulation. However, the effect on long-term appetite regulation is less clear, as studies have not demonstrated whether there is a relationship between excess adiposity and portion sizes. Yet, it is important to determine whether individuals with overweight or obesity habitually select larger portions of food, as this represents a key area for treatment intervention.

7.1.1 The relationship between BMI and portion size

Excess adiposity develops from the overconsumption of energy-rich foods, yet it is unclear whether meal frequency or large portions sizes contribute to a positive energy balance (Mattes, 2014). However, experimental studies have not consistently demonstrated a relationship between BMI and food portion sizes. In studies assessing the effect of large food portions on food intake (portion size effect [PSE]), similar responses were found between lean and overweight/obese participants (Hollands et al., 2017; McCrory et al., 2006; Rolls et al., 2007; Rolls et al., 2002), indicating that overweight individuals and individuals with obesity do not eat substantially more food compared to lean when provided with larger portions of food. Although a recent meta-analysis concluded that PSE might be attenuated in overweight or individuals with obesity (Zlatevska et al., 2014). These studies indicate that in the short-term, the presence of larger portion sizes of food does not cause individuals with obesity to eat substantially more food than lean individuals.

A key question remains as to whether large portion sizes influences long-term eating behaviour, or whether overweight or obese individual habitually select larger portions of food. Individuals with obesity report a higher overall energy intake (Howarth et al., 2007; Lindroos et al., 1997), and cross-sectional studies report that overweight individuals and individuals with obesity will habitually select larger portions of foods (Albar et al., 2014; Berg et al., 2009; Gouvea et al., 2012; Liebman et al., 2003).

However, several experimental studies have reported that BMI does not predict self-selected portion sizes. Using different experimental approach, where participants were asked to choose their ideal portion size from an array of food images of varying portion sizes (Brunstrom, 2014), no association was found between BMI and larger food portions (Brunstrom, Rogers, et al., 2008; Brunstrom & Shakeshaft, 2009; Fay et al., 2011; Reily et al., 2016; Wilkinson et al., 2012). In larger sample population groups, there has been an indication that BMI may be associated with self-selecting larger food portions; however the associations were not consistently observed across all population groups. Lewis et al. (2015) reported that individuals with obesity selected larger portions of food to serve themselves (referred to as a personal norm) compared to lean individuals, although the authors noted that the difference between the lean and obese groups was relatively small. Consistent with this observation, Labbe et al. (2017) reported that in a female population (n = 300), the association between self-selected large portion sizes and BMI was weak and non-significant. Spence et al. (2016) examined self-selected food portions in a sample of adults from Ireland and Denmark (n = 2075), yet found that BMI only predicted larger portion sizes in the Irish sample population. While it is important to consider methodological differences such as participant recruitment, test stimuli and sample size, overall these studies suggest that BMI is a relatively weak predictor of food portion sizes.

7.1.2 Waist to height ratio

In these studies, BMI was used as an indicator of obesity; however, it may be that a more definitive index of adiposity is needed to determine its effect on portion size, at least because BMI does not differentiate between excess adiposity or musculature (Ashwell & Lejeune, 2011; Ashwell, 2005). For instance, Blundell and colleagues have reported that BMI did not predict self-determined meal size amongst a sample of overweight individuals and individuals with obesity, however fat-free body mass and resting metabolic rate was positively associated with both meal size and energy intake (Blundell, Caudwell, Gibbons, Hopkins, Naslund, et al., 2012; Blundell, Caudwell, Gibbons, Hopkins, Näslund, et al., 2012; Caudwell et al., 2013). This finding has been confirmed in other studies (Cameron et al., 2016; Weise et al., 2014). Furthermore, in a sample of lean individuals, fat mass was negatively associated with energy intake (Blundell et al., 2015), indicating that body composition, namely fat-free and fat mass, play a more definitive role in directing food intake than bodyweight alone.

The measurement of body composition can be difficult in large sample sizes; however, waist circumference and height can be easily obtained and used to calculate the waist-to-height ratio (WHtR). The WHtR ratio is considered a more precise measure of excess adiposity (Ashwell, 2005; Ashwell & Hsieh, 2005; Ashwell & Lejeune, 1996) because it indicates the presence of central adiposity, a characteristic feature associated with obesity and several cardiovascular and metabolic health risks (Ashwell & Lejeune, 1996; Browning et al., 2010). WHtR is considered a better predictor of coronary (Hsieh & Muto, 2005; Lam et al., 2015) and metabolic risk factors (Ponnalagu et al., 2018), metabolic syndrome (Bi et al., 2019) and markers of insulin resistance (Benites-Zapata et al., 2019; Bi et al., 2019). WHtR is more strongly associated with BMI and percentage body fat than waist circumference (Anwar et al., 2019; Flegal et al., 2009). Ashwell (2005) has suggested that values of above 0.5 are associated with increased health risks, although consideration should be given to difference between men and women, as a cut-off value of 0.5 may underestimate central obesity in women (Csongová et al., 2018). Taken together, this suggests that measures such as WHtR and RMR may be a better predictor of food portion size.

7.1.3 Factors influencing decisions about portion size

Alongside body composition and metabolic rate, individual characteristics such as age and sex are associated with portion size. Men and women appear to respond similarly to the presence of larger portions of food (Hollands et al., 2017); Zlatevska et al. (2014) reported that the portion size effect was weaker in women, indicating that larger food portions may have a greater effect on men. When self-selecting portion sizes from food images, men habitually select larger portions of food to serve themselves (Lewis et al., 2015) and more specifically, will choose larger portions of main entrees and side dishes such as peas, rice and potatoes (Brunstrom, Rogers, et al., 2008). Men also have a greater tendency to clear their plate of food and indicate that they can eat more food at the end of a meal in comparison to women (Fay et al., 2011; Hinton et al., 2013). This behaviour may be related to the physiological differences, as men have relatively higher energy needs in men compared to women (Blundell et al., 2015). This suggests that men may consistently choose larger portions of food compared to women.

Age may also influence decisions about food portion sizes. Many studies have focused on the portion size effect on children. Younger children under 3 years old may be resistant to large food portions as they are more responsive to the internal homeostatic cues of hunger and satiety. Older children may be more sensitive to environmental stimuli, and therefore the presence of larger food portions influences eating behaviour (Benton, 2015; English et al., 2015; Rolls et al., 2000). Few studies have focused on the role of portion size across the adult lifespan; however, there is evidence that with ageing, older individuals choose smaller portions of food (Morley, 2001; Wysokiński et al., 2015). Furthermore Howarth et al. (2007reported that older individuals (between 60-90 years) consumed less energy, consumed fewer snacks and rarely skipped meals in comparison to younger (20-59 years) individuals. This suggests that compared to the younger, older individuals may estimate needing smaller portions of food.

The evidence suggests that WHtR and RMR may be better predictors of food portion size compared to BMI and that factors such as sex and age may moderate/influence the relationship between portion size and weight status. In the current study, participants were asked to choose the maximum portion size of a food that they could eat for lunch that would keep them full until dinner time. They were asked to select maximum portion sizes of five different foods using the study method provided by Brunstrom et al. (2009). The objective was to determine whether WHtR, RMR, age and sex were stronger predictors of maximum portion size compared to BMI. The test foods chosen were snack foods and side dishes, as described in (Brunstrom & Shakeshaft, 2009). The type of food (snack food or side dishes) were explicitly chosen to eliminate judgements on food portions based on expected norms.

I hypothesised that:

1. WHtR, RMR, age and sex would predict ideal chosen portion size
2. BMI would not be reliably associated with maximum portion size.
3. Higher WHtR and RMR would predict larger ideal portion sizes, while age (older participants) and sex (specifically females) would predict a smaller maximum portion sizes.

7.2 Methods

7.2.1 Participant characteristics

Participants were recruited from an opportunity sample of visitors to the Science Museum, London, and took part in a study entitled “How much do you like to eat?” (‘Live Science’ public engagement). A total of 555 adult participants (362 women, 193 men), aged between 18 and 85 years, took part in the study. The predominant language spoken at home was English (73.4%). Details of the study population are listed in table 6.2 in the results section. As in the study methodology reported in Chapter 6, three participants were excluded from the analysis. To control for food familiarity, participants were also excluded from analyses if they indicated that they were not familiar with the food item.

7.2.2 Procedure

The participants followed the study procedures as described in the Methods section 6.2 of chapter 6. The study was approved by the University of Roehampton Human Research Ethics Committee and Science Museum, London (PSYC 15_185)

7.2.3 Measures

7.2.3.1 Anthropometry

As described in Chapter 6, participants measured their height, weight and waist circumference under supervision. Resting metabolic rate was estimated using the Mifflin-St Jeor equation (Mifflin et al., 1990). Waist-to-height (WHtR) ratio was calculated using the following formula (Rönnecke et al., 2019)

$$\mathrm{WHtR} = \frac{\text{waist circumference (cm)}}{\text{height (cm)}}$$

7.2.3.2 Timing of last meal

The participants were asked to report when they had eaten their last meal as described in the Methods section of Chapter 6.

7.2.3.3 Estimation of portion size

7.2.3.3.1 Stimuli

The portion size task was a shorter version adapted from a portion size laboratory task published by Brunstrom and colleagues (Brunstrom & Rogers, 2009). Permission to adapt the task and use food images was obtained from Professor J.M. Brunstrom, Department of Experimental Psychology, University of Bristol. The task presented images of five different foods: peas (British garden peas, Sainsburys Supermarket Ltd), sweetcorn (Sainsburys Supermarket, Ltd), peanut M&M's (Mars Inc.), milk chocolate (Cadbury, Mondelez) and salted peanuts (Original salted peanuts, KPnuts, KP snacks). The macronutrient composition of the test foods was obtained from packaging and is detailed in table 7.1. Each food was photographed on a 255 mm diameter white plate as described (Brunstrom & Rogers, 2009). These foods were explicitly chosen to eliminate judgements on food portions based on habitual or expected norms. For each food portion, the first image displayed a 20 kcal portion. As the picture number increased, the portion size increased by 20 kcal, therefore picture 2 contained 40 kcal, picture 3 contained 60 kcal etc. The largest portion size depended on the amount of food that could be positioned on the plate. A total of between 40 to 70 food images for each food, providing a maximum of 800 kcal and 1400 kcal respectively. The name of the food and brand (where appropriate) was presented with the relevant image in the lower left-hand corner of the images.

Table 7.1

Nutritional information for five test foods

Food	Carbohydrate (g)	Protein (g)	Fat (g)	Total energy kcal /100g
Peas	9.1	5.9	0.9	68
Sweetcorn	19.6	4.2	2.3	116
Peanuts	9.9	27.5	49	590
M&M's	68.6	4.6	20.7	479
Chocolate	57	7.3	30	534

7.2.3.3.2 Measures

The maximum portion size was assessed by displaying an image of food portions in the middle of a 15-inch LCD-monitor. Portion size was measured in a one trial for each food item. In the trial, the food image was displayed on the monitor and participants could adjust the portion size by depressing the left or right arrow key on a keyboard. Depressing the left arrow key caused the portion size to decrease while pressing the right caused the portion size to increase, as described by Brunstrom & Rogers (2009). The participants were asked to choose a portion of food that would indicate the "maximum amount of that food they would choose to eat for lunch if no other food was available". The images of food and portion size were presented in a randomised order. After the participants had selected a portion size, participants were instructed to press a button marked 'continue'. Participants were asked to indicate the timing of their last meal as described in chapter 6.

7.2.4 Data analysis

Tests for normality and equal variance were conducted on dependent and independent (predictor) variables. Age, waist, WHtR, BMI, the timing of last meal, portion size for peas and sweetcorn were not normally distributed and transformed using the natural log function (Ln). If participants indicated they had 'never eaten' the food, they were not included in the data analysis for the respective food. Separate one-way ANOVAs were used to compare maximum portion size between individuals with WHtR greater or less than 0.5 and comparisons between men and women. A chi-squared test of homogeneity was used to test the proportional differences between men and women categorised according to WHtR < 0.5 or ≥ 0.5. Post hoc analysis employed pairwise comparisons using the z-test of two proportions.

Pearson’s correlations were used to determine the relationship between age, BMI, waist, WHtR and RMR and portion size of each food and the average portion size for all foods. From these results, variables with significant correlations were included in multiple regression models designed to test my hypotheses. Both age and RMR were significantly correlated (respectively, negatively and positively) with portion size for all foods (individual foods and average portion size) and included in the regression analyses (please refer to Table 7.4). BMI and waist were not correlated with the portion size of any food . WHtR correlated negatively with portion size of peas and M&M’s and average portion size for all foods and was included in the regression model. A multiple linear regression analysis was used to determine whether WHtR, age, sex and RMR were significant predictors of portion size. Sex was coded as females = 1, males = 2. Sex was strongly correlated with RMR; therefore, two separate models were performed: In model 1, age, RMR and WHtR were included as predictors in the regression equation. In model 2, age, WHtR and sex were included as predictors in the regression equation. All assumptions were met and detailed in Appendix I.

7.3 Results

7.3.1 Subject characteristics

Details of the participants' characteristics grouped by WHtR are listed in table 7.2. Individuals in the WHtR ≥ 0.5 group were older, had a larger waist circumference, and a higher BMI and resting metabolic rate (p<0.001). The high WHtR (≥ 0.5) group contained a greater proportion of men than women compared to the WHtR < 0.5 group (p < 0.001) (Table 7.2).

Table 7.2:

Comparison of the mean (standard deviation) age, waist circumference, body mass index (BMI), waist-to-height ratio (WHtR) and resting metabolic rate (RMR) for participants who were above and below cut off values of 0.5 for waist-to-height ratio

	All (N = 555)	< 0.5 (N = 362)	>= 0.5 (N = 193)
Age (years)	31.77 (11.9)	28.87 (9.93)	37.22 (13.32)**
Waist (cm)	83.78 (13.19)	76.34 (7.08)	97.12 (10.9)**
BMI (kg.m^2)	25.58 (5.11)	23.01 (2.54)	30.2 (5.31)**
WHtR	0.49 (0.08)	0.45 (0.03)	0.57 (0.06)**
RMR (kJ.kg^{-1}.day^1)	6564.45 (1106.92)	6273.45 (967.8)	7110.26 (1146.84)**
Sex (count)	198 (M) 362 (W)	133 (M) 229 (W)	107 (M) 91 (W)***
Sex (Percentage)		37% (M) 63.% (W)	54 % (M) 46% (W)***

***p < 0.001, *** p < 0.001 Chi-square test of homogeneity, greater proportion of men and smaller proportion of women classified WHtR ≥ 0.5 compared to WHtR < 0.5.*

7.3.2 Comparison of maximum portion size between individuals with WHtR < 0.5 or ≥ 0.5

Participants with WHtR ≥ 0.5 reported smaller portion sizes for peas, M&M's and chocolate, mean difference ± SE [95%CI]; peas (Ln Peas) -0.16 ± 0.077 [-0.31 to -0.012], one-way ANOVA p = 0.035, partial η^2 = 0.008; M&M's -16.38 ± 5.71 [-27.60 to -5.16], p = 0.004, partial η^2 = 0.015; Chocolate -10.45 ± 5.15 [-20.57 to -0.32], p =0.043, partial η^2 = 0.008 (please refer to figure 7.1)

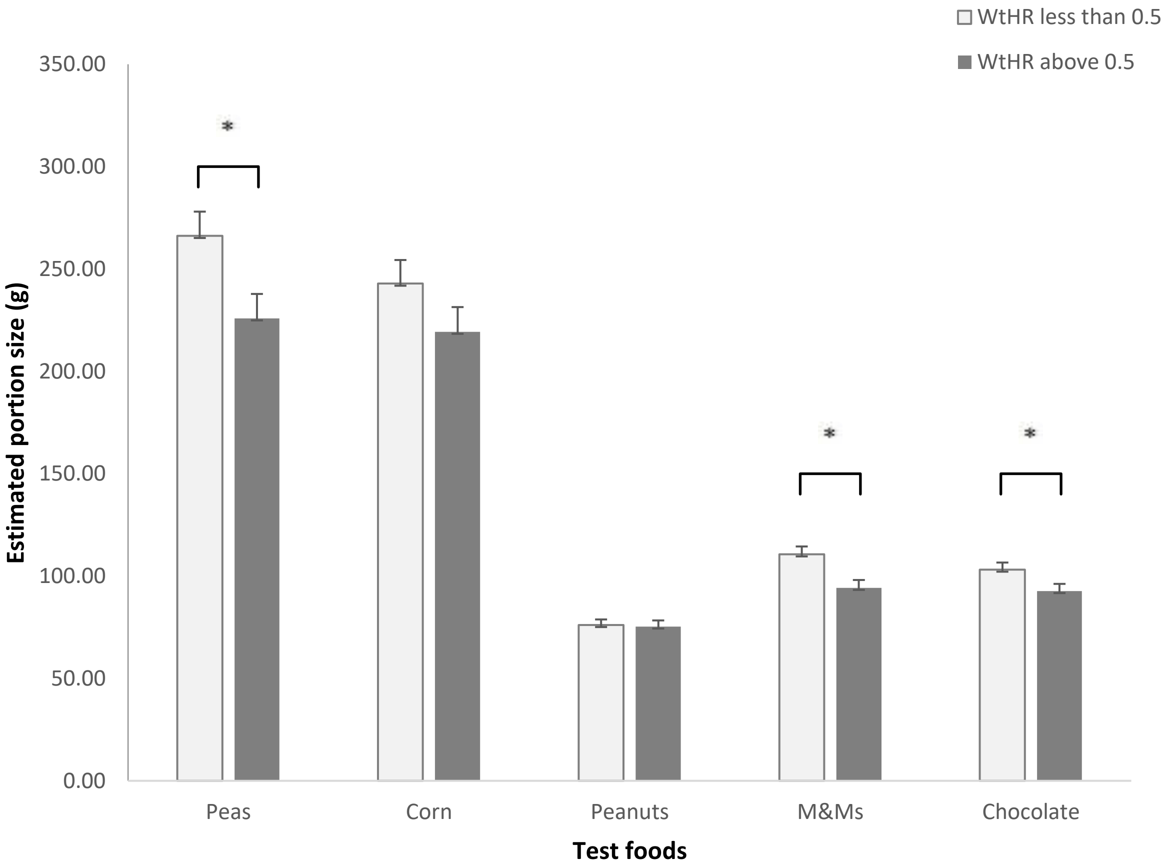


*Figure 7.1: Comparison of maximum portion size of peas, corn, peanuts, M&M's and chocolate between participants with WHtR of less than or greater than 0.5; *p < 0.05 portion size larger for individuals with WHtR less than 0.5. Mean ± 1SEM, (1-way ANOVA tests)*

7.3.3 Comparison of maximum portion sizes chosen between men and women

Men's chosen higher portion sizes for all foods, compared to women (please refer to table 7.3)

Table 7.3:

Comparison of maximum portion size (g) between men and women of peas, corns, peanuts, M&M's and chocolate mean, Mean ± 1SEM

Food (g)	All (N = 541)	Men (N = 235)	Women (N = 306)
Peas	252.8 ± 8.51	299.12 ± 13.86	217.22 ± 10.2**
Corn	234.51 ± 8.42	274.95 ± 14.09	203.98 ± 9.94**
Peanuts	75.91 ± 2.01	90.17 ± 3.06	65 ± 2.5**
M&M's	104.83 ± 2.77	117.59 ± 4.25	95.18 ± 3.55**
Chocolate	99.55 ± 2.49	108.13 ± 3.81	93.11 ± 3.24*

**p<0.01 and **p< 0.001 Men reported higher portion sizes compared to women*

7.3.4 Associations between predictor variables and maximum portion size for each food

BMI (LnBMI) and waist were not correlated with maximum portion size ($p > 0.05$) for all foods except peas. BMI and waist were excluded from subsequent analyses (largest *r* for BMI = -0.089, $p = 0.038$ for peas; largest *r* for waist = 0.052, $p = 0.22$). Older age (LnAge) and higher RMR were associated with decreased and increased portion sizes of all foods, respectively. Higher WHtR (LnWHtR) was associated with a smaller portion size for peas and M&M's (Table 7.4).

Table 7.4:

Pearson correlations between predictor variables BMI (LnBMI), Age (LnAge), Waist, WHtR (LnWHtR) and RMR for maximum portion of peas, corn, peanuts, M&M's, chocolate and average portion size for all foods (All)

Maximum portion (g)	**Peas**	**Corn**	**Peanuts**	**M&M's**	**Chocolate**	**All**
LnBMI	-0.075	-0.003	-0.029	-0.066	-0.023	-0.053
LnAge	-0.13**	-0.15**	-0.15**	-0.29**	-0.22**	-0.21**
LnWaist	-0.03	0.02	0.05	-0.03	0.02	0.00
LnWHtR	-0.14**	-0.079	-0.049	-0.10*	-0.057	-0.11**
RMR	0.18**	0.20**	0.24**	0.18**	0.18**	0.25**

**p<0.05; **p< 0.001; LnBMI = natural log body mass index (BMI), LnAge = natural log of age,LnWaist =natural log of waist; LnWHtR = Natural log of waist to height ratio*

7.3.5 Correlation between timing of last meal and maximum portion size

The timing of the last meal did not influence maximum portion size; peanuts *r* (539) = 0.024, $p = 0.58$; M&M's *r* (546) = -0.043, $p = 0.28$; Chocolate *r* (544) = -

0.051, p = 0.24; Peas r (546) = -0.029, p = 0.49 and Sweetcorn, r (542) = 0.006, p = 0.89.

7.3.6 Predictors of maximum portion size

7.3.6.1 Model 1

Model 1 included age, resting metabolic rate and waist-to-height ratio as predictors. Model 1 significantly predicted portion size; however it only explained a small proportion of the variance: peas 7.8 %; sweetcorn 6.7%; peanuts 8.7%; M&M's 11%; chocolate 7.1%, respectively. Higher RMR predicted a larger portion size for all foods, while older age predicted a smaller portion for sweetcorn, M&M's and chocolate. Higher WHtR predicted smaller portion sizes for all foods except chocolate. The model is detailed in table 7.5

7.3.6.2 Model 2

Model 2 included age, waist-to-height ratio and sex as predictors. Model 2 also significantly predicted portion size; however it also only explained a small proportion of the variance: peas 6.9%; Sweetcorn 5.7%; peanuts 8.7%; M&M's 11%; Chocolate 6.0%, respectively. Males chose larger portion sizes for all foods in comparison to females. Older age predicted a smaller portions of all foods. Higher WHtR predicted a smaller portion size for peas. Model 2 is detailed in table 7.6

Table 7.5: Unstandardized (*B*) and standardised (Beta) regression coefficients for Age (lnAge), resting metabolic rate (RMR) and WHtR (lnWHtR), *t* values, *p*-values, and the full model for standard regression models predicting maximum portion size (Model 1)

Food	Predictors					Full model		
		B	SE	Beta	*t*	Adj R^2	df	*F*
LnPeas	LnAge	-0.08	0.12	-0.03	-0.65			
	RMR	0.21	0.04	0.27	5.47***	0.078	3,532	16.04***
	LnWHtR	-1.42	0.30	-0.24	-4.68***			
LnCorn	LnAge	-0.27	0.12	-0.10	-2.21*			
	RMR	0.18	0.04	0.23	4.63***	0.067	3,528	13.63***
	LnWHtR	-0.87	0.32	-0.14	-2.77**			
Peanuts	LnAge	-6.47	6.25	-0.05	-1.04			
	RMR	12.90	2.03	0.31	6.37***	0.087	3,526	17.86***
	LnWHtR	-53.98	16.32	-0.17	-3.31*			
M&M's	LnAge	-43.57	8.51	-0.23	-5.12***			
	RMR	11.76	2.79	0.20	4.21***	0.11	3,533	23.45***
	LnWHtR	-50.51	22.44	-0.11	-2.25*			
Chocolate	LnAge	-29.36	7.82	-0.18	-3.76***			
	RMR	10.12	2.54	0.19	3.98***	0.07	3,531	15.22***
	LnWHtR	-30.50	20.36	-0.08	-1.50			

p< 0.05 **p < 0.01,* p < 0.001. LnAge = Natural log of Age, LnWHtR = Natural log of waist to height ratio, LnPeas = natural log of peas, LnCorn = natural log of sweetcorn.*

Table 7.6: Unstandardized (*B*) and standardised (Beta) regression coefficients for Age (lnAge), (Sex) and WHtR (lnWHtR), *t* values, *p*-values, and the full model for standard regression models predicting maximum portion size (Model 2)

Food	Predictors					Full model		
		B	SE	Beta	*t*	Adj R^2	df	*F*
LnPeas	LnAge	-0.23	0.11	-0.09	-2.09*			
	WHtR	-0.83	0.27	-0.14	-3.09**	0.069	3,532	14.13***
	Sex	0.36	0.07	0.21	4.94***			
LnCorn	LnAge	-0.40	0.11	-0.16	-3.52***			
	WHtR	-0.34	0.28	-0.05	-1.21	0.057	3,528	11.61***
	Sex	0.30	0.08	0.17	3.95***			
Peanuts	LnAge	-15.69	5.93	-0.12	-2.64**			
	WHtR	-18.85	14.40	-0.06	-1.31	0.087	3,536	17.75***
	Sex	25.17	3.97	0.27	6.34***			
M&M's	LnAge	-51.77	8.10	-0.28	-6.39***			
	WHtR	-18.04	19.61	-0.04	-0.92	0.11	3,533	23.66***
	Sex	23.06	5.39	0.18	4.28***			
Chocolate	LnAge	-37.31	7.49	-0.22	-4.98***			
	WHtR	0.21	18.06	0.00	0.012	0.06	3,531	12.71***
	Sex	14.71	4.99	0.13	2.95**			

p< 0.05 **p < 0.01,* p < 0.001. LnAge = Natural log of Age, LnWHtR = Natural log of waist to height ratio, LnPeas = natural log of peas, LnCorn = natural log of sweetcorn, Sex coding: Females = 1, Males = 2.*

7.4 Discussion

This study examined whether adiposity, especially the health-associated waist-to-height ratio (WHtR), and resting metabolic rate (RMR) predicted maximum portion size of five foods in a sample population of visitors to the Science Museum, London. I found that both WHtR and RMR explained a significantly proportion of the variation in maximum chosen portion size, while BMI did not. However, contrary to the study hypothesis, a higher WHtR predicted a smaller maximum chosen portion size of snack foods and side dishes.

7.4.1 RMR, WHtR, sex and age are better predictors of portion size than is BMI

The results of this study showed that RMR, WHtR, sex and age were significant predictors of portion size, while BMI was not. The inability of BMI to predict portion size is consistent with findings from other experimental studies. Whether participants choose their ideal portion size from an array of food images (Brunstrom, Rogers, et al., 2008; Fay et al., 2011; Wilkinson et al., 2012; Zuraikat et al., 2018) or participants food intake was measured (meal size or volume of food consumed, Blundell, Caudwell, Gibbons, Hopkins, Naslund, et al., 2012; Blundell, Caudwell, Gibbons, Hopkins, Näslund, et al., 2012; Caudwell et al., 2013), in these studies BMI was not associated with portion size or meal size.

In the current study, participants were asked to choose a maximum amount of that food they would choose to eat for lunch if no other food was available. Resting metabolic rate predicted larger portion sizes for all foods, meaning that individuals with higher energy needs estimated that they needed larger portions of snack foods and vegetables to maintain satiety. This finding is in line with a recent proposed Formulation for Appetite Control, that emphasises that fat-free body mass and RMR are better indicators of appetite and food intake than is BMI or fat mass (Blundell, 2018; Hopkins et al., 2016). My findings further suggest that RMR also plays a role in influencing decisions about portion size. Since RMR reflects the physiological demand for energy, it is proposed that these signals would influence both biological and behavioural processes to ensure adequate food intake (Blundell, 2018).

7.4.2 Higher adiposity associated with smaller portion size estimates

WHtR was used in this study as a measure of adiposity yet was found to be negatively related to maximum portion sizes for M&M's and peas and predicted a smaller portion size for all foods (except chocolate) when accounting for resting metabolic rate (energy requirements) and age. This was contrary to the study hypothesis. Other studies have shown that fat mass does not predict energy intake across the spectrum of adiposity (i.e for lean, overweight individuals and individuals with obesity) (Blundell et al., 2012; Blundell et al., 2015; Woodward et al., 2017), however in two studies fat mass index (fat mass expressed as a proportion of height: kg/m^2), was associated with a lower energy intake (Blundell et al., 2015; Weise et al., 2014). As fat mass index and WHtR are similar measures of adiposity (i.e. weight or circumference as a proportion of height), the findings of this study support the hypothesis that fat plays an inhibitory role on food intake (Blundell, 2018; MacLean et al., 2017). However, it is also important to consider that in this study, participants were responding to visual food cues, not the presence of food. Furthermore, behaviours such as dietary restraint, underreporting and differences in expectations of postingestive effects of food may have influenced the outcome of the present study.

The smaller predicted portion sizes could reflect a form of underreporting that is consistently observed in overweight/obesity study participants.

Overweight individuals and individuals with obesity are less likely to accurately report their food intake and preferences (De Castro, 2010), and under or misreport dietary intake by an average of 15 % (Poslusna et al., 2009). This behaviour has been observed for adults (Heitmann & Lissner, 1995; Merema et al., 2019; Visscher et al., 2006) and children (Abreu et al., 2014; Gomez-Bruton et al., 2019; Vieira et al., 2014). In the current study, the smaller portion estimates predicted by higher WHtR may reflect a form of underreporting whereby individuals with higher adiposity deliberately chose smaller portion sizes to provide a more socially desirable response (Merema et al., 2019).

Considering that testing took place in a public area, participants with higher adiposity may have felt more self-conscious about disclosing their desired food portions. Lewis et al. (2015) found that in comparison with lean, individuals with obesity selected similar portions of food considered normal by other people (social norms), whereas they chose a significantly larger portion of food (compared to lean) for what they considered normal for themselves (a personal norm). This suggests that overweight individuals and individuals with obesity are aware of portion sizes deemed normal or acceptable at a social level and may adjust estimates accordingly. Furthermore, similar studies highlight the role that underreporting may play a role in influencing the relationship between BMI and meal size (Brunstrom, Rogers, et al., 2008; Labbe et al., 2017; Spence et al., 2016).

It is also possible that individuals with higher WHtR were exercising dietary restraint when choosing maximum portion sizes. Dietary restraint describes the intention to restrict or control food intake to reduce body weight (Lowe, 2002). Individuals who report high levels of dietary restraint are found to choose smaller portions of food (Brunstrom, Rogers, et al., 2008; Labbe et al., 2017; Lewis et al., 2015; Spence et al., 2016). Faulkner et al., (2017) found that restrained eaters estimate smaller portion sizes and also reported feeling more guilty about eating standard portions of food compared to unrestrained eaters, suggesting that portion size control is a method restrained eaters use to reduce food intake.

Restrained eating may have influenced the relationship between portion size and adiposity in men and women. In this study, simple analyses (ANOVA) revealed that men chose larger food portions and men also presented with a higher WHtR compared to women. In the regression model, however, WHtR did not predict the portion size of sweetcorn, M&M's, peanuts and chocolate when sex was included as a predictor. This finding suggests sex confounds with WHtR and that the effect of adiposity on portion size differs between men and women. Women with higher WHtR chose smaller portions of energy-dense (peanuts, M&M's and chocolate) and sweet foods (sweetcorn) and these choices may be an expression of restrained eating and controlled food intake due to a higher level of adiposity. Although high dietary restraint is not consistently associated with BMI (Bellisle et al., 2009; Klesges et al., 1991; Provencher et al., 2003; Smith et al., 1998), overall dietary restraint is reported to be higher in women than men (Cornier et al., 2010; Goldfield & Lumb, 2008) and rigid restraint (more stringent approach to controlling food intake) is associated with higher BMI in women, not men (Provencher et al., 2003). Also, Brunstrom and Shakeshaft (2009) highlighted that there might be a restraint towards specific foods (food-specific restraint), particularly foods that are energy-dense and palatable (Lemmens et al., 2010). An interesting study reported that amongst women, restrained eaters were more vulnerable to the context of food exposure than unrestrained eaters. Viewing images of low or high energy-dense foods did not increase subsequent food intake in restrained eaters, relative to unrestrained eaters

(Kemps et al., 2016). The authors proposed that viewing pictures of these foods activated dietary goals for the restrained eaters, such that seeing images of palatable foods reminded of need to restrict these foods, which then resulted in the subsequent control of food intake. In this study, however, I did not measure dietary restraint, so it is difficult to assess the impact on the study outcomes.

Another explanation for the study findings is that individuals with higher adiposity may differ in their perceptions of appetite and attitudes towards foods, which then influenced their decisions about portion size. Factors such as palatability (how much the food is 'liked'), familiarity (how often the food is consumed), energy density (ED) of the food, and the expected feelings of fullness (satiety/satiation) that the food provides influences decisions about portion size (Brogden & Almiron-Roig, 2010; Brunstrom, 2011, 2014). I observed that across all participants, larger portions (or greater weight of food) of low ED foods (peas and sweetcorn), but smaller portions for high ED foods (peanuts, M&M's and chocolate) were chosen. However, it was estimated that the energy by high ED foods (energy provided per average portion size) would provide twice as much energy for the high ED foods in comparison with the low ED foods (High ED: average energy for peanuts, M&M, chocolate = 450.38kcal vs low ED foods: average energy for peas and sweetcorn = 222.87kcal). This finding is in line with the observation that per energy unit, high energy-dense foods provide less satiation and are likely to result in overconsumption (Almiron-roig et al., 2013; Brunstrom, Shakeshaft, et al., 2008; Ello-martin et al., 2005).

Although ED did not appear to influence portion size estimates for individuals with higher adiposity (i.e. the differences between maximum portion size of low and high energy foods were similar between groups), differences in liking and familiarity may play a relatively important role in influencing portion estimates. For instance, overweight individuals and individuals with obesity report consuming fruit and vegetables less frequently (Johnson et al., 2008; Ledikwe et al., 2006; Mendoza et al., 2007), therefore, arguably may be less familiar with the feeling of fullness that a portion of food may provide. Less familiar foods are expected to deliver poor satiation (Brunstrom, Shakeshaft, et al., 2008) and possibly chosen in smaller portion sizes (Keenan et al., 2015b). Familiarity improves with eating, mainly if a food is eaten to fullness (Irvine 2013). Although familiarity was controlled for in the study, foods consumed more regularly (once per week) are expected to provide more satiation than foods eaten less frequently (Brunstrom, Shakeshaft, et al., 2010). It may be that individuals with higher adiposity who consume low energy-dense foods less often are less familiar with the satiation value, and more likely to choose smaller portion sizes. Similarly, lower liking scores could also contribute to foods being chosen in smaller portion sizes (Spence et al., 2016), although the study did not assess liking for or perceived pleasantness of the test foods.

Another possibility that limits interpretation of my findings, highlighted by Labbe and colleagues (2017) is that individuals with higher adiposity may be more responsive to the presence of food, rather than viewing food images. Two recent studies demonstrate that the presence of food has a substantial influence on food evaluations and that compared with lean individuals, overweight individuals and individuals with obesity are susceptible to eating more food when presented with physical form of food than when evaluating food images alone (Medic et al., 2016; Romero et al., 2018). Overweight individuals and individuals with obesity are found to be more responsive to food cues, such as the sight and smell of food (Ferriday & Brunstrom, 2011) and heightened responsiveness that is directly associated with the size of food portions an individual will habitually serve themselves (Tetley et al., 2009). Thus, participants with higher adiposity may have found it easier to apply restraint to portion size when choosing from images rather than real foods.

This study did not find an association between the time since the last meal and maximum portion estimates, despite considerable variation across participants, suggesting that the task was unaffected by concurrent nutritional state. However, we did not measure hunger levels directly, and several studies have demonstrated that it does influence portion size estimates (Brogden et al., 2009; Brogden & Almiron-Roig, 2011; Spence et al., 2016). Moreover, the foods in this study are either eaten commonly as between-meal snacks, perhaps in the absence of high hunger, or as side dishes rather than on their own, which may limit the relevance of portion size to hunger relief.

7.4.3 Individual differences in portion size estimates

Consistent with the findings from other studies, men chose larger portions of food compared to women. It is known that men have a higher fat-free body mass and RMR compared to women, and these physiological differences are often cited as the reason for differences in eating behaviour between sexes (Blundell et al., 2015). However, it is also important to consider social and cultural expectations associated with gender and eating behaviour. For instance, smaller portion sizes are considered more feminine, while larger portion sizes more masculine (Bock & Kanarek, 1995; Chaiken & Pliner, 1987), although recent evidence indicates that the influence of gender stereotyping on portion sizes may be changing (Yantcheva & Brindal, 2013). Conventional beliefs about gender and eating behaviour may have influenced choices about portion size.

In both regression models, older age predicted smaller food portion sizes, meaning that older individuals chose smaller portions of food, a finding which is consistent with studies reporting a decline in food intake with ageing (Wakimoto & Block, 2011).

7.4.4 Limitations

This investigation was limited to five side dish or snack foods due to time constraints, and therefore do not reflect decisions about the portion size of main meals or entrees or reflect food choice across a spectrum of available foods. This study did not control for variables such as dieting, restraint and palatability, which may have influenced the study outcome. More specifically the finding that women with higher WHtR chose smaller portions of food suggests that the measurement of dietary restraint is key to understanding the relationship between excess adiposity and portion size.

Furthermore, body composition was not measured directly, but inferred from weight, height and waist circumference. This study was a cross-sectional survey, and the results cannot imply casualty, i.e. that higher levels of body fat cause individuals to choose smaller portions of food.

Participants for this study were obtained from a sample population of visitors to the Science Museum, London. The ethnicity profile of the sample population closely matched that of the UK population. The sample population was predominantly British, white adults (approximately 85%), which is similar to ethnicity population groups reported in the 2011 UK census (Office of National Statistics, 2018). However, this population would represent individuals who expressed an interested in science education.

7.4.5 Conclusion

This study demonstrated that resting metabolic rate and waist-to-height ratio significantly predicted estimated maximum portion size, while BMI and waist circumference were not associated with estimates. However, it is not clear whether individuals with higher adiposity do select larger portions of food, primarily because it was difficult to assess true estimates of portion size without controlling for dieting, restraint, liking or expected satiety. It may be that individuals with higher adiposity do select larger portions of actual food, however, because other factors such as eating rate, eating frequency and energy density may play an equal role in contributing to increased energy intake and the development of obesity (Herman et al., 2016; Mattes, 2014). This provides further support for the need to use measures of body composition (fat-free body mass, fat mass and resting metabolic rate) in the assessment of appetite and eating behaviour to understand the aetiology of obesity.

Chapter 8: General discussion:

8.1 Summary of findings

The thesis aimed to investigate the biopsychological factors associated with overeating and the development of obesity. These factors were explored under two themes: that the foods now available to humans are extremely palatable, energy-dense, with low protein value (or content), and served in large portion sizes. It was hypothesised that these factors profoundly impact appetite to encourage food intake and are associated with overeating and excess adiposity or obesity. The second theme explored the individual variability in biological, physiological, psychological and socio-economic traits or factors that would increase responsiveness to the obesogenic nature of the food environment. These factors were investigated in experimental intervention studies and larger population-level or cross-sectional studies.

Chapter 3 and 4 investigated the appetite responses to consuming foods containing high levels of fat and sugar. Combinations of fat and sweetness enhanced the palatability and desire to eat on initial tasting and sustained reward responses and feelings of hunger in the early stages of eating. These responses were associated with a higher intake of food.   Restrained eaters exhibited sustained pleasantness and desire to eat during the first part of the meal. Chapter 4 further investigated the postprandial responses in acyl-ghrelin to during palatable food consumption and found that the addition of sweetness to a high-fat food sustained acyl-ghrelin levels in the early stages of eating.

Chapter 5 investigated the individual responses to low protein intake and found that the level of physical activity did not influence the response to a low protein meal as only marginal differences were observed between the active, moderately active and sedentary groups. Across all participants and over both lunch meals body composition and resting metabolic rate was strongly associated with energy and protein intake.

Chapter 6 investigated the biological, psychological, anthropometric and socio-economic factors associated with obesity-related eating behaviours and attitudes toward food in a community-based sample. A heightened desire to eat, weaker satiety response and frequent intake of energy-dense foods are associated with weight gain and obesity. In this sample, individuals who were overweight or obese had the at-risk AA/AT allele and of the lower level of education, showed a greater motivation to eat energy-dense foods and reported eating these foods more often. Furthermore, when viewing images of controlled portions of food, overweight individuals and individuals with obesity reported lower anticipated satiation for energy-dense foods, confirming the findings observed in smaller experimental studies.

Chapter 7 investigated the relationship between portion size and obesity in a community-based sample. When accounting for age and sex, resting metabolic rate and adiposity (waist-to-height ratio) predicted the maximum food portion size chosen for lunch, while body mass index and fat mass did not significantly predict portion size. Individuals with a higher resting metabolic rate chose larger food portions, while a higher waist-to-height ratio predicted smaller portion sizes. And older individuals and women chose significantly smaller portions of food.

8.2 Environmental factors influencing appetite and eating behaviour: palatable, energy-dense, sweet foods heighten eating-related reward processes and undermine appetite control.

The modern food environment provides an abundance of palatable, energy-dense foods that contain high levels of fat and sugar and salt. Studies have demonstrated that food palatability, energy density and portion size are environmental factors that profoundly influence eating behaviour (Ledikwe et al., 2005; McCrory et al., 2006; Rolls, 2018) and evoke a heightened reward response (DiFeliceantonio et al., 2018; Stice, Burger, et al., 2013a). The findings from this thesis suggest that the mechanisms that evoke overeating may involve the following: that combinations of high levels of fat and sugar in foods provide a more palatable and rewarding eating experience than eating high-fat foods alone. The fat-sugar combinations not only enhance the pleasantness of food on first tasting, but these foods also evoke appetite response in the early stages of eating that sustains hunger, motivation to eat and hunger-related acyl ghrelin levels, leading to increased food intake. The work from this thesis largely suggests that it is the taste of the food strongly influences appetite and that the pleasant taste may stimulate orosensory reward processes to encourage food intake. Furthermore, these findings also suggest that fat-sugar combinations may elicit supra-normal appetite responses, because they may exploit the basic human liking for fat and sweet flavours (Drewnowski & Almiron-roig, 2010; Drewnowski et al., 2012). Naturally occurring foods do not contain high levels of fat and sugar or salt, therefore high levels of fat and sugar in food may have a synergistic effect on sensory perception, evoking a more powerful reward response to encourage food intake (DiFeliceantonio et al., 2018; Drewnowski & Greenwood, 1983; Gibson et al., 2008; Stice, Figlewicz, et al., 2013; Valkauskaite

& Gibson, 2010). The work from this thesis does not negate the role of post-ingestive responses in reinforcing food intake, as these processes have been shown to influence reward processing in the brain, particularly in response to the palatable taste of sweetness (Small & DiFeliceantonio, 2019; Thanarajah et al., 2019). Future studies should investigate how the post-ingestive response to combinations of fat and sugar may play a role in reinforcing acute food intake and influencing long-term eating behaviour.

The dissociation of the behavioural components of reward underlies many addictive behaviours and eating disorders and is characterised by a heightened ‘wanting’ or motivational salience independently of a heightened ‘liking’ or pleasantness (Berridge, 2009; Berridge & Robinson, 2016). The findings from this thesis demonstrated that a high-fat, sweet food sustained the motivation to eat while ratings for pleasantness decreased with food consumption. This finding suggests that acute overeating may be driven by a dissociation of reward responses demonstrated by a reduction in the liking or pleasantness of the food, but a sustained desire to eat. However, measuring the behavioural components of reward behaviour, namely ‘liking’ and ‘wanting’, remains a controversial topic in eating behaviour research (Finlayson & Dalton, 2012a; Havermans, 2011). Therefore, future studies should consider how these behaviours are operationalised as the individual’s perception of the sensory cue and associated reward (expected pleasantness) may play a stronger role in directing eating behaviour (Pool et al., 2016).

The sustained postprandial acyl-ghrelin responses with the intake of high-fat, sweet food (Chapter 4) also indicates that these foods alter appetite on a physiological level. Although the acyl-ghrelin responses were not directly linked to the sustained hunger and motivation to eat observed in Chapter 3, the changes in ghrelin may underlie the appetite and reward responses when eating high-fat, sweet food. Alongside its role in stimulating food intake, ghrelin also acts on dopaminergic reward neural circuits to drive the motivational reward processes of eating behaviour (King 2011, Kawahara 2013, Skibicka 2011), and, therefore, has been implicated in reward-based feeding and hyperphagia (Naleid et al., 2005; Wei et al., 2015). Collectively, the findings suggest the consumption of a high-fat, sweet food triggers the release of ghrelin, which then acts to increase hunger and motivational aspects of eating behaviour to drive food intake. These findings present an exciting opportunity for future research to investigate how ghrelin may be involved in acute and chronic overconsumption of high-fat, sweet foods. Furthermore, how postprandial changes in ghrelin also underlie the overconsumption of high-fat, salty or savoury foods (Bolhuis et al., 2016).

The thesis found that consumption of a high-fat, sweet food leads to a higher energy intake relative to an isoenergetic non-sweet food (Chapter 3) and that relative to lower energy-dense food, portions of high energy-dense foods chosen for lunch provide more energy (Chapter 7). These two findings suggest that it is easier to consume more calories from high energy-dense, palatable foods, increasing the likelihood that an individual will overeat. These findings support the suggestion that humans are unable to adapt to the modern, western-style food environment as we are unable to accurately determine the energy content of high energy-dense foods (Brunstrom et al., 2018). Therefore, frequent intake of these foods is likely to lead to overeating, positive energy balance and the development of excess adiposity.

8.3 Individual variation in body composition, metabolism and level of physical activity may influence responses to low protein intake

Chronic consumption of a diet that is highly processed and offers a relatively lower proportion of protein is implicated as a driver of overeating. The work sort to investigate whether the individual variation in body composition, estimated protein needs, and level of physical activity influenced appetite and eating responses to acute protein restriction. However, only minor differences in appetite and food intake were observed between activity groups, suggesting that either protein restriction is unaffected by activity status or that the degree of restriction was not enough to elicit substantial changes in eating behaviour. Although studies have demonstrated that both animals and humans respond to acute periods of protein restriction (Baker et al., 1987; Gibson & Booth, 1986; Gibson et al., 1995; Masic & Yeomans, 2017), the expected appetite responses, such as increased hunger and desire to eat savoury/salty foods, are more consistently observed with longer periods of restriction (Griffioen-Roose et al., 2012, 2014; Martens et al., 2014; Martens et al., 2014). On a wider level, studies show that energy homeostasis is relatively insensitive to acute perturbations in energy balance caused by exercise (Dorling et al., 2018; Schubert et al., 2013). It may be that similar mechanisms operate to regulate protein homeostasis and that perturbations in protein balance need to take place over successive meals or several days to have a substantial effect on eating behaviour.

The experimental findings of chapter 5 may warrant further discussion. Body composition and resting metabolic rate were associated with food intake across both lunch meals. It appeared that the active group, overall, exhibited a greater level of dietary regulation, as energy and protein intake at lunch as the association between body composition, metabolism and food intake was retained were these groups were subdivided according to activity status. These findings were underpowered; however it may suggest that despite protein restriction, active individuals balance food choices and choose the correct amount of food to meet their nutritional needs. This behaviour may occur with a greater level of accuracy in comparison with those who are moderately active and or sedentary, which is consistent with the recently updated perspective on physical activity and appetite control (Beaulieu et al., 2018). This finding does not exclude the possibility that active individuals present with a greater amino acid reserve that serves to buffer the effect of a single low protein meal (Poortmans et al., 2012).

In the active group, FFM and RMR were associated with protein intake, which indicates that fat-free mass exerts an influence on appetite mechanisms to direct both energy and protein intake, consistent with the Protein-stat model proposed by Millward (1995, 1997). The observation that this behaviour was evident only in the active group further suggests that physical activity heightens awareness for both energy and macronutrient requirements, such as dietary protein. Rationally, an individual with more lean tissue will require a greater amount of protein to support growth and maintenance. Therefore regular physical activity, particularly of a high intensity, serves to heighten appetite control to ensure nutritional requirements are met (Beaulieu et al., 2016; Blundell, 2011). However, it is important to consider that the activity groups were not matched for sex and therefore future studies will need to confirm this suggestion.

On a broader level, these findings may be relevant to overweight individuals and individuals with obesity who do present with greater levels of fat-free body mass, a higher resting metabolic rate, yet increased adiposity and increased sedentary behaviour (Oussaada et al., 2019; Shields & Tremblay, 2008; Blundell et al., 2012, 2015). A higher level of body fat may weaken appetite control (Blundell, 2018), while sedentary behaviour may increase the susceptibility to appetite dysregulation, or overeating fat-rich, carbohydrate-rich foods (Beaulieu et al., 2018). This interaction may become more acute when protein intake is restricted, such that a greater level of appetite dysregulation occurs with protein deprivation in moderately active, or less active individuals, and individuals consume more energy from non-protein foods.

8.4 Is obesity associated with choosing larger portions of food?

The provision of large food portions is another key factor implicated in the development and maintenance of obesity. However, because studies have not observed a consistent relationship between body mass index and portion size, this thesis posited that another index of adiposity, the waist-to-height ratio, would predict a relationship between excess adiposity and portion size. Contrary to the study hypothesis, however, in a large community sample, individuals with a higher WHtR choose smaller portions of food. Furthermore, when accounting for age and resting metabolic rate, WHtR predicted smaller portion sizes across the study sample. Despite the findings of the study, it cannot be concluded that increased adiposity is not associated with choosing larger portions of food. Overweight individuals and individuals with obesity are proposed to have higher metabolic requirements because of higher body weight and the support of increase adipose tissue (Oussaada et al., 2019; Schutz et al., 2002). Also, those with higher metabolic requirements exhibit higher levels of hunger, which would drive an increase in food intake (Blundell et al., 2012; Caudwell et al., 2013). Furthermore, numerous cross-sectional studies have shown that overweight and obesity are associated with consuming a higher energy intake (Austin et al., 2011; Ford & Dietz, 2016; Kant & Graubard, 2005;

Scarborough et al., 2011; Stubbs et al., 2004; Yancy et al., 2013). It is likely that the increased energy intake is caused either by consuming larger portions of food or eating more frequently (increased meal frequency of eating occasions) (Mattes, 2014). However, it must also be considered that the study was limited to five side dish or snack foods and therefore do not reflect decisions about the portion size of main meals or entrees (discussed further in 'Strengths and Limitations' subsection below). Consideration must be given for the fact that the investigation took place in a public setting. Therefore, factors such as social desirability, dietary underreporting and dietary restraint may have influenced the decisions about portion size in the study sample. Underreporting of energy intake is commonly reported in nutrition research (Livingstone & Black, 2003) and is particularly prevalent amongst overweight individuals and individuals with obesity (Heitmann & Lissner, 1995; Kelly, Rennie, et al., 2009). Furthermore, overweight individuals and individuals with obesity may choose a smaller portion of food that is deemed to be socially acceptable, over a portion of food they would serve themselves (Lewis et al., 2015).

The findings from Chapter 7 observed found that individuals with greater metabolic needs (higher resting metabolic rate) chose larger portions of food, while increased adiposity predicted choosing a smaller portion of food. Similarly, Chapter 5 observed that resting metabolic rate and fat-free mass predicted energy intake in physically active individuals. Collectively, these findings support the proposal that the components of energy expenditure exert a stimulatory effect on food intake and eating behaviour, while fat mass exerts an inhibitory effect on food intake (Blundell, 2018). These findings have important implications for future studies as it is important to include components of energy expenditure (resting metabolic rate and fat-free body mass) when assessing eating behaviour. Consideration may be given for the role these components play in reflecting either the excitatory or inhibitory aspects of eating behaviour. As such resting metabolic rate and fat-free body mass may serves as predictors of factors related to meal initiation (decisions about portion size, hunger, prospective food intake, desire to eat, desire for specific flavours etc.), while fat mass may be used to predict behaviours relating to meal termination (fullness, sensory-specific satiety, satiation and satiety (Hopkins et al., 2017).

8.5 Individual variation in eating styles, eating behaviours and personality traits influence the response to palatable foods

The individual variation in eating styles, eating behaviours and personality traits may predict how an individual will respond to palatable food cues. In this thesis, restrained eaters were particularly susceptible to the taste of palatable food, as they demonstrated a sustained reward response in the early stages of eating a high-fat, sweet food (Chapter 3). Although these responses did not lead to increased energy intake, these responses may predict a susceptibility or likelihood of overeating in the future. Restrained eaters face a perpetual conflict between the enjoyment of eating palatable food and the goal of attaining the desired body weight. Restrained eaters are characteristically overresponsive to palatable food cues (Burger & Stice, 2011; Fedoroff et al., 2003; Houben et al., 2010; Wang et al., 2016) and demonstrate a high degree of cognitive control to suppress heightened responses (Houben et al., 2012; Wang et al., 2016). This may explain why restrained eaters are prone to disinhibition, overeating and weight gain (Chaput et al., 2009; Drapeau et al., 2003; Snoek et al., 2013; Stice et al., 1999), because the effort required to suppress urges to overeat are easily undermined, and led to overeating.

In the thesis, other eating styles (emotional eating, disinhibition), propensity towards hedonic hunger (Power of Food Scale), and personality traits (sensitivity to reward and impulsivity) did not predict a greater reward response palatable food, alterations in postprandial acyl-ghrelin responses or increased food intake (Chapter 3 & 4). Sensitivity to reward did not predict reward responses when viewing pictures of palatable, energy-dense foods (Chapter 6). It is important to note that the absence of a finding does not indicate that individuals who exhibit these eating styles or personality traits are less susceptibility to palatable foods cues. As numerous studies have observed a strong relationship between sensitivity to reward, impulsivity, overeating energy-dense foods and development of obesity (Davis, 2009; Davis et al., 2007; Loxton & Tipman, 2017; Morris et al., 2015). In particular high levels of emotional eating, sensitivity to reward and impulsivity often predict the preference for food rich in sugar and fat (Davis et al., 2007; Gibson, 2012; Meule et al., 2014). It is acknowledged, particularly in the case of reward sensitivity, that global measures of reward sensitivity may not predict reward responses to specific foods, i.e. that individuals find some foods more rewarding than others (Stephens et al., 2010). Evidence for food-specific reward has been reported in individuals with obesity and those with binge eating disorder (Loxton, 2018). An individual who exhibits these behaviour and personality traits may still find energy-dense, palatable foods more rewarding, yet further studies are encouraged to examine the food-specific reward responses to palatable food cues.

8.6 The individual variation in age, sex, weight status and inheritable genetic traits predicts susceptibility to palatable food cues

Groups of individuals may be more susceptible to food than others. The thesis investigated the motivational and appetite aspects of eating behaviour across a sample population. The results at a population level largely reflected findings reported from laboratory-based studies: overweight individuals and individuals with obesity, who carry the at-risk AA/AT allele of the *FTO* gene, and have a lower level of education show an increased motivation and preference for high energy-dense foods, yet find these foods less satisfying (Chapter 6). This means that the theoretical concepts of motivational processes relating to food choice and eating are measurable and valid for a community sample of free-living adults.

8.6.1 Weight status and FTO polymorphism alleles AA/AT/TT

The heightened reward response and reduced satiety/increased satiation in OWOB has been reported in experimental studies (Epstein et al., 2012; Ferriday & Brunstrom, 2011; Finlayson et al., 2012; French et al., 2012; French et al., 2014). An individual who exhibits these behaviours maybe at greater risk of overeating because they gain more pleasure from eating and are more likely to eat for hedonic reasons than for reasons relating to energy homeostasis (Blundell & Finlayson, 2004). Similarly, weaker satiety with eating means OWOB may be more likely to consume more food or eat more frequently to achieve a level of fullness or food satisfaction (Delgado-Aros et al., 2004; Meyer-Gerspach et al., 2014). The thesis observed similar appetitive behaviour in individuals with AA/AT allele of the *FTO* gene at rs9939609; these individuals showed an increase preference for high-fat sweet foods, like ice-cream. Although the study cannot confer causality, it may be that an increased motivation to eat underlies the increased intake of high energy-dense foods observed in these individuals (Tanofsky-Kraff et al., 2009). Certainly, individuals with a higher proportion of at-risk obesity-related genetic polymorphisms are found to exhibit high levels of disinhibition and susceptibility to hunger (Jacob et al., 2018). This suggests that the genetic contribution towards the variability in body weight may be mediated in part by eating behaviour traits. It is noted, however, that not all individuals with the ‘at-risk’ AA/AT allele presented with obesity, which suggests several behaviour traits interact with genetic traits to increase susceptibility to palatable food cues.

8.6.2 Age and sex

This thesis observed that the age- and sex-related differences in eating behaviour traits are consistent with the variations in dietary patterns reported in cross-sectional studies. Older individuals chose smaller portions of food, preferred lower energy-dense foods and reported eating high energy-dense foods less frequently compared to younger. Although there was a greater proportion of older individuals who were overweight or obese, the preferences observed in the younger individuals (for instance, a greater motivation to eat and more frequent intake of energy-dense foods), may represent a susceptibility to overeating and weight gain in the future if continued to later stages of adulthood.

The sex-related differences in food preferences and food portion sizes have been reported in several cross-sectional studies (Knudsen et al., 2014; Mikkilä et al., 2005; Pinto de Souza Fernandes et al., 2017; Wardle et al., 2004). The findings from Chapter 6 and 7 observed that men chose larger portions of food and show a motivation to eat high-fat savoury foods like beef steak. Women chose smaller food portions yet demonstrate a greater desire to eat high-fat sweet foods and report eating them more frequently. The sex-related differences in food preferences and portion sizes may shape attitudes toward future food choices and may confer a susceptibility toward overeating specific foods. These findings are particularly relevant for creating targeted approaches to obesity treatment and prevention.

8.6.3 Socio-economic status

The increased availability of cheap, processed food, increased proximity to fast food outlets and to increase food disparity are some the key factors implicated for increased obesity rates in low socioeconomic communities (Drewnowski & Darmon, 2005a; Jeffery et al., 2006; Maguire et al., 2015; Reidpath et al., 2002; Taveras et al., 2005). The findings of this thesis provide a further contribution by demonstrating that there are behavioural traits that may predispose these individuals to overeat. The thesis found that individuals of a lower level of education showed an increased motivation to consume energy-dense foods, like a hotdog, yet perceive healthier foods, like salmon, to be less filling. Overall, this suggests that since education level is a proxy for socio-economic status, individuals of lower socioeconomic status are likely to choose high energy-dense foods because they find them more appealing and consume them more frequently, yet likely to avoid healthier foods as they find them less satisfying. It will be important to examine these findings in more detail using established psychometric tools. These insights provide an important understanding of the attitudes and behaviours are shaping eating habits in this population group. While the predominance of research has focused on investigating the environmental drivers of overeating (Jeffery et al., 2006; Larson et al., 2009; Mason et al., 2018; Riva et al., 2009), these findings highlight the role of psychological and behavioural factors that underlie food choices. Recently several qualitative studies have shown that social and psychological factors, such as social constructs relating to masculinity or femininity, nutrition knowledge, food awareness, cooking skills and attitudes toward healthy eating and exercise contribute towards food choices in individuals of a low socioeconomic status (Daborn et al., 2005; Romeike et al., 2016; Stephens et al., 2018). Understanding the relative contribution of the psychological and behavioural susceptibilities is not only important in understanding the specific pathways of overeating in this population group, but also in the development of effective intervention strategies that will address behaviour change.

8.7 Strengths and Limitations

This thesis investigated the biopsychological factors of overeating on a broad level using experimental and population-level studies in adults under free-living conditions. The methodological approach of this thesis has several strengths and limitations which influence the generalisability of the findings. This section provides a discussion of the general limitations relating to laboratory and field-based studies, followed by a discussion of the specific limitations relating to each thesis chapter.

Eating behaviour is a learnt response to environmental cues and therefore is guided both the physiological processes of taste, ingestion and assimilation, and non-automatic, cognitive processes such beliefs, perceptions and attitudes toward food. These factors will influence the measurement of eating behaviour in a laboratory or field setting (Brunstrom, 2005). On a general level, measuring food intake and eating behaviour for a single meal or at a single time point may not reflect habitual eating practices and behaviours. Therefore, one is limited to the generalisability of the findings. Many of the studies in this thesis require the individual to report their appetite and food preferences, and this requires an individual to have a degree of interoceptive awareness (Stevenson et al., 2015); in other words an awareness of their internal state and of subjective perception of physiological cues that relate to hunger, fullness, and satiety. Although eating is a repetitive and automatic behaviour, the use of Visual Analog Scales, which are commonly used to obtain an objective assessment of appetite sensations, may be limited by the participants understanding of them or, as discussed previously, the ability to evaluate appetite sensations (Lesdéma et al., 2016). Although the thesis investigated the association of several eating styles, eating behaviour traits and personality traits associated with overeating, there are other psychological and behavioural traits that were not measured that may have impacted the outcome of the thesis.

Experimental investigations that take place in a laboratory setting provide the investigator with an opportunity to control for extraneous variables that may impact the outcome of the study. However, a laboratory is an unnatural setting and may alter intake and food preference, thereby reducing the ability to capture authentic eating behaviour (Best et al., 2018). The setting itself may reduce the motivation and desire to eat (Best & Papies, 2017). The strength of the thesis was that the experimental studies reported in chapters 3, 4 and 5 followed the guidelines proposed for the more rigorous measurement of ingestive behaviour (Hetherington & Rolls, 2018). It is also acknowledged that in a controlled laboratory setting, there may be interactions between tester and participant or attributes relating to the participant that can vary the response to experimental investigations (Stubbs & Finlayson, 2018). The findings from a single meal or short term studies must be considered in light of longer-term or longitudinal studies to understanding the effect on eating behaviour and weight gain (Berthoud et al., 2011; Berthoud et al., 2017)

Cross-sectional investigations take place at a single time point, and therefore, the outcome may only represent eating behaviours in part, and the outcome cannot infer causality. In a public setting, several extraneous variables would influence the study outcome, such as time of day, emotional state, presence of families or large crowds (some presented with families, others were alone). A degree of dietary misreporting may have influenced participant responses, and this behaviour may have been more prevalent in weight-conscious individuals.

Limitations and strengths specific to each study are discussed in relevant chapter 3-7. To summarise: In Chapter 3 and 4, the strength of the study was that the comparison of a high-fat, sweet and non-sweet food allowed comparison of appetite mechanisms that occur through the eating process. However, in both studies there were several limitations that are important to discuss. The study design did not include a low-fat control as the design closely followed that of previous experiments undertaken in our laboratory (Gibson et al., 2008; Valkauskaite & Gibson, 2010). However, including a low-fat control would have determined the interaction between the taste of fat and sweetness in influencing appetite and eating behaviour, rather than the addition of sweetness alone.

The findings of both studies were underpowered to detect a medium effect size between the two experimental conditions for food intake, appetite ratings and changes in acyl ghrelin levels. Furthermore, the analyses did not control for multiple comparisons. Accordingly, future research directives would be to replicate the study in larger samples and include a low-fat control.

In chapter 3, study cannot infer overconsumption as an increased food intake of the high-fat, sweet condition at a single occasion may not reflect a risk factor for weight gain. An individual may initiate compensatory behaviours to mitigate the effects of high energy intake. Therefore, overconsumption is more accurately expressed as an increased intake that occurs over several eating occasions or overtime, where a higher energy intake occurs over and above the individual's energy requirements (Fay et al., 2012).

In Chapter 4 only one appetite hormone was measured, and the characterisation of postprandial responses of satiety hormones such as Peptide YY (PYY), Glucagon-like peptide-1 (GLP-1), Cholecystokinin (CCK) would have provided a clearer picture of the overall appetite responses with ingestion of high-fat sweet food. Furthermore, the hormones responses were measured over a short period

(60 minutes), but future studies would benefit in a longer post-prandial assessment to show responses as they relate to satiation, satiety and initiation of next meal (Chapter 3).

Several limitations were found for study reported in Chapter 5. The study design employed a single protein-restricted meal; however the protocol did not elicit the expected appetite and food intake responses across the sample population, nor in groups stratified for level of physical activity. It was concluded that a single low protein meal may not have created enough of a protein deficit to elicit a meaningful change in eating behaviour. However, consideration must be made for the small sample sizes and lack of heterogeneity between activity groups, as these factors could have contributed to the variability in ingestive response. Future research should consider restriction protein intake over several meals or days and also consider assessing habitual protein intake and overall dietary intake (for instance data collected using a dietary recall or food diary method), as food choices may be influenced by habitual intake (Masic & Yeomans, 2017). Also, the objective assessment of physical activity, for instance wearing an accelerometer would have provided a more accurate assessment of habitual physical activity and sedentary behaviour.

A strength of the studies reported for Chapter 6 and 7 was that the sample population could be considered a representation of the wider UK population. The sample population was compromised of predominantly British, white adults (approximately 85%), which is similar to ethnicity population groups reported in the 2011 UK census (Office of National Statistics, 2018). A limitation reported for Chapter 6 is that the food image questionnaire used for the study was not validated before testing. This was due to restrictions in the time each participant could engage in the study. However, the food images used in the questionnaire were validated for use in ingestive behaviour research (Blechert et al., 2014). Similarly, the questionnaire was limited to ten different foods, and the participant responses may reflect the attitudes towards specific foods, rather than categories of foods (high energy-dense, low energy-dense, sweet or savoury foods).

A strength of the study reported in Chapter 7 was the use of an established psychometric tool to assess expectations about portion size in adults (Brunstrom, Shakeshaft, et al., 2008). However, a limitation of the study is that there were only five snack foods or side dishes chosen for the study. As with the limitations of chapter 6, the participants were restricted for time. Factors related to food liking may have played an important role in driving responses to chosen portion size. The study did not control for eating behaviours or traits (such as restrained eating/dieting) that may have influenced decisions about portion size (Brunstrom, 2014).

8.8 Summary and Implication of findings

The findings of this thesis indicate that the environmental factors associated with a Western-style diet may play a role in influencing appetite and eating behaviour. Palatable, high energy-dense foods stimulate the sensory, appetite and reward responses to provide a pleasurable eating experience, and this may encourage further eating. Individuals find it difficult to accurately assess the energy content of these foods, making it more likely that they will consume too many calories. Importantly, the thesis observed that individuals do not respond to the obesogenic nature of the food environment in the same way. Decisions about portion size, energy intake and macronutrient balance are influenced more strongly by an individual's body composition and metabolic rate, rather than body fat or body mass index. Individuals who exercise dietary restraint, are overweight or obese or have inherited the obesity-related AA/AT allele show a heightened reward response to palatable food cues, meaning these individuals may be more susceptible to overeating. Individuals of lower socioeconomic status also show an increased motivation towards high energy-dense foods, yet find healthier foods to be less satisfying, which indicates that attitudes toward food may be shaping future food choices.

The work from this thesis contributes to a wider framework investigating the environmental determinants of appetite and eating behaviour and their role in the development of obesity. There is a growing interest in how the chronic intake of ultra- or highly-processed foods influence eating behaviour, overall health and mortality and obesity risk (Hall et al., 2019; Lawrence & Baker, 2019; Sjöblad, 2019). Although the level of food processing per se is not a primary contributing factor (as many nutritionally dense foods may also be highly processed) (Ludwig et al., 2019), these foods offer little nutritional value. They are stripped of micronutrients, protein and dietary fibre that would otherwise confer a nutritional benefit to the consumer (Steele et al., 2017; Monteiro et al., 2017), however, the clever combinations of high levels of fat and flavour (sugar and salt) offer an extremely palatable food product. The work from this thesis suggests that the unnaturally high levels of fat and sugar in food evoke supra-normal responses in appetite and food-reward processes that promote acute feeding behaviour. Furthermore, the enhanced palatability appears to be acting primarily on processes involved in the cephalic phase of eating, suggesting that taste and early sensory responses act to reinforce food intake. However, it is possible that both the taste and post-ingestive responses have a reinforcing effect on feeding behaviour (Small & DiFeliceantonio, 2019; Thanarajah et al., 2019). Therefore, future studies need to understand the role that ingestive processes play in reinforcing the consumption of fat-rich, sweetened foods, and the interplay between taste and ingestion that serves to strengthen a chronic intake of these foods.

This thesis also investigated the role of two other environmental determinants of overeating, namely protein leverage (restriction) and large portion sizes, and their association with overeating and excess adiposity. Although this thesis did not provide conclusive evidence that protein restriction influences eating behaviour, or that excess adiposity is associated with choosing larger portions of food, these environmental factors may still play a key role in promoting overeating. It has been acknowledged that protein leverage is incomplete, as inconsistent responses to appetite and feeding have been observed with various levels of dietary protein intake (Hall, 2019), yet a compromised intake of dietary protein may still play a primary role in the development of obesity (Gosby et al., 2014). Similarly, large food portion sizes may still contribute to the development of obesity (Young & Nestle, 2012) although the understanding of the relative contribution of increased meal frequency is also important (Mattes, 2014). One important implication for future research is that studies should consider including components of energy expenditure (fat-free body mass, daily energy expenditure, resting metabolic rate) as independent variables predicting eating behaviour. The work from this thesis confirms the observation from others that these indices serve as a valid and informative measure of eating behaviour (Blundell, 2018; Hopkins et al., 2016; McNeil et al., 2017).

The individual variation in response to a palatable food environment has important implications for guiding future research studies. Recently studies have characterised other eating behaviour traits associated with overeating, such as loss of control over eating (Latner et al., 2014), reward-based eating (Mason et al., 2017) and food cravings (Nijs et al., 2007), therefore individuals who exhibit these behaviours may also be more responsive to palatable food cues. Including these newer psychometric tools in investigations may provide valuable insight into the individual susceptibility to the obesogenic food environment. Furthermore, Vainik et al. (2019) suggest that many of the obesity-associated eating behaviours are related and broadly describe a single construct of uncontrolled eating. Their work identifies an uncontrolled eating phenotype: where components of body mass index, food intake, personality traits and neural responses are linked. Others have also identified specific phenotypes weak satiety phenotype (Drapeau et al., 2013a); high-fat phenotype (Blundell et al., 2005). The work from this thesis may be used to characterise how susceptible a specific phenotype is to palatable food cues (in other words, whether food cues provoke overeating in distinct phenotypes). The work from this thesis may also be useful in designing a targeted approach for obesity treatment and prevention. For instance, a restrained eater may benefit from a strategy that reduces exposure to palatable food cues.

While it is beyond the scope of this thesis to provide recommendations for public health policy, the knowledge gained from this thesis will contribute to a broader debate on issues of public health. The Food and Drink industry has been heavily criticised for their role in the development of the obesity epidemic (Monteiro & Cannon, 2019; Moodie et al., 2013) and failing to take a responsible role to support public health programmes and policies (The Lancet, 2011). Up until this point, the food and drink industry has profited enormously from favourable economic policies and free trade agreements allowing for the production and global distribution of their products (Stuckler et al., 2012). It is estimated that the industry has generated over $90 billion US dollars in products sales. Nestle’s profit increased by 42% to reach $10.3 billion US dollars (Forbes, 2019). Much of the growth in profit has come directly from increased sales in low-income and middle-income countries (Moodie et al., 2013).

Historically, governments have been reluctant impose strict regulations on the food and drink industry, instead have encouraged industry to self-regulate and engage in voluntary pledges to improve public health (Bryden et al., 2013; Durand et al., 2015; Panjwani & Caraher, 2014). As good as these pledges sound, they have yielded little real action (Bauman et al., 2019; Knai et al., 2015). Instead, the government has been heavily criticised for allowing the industry to have input to policy development and it has been revealed that, apart from profit motives, the industry seeks to influence policies to reduce the likelihood of stricter regulation (Durand et al., 2015; Flint & Oliver, 2019). Of greater concern is that the food, drink and alcohol industry have been found to use unethical strategies, such as lobbying politicians and public health officials, to dissuade industrial regulation (Monteiro & Cannon, 2019; Moodie et al., 2013).

The primary argument used by industry and government is that individuals should be free to make their own food choices and it's up to them to make the right ones (Brownell et al., 2010; Kent, 2009), however the sheer scale of the obesity epidemic implores abandonment of a such a simplified view of obesity. There have been calls to reframe our understanding of the drivers of the obesity epidemic and the level of personal responsibility in its development (Roberto et al., 2015). The finding from this thesis, together with observations from other research studies, primarily suggests that humans are unable to adapt to a highly palatable, energy-dense food environment fully; these foods are very appealing, available in abundance and challenging to resist. Eating them provides an immensely rewarding eating experience, which then encourages further consumption. While individual variability may predict susceptibility to overeating, the scale of the obesity crisis suggests that these environmental factors exploit the normal biological, physiological, psychological processes to encourage overeating and excess adiposity, exerting influencing on a population level, and not just the chosen few.

A political declaration at the UN high-level meeting on non-communicable diseases (UN General Assembly, 2011) and recommendations from the World Health Organization's Commission to Ending Childhood Obesity (World Health Organisation, 2017) has urged governments to recognise their responsibility in reducing the obesity risk. This responsibility would involve stricter regulations on the manufacturing, production and distribution of high energy-dense palatable food products (Monteiro & Cannon, 2019). Furthermore, work is needed to encourage the reduction in food portion sizes, food reformulations and reducing exposure to the consumer (Hetherington et al., 2018; Marteau et al., 2015).

The obesity epidemic and the associated health concerns present a substantial burden to countries across the globe. Given the prevalence of obesity remains high in developed countries, yet increasing in more vulnerable communities and population groups, there is an urgent need to prioritise obesity treatment and prevention strategies. A greater understanding of the environmental drivers of overeating and the individual susceptibility to the environment can be used to develop a more targeted approach to reducing the prevalence of the obesity condition.

Appendix A: Experimental measures

A1: BIS-BAS Scales **(Carver & White, 1994)**

Response options: Very false for me, Somewhat false for me, Somewhat true for me, Very true for me

1. A person's family is the most important thing in life.
2. Even if something bad is about to happen to me, I rarely experience fear or nervousness.
3. I go out of my way to get things I want.
4. When I'm doing well at something I love to keep at it.
5. I'm always willing to try something new if I think it will be fun.
6. How I dress is very important to me.
7. When I get something I want, I feel excited and energized.
8. Criticism or scolding hurts me quite a bit.
9. When I want something I usually go all-out to get it.
10. I will often do things for no other reason than that they might be fun.
11. It's hard for me to find the time to do things such as get a haircut.
12. If I see a chance to get something I want I move on it right away.
13. I feel pretty worried or upset when I think or know somebody is angry at me.
14. When I see an opportunity for something I like I get excited right away.
15. I often act on the spur of the moment.
16. If I think something unpleasant is going to happen I usually get pretty "worked up."
17. I often wonder why people act the way they do. 18. When good things happen to me, it affects me strongly
18. When good things happen to me, it affects me strongly.

19. I feel worried when I think I have done poorly at something important.

20. I crave excitement and new sensations.

21. When I go after something I use a “no holds barred” approach.

22. I have very few fears compared to my friends.

23. It would excite me to win a contest.

24. I worry about making mistakes.

A2: Three Factor Eating Questionnaire R-18 (Cappelleri, Bushmakin, Gerber, Leidy, Sexton, Lowe, et al., 2009)

Response options: definitely true, mostly true, mostly false, definitely false

1. I deliberately take small helpings as a means of controlling my weight.
2. I start to eat when I feel anxious.
3. Sometimes when I start eating I just can't seem to stop.
4. When I feel sad I often eat too much.
5. I do not eat some foods because they make me fat
6. Being with someone who is eating often makes me want to eat also.
7. When I feel tense or 'wound up' I often feel I need to eat.
8. I often get so hungry that my stomach feels like a bottomless pit.
9. I'm always so hungry that it's hard for me to stop eating before finishing all of the food on my plate.
10. When I feel lonely I console myself by eating.
11. I consciously hold back on how much I eat at meals to keep from gaining weight.
12. When I smell or see a really tasty, savoury food, I find it very difficult to keep from eating - even if I've just finished a meal.
13. I'm always hungry enough to eat at any time.
14. If I feel nervous I try to calm down by eating.

15. When I see something that looks very delicious, I often get so hungry that I have to eat right away.

16. When I feel depressed I want to eat.

Response options Never, rarely, sometimes, at least once a week

17. Do you go on binges even though you are not hungry?

Response options Only at mealtimes, sometimes between meals, often between meals, almost always

18. How often do you feel hungry?

A3: Power of Food scale (Michael R. Lowe et al., 2009)

Response options: I don't agree at all, I agree a little, I agree somewhat, I agree, I strongly agree

1. I find myself thinking about food even when I'm not physically hungry.
2. I get more pleasure from eating than I do from almost anything else
3. If I see or smell a food I like, I get a powerful urge to have some.
4. When I'm around a fattening food I love, it's hard to stop myself from at least tasting it.
5. It's scary to think of the power that food has over me.
6. When I know a delicious food is available, I can't help myself from thinking about having some.
7. I love the taste of certain foods so much that I can't avoid eating them even if they're bad for me.
8. Just before I taste a favourite food, I feel intense anticipation
9. When I eat delicious food I focus a lot on how good it tastes.
10. Sometimes, when I'm doing everyday activities, I get an urge to eat 'out of the blue' (for no apparent reason).
11. I think I enjoy eating a lot more than most people.

12. Hearing someone describe a great meal makes me really want to have something to eat.

13. It seems like I have food on my mind a lot.

14. It's very important to me that the food I eat are as delicious as possible.

15. Before I eat a favorite food my mouth tends to flood with saliva.

A4: Barratt Impulsivity Scale (A. Meule et al., 2015)

Response options: Rarely/Never, Sometimes, Often, Almost Always

1. I act on impulse.
2. I act on the spur of the moment.
3. I do things without thinking.
4. I say things without thinking.
5. I buy things on impulse.
6. I plan for job security.
7. I plan for the future.
8. I save regularly.
9. I plan tasks carefully.
10. I am a careful thinker.
11. I am restless at lectures or talks.
12. I squirm at plays or lectures.
13. I concentrate easily.
14. I don’t pay attention.
15. I am easily bored solving thought problems.

A5: Positive and Negative Affect Scores (PANAS) (Watson, Clark and Tellegen, 1988)

This scale consists of a number of words that describe different feelings and emotions. Read each item and then shade in a box in the appropriate column, next to that word, which indicates to what extent you feel this way right now, that is, at the present moment. Responses include: Very slightly or not at all, a little, moderately, quite a bit, extremely

1. Interested
2. Disinterested
3. Excited
4. Upset
5. Strong
6. Guilty
7. Scared
8. Hostile
9. Enthusiastic
10. Proud
11. Irritable
12. Alert
13. Ashamed
14. Inspired
15. Nervous
16. Determined
17. Attentive
18. Jittery
19. Active
20. Afraid

Appendix B: Sussex Ingestion Pattern Monitor (SIPM) version 2.0

B1: Screen prompts, appetite and sensory ratings

‘Today we will serve you rice pudding and you are welcome to eat as much or little as you like. While eating or rating, it is essential that you do no leave your spoon in the bowl at any time: if you want to put your spoon down, please use the small plate provided. Please also do not lean on the placemat. Occasionally, you may be asked to call you experimenter who will provide more food. If you have any questions, please call the experimenter now, otherwise click on ‘Start’ to begin.’

‘At this point in the study we simply need you to complete 3 ratings of your appetite. Each rating is on a simple line scale. Please take care to read the question and then scale your response between the two extreme points by moving the slide bar left or right. Click on ‘Start Rating’ button when you are ready to start.’

‘On the scale above, please indicate, by moving a vertical mark’

‘How full do you feel right now?’

‘How hungry do you feel right now?’

‘How sick do you feel right now?’

Sensory ratings: ‘In this part of the study we would like you to make a number of sensory ratings. In order to start this procedure, you will be asked to call you experimenter who will provide a bowl of rice pudding for you to taste and evaluate. All the evaluations are completed on the simple rating scales: use the mouse or touch pen to adjust the scale to the answer which best fits your evaluation. Please call your experimenter now for a bowl of rice pudding. When a bowl of rice pudding is served, please click on ‘Start’.’

‘Please taste on or two spoonfuls of rice pudding provided and then click on ‘Start Sensory Ratings’. WHILE RATINGS: Please do not lean on the placemat or leave your spoon in the bowl at any time: if you want to put your spoon down, please use the small plate provided.'

'On the scale, please indicate, by moving a horizontal mark, how SWEET/CREAMY the rice pudding tastes to you. N.B. Use the scale to indicate how INTENSE THE SWEET/CREAMY TASTE is relative to sensations you has experience of any kind, not just taste (e.g. pain, noise, etc.).'

'How close the SWEETNESS of the rice pudding tastes to your ideal sweetness in rice pudding.

'How CREAMY the rice pudding tastes to you.

'On the scale (above), please indicate, by moving a vertical mark, how close the SWEET?CREAMY of the rice pudding tastes to your ideal sweetness/creaminess in rice pudding.'

'How much PERCENT fat (by weight) do you think this rice pudding contains?'

'This completes the sensory ratings. Please call your experimenter now: they will provide another bowl of rice pudding for you to eat as much as you like at your own speed. While eating or rating, it is essential that you do not lean on the placemat, lift the bowl up or leave your spoon in the bowl at any time: if you want to put your spoon down, please use the small plate provided. Please do no click on the button below – it is for experimenter only'

Starting Eating Stage: 'In this part of the study please eat as much as you like at your own speed and then click on the 'Meal finished' when you have finished your meal. Click on 'Meal Finished' ONLY when you are sure you have had enough food.'

Within meal ratings ‘PLEASE PAUSE EATING, complete some ratings and then resume eating again. Please remember while rating: Do no lean on the placemat, lift the bowl up or leave your spoon in the bowl at any time: if you want to put your spoon down, please use the small plate provided. Please click on ‘Start Ratings’. On the scale above, please indicate, by moving a vertical mark.’

‘How much do you want to eat a spoonful right now?’

‘How hungry do you feel right now?

‘How pleasant do you find the taste of this rice pudding right now?

Stop eating

‘Please stop eating’

After meal ratings: ‘At this point in the study we simply need you to complete 3 ratings of your appetite. Each rating is on a simple line scale. Please take care to read the question and then scale your response between the two extreme points by moving the slide bar left or right. Click on ‘Start Ratings’ button when you are ready to start. On the scale above, please indicate, by moving a vertical mark’

‘How full do you feel right now?’

‘How sick do you feel right now?’

‘When you finish, click on ‘Completed’. Experiment is now completed. Please do not click on the button below – it is for experimenter only. Call your experimenter now.’

B2: SIPM VAS

B3: SIPM gLMS

B3: Image of rice pudding meal

Appendix C: Nutritional information for lunch buffet (Chapter 5)

C1: Nutritional information for food items served to participants in a buffet-style lunch

Table 5.2:
Nutritional information for food items served for lunch buffet (per 100 g and per portion)

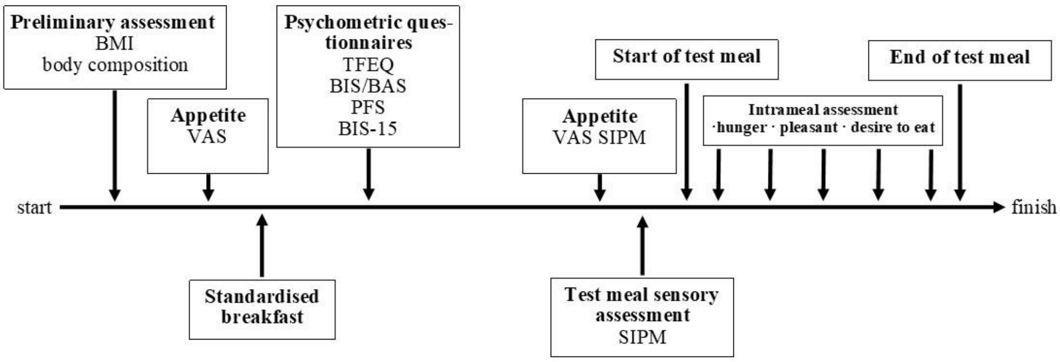
Food item	Energy (kJ)	Energy (kcal)	Fat (g)	Saturated fat (g)	Carbohydrate (g)	Sugars (g)	Fibre (g)	Protein (g)	Salt (g)	portion (g)	protein (g)	PE%
Chicken	477.0	113.0	1.6	0.5	0.5	0.5	0.8	23.9	0.5	80.0	19.1	83%
Tuna	478.0	113.0	0.5	0.1	0.5	0.5	0.5	27.0	0.8	60.0	16.2	98%
Meatballs	1000.0	240.0	17.0	7.0	8.5	1.3	1.0	12.0	1.4	100.0	12.0	21%
Yoghurt	341.0	80.0	0.2	0.1	11.6	10.9	0.2	7.9	0.1	150.0	11.9	40%
Pasta	733.0	173.0	1.0	0.2	35.0	1.0	2.0	5.0	0.0	200.0	10.2	13%
Bread	1164.0	275.0	4.1	0.7	47.8	1.9	3.0	10.3	0.8	89.0	9.3	15%
Peanuts	2569.0	620.0	50.6	6.3	12.5	6.0	6.2	25.6	0.7	30.0	7.7	17%
Cheese	1619.0	370.0	32.0	20.8	0.5	0.5	0.5	25.5	1.8	30.0	7.6	25%
Ice-cream	684.0	163.0	5.6	4.6	24.4	22.6	1.0	3.2	0.1	56.0	1.8	8%
Crisps	2242.0	537.0	32.2	2.8	55.4	0.5	2.3	5.3	1.2	30.0	1.6	4%
Tomato sauce	216.0	51.0	1.0	0.1	8.1	7.0	1.6	1.7	0.7			
Ketchup	435.0	102.0	0.1	0.0	23.2	22.8	0.0	1.2	1.8			
Creamy sauce	422.0	102.0	8.1	3.2	5.8	2.3	0.2	1.1	0.8			
Mayonnaise	2749.0	668.0	73.2	6.1	1.4	1.2	0.0	1.0	1.5			

kJ = kilojoules; kcal = kilocalories; g = grams; PE% = percentage energy from protein

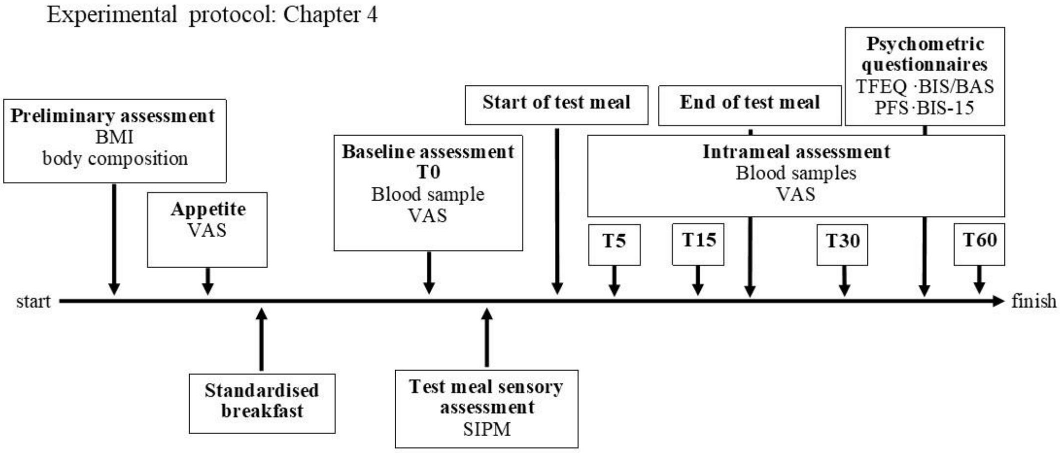
C2: Image of the lunch buffet

Appendix D: Systematics of experimental procedures

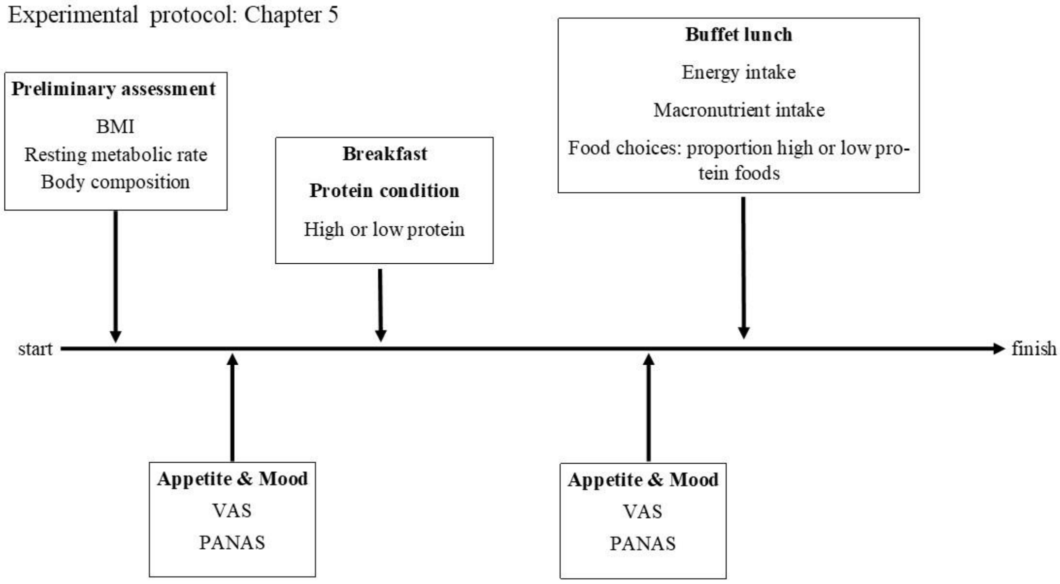
Experimental protocol: Chapter 3



Experimental protocol: Chapter 4



Experimental protocol: Chapter 5



Appendix E: Live Science at the Science Museum, London

E1: Lifestyle questionnaire: Science Museum: How Much Do You Like to Eat?

WELCOME

There are 3 parts to this task:

(i) Some brief questions about who you are (anonymous of course)

(ii) A few questions about your personality

(iii) A task involving ranking of pictures of foods on 3 different qualities

(i) About you:

a. How old are you (number of years)?
b. Now enter your body measurements:
c. Please enter your **HEIGHT** in cm:
d. Please enter your **WEIGHT** in kilogrammes:
e. Please enter your **WAIST** circumference in cm:
f. Who am I? Some questions about you:
g. What language do you speak most of the time at home? *(please type in)*
h. Please select the HIGHEST level of education you have completed from the dropdown list.
i. What is your gender
j. What is your ethnic group?

(ii) A few questions about your personality? (BAS scale from Carver & White BIS/BAS: please refer to Appendix A1)

(iii) A task involving ranking of pictures of foods on 3 different qualities

a. **How much do you want to eat these foods (in these portions)?**

Which of these 10 foods would you **most like to eat right now**, if it was available in this amount? Please drag these foods into the box, then order them to show how much you would like to eat each of them right now, with the most wanted at the top...(Rank by how much you want to eat each food now (you can reorder within this box)

b. **How filling are these foods (in these portions)?**

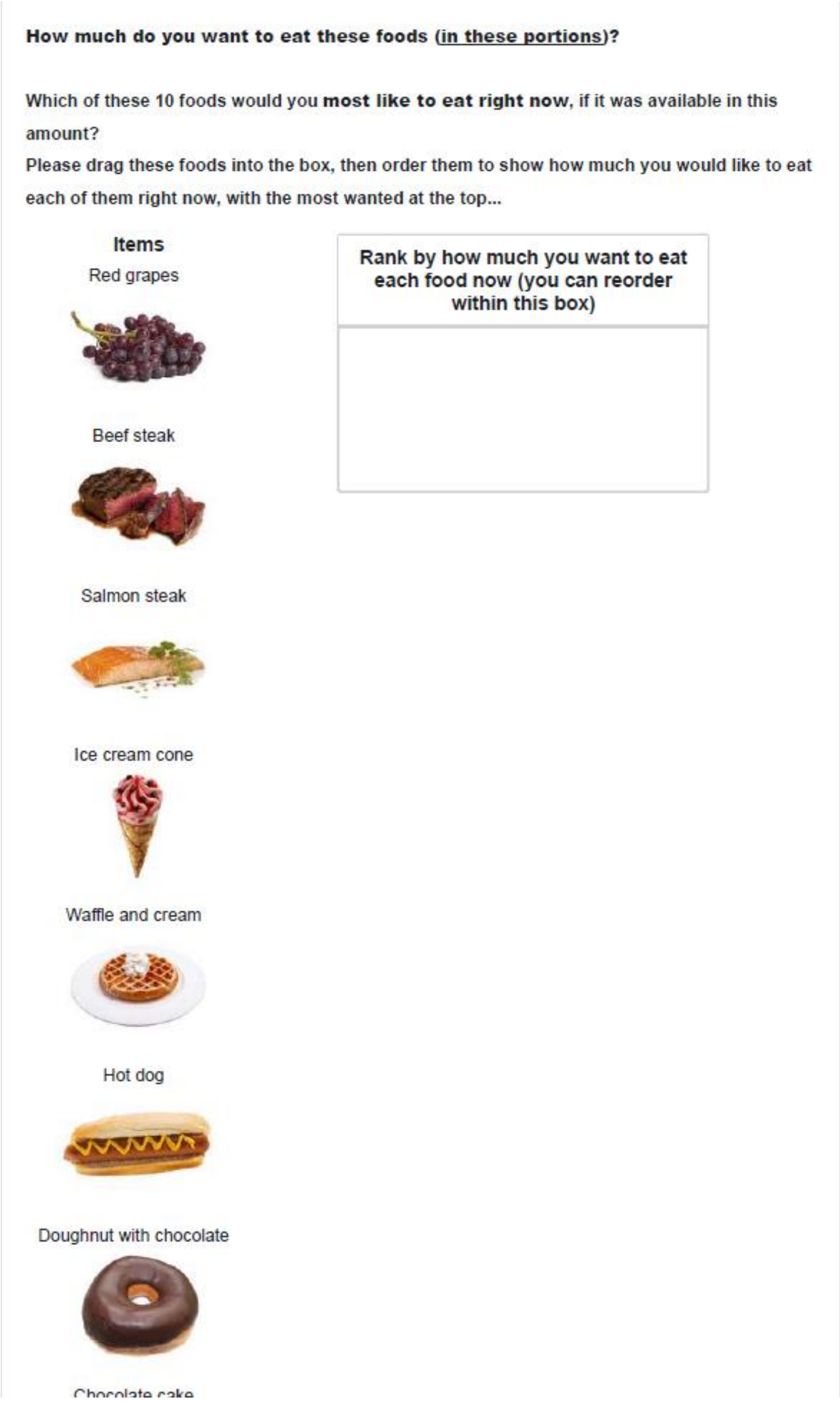
Which of these 10 foods would **most fill you up**? Please drag these foods into the box then order them to show how much they would fill you up, with the most filling at the top...

c. **How often do you eat these foods?**

Please drag these foods into the box, then order them to show how often you eat each food, with the one most often eaten at the top...

If you have never eaten a food, drag it into the bottom box.

Screenshot of food rank task in the lifestyle questionnaire



E2: Food images used for rank order task

Red grapes

Beefsteak

Salmon

Hotdog

Bread roll

Ice-cream

Waffle with whipped cream

Croissant

Donut with chocolate topping

Chocolate cake

E3: Nutritional information of ten food images

Table 6.1

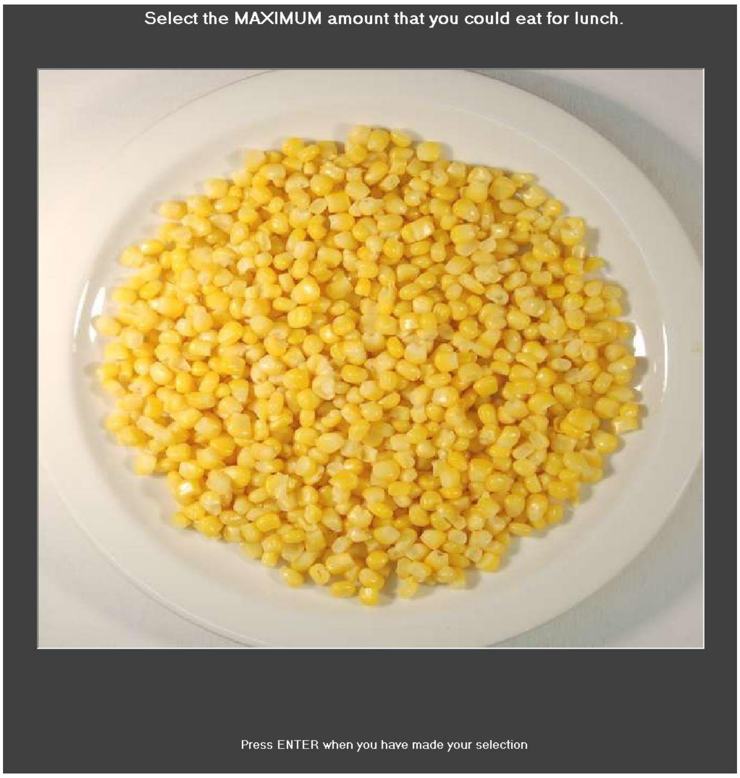
Nutritional information for the ten food items used for the online questionnaire images

Food item	**Weight (g)**	**Carbo-hydrate (g)**	**Fat (g)**	**Protein (g)**	**Energy (kcal)**	**Energy (kJ)**	**ED (kcal.g^{-1})**
Red grapes	300.0	46.8	0.9	2.1	213.0	873.3	0.3
Steak	200.0	0.0	8.0	42.0	242.0	992.2	1.2
Salmon fillet	125.0	1.6	12.0	25.0	212.5	871.25	1.7
Hot dog	115.0	30.2	11.4	10.6	266.8	1093.9	2.3
Bread roll	95.0	48.1	1.3	7.0	235.6	966.0	2.5
Strawberry ice cream cone	92.0	36.0	9.0	2.5	235.0	963.5	2.6
Plain croissant	70.0	25.1	13.2	3.6	233.1	955.7	3.3
Waffle with whipped cream	80.0	42.0	17.1	2.9	264.8	1085.7	3.3
Chocolate cake	65.0	33.5	10.0	3.5	243.8	999.6	3.8
Donut with chocolate topping	55.0	20.0	14.9	3.8	231.0	947.1	4.2

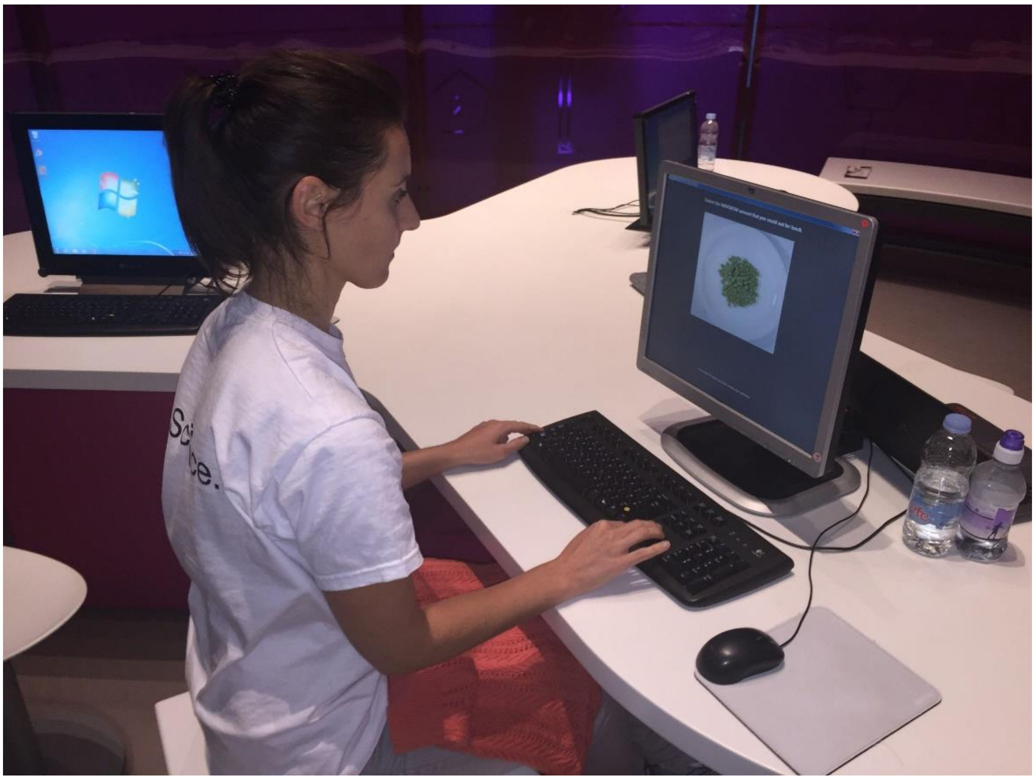
kcal = kilocalories; kJ = kilojoules, g = grammes, ED = energy density (kcal.g^{-1})

E4: Food images used for portion size task

Images of food portion sizes for milk chocolate and sweetcorn.



E5: Image of research station at the Science Museum



Appendix F: Supplementary data Chapter 4

Table F1: Descriptive statistics for variables Body Mass Index (BMI) and Fat Mass (FM) for initial sensory ratings, Sweetness, Creaminess for sweet rice condition (S) and Initial hunger and Adjusted ideal creaminess ratings for non-sweet condition (NS).

	N	Mean	Std. Deviation	Skewness	Std. Error	Kurtosis	Std. Error	Skewness z-score	Kurtosis z-score
Sweetness S	15	26.53	14.29	1.63	0.58	3.94	1.12	**2.82**	**3.51**
Creaminess S	15	33.60	13.65	1.24	0.58	3.28	1.12	**2.15**	**2.93**
Initial Hunger NS	15	61.73	21.39	-1.63	0.58	3.43	1.12	**-2.81**	**3.06**
Adjust Ideal Creamy NS	15	49.13	19.74	0.58	0.58	3.12	1.12	1.08	**2.79**
BMI	15	19.20	33.40	23.51	3.37	1.93	0.58	**4.92**	**1.12**
FM	15	12.80	48.00	21.03	8.49	2.60	0.58	**7.74**	**1.12**

Appendix G: Supplementary data for Chapter 5

Table G1:

Number of participants not included in appetite and mood analyses

Appetite measure	**Sedentary**	**Moderate**	**Active**
Hunger	0	0	3
Fullness	0	1	2
Satisfaction	2	2	2
Estimated intake	1	1	1
Sweet	2	4	3
Salty	1	1	0
Savoury	1	2	0
Fatty	2	2	1
PANAS			
Positive affect	1	0	0
Negative affect	1	0	0

Appendix H: Supplementary data for Chapter 6

Table H1, H2, H3 present Pearson's correlation coefficients of correlations between ranks scores for the desire to eat, expected satiation and frequency of intake of ten different food items.

Table H1:
Pearson correlation coefficients for rank scores of the desire to eat 10 food items:

	Chocolate cake	**Salmon**	**Ice-cream**	**Donut**	**Beef steak**	**Grapes**	**Bread**	**Waffle**	**Croissant**	**Hotdog**
Chocolate cake	1	-0.31**	0.02	0.30**	-0.17**	-0.24**	-0.27**	0.09**	-0.13**	-0.17**
Salmon			-0.22**	-0.40**	0.08	0.07	-0.01	-0.26**	-0.14**	-0.12**
Ice-cream				0.00	-0.25	0.12	-0.24	0.08	-0.14**	-0.18**
Donut					-0.24	-0.24**	-0.22**	0.10**	-0.07	-0.02
Beef steak						-0.30**	-0.12**	-0.26**	-0.27**	0.25**
Grapes							0.05	-0.06	0.00	-0.35**
Bread								-0.24**	0.17**	-0.15**
Waffle									-0.03	-0.19**
Croissant										-0.22**
Hotdog										1

**$p < 0.001$

Table H2:
Pearson correlation coefficients for rank scores of the expected satiation of 10 food items:

	Chocolate cake	**Salmon**	**Ice-cream**	**Donut**	**Beef steak**	**Grapes**	**Bread**	**Waffle**	**Croissant**	**Hotdog**
Chocolate cake	1	-0.45**	0.14**	0.38**	-0.23**	-0.29**	-0.28**	0.19**	-0.20**	-0.10*
Salmon			-0.21**	-0.43**	0.32**	0.10*	-0.09**	-0.37**	-0.16**	-0.01
Ice-cream				0.13**	-0.24**	0.01	-0.27**	0.06	-0.13**	-0.24**
Donut					-0.44**	-0.31**	-0.12**	0.16**	-0.06	-0.13**
Beef steak						-0.06	-0.14**	-0.27**	-0.25**	0.14**
Grapes							-0.07	-0.19**	-0.01	-0.28**
Bread								-0.26**	0.18	-0.10**
Waffle									-0.12**	-0.05
Croissant										-0.19**
Hotdog										1

**p < 0.001, *p < 0.05

Table H3:

Pearson correlation coefficients for rank scores of the frequency of intake of 10 food items:

	Chocolate cake	**Salmon**	**Ice-cream**	**Donut**	**Beef steak**	**Grapes**	**Bread**	**Waffle**	**Croissant**	**Hotdog**
Chocolate cake	1	-0.15**	0.04	0.15**	-0.26**	-0.10*	-0.02	0.02	-0.18**	-0.12**
Salmon			-0.20**	-0.31**	0.12**	0.04	-0.21**	-0.11**	-0.11*	-0.21**
Ice-cream				0.03	-0.18**	0.01	-0.15**	0.09	-0.14**	-0.20**
Donut					-0.23**	-0.19**	-0.10*	0.09	-0.02	0.12**
Beef steak						-0.20**	-0.16**	-0.16**	0.07	-0.15**
Grapes							-0.11**	-0.06	-0.21**	-0.20**
Bread								-0.04	-0.08	0.02
Waffle									-0.07	0.03
Croissant										-0.04
Hotdog										1

$**p < 0.001$, $*p < 0.05$

Appendix I: Supplementary data for Chapter 7

Regression equation: Statistical assumptions

Table I1 presents the intercorrelations for predictor variables BMI, age, WHtR, RMR and sex.

Table I1: Intercorrelations between predictor variables age, BMI, WHtR, RMR and sex

	LnAge	LnBMI	LnWHtR	RMR	Sex
LnAge	1	0.25**	0.34**	-0.14**	-0.03
LnBMI			0.86**	0.53**	0.13**
LnWHtR				0.41**	0.16**
RMR					0.78**
Sex					1

$^{**}p < 0.001$

Table I2 and I3 present the Durbin-Watson statistics and tolerance values for regression analysis (model 1& 2). The linearity of each predictor variable (BMI, age, waist, RMR) and the outcome variable (portion size) was assessed by partial regression plots and plots of the standardised residuals against unstandardized predicted values. There was independence of residuals as inspected by Durbin-Watson statistic (please refer to table I2 below). There was homoscedasticity as assessed by visual inspection of a plot of standardised residuals against unstandardized predicted values. There was no evidence of multicollinearity as assessed by tolerance values greater than 0.1. There were no studentized deleted residuals greater than ±3 standard deviations, no leverage values greater than 0.2, or Cook's distance values greater than 1. The assumption of normality was met as assessed by visual inspection of the Q-Q plot.

Table I2: Durbain-Watson statistic and tolerance values for predictor variables used in regression model 1

Model 1	Durbin-Watson statistic	Tolerance values		
Predictor variable		WHtR	Age	RMR
Peas	1.96	0.66	0.79	0.73
Corn	1.95	0.67	0.80	0.73
Peanuts	1.95	0.67	0.79	0.73
M&M's	2.01	1.52	1.25	1.38
Chocolate	2.05	0.66	0.80	0.74

Table I3: Durbain-Watson statistic and tolerance values for predictor variables used in regression model 2

Model 2	Durbin-Watson statistic	Tolerance values		
Predictor variable		Age	WHtR	Sex
Peas	2.09	0.88	0.86	0.97
Corn	2.20	0.88	0.86	0.97
Peanuts	2.12	0.88	0.86	0.97
M&M's	2.25	0.89	0.86	0.96
Chocolate	1.98	0.88	0.86	0.97

Appendix J: Ethics Committee Documents

J1: Sample participant consent form consent for Chapter 4

University of Roehampton London

PARTICIPANT CONSENT FORM

Title of Research Project:

The influence of food sensory qualities on appetite-related hormones

Brief Description of Research Project:

The aim of this study is to look at the how the different properties of food (taste, flavour, nutrient content) influence hormones involved in controlling appetite and food intake. The trial will test 20 participants and take place over two mornings and lunchtimes at Whitelands College Campus, University of Roehampton, London.

On the first day, you will be provided with a standard breakfast meal. Approximately 3 hours later, you will be provided with either a savoury meal or sweet dessert. Shortly before the meal, we will ask permission to obtain a blood sample by venous cannulation. The cannula will remain in your arm so that we will be able to obtain further blood samples taken before the meal (baseline 0 min), early-meal (+5 minutes), then at 15, 30 and 60 min following the meal. After that, the cannula will be removed. On the second trial day, we will repeat the same procedure. There will be a 7-day interval between the first and second trial day. The blood samples donated will be destroyed after completion of all analyses or upon withdrawal from the project.

As part of this study, you will be required to complete a medical health history questionnaire; some brief personality questionnaires; a body composition assessment where your weight, height and body fat and lean body mass will be measured. For this assessment we will use the BOD POD and the assessment involves sitting in an enclosed chamber with a viewing window for approximately 40 seconds while the BOD POD records changes in air pressure and volume inside the chamber. These values will be used to calculate your body composition. The assessment also requires that you wear minimal, tight-fitting clothing or a swimsuit. You will ask be asked to provide a dietary record of all the food you have eaten over last 24 hours; visit the university laboratory on the morning of the requested days, and consume two breakfast and lunch meals on these testing days.

Investigator Contact Details:

Name: Christle Coxon

Department: Psychology

University address: Whitelands College, Holybourne Avenue, London Postcode: SW15 4JD

Email: coxonc@roehampton.ac.uk

Tel : 020 8392 6005

Consent Statement:

I agree to take part in this research and am aware that I am free to withdraw at any point without giving a reason, although if I do so I understand that my data might still be used in a collated form. I understand that the information I provide will be treated in confidence by the investigator and that my identity will be protected in the publication of any findings, and that data will be collected and processed in accordance with the Data Protection Act 1998, the Human Tissue Act (2004) and with the University's Data Protection Policy.

Name ………………………………… Signature ……………………………… Date ……………………………………

Please note: if you have a concern about any aspect of your participation or any other queries please raise this with the investigator (or if the researcher is a student you can also contact the Director of Studies.) However, if you would like to contact an independent party please contact the Head of Department.

Study Supervisor Contact Details:

Dr Leigh Gibson

University of Roehampton,

Department of Psychology,

Whitelands College,

Holybourne Ave,

London

SW15 4JD

020 8392 3744

l.gibson@roehampton.ac.uk

Head of Department Contact Details:

Dr Diane Bray

University of Roehampton

Department of Psychology,

Whitelands College

Holybourne Ave,

London

SW15 4JD

020 8392 3627

d.bray@roehampton.ac.uk

Appendix K: Published abstracts

Coxon C., Gibson, E.L., Chhina, N., Scholtz, S., Purkayastha, S., Moorthy, K., Hakky, S., Ahmed, A. & Goldstone, A.P. (2016). Reduced desire to eat and ideal creaminess of food following gastric bypass surgery, Appetite, 107, 679-680.

Gibson, E.L., Coxon, C., Crossman, M., Norbury, R., Bakic, D., Elias-Stagg, F., Brunstrom, J.M. (2016). Family influence on portion size? sibling number and birth position are inversely related to maximum portions chosen across a range of foods. Appetite, 107, 681

Coxon, C.; Halsey, L.; Gibson, E.L. (June, 2016) Sugar and fat interact to encourage overeating. Poster presentation at the International Society for Behavioural Nutrition and Physical Activity (ISBNPA, 2016).

Gibson, E.L.; Coxon, C.; Crossman, M.; Norbury, R.; Bakic, D.; Elias-Stagg, F.; Brunstrom, J.M. (June, 2016) Eating rate is positively related to adiposity in adults and children independently of birth order, sibling number, reward sensitivity and FTO rs9939609 polymorphism. Poster presentation at the International Society for Behavioural Nutrition and Physical Activity (ISBNPA, 2016).

Abstract presentations: Obesity week 2016

Are reductions in reward responses to high-energy foods after gastric bypass surgery related to aversive symptoms and dumping syndrome?

B Zaki [1], N Chhina [1], C Coxon [2], N Onokwai [1], S Scholtz [3], S Purkayastha [3], K Moorthy [3], S Hakky [3], A Ahmed [3], AP Goldstone [1]

[1] Division of Brain Sciences, Imperial College London, UK; [2] Dept. of Psychology, University of Roehampton, London, UK; [3] Imperial Weight Centre, St. Mary's Hospital, London, UK

Healthier food hedonics and emotional eating link with orbitofrontal cortex and amygdala responses to food and unpleasant images after gastric bypass surgery

Chhina N[1], Coxon C[2], Onokwai N[1], Scholtz S[3], Purkayastha S[3], Moorthy K[3], Hakky S[3], Ahmed A[3], Goldstone AP[1]

[1]Division of Brain Sciences, Imperial College London, UK; [2]Dept. of Psychology, University of Roehampton, London, UK; [3]Imperial Weight Centre, St. Mary’s Hospital, London, UK

Manuscripts in preparation

Potter, C[1*]., Gibson, E.L.[2], Griggs, R.L[1]., Ferriday, D.[1], Coxon, C.[2], Crossman, M.[2], Norbury, R.[2], Rogers, P.J.[1,3], Brunstrom, J.M.[1,3] (2019) Eating rate, sibling number, birth order and adiposity: Exploring associations between number of siblings, birth order, eating rate and adiposity in children and adults.

[1] Nutrition and Behaviour Unit, School of Psychological Science, University of Bristol, 12a Priory Road, Bristol, BS8 1TU, UK. [2] Department of Psychology, University of Roehampton, London, SW15 4JD, UK. [3] National Institute for Health Research Bristol Biomedical Research Centre, University Hospitals Bristol NHS Foundation Trust and University of Bristol, UK

Appendix L: Post-hoc power analysis of experimental studies

Chapter 3:

Change in hunger: For the number of participants (N = 19) to detect a medium size effect (*d* =0.5), statistical difference (power (1 – **B**) = 0.64, **a** = 0.05, GPOWER 3.1). To achieve adequate power > 0.8, 30 participants would need to be tested.

Change in wanting: For the number of participants (N = 19) to detect a medium size effect (*d* =0.5), statistical difference (power (1 – **B**) = 0.49, **a** = 0.05, GPOWER 3.1). To achieve adequate power > 0.8, 42 participants would need to be tested.

Chapter 4:

Intake: For the number of participants (N = 14) with a small effect size (*d* =0.27), statistical difference (power (1 – **B**) = 0.15, **a** = 0.05, GPOWER 3.1). To achieve adequate power > 0.8, with a medium effect size (*d*= 0.5), 35 participants would need to be tested.

Changes in acyl-ghrelin levels at T5: For the number of participants (N = 10) with a small effect size (*d* =0.27), statistical difference (power (1 – **B**) = 0.43, **a** = 0.05, GPOWER 3.1). To achieve adequate power > 0.8, with a small effect size (*d*= 0.3), 20 participants would need to be tested.

Chapter 5

Intake: For the number of participants in each activity group **(N =** 8**) with a** medium **effect size (d =0.**48**), statistical difference (power (1 –** B**) =** 0.25**,** a **= 0.05, GPOWER 3.1). To achieve adequate power** > 0.8, with a medium effect size (*d*= 0.5), 20 participants would need to be tested.

References

Abizaid, A., Liu, Z. W., Andrews, Z. B., Shanabrough, M., Borok, E., Elsworth, J. D., … Horvath, T. L. (2006). Ghrelin modulates the activity and synaptic input organization of midbrain dopamine neurons while promoting appetite. *Journal of Clinical Investigation*, *116*(12), 3229–3239.

Abreu, S., Santos, R., Moreira, C., Santos, P. C., Mota, J., & Moreira, P. (2014). Food consumption, physical activity and socio-economic status related to BMI, waist circumference and waist-to-height ratio in adolescents. *Public Health Nutrition*, *17*(8), 1834–1849.

Adam, T. C. M., & Westerterp-Plantenga, M. S. (2005). Glucagon-like peptide-1 release and satiety after a nutrient challenge in normal-weight and obese subjects. *The British Journal of Nutrition*, *93*(6), 845–851.

Ahmad, T., I-Min, L. E. E., Paré, G., Chasman, D. I., Rose, L., Ridker, P. M., & Mora, S. (2011). Lifestyle interaction with fat mass and obesity-associated (FTO) genotype and risk of obesity in apparently healthy U.S. women. *Diabetes Care*, *34*(3), 675–680.

Albar, S. A., Alwan, N. A., Evans, C. E. L., & Cade, J. E. (2014). Is there an association between food portion size and BMI among British adolescents? *British Journal of Nutrition*, *112*(5), 841–851.

Almiron-roig, E., Solis-trapala, I., Dodd, J., & Jebb, S. A. (2013). Estimating food portions. Influence of unit number, meal type and energy density. *Appetite*, *71*, 95–103.

Andersen, J. L. (2010). Structural and Molecular Adaptations to Training. In K. Nosaka, R. U. Newton, & M. Cardinale (Eds.), *Strength and conditioning: biological principles and practical applications* (pp. 125–136). Chichester:

Wiley-Blackwell.

Andreasen, C. H., Stender-Petersen, K. L., Mogensen, M. S., Torekov, S. S., Wegner, L., Andersen, G., … Hansen, T. (2008). Low physical activity accentuates the effect of the FTO rs9939609 polymorphism on body fat accumulation. *Diabetes*, *57*(1), 95–101.

Anglé, S., Engblom, J., Eriksson, T., Kautiainen, S., Saha, M.-T., Lindfors, P., … Rimpelä, A. (2009). Three factor eating questionnaire-R18 as a measure of cognitive restraint, uncontrolled eating and emotional eating in a sample of young Finnish females. *The International Journal of Behavioral Nutrition and Physical Activity*, *6*, 41.

Anwar, S., Aleem, B., Rashid, H. H., & Moslhey, G. J. (2019). Which is a better marker for overweight: waist height ratio or waist circumference? *International Journal of Research in Medical Sciences*, *7*(2), 462.

Arganini, C., & Saba, A. (2012). Gender differences in food choice and dietary intake in modern Western societies. *Public Health - Social and Behavioral Health*, 83–103.

Arnold, M., Leitzmann, M., Freisling, H., Bray, F., Romieu, I., Renehan, A., & Soerjomataram, I. (2016). Obesity and cancer: An update of the global impact. *Cancer Epidemiology*, *41*, 8–15.

Arosio, M., Ronchi, C. L., Beck-Peccoz, P., Gebbia, C., Giavoli, C., Cappiello, V., … Peracchi, M. (2004). Effects of Modified Sham Feeding on Ghrelin Levels in Healthy Human Subjects. *The Journal of Clinical Endocrinology & Metabolism*, *89*(10), 5101–5104.

Arvaniti, K., Richard, D., & Tremblay, A. (2000). Reproducibility of energy and macronutrient intake and related substrate oxidation rates in a buffet-type meal.

The British Journal of Nutrition, *83*(5), 489–495.

Ashwell, M. (2005). Waist to height ratio and the Ashwell® shape chart could predict the health risks of obesity in adults and children in all ethnic groups. *Nutrition and Food Science*, *35*(5), 359–364.

Ashwell, M., & Hsieh, S. D. (2005). Six reasons why the waist-to-height ratio is a rapid and effective global indicator for health risks of obesity and how its use could simplify the international public health message on obesity. *International Journal of Food Sciences and Nutrition*, *56*(5), 303–307.

Ashwell, M., & Lejeune, S. (1996). Ratio of waist circumference to height may be better indicator of need for weight management. *British Medical Journal (Clinical Research Ed.)*, *312*, 377–377.

Atella, V., & Kopinska, J. (2012). Body Weight of Italians: The Weight of Education. *SSRN Electronic Journal*, *9*(3).

Austin, G. L., & Krueger, P. M. (2013). Increasing the Percentage of Energy from Dietary Sugar, Fats, and Alcohol in Adults Is Associated with Increased Energy Intake but Has Minimal Association with Biomarkers of Cardiovascular Risk. *The Journal of Nutrition*, *143*(10), 1651–1658.

Austin, G. L., Ogden, L. G., & Hill, J. O. (2011). Trends in carbohydrate, fat, and protein intakes and association with energy intake in normal-weight, overweight, and obese individuals: 1971-2006. *American Journal of Clinical Nutrition*, *93*(4), 836–843.

Ax, E., Warensjö Lemming, E., Becker, W., Andersson, A., Lindroos, A. K., Cederholm, T., … Fung, T. T. (2016). Dietary patterns in Swedish adults; Results from a national dietary survey. *British Journal of Nutrition*, *115*(1), 95–104.

Baker, A. H., & Wardle, J. (2003). Sex differences in fruit and vegetable intake in older adults. *Appetite, 40*(3), 269–275.

Baker, B. J., Booth, D. A., Duggan, J. P., & Gibson, E. L. (1987). Protein appetite demonstrated: learned specificity of protein-cue preference to protein need in adult rats. *Nutrition Research*, *7*, 481–487.

Ball, K., & Crawford, D. (2005). Socioeconomic status and weight change in adults: A review. *Social Science and Medicine*, *60*(9), 1987–2010.

Ballard, O., & Morrow, A. L. (2013). Human milk composition: Nutrients and bioactive factors. *Pediatric Clinics of North America*, *7*(4), 304–312.

Bamia, C., Trichopoulos, D., Ferrari, P., Overvad, K., Bjerregaard, L., Tjønneland, A., … Trichopoulou, A. (2007). Dietary patterns and survival of older Europeans: The EPIC-Elderly Study (European Prospective Investigation into Cancer and Nutrition). *Public Health Nutrition*, *10*(6), 590–598.

Barkeling, B., King, N. A., Näslund, E., & Blundell, J. E. (2007). Characterization of obese individuals who claim to detect no relationship between their eating pattern and sensations of hunger or fullness. *International Journal of Obesity*, *31*(3), 435–439.

Bartoshuk, L. M., Duffy, V. B., Hayes, J. E., Moskowitz, H. R., & Snyder, D. J. (2006). Psychophysics of sweet and fat perception in obesity: problems, solutions and new perspectives. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *361*(1471), 1137–1148.

Batterham, R. L., Heffron, H., Kapoor, S., Chivers, J. E., Chandarana, K., Herzog, H., … Withers, D. J. (2006). Critical role for peptide YY in protein-mediated satiation and body-weight regulation. *Cell Metabolism*, *4*(September 2006), 223–233.

Baum, C. L., & Ruhm, C. J. (2009). Age, socioeconomic status and obesity growth. *Journal of Health Economics*, *28*(3), 635–648.

Bauman, A., Rutter, H., & Baur, L. (2019). Too little, too slowly: international perspectives on childhood obesity. *Public Health Research & Practice*, *29*(1), 1–5.

Baumann, A. A., & Odum, A. L. (2012). Impulsivity, risk taking, and timing. *Behavioural Processes*, *90*(3), 408–414.

Beals, K. A., & Mitchell, A. (2015). Recent recommendations and current controversies in sport nutrition. *American Journal of Lifestyle Medicine*, *9*(4), 288–297.

Beauchamp, G.K., & Mennella, J. A. (2009). Early flavor learning and its impact on later feeding behavior. *Journal of Pediatric Gastroenterology and Nutrition*, *48 Suppl 1*(10), S25-30.

Beauchamp, Gary K. (2016). Why do we like sweet taste: A bitter tale? *Physiology & Behavior*, *164*(Part B), 432–437.

Beaudart, C., Locquet, M., Touvier, M., Reginster, J. Y., & Bruyère, O. (2019). Association between dietary nutrient intake and sarcopenia in the SarcoPhAge study. *Aging Clinical and Experimental Research*, *31*(6), 815–824.

Beaulieu, K., Hopkins, M., Blundell, J. E., & Finlayson, G. (2016). Does Habitual Physical Activity Increase the Sensitivity of the Appetite Control System? A Systematic Review. *Sports Medicine*, *46*(12), 1897–1919.

Beaulieu, K., Hopkins, M., Blundell, J. E., & Finlayson, G. (2018). Homeostatic and non-homeostatic appetite control along the spectrum of physical activity levels: An updated perspective. *Physiology and Behavior*, *192*(Aug 1), 23–29.

Beaulieu, Kristine, Hopkins, M., Blundell, J., & Finlayson, G. (2017). Impact of physical activity level and dietary fat content on passive overconsumption of energy in non-obese adults. *International Journal of Behavioral Nutrition and Physical Activity*, *14*(1), doi: 10.1186/s12966-017-0473-3.

Beaulieu, Kristine, Hopkins, M., Long, C., Blundell, J., & Finlayson, G. (2017). High Habitual Physical Activity Improves Acute Energy Compensation in Nonobese Adults. *Medicine and Science in Sports and Exercise*, *49*(11), 2268–2275.

Beaver, J. D., Lawrence, A. D., van Ditzhuijzen, J., Davis, M. H., Woods, A., & Calder, A. J. (2006). Individual differences in reward drive predict neural responses to images of food. *The Journal of Neuroscience*, *26*(19), 5160–5166.

Beck, K. L., Jones, B., Ullah, I., McNaughton, S. A., Haslett, S. J., & Stonehouse, W. (2018). Associations between dietary patterns, socio-demographic factors and anthropometric measurements in adult New Zealanders: an analysis of data from the 2008/09 New Zealand Adult Nutrition Survey. *European Journal of Nutrition*, *57*(4), 1421–1433.

Bellisle, F., & Dalix, a. M. (2001). Cognitive restraint can be offset by distraction, leading to increased meal intake in women. *American Journal of Clinical Nutrition*, *74*(2), 197–200.

Bellisle, France, Clement, K., Le Barzic, M., Le Gall, A., Guy-Grand, B., & Basdevant, A. (2004). The eating inventory and body adiposity from leanness to massive obesity: A study of 2509 adults. *Obesity Research*, *12*(12), 2023–2030.

Bellisle, France, Dalix, A. M., Airinei, G., Hercberg, S., & Péneau, S. (2009). Influence of dietary restraint and environmental factors on meal size in normal-weight women. A laboratory study. *Appetite*, *53*(3), 309–313.

Bellissimo, N., Desantadina, M. V, Pencharz, P. B., Berall, G. B., Thomas, S. G., &

Anderson, G. H. (2008). A comparison of short-term appetite and energy intakes in normal weight and obese boys following glucose and whey-protein drinks. *International Journal of Obesity*, *32*(2), 362–371.

Benites-Zapata, V. A., Toro-Huamanchumo, C. J., Urrunaga-Pastor, D., Guarnizo-Poma, M., Lazaro-Alcantara, H., Paico-Palacios, S., … Ranilla-Seguin, V. del C. (2019). High waist-to-hip ratio levels are associated with insulin resistance markers in normal-weight women. *Diabetes and Metabolic Syndrome: Clinical Research and Reviews*, *13*(1), 636–642.

Benton, D. (2015). Portion Size: What We Know and What We Need to Know. *Critical Reviews in Food Science and Nutrition*, *55*(7), 988–1004.

Bere, E., Brug, J., & Klepp, K. I. (2008). Why do boys eat less fruit and vegetables than girls? *Public Health Nutrition*, *11*(3), 321–325.

Berg, C., Lappas, G., Wolk, A., Strandhagen, E., Torén, K., Rosengren, A., … Lissner, L. (2009). Eating patterns and portion size associated with obesity in a Swedish population. *Appetite*, *52*(1), 21–26.

Berridge, K.C., & Kringelbach, M. L. (2013). Neuroscience of affect: Brain mechanisms of pleasure and displeasure. *Current Opinion in Neurobiology*, *23*(3), 294–303.

Berridge, Kent C. (2009). “Liking” and “wanting” food rewards: Brain substrates and roles in eating disorders. *Physiology and Behavior*, *97*(5), 537–550.

Berridge, Kent 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.

Berridge, Kent C., & Robinson, T. E. (2016). Liking, wanting, and the incentive-sensitization theory of addiction. *American Psychologist*, *71*(8), 670–679.

Berridge, Kent C., Robinson, T. E., & Aldridge, J. W. (2009). Dissecting components of reward: “liking”, “wanting”, and learning. *Current Opinion in Pharmacology*, *9*(1), 65–73.

Berthoud, H.-R. (2006). Homeostatic and non-homeostatic pathways involved in the control of food intake and energy balance. *Obesity (Silver Spring, Md.)*, *14 Suppl 5*(August), 197S-200S.

Berthoud, H.-R., Lenard, N. R., & Shin, A. C. (2011). Food reward, hyperphagia, and obesity. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, *300*(6), R1266–R1277.

Berthoud, H.-R., Münzberg, H., Richards, B. K., & Morrison, C. D. (2012). Neural and metabolic regulation of macronutrient intake and selection. *Proceedings of the Nutrition Society*, *71*(3), 390–400.

Berthoud, H. R. (2007). Interactions between the “cognitive” and “metabolic” brain in the control of food intake. *Physiology and Behavior*, *91*(5), 486–498.

Berthoud, H. R., Münzberg, H., & Morrison, C. D. (2017). Blaming the Brain for Obesity: Integration of Hedonic and Homeostatic Mechanisms. *Gastroenterology*, *152*(7), 1728–1738.

Bertin, M., Touvier, M., Dubuisson, C., Dufour, A., Havard, S., Lafay, L., … Lioret, S. (2016). Dietary patterns of French adults: Associations with demographic, socio-economic and behavioural factors. *Journal of Human Nutrition and Dietetics*, *29*(2), 241–254.

Bertoli, S. (2014). Taste sensitivity, nutritional status and metabolic syndrome: Implication in weight loss dietary interventions. *World Journal of Diabetes*, *5*(5), 717.

Besnard, P., Passilly-Degrace, P., & Khan, N. A. (2015). Taste of fat: A sixth taste modality? *Physiological Reviews*, *96*(1), 151–176.

Best, M., Barsalou, L. W., & Papies, E. K. (2018). Studying human eating behaviour in the laboratory: Theoretical considerations and practical suggestions. *Appetite, 130*(1), 339–343.

Best, M., & Papies, E. K. (2017). Right Here, Right Now: Situated Interventions to Change Consumer Habits. *Journal of the Association for Consumer Research*, *2*(3), 333–358.

Beunza, J. J., Toledo, E., Hu, F. B., Bes-Rastrollo, M., Serrano-Martínez, M. Sánchez-Villegas, A., Martínez, J. A., & Martínez-González, M. (2010). Adherence to the Mediterranean diet, long-term weight change, and incident overweight or obesity: the Seguimiento Universidad de Navarra (SUN) cohort. *American Journal of Clinical Nutrition*, *92*(3), 1484–1493.

Bhaskaran, K., Dos-Santos-Silva, I., Leon, D. A., Douglas, I. J., & Smeeth, L. (2018). Association of BMI with overall and cause-specific mortality: a population-based cohort study of 3.6 million adults in the UK. *The Lancet Diabetes and Endocrinology*, *6*(12), 944–953.

Bi, X., Loo, Y. T., & Henry, C. J. (2019). Android fat as a determinant of metabolic syndrome: Sex differences. *Nutrition*, *57*, 127–132.

Blatt, A. D., Roe, L. S., & Rolls, B. J. (2011). Increasing the Protein Content of Meals and Its Effect on Daily Energy Intake. *Journal of the American Dietetic Association*, *111*(2), 290–294.

Blechert, J., Meule, A., Busch, N. A., & Ohla, K. (2014). Food-pics: An image database for experimental research on eating and appetite. *Frontiers in Psychology*, *5*(JUN), 1–10.

Blüher, M. (2019). Obesity: global epidemiology and pathogenesis. *Nature Reviews*

Endocrinology, *15*(5), 288–298.

Blundell, J. E. (2001). Regulation of Appetite and the Management of Obesity. *International Textbook of Obesity*, 113–124.

Blundell, J. E. (2011). Physical activity and appetite control: Can we close the energy gap? *Nutrition Bulletin*, *36*(3), 356–366.

Blundell, J. E. (2018). Biology of the drive to eat: Implications for understanding human appetite and obesity. *Ernahrungs Umschau*, *65*(7), 126–133.

Blundell, J. E., Caudwell, P., Gibbons, C., Hopkins, M., Naslund, E., King, N. A., & Finlayson, G. (2012). Role of resting metabolic rate and energy expenditure in hunger and appetite control: a new formulation. *Disease Models & Mechanisms*, *5*(5), 608–613.

Blundell, J. E., Caudwell, P., Gibbons, C., Hopkins, M., Näslund, E., King, N. A., & Finlayson, G. (2012). Body composition and appetite: fat-free mass (but not fat mass or BMI) is positively associated with self-determined meal size and daily energy intake in humans. *The British Journal of Nutrition*, *107*(3), 445–449.

Blundell, J. E., & Finlayson, G. (2004). Is susceptibility to weight gain characterized by homeostatic or hedonic risk factors for overconsumption? *Physiology and Behavior*, *82*(1), 21–25.

Blundell, J. E., Finlayson, G., Gibbons, C., Caudwell, P., & Hopkins, M. (2015). The biology of appetite control: Do resting metabolic rate and fat-free mass drive energy intake? *Physiology and Behavior*, *152*, 473–478.

Blundell, J. E., & Macdiarmid, J. I. (1997). Fat as a risk factor for overconsumption: Satiation, satiety, and patterns of eating. *Journal of Amercian Dietetic Association*, *97*, S63–S69.

Blundell, J. E., Stubbs, R. J., Golding, C., Croden, F., Alam, R., Whybrow, S., …

Lawton, C. L. (2005). Resistance and susceptibility to weight gain: Individual variability in response to a high-fat diet. *Physiology and Behavior*, *86*(5), 614–622.

Bock, B. C., & Kanarek, R. B. (1995). Women and men are what they eat: The effects of gender and reported meal size on perceived characteristics. *Sex Roles*, *33*(1–2), 109–119.

Boeing, H. (2013). Obesity and cancer - The update 2013. *Best Practice and Research: Clinical Endocrinology and Metabolism*, *27*(2), 219–227.

Boitard, C., Cavaroc, A., Sauvant, J., Aubert, A., Castanon, N., Layé, S., & Ferreira, G. (2014). Impairment of hippocampal-dependent memory induced by juvenile high-fat diet intake is associated with enhanced hippocampal inflammation in rats. *Brain, Behavior, and Immunity*, *40*, 9–17.

Bolhuis, D. P., Costanzo, A., Newman, L. P., & Keast, R. S. J. (2016). Salt Promotes Passive Overconsumption of Dietary Fat in Humans. *Journal of Nutrition*, *146*(4), 838–845.

Bolhuis, Dieuwerke P., Costanzo, A., & Keast, R. S. J. (2018). Preference and perception of fat in salty and sweet foods. *Food Quality and Preference*, *64*(September 2017), 131–137.

Bolhuis, Dieuwerke P., Newman, L. P., & Keast, R. S. J. (2016). Effects of salt and fat combinations on taste preference and perception. *Chemical Senses*, *41*(3), 189–195.

Bongers, P., de Graaff, A., & Jansen, A. (2016). “Emotional” does not even start to cover it: Generalization of overeating in emotional eaters. *Appetite*, *96*, 611–616.

Booth, D. A. (2009). Lines, dashed lines and “scale” ex-tricks. Objective measurements of appetite versus subjective tests of intake. *Appetite*, *53*(3), 434–437.

Booth, D. A., & Toates, F. M. (1974). A physiological control theory of food intake in the rat: Mark 1. *Bulletin of the Psychonomic Society*, *3*(6), 442–444.

Booth, H. P., Charlton, J., & Gulliford, M. C. (2017). Socioeconomic inequality in morbid obesity with body mass index more than 40 kg/m2 in the United States and England. *SSM - Population Health*, *3*(December 2016), 172–178.

Borodulin, K., Zimmer, C., Sippola, R., Mäkinen, T. E., Laatikainen, T., & Prättälä, R. (2012). Health behaviours as mediating pathways between socioeconomic position and body mass index. *International Journal of Behavioral Medicine, 19*(1), 14–22.

Bou, R., Cofrades, S., & Jiménez-Colmenero, F. (2014). Physicochemical properties and riboflavin encapsulation in double emulsions with different lipid sources. *LWT - Food Science and Technology*, *59*(2P1), 621–628.

Bowen, J., Noakes, M., Trenerry, C., & Clifton, P. M. (2006). Energy intake, ghrelin, and cholecystokinin after different carbohydrate and protein preloads in overweight men. *Journal of Clinical Endocrinology and Metabolism*, *91*(4), 1477–1483.

Boylan, S. M., Gill, T. P., Hare-Bruun, H., Andersen, L. B., & Heitmann, B. L. (2014). Associations between adolescent and adult socioeconomic status and risk of obesity and overweight in Danish adults. *Obesity Research and Clinical Practice*, *8*(2), e163–e171.

Breen, F. M., Plomin, R., & Wardle, J. (2006). Heritability of food preferences in young children. *Physiology and Behavior*, *88*(4–5), 443–447.

Brennan, I., Luscombe-Marsh, ND Seimon, R., Otto, B., Horowitz, M., Wishart, J.,

& Feinle-Bisset, C. (2006). Effects of fat, protein, and carbohydrate and protein load on appetite, plasma cholecystokinin, peptide YY, and ghrelin, and energy intake in lean and obese men. *Am J Physiol Gastrointest Liver Physiol*, *83*(1), 89–94.

Breslin, P. A. S. (2013). An evolutionary perspective on food and human taste. *Current Biology*, *23*(9), R409–R418.

British Heart Foundation. (2013). Portion Distortion Report:How Much Are We Really Eating? https://www.bhf.org.uk/informationsupport/publications/policy-documents/portion-distortion-report-2013. Accessed 7th September 2019

British Nutrition Foundation. (2018). What is energy density? https://www.nutrition.org.uk/healthyliving/fuller/what-is-energy-density.html. Accessed 9th February 2019

Brogden, N., Sinclair, C., & Almiron-roig, E. (2009). Effects of hunger and experience on portion size estimation by men (abstract). *Appetite*, *52*, 821.

Brogden, Nina, & Almiron-Roig, E. (2010). Food liking, familiarity and expected satiation selectively influence portion size estimation of snacks and caloric beverages in men. *Appetite*, *55*(3), 551–555.

Brogden, Nina, & Almiron-Roig, E. (2011). Estimated portion sizes of snacks and beverages differ from reference amounts and are affected by appetite status in non-obese men. *Public Health Nutrition*, *14*(10), 1743–1751.

Brownell, K. D., Kersh, R., Ludwig, D. S., Post, R. C., Puhl, R. M., Schwartz, M. B., & Willett, W. C. (2010). Personal responsibility and obesity: A constructive approach to a controversial issue. *Health Affairs*, *29*(3), 379–387.

Browning, L. M., Hsieh, S. D., & Ashwell, M. (2010). A systematic review of waist-to-height ratio as a screening tool for the prediction of cardiovascular disease and diabetes: 05 could be a suitable global boundary value. *Nutrition Research Reviews*, *23*(2), 247–269.

Brunstrom, J. M. (2005). Dietary learning in humans: Directions for future research. *Physiology and Behavior, 85*(1), 57–65.

Brunstrom, J. M. (2011). The control of meal size in human subjects: A role for expected satiety, expected satiation and premeal planning. *Proceedings of the Nutrition Society*, *70*(2), 155–161.

Brunstrom, J. M. (2014). Mind over platter: Pre-meal planning and the control of meal size in humans. *International Journal of Obesity, 38*(SUPPL. 1), S9–S12. Nature Publishing Group.

Brunstrom, J. M., Collingwood, J., & Rogers, P. J. (2010). Perceived volume, expected satiation, and the energy content of self-selected meals. *Appetite*, *55*(1), 25–29.

Brunstrom, J. M., Drake, A. C. L., Forde, C. G., & Rogers, P. J. (2018). Undervalued and ignored: Are humans poorly adapted to energy-dense foods? *Appetite*, *120*, 589–595.

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.

Brunstrom, J. M., Rogers, P. J., Pothos, E. M., Calitri, R., & Tapper, K. (2008). Estimating everyday portion size using a ‘method of constant stimuli : In a student sample, portion size is predicted by gender, dietary behaviour, and hunger, but not BMI. *Appetite*, *51*, 296–301.

Brunstrom, J. M., & Shakeshaft, N. G. (2009). Measuring affective (liking) and non-affective (expected satiety) determinants of portion size and food reward.

Appetite, 52(1), 108–114.

Brunstrom, J. M., Shakeshaft, N. G., & Alexander, E. (2010). Familiarity changes expectations about fullness. *Appetite, 54*(3), 587–590.

Brunstrom, J. M., Shakeshaft, N. G., & Scott-Samuel, N. E. (2008). Measuring “expected satiety” in a range of common foods using a method of constant stimuli. *Appetite, 51*(3), 604–614.

Bryant, E. J., King, N. A., & Blundell, J. E. (2008). Disinhibition: its effect on appetite and weight regulation. *Obesity Reviews*, *9*(5), 409–419.

Bryden, A., Petticrew, M., Mays, N., Eastmure, E., & Knai, C. (2013). Voluntary agreements between government and business-A scoping review of the literature with specific reference to the Public Health Responsibility Deal. *Health Policy*, *110*(2–3), 186–197.

Buckland, N. J., James Stubbs, R., & Finlayson, G. (2015). Towards a satiety map of common foods: Associations between perceived satiety value of 100 foods and their objective and subjective attributes. *Physiology & Behavior, 152*, 340–346.

Buckley, C. M., Austin, S., Corfe, B. M., Green, M. A., Johnstone, A. M., Stevenson, E. J., … Brunstrom, J. M. (2019). Protein valuation in food choice is positively associated with lean mass in older adults. *The Journal of Nutrition: Ingestive Behavior and Neurosciences, 00,* 1–9.

Burger, K. S., & Berner, L. A. (2014). A functional neuroimaging review of obesity, appetitive hormones and ingestive behavior. *Physiology & Behavior*, *136,* 121–127.

Burger, K. S., & Stice, E. (2011). Relation of dietary restraint scores to activation of reward-related brain regions in response to food intake, anticipated intake, and food pictures. *NeuroImage*, *55*(1), 233–239.

Burger, K. S., & Stice, E. (2012). Frequent ice cream consumption is associated with reduced striatal response to receipt of an ice cream-based milkshake. *American Journal of Clinical Nutrition*, *95*, 810–817.

Burgoine, T., Forouhi, N. G., Griffin, S. J., Brage, S., Wareham, N. J., & Monsivais, P. (2016). Does neighborhood fast-food outlet exposure amplify inequalities in diet and obesity? A cross sectional study. *American Journal of Clinical Nutrition*, *103*(6), 1540–1547.

Cadbury. (2020). Cadbury Dairy Milk.

Callahan, H. S., Cummings, D. E., Pepe, M. S., Breen, P. A., Matthys, C. C., & Weigle, D. S. (2004). Postprandial suppression of plasma ghrelin level is proportional to ingested caloric load but does not predict intermeal interval in humans. *Journal of Clinical Endocrinology and Metabolism*, *89*(3), 1319–1324.

Calviño, A. M., García-Medina, M. R., Cometto-muñiz, J. E., Rodríguez, M. B., & de Fisiología, C. (1993). Perception of sweetness and bitterness in different vehicles. *Perception & Psychophysics*, *54*(6), 751–758.

Cameron, J. D., Sigal, R. J., Kenny, G. P., Alberga, A. S., Prud'homme, D., Phillips, P., … Goldfield, G. (2016). Body composition and energy intake — skeletal muscle mass is the strongest predictor of food intake in obese adolescents: The HEARTY trial. *Applied Physiology, Nutrition, and Metabolism*, *41*(6), 611–617.

Camilleri, G. M., Méjean, C., Kesse-Guyot, E., Andreeva, V. A., Bellisle, F., Hercberg, S., & Péneau, S. (2014). The Associations between Emotional Eating and Consumption of Energy-Dense Snack Foods Are Modified by Sex and Depressive Symptomatology. *The Journal of Nutrition*, *144*(8), 1264–1273.

Camiña, J. P. (2006). Cell biology of the ghrelin receptor. *Journal of*

Neuroendocrinology, *18*(1), 65–76.

Cappelleri, J. C., Bushmakin, A. G., Gerber, R. A., Leidy, N. K. ., Sexton, C. C., Lowe, M. R., & Karlsson, J. (2009). Psychometric analysis of the Three-Factor Eating Questionnaire-R21: results from a large diverse sample of obese and non-obese participants. *International Journal of Obesity (2005)*, *33*(6), 611–620.

Cappelleri, J. C., Bushmakin, A. G., Gerber, R. A., Leidy, N. K., Sexton, C. C., Karlsson, J., & Lowe, M. R. (2009). Evaluating the Power of Food Scale in obese subjects and a general sample of individuals: development and measurement properties. *International Journal of Obesity (2005)*, *33*(8), 913–922.

Cardi, V., Leppanen, J., & Treasure, J. (2015). The effects of negative and positive mood induction on eating behaviour: A meta-analysis of laboratory studies in the healthy population and eating and weight disorders. *Neuroscience and Biobehavioral Reviews*, *57*, 299–309.

Carr, K. A., Lin, H., Fletcher, K. D., & Epstein, L. H. (2014). Food reinforcement, dietary disinhibition and weight gain in nonobese adults. *Obesity*, *22*(1), 254–259.

Carter, F. A., & Jansen, A. (2012). Improving psychological treatment for obesity. Which eating behaviours should we target? *Appetite*, *58*(3), 1063–1069.

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*, *67*(2), 319–333.

Casanova, N., Beaulieu, K., Oustric, P., Gibbons, C., Finlayson, G., & Hopkins, M. (2019). The relationship between resting metabolic rate and free-living daily energy intake is moderated by body fat percentage in women with overweight and obesity. *Obesity Abstracts*, *1 P37*, DOI: 10.1530/obabs.01.P37.

Caudwell, P., Finlayson, G., Gibbons, C., Hopkins, M., King, N. A., Näslund, E., & Blundell, J. E. (2013). Resting metabolic rate is associated with hunger, self-determined meal size, and daily energy intake and may represent a marker for appetite. *American Journal of Clinical Nutrition*, *97*(1), 7–14.

Caudwell, P., Gibbons, C., Hopkins, M., Naslund, E., King, N. A., Finlayson, G., & Blundell, J. E. (2011). The influence of physical activity on appetite control: An experimental system to understand the relationship between exercise-induced energy expenditure and energy intake. *Proceedings of the Nutrition Society*, *70*(2), 171–180.

Cecil, J. E., Tavendale, R., Watt, P., Hetherington, M. M., & Palmer, C. N. A. (2008). An Obesity-Associated FTO Gene Variant and Increased Energy Intake in Children. *The New England Journal of Medicine*, *359*, 2558–2566.

Celis-Morales, C., Marsaux, C. F. M., Livingstone, K. M., Navas-Carretero, S., San-Cristobal, R., O'Donovan, C. B., … Mathers, J. C. (2016). Physical activity attenuates the effect of the FTO genotype on obesity traits in European adults: The Food4Me study. *Obesity*, *24*(4), 962–969.

Cerit, H., Christensen, K., Moondra, P., Klibanski, A., Goldstein, J. M., & Holsen, L. M. (2019). Divergent associations between ghrelin and neural responsivity to palatable food in hyperphagic and hypophagic depression. *Journal of Affective Disorders*, *242*(November 2017), 29–38.

Cermak, N. M., Res, P. T., de Groot, L. C., Saris, W. H., & van Loon, L. J. (2012). Protein supplementation augments the adaptive response of skeletal muscle to resistance-type exercise training: a meta-analysis. *The American Journal of*

Clinical Nutrition, *96*, 1454–1464.

Chaiken, S., & Pliner, P. (1987). Women, but not Men, Are What They Eat. *Personality and Social Psychology Bulletin*, *13*(2), 166–176.

Chaput, J. P., Leblanc, C., Pérusse, L., Després, J. P., Bouchard, C., & Tremblay, A. (2009). Risk factors for adult overweight and obesity in the quebec family study: Have we been barking up the wrong tree. *Obesity*, *17*(10), 1964–1970.

Chaudhri, O. B., Field, B. C. T., & Bloom, S. R. (2008). Gastrointestinal satiety signals. *International Journal of Obesity (2005)*, *32 Suppl 7*, S28–S31.

Choi, J. H., & Chung, S. J. (2015). Sweetness potency and sweetness synergism of sweeteners in milk and coffee systems. *Food Research International*, *74*, 168–176.

Chrostowska, M., Szyndler, A., Hoffmann, M., & Narkiewicz, K. (2013). Impact of obesity on cardiovascular health. *Best Practice & Research. Clinical Endocrinology & Metabolism*, *27*(2), 147–156.

Church, T. S., Thomas, D. M., Tudor-Locke, C., Katzmarzyk, P. T., Earnest, C. P., Rodarte, R. Q., … Bouchard, C. (2011). Trends over 5 decades in U.S. occupation-related physical activity and their associations with obesity. *PLoS ONE*, *6*(5), 1–7.

Claussnitzer, M., Dankel, S., Kim, K., Quon, G., Meuleman, W., Haugen, C., … Kellis, M. (2015). FTO Obesity Variant Circuitry and Adipocyte Browning in Humans. *New England Journal of Medicine*, *373*(10), 895–907.

Colquhoun, D. (2017). *The Reproducibility Of Research And The Misinterpretation Of P Values | bioRxiv*.

Cornier, M. A., Salzberg, A. K., Endly, D. C., Bessesen, D. H., & Tregellas, J. R. (2010). Sex-based differences in the behavioral and neuronal responses to food.

Physiology and Behavior, *99*(4), 538–543.

Covasa, M., Grahn, J., & Ritter, R. C. (2000). High fat maintenance diet attenuates hindbrain neuronal response to CCK. *Regulatory Peptides*, *86*(1–3), 83–88.

Cox, D. N., Perry, L., Moore, P. B., Vallis, L., & Mela, D. J. (1999). Sensory and hedonic associations with macronutrient and energy intakes of lean and obese consumers. *International Journal of Obesity*, *23*, 403–410.

Craig, C. L., Marshall, A. L., Sjöström, M., Bauman, A. E., Booth, M. L., Ainsworth, B. E., … Oja, P. (2003). International physical activity questionnaire: 12-Country reliability and validity. *Medicine and Science in Sports and Exercise*, *35*(8), 1381–1395.

Crino, M., Sacks, G., Vandevijvere, S., Swinburn, B., & Neal, B. (2015). The Influence on Population Weight Gain and Obesity of the Macronutrient Composition and Energy Density of the Food Supply. *Current Obesity Reports*, *4*(1), 1–10.

Crum, A. J., Corbin, W. R., Brownell, K. D., & Salovey, P. (2011). Mind over milkshakes: mindsets, not just nutrients, determine ghrelin response. *Health Psychology : Official Journal of the Division of Health Psychology, American Psychological Association*, *30*(4), 424–429; discussion 430-431.

Crystal, S. R., & Teff, K. L. (2006). Tasting fat: Cephalic phase hormonal responses and food intake in restrained and unrestrained eaters. *Physiology and Behavior*, *89*(2), 213–220.

Csongová, M., Volkovová, K., Gajdoš, M., Gurecká, R., Koborová, I., Líšková, A., & Šebeková, K. (2018). Gender-associated differences in the prevalence of central obesity using waist circumference and waist-to-height ratio, and that of general obesity, in Slovak adults. *Central European Journal of Public Health*, *26*(3), 228–233.

Cugini, P., Salandri, A., Cilli, M., Ceccotti, P., Di Marzo, A., Rodio, A., … Giovannini, C. (1998). Daily hunger sensation and body composition: I. Their relationships in clinically healthy subjects. *Eating and Weight Disorders : EWD*, *3*(4), 168–172.

Cugini, P., Salandri, A., Cilli, M., Ceccotti, P., Di Marzo, A., Rodio, A., … Giovannini, C. (1999). Daily hunger sensation and body compartments: II. Their relationships in obese patients. *Eating and Weight Disorders*, *4*(2), 81–88.

Cummings, D. E. (2006). Ghrelin and the short- and long-term regulation of appetite and body weight. *Physiology and Behavior*, *89*(1), 71–84.

Cummings, D. E., Foster-Schubert, K., & Overduin, J. (2005). Ghrelin and Energy Balance: Focus on Current Controversies. *Current Drug Targets, 6*(2), 153–169.

Cummings, D. E., & Overduin, J. (2007). Gastrointestinal regulation of food intake. *Health Care*, *117*(1), 13–23.

Cummings, D. E., Purnell, J. ., Frayo, R. ., Schmidova, K., Wisse, B. ., & Weigle, D. . (2001). A Preprandial Rise in Plasma Ghrelin Levels Suggests a Role in Meal Initiation in Humans. *Diabetes*, *50*, 1714–1719.

Cummings, D. E., Scott Frayo, R., Marmonier, C., Aubert, R., & Chapelot, D. (2004). Plasma ghrelin levels and hunger scores in humans initiating meals voluntarily without time-and food-related cues. *American Journal of Physiology Endocrinology and Metabolism*, *287*, E297–E304.

Cummings, D. E., Weigle, D. S., Frayo, R. S., Breen, P. A., Ma, M. K., Dellinger, E. P., & Purnell, J. Q. (2002). Plasma ghrelin levels after diet-induced weight loss or gastric bypass surgery. *New England Journal of Medicine, 346*(21), 1623–

1630.

Cyders, M. A., & Coskunpinar, A. (2012). The relationship between self-report and lab task conceptualizations of impulsivity. *Journal of Research in Personality*, *46*(1), 121–124.

Daborn, C., Dibsall, L., & Lambert, N. (2005). Understanding the food related experiences and beliefs of a specific group of low-income men in the UK. *Health Education*, *105*(2), 109–125.

Dalton, M., Blundell, J. E., & 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.

Dalton, M., & Finlayson, G. (2014). Psychobiological examination of liking and wanting for fat and sweet taste in trait binge eating females. *Physiology and Behavior*, *136*, 128–134.

Dalton, M., Finlayson, G., Esdaile, E., & King, N. A. (2013). Appetite, Satiety, and Food Reward in Obese Individuals: A Behavioral Phenotype Approach. *Current Nutrition Reports*, *2*(4), 207–215.

Dalton, M., Hollingworth, S., Blundell, J. E., & Finlayson, G. (2015). Weak satiety responsiveness is a reliable trait associated with hedonic risk factors for overeating among women. *Nutrients*, *7*(9), 7421–7436.

Das, J. K., Salam, R. A., Thornburg, K. L., Prentice, A. M., Campisi, S., Lassi, Z. S., … Bhutta, Z. A. (2017). Nutrition in adolescents: physiology, metabolism, and nutritional needs. *Annals of the New York Academy of Sciences*, *1393*(1), 21–33.

Date, Y., Kojima, M., Hosoda, H., Sawaguchi, A., Mondal, M. S., Suganuma, T., … Nakazato, M. (2000). Ghrelin, a Novel Growth Hormone-Releasing Acylated

Peptide, Is Synthesized in a Distinct Endocrine Cell Type in the Gastrointestinal Tracts of Rats and Humans. *Endocrinology*, *141*(11), 4255–4261.

Davillas, A., & Benzeval, M. (2016). Alternative measures to BMI: Exploring income-related inequalities in adiposity in Great Britain. *Social Science and Medicine*, *166*, 223–232.

Davis, C. (2009). Psychobiological traits in the risk profile for overeating and weight gain. *International Journal of Obesity (2005)*, *33 Suppl 2*(S2), S49–S53.

Davis, Caroline. (2013). From Passive Overeating to “Food Addiction”: A Spectrum of Compulsion and Severity. *International Scholarly Research Notices: Obesity*, *Article ID*, 1–20.

Davis, Caroline, 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, Caroline, & Fox, J. (2008). Sensitivity to reward and body mass index (BMI): Evidence for a non-linear relationship. *Appetite*, *50*(1), 43–49.

Davis, Caroline, Patte, K., Levitan, R., Reid, C., Tweed, S., & Curtis, C. (2007). From motivation to behaviour: A model of reward sensitivity, overeating, and food preferences in the risk profile for obesity. *Appetite*, *48*(1), 12–19.

Davis, Caroline, Strachan, S., & Berkson, M. (2004). Sensitivity to reward: Implications for overeating and overweight. *Appetite*, *42*(2), 131–138.

Dawe, S., Gullo, M. J., & Loxton, N. J. (2004). Reward drive and rash impulsiveness as dimensions of impulsivity: Implications for substance misuse. *Addictive Behaviors*, *29*(7), 1389–1405.

Dawe, S., & Loxton, N. J. (2004). The role of impulsivity in the development of substance use and eating disorders. *Neuroscience and Biobehavioral Reviews*, *28*(3), 343–351.

de Abreu, D., Guessous, I., Vaucher, J., Preisig, M., Waeber, G., Vollenweider, P., & Marques-Vidal, P. (2013). Low compliance with dietary recommendations for food intake among adults. *Clinical Nutrition*, *32*(5), 783–788.

de Araujo, I. E., & Rolls, E. T. (2004). Representation in the Human Brain of Food Texture and Oral Fat. *The Journal of Neuroscience : The Official Journal of the Society for Neuroscience*, *24*(12), 3086–3093.

de Araujo, I. E. T., Rolls, E. T., Kringelbach, M. L., McGlone, F., & Phillips, N. (2003). Taste-olfactory convergence, and the representation of the pleasantness of flavour, in the human brain. *European Journal of Neuroscience*, *18*(7), 2059–2068.

De Castro, J. M. (2010). The control of food intake of free-living humans: putting the pieces back together. *Physiology & Behavior*, *100*(5), 446–453.

De Graaf, C., De Jong, L. S., & Lambers, A. C. (1999). Palatability affects satiation but not satiety. *Physiology and Behavior*, *66*(4), 681–688.

De Lauzon-Guillain, B., Basdevant, A., Romon, M., Karlsson, J., Borys, J. M., & Charles, M. A. (2006). Is restrained eating a risk factor for weight gain in a general population? *American Journal of Clinical Nutrition*, *83*(3), 132–138.

De Silva, A., & Bloom, S. R. (2012). Gut hormones and appetite control: A focus on PYY and GLP-1 as therapeutic targets in obesity. *Gut and Liver*, *6*(1), 10–20.

De Silva, P. H. G. J., Weerasinghe, W. M. C. B., & Seresinhe, T. (2011). Socio-Demographic Determinants of Perceived Influences on Household Food Choice in Galle, Kalutara, Matara and Moneragala Districts. *Pakistan Journal of Social Sciences*, *8*(3), 142–145.

Deglaire, A, Méjean, C., Castetbon, K., Hercberg, S., & Schlich, P. (2015).

Associations between weight status and liking scores for sweet , salt and fat according to the gender in adults (The Nutrinet-Santé study). *European Journal of Clinical Nutrition*, *69*, 40–46.

Deglaire, Amélie, Méjean, C., Castetbon, K., Kesse-Guyot, E., Urbano, C., Hercberg, S., & Schlich, P. (2012). Development of a questionnaire to assay recalled liking for salt, sweet and fat. *Food Quality and Preference*, *23*, 110–124.

Dekker, L. H., van Dam, R. M., Snijder, M. B., Peters, R. J., Dekker, J. M., de Vries, J. H., … Nicolaou, M. (2015). Comparable Dietary Patterns Describe Dietary Behavior across Ethnic Groups in the Netherlands, but Different Elements in the Diet Are Associated with Glycated Hemoglobin and Fasting Glucose Concentrations. *Journal of Nutrition*, *145*(8), 1884–1891.

Del Parigi, A., Chen, K., Gautier, J. F., Salbe, A. D., Pratley, R. E., Ravussin, E., … Antonio Tataranni, P. (2002). Sex differences in the human brain's response to hunger and satiation. *American Journal of Clinical Nutrition*, *75*(6), 1017–1022.

Delgado-Aros, S., Cremonini, F., Castillo, J. E., Chial, H. J., Burton, D. D., Ferber, I., & Camilleri, M. (2004). *Independent Influences of Body Mass and Gastric Volumes*. 432–440.

Den Hoed, M., Westerterp-Plantenga, M. S., Mariman, E. C. M., & Westerterp, K. R. (2009). Postprandial responses in hunger and satiety are associated with a genetic variant in the FTO gene. *Appetite*, *52*(3), 826.

Deshmukh-Taskar, P., Nicklas, T. A., Yang, S. J., & Berenson, G. S. (2007). Does Food Group Consumption Vary by Differences in Socioeconomic, Demographic, and Lifestyle Factors in Young Adults? The Bogalusa Heart

Study. *Journal of the American Dietetic Association*, *107*(2), 223–234.

Devoto, F., Zapparoli, L., Bonandrini, R., Berlingeri, M., Ferrulli, A., Luzi, L., … Paulesu, E. (2018). Hungry Brains: A Meta-Analytical Review of Brain Activation Imaging Studies On Food Perception and Appetite in Obese Individuals. *Neuroscience & Biobehavioral Reviews*.

Dibsdall, L., Lambert, N., Bobbin, R., & Frewer, L. (2003). Low-income consumers' attitudes and behaviour towards access, availability and motivation to eat fruit and vegetables. *Public Health Nutrition*, *6*(02), 159–168.

DiFeliceantonio, A. G., Coppin, G., Rigoux, L., Edwin Thanarajah, S., Dagher, A., Tittgemeyer, M., & Small, D. M. (2018). Supra-Additive Effects of Combining Fat and Carbohydrate on Food Reward. *Cell Metabolism*, *28*(1), 33-44.e3.

Dilberti, N., Bordi, P. L., Concklin, M. T., Roe, L. S., & Rolls, B. J. (2004). Increased portion size leads to increased consumption in a restaurant meal. *Obes. Res.*, *12*(3), 562.

Dina, C., Meyre, D., Gallina, S., Durand, E., Körner, A., Jacobson, P., … Froguel, P. (2007). Variation in FTO contributes to childhood obesity and severe adult obesity. *Nature Genetics*, *39*(6), 724–726.

Disse, E., Bussier, A. L., Veyrat-Durebex, C., Deblon, N., Pfluger, P. T., Tschöp, M. H., … Rohner-Jeanrenaud, F. (2010). Peripheral ghrelin enhances sweet taste food consumption and preference, regardless of its caloric content. *Physiology and Behavior*, *101*(2), 277–281.

Dit El Khoury, D. T., Obeid, O., Azar, S. T., & Hwalla, N. (2006). Variations in postprandial ghrelin status following ingestion of high-carbohydrate, high-fat, and high-protein meals in males. *Annals of Nutrition and Metabolism*, *50*(3), 260–269.

Do, R., Bailey, S. D., Desbiens, K., Belisle, A., Montpetit, A., Vohl, M., … West, P. A. (2008). *Genetic Variants of FTO Influence Adiposity , Insulin Sensitivity , Leptin Levels , and Resting Metabolic Rate in the Quebec Family Study*. *57*(April), 1147–1150.

Dobbs, R., Sawyer, C., Thompson, F., Manyika, J., Woetzel, J., Child, P., … Spatharou, A. (2014). Overcoming obesity : An initial economic analysis Executive summary Discussion paper. *The McKinsey Global Institute*, (November).

Dobson, P. W., Chakraborty, R., & Seaton, J. S. (2017). Containing big soda: Countering inducements to buy large-size sugary drinks. *Journal of Business Research*, *75*, 185–191.

Dorling, J., Broom, D. R., Burns, S. F., Clayton, D. J., Deighton, K., James, L. J., … Stensel, D. J. (2018). Acute and chronic effects of exercise on appetite, energy intake, and appetite-related hormones: The modulating effect of adiposity, sex, and habitual physical activity. *Nutrients*, *10*(9).

Dougkas, A., Yaqoob, P., Givens, D. I., Reynolds, C. K., & Minihane, A. M. (2013). The impact of obesity-related SNP on appetite and energy intake. *British Journal of Nutrition*, *110*(6), 1151–1156.

Drapeau, V., Blundell, J. E., Gallant, A. R., Arguin, H., Després, J., Lamarche, B., & Tremblay, A. (2013a). Behavioural and metabolic characterisation of the low satiety. *Appetite*, *70*, 67–72.

Drapeau, V., Blundell, J. E., Gallant, A. R., Arguin, H., Després, J. P., Lamarche, B., & Tremblay, A. (2013b). Behavioural and metabolic characterisation of the low satiety phenotype. *Appetite*, *70*, 67–72.

Drapeau, V., Hetherington, M., & Tremblay, A. (2011). Impact of eating and lifestyle behaviours on body weight: Beyond energy value. In V. R. Preedy, R. R. Watson, & C. R. Martin (Eds.), *Handbook of Behavior, Food and Nutrition* (Vol. 1, pp. 693–706).

Drapeau, V., Provencher, V., Lemieux, S., Després, J. P., Bouchard, C., & Tremblay, A. (2003). Do 6-y changes in eating behaviors predict changes in body weight? Results from the Quebec Family Study. *International Journal of Obesity*, *27*(7), 808–814.

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.

Dressler, Heidi, & Smith, C. (2013). Food choice, eating behavior, and food liking differs between lean/normal and overweight/obese, low-income women. *Appetite*, *65*, 145–152.

Drewnowski, A. (1998). Energy density, palatability, and satiety: implications for weight control. *Nutrition Reviews*, *56*(12), 347–353.

Drewnowski, A. (2007). The real contribution of added sugars and fats to obesity. *Epidemiologic Reviews*, *29*(1), 160–171.

Drewnowski, A., & Almiron-roig, E. (2010). Human Perceptions and Preferences for Fat-Rich Foods. In *Fat Detection: Taste, Texture, and Post Ingestive Effects* (pp. 1–23).

Drewnowski, A., & Darmon, N. (2005a). Symposium: Modifying the Food Environment: Energy Density, Food Costs, and Portion Size Portion Sizes and the Obesity Epidemic. *The Journal of Nutrition*, (11), 900–904.

Drewnowski, A., & Darmon, N. (2005b). The economics of obesity : dietary energy density and energy cost. *American Journal of Clinical Nutrition*, *82*((suppl)), 265S-273S.

Drewnowski, A., & Greenwood, M. R. (1983). Cream and sugar: human preferences for high-fat foods. *Physiology & Behavior*, Vol. 30, pp. 629–633.

Drewnowski, A., Henderson, S. A., Shore, A. B., & Barratt-Fornell, A. (1997). Nontasters, tasters, and supertasters of 6-n-propylthiouracil (PROP) and hedonic response to sweet. *Physiology and Behavior*, *62*(3), 649–655.

Drewnowski, A., Krahn, D. D., Demitrack, M. A., Nairn, K., & Gosnell, B. A. (1992). Taste responses and preferences for sweet high-fat foods: Evidence for opioid involvement. *Physiology and Behavior*, *51*(2), 371–379.

Drewnowski, A., Kurth, C., Holden-Wiltse, J., & Saari, J. (1992). Food preferences in human obesity: carbohydrates versus fats. *Appetite*, *18*, 207–221.

Drewnowski, A., Mennella, J. A., Johnson, S. L., & Bellisle, F. (2012). Sweetness and Food Preference. *The Journal of Nutrition*, *142*(7), 1142–1148.

Drewnowski, A., & Schwartz, M. (1990). Invisible fats: sensory assessment of sugar/fat mixtures. *Appetite*, *14*, 203–217.

Drewnowski, A., Shrager, E. E., Lipsky, C., Stellar, E., & Greenwood, M. R. (1989). Sugar and fat: sensory and hedonic evaluation of liquid and solid foods. *Physiology & Behavior*, *45*(16), 177–183.

Duffey, K. J., & Popkin, B. M. (2011). Energy density, portion size, and eating occasions: Contributions to increased energy intake in the United States, 1977-2006. *PLoS Medicine*, *8*(6).

Dulloo, A. G., Jacquet, J., Miles-Chan, J. L., & Schutz, Y. (2017). Passive and active roles of fat-free mass in the control of energy intake and body composition regulation. *European Journal of Clinical Nutrition*, *71*(3), 353–357.

Durand, M. A., Petticrew, M., Goulding, L., Eastmure, E., Knai, C., & Mays, N.

(2015). An evaluation of the Public Health Responsibility Deal: Informants' experiences and views of the development, implementation and achievements of a pledge-based, public-private partnership to improve population health in England. *Health Policy*, *119*(11), 1506–1514.

Dyck, D. J. (2005). Leptin sensitivity in skeletal muscle is modulated by diet and exercise. *Exercise and Sport Sciences Reviews*, *33*(4), 189–194.

Dykes, J., Brunner, E. J., Martikainen, P. T., & Wardle, J. (2004). Socioeconomic gradient in body size and obesity among women: The role of dietary restraint, disinhibition and hunger in the Whitehall II study. *International Journal of Obesity*, *28*(2), 262–268.

Economic & Social Research Council. (2014). Food price promotions and public health. https://esrc.ukri.org/news-events-and-publications/evidence-briefings/food-price-promotions-and-public-health/. Accesssed 7th September 2019

Edholm, O. G., Adam, J. M., Healy, M. J. R., Wolff, H. S., Goldsmith, R., & Best, T. W. (1970). Food Intake and Energy Expenditure of Army Recruits. *British Journal of Nutrition*, *24*(1970), 1091–1107.

Elfhag, K., Tynelius, P., & Rasmussen, F. (2007). Sugar-sweetened and artificially sweetened soft drinks in association to restrained, external and emotional eating. *Physiology and Behavior*, *91*(2–3), 191–195.

Elia, M., Ritz, P., & Stubbs, R. J. (2000). Total energy expenditure in the elderly. *European Journal of Clinical Nutrition*, *54*, S92–S103.

Ello-martin, J. A., Ledikwe, J. H., & Rolls, B. J. (2005). The influence of food portion size and energy density on energy intake : implications for weight management 1 – 4. *Obesity Research*, *82*, 236–241.

English, P. J.; Ghatei, M. A.; Malik, I. A.; Bloom, S. R. (2002). Food Fails to Suppress Ghrelin Levels in Obese Humans. *The Journal of Clinical Endocrinology & Metabolism*, *87*(6), 2984–2987.

English, L., Lasschuijt, M., & Keller, K. L. (2015). Mechanisms of the portion size effect. What is known and where do we go from here? *Appetite*, *88*, 39–49.

Epstein, L.H., Carr, K. A., Lin, H., & Fletcher, K. D. (2011). Food reinforcement, energy intake, and macronutrient choice. *American Journal of Clinical Nutrition*, *94*(1), 12–18.

Epstein, L.H., Jankowiak, N., Fletcher, K. D., Carr, K. A., Nederkoorn, C., Raynor, H. A., & Finkelstein, E. (2014). Women who are motivated to eat and discount the future are more obese. *Obesity*, *22*(6), 1394–1399.

Epstein, L.H., & Leddy, J. J. (2006). Food reinforcement. *Appetite*, *46*(1), 22–25.

Epstein, L.H., Lin, H., Carr, K. A., & Fletcher, K. D. (2012). Food reinforcement and obesity. Psychological moderators. *Appetite*, *58*(1), 157–162.

Epstein, L.H., Salvy, S. J., Carr, K. A., Dearing, K. K., & Bickel, W. K. (2010). Food reinforcement, delay discounting and obesity. *Physiology and Behavior*, *100*(5), 438–445.

Epstein, Leonard H., Paluch, R. A., Carr, K. A., Temple, J. L., Bickel, W. K., & MacKillop, J. (2018). Reinforcing value and hypothetical behavioral economic demand for food and their relation to BMI. *Eating Behaviors*, *29*(March), 120–127.

Epstein, Leonard H, Leddy, J. J., Temple, J. L., Faith, M. S., & Epstein, Leonard H.; Leddy, J.J.; Temple, J.L.; Faith, M. S. (2007). Food reinforcement and eating: A multilevel analysis. *Psychological Bulletin*, *133*(5), 884–906.

Erlanson-Albertsson, C. (2005). How Palatable Food Disrupts Appetite Regulation.

Basic & Clinical Pharmacology & Toxicology, *97*(2), 61–73.

European Food Information Council. (2012). Fruit and vegetable consumption in Europe – do Europeans get enough? https://www.eufic.org/en/healthy-living/article/fruit-and-vegetable-consumption-in-europe-do-europeans-get-enough. Accessed 16th January 2019

Evans, D. E., & Rothbart, M. K. (2007). Developing a model for adult temperament. *Journal of Research in Personality*, *41*(4), 868–888.

Faulkner, G. P., Livingstone, M. B. E., Pourshahidi, L. K., Spence, M., Dean, M., O'Brien, S., … Kerr, M. A. (2017). An evaluation of portion size estimation aids: Consumer perspectives on their effectiveness. *Appetite*, *114*, 200–208.

Fay, S.H., Finlayson, G., & King, N. A. (2012). Diet-Induced Obesity: When Does Consumption Become Overconsumption? *Current Obesity Reports*, 104–106.

Fay, Stephanie H., Ferriday, D., Hinton, E. C., Shakeshaft, N. G., Rogers, P. J., & Brunstrom, J. M. (2011). What determines real-world meal size? Evidence for pre-meal planning. *Appetite*, *56*(2), 284–289.

Fedoroff, I., Polivy, J., & Herman, C. P. (2003). The specificity of restrained versus unrestrained eaters' responses to food cues: General desire to eat, or craving for the cued food? *Appetite*, *41*(1), 7–13.

Felton, A. M. (2008). The Nutritional Ecology of Spider Monkeys (Ateles chamek) in the Context of Reduced-Impact Logging (Doctoral dissertation). *Fenner School of Environment and Society, Australian National University*, p. 163.

Fernstrom, J. D. (2005). Branched-Chain Amino Acids and Brain Function. *Journal of Nutrition*, (7), 1585–1590.

Ferriday, D., & Brunstrom, J. M. (2011). 'I just can't help myself': effects of food-cue exposure in overweight and lean individuals. *International Journal of*

Obesity, *35*(1), 142–149.

Field, A. (2013). Exploring Data:The Beast of Bias Sources of Bias Outliers. In *Discovering Statistics Using IBM SPSS Statistics* (4th editio, pp. 1–21). Sage Publications.

Fields, S. A., Sabet, M., & Reynolds, B. (2013). Dimensions of impulsive behavior in obese, overweight, and healthy-weight adolescents. *Appetite*, *70*, 60–66.

Finch, L. E., & Tomiyama, A. J. (2015). Comfort eating, psychological stress, and depressive symptoms in young adult women. *Appetite*, *95*, 239–244.

Finegood, D. T., Merth, T. D. N., & Rutter, H. (2010). Implications of the foresight obesity system map for solutions to childhood obesity. *Obesity*, *18*(Suppl 1), 13–16.

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., Cecil, J., Higgs, S., Hill, A., & Hetherington, M. (2012). Susceptibility to weight gain. Eating behaviour traits and physical activity as predictors of weight gain during the first year of university. *Appetite*, *58*(3), 1091–1098.

Finlayson, G., & Dalton, M. (2012a). Current progress in the assessment of “liking” vs. “wanting” food in human appetite. Comment on “ ‘You Say it’s Liking, I Say it’s Wanting...’ On the difficulty of disentangling food reward in man.” *Appetite*, *58*(1), 373–378.

Finlayson, G., & Dalton, M. (2012b). Hedonics of Food Consumption: Are Food ‘Liking’ and ‘Wanting’ Viable Targets for Appetite Control in the Obese? *Current Obesity Reports*, *1*(1), 42–49.

Finlayson, G., King, N. A., & Blundell, J. E. (2007a). Is it possible to dissociate “liking” and “wanting” for foods in humans? A novel experimental procedure. *Physiology and Behavior*, *90*(1), 36–42.

Finlayson, G., King, N. A., & Blundell, J. E. (2007b). Liking vs. wanting food: importance for human appetite control and weight regulation. *Neuroscience and Biobehavioral Reviews*, *31*(7), 987–1002.

Finlayson, G., King, N. A., & Blundell, J. E. (2008). The role of implicit wanting in relation to explicit liking and wanting for food: Implications for appetite control. *Appetite*, *50*, 120–127.

Firk, C., & Markus, C. R. (2009). Mood and cortisol responses following tryptophan-rich hydrolyzed protein and acute stress in healthy subjects with high and low cognitive reactivity to depression. *Clinical Nutrition*, *28*(3), 266–271.

Flegal, K. M., Shepherd, J. A., Looker, A. C., Graubard, B. I., Borrud, L. G., Ogden, C. L., … Schenker, N. (2009). Comparisons of percentage body fat, body mass index, waist circumference, and waist-stature ratio in adults. *The American Journal of Clinical Nutrition*, *89*(3), 500–508.

Flint, S. W., & Oliver, E. J. (2019). Seven-year itch: The UK Government’s difficult relationship with the food and drink industry since Healthy Lives, Healthy People: A Call to Action on Obesity in England (2011). *Public Health Nutrition*, *22*(7), 1326–1329.

Fogelholm, M., Anderssen, S., Gunnarsdottir, I., & Lahti-Koski, M. (2012). Dietary macronutrients and food consumption as determinants of long-term weight change in adult populations: a systematic literature review. *Food & Nutrition Research*, *56*(1), 19103.

Foong Fong Chong, M., Ayob, M. N. im M., Chong, K. J., Tai, E. S., Khoo, C. M., Leow, M. K. S., … Khoo, E. Y. H. (2016). Psychometric analysis of an eating behaviour questionnaire for an overweight and obese Chinese population in Singapore. *Appetite*, *101*, 119–124.

Forbes. (2019). The World's Largest Food and Restaurant Companies in 2019. https://www.forbes.com/sites/chloesorvino/2019/05/15/worlds-largest-food-restaurant-companies-2019/. Date accessed 3rd October 2019

Ford, E.S., & Dietz, W. H. (2016). Trends in energy intake among adults in the United States: findings from NHANES. *American Journal of Clinical Nutrition, 118*(24), 6072–6078.

Ford, Earl S., & Mokdad, A. H. (2008). Epidemiology of obesity in the Western Hemisphere. *Journal of Clinical Endocrinology and Metabolism*, *93*(November 2008), 1–8.

Foresight. (2007). Tackling Obesities : Future Choices – Building the Obesity System Map. https://www.gov.uk/government/publications/reducing-obesity-obesity-system-map. Accessed 3rd October 2019

Forhan, M., & Gill, S. V. (2013). Obesity, functional mobility and quality of life. *Best Practice & Research Clinical Endocrinology & Metabolism*, *27*(2), 129–137.

Foster-Schubert, K. E., Overduin, J., Prudom, C. E., Liu, J., Callahan, H. S., Gaylinn, B. D., … Cummings, D. E. (2008). Acyl and total ghrelin are suppressed strongly by ingested proteins, weakly by lipids, and biphasically by carbohydrates. *Journal of Clinical Endocrinology and Metabolism*, *93*(5), 1971–1979.

Francaux, M., & Deldicque, L. (2019). Exercise and the control of muscle mass in human. *Pflugers Archiv European Journal of Physiology*, *471*, 397–411.

Franken, I. H. A., & Muris, P. (2005). Individual differences in reward sensitivity are related to food craving and relative body weight in healthy women. *Appetite*, *45*(2), 198–201.

Frankort, A., Roefs, A., Siep, N., Roebroeck, A., Havermans, R., & Jansen, A. (2012). Reward activity in satiated overweight women is decreased during unbiased viewing but increased when imagining taste: An event-related fMRI study. *International Journal of Obesity*, *36*(5), 627–637.

Fraser, G. E., Welch, A., Luben, R., Bingham, S. A., & Day, N. E. (2000). The effect of age, sex, and education on food consumption of a middle- aged English cohort - EPIC in East Anglia. *Preventive Medicine*, *30*(1), 26–34.

Frayling, T. M., Timpson, N. J., Weedon, M. N., Freathy, R. M., Lindgren, C. M., Perry, J. R. B., … Mccarthy, M. I. (2007). A Common Variant in the FTO Gene Is Associated with Body Mass Index and Predisposes to Childhood and Adult Obesity. *Science*, *316*(5826), 889–894.

Frecka, J. M., & Mattes, R. D. (2008). Possible entrainment of ghrelin to habitual meal patterns in humans. *AJP: Gastrointestinal and Liver Physiology*, *294*(3), G699–G707.

Fredriksson, R., Hägglund, M., Olszewski, P. K., Stephansson, O., Jacobsson, J. A., Olszewska, A. M., … Schiöth, H. B. (2008). The obesity gene, FTO, is of ancient origin, up-regulated during food deprivation and expressed in neurons of feeding-related nuclei of the brain. *Endocrinology*, *149*(5), 2062–2071.

French, S. A., Epstein, L. H., Jeffery, R. W., Blundell, J. E., & Wardle, J. (2012). Eating behavior dimensions. Associations with energy intake and body weight. A review. *Appetite*, *59*(2), 541–549.

French, S. A., Harnack, L., & Jeffery, R. W. (2000). Fast food restaurant use among women in the pound of prevention study: Dietary, behavioral and demographic correlates. *International Journal of Obesity*, *24*(10), 1353–1359.

French, S. A., Mitchell, N. R., Finlayson, G., Blundell, J. E., & Jeffery, R. W. (2014). Questionnaire and laboratory measures of eating behavior: Associations with energy intake and BMI in a community sample of working adults. *Appetite*, *72*, 50–58.

French, S. A., Mitchell, N. R., Wolfson, J., Harnack, L. J., Jeffery, R. W., Gerlach, A. F., … Pentel, P. R. (2014). Portion Size Effects on Weight Gain in a Free Living Setting. *Obesity*, *22*, 1400–1405.

Friel, S., Newell, J., & Kelleher, C. (2005). Who eats four or more servings of fruit and vegetables per day? Multivariate classification tree analysis of data from the 1998 Survey of Lifestyle, Attitudes and Nutrition in the Republic of Ireland. *Public Health Nutrition*, *8*(02), 159–169.

Fukagawa, N. K., & Yu, Y.-M. (2009). Nutrition and Metabolism of Proteins and Amino Acids. In M. Gibney (Ed.), *Introduction to Human Nutrition* (2nd Ed, pp. 49–73). Wiley-Blackwell.

Gan, L., England, E., Yang, J. Y., Toulme, N., Ambati, S., Hartzell, D. L., … Baile, C. A. (2015). A 72-hour high fat diet increases transcript levels of the neuropeptide galanin in the dorsal hippocampus of the rat. *BMC Neuroscience*, *16*(1), 1–10.

Garneau, N. L., Nuessle, T. M., Mendelsberg, B. J., Shepard, S., & Tucker, R. M. (2018). Sweet liker status in children and adults: Consequences for beverage intake in adults. *Food Quality and Preference*, *65*(July 2017), 175–180.

Gautier, J., Chen, K., Salbe, A. D., Bandy, D., Pratley, R. E., Heiman, M., …

Tataranni, P. A. (2000). Differential brain responses to satiation in obese and lean mean. *Diabetes*, *49*, 838–846.

Gautier, J. F., Del Parigi, A., Chen, K., Salbe, A. D., Bandy, D., Pratley, R. E., … Tataranni, P. A. (2001). Effect of satiation on brain activity in obese and lean women. *Obesity Research*, *9*(11), 676–684.

Gazan, R., Béchaux, C., Crépet, A., Sirot, V., Drouillet-Pinard, P., Dubuisson, C., & Havard, S. (2016). Dietary patterns in the French adult population: A study from the second French national cross-sectional dietary survey (INCA2) (2006-2007). *British Journal of Nutrition*, *116*(2), 300–315.

Gearhardt, A. N., Rizk, M. T., & Treat, T. A. (2014). The association of food characteristics and individual differences with ratings of craving and liking. *Appetite*, *79*, 166–173.

Geisser, S., & Greenhouse, S. W. (1959). On methods in the analysis of profile data. *Psychometrika*, *24*(2), 95–111.

Genton, L., Melzer, K., & Pichard, C. (2010). Energy and macronutrient requirements for physical fitness in exercising subjects. *Clinical Nutrition*, *29*(4), 413–423.

Gerlach, G., Herpertz, S., & Loeber, S. (2015). Personality traits and obesity: a systematic review. *Obesity Reviews*, *16*(1), 32–63.

Giallonardo, T., & Warburton, D. E. R. (2016). Sit less, move more. *Health and Fitness Journal of Canada*, *9*(1), 15–19.

Gibbons, C., Caudwell, P., Finlayson, G., Webb, D. L., Hellström, P. M., Näslund, E., & Blundell, J. E. (2013). Comparison of postprandial profiles of ghrelin, active GLP-1, and total PYY to meals varying in fat and carbohydrate and their association with hunger and the phases of satiety. *Journal of Clinical*

Endocrinology and Metabolism, *98*(5), 847–855.

Gibson, E. L. (2006). Emotional influences on food choice: Sensory, physiological and psychological pathways. *Physiology and Behavior, 89*, 53–61.

Gibson, E. L. (2011). Emotional and Behavioral Aspects of Chocolate Eating. In Victor R Preedy, R. R. Watson, & C. R. Martin (Eds.), *Handbook of Behavior, Food and Nutrition* (pp. 601–620). New York, NY: Springer New York.

Gibson, E. L. (2012). The psychobiology of comfort eating: implications for neuropharmacological interventions. *Behavioural Pharmacology*, *23*(5–6), 442–460.

Gibson, E. L., Barr, S., & Jeanes, Y. M. (2011). Habitual fat intake predicts cognitive function in young women. *Appetite, 57*(2), 542.

Gibson, E. L., & Booth, D. A. (1986). Acquired protein appetite in rats: Dependence on a protein-specific need state. *Experiential*, *42*, 1003–1004.

Gibson, E. L., & Brunstrom, J. M. (2007). Learned Influences on Appetite, Food Choice, and Intake: Evidence in Human Beings. In T. Kirkham & S. Cooper (Eds.), *Appetite and Body Weight: Integrative Systems and the Development of Anti-Obesity Drugs* (pp. 271–300). Academic Press.

Gibson, E. L., Checkley, S., Papadopoulos, A., Poon, L., Daley, S., & Wardle, J. (1999). Increased salivary cortisol reliably induced by a protein-rich midday meal. *Psychosomatic Medicine*, *61*(2), 214–224.

Gibson, E. L., Latif, R. H., Laurens, A., & Nercessian, A. (2008). Overeating by sweetening a fat-rich food: Independence from postingestive cholescystokinin and insulin release. *Appetite, 51*(2), 368 (Abstract only).

Gibson, E. L., Vargas, K., Hogan, E., Holmes, A., Rogers, P. J., Wittwer, J., … Mohajeri, M. H. (2014). Effects of acute treatment with a tryptophan-rich protein hydrolysate on plasma amino acids, mood and emotional functioning in older women. *Psychopharmacology*, *231*(24), 4595–4610.

Gibson, E. L., Wainwright, C. J., & Booth, D. A. (1995). Disguised protein in lunch after low-protein breakfast conditions food-flavor preferences dependent on recent lack of protein intake. *Physiology & Behavior*, *58*(2), 363–371.

Gibson, E. L., & Wardle, J. (2003). Energy density predicts preferences for fruit and vegetables in 4-year-old children. *Appetite*, *41*(1), 97–98.

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.

Gietzen, D. W., & Aja, S. M. (2012). The brain's response to an essential amino acid-deficient diet and the circuitous route to a better meal. *Molecular Neurobiology*, *46*(2), 332–348.

Gietzen, D. W., & Rogers, Q. R. (2006). Nutritional homeostasis and indispensable amino acid sensing: A new solution to an old puzzle. *Trends in Neurosciences*, *29*(2), 91–99.

Gille, D., Bütikofer, U., Chollet, M., Schmid, A., Altintzoglou, T., Honkanen, P., … Piccinali, P. (2016). Nutrition behavior of the middle-aged and elderly: Compliance with dietary recommendations of the food pyramid. *Clinical Nutrition*, *35*(3), 638–644.

Giskes, K., van Lenthe, F., Avendano-Pabon, M., & Brug, J. (2011). A systematic review of environmental factors and obesogenic dietary intakes among adults: Are we getting closer to understanding obesogenic environments? *Obesity Reviews*, *12*(501), 95–106.

Glanz, K., Basil, M., Maibach, E., Goldberg, J., & Snyder, D. (1998). Why

Americans eat what they do: Taste, nutrition, cost, convenience, and weight control concerns as influences on food consumption. *Journal of the American Dietetic Association*, *98*(10), 1118–1126.

Goldfield, G. S., & Lumb, A. (2008). Smoking, dietary restraint, gender, and the relative reinforcing value of snack food in a large university sample. *Appetite*, *50*(2–3), 278–289.

Goldstone, A. P., Prechtl, C. G., Scholtz, S., Miras, A. D., Chhina, N., Durighel, G., … Bell, J. D. (2014). Ghrelin mimics fasting to enhance human hedonic, orbitofrontal cortex, and hippocampal responses to food. *American Journal of Clinical Nutrition*, *99*(6), 1319–1330.

Goldstone, A. P., Prechtl de Hernandez, C. G., Beaver, J. D., Muhammed, K., Croese, C., Bell, G., … Bell, J. D. (2009). Fasting biases brain reward systems towards high-calorie foods. *The European Journal of Neuroscience*, *30*(8), 1625–1635.

Gomez-Bruton, A., Arenaza, L., Medrano, M., Mora-Gonzalez, J., Cadenas-Sanchez, C., Migueles, J. H., … Labayen, I. (2019). Associations of dietary energy density with body composition and cardiometabolic risk in children with overweight and obesity: role of energy density calculations, under-reporting energy intake and physical activity. *British Journal of Nutrition*, 1–28.

Goodyear, L. J., & Kahn, B. B. (1998). Exercise, glucose transport and insulin sensitivity. *Annual Review of Medicine*, *49*(1), 235–261.

Gosby, A. K., Conigrave, A. D., Lau, N. S., Iglesias, M. A., Hall, R. M., Jebb, S. A., … Simpson, S. J. (2011). Testing protein leverage in lean humans: a randomised controlled experimental study. *PloS One*, *6*(10), e25929.

Gosby, A. K., Conigrave, A. D., Raubenheimer, D., & Simpson, S. J. (2014). Protein leverage and energy intake. *Obesity Reviews : An Official Journal of the International Association for the Study of Obesity*, *15*(3), 183–191.

Gouvea, A., Rodrigues, M., Pacheco, R., Cristina, M., Calvo, M., Medeiros, G., & Fiates, R. (2012). Overweight / obesity is associated with food choices related to rice and beans , colors of salads , and portion size among consumers at a restaurant serving buffet-by-weight in Brazil. *Appetite*, *59*(2), 305–311.

Graaf, C. De, & Frijters, J. E. R. (1989). Interrelationships among sweetness, saltiness and total taste intensity of sucrose, NaCl and sucrose/NaCl mixtures. *Chemical Senses*, *14*(1), 81–102.

Grabenhorst, F., Rolls, E. T., Parris, B. A., & D'Souza, A. A. (2010). How the brain represents the reward value of fat in the mouth. *Cerebral Cortex*, *20*(5), 1082–1091.

Gray, J. A., & Mcnaughton, N. (2000). *The Neuropsychology of anxiety: An enquiry into the functions of the septo-hippocampal system 2nd ed* (N. J. Mackintosh, T. Shallice, A. Treisman, J. L. McGaugh, D. Schacter, & L. Weiskrantz, Eds.). Oxford University Press.

Green, E., & Murphy, C. (2012). Altered processing of sweet taste in the brain of diet soda drinkers. *Physiology and Behavior*, *107*(4), 560–567.

Green, S. M., Delargy, H. J., Joanes, D., & Blundell, J. E. (1997). A satiety quotient: a formulation to assess the satiating effect of food. *Appetite*, *29*(3), 291–304.

Green, S. M., Wales, J. K., Lawton, C. L., & Blundell, J. E. (2000). Comparison of high-fat and high-carbohydrate foods in a meal or snack on short-term fat and energy intakes in obese women. *British Journal of Nutrition*, *84*(June 1997), 521–530.

Gregersen, N. T., Flint, A., Bitz, C., Blundell, J. E., Raben, A., & Astrup, A. (2008).

Reproducibility and power of ad libitum energy intake assessed by repeated single meals. *American Journal of Clinical Nutrition*, *87*(5), 1277–1281.

Gregersen, N. T., Møller, B. K., Raben, A., Kristensen, S. T., Holm, L., Flint, A., & Astrup, A. (2011). Determinants of appetite ratings: The role of age, gender, BMI, physical activity, smoking habits, and diet/weight concern. *Food and Nutrition Research*, *55*, 1–10.

Griffioen-Roose, S., Finlayson, G., Mars, M., Blundell, J. E., & de Graaf, C. (2010). Measuring food reward and the transfer effect of sensory specific satiety. *Appetite*, *55*(3), 648–655.

Griffioen-Roose, S., Mars, M., Finlayson, G., Blundell, J. E., & de Graaf, C. (2011). The effect of within-meal protein content and taste on subsequent food choice and satiety. *British Journal of Nutrition*, *106*(5), 779–788.

Griffioen-Roose, S., Mars, M., Siebelink, E., Finlayson, G., Tomé, D., & De Graaf, C. (2012). Protein status elicits compensatory changes in food intake and food preferences. *American Journal of Clinical Nutrition*, *95*(1), 32–38.

Griffioen-Roose, S., Smeets, P. A. M., Van Den Heuvel, E., Boesveldt, S., Finlayson, G., & De Graaf, C. (2014). Human Protein status modulates brain reward responses to food cues. *American Journal of Clinical Nutrition*, *100*(1), 113–122.

Grimm, E. R., & Steinle, N. I. (2011). Genetics of eating behavior: Established and emerging concepts. *Nutrition Reviews*, *69*(1), 52–60.

Guan, X. M., Yu, H., Palyha, O. C., McKee, K. K., Feighner, S. D., Sirinathsinghji, D. J. S., … Howard, A. D. (1997). Distribution of mRNA encoding the growth hormone secretagogue receptor in brain and peripheral tissues. *Molecular Brain Research*, *48*(1), 23–29.

Guelfi, K. J., Donges, C. E., & Duffield, R. (2013). Beneficial effects of 12 weeks of aerobic compared with resistance exercise training on perceived appetite in previously sedentary overweight and obese men. *Metabolism: Clinical and Experimental*, *62*(2), 235–243.

Guerrieri, R., Nederkoorn, C., & Jansen, A. (2008). The interaction between impulsivity and a varied food environment: its influence on food intake and overweight. *International Journal of Obesity*, *32*, 708–714.

Guerrieri, Ramona, Nederkoorn, C., & Jansen, A. (2007). How impulsiveness and variety influence food intake in a sample of healthy women. *Appetite*, *48*(1), 119–122.

Guerrieri, Ramona, Nederkoorn, C., Stankiewicz, K., Alberts, H., Geschwind, N., Martijn, C., & Jansen, A. (2007). The influence of trait and induced state impulsivity on food intake in normal-weight healthy women. *Appetite*, *49*(1), 66–73.

Habhab, S., Sheldon, J. P., & Loeb, R. C. (2009). The relationship between stress, dietary restraint, and food preferences in women. *Appetite*, *52*(2), 437–444.

Hajnal, A., Smith, G. P., & Norgren, R. (2004). Oral sucrose stimulation increases accumbens dopamine in the rat. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology*, *286*(1), R31–R37.

Hall, K.D. (2019). The potential role of protein leverage in the US obesity epidemic. *Obesity*, *27*, 1222–1224.

Hall, K.D., Ayuketah, A., Bernstein, S., Brychta, R., Cai, H., Cassimatis, T., … Zhou, M. (2019). Ultra-processed diets cause excess calorie intake and weight gain: A one-month inpatient randomized controlled trial of ad libitum food intake.

Hall, Kevin D. (2018). Did the Food Environment Cause the Obesity Epidemic? *Obesity*, *26*(1), 11–13.

Hall, Kevin D. (2019). The Potential Role of Protein Leverage in the US Obesity Epidemic. *Obesity*.

Hall, Kevin D., Heymsfield, S. B., Kemnitz, J. W., Klein, S., Schoeller, D. A., & Speakman, J. R. (2012). Energy balance and its components: Implications for body weight regulation. *American Journal of Clinical Nutrition*, *95*(4), 989–994.

Halton, T. L., & Hu, F. B. (2004). The Effects of High Protein Diets on Thermogenesis, Satiety and Weight Loss: A Critical Review. *Journal of the American College of Nutrition*, *23*(5), 373–385.

Hao, S., Ross-Inta, C. M., & Gietzen, D. W. (2010). The sensing of essential amino acid deficiency in the anterior piriform cortex, that requires the uncharged tRNA/GCN2 pathway, is sensitive to wortmannin but not rapamycin. *Pharmacology Biochemistry and Behavior*, *94*(3), 333–340.

Harbron, J., Van der Merwe, L., Zaahl, M. G., Kotze, M. J., & Senekal, M. (2014). Fat mass and obesity-associated (FTO) gene polymorphisms are associated with physical activity, food intake, eating behaviors, psychological health, and modeled change in body mass index in overweight/obese caucasian adults. *Nutrients*, *6*(8), 3130–3152.

Hardikar, S., Höchenberger, R., Villringer, A., & Ohla, K. (2017). Higher sensitivity to sweet and salty taste in obese compared to lean individuals. *Appetite*, *111*, 158–165.

Haryono, R. Y., Sprajcer, M. A., & Keast, R. S. J. (2014). Measuring Oral Fatty Acid Thresholds, Fat Perception, Fatty Food Liking, and Papillae Density in

Humans Video Link. *Journal of Visualized Experiments*, *88*, e51236.

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), 286–294.

Havermans, R. C., Giesen, J. C. A. H., Houben, K., & Jansen, A. (2011). Weight, gender, and snack appeal. *Eating Behaviors*, *12*(2), 126–130.

Hayes, J. E., & Duffy, V. B. (2008). Oral sensory phenotype identifies level of sugar and fat required for maximal liking. *Physiology and Behavior*, *3*(95(1-2)), 77–87.

Hays, N. P., Bathalon, G. P., McCrory, M. A., Roberts, S. B., Roubenoff, R., & Lipman, R. (2002). Eating behavior correlates of adult weight gain and obesity in healthy women aged 55–65 y. *The American Journal of Clinical Nutrition*, *75*(3), 476–483.

Hays, N. P., & Roberts, S. B. (2008). Aspects of Eating Behaviors “Disinhibition” and “Restraint” Are Related to Weight Gain and BMI in Women. *Obesity*, *16*(1), 52–58.

Heitmann, B. L., & Lissner, L. (1995). Dietary underreporting by obese individuals - is it specific or non-specific? *British Medical Journal*, *311*, 986–989.

Herman, C. P., Polivy, J., Vartanian, L. R., & Pliner, P. (2016). Are large portions responsible for the obesity epidemic? *Physiology and Behavior*, Vol. 156, pp. 177–181. Elsevier Inc.

Hetherington, M. M., Blundell-Birtill, P., Caton, S. J., Cecil, J. E., Evans, C. E., Rolls, B. J., & Tang, T. (2018). Understanding the science of portion control and the art of downsizing. *Proceedings of the Nutrition Society*, *77*(3), 347–355.

Hetherington, M. M., & Rolls, B. J. (2018). Favouring more rigour when investigating human eating behaviour is like supporting motherhood and apple pie: A response to Robinson, Bevelander, Field, and Jones (2018). *Appetite*, *130*(April), 330–333.

Hill, C. M., Berthoud, H. R., Münzberg, H., & Morrison, C. D. (2018). Homeostatic sensing of dietary protein restriction: A case for FGF21. *Frontiers in Neuroendocrinology*, *51*(May), 125–131.

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 7-10-y-old children. *American Journal of Clinical Nutrition*, *90*(2), 276–281.

Hill, J. O. (2006). Understanding and addressing the epidemic of obesity: An energy balance perspective. *Endocrine Reviews*, *27*(7), 750–761.

Hill, J. O., Wyatt, H. R., & Peters, J. C. (2012). Energy Balance and Obesity. *Circulation*, *126*, 126–132.

Hinney, A., Nguyen, T. T., Scherag, A., Friedel, S., Brönner, G., Müller, T. D., … Hebebrand, J. (2007). Genome Wide Association (GWA) study for early onset extreme obesity supports the role of fat mass and obesity associated gene (FTO) variants. *PLoS ONE*, *2*(12).

Hinton, E. C., Brunstrom, J. M., Fay, S. H., Wilkinson, L. L., Ferriday, D., Rogers, P. J., & De Wijk, R. (2013). Using photography in “The Restaurant of the Future”: A useful way to assess portion selection and plate cleaning? *Appetite*, *63*, 31–35.

Hollands, G. J., Shemilt, I., Marteau, T. M., Jebb, S. A., Lewis, H. B., Wei, Y., … Ogilvie, D. (2017). Portion, package or tableware size for changing selection and consumption of food, alcohol and tobacco (Review). *Cochrane Database Systematic Reviews*, (9), CD011045.

Holmes, M. D., Dalal, S., Sewram, V., Diamond, M. B., Adebamowo, S. N., Ajayi,

I. O., … Fung, T. T. (2018). Consumption of processed food dietary patterns in four African populations. *Public Health Nutrition*, *21*(8), 1529–1537.

Hopkins, M., & Blundell, J. E. (2016). Energy balance, body composition, sedentariness and appetite regulation: pathways to obesity. *Clinical Science*, *130*(18), 1615–1628.

Hopkins, M., Finlayson, G., Duarte, C., Whybrow, S., Ritz, P., Horgan, G. W., … Stubbs, R. J. (2016). Modelling the associations between fat-free mass, resting metabolic rate and energy intake in the context of total energy balance. *International Journal of Obesity*, *40*(2), 312–318.

Hopkins, Mark, Beaulieu, K., Myers, A., Gibbons, C., & Blundell, J. E. (2017, November 2). Mechanisms responsible for homeostatic appetite control: theoretical advances and practical implications. *Expert Review of Endocrinology and Metabolism*, Vol. 12, pp. 401–415. Taylor and Francis Ltd.

Horner, K. M., Byrne, N. M., Cleghorn, G. J., & King, N. A. (2015). Influence of habitual physical activity on gastric emptying in healthy males and relationships with body composition and energy expenditure. *British Journal of Nutrition*, *114*(3), 489–496.

Hou, R., Mogg, K., Bradley, B. P., Moss-Morris, R., Peveler, R., & Roefs, A. (2011). External eating, impulsivity and attentional bias to food cues. *Appetite*, *56*(2), 424–427.

Hou, Z., Miao, Y., Gao, L., Pan, H., & Zhu, S. (2006). Ghrelin-containing neuron in cerebral cortex and hypothalamus linked with the DVC of brainstem in rat. *Regulatory Peptides*, *134*(2–3), 126–131.

Houben, K., Nederkoorn, C., & Jansen, A. (2012). Too tempting to resist? Past success at weight control rather than dietary restraint determines exposure-induced disinhibited eating. *Appetite*, *59*(2), 550–555.

Houben, K., Nederkoorn, C., & Jansen, A. (2014). Eating on impulse: The relation between overweight and food-specific inhibitory control. *Obesity*, *22*(5), 2013–2015.

Houben, K., Roefs, A., & Jansen, A. (2010). Guilty pleasures. Implicit preferences for high calorie food in restrained eating. *Appetite*, *55*(1), 18–24.

Howarth, N. C., Huang, T. T. K., Roberts, S. B., Lin, B. H., & McCrory, M. A. (2007). Eating patterns and dietary composition in relation to BMI in younger and older adults. *International Journal of Obesity*, *31*(4), 675–684.

Hsieh, S. D., & Muto, T. (2005). The superiority of waist-to-height ratio as an anthropometric index to evaluate clustering of coronary risk factors among non-obese men and women. *Preventive Medicine*, *40*(2), 216–220.

Hsu, T. M., Suarez, A. N., & Kanoski, S. E. (2016). Ghrelin: A link between memory and ingestive behavior. *Physiology and Behavior*, *162*, 1–8.

Huang, X. F., Han, M., South, T., & Storlien, L. (2003). Altered levels of POMC, AgRP and MC4-R mRNA expression in the hypothalamus and other parts of the limbic system of mice prone or resistant to chronic high-energy diet-induced obesity. *Brain Research*, *992*(1), 9–19.

Huang, X. F., Huang, X., Han, M., Chen, F., Storlien, L., & Lawrence, A. J. (2004). 5-HT2A/2C receptor and 5-HT transporter densities in mice prone or resistant to chronic high-fat diet-induced obesity: A quantitative autoradiography study. *Brain Research*, *1018*(2), 227–235.

Huang, X., Hancock, D., Gosby, A., McMahon, A., Solon, S., Le Couteur, D., … Simpson, S. (2013). Effects of Dietary Protein to Carbohydrate Balance on Energy Intake, Fat Storage, and Heat Production in Mice. *Obesity*, *21*(1), 85–

92.

Hughes, V. A., Frontera, W. R., Roubenoff, R., Evans, W. J., & Fiatarone Singh, M. A. (2002). Longitudinal changes in body composition in older men and women: Role of body weight change and physical activity. *American Journal of Clinical Nutrition*, *76*(2), 473–481.

Hung, Y., Wijnhoven, H., Visser, M., & Verbeke, W. (2019). Appetite and Protein Intake Strata of Older Adults in the European Union: Socio-Demographic and Health Characteristics, Diet-Related and Physical Activity Behaviours. *Nutrients*, *11*(4), 777.

Hunt, S. C., Stone, S., Xin, Y., Scherer, C. A., Magness, C. L., Iadonato, S. P., … Adams, T. D. (2008). Association of the FTO gene with BMI. *Obesity*, *16*(4), 902–904.

Iatridi, V., Hayes, J. E., & Yeomans, M. R. (2019). Reconsidering the classification of sweet taste liker phenotypes: A methodological review. *Food Quality and Preference*, *72*(August 2018), 56–76.

Illner, K., Brinkmann, G., Heller, M., Bosy-Westphal, A., & Müller, M. J. (2000). Metabolically active components of fat free mass and resting energy expenditure in nonobese adults. *American Journal of Physiology-Endocrinology and Metabolism*, *278*(2), E308–E315.

Institute of Medicine. (2005). Protein and amino acids. In F. and N. Board (Ed.), *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients)* (pp. 589–768). National Academy Sciences, National Research Council.

IPAQ Research Committee. (2005). Guidelines for Data Processing and Analysis of the International Physical Activity Questionnaire (IPAQ) – Short and Long

Forms. *IPAQ*, (November), 1–15.

Jaacks, L. M., Vandevijvere, S., Pan, A., McGowan, C. J., Wallace, C., Imamura, F., … Ezzati, M. (2019). The obesity transition: stages of the global epidemic. *The Lancet Diabetes and Endocrinology*, *7*(3), 231–240.

Jacob, R., Drapeau, V., Tremblay, A., Provencher, V., Bouchard, C., & Pérusse, L. (2018). The role of eating behavior traits in mediating genetic susceptibility to obesity. *American Journal of Clinical Nutrition*, *108*(3), 445–452.

James, W. P. T. (2008). The fundamental drivers of the obesity epidemic. *Obesity Reviews*, *9 Suppl 1*, 6–13.

Jean, C., Fromentin, G., Tomé, D., & Larue-Achagiotis, C. (2002). Wistar rats allowed to self-select macronutrients from weaning to maturity choose a high-protein, high-lipid diet. *Physiology and Behavior*, *76*(1), 65–73.

Jebb, S. (2017). Dusting off Foresight's obesity report - Foresight.

Jeffery, R. W., Baxter, J., McGuire, M., & Linde, J. (2006). Are fast food restaurants an environmental risk factor for obesity? *International Journal of Behavioral Nutrition and Physical Activity*, *3*, 2.

Jeffery, R. W., Rydell, S., Dunn, C. L., Harnack, L. J., Levine, A. S., Pentel, P. R., … Walsh, E. M. (2007). Effects of portion size on chronic energy intake. *International Journal of Behavioral Nutrition and Physical Activity*, *4*(1), 27.

Ji Yoon, K., Prescott, J., & Kwang Ok, K. (2014). Patterns of sweet liking in sucrose solutions and beverages. *Food Quality and Preference*, *36*, 96–103.

Johnson, F., Pratt, M., & Wardle, J. (2012). Dietary restraint and self-regulation in eating behavior. *International Journal of Obesity (2005)*, *36*(5), 665–674.

Johnson, L., Mander, A. P., Jones, L. R., Emmett, P. M., & Jebb, S. A. (2008). Energy-dense, low-fiber, high-fat dietary pattern is associated with increased fatness in childhood. *The American Journal of Clinical Nutrition*, *87*(4), 846–854.

Kagan, J. (1966). The generality and dynamics of conceptual tempo. *Journal of Abnormal Psychology*, *71*(1), 17–24.

Kant, A. K., & Graubard, B. I. (2005). Energy density of diets reported by American adults: Association with food group intake, nutrient intake, and body weight. *International Journal of Obesity*, *29*(8), 950–956.

Karelis, A. D., Lavoie, M. E., Fontaine, J., Messier, V., Strychar, I., Rabasa-Lhoret, R., & Doucet, É. (2010). Anthropometric, metabolic, dietary and psychosocial profiles of underreporters of energy intake: A doubly labeled water study among overweight/obese postmenopausal womena Montreal Ottawa New Emerging Team study. *European Journal of Clinical Nutrition*, *64*(1), 68–74.

Karra, E., O'Daly, O. G., Choudhury, A. I., Yousseif, A., Millership, S., Neary, M. T., … Batterham, R. L. (2013). A link between FTO, ghrelin, and impaired brain food-cue responsivity. *Journal of Clinical Investigation*, *123*(8), 3539–3551.

Kearney, M., Kearney, J., Dunne, A., & Gibney, M. J. (1999). Sociodemographic determinants of perceived in¯uences on food choice in a nationally representative sample of Irish adults. *Public Health Nutrition*, *3*(2), 219–226.

Keast, R. S., & Costanzo, A. (2015). Is fat the sixth taste primary? Evidence and implications. *Flavour*, *4*(1), 1–7.

Keenan, G. S., Brunstrom, J. M., & Ferriday, D. (2015a). Effects of meal variety on expected satiation: Evidence for a 'perceived volume' heuristic. *Appetite*, *89*, 10–15.

Keenan, G. S., Brunstrom, J. M., & Ferriday, D. (2015b). Effects of meal variety on expected satiation: Evidence for a “perceived volume” heuristic. *Appetite*, *89*, 10–15.

Keller, C., & Siegrist, M. (2015). Does personality influence eating styles and food choices? Direct and indirect effects. *Appetite*, *84*, 128–138.

Kelly, M. T., Rennie, K. L., Wallace, J. M. W., Robson, P. J., Welch, R. W., Hannon-Fletcher, M. P., & Livingstone, M. B. E. (2009). Associations between the portion sizes of food groups consumed and measures of adiposity in the British national diet and nutrition survey. *British Journal of Nutrition*, *101*(9), 1413–1420.

Kelly, M. T., Wallace, J. M. W., Robson, P. J., Rennie, K. L., Welch, R. W., Hannon-Fletcher, M. P., … Livingstone, M. B. E. (2009). Increased portion size leads to a sustained increase in energy intake over 4d in normal-weight and overweight men and women. *British Journal of Nutrition*, *102*(3), 470–477.

Kemps, E., Herman, C. P., Hollitt, S., Polivy, J., Prichard, I., & Tiggemann, M. (2016). Contextual cue exposure effects on food intake in restrained eaters. *Physiology and Behavior*, *167*, 71–75.

Kemps, E., & Tiggemann, M. (2015). Approach bias for food cues in obese individuals. *Psychology and Health*, *30*(3), 370–380.

Kenny, P. J. (2011). Reward Mechanisms in Obesity: New Insights and Future Directions. *Neuron*, *69*(4), 664–679.

Kent, M. (2009). Coke Didn’t Make America Fat. https://www.wsj.com/articles/SB10001424052748703298004574455464120581696. Accessed 3rd August 2019

Kent, S., Fusco, F., Gray, A., Jebb, S. A., Cairns, B. J., & Mihaylova, B. (2017). Body mass index and healthcare costs: a systematic literature review of individual participant data studies. *Obesity Reviews*, *18*(8), 869–879.

Keskitalo, K., Tuorila, H., Spector, T. D., Cherkas, L. F., Knaapila, A., Kaprio, J., … Perola, M. (2008). The Three-Factor Eating Questionnaire, body mass index, and responses to sweet and salty fatty foods: A twin study of genetic and environmental associations. *American Journal of Clinical Nutrition*.

Kesse-Guyot, E., Bertrais, S., Péneau, S., Estaquio, C., Dauchet, L., Vergnaud, A. C., … Bellisle, F. (2009). Dietary patterns and their sociodemographic and behavioural correlates in French middle-aged adults from the SU.VI.MAX cohort. *European Journal of Clinical Nutrition*, *63*(4), 521–528.

Khoury, J. E., Gonzalez, A., Levitan, R. D., Pruessner, J. C., Chopra, K., Basile, V. S., … Atkinson, L. (2015). Summary cortisol reactivity indicators: Interrelations and meaning. *Neurobiology of Stress*, *2*, 34–43.

Killgore, W. D. S. S., Young, A. D., Femia, L. A., Bogorodzki, P., Rogowska, J., & Yurgelun-Todd, D. A. (2003). Cortical and limbic activation during viewing of high- versus low-calorie foods. *NeuroImage*, *19*(4), 1381–1394.

Kilpeläinen, T. O., Qi, L., Brage, S., Sharp, S. J., Sonestedt, E., Demerath, E., … Loos, R. J. F. (2011). Physical activity attenuates the influence of FTO variants on obesity risk: A meta-analysis of 218,166 adults and 19,268 children. *PLoS Medicine*, *8*(11).

Kim, J. Y., Prescott, J., & Kim, K. O. (2014). Patterns of sweet liking in sucrose solutions and beverages. *Food Quality and Preference*, *36*, 96–103.

Kim, J. Y., Prescott, J., & Kim, K. O. (2017). Emotional responses to sweet foods according to sweet liker status. *Food Quality and Preference*, *59*, 1–7.

King, J. A., Deighton, K., Broom, D. R., Wasse, L. K., Douglas, J. A., Burns, S. F., … Stensel, D. J. (2017). Individual variation in hunger, energy intake, and ghrelin responses to acute exercise. *Medicine and Science in Sports and Exercise*, *49*(6), 1219–1228.

King, N. A., Caudwell, P., Hopkins, M., Stubbs, J. R., Naslund, E., & Blundell, J. E. (2009). Dual-process action of exercise on appetite control: Increase in orexigenic drive but improvement in meal-induced satiety. *American Journal of Clinical Nutrition*, *90*(4), 921–927.

King, N. A., Horner, K., Hills, A. P., Byrne, N. M., Wood, R. E., Bryant, E., … Blundell, J. E. (2012). Exercise, appetite and weight management: understanding the compensatory responses in eating behaviour and how they contribute to variability in exercise-induced weight loss. *British Journal of Sports Medicine*, *46*(5), 315–322.

Klausen, B., Toubro, S., & Astrup, A. (1997). Age and sex effects on energy expenditure. *American Journal of Clinical Nutrition*, *65*(4), 895–907.

Klesges, R. C., Klem, M. L., Epkins, C. C., & Klesges, L. M. (1991). A longitudinal evaluation of dietary restraint and its relationship to changes in body weight. *Addictive Behaviors*, *16*(5), 363–368.

Knai, C., Petticrew, M., Durand, M. A., Eastmure, E., James, L., Mehrotra, A., … Mays, N. (2015). Has a public-private partnership resulted in action on healthier diets in England? An analysis of the Public Health Responsibility Deal food pledges. *Food Policy*, *54*(March 2011), 1–10.

Knudsen, V. K. K., Matthiessen, J., Biltoft-Jensen, A., Sørensen, M. R., Groth, M. V., Trolle, E., … Fagt, S. (2014). Identifying dietary patterns and associated health-related lifestyle factors in the adult Danish population. *European Journal of Clinical Nutrition*, *68*(6), 736–740.

Kojima, M., Hosoda, H., Date, Y., Nakazato, M., Matsuo, H., & Kangawa, K.

(1999). Ghrelin is a growth-hormone-releasing acylated peptide from stomach. *Nature*, *402*(6762), 656–660.

Komatsu, S., & Aoyama, K. (2014). Food Craving and Its Relationship with Restriction and Liking in Japanese Females. *Foods, 3*(2), 208–216.

Konttinen, H., Männistö, S., Sarlio-Lähteenkorva, S., Silventoinen, K., & Haukkala, A. (2010). Emotional eating, depressive symptoms and self-reported food consumption. A population-based study. *Appetite*, *54*(3), 473–479.

Kraemer, W. J., & Ratamess, N. a. (2005). Hormonal responses and adaptations to resistance exercise and training. *Sports Medicine (Auckland, N.Z.)*, *35*(4), 339–361.

Kral, T. V. E., & Rolls, B. J. (2004). Energy density and portion size: Their independent and combined effects on energy intake. *Physiology and Behavior*, *82*(1), 131–138.

Kringelbach, M.L., & Berridge, K. C. (2010). The functional neuroanatomy of pleasure and happiness. *Discovery Medicine*, *9*(49), 579–587.

Kringelbach, Morten L. (2007). Cortical Svstems Involved in Appetite and Food Consumption. In *Appetite and Body Weight: Integrative Systems and the Development of Anti-Obesity Drugs* (pp. 5–26). Academic Press, Burlington.

Kroemer, N. B., Krebs, L., Kobiella, A., Grimm, O., Pilhatsch, M., Bidlingmaier, M., … Smolka, M. N. (2013). Fasting levels of ghrelin covary with the brain response to food pictures. *Addiction Biology*, *18*(5), 855–862.

Krølner, R., Rasmussen, M., Brug, J., Klepp, K. I., Wind, M., & Due, P. (2011). Determinants of fruit and vegetable consumption among children and adolescents: a review of the literature. Part II: qualitative studies. *International Journal of Behavioral Nutrition and Physical Activity*, *8*(1), 112.

Labbe, D., Rytz, A., Brunstrom, J. M., Forde, C. G., & Martin, N. (2017). Influence of BMI and dietary restraint on self-selected portions of prepared meals in US women. *Appetite*, *111*, 203–207.

Lähteenmäki, L., & Tuorila, H. (1995). Three-factor eating questionnaire and the use and liking of sweet and fat among dieters. *Physiology & Behavior*, *57*(1), 81–88.

Lahti-Koski, M., Pietinen, P., Heliovaara, M., & Vartiainen, E. (2002). Associations of body mass index and obesity with physical activity. *Food Choices, Alcohol Intake, and Smoking in the 1982-1997 {FINRISK} Studies*, *75*(1), 809–817.

Lam, B. C., Koh, G. C., Chen, C., Wong, M. T., & Fallows, S. J. (2015). Comparison of Body Mass Index (BMI), Body Adiposity Index (BAI), Waist Circumference (WC), Waist-To-Hip Ratio (WHR) and Waist- To-Height Ratio (WHtR) as Predictors of Cardiovascular Disease Risk Factors in an Adult Population in Singapore. *CME Journal of Gynecologic Oncology*, *9*(2), 133–152.

Lampuré, A., Castetbon, K., Deglaire, A., Schlich, P., Péneau, S., Hercberg, S., & Méjean, C. (2016). Associations between liking for fat, sweet or salt and obesity risk in French adults: a prospective cohort study. *The International Journal of Behavioral Nutrition and Physical Activity*, *13*(1), 74.

Lampuré, A., Deglaire, A., Schlich, P., Castetbon, K., Péneau, S., Hercberg, S., & Méjean, C. (2014). Liking for fat is associated with sociodemographic, psychological, lifestyle and health characteristics. *The British Journal of Nutrition*, *112*(8), 1353–1363.

Lampuré, A., Schlich, P., Deglaire, A., Castetbon, K., Péneau, S., Hercberg, S., & Méjean, C. (2015). Sociodemographic, Psychological, and Lifestyle

Characteristics Are Associated with a Liking for Salty and Sweet Tastes in French Adults. *The Journal of Nutrition*, *145*(3), 587–594.

Landgren, S., Jerlhag, E., Hallman, J., Oreland, L., Lissner, L., Strandhagen, E., … Engel, J. A. (2010). Genetic variation of the ghrelin signaling system in females with severe alcohol dependence. *Alcoholism: Clinical and Experimental Research*, *34*(9), 1519–1524.

Larson, N. I., Story, M. T., & Nelson, M. C. (2009). Neighborhood Environments. Disparities in Access to Healthy Foods in the U.S. *American Journal of Preventive Medicine*, *36*(1), 74-81.e10.

Latner, J., & Schwartz, M. (1999). The effect of a high-carbohydrate, high protein or balance lunch upon later food intake and hunger rating. *Appetite*, *33*, 119–128.

Latner, J D, & Schwartz, M. (1999). The effects of a high-carbohydrate, high-protein or balanced lunch upon later food intake and hunger ratings. *Appetite*, *33*(1), 119–128.

Latner, Janet D., Mond, J. M., Kelly, M. C., Haynes, S. N., & Hay, P. J. (2014). The loss of control over eating scale: Development and psychometric evaluation. *International Journal of Eating Disorders*, *47*(6), 647–659.

Lattimore, P., & Caswell, N. (2004). Differential effects of active and passive stress on food intake in restrained and unrestrained eaters. *Appetite*, *42*(2), 167–173.

Lau, C., Glümer, C., Toft, U., Tetens, I., Carstensen, B., Jørgensen, T., & Borch-Johnsen, K. (2008). Identification and reproducibility of dietary patterns in a Danish cohort: The Inter99 study. *British Journal of Nutrition*, *99*(5), 1089–1098.

Lawrence, M. A., & Baker, P. I. (2019). Ultra-processed food and adverse health outcomes. *The British Medical Journal*, *365*, l2289.

Le Roux, C. W., Batterham, R. L., Aylwin, S. J. B., Patterson, M., Borg, C. M., Wynne, K. J., … Bloom, S. R. (2006). Attenuated peptide YY release in obese subjects is associated with reduced satiety. *Endocrinology, 147*(1), 3–8.

Ledikwe, J. H., Blanck, H. M., Khan, L. K., Serdula, M. K., Seymour, J. D., Tohill, B. C., & Rolls, B. J. (2006). Dietary Energy Density Is Associated with Body Weight Status and Vegetable Intake in U.S. Children. *American Journal of Clinical Nutrition*, *83*, 1362–1368.

Ledikwe, J. H., Ello-martin, J. A., & Rolls, B. J. (2005). Symposium: Modifying the Food Environment: Energy Density, Food Costs, and Portion Size Public Health Strategies for Dietary Change: Schools and Workplaces. *Journal of Nutrition*, *135*, 905–909.

Lee, K. P., Simpson, S. J., Clissold, F. J., Brooks, R., Ballard, J. W. O., Taylor, P. W., … Raubenheimer, D. (2008). Lifespan and reproduction in Drosophila: New insights from nutritional geometry. *Proceedings of the National Academy of Sciences of the United States of America*, *105*(7), 2498–2503.

Lehnert, T., Sonntag, D., Konnopka, A., Riedel-Heller, S., & König, H.-H. H. (2013). Economic costs of overweight and obesity. *Best Practice and Research: Clinical Endocrinology and Metabolism*, *27*(2), 105–115.

Leidy, H. J., Clifton, P. M., Astrup, A., Wycherley, T. P., Westerterp-Plantenga, M. S., Luscombe-Marsh, N. D., … Mattes, R. D. (2015). The role of protein in weight loss and maintenance. *American Journal of Clinical Nutrition*.

Leitch, M. A., Morgan, M. J., & Yeomans, M. R. (2013). Different subtypes of impulsivity differentiate uncontrolled eating and dietary restraint. *Appetite*, *69*, 54–63.

Lemmens, S. G., Born, J. M., Rutters, F., Schoffelen, P. F., Wouters, L., &

Westerterp-Plantenga, M. S. (2010). Dietary restraint and control over wanting following consumption of forbidden food. *Obesity*, *18*(10), 1926–1931.

Lesdéma, A., Marsset-Baglieri, A., Talbot, L., Arlotti, A., Delarue, J., Fromentin, G., … Vinoy, S. (2016). When satiety evaluation is inspired by sensory analysis: A new approach. *Food Quality and Preference*, *49*, 106–118.

Lewis, H. B., Forwood, S. E., Ahern, A. L., Verlaers, K., Robinson, E., Higgs, S., & Jebb, S. A. (2015). Personal and social norms for food portion sizes in lean and obese adults. *International Journal of Obesity*, *39*(8), 1319–1324.

Li, Q., Blume, S. W., Huang, J. C., Hammer, M., & Ganz, M. L. (2015). Prevalence and healthcare costs of obesity-related comorbidities: evidence from an electronic medical records system in the United States. *Journal Of Medical Economics*, *6998*(October), 1–37.

Li, X., Tao, Q., Fang, Y., Cheng, C., Hao, Y., Qi, J., … Zhang, X. (2015). Reward sensitivity predicts ice cream-related attentional bias assessed by inattentional blindness. *Appetite*, *89*, 258–264.

Liebman, M., Pelican, S., Moore, S. A., Holmes, B., Wardlaw, M. K., Melcher, L. M., … Haynes, G. W. (2003). Dietary intake, eating behavior, and physical activity-related determinants of high body mass index in rural communities in Wyoming, Montana, and Idaho. *International Journal of Obesity*, *27*(6), 684–692.

Lindroos, A. K., Lissner, L., Mathiassen, M. E., Karlsson, J., Sullivan, M., Bengtsson, C., & Sjöström, L. (1997). Dietary intake in relation to restrained eating, disinhibition, and hunger in obese and nonobese swedish women. *Obesity Research*, *5*(3), 175–182.

Lissner, L., Habicht, J. P., Strupp, B. J., Levitsky, D. A., Haas, J. D., & Roe, D. A.

(1989). Body composition and energy intake: Do overweight women overeat and underreport? *American Journal of Clinical Nutrition*, *49*(2), 320–325.

Liu, G., Zhu, H., Lagou, V., Gutin, B., Stallmann-Jorgensen, I. S., Treiber, F. A., … Snieder, H. (2010a). FTO variant rs9939609 is associated with body mass index and waist circumference, but not with energy intake or physical activity in European- and African-American youth. *BMC Medical Genetics*, *11*(1).

Liu, G., Zhu, H., Lagou, V., Gutin, B., Stallmann-Jorgensen, I. S., Treiber, F. A., … Snieder, H. (2010b). FTO variant rs9939609 is associated with body mass index and waist circumference, but not with energy intake or physical activity in European- and African-American youth. *BMC Medical Genetics*, *11*(1).

Livingstone, K. M., Celis-Morales, C., Navas-Carretero, S., San-Cristoba, R., MacReady, A. L., Fallaize, R., … Mathers, J. C. (2016). Effect of an Internet-based, personalized nutrition randomized trial on dietary changes associated with the Mediterranean diet: The Food4Me Study. *American Journal of Clinical Nutrition*, *104*(2), 288–297.

Livingstone, M. B. E., & Black, A. E. (2003). Biomarkers of Nutritional Exposure and Nutritional Status. *Journal of Nutrition*, *133*(3), 895S-920S.

Lluch, A., Herbeth, B., Méjean, L., & Siest, G. (2000). Dietary intakes, eating style and overweight in the Stanislas Family Study. *International Journal of Obesity*, *24*(11), 1493–1499.

Locke, A. E., Kahali, B., Berndt, S. I., Justice, A. E., Pers, T. H., Day, F. R., Powell, C., Vedantam, S., Buchkovich, M. L., Yang, J., Croteau-Chonka, D. C., Esko, T., Fall, T., Ferreira, T., Gustafsson, S., Kutalik, Z., Luan, J., Mägi, R., Randall, J. (2015). Genetic studies of body mass index yield new insights for obesity biology. *Nature*, *518*(7538), 197–206.

Logan, G. D., Schachar, R. J., & Tannock, R. (1997). Impulsivity and inhibitory control. *Psychological Science, 8*(1), 60–64.

Long, S. J., Hart, K., & Morgan, L. M. (2002). The ability of habitual exercise to influence appetite and food intake in response to high- and low-energy preloads in man. *British Journal of Nutrition*, *87*(05), 517–523.

Long, S. J., Jeffcoat, A. R., & Millward, D. J. (2000). Effect of habitual dietary-protein intake on appetite and satiety. *Appetite*, *35*(1), 79–88.

Low, Y. Q., Lacy, K., & Keast, R. S. J. (2014). The role of sweet taste in satiation and satiety. *Nutrients*, *6*(9), 3431–3450.

Lowe, M.R. (2002). Dietary Restraint and Overeating. In C. G. Fairburn & K. D. Brownell (Eds.), *Eating disorders and Obesity: A Comprehensive Handbook* (2nd ed., pp. 88–92). New York: Guildford Press.

Lowe, Michael R., Butryn, M. L., Didie, E. R., Annunziato, R. A., Thomas, J. G., Crerand, C. E., … Halford, J. (2009). The Power of Food Scale. A new measure of the psychological influence of the food environment. *Appetite*, *53*(1), 114–118.

Loxton, N. J. (2018). The Role of Reward Sensitivity and Impulsivity in Overeating and Food Addiction. *Current Addiction Reports*, 1–11.

Loxton, N. J., & Tipman, R. J. (2017). Reward sensitivity and food addiction in women. *Appetite*, *115*, 28–35.

Ludwig, D. S., Astrup, A., Bazzano, L. A., Ebbeling, C. B., Heymsfield, S. B., King, J. C., & Willett, W. C. (2019). Ultra-Processed Food and Obesity: The Pitfalls of Extrapolation from Short Studies. *Cell Metabolism*, *30*(1), 3–4.

Luscombe-Marsh, N. D., Smeets, A. J. P. G., & Westerterp-Plantenga, M. S. (2008). Taste sensitivity for monosodium glutamate and an increased liking of dietary protein. *British Journal of Nutrition*, *99*(4), 904–908.

Luscombe-Marsh, N. D., Smeets, A. J. P. G., & Westerterp-Plantenga, M. S. (2009). The addition of monosodium glutamate and inosine monophosphate-5 to high-protein meals: Effects on satiety, and energy and macronutrient intakes. *British Journal of Nutrition*, *102*(6), 929–937.

Macht, M. (2008). How emotions affect eating: A five-way model. *Appetite*, *50*(1), 1–11.

Mackelvie, K. J., Meneilly, G. S., Elahi, D., Wong, A. C. K., Barr, S. I., & Chanoine, J. P. (2007). Regulation of appetite in lean and obese adolescents after exercise: Role of acylated and desacyl ghrelin. *Journal of Clinical Endocrinology and Metabolism*, *92*(2), 648–654.

Mackey, L., White, M. J., Tyack, Z., Finlayson, G., Dalton, M., & King, N. A. (2019). A dual-process psychobiological model of temperament predicts liking and wanting for food and trait disinhibition. *Appetite*, *134*(December 2018), 9–16.

MacLean, P. S., Blundell, J. E., Mennella, J. A., & Batterham, R. L. (2017). Biological control of appetite: A daunting complexity. *Obesity*, *25*(December 2016), S8–S16.

Maffeis, C., Surano, M. G., Cordioli, S., Gasperotti, S., Corradi, M., & Pinelli, L. (2010). A High-fat vs a Moderate-fat Meal in Obese Boys: Nutrient Balance, Appetite, and Gastrointestinal Hormone Changes. *Obesity*, *18*(3), 449–455.

Maguire, E. R., Burgoine, T., & Monsivais, P. (2015). Area deprivation and the food environment over time: A repeated cross-sectional study on takeaway outlet density and supermarket presence in Norfolk, UK, 1990-2008. *Health and Place*, *33*, 142–147.

Malik, S., McGlone, F., Bedrossian, D., & Dagher, A. (2008). Ghrelin Modulates Brain Activity in Areas that Control Appetitive Behavior. *Cell Metabolism*, *7*(5), 400–409.

Markussen, M. S., Veierod, M. B., Kristiansen, A. L., Ursin, G., & Andersen, L. F. (2016). Dietary patterns of women aged 50-69 years and associations with nutrient intake, sociodemographic factors and key risk factors for non-communicable diseases. *Public Health Nutrition*, *19*(11), 2024–2032.

Marteau, T. M., Hollands, G. J., Shemilt, I., & Jebb, S. A. (2015). Downsizing: Policy options to reduce portion sizes to help tackle obesity. *BMJ (Online)*, *351*(December), 1–5.

Martens, E. A., Lemmens, S. G., & Westerterp-Plantenga, M. S. (2013). Protein leverage affects energy intake of high-protein diets in humans. *American Journal of Clinical Nutrition*, *97*(1), 86–93.

Martens, E. A. P., Tan, S.-Y., Mattes, R. D., & Westerterp-Plantenga, M. S. (2014). No protein intake compensation for insufficient indispensable amino acid intake with a low-protein diet for 12 days. *Nutrition and Metabolism*, *11*(1).

Martens, E. A. P., & Westerterp-Plantenga, M. S. (2014). Protein diets, weight loss and weight maintenance. *Current Opinion in Clinical Nutrition and Metabolic Care*, *17*(75–79).

Martens, E. A., Tan, S.-Y., Dunlop, M. V., Mattes, R. D., & Westerterp-Plantenga, M. S. (2014). Protein leverage effects of beef protein on energy intake in humans. *American Journal of Clinical Nutrition*, *99*(6), 1397–1406.

Martinez-Cordero, C., Kuzawa, C. W., Sloboda, D. M., Stewart, J., Simpson, S. J., & Raubenheimer, D. (2012). Testing the Protein Leverage Hypothesis in a free-living human population. *Appetite*, *59*(2), 312–315.

Martínez Steele, E., Popkin, B. M., Swinburn, B., & Monteiro, C. A. (2017). The share of ultra-processed foods and the overall nutritional quality of diets in the US: Evidence from a nationally representative cross-sectional study. *Population Health Metrics*, *15*(1), 1–11.

Martínez Steele, E., Raubenheimer, D., Simpson, S. J., Baraldi, L. G., & Monteiro, C. A. (2018). Ultra-processed foods, protein leverage and energy intake in the USA. *Public Health Nutrition*, *21*(1), 114–124.

Martins, C., Kulseng, B., King, N. A., Holst, J. J., & Blundell, J. E. (2010). The effects of exercise-induced weight loss on appetite-related peptides and motivation to eat. *The Journal of Clinical Endocrinology and Metabolism*, *95*(4), 1609–1616.

Martins, C., Morgan, L. M., Bloom, S. R., & Robertson, M. D. (2007). Effects of exercise on gut peptides, energy intake and appetite. *The Journal of Endocrinology*, *193*(2), 251–258.

Martins, C., Morgan, L., & Truby, H. (2008). A review of the effects of exercise on appetite regulation: an obesity perspective. *International Journal of Obesity (2005)*, *32*(9), 1337–1347.

Masic, U., & Yeomans, M. R. (2014a). Monosodium glutamate delivered in a protein-rich soup improves subsequent energy compensation. *Journal of Nutritional Science*, *3*, 1–9.

Masic, U., & Yeomans, M. R. (2014b). Umami flavor enhances appetite but also increases satiety. *American Journal of Clinical Nutrition*, *100*(2), 532–538.

Masic, U., & Yeomans, M. R. (2017). Does acute or habitual protein deprivation influence liking for monosodium glutamate? *Physiology and Behavior*, *171*, 79–86.

Mason, A. E., Vainik, U., Acree, M., Tomiyama, A. J., Dagher, A., Epel, E. S., & Hecht, F. M. (2017). Improving assessment of the spectrum of reward-related eating: The RED-13. *Frontiers in Psychology*, *8*(MAY), 1–14.

Mason, K. E., Pearce, N., & Cummins, S. (2018). Associations between fast food and physical activity environments and adiposity in mid-life: cross-sectional, observational evidence from UK Biobank. *The Lancet Public Health*, *3*(1), e16–e23.

Masuda, Y., Tanaka, T., Inomata, N., Ohnuma, N., Tanaka, S., Itoh, Z., … Kangawa, K. (2000). Ghrelin stimulates gastric acid secretion and motility in rats. *Biochemical and Biophysical Research Communications*, *276*(3), 905–908.

Mattes, R. (2014). Energy intake and obesity: Ingestive frequency outweighs portion size. *Physiology and Behavior*, *134*(C), 110–118.

Mattes, R. D. (2009). Oral thresholds and suprathreshold intensity ratings for free fatty acids on 3 tongue sites in humans: Implications for transduction mechanisms. *Chemical Senses*, *34*, 415–423.

Mayer, J., Roy, P., & Mitra, K. P. (1956). Relation between caloric intake, body weight, and physical work: studies in an industrial male population in West Bengal. *The American Journal of Clinical Nutrition*, *4*(2), 169–175.

Mayntz, D., Nielsen, V. H., Sørensen, A., Toft, S., Raubenheimer, D., Hejlesen, C., & Simpson, S. J. (2009). Balancing of protein and lipid intake by a mammalian carnivore, the mink, Mustela vison. *Animal Behaviour*, *77*(2), 349–355.

McCaffery, J. M., Papandonatos, G. D., Peter, I., Huggins, G. S., Raynor, H. A., Delahanty, L. M., … Wing, R. R. (2012). Obesity susceptibility loci and dietary intake in the Look AHEAD Trial. *American Journal of Clinical Nutrition*, *95*(6), 1477–1486.

McCrory, M. a, Saltzman, E., Rolls, B. J., & Roberts, S. B. (2006). A twin study of the effects of energy density and palatability on energy intake of individual foods. *Physiology & Behavior, 87*(3), 451–459.

McGlory, C., van Vliet, S., Stokes, T., Mittendorfer, B., & Phillips, S. M. (2019). The impact of exercise and nutrition on the regulation of skeletal muscle mass. *Journal of Physiology*, *597*(5), 1251–1258.

McLaren, L. (2007). Socioeconomic status and obesity. *Epidemiologic Reviews*, *29*(1), 29–48.

McNeil, J., Lamothe, G., Cameron, J. D., Riou, M. E., Cadieux, S., Lafrenière, J., … Doucet, É. (2017). Investigating predictors of eating: Is resting metabolic rate really the strongest proxy of energy intake? *American Journal of Clinical Nutrition*, *106*(5), 1206–1212.

McTaggart, J. S., Lee, S., Iberl, M., Church, C., Cox, R. D., & Ashcroft, F. M. (2011). FTO is expressed in neurones throughout the brain and its expression is unaltered by fasting. *PLoS ONE*, *6*(11), e27968.

Medic, N., Ziauddeen, H., Forwood, S. E., Davies, K. M., Ahern, A. L., Jebb, S. A., … Fletcher, P. C. (2016). The presence of real food usurps hypothetical health value judgment in overweight people. *Society for Neuroscience*, *3*(2), 412–427.

Méjean, C., MacOuillard, P., Castetbon, K., Kesse-Guyot, E., & Hercberg, S. (2011). Socio-economic, demographic, lifestyle and health characteristics associated with consumption of fatty-sweetened and fatty-salted foods in middle-aged French adults. *British Journal of Nutrition*, *105*(5), 776–786.

Mela, D. J. (2001). Determinants of food choice: relationships with obesity and weight control. *Obesity Research*, *9*(November), 249S-255S.

Mela, David J. (2006). Eating for pleasure or just wanting to eat? Reconsidering sensory hedonic responses as a driver of obesity. *Appetite*, Vol. 47, pp. 10–17.

Melhorn, S. J., Askren, M. K., Chung, W. K., Kratz, M., Bosch, T. A., Tyagi, V., … Schur, E. A. (2018). FTO genotype impacts food intake and corticolimbic activation. *American Journal of Clinical Nutrition*, *107*(2), 145–154.

Mendoza, J. A., Drewnowski, A., & Christakis, D. A. (2007). Dietary energy density is associated with obesity and the metabolic syndrome in US adults. *Diabetes Care*, *30*(4), 974–979.

Mennella, I., Ferracane, R., Zucco, F., Fogliano, V., & Vitaglione, P. (2015). Food Liking Enhances the Plasma Response of 2-Arachidonoylglycerol and of Pancreatic Polypeptide upon Modified Sham Feeding in Humans. *Journal of Nutrition*, *145*(9), 2169–2175.

Mennella, J. A., & Bobowski, N. K. (2015). The sweetness and bitterness of childhood: Insights from basic research on taste preferences. *Physiology & Behavior*, *152*(Part B), 502–507.

Merema, M., O’Connell, E., Joyce, S., Woods, J., & Sullivan, D. (2019). Trends in body mass index and obesity prevalence in Western Australian adults, 2002 to 2015. *Health Promotion Journal of Australia*, *30*(1), 60–65.

Mesas, A. E., Muñoz-Pareja, M., López-García, E., & Rodríguez-Artalejo, F. (2012). Selected eating behaviours and excess body weight: A systematic review. *Obesity Reviews*, *13*(2), 106–135.

Methven, L., Xiao, C., Cai, M., & Prescott, J. (2016). Rejection thresholds (RjT) of sweet likers and dislikers. *Food Quality and Preference*, *52*, 74–80.

Meule, A., Mayerhofer, M., Grundel, T., Berker, J., Beck Teran, C., & Platte, P. (2015). Half-Year Retest-Reliability of the Barratt Impulsiveness Scale-Short Form (BIS-15). *SAGE Open*, *5*(1), 10–12.

Meule, Adrian. (2013). Impulsivity and overeating: A closer look at the subscales of the Barratt Impulsiveness Scale. *Frontiers in Psychology*, *4*(APR), 1–4.

Meule, Adrian, & Kübler, A. (2014). Double trouble. Trait food craving and impulsivity interactively predict food-cue affected behavioral inhibition. *Appetite*, *79*, 174–182.

Meule, Adrian, Lutz, A. P. C., Vögele, C., & Kübler, A. (2014). Impulsive reactions to food-cues predict subsequent food craving. *Eating Behaviors*, *15*(1), 99–105.

Meyer-Gerspach, A. C., Wölnerhanssen, B., Beglinger, B., Nessenius, F., Napitupulu, M., Schulte, F. H., … Beglinger, C. (2014). Gastric and intestinal satiation in obese and normal weight healthy people. *Physiology and Behavior*, *129*, 265–271.

Miech, R., Kumanyika, S., Stettler, N., Link, B., Phelan JC, C., & Hang, V. (2006). Trends in the Association of Poverty. *Journal of American Medical Association*, *295*(20), 2385–2393.

Mifflin, M. D., St Jeor, S. T., Hill, L. A., Scott, B. J., Daugherty, S. A., & Koh, Y. O. (1990). A new predictive equation in healthy individuals for resting energy. *The American Journal of Clinical Nutrition*, *51*, 241–247.

Mikkilä, V., Räsänen, L., Raitakari, O. T., Pietinen, P., & Viikari, J. (2005). Consistent dietary patterns identified from childhood to adulthood: The Cardiovascular Risk in Young Finns Study. *British Journal of Nutrition*, *93*(06), 923.

Millward, D. J. (1995). A protein-stat mechanism for regulation of growth and maintenance of the lean body mass. *Nutrition Research Reviews*, *8*(1995), 93–120.

Millward, D. J. (1997). Human Amino Acid Requirements. *The Journal of Nutrition*, *127*(June), 1842–1846.

Millward, D. J. (1998). Nutrition for Health Metabolic Demands for Amino Acids and the Human Dietary Requirement: Millward and Rivers (1988) Revisited 1,2. *Journal of Nutrition*, *128*(1988), 2563–2576.

Miras, A. D., Jackson, R. N., Jackson, S. N., Goldstone, A. P., Olbers, T., Hackenberg, T., … le Roux, C. W. (2007). Gastric bypass surgery for obesity decreases the reward value of a sweet-fat stimulus as assessed in a progressive ration task. *American Journal of Clinical Nutrition*, *96*, 467–473.

Mishra, G., Ball, K., Arbuckle, J., & Crawford, D. (2002). Dietary patterns and their association with socioeconomic status: Factor analysis of the 1995 Australian National Nutrition Survey. *European Journal of Clinical Nutrition*, *56*, 687–693.

Mittelman, S. D., Klier, K., Braun, S., Azen, C., Geffner, M. E., & Buchanan, T. A. (2010). Obese adolescents show impaired meal responses of the appetite-regulating hormones ghrelin and PYY. *Obesity*, *18*(5), 918–925.

Mobbs, O., Crépin, C., Thiéry, C., Golay, A., & Van der Linden, M. (2010). Obesity and the four facets of impulsivity. *Patient Education and Counseling*, *79*(3), 372–377.

Mobini, S., Grant, A., Kass, A. E., & Yeomans, M. R. (2007). Relationships between functional and dysfunctional impulsivity, delay discounting and cognitive distortions. *Personality and Individual Differences*, *43*(6), 1517–1528.

Moeller, F. G., Barratt, E. S., Ph, D., Dougherty, D. M., Schmitz, J. M., & Swann, A. C. (2001). Psychiatric Aspects of Impulsivity. *Psychiatry Interpersonal and Biological Processes*, *158*(November), 1783–1793.

Moesgaard, S. G., Ahrén, B., Carr, R. D., Gram, D. X., Brand, C. L., & Sundler, F.

(2004). Effects of high-fat feeding and fasting on ghrelin expression in the mouse stomach. *Regulatory Peptides*, *120*(1–3), 261–267.

Møller, P. (2014). Orosensory perception. In D. Bar-Shalom & K. Rose (Eds.), *Pediatric formulations: a roadmap* (Vol. 11, pp. 105–121). Springer. AAPS Advances in the Pharmaceutical Sciences Series, Vol.. 11 https://doi.org/10.1007/978-1-4899-8011-3_8.

Molteni, R., Barnard, R. J., Ying, Z., Roberts, C. K., & Gómez-Pinilla, F. (2002). A high-fat, refined sugar diet reduces hippocampal brain-derived neurotrophic factor, neuronal plasticity, and learning. *Neuroscience*, *112*(4), 803–814.

Monsivais, P., & Drewnowski, A. (2009). Lower-Energy-Density Diets Are Associated with Higher Monetary Costs per Kilocalorie and Are Consumed by Women of Higher Socioeconomic Status. *Journal of the American Dietetic Association*, *109*(5), 814–822.

Monteiro, Carlos A, & Cannon, G. J. (2019). The role of the transnational ultra-processed food industry in the pandemic of obesity and its associated diseases: problems and solutions. *World Nutrition*, *10*(1), 89–99.

Monteiro, Carlos A, Moura, E. C., Conde, W. L., & Popkin, B. M. (2004). Public Health Reviews Socioeconomic status and obesity in adult populations of developing countries : a review. *Public Health Reviews*, *82*(12), 940–946.

Monteiro, Carlos Augusto, Cannon, G., Moubarac, J.-C., Levy, R. B., Louzada, M. L. C., & Jaime, P. C. (2017). The UN Decade of Nutrition, the NOVA food classification and the trouble with ultra-processing. *Public Health Nutrition*, *21*(01), 5–17.

Monteleone, A. M., Monteleone, P., Dalle Grave, R., Nigro, M., El Ghoch, M., Calugi, S., … Maj, M. (2015). Ghrelin response to hedonic eating in underweight and short-term weight restored patients with anorexia nervosa. *Psychiatry Research*, *235*, 55–60.

Monteleone, P., Bencivenga, R., Longobardi, N., Serritella, C., & Maj, M. (2003). Differential Responses of Circulating Ghrelin to High-Fat or High-Carbohydrate Meal in Healthy Women. *Journal of Clinical Endocrinology and Metabolism*, *88*(11), 5510–5514.

Monteleone, P., Piscitelli, F., Scognamiglio, P., Monteleone, A. M., Canestrelli, B., Di Marzo, V., & Maj, M. (2012). Hedonic eating is associated with increased peripheral levels of ghrelin and the endocannabinoid 2-arachidonoyl-glycerol in healthy humans: A pilot study. *Journal of Clinical Endocrinology and Metabolism*, *97*(6), 917–924.

Monteleone, P., Scognamiglio, P., Monteleone, A. M., Perillo, D., Canestrelli, B., & Maj, M. (2013). Gastroenteric hormone responses to hedonic eating in healthy humans. *Psychoneuroendocrinology*, *38*(8), 1435–1441.

Moodie, R., Stuckler, D., Monteiro, C., Sheron, N., Neal, B., Thamarangsi, T., … Casswell, S. (2013). Profits and pandemics: Prevention of harmful effects of tobacco, alcohol, and ultra-processed food and drink industries. *The Lancet*, *381*(9867), 670–679.

Morley, J. E. (2001). Decreased Food Intake With Aging. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, *56*(Supplement 2), 81–88.

Morris, M. J., Beilharz, J. E., Maniam, J., Reichelt, A. C., & Westbrook, R. F. (2015). Why is obesity such a problem in the 21st century? The intersection of palatable food, cues and reward pathways, stress, and cognition. *Neuroscience and Biobehavioral Reviews*, *58*, 36–45.

Morrison, C. D., & Laeger, T. (2015). Protein-dependent regulation of feeding and metabolism. *Trends in Endocrinology and Metabolism*, *26*(5), 256–262.

Morrison, C. D., Reed, S. D., & Henagan, T. M. (2012). Homeostatic regulation of protein intake: in search of a mechanism. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology*, *302*(8), R917-28.

Mozaffarian, D., Hao, T., Rimm, E. B., Willett, W. C., & Hu, F. B. (2011). Changes in Diet and Lifestyle and Long-Term Weight Gain in Women and Men. *New England Journal of Medicine*, *364*(25), 2392–2404.

Murphy, Maureen, Badland, H., Jordan, H., Koohsari, M. J., & Giles-Corti, B. (2018). Local food environments, suburban development, and BMI: A mixed methods study. *International Journal of Environmental Research and Public Health*, *15*(7), 1–19.

Murphy, Michelle, Peters, K. Z., Denton, B. S., Lee, K. A., Chadchankar, H., & McCutcheon, J. E. (2018). Restriction of dietary protein leads to conditioned protein preference and elevated palatability of protein-containing food in rats. *Physiology and Behavior*, *184*(December 2017), 235–241.

Naleid, A. M., Grace, M. K., Cummings, D. E., & Levine, A. S. (2005). Ghrelin induces feeding in the mesolimbic reward pathway between the ventral tegmental area and the nucleus accumbens. *Peptides*, *26*(11), 2274–2279.

Nasser, J. A., Gluck, M. E., & Geliebter, A. (2004). Impulsivity and test meal intake in obese binge eating women. *Appetite*, *43*(3), 303–307.

NCD Risk Factor Collaboration (NCD-RisC). (2016). Trends in adult body-mass index in 200 countries from 1975 to 2014: A pooled analysis of 1698 population-based measurement studies with 19.2 million participants. *The Lancet*, *387*(10026), 1377–1396.

NCD Risk Factor Collaboration (NCD-RisC). (2017). Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. *The Lancet*, *390*(10113), 2627–2642.

Nederkoorn, C., Smulders, F. T. Y., Havermans, R. C., Roefs, A., & Jansen, A. (2006). Impulsivity in obese women. *Appetite*, *47*(2), 253–256.

Newby, P. K., Muller, D., Hallfrisch, J., Andres, R., & Tucker, K. L. (2004). Food patterns measured by factor analysis and anthropometric changes in adults. *The American Journal of Clinical Nutrition*, *80*(2), 504–513.

Newby, P. K., & Tucker, K. L. (2004). Empirically Derived Eating Patterns Using Factor or Cluster Analysis : A Review. *Nutrition Reviews*, *62*(5), 177–203.

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.

Ng, S. W., & Popkin, B. M. (2012). Time Use and Physical Activity: A Shift Away from Movement across the Globe. *Obesity Reviews*, *13*(8), 659–680.

NHS Digital. (2018). *Health Survey for England 2017: Adult and child Overweight and Obesity*.

Nicklett, E. J., & Kadell, A. R. (2013). Fruit and vegetable intake among older adults: A scoping review. *Maturitas*, *75*, 305–312.

Nielsen, S. J., & Popkin, B. M. (2003). Patterns and Trends in Food Portion Sizes, 1977-1998. *Journal of American Medical Association*, *289*(4), 450–453.

Nijs, I. M. T., Franken, I. H. A., & Muris, P. (2007). The modified Trait and State Food-Cravings Questionnaires: Development and validation of a general index of food craving. *Appetite*, *49*(1), 38–46.

Ningombam, S. S., Chhungi, V., Newmei, M. K., Rajkumari, S., Devi, N. K., Mondal, P. R., & Saraswathy, K. N. (2018). Differential distribution and association of FTO rs9939609 gene polymorphism with obesity: A cross-sectional study among two tribal populations of India with East-Asian ancestry. *Gene*, *647*(December 2017), 198–204.

Nolan-Pouparta, S., Veldhuizen, M. G., Geha, P., & Small, D. M. (2013). Midbrain Response to Milkshake Correlates with Ad Libitum Milkshake Intake in the Absence of Hunger. *Appetite*, *60*(1), 168–174.

Nolan, L. J. (2012). Association between impulsiveness and pleasantness ratings for food and drugs. *Appetite*, *59*, e43.

O'Connor, D., Jones, F., Conner, M., McMillan, B., & Ferguson, E. (2008). Effects of Daily Hassles and Eating Style on Eating Behaviour. *Health Psychology*, *27*(1), 20–31.

Obregón Rivas, A. M., Santos, J. L., Valladares, M. A., Cameron, J., & Goldfield, G. (2018). Association of the FTO fat mass and obesity–associated gene rs9939609 polymorphism with rewarding value of food and eating behavior in Chilean children. *Nutrition*, *54,* 105–110.

Ochner, C. N., Stice, E., Hutchins, E., Afifi, L., Geliebter, A., Hirsch, J., & Teixeira, J. (2012). Relation between changes in neural responsivity and reductions in desire to eat high-calorie foods following gastric bypass surgery. *Neuroscience*, *209*, 128–135.

Office of National Statistics. (2018). *Population of England and Wales*. https://www.ethnicity-facts-figures.service.gov.uk/uk-population-by-ethnicity/national-and-regional-populations/population-of-england-and-wales/latest. Accessed 2nd October 2019

Ohkuma, T., Hirakawa, Y., Nakamura, U., Kiyohara, Y., Kitazono, T., & Ninomiya, T. (2015). Association between eating rate and obesity: A systematic review and meta-analysis. *International Journal of Obesity*, *39*(11), 1589–1596.

Oussaada, S. M., Galen, K. A. van, Cooiman, M. I., Kleinendorst, L., Hazebroek, E. J., Haelst, M. M. van, … Serlie, M. J. (2019). The pathogenesis of obesity. *Metabolism: Clinical and Experimental*, *92*(1), 26–36.

Ouwens, M. a., Van Strien, T., & Van Der Staak, C. P. F. (2003). Tendency toward overeating and restraint as predictors of food consumption. *Appetite*, *40*(3), 291–298.

Overduin, J., Figlewicz, D. P., Bennett-Jay, J., Kittleson, S., & Cummings, D. E. (2012). Ghrelin increases the motivation to eat, but does not alter food palatability. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology*, *303*(3), R259–R269.

Padulo, C., Carlucci, L., Manippa, V., Marzoli, D., Saggino, A., Tommasi, L., … Brancucci, A. (2017). Valence, familiarity and arousal of different foods in relation to age, sex and weight. *Food Quality and Preference*, *57*, 104–113.

Pampel, F. C., Denney, J. T., & Krueger, P. M. (2012). Obesity, SES, and economic development: A test of the reversal hypothesis. *Social Science and Medicine*, *74*(7), 1073–1081.

Panjwani, C., & Caraher, M. (2014). The Public Health Responsibility Deal: Brokering a deal for public health, but on whose terms? *Health Policy*, *114*(2–3), 163–173.

Park, S. L., Cheng, I., Pendergrass, S. A., Kucharska-Newton, A. M., Lim, U., Ambite, J. L., … Le Marchand, L. (2013). Association of the FTO obesity risk variant rs8050136 with percentage of energy intake from fat in multiple racial/ethnic populations. *American Journal of Epidemiology*, *178*(5), 780–790.

Pasquet, P., Frelut, M. L., Simmen, B., Hladik, C. M., & Monneuse, M. O. (2007). Taste perception in massively obese and in non-obese adolescents. *International Journal of Pediatric Obesity*, *2*(4), 242–248.

Patton, J. H., Stanford, M., & Barratt, E. S. (1995). Factor structure of the Barratt Impulsiveness Scale. J Clin Psychol 51: 768-774. *Journal of Clinical Psychology,* Vol. 51, pp. 768–774.

Peciña, S, & Berridge, K. C. (2000). Opioid eating site in accumbens shell mediates food intake and hedonic "liking": map based on microinjection Fos plumes. *Brain Research*, *863*, 71–86.

Peciña, Susana. (2008). Opioid reward "liking" and "wanting" in the nucleus accumbens. *Physiology and Behavior*, *94*(5), 675–680.

Pedersen, S. D. (2013). Metabolic complications of obesity. *Best Practice and Research: Clinical Endocrinology and Metabolism*, *27*(2), 179–193.

Peng, M., Hautus, M. J., Oey, I., & Silcock, P. (2016). Is there a generalized sweetness sensitivity for an individual? A psychophysical investigation of inter-individual differences in detectability and discriminability for sucrose and fructose. *Physiology and Behavior*, *165*, 239–248.

Peng, S., Zhu, Y., Xu, F., Ren, X., Li, X., & Lai, M. (2011). FTO gene polymorphisms and obesity risk: a meta-analysis. *BMC Medicine*, *9*(1), 71.

Penney, T., Burgoine, T., & Monsivais, P. (2018). Relative Density of Away from Home Food Establishments and Food Spend for 24,047 Households in England: A Cross-Sectional Study. *International Journal of Environmental Research and Public Health*, *15*(12), 2821.

Pepino, M. Y., Finkbeiner, S., Beauchamp, G. K., & Mennella, J. A. (2010). Obese women have lower monosodium glutamate taste sensitivity and prefer higher concentrations than do normal-weight women. *Obesity*, *18*(5), 959–965.

Perello, M., Sakata, I., Birnbaum, S., Chuang, J. C., Osborne-Lawrence, S., Rovinsky, S. A., … Zigman, J. M. (2010). Ghrelin Increases the Rewarding Value of High-Fat Diet in an Orexin-Dependent Manner. *Biological Psychiatry*, *67*(9), 880–886.

Perelló, M., & Zigman, J. M. (2012). The role of ghrelin in reward-based eating. *Biological Psychiatry*, *72*(5), 347–353.

Pérez, C., Ackroff, K., & Sclafani, A. (1996). Carbohydrate- and protein-conditioned flavor preferences: Effects of nutrient preloads. *Physiology and Behavior*, *59*(3), 467–474.

Perrin, A.-E., Dallongeville, J., Ducimetière, P., Ruidavets, J.-B., Schlienger, J.-L., Arveiler, D., & Simon, C. (2005). Interactions between traditional regional determinants and socio-economic status on dietary patterns in a sample of French men. *British Journal of Nutrition*, *93*(01), 109.

Pezeshki, A., Zapata, R., Singh, A., Yee, N., & Chelikani, P. (2016). Low protein diets produce divergent effects on energy balance. *Scientific Reports*, *6*, 1–13.

Phositlimpagul, A., Edwards, G. L., & Azain, M. J. (2002). *Hepatic vagotomy disrupts somatotropin-induced protein selection*. *75*, 193–200.

Piaggi, P., Thearle, M. S., Krakoff, J., & Votruba, S. B. (2015). Higher daily energy expenditure and respiratory quotient, rather than fat-free mass, independently determine greater ad libitum overeating. *Journal of Clinical Endocrinology and Metabolism*, *100*(8), 3011–3020.

Pickering, A., & Corr, P. (2008). J.A.Gray's reinforcement sensitivity theory (RST) of personality. In G. Boyle, G. Matthews, & D. Saklofske (Eds.), *The SAGE*

Handbook of Personality: Theory and Assessment Personality Measurement and Testing (Volume 2, pp. 239–255). London, New Delhi and Thousand Oaks: Sage.

Pieroni, L., & Salmasi, L. (2014). Fast-food consumption and body weight. Evidence from the UK. *Food Policy*, *46*, 94–105.

Pinto de Souza Fernandes, D., Duarte, M. S. L., Pessoa, M. C., Franceschini, S. D. C. C., & Ribeiro, A. Q. (2017). Evaluation of diet quality of the elderly and associated factors. *Archives of Gerontology and Geriatrics*, *72*(October 2016), 174–180.

Polivy, J., Coleman, J., & Herman, C. P. (2005). The effect of deprivation on food cravings and eating behavior in restrained and unrestrained eaters. *International Journal of Eating Disorders*, *38*(4), 301–309.

Polivy, J., & Herman, C. P. (1985). Dieting and Binging. A Causal Analysis. *American Psychologist*, *40*(2), 193–201.

Polk, S. E., Schulte, E. M., Furman, C. R., & Gearhardt, A. N. (2017). Wanting and liking: Separable components in problematic eating behavior? *Appetite*, *115*, 45–53.

Ponnalagu, S., Bi, X., & Henry, C. J. (2018). Is waist circumference more strongly associated with metabolic risk factors in Asians than waist-to-height ratio? *Nutrition*, *60*, 30–34.

Pool, E., Sennwald, V., Delplanque, S., Brosch, T., & Sander, D. (2016). Measuring wanting and liking from animals to humans: A systematic review. *Neuroscience and Biobehavioral Reviews*, *63*, 124–142.

Poortmans, J. R., Carpentier, A., Pereira-Lancha, L. O., & Lancha, A. (2012). Protein turnover, amino acid requirements and recommendations for athletes and active populations. *Brazilian Journal of Medical and Biological Research*, *45*(10), 875–890.

Popkin, B. M. (2006). Global nutrition dynamics: the world is shifting rapidly toward a diet linked with noncommunicable diseases. *The American Journal of Clinical Nutrition*, *84*(2), 289–298.

Popkin, B. M. (2015). Nutrition Transition and the Global Diabetes Epidemic. *Current Diabetes Reports*, *15*(9).

Popkin, B. M. (2017). Relationship between shifts in food system dynamics and acceleration of the global nutrition transition. *Nutrition Reviews*, *75*(2), 73–82.

Popkin, B. M., Adair, L. S., & Ng, S. W. (2012). Global nutrition transition and the pandemic of obesity in developing countries. *Nutrition Reviews*, *70*(1), 3–21.

Poslusna, K., Ruprich, J., De Vries, J. H. M., Jakubikova, M., & van't Veer, P. (2009). Misreporting of energy and micronutrient intake estimated by food records and 24 hour recalls, control and adjustment methods in practice. *British Journal of Nutrition*, *101*(Suppl 2), S73–S85.

Power, M. L., & Schulkin, J. (2008). Anticipatory physiological regulation in feeding biology: Cephalic phase responses. *Appetite*, *50*(2–3), 194–206.

Powley, T. L. (2000). Vagal circuitry mediating cephalic-phase responses to food. *Appetite*, *34*(2), 184–188.

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.

Provencher, V., Drapeau, V., Tremblay, A., Després, J.-P., Bouchard, C., & Lemieux, S. (2004). Eating behaviours, dietary profile and body composition according to dieting history in men and women of the Québec Family Study.

British Journal of Nutrition, *91*(06), 997.

Provencher, V., Drapeau, V., Tremblay, A., Després, J. P., & Lemieux, S. (2003). Eating behaviors and indexes of body composition in men and women from the Québec family study. *Obesity Research*, *11*(6), 783–792.

Pruessner, J. C., Kirschbaum, C., Meinlschmid, G., & Hellhammer, D. H. (2003). Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. *Psychoneuroendocrinology*, *28*(7), 916–931.

Qi, Q., Chu, A. Y., Kang, J. H., Huang, J., Rose, L. M., Jensen, M. K., … Qi, L. (2014). Fried food consumption, genetic risk, and body mass index: Gene-diet interaction analysis in three US cohort studies. *British Medical Journal*, *348*(March), 1–12.

Ralevski, E., Shanabrough, M., Newcomb, J., Gandelman, E., Hayden, R., Horvath, T. L., & Petrakis, I. (2018). Ghrelin is related to personality differences in reward sensitivity and impulsivity. *Alcohol and Alcoholism*, *53*(1), 52–56.

Rasmussen, E. B., Lawyer, S. R., & Reilly, W. (2010). Percent body fat is related to delay and probability discounting for food in humans. *Behavioural Processes*, *83*(1), 23–30.

Rasmussen, M., Krølner, R., Klepp, K. I., Lytle, L., Brug, J., Bere, E., & Due, P. (2006). Determinants of fruit and vegetable consumption among children and adolescents: A review of the literature. Part I: Quantitative studies. *International Journal of Behavioral Nutrition and Physical Activity*, *3*, 1–19.

Raubenheimer, D., & Jones, S. A. (2006). Nutritional imbalance in an extreme generalist omnivore: tolerance and recovery through complementary food selection. *Animal Behaviour*, *71*(6), 1253–1262.

Raubenheimer, D., Machovsky-Capuska, G. E., Gosby, A. K., & Simpson, S. J. (2015). Nutritional ecology of obesity: from humans to companion animals. *The British Journal of Nutrition*, *113*(2015), S26–S39.

Raubenheimer, David, & Simpson, S. J. (2018). Nutritional ecology and foraging theory. *Current Opinion in Insect Science*, *27*, 38–45.

Ravussin, E., Lillioja, S., Anderson, T. E., Christin, L., & Bogardus, C. (1986). Determinants of 24-hour energy expenditure in man. *The Journal of Clinical Investigation*, *78*(December), 1568–1578.

Redman, L. M., Kraus, W. E., Bhapkar, M., Das, S. K., Racette, S. B., Martin, C. K., … Study, C. (2014). Energy requirements in nonobese men and women : results from. *American Journal of Clinical Nutrition*, *99*(1), 71–78.

Reidpath, D. D., Burns, C., Garrard, J., Mahoney, M., & Townsend, M. (2002). An ecological study of the relationship between social and environmental determinants of obesity. *Health & Place*, *8*(2), 141–145.

Reily, N. M., Herman, C. P., & Vartanian, L. R. (2016). Portion-size preference as a function of individuals' body mass index. *Obesity Science & Practice*, *2*(3), 241–247.

Reynolds, B., Ortengren, A., Richards, J. B., & de Wit, H. (2006). Dimensions of impulsive behavior: Personality and behavioral measures. *Personality and Individual Differences*, *40*(2), 305–315.

Ribeiro, R. V., Hirani, V., Senior, A. M., Gosby, A. K., Cumming, R. G., Blyth, F. M., … Le Couteur, D. G. (2017). Diet quality and its implications on the cardio-metabolic, physical and general health of older men: The Concord Health and Ageing in Men Project (CHAMP). *British Journal of Nutrition*, *118*(2), 130–143.

Riva, M., Curtis, S., Gauvin, L., & Fagg, J. (2009). Unravelling the extent of inequalities in health across urban and rural areas: Evidence from a national sample in England. *Social Science and Medicine*, *68*(4), 654–663.

Rizi, E. P., Loh, T. P., Baig, S., Chhay, V., Huang, S., Quek, J. C., … Khoo, C. M. (2018). A high carbohydrate, but not fat or protein meal attenuates postprandial ghrelin, PYY and GLP-1 responses in Chinese men. *PLoS ONE*, *13*(1), 1–12.

Roberto, C. A., Swinburn, B., Hawkes, C., Huang, T. T. K., Costa, S. A., Ashe, M., … Brownell, K. D. (2015). Patchy progress on obesity prevention: Emerging examples, entrenched barriers, and new thinking. *The Lancet*, *385*(9985), 2400–2409.

Roberts, T. J., & Azain, M. J. (1997). Somatotropin Treatment Reduces Energy Intake without Altering Protein Intake in Pigs Selecting between High and Low Protein Diets. *The Journal of Nutrition*, *127*(10), 2047–2053.

Robinson, E., Kersbergen, I., Brunstrom, J. M., & Field, M. (2014). I'm watching you. Awareness that food consumption is being monitored is a demand characteristic in eating-behaviour experiments. *Appetite*, *83*, 19–25.

Rodriguez, N. N. R., Di Marco, N., Langley, S., & DiMarco, N. M. (2009). American College of Sports Medicine, American Dietetic Association, and Dietitians of Canada joint position statement: Nutrition and athletic performance. *Medicine and Science in Sports and Exercise*, *41*(3), 709–731.

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., Savage, J. S., & Birch, L. L. (2014). Measurement of food reinforcement in preschool children: Associations with food intake, BMI, and reward sensitivity. *Appetite*, *72*, 21–27.

Rolls, B. (2000). The role of energy density in overconsumption of fat. *Journal of Nutrition*, *130*, 272–275.

Rolls, B. J. (2014). What is the role of portion control in weight management. *International Journal of Obesity*, *38*(SUPPL. 1), S1–S8.

Rolls, B.J. (2018). The Role of Portion Size, Energy Density, and Variety in Obesity and Weight Management. In T. A. Wadden & G. A. Bray (Eds.), *Handbook of Obesity Treament* (2nd editio, pp. 93–104). New York: The Guildford Press.

Rolls, B.J., Engell, D., & Birch, L. L. (2000). Serving portion sizes influences 5-year-old but not 3-year-old children's food intakes. *Journal of the American Dietetic Association*, *100*(2), 232–234.

Rolls, B.J., Morris, E. L., & Roe, L. S. (2002). Portion size of food affects energy intake in normal-weight and overweight men and women. *American Journal of Clinical Nutrition*, *76*, 1207–1213.

Rolls, Barbara J., Roe, L. S., Kral, T. V. E., Meengs, J. S., & Wall, D. E. (2004). Increasing the portion size of a packaged snack increases energy intake in men and women. *Appetite*, *42*(1), 63–69.

Rolls, Barbara J., Roe, L. S., & Meengs, J. S. (2006). Larger portion sizes lead to a sustained increase in energy intake over 2 days. *Journal of the American Dietetic Association*, *106*(4), 543–549.

Rolls, Barbara J., Roe, L. S., Meengs, J. S., & Wall, D. E. (2004). Increasing the portion size of a sandwich increases energy intake. *Journal of the American Dietetic Association*, *104*(3), 367–372.

Rolls, Barbara J, Roe, L. S., & Meengs, J. S. (2007). The effect of large portion sizes on energy intake is sustained for 11 days. *Obesity (Silver Spring, Md.)*, *15*(6), 1535–1543.

Rolls, E. T. (2011). The Neural Representation Of Oral Texture Including Fat Texture. *Journal of Texture Studies*, *42*(2), 137–156.

Rolls, E. T. (2016). Reward Systems in the Brain and Nutrition. *Annual Review of Nutrition*, *36*(1), 435–470.

Romeike, K., Abidi, L., Lechner, L., De Vries, H., & Oenema, A. (2016). Similarities and differences in underlying beliefs of socio-cognitive factors related to diet and physical activity in lower-educated Dutch, Turkish, and Moroccan adults in the Netherlands: A focus group study. *BMC Public Health*, *16*(1), 1–15.

Romero, C. A., Compton, M. T., Yang, Y., & Snow, J. C. (2018). The real deal: Willingness-to-pay and satiety expectations are greater for real foods versus their images. *Cortex*, *107*(0296), 78–91.

Rönnecke, E., Vogel, M., Bussler, S., Grafe, N., Jurkutat, A., Schlingmann, M., … Kiess, W. (2019). Age- and Sex-Related Percentiles of Skinfold Thickness, Waist and Hip Circumference, Waist-to-Hip Ratio and Waist-to-Height Ratio: Results from a Population-Based Pediatric Cohort in Germany (LIFE Child). *Obesity Facts*, *12*(1), 25–39.

Rossi, M. A., & Stuber, G. D. (2018). Overlapping Brain Circuits for Homeostatic and Hedonic Feeding. *Cell Metabolism*, *27*(1), 42–56.

Roswall, N., Ängquist, L., Ahluwalia, T. S., Romaguera, D., Larsen, S. C., Østergaard, J. N., … Tjønneland, A. (2014). Association between Mediterranean and Nordic diet scores and changes in weight and waist circumference: Influence of FTO and TCF7L2 loci. *American Journal of Clinical Nutrition*, *100*(4), 1188–1197.

Rothbart, M. K., Evans, D. E., & Ahadi, S. A. (2000). Temperament and personality: Origins and outcomes. *Journal of Personality and Social Psychology*, *78*(1), 122–135.

Roux, Carel W. le, Aylwin, S. J. B., Batterham, R. L., Borg, C. M., Coyle, F., Prasad, V., … Bloom, S. R. (2006). Gut Hormone Profiles Following Bariatric Surgery Favor an Anorectic State, Facilitate Weight Loss, and Improve Metabolic Parameters. *Annals of Surgery*, *243*(1), 108–114.

Rozin, P. (2002). Human Food Intake and Choice : Biological , Psychological and Cultural Perspectives. In *Food selection from genes to culture* (pp. 7–26). Levallois-Perret, France, Danone Institute.

Rudenga, K. J., & Small, D. M. (2012). Amygdala response to sucrose consumption is inversely related to artificial sweetener use. *Appetite*, *58*(2), 504–507.

Ruegsegger, G. N., & Booth, F. W. (2018). Health Benefits of Exercise: The Facts. *Cold Spring Harbour Perspective Medicine, 8*(a029694), 1–15.

Rutters, F., Lemmens, S. G. T., Born, J. M., Bouwman, F., Nieuwenhuizen, A. G., Mariman, E., & Westerterp-Plantenga, M. S. (2010). Genetic associations with acute stress-related changes in eating in the absence of hunger. *Patient Education and Counseling*, *79*(3), 367–371.

Rydén, A., Sullivan, M., Torgerson, J. S., Karlsson, J., Lindroos, A. K., & Taft, C. (2003). Severe obesity and personality: A comparative controlled study of personality traits. *International Journal of Obesity*, *27*(12), 1534–1540.

Saelens, B. E., & Epstein, L. H. (1996). Reinforcing value of food in obese and non-obese women. *Appetite*, *27*, 41–50.

Salbe, A. D., DelParigi, A., Pratley, R. E., Drewnowski, A., & Tataranni, A. P. (2004). Taste preferences and body weight changes in an obesity-prone population. *American Journal of Clinical Nutrition*, *79*(3), 372–378.

Sample, C. H., Jones, S., Hargrave, S. L., Jarrad, L. E., & Davidson, T. L. (2016). Western Diet and the Weakening of the Interoceptive Stimulus Control of Appetitive Behavior. *Physiology & Behavior*, *312*(1), 219–230.

Sánchez-Villegas, A., Delgado-Rodríguez, M., Martínez-González, M. Á., de Irala-Estévez, J., Martínez, J. A., De la Fuente, C., … Willett, W. C. (2003). Gender, age, socio-demographic and lifestyle factors associated with major dietary patterns in the Spanish project SUN (Seguimiento Universidad de Navarra). *European Journal of Clinical Nutrition*, *57*(2), 285–292.

Savage, J. S., Hoffman, L., & Birch, L. L. (2009). Dieting, restraint, and disinhibition predict women's weight change over 6 y. *American Journal of Clinical Nutrition*, *90*(1), 33–40.

Scarborough, P., Burg, M. R., Foster, C., Swinburn, B., Sacks, G., Rayner, M., … Allender, S. (2011). Increased energy intake entirely accounts for increase in body weight in women but not in men in the UK between 1986 and 2000. *British Journal of Nutrition*, *105*(09), 1399–1404.

Schag, K., Schönleber, J., Teufel, M., Zipfel, S., & Giel, K. E. (2013). Food-related impulsivity in obesity and binge eating disorder: A systematic review. *Obesity Reviews : An Official Journal of the International Association for the Study of Obesity*, *14*(June), 477–495.

Scheid, J. L., Carr, K. A., Lin, H., Fletcher, K. D., Sucheston, L., Singh, P. K., … Epstein, L. H. (2014). FTO polymorphisms moderate the association of food reinforcement with energy intake. *Physiology and Behavior*, *132*, 51–56.

Scholtz, S., Miras, A. D., Chhina, N., Prechtl, C. G., Sleeth, M. L., Daud, N. M., … Goldstone, A. P. (2014). Obese patients after gastric bypass surgery have lower brain-hedonic responses to food than after gastric banding. *Gut*, *63*(6), 891–902.

Schubert, M. M., Sabapathy, S., Leveritt, M., & Desbrow, B. (2013). Acute Exercise and Hormones Related to Appetite Regulation: A Meta-Analysis. *Sports Medicine*, *44*(3), 387–403.

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), 1–18.

Schutz, Y., Kyle, U. U. G., & Pichard, C. (2002). Fat-free mass index and fat mass index percentiles in Caucasians aged 18-98y. *International Journal of Obesity*, *26*, 953–960.

Schwartz, M. W., Seeley, R. J., Zeltser, L. M., Drewnowski, A., Ravussin, E., Redman, L. M., & Leibel, R. L. (2017). Obesity Pathogenesis: An Endocrine Society Scientific Statement. *Endocrine Reviews*, *38*(September), 267–296.

Schwedhelm, C., Iqbal, K., Knüppel, S., Schwingshackl, L., & Boeing, H. (2018). Contribution to the understanding of how principal component analysis–derived dietary patterns emerge from habitual data on food consumption. *American Journal of Clinical Nutrition*, *107*(2), 227–235.

Sclafani, A. (1997). Learned controls of ingestive behaviour. *Appetite*, *29*(2), 153–158.

Scuteri, A., Sanna, S., Chen, W. M., Uda, M., Albai, G., Strait, J., … Abecasis, G. R. (2007). Genome-wide association scan shows genetic variants in the FTO gene are associated with obesity-related traits. *PLoS Genetics*, *3*(7), 1200–1210.

Serra, M. C., & Ryan, A. S. (2016). Resting energy expenditure: Determinants and Practical Applications for Athletes. In *Sports Medicine* (pp. 1–13). SMGroup.

Shi, H., & Clegg, D. J. (2009). Sex differences in the regulation of body weight. *Physiology and Behavior*, *97*(2), 199–204.

Shi, Haifei, Seeley, R. J., & Clegg, D. J. (2009). Sexual differences in the control of energy homeostasis. *Frontiers in Neuroendocrinology*, *30*(3), 396–404.

Shields, M., & Tremblay, M. S. (2008). Sedentary behaviour and obesity. *Health Reports*, *19*(2), 1–39.

Shimbara, T., Mondal, M. S., Kawagoe, T., Toshinai, K., Koda, S., Yamaguchi, H., … Nakazato, M. (2004). Central administration of ghrelin preferentially enhances fat ingestion. *Neuroscience Letters*, *369*(1), 75–79.

Shook, R. P., Hand, G. A., Drenowatz, C., Hebert, J. R., Paluch, A. E., Blundell, J. E., … Blair, S. N. (2015). Low levels of physical activity are associated with dysregulation of energy intake and fat mass gain over 1 year 1 , 2. *Am J Clin Nutr*, *102*(March), 1332–8.

Si Hassen, W., Castetbon, K., Péneau, S., Tichit, C., Nechba, A., Lampuré, A., … Méjean, C. (2018). Socio-economic and demographic factors associated with snacking behavior in a large sample of French adults. *International Journal of Behavioral Nutrition and Physical Activity*, *15*(1), 1–12.

Siep, N., Roefs, A., Roebroeck, A., Havermans, R., Bonte, M. L., & Jansen, A. (2009). Hunger is the best spice: An fMRI study of the effects of attention, hunger and calorie content on food reward processing in the amygdala and orbitofrontal cortex. *Behavioural Brain Research*, *198*(1), 149–158.

Sim, A. Y., Wallman, K. E., Fairchild, T. J., & Guelfi, K. J. (2015). Effects of high-intensity intermittent exercise training on appetite regulation. *Medicine and Science in Sports and Exercise*, *47*(11), 2441–2449.

Simchen, U., Koebnick, C., Hoyer, S., Issanchou, S., & Zunft, H.-J. (2006). Odour and taste sensitivity is associated with body weight and extent of misreporting of body weight. *European Journal of Clinical Nutrition*, *60*(6), 698–705.

Simonian, H. P., Kresge, K. M., Boden, G. H., & Parkman, H. P. (2005). Differential effects of sham feeding and meal ingestion on ghrelin and pancreatic polypeptide levels: evidence for vagal efferent stimulation mediating ghrelin release. *Neurogastroenterology and Motility*, *17*(3), 348–354.

Simpson, K. A., Martin, N. M., & Bloom, S. R. (2009). Hypothalamic regulation of food intake and clinical therapeutic applications. *Arquivos Brasileiros de Endocrinologia e Metabologia*, *53*(2), 120–128.

Simpson, S. J., Batley, R., & Raubenheimer, D. (2003). Geometric analysis of macronutrient intake in humans: The power of protein? *Appetite*, *41*(2), 123–140.

Simpson, S. J., Le Couteur, D. G., James, D. E., George, J., Gunton, J. E., Solon-Biet, S. M., & Raubenheimer, D. (2017). The Geometric Framework for Nutrition as a tool in precision medicine. *Nutrition and Healthy Aging*, *4*(3), 217–226.

Simpson, S. J., & Raubenheimer, D. (1997). Geometric analysis of macronutrient selection in the rat. *Appetite*, *28*(3), 201–213.

Simpson, S. J., & Raubenheimer, D. (2005). Obesity: The protein leverage hypothesis. *Obesity Reviews*, *6*(2), 133–142.

Simpson, S. J., & Raubenheimer, D. (2012). The geometry of human nutrition. In L. A. Baur, S. M. Twigg, & R. S. Magnusson (Eds.), *A modern epidemic: expert perspectives on obesity and diabetes* (pp. 20–45). Sydney University Press.

Sjöblad, S. (2019). Could the high consumption of high glycaemic index carbohydrates and sugars, associated with the nutritional transition to the

Western type of diet, be the common cause of the obesity epidemic and the worldwide increasing incidences of Type 1 and Type 2 d. *Medical Hypotheses*, *125*, 41–50.

Small, D. M., & DiFeliceantonio, A. G. (2019). Processed foods and food reward. *Science*, *363*(6425), 346–347.

Small, D. M., Gregory, M. D., Mak, Y. E., Gitelman, D., Mesulam, M. M., & Parrish, T. (2003). Dissociation of neural representation of intensity and affective valuation in human gustation. *Neuron*, *39*(4), 701–711.

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.

Small, D. M., Zatorre, R. J., Dagher, A., Evans, A. C., & Jones-gotman, M. (2001). Changes in brain activity related to eating chocolate. *Brain*, *124*, 1720–1733.

Smeets, A. J., Lejeune, M. P., & Westerterp-Plantenga, M. S. (2009). Effects of oral fat perception by modified sham feeding on energy expenditure, hormones and appetite profile in the postprandial state. *British Journal of Nutrition*, *101*(9), 1360–1368.

Smeets, P. A. M., Erkner, A., & De Graaf, C. (2010). Cephalic phase responses and appetite. *Nutrition Reviews*, *68*(11), 643–655.

Smith, C. F., Geiselman, P. J., Williamson, D. A., Champagne, C. M., Bray, G. A., & Ryan, D. H. (1998). Association of dietary restraint and disinhibition with eating behavior, body mass, and hunger. *Eating and Weight Disorders*, *3*(1), 7–15.

Snoek, H. M., Engels, R. C. M. E., van Strien, T., & Otten, R. (2013). Emotional, external and restrained eating behaviour and BMI trajectories in adolescence.

Appetite, *67*, 81–87.

Sobesky, J. L., Barrientos, R. M., De May, H. S., Thompson, B. M., Weber, M. D., Watkins, L. R., & Maier, S. F. (2014). High-fat diet consumption disrupts memory and primes elevations in hippocampal IL-1β, an effect that can be prevented with dietary reversal or IL-1 receptor antagonism. *Brain, Behavior, and Immunity*, *42*, 22–32.

Soenen, S., & Westerterp-Plantenga, M. S. (2008). Proteins and satiety: Implications for weight management. *Current Opinion in Clinical Nutrition and Metabolic Care*, *11*(6), 747–751.

Solon-Biet, S. M., Mitchell, S. J., Coogan, S. C. P., Cogger, V. C., Gokarn, R., McMahon, A. C., … Le Couteur, D. G. (2015). Dietary Protein to Carbohydrate Ratio and Caloric Restriction: Comparing Metabolic Outcomes in Mice. *Cell Reports*, *11*(10), 1529–1534.

Solon-Biet, S. M., McMahon, A. C., Ballard, J. W. O., Ruohonen, K., Wu, L. E., Cogger, V. C., … Simpson, S. J. (2014). The ratio of macronutrients, not caloric intake, dictates cardiometabolic health, aging, and longevity in ad libitum-fed mice. *Cell Metabolism*, *19*(3), 418–430.

Sørensen, A, Mayntz, D., Raubenheimer, D., & Simpson, S. (2008). Protein-leverage in Mice: The Geometry of Macronutrient Balancing and Consequences for Fat Deposition. *Obesity*, *16*, 566–571.

Sørensen, Allan, Mayntz, D., Raubenheimer, D., & Simpson, S. (2008). Protein-leverage in Mice: The Geometry of Macronutrient Balancing and Consequences for Fat Deposition. *Obesity*, *16*(3), 566–571.

Sørensen, L. B., Møller, P., Flint, A., Martens, M., & Raben, A. (2003). Effect of sensory perception of foods on appetite and food intake: a review of studies on humans. *International Journal of Obesity*, *27*, 1152–1166.

Sparti, A., Delany, J. P., De La Bretonne, J. A., Sander, G. E., & Bray, G. A. (1997). Relationship between resting metabolic rate and the composition of the fat-free mass. *Metabolism: Clinical and Experimental*, *46*(10), 1225–1230.

Speakman, J. R. (2015). The ‘Fat Mass and Obesity Related’ (FTO) gene: Mechanisms of Impact on Obesity and Energy Balance. *Current Obesity Reports*, *4*(1), 73–91.

Speakman, J. R., Rance, K. A., & Johnstone, A. M. (2008). Polymorphisms of the FTO gene are associated with variation in energy intake, but not energy expenditure. *Obesity*, *16*(8), 1961–1965.

Specter, S. E., & Drewnowski, A. (2004). Special Article Poverty and obesity: the role of energy density and energy costs. *American Journal of Clinical Nutrition*, *79*(1), 6–16.

Spence, M., Stancu, V., Dean, M., Livingstone, M. B. E., Gibney, E. R., & Lähteenmäki, L. (2016). Are food-related perceptions associated with meal portion size decisions? A cross-sectional study. *Appetite*, *103*, 377–385.

Spinella, M. (2007). Normative data and a short form of the Barratt Impulsiveness Scale. *The International Journal of Neuroscience*, *117*(3), 359–368.

Stafford, M., Cummins, S., Ellaway, A., Sacker, A., Wiggins, R. D., & Macintyre, S. (2007). Pathways to obesity: Identifying local, modifiable determinants of physical activity and diet. *Social Science and Medicine*, *65*(9), 1882–1897.

Stamatakis, E., Primatesta, P., Chinn, S., Rona, R., & Falascheti, E. (2005). Overweight and obesity trends from 1974 to 2003 in English children: What is the role of socioeconomic factors? *Archives of Disease in Childhood*, *90*(10), 999–1004.

Steinberg, G. R., Smith, A. C., Wormald, S., Malenfant, P., Collier, C., & Dyck, D. J. (2004). Endurance training partially reverses dietary-induced leptin resistance in rodent skeletal muscle. *American Journal of Physiology-Endocrinology and Metabolism*, *286*(1), E57–E63.

Stephens, D. N., Duka, T., Crombag, H. S., Cunningham, C. L., Heilig, M., & Crabbe, J. C. (2010). Reward sensitivity: Issues of measurement, and achieving consilience between human and animal phenotypes. *Addiction Biology*, *15*(2), 146–168.

Stephens, L. D., Crawford, D., Thornton, L., Olstad, D. L., Morgan, P. J., van Lenthe, F. J., & Ball, K. (2018). A qualitative study of the drivers of socioeconomic inequalities in men's eating behaviours. *BMC Public Health*, *18*(1), 1257.

Stevenson, R. J., Mahmut, M., & Rooney, K. (2015). Individual differences in the interoceptive states of hunger, fullness and thirst. *Appetite*, *95*, 44–57.

Stice, E., & Burger, K. (2019). Neural vulnerability factors for obesity. *Clinical Psychology Review*, *68*(October 2018), 38–53.

Stice, E., Burger, K. S., & Yokum, S. (2013a). Relative ability of fat and sugar tastes to activate reward, gustatory, and somatosensory regions. *American Journal of Clinical Nutrition*, *98*(6), 1377–1384.

Stice, E., Burger, K., & Yokum, S. (2013b). Caloric deprivation increases responsivity of attention and reward brain regions to intake, anticipated intake, and images of palatable foods. *NeuroImage*, *67*, 322–330.

Stice, E., Cameron, R. P., Killen, J. D., Hayward, C., & Taylor, C. B. (1999). Naturalistic weight-reduction efforts prospectively predict growth in relative weight and onset of obesity among female adolescents. *Journal of Consulting* *and Clinical Psychology*, *67*(6), 967–974.

Stice, E., Figlewicz, D. P., Gosnell, B. A., Levine, A. S., & Pratt, W. E. (2013). The contribution of brain reward circuits to the obesity epidemic. *Neuroscience and Biobehavioral Reviews*, *37*(9), 2047–2058.

Stice, E., Spoor, S., Ng, J., & Zald, D. H. (2009). Relation of obesity to consummatory and anticipatory food reward. *Physiology and Behavior*, *97*(5), 551–560.

Stice, E., Yokum, S., Blum, K., & Bohon, C. (2010). Weight gain is associated with reduced striatal response to palatable food. *Journal of Neuroscience*, *30*(39), 13105–13109.

Stinson, E., Piaggi, P., Ibrahim, M., Venti, C., Krakoff, J., & Votruba, S. (2018). High fat and sugar consumption during ad libitum intake predicts weight gain. *Obesity*, *26*(4), 689–695.

Stubbs, R. . J., Hughes, D. A., Johnstone, A. M., Whybrow, S., Horgan, G. W., King, N. A., & Blundell, J. E. (2004). Rate and extent of compensatory changes in energy intake and expenditure in response to altered exercise and diet composition in humans. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology*, *286*(2), R350-8.

Stubbs, R. J., & Finlayson, G. (2018). Improving laboratory studies of human eating behaviour: Energy balance and real-world considerations. *Appetite*, *130*(April), 334–335.

Stubbs, R. J., Harborn, C. G., Murgatroyd, P. R., & Prentice, A. M. (1995). Covert manipulation of dietary fat and energy density: effect on substrate flux and food intake in men eating ad libitum. *American Journal of Clinical Nutrition*, *62*, 316–329.

Stubbs, R. J., Hopkins, M., Finlayson, G., Duarte, C., Gibbons, C., & Blundell, J. E. (2018). Potential effects of fat mass and fat-free mass on energy intake in different states of energy balance. *European Journal of Clinical Nutrition*, *72*, 698–709.

Stuckler, D., McKee, M., Ebrahim, S., & Basu, S. (2012). Manufacturing epidemics: The role of global producers in increased consumption of unhealthy commodities including processed foods, alcohol, and tobacco. *PLoS Medicine*, *9*(6), 10.

Suchankova, P., Jerlhag, E., Jayaram-Lindström, N., Nilsson, S., Toren, K., Rosengren, A., … Franck, J. (2013). Genetic Variation of the Ghrelin Signalling System in Individuals with Amphetamine Dependence. *PLoS ONE*, *8*(4).

Sun, Y., Wang, P., Zheng, H., & Smith, R. G. (2004). Ghrelin stimulation of growth hormone release and appetite is mediated through the growth hormone secretagogue receptor. *Proceedings of the National Academy of Sciences*, *101*(13), 4679–4684.

Swinburn, B. A., Sacks, G., Hall, K. D., McPherson, K., Finegood, D. T., Moodie, M. L., & Gortmaker, S. L. (2011). The global obesity pandemic: Shaped by global drivers and local environments. *The Lancet*, *378*(9793), 804–814.

Swinburn, B. A., Sacks, G., & Ravussin, E. (2009). Increased food energy supply is more than sufficient to explain the US epidemic of obesity. *American Journal of Clinical Nutrition*, *90*, 1453–1456.

Tan, C. C., & Chow, C. M. (2014). Stress and emotional eating: The mediating role of eating dysregulation. *Personality and Individual Differences*, *66*, 1–4.

Tanaka, T., Ngwa, J. S., Van Rooij, F. J. A., Zillikens, M. C., Wojczynski, M. K., Frazier-Wood, A. C., … Nettleton, J. A. (2013). Genome-wide meta-analysis of observational studies shows common genetic variants associated with macronutrient intake. *American Journal of Clinical Nutrition*, *97*(6), 1395–1402.

Tanofsky-Kraff, M., Han, J. C., Anandalingam, K., Shomaker, L. B., Columbo, K. M., Wolkoff, L. E., … Yanovski, J. A. (2009). The FTO gene rs9939609 obesity-risk allele and loss of control over eating. *American Journal of Clinical Nutrition*, *90*, 1483–1488.

Tapper, K., Baker, L., Jiga-Boy, G., Haddock, G., Maio, G. R. (2014). Sensitivity to reward and punishment: Associations with diet, alcohol consumption, and smoking. *Personality and Individual Differences*, *72*, 79–84.

Tarnopolsky, M. (2004). Protein requirements for endurance athletes. *Nutrition*, *20*(7–8), 662–668.

Taveras, E. M., Berkey, C. S., Rifas-Shiman, S. L., Ludwig, D. S., Rockett, H. R. H., Field, A. E., … Gillman, M. W. (2005). Association of Consumption of Fried Food Away From Home With Body Mass Index and Diet Quality in Older Children and Adolescents. *Pediatrics*, *116*(4), e518–e524.

Taylor, V. H., Forhan, M., Vigod, S. N., McIntyre, R. S., & Morrison, K. M. (2013). The impact of obesity on quality of life. *Best Practice & Research Clinical Endocrinology & Metabolism*, *27*(2), 139–146.

Teff, K. L. (2010). Cephalic phase pancreatic polypeptide responses to liquid and solid stimuli in humans. *Physiology and Behavior*, *99*(3), 317–323.

Teff, K. L., Devine, J., & Engelman, K. (1995). Sweet taste: Effect on cephalic phase insulin release in men. *Physiology and Behavior*, *57*(6), 1089–1095.

Temple, J., Bulkley, a, Badawy, R., Krause, N., McCann, S., & Epstein, L. (2008).

Differential Effects of Daily Snack Food Consumption on Food Reinforcement in Obese and Non-Obese Women. *Obesity*, *16*, S67–S67.

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. *American Journal of Clinical Nutrition*, *87*(5), 1121–1127.

Tentolouris, N., Kokkinos, A., Tsigos, C., Kyriaki, D., Doupis, J., Raptis, S. A., & Katsilambros, N. (2004). Differential effects of high-fat and high-carbohydrate content isoenergetic meals on plasma active ghrelin concentrations in lean and obese women. *Hormone and Metabolic Research*, *36*(8), 559–563.

Tetley, A., Brunstrom, J., & Griffiths, P. (2009). Individual differences in food-cue reactivity. The role of BMI and everyday portion-size selections. *Appetite*, *52*(3), 614–620.

Thanarajah, S. E., Backes, H., DiFeliceantonio, A. G., Albus, K., Cremer, A. L., Hanssen, R., … Tittgemeyer, M. (2019). Food Intake Recruits Orosensory and Post-ingestive Dopaminergic Circuits to Affect Eating Desire in Humans. *Cell Metabolism*, *29*(3), 695-706.e4.

The Health and Social Care Information Centre (NHS Digital). (2018a). *National Child Measurement Programme, England - 2017/18 School Year*.

The Health and Social Care Information Centre (NHS Digital). (2018b). Statistics on obesity, physical activity and diet. In *National Office of Statistics* (Vol. 1).

The Lancet. (2011). Urgently needed: A framework convention for obesity control. *The Lancet*, *378*(9793), 741.

Timpson, N. J., Emmett, P. M., Frayling, T. M., Rogers, I., Hattersley, A. T., McCarthy, M. I., & Smith, G. D. (2008). The fat mass- and obesity-associated locus and dietary intake in children. *American Journal of Clinical Nutrition*, *88*(4), 971–978.

Togo, P., Osler, M., Sørensen, T. I. A., & Heitmann, B. L. (2004). A longitudinal study of food intake patterns and obesity in adult Danish men and women. *International Journal of Obesity*, *28*(4), 583–593.

Torrubia, R., Ávila, C., Moltó, J., & Caseras, X. (2001). The Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ) as a measure of Gray’s anxiety and impulsivity dimensions. *Personality and Individual Differences, 31*, 837–862.

Tremblay, A., & Bellisle, F. (2015). Nutrients, satiety, and control of energy intake. *Applied Physiology, Nutrition, and Metabolism*, *40*(10), 971–979.

Turrell, G., & Kavanagh, A. M. (2006). *Socio-economic pathways to diet : modelling the association between socio-economic position and food purchasing behaviour*. *9*(3), 375–383.

U.S. Department of Agriculture Center for Nutrition Policy and Promotion. (2007). Fruit and Vegetable Consumption by Older Americans. In *Nutrition Insight* (Vol. 34). http://www.cnpp.usda.gov/sites/default/files/nutrition_insights_uploads/Insight34.pdf. Accessed 30th September 2019

U.S. Department of Health and Human Services. (1996). CHAPTER 3: Physiologic Responses and Long-Term Adaptations to Exercise. In *Physical Activity and Health: A report of the Surgeon General* (Vol. 81, pp. 61–77). Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion.

Uher, R., Treasure, J., Heining, M., Brammer, M. J., & Campbell, I. C. (2006). Cerebral processing of food-related stimuli: Effects of fasting and gender. *Behavioural Brain Research*, *169*(1), 111–119.

United Nations. (2012). *Resolution adopted by the General Assembly on 19 September 2011. Political declaration of the high-level meeting of the general assembly on the prevention and control of non-communicable diseases.* https://www.un.org/en/ga/ncdmeeting2011/. Accessed 3rd August 2019

Urbano, C., Deglaire, A., Cartier-Lange, E., Herbreteau, V., Cordelle, S., & Schlich, P. (2016). Development of a sensory tool to assess overall liking for the fatty, salty and sweet sensations. *Food Quality and Preference*, *48*, 23–32.

Vainik, U., García-García, I., & Dagher, A. (2019). Uncontrolled eating: a unifying heritable trait linked with obesity, overeating, personality and the brain. *European Journal of Neuroscience,* (December 2018), 1–16.

Valassi, E., Scacchi, M., & Cavagnini, F. (2008). Neuroendocrine control of food intake. *Nutrition, Metabolism and Cardiovascular Diseases*, *18*(2), 158–168.

Valkauskaite, J., & Gibson, E. L. (2010). Microstructural analysis and personality predictors of overeating a high-fat food induced by sweetness. *Obesity*, S159.

Van den Berg, I., Franken, I. H. a, & Muris, P. (2010). A new scale for measuring reward responsiveness. *Frontiers in Psychology*, *1*, 1–7.

Van Der Klaauw, A. A., & Farooqi, I. S. (2015). The hunger genes: Pathways to obesity. *Cell*, *161*(1), 119–132.

Van Der Klaauw, A. A., Keogh, J. M., Henning, E., Trowse, V. M., Dhillo, W. S., Ghatei, M. A., & Farooqi, I. S. (2013). High protein intake stimulates postprandial GLP1 and PYY release. *Obesity*, *21*(8), 1602–1607.

Van Der Lely, A. J., Tschöp, M., Heiman, M. L., & Ghigo, E. (2004). Biological, physiological, pathophysiological, and pharmacological aspects of ghrelin. *Endocrine Reviews*, *25*(3), 426–457.

Van Dongen, M. V., Van Den Berg, M. C., Vink, N., Kok, F. J., & De Graaf, C. (2012). Taste-nutrient relationships in commonly consumed foods. *British Journal of Nutrition*, *108*(1), 140–147.

van Koningsbruggen, G. M., Stroebe, W., & Aarts, H. (2013). Successful restrained eating and trait impulsiveness. *Appetite*, *60*(1), 81–84.

Van Ree, J. M., Niesink, R. J. M., Van Wolfswinkel, L., Ramsey, N. F., Kornet, M. L. M. W., Van Furth, W. R., … Van den Berg, C. L. (2000). Endogenous opioids and reward. *European Journal of Pharmacology*, *405*(1–3), 89–101.

van Strien, T, Frijters, J., Bergers, G., & Defares, P. (1986). The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *International Journal of Eating Disorders*, *5*(2), 295–315.

van Strien, T, Peter Herman, C., & Verheijden, M. (2012). Eating style, overeating and weight gain. A prospective 2-year follow-up study in a representative Dutch sample. *Appetite*, *59*(3), 782–789.

van Strien, Tatjana, Herman, C. P., & Verheijden, M. W. (2009). Eating style, overeating, and overweight in a representative Dutch sample. Does external eating play a role? *Appetite*, *52*(2), 380–387.

van Strien, Tatjana, Konttinen, H., Homberg, J. R., Engels, R. C. M. E., & Winkens, L. H. H. (2016). Emotional eating as a mediator between depression and weight gain. *Appetite*, *100*, 216–224.

Van Strien, Tatjana, Roelofs, K., & De Weerth, C. (2013). Cortisol reactivity and distress-induced emotional eating. *Psychoneuroendocrinology*, *38*(5), 677–684.

Ventura, A. K., & Worobey, J. (2013). Early influences on the development of food preferences. *Current Biology*, *23*(9), R401–R408.

Verbeken, S., Braet, C., Lammertyn, J., Goossens, L., & Moens, E. (2012). How is reward sensitivity related to bodyweight in children? *Appetite, 58*(2), 478–483.

Verdich, C., Toubro, S., Buemann, B., Lysgård Madsen, J., Juul Holst, J., & Astrup, a. (2001). The role of postprandial releases of insulin and incretin hormones in meal-induced satiety--effect of obesity and weight reduction. *International Journal of Obesity and Related Metabolic Disorders : Journal of the International Association for the Study of Obesity*, *25*(8), 1206–1214.

Vieira, M. V., Del Ciampo, I. R. L., & Del Ciampo, L. A. (2014). Food consumption among healthy and overweight adolescents. *Journal of Human Growth and Development*, *24*(2), 157–162.

Villareal, D. T., Apovian, C. M., Kushner, R. F., & Klein, S. (2005). Obesity in older adults: Technical review and position statement of the American Society for Nutrition and NAASO, the Obesity Society. *Obesity Research*, *13*(11), 1849–1863.

Visscher, T. L. S., Viet, A. L., Kroesbergen, H. T., & Seidell, J. C. (2006). Underreporting of BMI in adults and its effect on obesity prevalence estimations in the period 1998 to 2001. *Obesity, 14*(11), 2054–2063.

Volkow, N.; 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.

Wåhlén, K., Sjölin, E., & Hoffstedt, J. (2008). The common rs9939609 gene variant of the fat mass- and obesity-associated gene FTO is related to fat cell lipolysis. *Journal of Lipid Research*, *49*(3), 607–611.

Wakimoto, P., & Block, G. (2011). Dietary Intake, Dietary Patterns, and Changes With Age: An Epidemiological Perspective. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, *56*(Supplement 2), 65–80.

Walker, R. E., Keane, C. R., & Burke, J. G. (2010). Disparities and access to healthy food in the United States: A review of food deserts literature. *Health and Place*, *16*(5), 876–884.

Wallis, D. J., & Hetherington, M. M. (2004). Stress and eating: The effects of ego-threat and cognitive demand on food intake in restrained and emotional eaters. *Appetite*, *43*(1), 39–46.

Wang, Y. C., McPherson, K., Marsh, T., Gortmaker, S. L., & Brown, M. (2011). Health and economic burden of the projected obesity trends in the USA and the UK. *The Lancet*, *378*(9793), 815–825.

Wang, Y., Dong, D., Todd, J., Du, J., Yang, Z., Lu, H., & Chen, H. (2016). Neural correlates of restrained eaters' high susceptibility to food cues: An fMRI study. *Neuroscience Letters*, *631*, 56–62.

Wansink, B., & Kim, J. (2005). Bad Popcorn in Big Buckets: Portion Size Can Influence Intake as Much as Taste. *Journal of Nutrition Education and Behavior*, *37*, 242–245.

Wansink, B. (2000). At the Movies: How External Cues and Perceived Taste Impact Consumption Volume. *Food Quality and Preference*, *12*(1), 69–74.

Wansink, Brian, Cheney, M. M., & Chan, N. (2003). Exploring comfort food preferences across age and gender. *Physiology and Behavior*, *79*(4–5), 739–747.

Ward, A., & Mann, T. (2000). Don't Mind If I Do: Disinhibited eating under cognitive load. *Journal of Personality and Social Psychology*, *78*(4), 753–763.

Wardle, J., & Gibson, E. L. (2016). Interactions with Emotions and Behavior. In F. George (Ed.), *Stress: Concepts, Cognition, Emotion, and Behavior*: *Vol. 1 of the H* (pp. 435–443). Academic Press, Burlington.

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.

Wardle, J., Llewellyn, C., Sanderson, S., & Plomin, R. (2009). The FTO gene and measured food intake in children. *International Journal of Obesity*, *33*(1), 42–45.

Wardle, J., Waller, J., & Jarvis, M. J. (2002). Sex Differences in the Association of Socioconomic Status With Obesity. *Research and Practice*, *92*(8), 1299–1304.

Wardle, J, Guthrie, C., Sanderson, S., Birch, L., & Plomin, R. (2001). Food and activity preferences in children of lean and obese parents. *International Journal of Obesity*, *25*(7), 971–977.

Wardle, Jane, Carnell, S., Haworth, C. M. A. a, Farooqi, I. S., O'Rahilly, S., & Plomin, R. (2008). Obesity associated genetic variation in FTO is associated with diminished satiety. *Journal of Clinical Endocrinology and Metabolism*, *93*(9), 3640–3643.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of Personality and Social Psychology*, *54*(6), 1063–1070.

Weafer, J., Burkhardt, A., & de Wit, H. (2014). Sweet taste liking is associated with impulsive behaviors in humans. *Frontiers in Behavioral Neuroscience, 8*(228), 1–6.

Webb, K. L., Scholfield, W. N., Lazarus, R., Smith, W., Mitchell, P., & Leeder, S. R.

(1999). Prevalence and predictors of dietary supplement use in an older Australian population. *Australian Journal of Nutrition & Dietetics*, *56*(2), 69-75 7p.

Wei, X. J., Sun, B., Chen, K., Lv, B., Luo, X., & Yan, J. Q. (2015). Ghrelin signaling in the ventral tegmental area mediates both reward-based feeding and fasting-induced hyperphagia on high-fat diet. *Neuroscience*, *300*, 53–62.

Weigle, D. S., Breen, P. A., Matthys, C. C., Callahan, H. S., Meeuws, K. E., Burden, V. R., & Purnell, J. Q. (2005). A high-protein diet induces sustained reductions in appetite, ad libitum caloric intake, and body weight despite compensatory changes in diurnal plasma leptin and ghrelin concentrations. *American Journal of Clinical Nutrition*, *82*(1), 41–48.

Weinstein, S. E., Shide, D. J., & Rolls, B. J. (1997). Changes in food intake in response to stress in men and women: psychological factors. *Appetite*, *28*(1), 7–18.

Weise, C. M., Hohenadel, M. G., Krakoff, J., & Votruba, S. B. (2014). Body composition and energy expenditure predict ad-libitum food and macronutrient intake in humans. *International Journal of Obesity*, *38*(2), 243–251.

Weller, R. E., Cook, E. W., Avsar, K. B., & Cox, J. E. (2008). Obese women show greater delay discounting than healthy-weight women. *Appetite*, *51*(3), 563–569.

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.

Westerterp-Plantenga, M. S., Lemmens, S. G., & Westerterp, K. R. (2012). Dietary protein - its role in satiety, energetics, weight loss and health. *The British*

Journal of Nutrition, *108*, S105–S112.

Westerterp, K. R., & Speakman, J. R. (2008). Physical activity energy expenditure has not declined since the 1980s and matches energy expenditures of wild mammals. *International Journal of Obesity*, *32*(8), 1256–1263.

Whichelow, M. J., & Prevost, A. T. (1996). Dietary patterns and their associations with demographic, lifestyle and health variables in a random sample of British adults. *British Journal of Nutrition*, *76*, 17–30.

White, B. D., Porter, M. H., & Martin, R. J. (2000a). Effects of age on the feeding response to moderately low dietary protein in rats. *Physiology and Behavior*, *68*(5), 673–681.

White, B. D., Porter, M. H., & Martin, R. J. (2000b). Protein selection, food intake, and body composition in response to the amount of dietary protein. *Physiology and Behavior*, *69*(4–5), 383–389.

Whiteside, S.P., Lynam, D. R., Miller, J. D., & Reynolds, S. K. (2005). Validation of the UPPS impulsive behaviour scale: a four-factor model of impulsivity. *European Journal of Personality*, *19*(7), 559.

Whiteside, Stephen P., & Lynam, D. R. (2001). The five factor model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality and Individual Differences, 30*(4), 669–689.

Wiig Dammann, K., & Smith, C. (2009). Factors Affecting Low-income Women's Food Choices and the Perceived Impact of Dietary Intake and Socioeconomic Status on Their Health and Weight. *Journal of Nutrition Education and Behavior*, *41*(4), 242–253.

Wilkinson, L. L., Hinton, E. C., Fay, S. H., Ferriday, D., Rogers, P. J., & Brunstrom, J. M. (2012). Computer-based assessments of expected satiety predict behavioural measures of portion-size selection and food intake. *Appetite*, *59*(3), 933–938.

Williamson, D. A., Lawson, O. J., Brooks, E. R., Wozniak, P. J., Ryan, D. H., Bray, G. A., & Duchmann, E. G. (1995). Association of body mass with dietary restraint and disinhibition. *Appetite*, *25*(1), 31–41.

Wise, R. A. (2006). Role of brain dopamine in food reward and reinforcement. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *361*(1471), 1149–1158.

Woods, S.C., & Ramsay, D. S. (2011). Food intake, metabolism and homeostasis. *Physiology & Behavior*, *104*(1), 4–7.

Woods, Stephen C., May-Zhang, A. A., & Begg, D. P. (2018). How and why do gastrointestinal peptides influence food intake? *Physiology & Behavior*, *193*, 218–222.

Woods, Stephen C, Seeley, R. J., Rushing, P. A., D'Alessio, D., & Tso, P. (2003). A controlled high-fat diet induces an obese syndrome in rats. *The Journal of Nutrition*, *133*(4), 1081–1087.

Woodward, H. E., Treat, T. A., Cameron, C. D., & Yegorova, V. (2017). Valence and arousal-based affective evaluations of foods. *Eating Behaviors*, *24*, 26–33.

World Health Organisation. (2017). Report of the Commission on Ending Childhood Obesity: implementation plan. In *World Health Organization*. Geneva.

World Health Organisation. (2019). Fact Sheets: Obesity and overweight.

Wortley, K. E., Chang, G.-Q., Davydova, Z., & Leibowitz, S. F. (2003). Peptides that regulate food intake: orexin gene expression is increased during states of hypertriglyceridemia. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, *284*(6), R1454-65.

Wouters, S., Jacobs, N., Duif, M., Lechner, L., & Thewissen, V. (2017). Affect and between-meal snacking in daily life: the moderating role of gender and age. *Psychology & Health*, *0446*, 1–18.

Wren, A. M., & Bloom, S. R. (2007). Gut hormones and appetite control. *Gastroenterology*, *132*(6), 2116–2130.

Wren, A. M., Seal, L. J., Cohen, M. A., Brynes, A. E., Frost, G. S., Murphy, K. G., … Bloom, S. R. (2001). Ghrelin enhances appetite and increases food intake in humans. *Journal of Clinical Endocrinology and Metabolism*, *86*(12), 5992–5995.

Wu, G. (2009). Amino acids: Metabolism, functions, and nutrition. *Amino Acids*, *37*(1), 1–17.

Wynne, K. (2005). Appetite control. *Journal of Endocrinology*, *184*(2), 291–318.

Wysokiński, A., Sobów, T., Kłoszewska, I., & Kostka, T. (2015). Mechanisms of the anorexia of aging—a review. *Age*, *37*(4), 81.

Yancy, W. S., Wang, C. C., & MacIejewski, M. L. (2013). Trends in energy and macronutrient intakes by weight status over four decades. *Public Health Nutrition*, *17*(2), 256–265.

Yantcheva, B., & Brindal, E. (2013). How much does what you eat matter? The potential role of meal size, fat content, and gender on ratings of desirability. *Eating Behaviors*, *14*(3), 285–290.

Yaribeygi, H., Atkin, S. L., Simental-Mendía, L. E., & Sahebkar, A. (2019). Molecular mechanisms by which aerobic exercise induces insulin sensitivity. *Journal of Cellular Physiology*, Vol. 234, pp. 12385–12392. Wiley-Liss Inc.

Yengo, L., Sidorenko, J., Kemper, K. E., Zheng, Z., Wood, A. R., Weedon, M. N., … Visscher, P. M. (2018). Meta-analysis of genome-wide association studies for height and body mass index in ~700 000 individuals of European ancestry. *Human Molecular Genetics*, *27*(20), 3641–3649.

Yeo, G. S. H., & O'Rahilly, S. (2012). Uncovering the biology of FTO. *Molecular Metabolism*, *1*(1–2), 32–36.

Yeomans, M. R. (1996). Palatability and the micro-structure of feeding in humans: the appetizer effect. *Appetite*, *27*(2), 119–133.

Yeomans, M. R. (2000). Rating changes over the course of meals: What do they tell us about motivation to eat? *Neuroscience and Biobehavioral Reviews*, *24*(2), 249–259.

Yeomans, M. R., Tovey, H. M., Tinley, E. M., & Haynes, C. J. (2004). Effects of manipulated palatability on appetite depend on restraint and disinhibition scores from the Three-Factor Eating Questionnaire. *International Journal of Obesity*, *28*(1), 144–151.

Yeomans, M.R. (2017). Adverse effects of consuming high fat–sugar diets on cognition: implications for understanding obesity. *Proceedings of the Nutrition Society*, (December 2016), 1–11.

Yeomans, M.R., Blundell, J. E., & Leshem, M. (2004). Palatability: response to nutritional need or need-free stimulation of appetite? *The British Journal of Nutrition*, *92 Suppl 1*(August 2015), S3–S14.

Yeomans, M.R., Gould, N. J., Leitch, M., & Mobini, S. (2009). Effects of energy density and portion size on development of acquired flavour liking and learned satiety. *Appetite*, *52*(2), 469–478.

Yeomans, M.R., & Gray, R. W. (1997). Effects of naltrexone on food intake and changes in subjective appetite during eating: Evidence for opioid involvement in the appetizer effect. *Physiology and Behavior*, *62*(1), 15–21.

Yeomans, M.R., Gray, R. W., Mitchell, C. J., & True, S. (1997). Independent effects of palatability and within-meal pauses on intake and appetite ratings in human volunteers. *Appetite*, *29*(1), 61–76.

Yeomans, M.R., Lee, M. D., Gray, R. W., & French, S. J. (2001). Effects of test-meal palatability on compensatory eating following disguised fat and carbohydrate preloads. *International Journal of Obesity*, *25*, 1215–1224.

Yeomans, M.R., Leitch, M., & Mobini, S. (2008). Impulsivity is associated with the disinhibition but not restraint factor from the Three Factor Eating Questionnaire. *Appetite*, *50*(2–3), 469–476.

Yeomans, M.R., Tepper, B. J., Rietzschel, J., & Prescott, J. (2007). Human hedonic responses to sweetness: Role of taste genetics and anatomy. *Physiology and Behavior*, *91*(2–3), 264–273.

Yeomans, M.R., Weinberg, L., & James, S. (2005). Effects of palatability and learned satiety on energy density influences on breakfast intake in humans. *Physiology and Behavior*, *86*(4), 487–499.

Yeomans, Martin R., Prescott, J., & Gould, N. J. (2009). Acquired hedonic and sensory characteristics of odours: Influence of sweet liker and propylthiouracil taster status. *Quarterly Journal of Experimental Psychology*, *62*(8), 1648–1664.

Young, L. R., & Nestle, M. (2002). The contribution of expanding portion sizes to the US obesity epidemic. *American Journal of Public Health*, *92*(2), 246–249.

Young, L. R., & Nestle, M. (2003). Expanding portion sizes in the US marketplace: Implications for nutrition counseling. *Journal of the American Dietetic Association*, *103*(2), 231–240.

Young, L. R., & Nestle, M. (2012). Reducing portion sizes to prevent obesity: A call to action. *American Journal of Preventive Medicine*, *43*(5), 565–568.

Yu, Z. M., DeClercq, V., Cui, Y., Forbes, C., Grandy, S., Keats, M., … Dummer, T. J. B. (2018). Fruit and vegetable intake and body adiposity among populations in Eastern Canada: the Atlantic Partnership for Tomorrow's Health Study. *BMJ Open*, *8*(4), e018060.

Zhang, M., Balmadrid, C., & Kelley, A. E. (2003). Nucleus accumbens opioid, GABAergic, and dopaminergic modulation of palatable food motivation: Contrasting effects revealed by a progressive ratio study in the rat. *Behavioral Neuroscience*, *117*(2), 202–211.

Zheng, H., Lenard, N., Shin, A., & Berthoud, H. R. (2009). Appetite control and energy balance regulation in the modern world: Reward-driven brain overrides repletion signals. *International Journal of Obesity*, *33*(2), S8-13.

Zhou, Y., Gao, X., Chen, H., & Kong, F. (2017). High-disinhibition restrained eaters are disinhibited by self-regulatory depletion in the food-related inhibitory control. *Eating Behaviors*, *26*, 70–75.

Zhu, Y., Hsu, W. H., & Hollis, J. H. (2014). Modified sham feeding of foods with different macronutrient compositions differentially influences cephalic change of insulin, ghrelin, and NMR-based metabolomic profiles. *Physiology and Behavior*, *135*, 135–142.

Zigman, J. M., Jones, J. E., Lee, C. E., Saper, C. B., & Elmquist, J. K. (2006). Expression of Ghrelin Receptor mRNA in the Rat and the Mouse Brain. *Journal of Comparative Neurology2*, *20*(494(3)), 528–548.

Zizza, C., Siega-Riz, A. M., & Popkin, B. M. (2001). Significant Increase in Young Adults' Snacking between 1977–1978 and 1994–1996 Represents a Cause for Concern! Significant Increase in Young Adults' Snacking between 1977–1978 and 1994–1996 Represents a Cause for Concern! *Preventive Medicine, 32*, 303–

310.

Zlatevska, N., Dubelaar, C., & Holden, S. S. (2014). Sizing Up the Effect of Portion Size on Consumption: A Meta-Analytic Review. *Journal of Marketing*, *78*(3), 140–154.

Zuraikat, F. M., Roe, L. S., Smethers, A. D., Reihart, L. W., & Rolls, B. J. (2018). Does the cost of a meal influence the portion size effect? *Appetite*, *127*(January), 341–348.